SOCIEDAD MEXICANA
DE INMUNOLOGÍA
En la lucha contra las enfermedades
infecciosas, autoinmunes, alergias y el cáncer

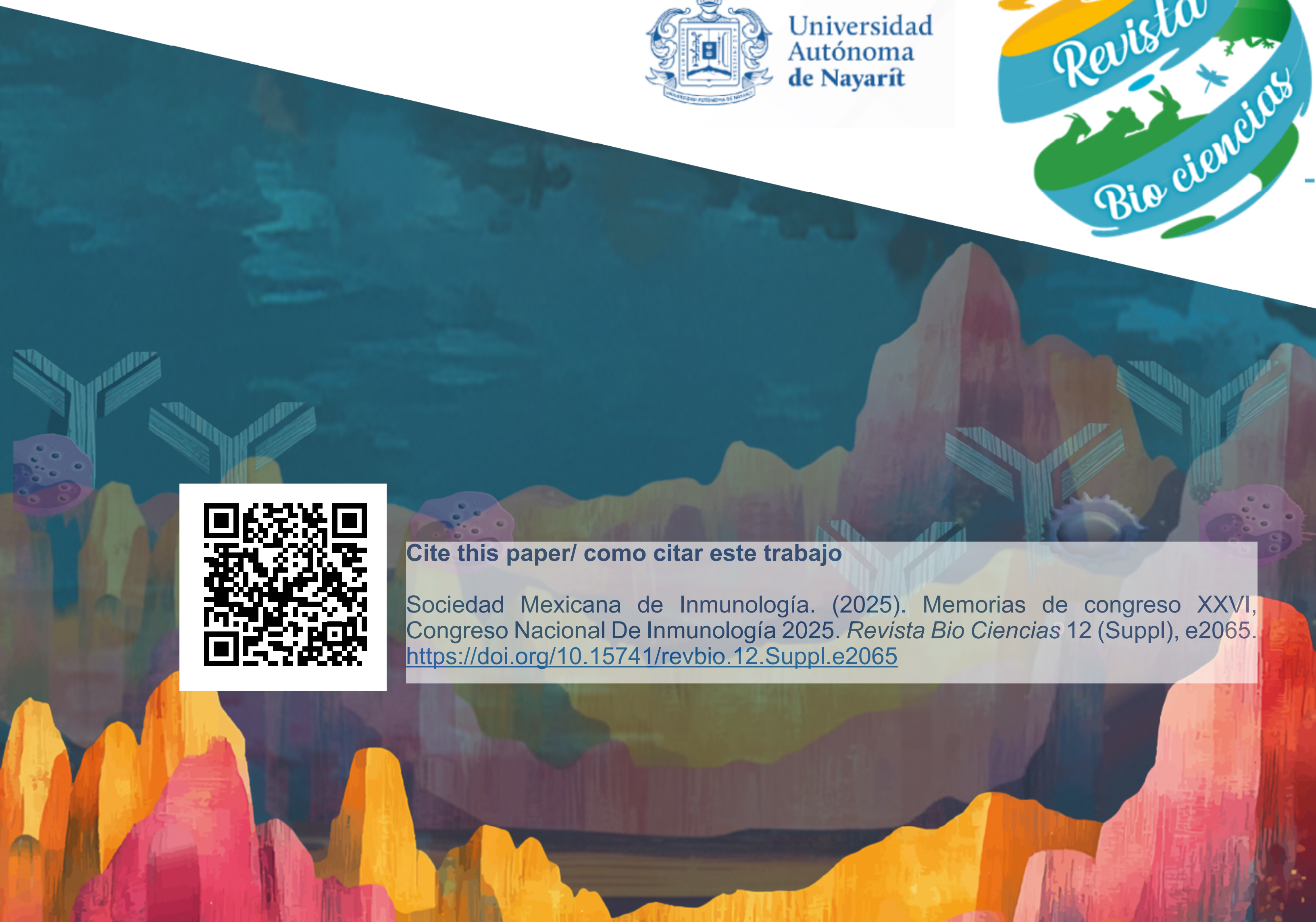
Memorias de Congreso

XXVI CONGRESO DE INMUNOLOGÍA

Chihuahua, Chihuahua, 2025

Universidad
Autónoma
de Nayarit

Revista Bio ciencias



Cite this paper/ como citar este trabajo

Sociedad Mexicana de Inmunología. (2025). Memorias de congreso XXVI, Congreso Nacional De Inmunología 2025. *Revista Bio Ciencias* 12 (Suppl), e2065. https://doi.org/10.15741/revbio.12.Suppl.e2065

TABLA DE CONTENIDO

En orden alfabético según el primer autor o autor corresponsal.

A - B - C - D - E - F - G - H - I - J- K - L - M - N - O - P - Q - R - S - T - U

V -W-Y- Z.

A

Acevedo-Domínguez *et al*

Betaglycan promotes the in vitro induction of regulatory T cells with diminished suppressive function. 38

Aguilar Fuentes *et al*

Development Of CAR.ME.LA For The Treatment Of Patients With Acute Lymphoblastic Leukemia 39

Aguilar-Garduño *et al*

Alternatively activated macrophages: A promising therapy on dry eye disease 40

Aguilar-González *et al*

Exercise reduces the risk of developing periodontal disease caused by aging 41

Albarrán-Godínez, *et al*

Dietary antigens gradually inactivate CD4+ T cells to produce oral tolerance 42

Alemán-Navarro *et al*

CD43 impacts the immunopathology of tuberculosis according to the host's inflammatory phenotype 43

Alva-Murillo *et al*

Immune response of Zophoba morio larvae exposed to polyethylene terephthalate (PET) microplastics 44

Álvarez-Carrasco *et al*

Immunomodulation of P-glycoprotein to Enhance Natural Killer Cell Survival in Acute Lymphoblastic Leukemia 45

Álvarez-Fosado *et al*

Discovery and Functional Characterization of Anti-TNFα Antibodies from Murine Immune Libraries 46

Ángeles-Floriano *et al*

Inflammation and endoplasmic reticulum stress association in bone marrow of pediatric acute lymphoblastic leukemia (B-ALL) patients 47

Antonio-Buendia & Santos-Argumedo

Myo1g controls B–T cell conjugate dynamics in antigen presentation. 48

Antonio-Mendoza *et al*

Characterization of the immunoglobulin lambda chain locus in native Mexican populations 49

Aragón-Franco *et al*

Intratumoral injection EST of the human parasite T. canis increase lung metastasis 50

Aranda-Nieto *et al*

Cytotoxic and Immunomodulatory Effects of the Immunopotentiator RTLV-1424 on HER2+ Breast Cancer Cells 51

Immunomodulatory and Cytotoxic Effect of Immunostimulant RTLV-1424 over Luminal Breast Cancer Cells 52

Aranda -Uribe *et al*

Association of SNVs in CTLA-4 and Galectin-3 in SLE in Southeastern Mexico. 53

Arce -Aceves *et al*

Effect of a defensin (Def1.3) from Chagas disease vector against Trypanosomatids 54

Arce-Mendoza *et al*

A novel methodology for NETs visualization under light microscopy 55

Arias-Díaz *et al*

Analysis of microplastic contamination in the grasshoper (Sphenarium purpurascens) and exploration of its effects on the immune response 56

Arroyo-Olarte *et al*

STAT6 Inhibition Promotes iTreg Stability and Therapeutic Efficacy in Inflammatory Bowel Disease 57

Arteaga-Cruz *et al*

Molecular characterization of expanded allospecific Tr1 cells with therapeutic potential in transplantation 58

Autran-Martínez *et al*

Development and validation of immunoassays for the characterization of IgG’s and IgA’s in dairy cattle 59

Ávila-Estrada *et al*

Modulation of immunological gene expression in sheep supplemented with a polyherbal additive 60

Ayala-Rodríguez *et al*

Evaluation of B10 regulatory cells in idiopathic inflammatory myopathies. 61

B

Báez-Magaña *et al*

Evaluation of candidate inducers of the trained immune response in bovine PBMC's 62

Báez-Solano *et al*

Potential Roles of FAM3A, SPG7, and MDH1 in Asthma Mitochondrial Regulation 63

Banda-Morales *et al*

Transcriptomic meta-analysis IL7R/STAT3 Axis Dysregulation in Pediatric Multiple Sclerosis 64

Barraza-Ramos *et al*

Stage-specific immune remodeling in progressive cutaneous lupus 65

Barrera-Osorio *et al*

Impact of Obesity on IgG and IgA Production in Colostrum of Female Mice 66

Bautista -Jacobo *et al*

Transient Effects of Cry1Ac Toxin and Protoxin on Intestinal Barrier Integrity via MAPK Signaling in a Caco-2 Cell Monolayer 67

Bautista-Sebastián *et al*

Interaction between MSCs with tumor cells in cervical cancer favours macrophage polarization 68

Becerril-García *et al*

Pathophysiological dynamics during neonatal murine neurocandidiasis 69

Beltrán-Calderón *et al*

MSC protect leukaemia cells from elimination by PTL and DMAPT favouring inflammation. 70

Beltrán-López *et al*

Identification of immunogenic P. gingivalis proteins using a murine immunochemical approach 71

Benítez-Guzmán *et al*

Bovine Viral Diarrhea Virus interaction prediction with caspase 1 support to viral replication 72

Borges-García & Rodríguez-Sanoja

Intranasally administered SMPs change the immune landscape in a TNBC mouse model 73

Borrego-Moreno *et al*

Identify the SARS-CoV-2 viral load in rt-PCR could prevent death in COVID-19 74

Bravo -Ramírez *et al*

CD39 and CD73 are key enzymes in the adenosine (ADO) pathway 75

Briones-Bautista *et al*

Standardization of an ELISA to identify anti-Actinomadura madurae IgM and IgG antibodies 76

C

Cabral-Venegas *et al*

Design of Antimicrobial Peptides Using Artificial Intelligence and Their Characterization by Surface Plasmon Resonance 77

Calitl-Flores *et al*

Generation and validation by ELISA of blocking the anti-ubiquitin scFv C1-Fc tandem 78

Campos López *et al*

Interaction between vitamin D SNVs with rheumatoid arthritis and hypovitaminosis D risk 79

Cantera-Bravo *et al*

Bovine Viral Diarrhea Virus Activates the NLRP3 Inflammasome Through Calcium Mobilization. 80

Caraveo-Centeno *et al*

Effect of prostaglandin inhibition on DENV replication in Anopheles albimanus 81

Caraza-Sánchez *et al*

Inmunophenotyping as a tool for classification and monitoring of MASLD in Veracruz population 82

Carballo-Uicab *et al*

Neutralization dose-response in sera from mice immunized with the AVX/COVID-12 vectorized vaccine 83

Cárdenas-Juárez *et al*

Expression of FOXP3 in B-Cell Subtypes (CD19+CD39+, CD19+CD39-) and Their Regulatory Function 84

Cardoso-Jaime & Dimopoulos

Non-canonical functions of Anopheles gambiae hemocytes during Plasmodium falciparum Infections. **85**

Cardoso-Jaime *et al*

New Insights into Antiviral Immune Response of Aedes aegypti Hemocytes (Macrophage-like cells) **86**

Carranza-Salazar *et al*

Implementation an efficient biobank in a research laboratory: ISO 9001:2015 certified **87**

The effect of Mycobacterium tuberculosis-infected macrophages in the development of lung cancer **88**

Carrasco-Ramírez *et al*

Early Development of Cancer During Acute Terminal Infection in Experimental Malaria. **89**

Carreón -Cortés *et al*

Effect of L-arginine and L-serine supplementation on neonatal T lymphocyte function **90**

Carrillo-Carlos *et al*

Exploring the role of SPN-205: a nonsense-mediated decay targeted CD43 isoform **91**

Carvajal-Ruiz *et al*

Characterization of Immune Populations in Gastric Mucosa during HIV/Helicobacter pylori Coinfection **92**

Castañeda Casimiro *et al*

Study of the humoral response in patients with active pulmonary tuberculosis against mycobacterial antigens **93**

Study of biological effect of extracellular vesicles from neutrophils during bacterial growth **94**

Castillo -Cruz *et al*

Autophagy regulates Nakaseomyces glabrata clearance in human THP-1 macrophages **95**

Castillo-Gálvez *et al*

Evaluation of miRNA expression in macrophages infected in vitro with Nocardia brasiliensis **96**

Castillo-Sánchez *et al*

Cytokines and Chemokines involved in the upregulation of CXCR4 in resting lymphocytes **97**

Castillo-Velázquez *et al*

Identification of microRNA drug targets for Alzheimer's and Diabetes Mellitus by network medicine. **98**

Drug repositioning of potential GPR15 inhibitors by molecular docking identifies effects on the signaling pathway and cell migration mediated by GPR15-GPR15L. **99**

Castillo-Vera *et al*

Altered CD4+ and CD8+ T-cell Phenotype in HIV+ Individuals Convalescent from COVID-19 **100**

Castrejón -Jiménez *et al*

Effect of Polysaccharide-Modified Hybrid Materials on the viability of macrophages **101**

Cázares-Preciado *et al*

Evaluation of the impact of culture media during DMSO neutrophilic differentiation of HL-60 cells **102**

Cedillo -Baños *et al*

Unlocking the Potential of Neonatal CD4+ T Cells with Cytokine Therapy **103**

Ceja -Garcia *et al*

Descubrimiento de un panel de anticuerpos de dominio único (SDAB) anti-PD-1 **104**

Cenobio-Hernández *et al*

Implementation of a platform for CAR-T cell manufacture and evaluation **105**

Cervantes-Candelas & Legorreta-Herrera

Tamoxifen induces sexually dimorphic patterns in the immune response to malaria **106**

Chacón-Salinas *et al*

BTK Inhibition by Valproic Acid: In Silico approach for Allergy Treatment **107**

Chavarría-Bencomo *et al*

Antibodies responses against SARS-CoV-2 vaccines in teachers and students from Chihuahua, Mexico. **108**

Chávez-Mireles *et al*

Association of PD-1/PD-L Axis in cTfh, Tph, and atBm cells in SLE. **109**

Chipres-Naranjo *et al*

The CD43 sialomucin promotes GLUT-1 expression in T cells 110

Chiyean-Acosta *et al*

Immunological profile of Leishmania mexicana and Brichromyia olmeca in inhabitants of Yucatan 111

Cocoletzi -Bautista *et al*

Transcriptional regulation of CCR6 by HIF-1 and its implication in pediatric Medulloblastoma 112

Consuegra-Sierra *et al*

Establishment of a Murine Model of B-ALL for the Preclinical Evaluation of CAR-T Cells 113

Contreras-Briones *et al*

Elevated HIF-1α correlates with mitochondrial activity in CD14+IRF8+ monocytes and exhausted CD8+PD1+ T-lymphocyte phenotype in breast cancer patients 114

Cordero-Ortiz *et al*

Production and isolation of porcine monoclonal antibodies from single B cells 115

Correa Becerril *et al*

Role of CCL28 in the development of colorectal cancer associated with colitis. 116

Covantes-Rosales *et al*

nAChR-α7 in Nile tilapia leukocytes exposed to diazinon and Aeromonas hydrophila 117

Cristerna Ortega *et al*

Characterization of Peripheral Blood Leukocytes in the Mexican Elderly Population 118

Cruz-Quintá *et al*

Immunoepigenetic and Molecular Impact of PAHs in B-Cell Acute Lymphoblastic Leukemia 119

Cubillo-Cervantes *et al*

Evaluation of Methods for Quantifying Cell Viability: Spectrophotometry, Microscopy, and Flow Cytometry 120

D

De la Rosa-Salazar *et al*

Resistance mechanisms to immunotherapy in her2+ breast cancer: systematic review and meta-analysis resistance 121

De León-Rodríguez *et al*

Composition of Immune Niches Present in Tumor Microenvironment Associate with Disease Control or Progression in Melanoma 122

Del Aguila -Velasco *et al*

Isolation and Characterization of a Neutralizing VNAR Fragment Against PD-L1 in cancer 123

Delgado-Benedey *et al*

Oxidative stress and Nrf2 activation under an exercise protocol in women recovered from breast cancer. 124

Delgado-Zaldívar *et al*

TGF-β3 Induces Oral Tolerance by Imprinting Gut-Homing Capacity in Regulatory T cells 125

Diupotex *et al*

Anti-PD-1 therapy enhances progenitor-like and intermediate exhausted T cells in chronic leishmaniasis. 126

Domínguez-Gómez, *et al*

Antioxidant effect of grape pomace on murine macrophages 127

Donis -Maturano *et al*

Evaluation of the Cytotoxic Effect of Anticancer-Potential Nanopolymers on Human Peripheral Blood Mononuclear Cells 128

Duarte-Mata *et al*

Dermaseptin DMS-DA6: a promising alternative to antibiotics for N. brasiliensis actinomycetoma 129

E

Esparza -Reyes *et al*

Lyn Kinase as a Key Regulator in Replicative Senescence of Mast Cells 130

Esperante *et al*

Development of an immunotherapy-resistant human melanoma model to establish therapeutic response biomarkers 131

Espino -Solís *et al*

Immune Profiling of Pediatric Patients Infected with Rickettsia and Ehrlichia 132

Espinoza-Hernández *et al*

Innovative Humanized NSG Mouse Platform for Cutting-Edge Immunotechnology and Biotherapeutic Evaluation 133

Esteban-Maldonado *et al*

Piezo1 expression in reactive astrocytes in a murine model of Alzheimer's disease 134

Estepa-San Nicolás *et al*

Altered expression of purinergic receptor genes in mast cells from IBD mice 135

Estevez-Mieres *et al*

Molecular characterization of a genetic variant in JAK3 136

F

Favela-Maldonado *et al*

CRLF2 expression in pediatric Ph-like and Ph^{+} B-cell acute lymphoblastic leukemia 137

Federico-Guerrero *et al*

PD-L1 as prognostic predictor for treatment of non-small cell lung cancer. 138

Félix-Arellano *et al*

Bioinformatic identification of immune receptors in tuberculosis targeted by progranulin 139

Figueroa -Miranda *et al*

Genotypic Variability of HEV Influences Renal Manifestations and CytokineProfile 140

Fisher-Bautista *et al*

Systemic inflammatory markers in ST-elevation myocardial infarction patients 141

IL-37 against ferroptosis induced during ischemia/reperfusion in an ***in vitro*** model 142

Flores-Castro *et al*

Interaction among polymorphisms of cytokines reveals synergistic influence on susceptibility to RA 143

Flores-Correa *et al*

Combination therapy with doxorubicin and VLP-rME in a mouse model of TNBC 144

Flores-Durand *et al*

Engineering a Safe Minicell-Producing Salmonella Typhimurium for Vaccine Delivery Vector purposes 145

Flores-Guzmán *et al*

Bone marrow dormant tumor cells maintain reactive memory CD8 T cells 146

Flores -Martínez *et al*

Cell-permeable Bax BH3 peptide induces chemosensitization in Head Neck cancer cells 147

Flores-Mejía *et al*

Effect of Gold Nanoparticles on the Biological Function of Human Basophils 148

Western Diet-Induced Obesity on the Response of Mast Cells from C57BL/6 Mice 149

Flores-Rabasa *et al*

B-1 lymphocytes and sepsis 150

Fonseca -Camarillo *et al*

Transcriptional analysis of Intestinal Wound Healing in patients with Ulcerative Colitis. 151

Participation of T Cell Factor -1 (TCF1) in Sjôgren's Syndrome. 152

Participation of the Extracellular Matrix Metalloproteinase Inducer (EMMPRIN) in dermatomyositis. 153

Participation of transcription factor NR4A and NDRG in patients with Inflammatory Bowel Disease. 154

Fournier Pierrick *et al*

Turning breast cancer bone metastases hot using 5-fluorouracil against MDSCs 155

Fragozo-Ortiz *et al*

Discovery and characterization of Anti-Ubiquitin scFvs via Phage Display Using ALTHEA Platinum VH:VL 4x3 156

Fuentes-Rodríguez *et al*

Validation process in leukemia-specialized labs from collaborative network "Mexico in alliance with St. Jude" 157

Fuerte-Pérez *et al*

CRTAM is essential for the regulation of the innate immune cell during bacterial infections 158

G

Gajón *et al*

Wnt3a-Expressing Cancer Stem Cells Impair CD8+ TRM Function, Contributing to Immunotherapy Resistance in Melanoma 159

Galarce- Sosa *et al*

γδ T Cells Drive Anti-NPA Autoantibodies in Lupus Mouse Model 160

Galindo Hernández *et al*

Effect of Acute Intranasal Administration of Cry1Ac Proteins (Protoxin and Toxin) on Pulmonary Mucosa 161

Gallegos Rodarte *et al*

Bovine Viral Diarrhea Virus induces activation of IFI16 in bovine macrophages. 162

Gamboa-Meraz *et al*

CXCR4 Upregulation by Soluble Factors Enhances HIV-1 Syncytia in Memory T Cells 163

Garay Canales *et al*

Perinatal di-2-ethylhexyl-phthalate (DEHP) Exposure Increases Breast Cancer Metastasis to the Lung and Liver 164

García-Cruz *et al*

Immunophenotype characterization of T-cell populations in a case of an orphan form of ichthyosis 165

García-Gómez *et al*

Monocytes from type 2 diabetic patients show metabolic and immune dysfunction 166

García-González *et al*

Comprehensive RNA Sequencing Analysis of CD43 (Sialophorin) Expression in Human Cancers 167

Evaluation of fibrosis biomarkers in patients with Metabolic Dysfunction-Associated Steatotic Liver Disease 168

García Hernández *et al*

Serological Prevalence of Orthoflaviviruses in Wild Birds and Rodents from the Yucatán Peninsula 169

García-Huerta *et al*

Impaired expression of autophagic protein ATG16L1 attenuates intestinal pathology evoked by ZIKV 170

García -Nava *et al*

Cell-permeable Bax BH3 Peptide induces Apoptosis and Chemosensitization in Lymphoma cells 171

García-Rasilla *et al*

Smad7 deletion as a strategy to enhance immune regulation via TGF-β in CD8^{+} T cells 172

García-Valeriano *et al*

Evaluation of T-cell responses induced by AVX/COVID-12 'Patria' COVID-19 vaccine in pigs 173

García-Vargas *et al*

Systematic Review and Meta-Analysis of Predictive Biomarkers for Rheumatoid Arthritis 174

Multibiomarker Model to Predict Rheumatoid Arthritis Risk in At-Risk Individuals 175

Garduño-Espinoza *et al*

Anticancer activity of antimicrobial peptides from Bacteria Lactic-Acid on epithelial cancer cells 176

Garza-García *et al*

Analysis of -2518 A>G (rs1024611) CCL2 with cardiometabolic variables in rheumatoid arthritis 177

Girón- Pérez *et al*

Salmonella typhimurium infection increased the necrosis of diazoxon-exposed mononuclear cells 178

Godínez-Mejía *et al*

Immunopotentiator "RTLV-1424" Modulates Mesenchymal Stem Cells Immunosuppressive Capacity Over T Cells. 179

Godínez-Victoria *et al*

Analysis of gut barrier markers in mice fed high-fat diet plus butyrate 180

Gómez-Castellano *et al*

Discovery and characterization of two anti-PD-1 antibodies with a unique PD-1 binding 181

Gómez Frías *et al*

Vitiligo: association between physical activity, strength, and body composition with oxidative stress 182

Gómez-Medina *et al*

Molecular mimicry between gut microbiome and ghrelin: in-silico exploration of anti-GRL antibodies-origin 183

Gómez-Méndez *et al*

Identification of Cry1Ac Toxin Receptors in Plasma Membranes of Macrophages 184

Gómez-Montañez *et al*

Role of the Complement System in Inflammation and Autoimmune Diseases: Perspectives for Future Therapies **185**

The Role of Dendritic Cells in Antigen Presentation and Immune Tolerance **186**

González-Barragán *et al*

Evaluation of Piezo2 as a potential therapeutic target in chronic myeloid leukemia **187**

González-Chávez *et al*

Joint Microbiota Dysbiosis Contributes to Immune Responses and Inflammation in Spondyloarthritis **188**

NPY Links Inflammation and Senescence in Experimental Rheumatoid Arthritis via mTOR Modulation **189**

González -Domínguez *et al*

Cytotoxic effect of an anti-CD44v6/CD54 CAR-T against gastric cancer **190**

González -Hernández *et al*

Purinergic Receptors and Exhaustion Marker Levels in T Lymphocytes in Breast Cancer **191**

González-Reyes *et al*

Development of a 3D Co-Culture Platform for Chemoresistance Prediction in B-ALL. **192**

González-Sánchez *et al*

Functional Role and Molecular Mechanisms of NTB-A (SLAMF6) in Human Natural Killer Cells **193**

Gorgonio-Meza *et al*

Acute Myeloid Leukemia and Immunodeficiency With Multisystemic Involvement in Fanconi Anemia **194**

Gress-Mogica *et al*

Epicatechin regulates autophagy and PD-L1 expression in hepatocellular carcinoma cell line **195**

Güemes-González *et al*

Phenotypic analysis of T lymphocytes and monocytes in patients with acute pancreatitis **196**

Gutiérrez-Iñiguez *et al*

Compatibility of NK cell immunotherapy with cannabidiol in leukemia treatment **197**

Gutiérrez-Xicotencatl *et al*

Targeting HPV16-E5 to DCs by prophylactic vaccination reduces tumor growth and metastasis. 198

Gutiérrez-Pérez *et al*

The neutrophil-to-hemoglobin and lymphocyte score and clinical profile of Rheumatoid Arthritis 199

Guzmán-Barrenechea *et al*

Integrated Epigenomic and Transcriptomic Profiling of TNF-α-Mediated Dysregulation in CD4+ T Cells 200

Guzmán -García *et al*

Melittin and methotrexate: Combined effect on leukemic cells 201

Guzmán-Guzmán *et al*

Interleukin 27 serum levels are associated with radiographic progression in rheumatoid arthritis 202

H

Hasing-Macías *et al*

Optimizing Methotrexate and L-Asparaginase Therapy for B-Cell Acute Lymphoblastic Leukemia. 203

Heredia-Murillo *et al*

IL-36 and VHL as possible mechanisms of VEGF production in mesangial cells 204

Hernández-Arvizu *et al*

Reactive Oxygen Species mediate the antibabesial effect of Aquiluscidin 205

Hernández-Benítez *et al*

Immunomodulatory Effects of Hydroxycinnamic Acid and Epicatechin on NK Cells in a Breast Cancer Co-Culture Model 206

Hernández-Campos *et al*

Quality of life in patients with Guillain-Barre syndrome and dysautonomia 207

Modified Rankin scale as a tool for functional follow-up in Guillain-Barre syndrome 208

Hernández -Doroteo *et al*

Administration of Anti-inflammatory Peptides in EAE Mice and Disease Progression Analysis 209

Hernández -González *et al*

Impact of CRR3 Inhibition on Neutrophil Extracellular Trap Formation and Proinflammatory Cytokine Release in Influenza Infection 210

Hernández-Hernández *et al*

Prevalent HLA-DRB1 alleles in México confer protection against symptomatic SARS-CoV-2 infection. **211**

Hernández-Jiménez *et al*

Immunological Evaluation of an mRNA Vaccine Against Salmonella Typhimurium in Mice **212**

Hernández-Martínez *et al*

The immune response modifies the allatotropin levels in Aedes aegypti **213**

Hernández-Mercado *et al*

Effects of fermentation products from Agave tequilana inulin on allergic inflammation **214**

Hernández-Moran *et al*

Combinatorial neuroprotective therapy in a rat model of traumatic spinal cord injury **215**

Hernández- Navia *et al*

Unmasking a Tumor Suppressor Role: mMGL1 Loss Enhances Anti-Tumor Inflammation in Colorectal-Cancer **216**

Hernández-Villegas *et al*

Comparative efficacy of Tocilizumab versus Azathioprine and other empirical disease - modifying drugs. **217**

Herrera-Torres *et al*

Extracellular Ubiquitin induces monocyte activation in a CXCR4-independent manner **218**

Huerta-Hernández *et al*

Modelling the pro- and anti-inflammatory macrophage differentiation: role of NFkB and CREB **219**

I

Ilhuicatzi -Alvarado *et al*

Effects of the oral administration of Cry1Ac toxin in gut mucosal tissue **220**

Islas-Saldívar *et al*

Evaluation of MAMDC2 protein as a potential therapeutic target in Chronic Myeloid Leukemia **221**

J

Jaimes-Ortega *et al*

Isthmin1 Upregulation in the Intestinal Microenvironment During *Salmonella Typhimuriu*m Infection 222

Jiménez -Rosas *et al*

Evaluation of a vaccine prototype against Salmonella Typhimurium in mice 223

Jiménez-Vargas *et al*

Dioscorea remotiflora reduces the inflammatory response in experimental allergic urticaria 224

Jiménez-López *et al*

Leukocytes in human breastmilk: a scoping review 225

Juárez-Carvajal *et al*

Immunomodulation by bacterial products promotes innate signatures favorable to tuberculosis host response 226

Juárez-Rivera *et al*

Evaluation of the inflammatory profile in a population of older adults with no clinical history of disease 227

Juárez-Valderrama *et al*

Molecular Mimicry Between Food Antigens and Metabolic Hormones: Role on Anti-LEP/Anti-GRL Antibodies 228

K

Kempis -Calanis *et al*

Impact of Delivery Mode on Neonatal CD4+ T Cell Gene Expression Profiles. 229

Ko *et al*

In vitro–generated TRM T cells show enhanced effector function and ICB response in human melanoma 230

L

Laredo-Tiscareño *et al*

New Evidence of Emerging Arboviruses Circulating in Mexico 231

Legorreta-Herrera *et al*

Testosterone differentially regulates Bmal1 and Clock expression and the malaria immune response 232

León-Cabrera *et al*

STAT6 Deficiency Accelerates Skin Carcinogenesis and Promotes Immunosuppressive Responses in a Murine Model **233**

Lopez -Pantoja *et al*

Interspecies cross-immunity as treatment for canine distemper virus infection **234**

Lopez -Alvarez *et al*

Base deficit and leukocyte count as predictors of mortality during sepsis **235**

López-Ávila *et al*

HIF-1 activation modulates CCR6 expression in vitro and in vivo models. **236**

López-Camacho *et al*

Validation of a serological ELISA method to detect anti-SARS-CoV-2 IgG and IgM antibodies in Mexican population. **237**

López-Castillo *et al*

Neuroinflammation sex-differences in the mouse striatum induced by aging and excitotoxic damage **238**

López-García *et al*

Autophagy-dependent cytotoxicity induced by oleanolic and ursolic acids in lung cancer cells **239**

López-Huerta *et al*

Single-cell analysis identifies inflammatory and tissue-remodeling tumor macrophages distinct from M1/M2 **240**

López-Leal *et al*

CCR3 blockade reduces neutrophil lung recruitment during acute influenza A H1N1 infection **241**

López-Montaño *et al*

Potential association of B regulatory cells in bovine leukemiavirus infection **242**

López-Moreno *et al*

In silico design of DNA vaccine against T. gondii targeting ROP35 epitopes **243**

In silico design of a DNA vaccine against Toxoplasma gondii using GRA6 **244**

López-Valencia *et al*

Effect of premature birth on the Gene Expression Profile of Neonatal CD8+ T Lymphocytes **245**

López-Pacheco *et al*

Comprehensive profiling of breast cancer-derived extracellular vesicles associated with metastasis and immunomodulation 246

Loya-Rivera *et al*

Therapeutic Potential of Nanodiamonds as Nanocarriers of Tofacitinib in Cellular Models of Arthritis 247

Lozano-Cisneros *et al*

Intratumoral Administration of RTLV-1424 Modulates Immune Infiltrate in Breast cancer Mouse Model 248

M

Macedo -Hernández *et al*

The protein ATG16L1 regulates myeloid cells in the peritoneal cavity 249

Machucho-Guzmán *et al*

Antileukemic potential of Ganoderma lucidum: Cytotoxicity on THP-1 and K562 cell lines 250

Macias -Almanza *et al*

Methyl-donor nutrient intake and global leukocyte DNA methylation in rheumatoid arthritis patients 251

Madera-Salcedo *et al*

Senescence Enhances Pro-Inflammatory Signaling in Mast Cells 252

Magaña -Guerrero *et al*

Peripheral Neutrophil NFAT5-Aldose Reductase Pathway: A Contributor to Inflammation in Diabetic Retinopathy 253

Anti-inflammatory and corneal repair properties of amniotic-membrane extract (AMEx) in a chemical de-epithelization model. 254

Magaño -Bocanegra *et al*

Surface display of scFv-TfR1 enhances bacterial adhesion for bacteria-based anti-lymphoma therapy purposes 255

Magdaleno-Villanueva *et al*

Association of number and proportion of B1 lymphocytes with emergency abdominal surgery outcomes 256

Malvaez-Luis *et al*

B55β: Critical mediator of arthritis pathogenesis and modulator of TNF-α inhibitor efficacy 257

Mandujano-López *et al*

Evaluation of the biomarker potential of the tetraspanin33 expression in B-cell lymphomas **258**

Novel B Cell Subsets as Potential Biomarkers in Idiopathic Inflammatory Myopathies: Insights into Disease Pathogenesis and Disease Activity **259**

Marcial -Medina *et al*

Dissecting TGF-β Pathway in Melanoma Immunity Using CRISPR/Cas9 Gene Editing **260**

Martínez Gutiérrez *et al*

Human B-1-like lymphocytes: truly similar to murine B-1 cells? **261**

Martínez -Iturbe *et al*

Cytotoxic Molecules as Potential Biomarkers for Active and Inactive Systemic Lupus Erythematosus **262**

Martínez-Leija *et al*

Low levels of miRNA-125b are associated with a poor response to neoadjuvant treatment in patients with breast cancer **263**

Martínez Martínez *et al*

Valproic Acid: Novel antitumor strategy for the treatment of canine mastocytoma **264**

Martínez-Meza *et al*

Immunophenotypic alterations in people occupationally exposed to pesticides in Monte Blanco, Veracruz **265**

Martínez-Morales *et al*

Effect of acute stress on mast cells and ileum epithelium of mice. **266**

Martínez-Pérez *et al*

Diazinon-induced immunotoxicity on Oreochromis niloticus challenged with Aeromonas hydrophila **267**

Martínez-Sarabia *et al*

$CD4^+$ T Cell Phenotypes May Distinguish ICB Responders from Non-Responders in Melanoma **268**

Mateos -Arenas *et al*

The peritoneal cestode Taenia crassiceps restructures gut bacterial communities in the mouse host **269**

Maya-Maldonado *et al*

LITAF-like Transcription Factor, a new regulator of the immune response in Drosophila **270**

Medina-Sosa *et al*

Systematic review for GPR15 / GPR15/L as a biomarker or drug target in inflammatory diseases. **271**

Meester *et al*

Improving fibromyalgia diagnosis: optimizing tests **272**

Mejía -Muñoz *et al*

Perinatal Sucralose Intake Impairs Antitumor Immunity and Promotes Colitis-Associated Tumorigenesis **273**

Mendiola *et al*

ATG16L1 is a negative regulator of the IL-6-Muc4 axis during enteric inflammation **274**

Mendoza-Ramírez *et al*

Immunization with plasmids that codify SARS-CoV-2 fusion proteins induced an immune response in a preclinical model **275**

Mendoza -Roldán *et al*

Inhibition of WNT/βcatenin in melanoma mouse model. Strategy to reverse immunotherapy resistance **276**

Mendoza-Sánchez *et al*

Impact of MIF Inhibition by CPSI-1306 in Colitis-Associated Colorectal Cancer **277**

Meneses-Preza *et al*

Study of mast cells during COVID-19 and its fibrotic lung sequelae **278**

Meza-de la Rosa *et al*

AI-biotechnology convergence and the emergence of a biodigital regime in vaccines: a sectoral innovation proposal **279**

Meza -Peña *et al*

Effects of diet on the Leptin-Anti-LEP antibody axis in Wistar rats **280**

Mijares-Guevara *et al*

Regulatory networks in neonatal T cells: vaginal, term and preterm cesarean delivery **281**

Moctezuma Rocha *et al*

Immunomodulatory and Antitumor Properties of the GK-1 Peptide: Possible Involvement of TLR4 Signaling. **282**

Mojica-Villa *et al*

NETS from neutrophils of patients with spondyloarthritis and their interaction with osteoblasts **283**

Molina-Afanador *et al*

Alleviation of indomethacin-induced enteric damage in Wistar rat by kombucha supplementation **284**

Molina-Guillén *et al*

Determination of antimicrobial peptides in marsh turtle microbiota for the analysis of its immunological resistance **285**

Monroy-García *et al*

HPV-binding function of CD73 in cervical cancer cells **286**

Montes-Zapata *et al*

Activation markers in NKG2C^{+}NKp46^{+} Memory-Like NK Cells Subset against Influenza A Nucleoprotein **287**

Metabolic gene dysregulation in NK cells from patients with severe COVID-19 **288**

Montiel-Martínez *et al*

Development of a recombinant viral vector as non-replicative rotavirus VP6 vaccine platform **289**

Mora-García *et al*

Association of CVD phenotypes with clinical activity and metabolic endotoxemia in SLE **290**

Morales-Núñez *et al*

Differential IL-17R family expression in B lymphocytes: potential targets for rheumatoid arthritis immunotherapy **291**

Morelos-Cruz *et al*

Analysis of the effect of cannabidiol on the immune response in horses **292**

Moreno-Eutimio *et al*

Immune and Inflammatory Profiling in Elite Taekwondo Athletes During the Preparation and Recovery Phases of the France 2024 Olympic Games 293

Moreno-Lafont *et al*

Use of Imipramine in a murine model of Brucella abortus 2308 294

Mosso-Pani *et al*

Metabolic Cross-Talk: Platelets as Dynamic Regulators of Neutrophil Immunometabolism 295

Muñoz Cano-Villalpando *et al*

Effect of polyherbal supplementation on intestinal microbiota and immune response in horses. 296

Mendoza-Montiel *et al*

PD-L1 expression in cervical cancer tissue is strongly associated with the expression of CD73/TGF-β the percentage of CD8+/PD-1+ T cells and disease progression 297

Muñoz-Valdivia *et al*

Relationship of anti-dsDNA autoantibodies and TLR9 haplotypes with risk in SLE patients 298

Muro-Dávalos *et al*

Association of C-reactive protein levels and CRP haplotypes with rheumatoid arthritis risk 299

N

Niño -Herrera *et al*

Repositioned Drugs Treatment for Suppressing Dengue Infection and Cytokine Production in Macrophages 300

O

Ocampo-Hernández *et al*

Immunomodulatory Effects of Purified and Encrypted Axolotl Peptides 301

Ogonaga-Borja *et al*

BCG as an inducer of trained immunity in bone marrow-derived mast cells 302

Ojeda -Vázquez *et al*

Immune expression of CD38 as an important biomarker of diagnosis, prognosis, and monitoring of MASLD 303

Olalde-Rodríguez *et al*

TLR4 expression in metabolically activated macrophages from patients with dyslipidemia. 304

Olguín-Alor *et al*

Inhibins are required for T-Cell migration and homing to peripheral lymphoid organs 305

Olivas-Aguirre *et al*

Expanding the Landscape of Ca^{2+} signaling in NK Cells: Evidence of Unexplored Ion Channels with Clinical Relevance 306

Olvera-Gómez *et al*

Detección de linfocitos T CD8 WT-1 e IE-1 específicos en pacientes con Cáncer de mama previo a la mastectomía. 307

Ordoñez-Rodriguez *et al*

“Computational insights into the mechanisms of haptenization of HSA-SMX” 308

Orozco -Alvarez *et al*

Nickel-Cobalt Nanocrystals and Their Anti-Inflammatory Potential 309

Orozco-Córdoba *et al*

Trim33 regulates Th9 Lymphocyte Differentiation and In Vivo Function. 310

Orozco-Jacobo *et al*

Decoding CMTM6 in Cervical Cancer: Subcellular Distribution and PD-L1 Binding Insights 311

Orozco-Ruiz *et al*

Modulation of Erythrocyte Oxidative Stress by Paenibacillus sp. Extracts at Different pH 312

Ortega-Díaz *et al*

Inhibition of autophagic flux as strategy to reduce the Raji cells proliferation 313

Ortega Francisco *et al*

COH04S1 vaccine induces cross-reactive and protective immunity against mpox virus infection. 314

Ortega-Francisco *et al*

Functional characterization of anti-Spike monoclonal antibody for Fc effector activity 315

Ortega-Tirado *et al*

Evaluation of VSP 5G8 Expression in Giardia lamblia Under Different Experimental Conditions 316

Ortiz-Jiménez *et al*

Evaluation of Renin-Angiotensin System in individuals with normal weight, obesity, and T2DM 317

Ortiz-Santos *et al*

Induction of Mast Cell Senescence by murine melanoma B16-F10 cells-conditioned media 318

P

Pacheco-García & Serafín-López

Immunization against SARS-CoV-2 in immunostimulated rats with imiquimod 319

Pastelin-Palacios *et al*

Virulence Factors of Staphylococcus aureus Isolates from Atopic Dermatitis Patients Correlate with Disease Severity in a Mexican Cohort 320

Pacheco-Olvera *et al*

Genomic Characterization of the T-Cell Receptor loci in Ambystoma mexicanum 321

Patiño-López *et al*

Early and transient CD40 expression in T cells 322

Paz-Rodríguez *et al*

Impact of Arylamine N-Acetyltransferase Silencing on CD4$^+$ T Cell Polarization and Immunometabolism 323

Pelcastre-Gómez *et al*

“Comparative analysis of immunogenic proteins of Mycobacterium tuberculosis recognized by patients in active and latent phases” 324

Pérez-Eguía *et al*

Function of pVHL in the Apoptosis of Pancreatic β Cells 325

Pérez-Lara *et al*

Identification of Non-Replicating Persistent Salmonella in B cells during ***in vitro*** Infections 326

Pérez López *et al*

Role of CCR3 in neutrophil activation during interaction with influenza virus ***in vitro*** 327

Pérez-Méndez *et al*

Development and optimization of cancer vaccines for breast cancer treatment 328

Pérez-Saucedo *et al*

Preclinical Evaluation of a Multi-Epitope HIV-1 Vaccine Candidate Engineered on a B19 Parvovirus VLP Platform 329

Pérez-Torres *et al*

Role of autophagy in PD-L1 expression in a lung cancer cell model 330

Pérez-Vergara *et al*

“Evaluation of the immune response induced by vaccine formulations against Salmonella Typhi.” 331

Pesqueda Cendejas *et al*

CRP variants and serum levels as cardiovascular and disease activity biomarkers in SLE 332

Prieto-Carraco *et al*

Mitochondrial Dysfunction Drives Immune Dysregulation and Oxidative Stress in Spondyloarthritis 333

Q

Quinto-Manzanares *et al*

Targeting B cell activation in vitro using antigen-decorated liposomes carrying Bcl-6 mRNA 334

Quiñones-Acuña *et al*

Immunopathogenesis and Novel Therapeutic Strategies in Rheumatoid Arthritis. 335

R

Ramírez -Álvarez *et al*

Optic Neuritis as Initial Manifestation of Multiple Sclerosis: A Case Report 336

Ramírez-Florencio *et al*

Generation of an Anti-CD19 scFv Panel Using the ALTHEA Platinum III Phage Display Library 337

Ramírez-Pérez & Santos-Argumedo

Dynamics of immunoglobulin production in milk: IgA and IgG across lactation stages in C57BL/6 mice 338

Ramírez Quintero *et al*

Characterization of B cells subsets in a murine model of accelerated aging 339

Ramírez-Vilchis *et al*

Role of CD43 in erythroid differentiation 340

Raya -Malvaez *et al*

Identification of receptors for protoxin Cry1Ac in macrophage plasma membrane proteins 341

Rea-Ortega *et al*

Distribution and Functional Capacity of Monocytes in Compensated and Decompensated Cirrhosis Patients 342

Rechy-Ahumada *et al*

Bacterial Extract-Mediated Activation and Immune Training of Mast Cells 343

Rendón-Bautista *et al*

Immune infiltrate and genome structure of TNBC sample by scRNA-seq and Hi-C 344

Reyes Hernández et al

The NLRP3-Interleukin 15 Axis Transiently Regulates the Resistance in Oral Cavity Carcinogenesis 345

Reyes-Martínez *et al*

Mycobacterium tuberculosis alters cytoskeletal dynamics and Induces ROS and NOS in hepatocytes 346

Reyna-Varela *et al*

Characterization of mast cells of the Lmna G609G progeric mouse 347

Ríos-Ramírez *et al*

The role of TGF-beta induced TIF1-gamma and Smad4 on regulatory T lymphocytes within the tumor microenvironment. 348

Rivas-Saucedo *et al*

Optimized IR-fluorophore platform for autoantibody/biomarker detection in TDM1 and LADA 349

Rivera Delgado *et al*

Evaluation of Microorganism Derived Adjuvants in induction of Trained Immunity in BMDCs 350

Rivera-Ortega *et al*

Humoral immune proteins in the mucus of two evolutionarily basal marine animals. 351

Robles-Reyes *et al*

Effect of amino acids on adult and neonatal CD4+ T cell responses 352

Rocha-González *et al*

Analysis of the Autophagy-Ferroptosis Crosstalk in a Cancer Cell Model **353**

Rodríguez-Cortés *et al*

Nitazoxanide shows an immunomodulatory effect in IFN-γ production in Vγ9Vδ2 T cells **354**

Rodríguez-Guerrero *et al*

Anti-inflammatory activity of Rhus trilobata subbractions in lipopolysaccharide-stimulated J774A.1 macrophages. **355**

Rodríguez-Hernández *et al*

MIF Deficiency Alters Gut Microbiota and Contributes to Colitis-Associated Colorectal Cancer Development **356**

Rodríguez-Jorge *et al*

Metabolic regulation of neonatal T cell activation and its therapeutic implications. **357**

Rodriguez-Lopez *et al*

Alterations in Immune Cell Populations in the Blood of Transgenic Mice with Alzheimer's-Like Pathology and Cerebral Amyloid Angiopathy (Tg-SwDI). **358**

Rodríguez-Santiago *et al*

Sex differences of immunological response in the induction of colorectal tumors **359**

Rodriguez-Villalpando *et al*

Differential Effects of Agave Fructans on Inflammation, Pruritus and Gut Microbiota in Rats **360**

Rojas-Guillen *et al*

Coagulation Times as Predictors of Mortality in Sepsis **361**

Rojas-Gutiérrez *et al*

Temporal transcriptomic metaanalysis of peripheral blood in early Ischemic Stroke **362**

Identification of transcriptomic biomarkers in early preeclampsia in systemic lupus erythematosus **363**

Biomarkers of fetal loss and infertility in rheumatic diseases: An integrative analysis **364**

Rojas-Salazar *et al*

Role of Neutrophil Extracellular Traps (NETs) in Inflammation and Thrombosis: New Horizons in Clinical Immunology 365

Cytokine Storms and Immune Dysregulation in Severe Viral Infections 366

Anti-PD-L1 Functionalized Nanoparticles to Enhance Immunotherapy in Resistant Murine Melanoma Models 367

Role of the Gut Microbiota in Modulating the Immune Response and Its Clinical Implications 368

Checkpoint Inhibitors and Immune Regulation in Cancer Therapy 369

Rojo-León *et al*

CD43 regulates T-cell function and survival in a context-dependent manner 370

Román-Anguiano *et al*

Viability of human leukemia cells with csGRP78 in an in vivo model 371

Romero-Ramírez *et al*

Myo1g participates in the expression of NK1.1 molecule and the cytotoxicity of NK cells. 372

Romero-Terrazas *et al*

Serological Profiling in Rickettsiosis Using a Multi-Species Recombinant Antigen 373

Romo-García *et al*

Cytokine alterations in the pediatric population chronically exposed to high concentrations of Particulate Matter 374

Romo -Sáenz *et al*

Synthetic Peptides of GP45 and GP90 as Biomarkers for Early EIAV Detection 375

Rosales *et al*

Low-density neutrophils increase during obesity and display less phagocytosis 376

Rosas-García *et al*

Expression and regulation of MPP1 in human monocyte-derived macrophages (MDM) 377

Silencing of IL-16 Expression in Human Monocyte-Derived Macrophages 378

Diet quality and rs1800795 IL6 interact with cardiovascular risk in rheumatoid arthritis 379

Rosas-González *et al*

MIF inhibitor (ISO-1) modulate ocular surface Inflammation in a Dry Eye model 380

Rubio-Blancas *et al*

Role of the CD43 sialomucin in macrophage differentiation in contrasting inflammatory backgrounds 381

Rubio-Robles *et al*

Helios limits CD8 T cell antitumor capacity 382

Ruedas-Montero *et al*

Tolerogenic innate immune cells compensate for regulatory T cells in Kawasaki disease 383

Ruelas-Ruiz *et al*

Cytokines and alpha-interferon response of A549 cells against influenza A virus 384

Influenza vaccination modulates monocyte subsets and cytokine expresión upon viral challenge 385

Ruíz-Cruz *et al*

Human parvovirus B19 VLP's Induce SARS-CoV-2 neutralizing antibodies in mice. 386

Ruiz-de la Cruz *et al*

Pathway Polarization in Human Leprosy Biopsies: From Immune Containment to Exhaustion 387

PD-1/PD-L1 blockade exacerbates inflammatory tissue damage in experimental Nocardia brasiliensis mycetoma 388

Immune dysregulation and bacterial structural adaptation in chronic Nocardia brasiliensis murine mycetoma 389

Ruiz *et al*

A Long-Term Culture Model for Investigating Senescence-Associated Dysregulation in Macrophages 390

S

Saad-Manzanera *et al*

Recombinant Scorpion Toxin Targeting Ion Channels as a Strategy Against Breast Cancer 391

Saint Martin-Castellanos *et al*

The ITIM domain of CD5 modulates Th1 versus Th17 T cell differentiation by regulating cytokine dependent STAT signaling. 392

Saint Remy-Hernández *et al*

An update of the description of immunoglobulin loci in Ambystoma mexicanum 393

Salcedo-Magaña *et al*

Characterization of tumor-infiltrating regulatory T cells 394

Salgado-López *et al*

Generation of Stable CD19+ Cells to Identify Anti-CD19 scFv for CAR-T" 395

Salinas-Guardado *et al*

Antipruritic effect of Agave tequilana fructans in experimental models of allergic itch 396

Salinas-Miralles *et al*

Anti-inflammatory and immunomodulatory effects of Agave tequilana fructans in experimental atopic dermatitis 397

Sánchez-Barrera *et al*

Removing IgG from excreted/secreted helminth products enhances their antitumor effects 398

Sánchez-Centeno *et al*

Expression of anti-CD19 chimeric antigen receptor (CAR) by neutrophil-like cells 399

Sánchez -Fragoso *et al*

VLPs are a promising platform for multiepitope vaccines targeting SARS-CoV-2 400

Sánchez -Torres *et al*

Butyric acid produced by Staphylococcus epidermidis attenuates the expression of IL-36γ and improves murine psoriasis 401

Sánchez-Villalobos *et al*

Glycomacropeptide modifies staphylococcal abundance and adhesion in experimental atopic dermatitis 402

Sánchez-Ramírez *et al*

Development of an innovative carbon nanotubes-based nanoconjugate for ovarian cancer immunotherapy 403

Santana-Sánchez *et al*

Characterization of exhausted T cells in systemic lupus erythematosus 404

Sandoval-Juárez *et al*

Cervical cancer mesenchymal stromal cells release PD-L1^high extracellular vesicles via adenosinergic signaling 405

Santana-Sánchez *et al*

Characterization of exhausted T cells in systemic lupus erythematosus 406

Sepúlveda -Cuéllar *et al*

Glial dysfunction links chronic Toxoplasma gondii infection to spatial memory deficits 407

Serna -Pérez *et al*

Internalization of Candida glabrata from biofilm established on biotic surface by macrophages 408

Sifontes-Rodríguez *et al*

Toxicological profile of the immunomodulatory, antitumor, and antimetastatic peptide GK-1 409

Solares-Rioja *et al*

Senescence-associated changes in electrophysiological properties of mast cells 410

Solís-Gómez *et al*

Prevalence and diagnostic approaches to autonomic dysfunction in Guillain-Barre syndrome 411

Solís-Torres *et al*

Effect of long-term exposure to particulate matter on pulmonary immune response against P. aeruginosa in a murine model 412

Solorio-Cárdenas *et al*

Immunopotentiator “RTLV14-24” modulates intratumoral T cells phenotype of cancer mouce model 413

Soria -Medina *et al*

Microglia’s dual role in neurogenesis and behavior in adult rodents: literature review 414

Soriano -Cruz *et al*

Alterations of endothelial progenitor cells in Mexican women with cardiovascular risk factors 415

Sosa Flores *et al*

Mapping HIV epitopes restricted by Amerindian HLA-B*35 subtypes using IFNγ ELISpot and synthetic HAP-containing peptides 416

Sta.Maria-Peregrino *et al*

Phenotype and immunogenicity of plasma extracellular vesicles in latent and active tuberculosis 417

Suárez-Rojas *et al*

Helios favors CD8 T cell effector differentiation by modulating IL-2 signaling 418

Suárez -Vázquez *et al*

Comparative Analysis of Exhaustion Markers in Blood and Tumor in Renal Cell Carcinoma 419

Immune Exhaustion Marker Dynamics Following Nephrectomy in Patients with Renal Cell Carcinoma 420

Mast cell deficiency affects the development of actinomycetoma caused by Nocardia brasiliensis 421

Mast Cell Activation by Nocardia brasiliensis Drives Immunopathology in Actinomycetoma 422

Sumoza-Toledo & Herrera-Hernández

The store-operated calcium entry inhibitor 2-aminoethyl diphenylborinate suppresses NOX-dependent netosis 423

T

Tapia-Rodríguez *et al*

Analysis of DNMT2 function during dengue virus infection in ***Aedes aegypti*** mosquitoes 424

Torres-Flores *et al*

Hybrid Immunity Enhances Effector Function and Stem-like Memory T Cells Against SARS-CoV-2 425

Torres- García *et al*

Peripheral Immune Biomarkers for Alzheimer's Diagnosis: Transcriptomic and Digital Cytometry Analysis 426

Torres -Salgado *et al*

Dietary Fiber: Impact on Immunity Mediated by the Gut Microbiome 427

Torres-Reyes *et al*

HPV E6/E7 regulate Grainyhead-like 2 and 3 expression in a CaCU model 428

Trejo-De la Mora *et al*

Combination therapy with Doxorubicin and Cry1Ac in a murine model of TNBC 429

Turiján-Espinoza *et al*

Effect of microRNA overexpression on macrophage polarization markers 430

U

Ulloa-Ramírez *et al*

Diazinon-induced neuroimmunotoxicity mediated through mAChR in Oreochromis niloticus challenged with Aeromonas hydrophila 431

Unzueta-Villalobos *et al*

Cytokine Signatures in Immune Thrombocytopenia Across Clinical Stages 432

Urbina-Rodríguez *et al*

In-silico transcriptomic study of P2X$_7$ and PA$_{2A}$ in T lymphocytes in metabolic syndrome 433

Urenda-Castañeda & Urenda-Castañeda

Effect of Probiotics on Secretory Immunoglobulin A in Saliva of Healthy Adults 434

Urieta-Benítez *et al*

Helicobacter pylori induces proteomic changes in human PBMCs and their exosomes 435

Urrutia-Medina & Sánchez-Ramírez

Therapeutic vaccine protocol against breast cancer 436

V

Valencia-Escamilla *et al*

Loss of arpin expression in acute myeloid leukemia cells triggers bone marrow spheroid colonization 437

Valenzuela-Padilla *et al*

The role of FMNL-2 during inflammation in cremasteric postcapillary venules 438

Vaquero-García *et al*

Characterization of Exhausted T Cell Subpopulations in Type 1 Diabetes Mellitus 439

Vargas-Villavicencio *et al*

Both the TNF-α SNP and the IL-12 SNP are important for congenital transmission and disease caused by Toxoplasma gondii in humans 440

Vázquez-Fuerte *et al*

Influence of Pathogen Virulence on the Proteomics of Immune Memory in Tenebrio molitor 441

Vázquez -Solórzano *et al*

IgG-ghrelin and leptin-reactive antibodies differ in individuals according to types of diet 442

Vázquez-Toledo *et al*

Evaluation of CD147, MMPs, TIMPs in Antibody-Mediated Renal Rejection: CD147-Induced MMP Production 443

Vega-Balderrama *et al*

In vitro modulation of inflammatory biomarkers by metabolites from probiotic microorganisms 444

Vega-Muñoz *et al*

Innate Immune Elements of Prostate Epithelial Cells Modulated by Trichomonas vaginalis 445

Trichomonas vaginalis modulates innate immune elements of vaginal epithelial cells 446

Velarde-Rocha *et al*

Cryopreservation induce changes in phenotype and functionality of antigen-specific NK cells 447

Velásquez-Ortiz *et al*

Evaluation of interactions between CD40 and CD40L complex in missense (p.Ala123Glu, p.Ser222Phe, p.Gli257Arg) mutations in patients with Hyper-IgM syndrome. 448

Ventura -Martínez *et al*

Influence of gestational age and birth type on neonatal CD4+ T cell activation 449

Vigil-Mora *et al*

CXCL10-Mediated Migration and Phenotypic Stability of Alloantigen-Specific Tregs 450

Vigueras-Galván *et al*

Seroprevalence of orthohantavirus in rodents from the Trans-Mexican Volcanic Belt. 451

Hantavirus seroprevalence in rodents from the Yucatan peninsula, Mexico. 452

Villa-Villaseñor *et al*

Impact of water pollution on the immune system and oxidative stress in the gills of Skiffia lermae 453

Villagómez-Olea *et al*

Origin and Heterogeneity of Neutrophils in Oral Gingiva 454

Villanueva -López *et al*

Development and Characterization of D119E, a Novel Bispecific Immune Checkpoint Inhibitor: Simultaneous Targeting of PD-1 and CTLA-4 455

Vizcarra-Soto *et al*

Evaluation of the NOTCH pathway during DSS-induced ileal enteritis 456

W

Wong-Baeza *et al*

Mitochondrial dynamics of germinal center B cells and plasma cells in response to a lipid antigen 457

Y

Yáñez-Mendoza *et al*

Characterization of the immunoglobulin heavy chain locus in native Mexican populations 458

Z

Zacarías-García *et al*

Effect of pharmacological inhibition of MIF in a diabetic retinopathy mouse model. 459

Zambrano-Romero *et al*

TGF-β via TIF1γ modulates the activation and proliferation of CD8+ T cells. 460

Zamora-Herrera *et al*

Bone marrow immune microenvironment in childhood leukemia: building a prognostic test 461

Zapata-Zúñiga *et al*

HMGB1 is released during ACPA-positive serum-NET induction and correlates with disease stage and severity in Rheumatoid Arthritis **462**

Zaragoza-García *et al*

Intestinal Protozoa Infection and systemic inflammation markers in rheumatoid arthritis **463**

Zaragoza-Jiménez *et al*

Inflammatory Profile Assessment in Veracruz Population with Metabolic Dysfunction-Associated Steatotic Liver Disease **464**

Zárate-Ayón *et al*

Chronic stress decreases IgA and its pIgR-mediated transport in mice lungs. **465**

Zavala-Reyes *et al*

Bioinformatic identification of a CD8Tex lymphocyte signature in an aggressive breast cancer **466**

Área del artículo: **Regulación inmune**

Betaglycan promotes the in vitro induction of regulatory T cells with diminished suppressive function.

Acevedo-Domínguez, Naray Alejandra [1]; Alvarado-Luis, Brenda Lizeth [1]; Hernández-Campos, Oscar [1]; Raman, Chander [3]; Soldevila, Gloria [1 2].

[1]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, UNAM, CDMX, México. [2]Laboratorio Nacional de Citometría de Flujo, Instituto de Investigaciones Biomedicas, UNAM, CDMX, México. [3]Department of Rheumatology and Medicine, University of Alabama at Birmingham, ALA, USA.

E-mail: nara_2305@hotmail.com

GFBR3 or betaglycan is a widely expressed glycoprotein that facilitates high-affinity TGF-β binding to TGFBR1 and TGFBR2. Betaglycan has been described as an important regulator of fetal development, reproduction, tumor suppression, and T cell maturation and effector differentiation, but its role in the regulation of immune responses remains poorly understood. Our group has previously reported that TGFBR3 expression is downregulated during iTreg induction and that TGFBR3 blocking reduces iTreg differentiation. Since TGF-β signaling is crucial for the induction and suppressive function of regulatory T cells (Treg), we sought to determine if betaglycan could be crucial to these processes. Due to the embryonic lethality of betaglycan knock-out mice, we generated a conditional knock-out mouse model in which betaglycan expression is abrogated in peripheral mature T cells by using the distal promoter of Lck (Tgfbr3fl/fl.dLcKCre). Tgfbr3fl/fl and Tgfbr3fl/fl.dLcKCre naïve T cells were FACS sorted and cultured in the presence of anti-CD3/CD28 Dynabeads and TGF-β1 (1 ng/mL) and iTreg induction was evaluated at 3 days after stimulation. For in vitro suppression assays, Tgfbr3fl/fl and Tgfbr3fl/fl.dLcKCre iTreg were FACS sorted and co-cultured with different ratios of Tgfbr3fl/fl Tresp for 3 days. Interestingly, iTreg induction was 20% lower in Tgfbr3fl/fl.dLckCre cells compared to Tgfbr3fl/fl cells (0.80 RI %CD25+ FoxP3+, p=0.0079). Furthermore, we also observed a reduction in FoxP3 expression (0.93 RI MFI FoxP3, p=0.0286) in TGFBR3 deficient iTreg. Preliminary data suggest that Tgfbr3fl/fl.dLckCre iTreg might suppress Tresp proliferation more efficiently than their Tgfbr3fl/fl counterparts. Our data show that betaglycan supports iTreg induction and could potentially restrict their suppressive function, which plays an important role in the regulation of immune responses in various diseases.

Work supported by PAPIIT-DGAPA Grant # 210224.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Development Of CAR.ME.LA For The Treatment Of Patients With Acute Lymphoblastic Leukemia

Rosa E. Aguilar, Luis A. Cenobio, Rosa M. Rubio, Juan L. Ontiveros, Roberta Demichelis, Iris K. Madera – Salcedo, Florencia Rosetti, José Carlos Crispín

Monterrey Institute of Technology and Higher Education, Mexico. Department of Rheumatology & Immunology , Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Mexico City, Mexico. Department of Hematology & Oncology, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Mexico City, Mexico.

E-mail: A01372989@tec.mx

Chimeric Antigen Receptor (CAR) T cells are a novel therapeutic approach for the treatment of hematologic malignancies, wich relies on the addition of engineered receptors into human T cells. CAR T cells eliminate their targets by conventional effector and citotoxic mechanisms. This cellular therapy was approved by the FDA and is comercially available in certain countries, albeit at high cost. However, in Mexico, CAR T cell therapies are still not produced or used to treat patients, representing an opportunity for its development. To this end, we generated a CD19-specific CAR to treat candidate patients with acute lymphoblastic leukemia (ALL) in Mexico (CAR.ME.LA).

We designed a third-generation CAR vector based on a scFv directed against CD19. Our CAR construct incorporates a hinge and transmembrane domains cloned from CD8a; an intracellular signaling domain composed of the co-stimulatory molecules CD28 and CD137, along with the activation molecule CD3z. All these components were introduced into a third-generation lentiviral plasmid backbone. The transgene is expressed under the regulation of the constitutive human EF-1α promoter. The lentiviral particles were produced by transient transfection of HEK 293 T cells. We confirmed the signaling capacity of our construct using a NFAT- reporter Jurkat T cell line.

In summary, we designed and generated a novel CAR construct that efficently signals in an in vitro system. Our construct is currently being tested in preclinical models and will be further produced following good manufacturing practice (GMP) guidelines to enable the future clinical use of CAR T cells in mexican patients with ALL.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Alternatively activated macrophages: A promising therapy on dry eye disease

Aguilar-Garduño, Arturo [1]; Rosas -Gonzáles , Karla [1];
Zacarías-García, Mónica [1]; Ramos-Montes , Oscar A [1];
Gabriel-Gutiérrez, Ospina [2]; Mendoza-Rodríguez , Mónica G [3];
Terrazas-Valdés , Luis I [3]; Vázquez-Mendoza, Alicia [1].

[1]Laboratorio de Inmunología Ocular, Carrera de Optometría, Facultad de Estudios Superiores Iztacala UNAM, Tlalnepantla de Baz, Estado de México, México. [2]Laboratorio de Biología de Sistemas. Departamento de Biología Celular y Fisiología, Instituto de investigación Biomédicas, UNAM. [3]Unidad de Investigación en Biomedicina (UBIMED), FES-Iztacala UNAM.

E-mail: arturo.aguilar99@comunidad.unam.mx

Dry eye disease (DED) is an inflammatory disease of the ocular surface (OS) of multifactorial origin, characterized by loss of homeostasis and hyperosmolarity of the tear film. Its progression is associated with the production of inflammatory cytokines (IL-6, IL-1β, IL-17, TNF-α), the recruitment and infiltration of neutrophils, monocytes, macrophages and T cells, which together perpetuate the inflammatory process and promote tissue damage in OS. One of the challenges of this pathology is its treatment. because the main therapy only alleviates the symptoms through the use of artificial tears, but does not modulate the underlying inflammatory process. For this reason, the effects of alternatively activated macrophage (M2)-mediated cell therapy are currently being explored. This therapy has proven effective in models of multiple sclerosis, diabetes, and colitis. However, has not been extensively studied about its effect on inflammatory ocular diseases such as DED. Therefore, the aim of this study was to assess whether the transfer of M2 macrophages can modulate the inflammatory process and the characteristic signs of this disease. DED was induced to Balb/c male mice 6-9 weeks of age with 0.5 mg scopolamine administration three times at day for 28 days. On day 14 post induction, 5x106 M2 macrophages were transferred intraperitoneally. The evaluation from the tear film revealed that M2 macrophage transference promotes the recovery of tear volume and the mucin arborization pattern. These results were associated with increased expression of tgf-β and IL4 production in the cornea and conjunctiva. Together, these results suggest that M2 macrophage which suggests that M2 macrophage could be a possible therapy in DED

This Project Is currently funded by PAPIIT – IN228225 and CONAHCyT–CBF-2023-2024-405

Acknowledgments: CONAHCyT for the postgraduate scholarship #2049036.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Exercise reduces the risk of developing periodontal disease caused by aging

Aguilar-González, José Isaías [1]; García-Hernández, Ana Lilia [2];
Cifuentes -Mendiola, Saúl Ernesto [2]; Martínez-Dávalos, Arnulfo [3];
Drago-Serrano, María Elisa [4]; Godínez-Victoria, Marycarmen [1].

[1]Sección de Estudios de Posgrado e Investigación, Escuela Superior de Medicina, Instituto Politécnico Nacional. [2]FES Iztacala, UNAM. [3]Instituto de Física, UNAM. [4]Laboratorio de Homeostasis Intestinal, Depto. Sistemas Biológicos UAM-Xochimilco.

E-mail: blaze6400@gmail.com

With age, the risk of developing different diseases increases, such as periodontitis, which causes the progressive destruction of the tissues supporting teeth, which ultimately results in the loss of teeth and the failure of dental implants, as well as a subsequent deterioration of oral function and quality of life. This is due to the chronic state of low-grade inflammation that develops with age and is associated with the activation of osteoclasts responsible for reabsorbing bone. The activation of the NLRP3 inflammasome is a reported mechanism by which bone resorption and loss due to age is favored. Our objective was to determine whether long-term moderate aerobic exercise can counteract alveolar bone loss by decreasing NLRP3 inflammasome activation in an aged mouse model. BALB/c mice, sedentary and exercised, were used from 3 to 12 months. The exercise program consisted of running 30 min/day on an endless treadmill, 5 times a week. Bone resorption was investigated by microcomputed tomography (microCT), and the expression of NLRP3, IL-1β, and Caspase-1 was measured by immunohistochemistry, while Th1/Th2/Th17 cytokines were evaluated by cytometry. The trained mice showed a marked reduction in alveolar bone loss, associated with increased bone density and volume, lower expression of TNF-α and IL-17, and increased IL-10 expression. A reduction in the expression of the NLRP3 inflammasome and its components in periodontal tissues was also observed. The findings indicate that moderate aerobic exercise could improve age-associated inflammation and decrease bone loss, at least in part, by suppressing the NLRP3 inflammasome.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Dietary antigens gradually inactivate CD4+ T cells to produce oral tolerance

Albarrán-Godínez, Adrián [1 2]; Delgado-Zaldívar, Diego [1 2];
Suárez-Rojas, Gerardo [1 2]; Salcedo-Magaña, Patricia [1 3];
Guerra-Arias, Mónica Liliana [1 4]; Rubio-, Rosa M [1];
Madera-Salcedo, Iris K [1]; Rosetti, Florencia [1]; Crispín, José C [1 4].

[1]Departamento de Inmunología y Reumatología, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Ciudad de México, México. [2]Programa de Doctorado en Ciencias Biomédicas, UNAM, Ciudad de México, México. [3]Posgrado en Ciencias Bioquímicas, UNAM, Ciudad de México, México. [4]Tecnológico de Monterrey, Escuela de Medicina y Ciencias de la Salud, Monterrey, Monterrey, México.

E-mail: adrianbiomed@gmail.com

CD4 T cells specific to dietary antigens become inactivated through different mechanisms that include deletion, anergy, and acquisition of regulatory capacities. The factors that influence these different outcomes are not known. The aim of this work was to analyze the role of antigen-associated factors (e.g., concentration, frequency, TCR affinity) in determining CD4 T cell fates during oral tolerance induction. We adoptively transferred naïve CD45.1 FoxP3GFP OT-II CD4 T cells and fed the mice with different regimes of the model antigen ovalbumin (OVA) recognized by OT-II cells. A single dose of OVA fed to the mice induced OT-II proliferation accompanied by upregulation of FoxP3, peaking at 48 hours after OVA administration. At later time points, FoxP3+ cells decreased and anergic cells gradually became the dominant population. Fate mapping experiments revealed Foxp3 expression is unstable, leading to adoption of an anergic phenotype. Ultimately, cellular death followed until adoptively transferred cells were not detected. Repetitive administration of OVA induced the expression of anergy-associated molecules (e.g., Ctla-4, Nrp-1) and transcription factors (e.g., TOX, Helios) in a dose-dependent manner, suggesting that repetitive exposure to cognate antigen gradually induces a more profound CD4 T cell inactivation. Additionally, repetitive exposure to OVA promoted homing of anergic CD4 T cells into the lamina propria. However, tolerance was a short-lived process, as its effect was not detected 3 weeks after OVA was discontinued. In vitro examination of tolerogenic fates using ligands of different affinities showed that weak TCR activation was associated with the development of anergy. Collectively, these results indicate that induction of oral tolerance is established through a gradual process where CD4 T cells acquire FoxP3 expression, progress into an anergic phenotype and are ultimately deleted. These results denote oral antigens is a dynamic process, in which T cells modulate their function according to antigen persistence and affinity.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

CD43 impacts the immunopathology of tuberculosis according to the host's inflammatory phenotype

Alemán-Navarro, Estefanía [1 2]; López-Torres, Manuel Othoniel [3];
Mata-Espinosa, Dulce Adriana [3]; Barrios-Payán, Jorge Alberto [3];
Flores-Alcántar, Ángel Francisco [1]; Melchy-Pérez, Erika Isabel [1];
Hernández-Pando, Rogelio Enrique [3]; Rosenstein-Azoulay, Yvonne Jane [1].

[1]Instituto de Biotecnología, UNAM Campus Morelos, Departamento de Medicina Molecular y Bioprocesos, Cuernavaca, Morelos, México. [2]Posgrado en Ciencias Bioquímicas, UNAM. [3]Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Sección de Patología Experimental, Ciudad de México, México.

E-mail: estefania.aleman@ibt.unam.mx

After the COVID-19 pandemic, tuberculosis (TB) has re-emerged as the leading global cause of death due to a single infectious agent. Approximately one-fourth of the world's population is latently infected with Mycobacterium tuberculosis (Mtb), with 5-10 % progressing to active disease over their lifetime. The interplay between Mtb and the host's immune system determines TB's evolution and outcome. Notably, the immune response of exposed individuals is heterogeneous, with individuals in developing countries often exhibiting a more anti-inflammatory response against Mtb due to environmental factors. Therefore, exploring the immunopathology of TB in different inflammatory contexts is crucial for developing effective treatments. The interaction between Mtb and the immune system triggers a complex inflammatory response mediated by multiple receptors in immune cells. One key receptor is CD43, a long transmembrane sialomucin that is abundantly expressed on the surface of immune cells, and monitors the extracellular environment to direct adhesion, activation, proliferation, survival, and cytokine/chemokine secretion processes. In this study, we evaluated the influence of CD43 on the immune response against Mtb infection in vivo using mouse models. Pulmonary Mtb load, lung histopathology, systemic and pulmonary cytokine and chemokine production, and survival, were evaluated in WT and CD43KO mice with a prototypic anti- (BALB/c) or pro-inflammatory (C57BL/6) immune response. Our results demonstrate that CD43 plays a crucial role in shaping the immune response to Mtb, with its impact strongly influenced by the host's predominant immune response type. CD43 modulates the cytokine and chemokine environment, as well as the quantity and proportion of immune cell populations in the lungs, ultimately affecting the host's ability to control pulmonary Mtb burden, cellular infiltration, inflammation-induced tissue damage, and survival. Notably, the interplay between CD43 expression and the host's immune response profile (pro- or anti-inflammatory) determines the outcome of the infection.

Supported by CONAHCYT and DGAPA/UNAM.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y comparada**

Immune response of Zophoba morio larvae exposed to polyethylene terephthalate (PET) microplastics

Alva-Murillo, Patricia Nayeli [1]; Ramírez -Dávila, Kevin Said [1];
Arias-Díaz, María Fernanda [1]; Hernández-Villanueva, Hugo
Abraham [1]; González-Evaristo, Alma Margarita [1];
Sánchez-Leyva, María del Carmen; Huchin-Mian, Juan Pablo [1].

[1]Departamento de Biología, División de Ciencias Naturales y Exactas, Campus Guanajuato,
Universidad de Guanajuato, Noria Alta S/N; CP 36050, Guanajuato, Guanajuato, México.

E-mail: pn.alva@ugto.mx

The mass production of plastics such as PET generates microplastics that contaminate terrestrial biomes and can be easily ingested by various organisms. Zophoba morio larvae, known for their ability to consume polymers such as polystyrene, represents an ideal model for studying the effects of PET on invertebrates, helping to predict impacts on their immune response and survival. This study aims to evaluate the humoral and cellular immune responses in Z. morio larvae exposed to PET and to predict the potential risks of plastic pollution to invertebrates. To this end, groups of 80 larvae were fed lettuce with PET (2.5% and 5% w/w, and control group) ad libitum. The immune response was evaluated on days 1, 3, 5, 8, 16 and 32 through: (1) analysis of humoral immunity (phenoloxidase [PO], lysozyme [Lz] and catalase [Cat] activities in hemolymph), and (2) evaluation of cellular immunity via nodular body formation in histological sections and circulating hemocyte counts. Results showed that both experimental groups (2.5% and 5%) exhibited significant weight and size increases. PO peaked on day 5 (highest activity in 5% group), Lz peaked on day 8 with similar levels in both treated groups, and Cat activity remained elevated until day 16. Histologically, nodular body formation with cell migration was observed in both groups, along with time-dependent changes in differential hemocyte counts. These findings demonstrate that the ingestion of PET microplastics significantly modulates both humoral and cellular immune responses in Z. morio, suggesting the development of an adaptive mechanism to PET ingestion, with no associated mortality.

Acknowledgments: this project was supported by grants from University of Guanajuato (DAIP 74/2024) to JPHM.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Immunomodulation of P-glycoprotein to Enhance Natural Killer Cell Survival in Acute Lymphoblastic Leukemia

Álvarez-Carrasco, Pablo Arturo [1 2]; Jiménez-Hernández, Elva [3];
Medina-Sansón, Aurora [3]; Gil-Guevara, Ricardo [4];
Contreras-Ramos, Alejandra [6]; Maldonado-Bernal, Carmen [2].

[1]Universidad Nacional Autónoma de México, Doctorado en Ciencias Biomédicas, Ciudad de México, México. [2]Hospital Infantil de México Federico Gómez, Unidad de Investigación en Inmunología y Proteómica, Ciudad de México, México. [3]Hospital Pediátrico Moctezuma, Departamento de Hematología, Ciudad de México, México. [4]Hospital Infantil de México Federico Gómez, Departamento de Hemato-Oncológica, Ciudad de México, México. [5]Hospital Infantil de México Federico Gómez, Servicio de Urgencias, Ciudad de México, México. [6]Hospital Infantil de México Federico Gómez, Laboratorio de Biología Molecular, Ciudad de México, México.

E-mail: p.arturo91@gmail.com

Natural Killer (NK) cells are crucial innate immune effectors against acute lymphoblastic leukemia (ALL) due to their malignant cell-killing capacity. However, chemotherapy resistance in leukemic cells, primarily mediated by P-glycoprotein (P-gp), limits standard ALL treatment. NK cells also express P-gp, but their basal activity is insufficient to protect them from chemotherapy, leading to reduced numbers and function. Since NK cells express TLR2, and its activation in other immune cells enhances P-gp activity, we hypothesized that TLR2 activation in NK cells could boost their chemotherapy resistance, promoting survival and anti-leukemic function. Our objective was to determine if TLR2 activation enhances P-gp activity in NK cells, preventing the deleterious effects of antineoplastic agents and increasing their survival. Peripheral blood mononuclear cells (PBMCs) were isolated from pediatric ALL patients , and NK cells were enriched for immunophenotyping, P-gp, and TLR2 quantification by flow cytometry. Cells were cultured with TLR2 ligands (PCSK, LTA, MALP-2). NK cell activation was assessed by measuring IFN-γ and CD107a (cytometry/ELISA), and P-gp activity via Rhodamine 123 efflux assays by flow cytometry. Stimulation of NK cells with PCSK showed trends towards increased activation (IFN-γ, CD107a) and P-gp activity (higher Rhodamine 123 efflux), while LTA and MALP-2 were less effective. Immunophenotyping of ALL patients revealed a predominance of the CD16+ subpopulation and confirmed TLR2 expression on total NK cells and their subpopulations, with higher expression on CD16+ cells. In conclusion, TLR2 activation in NK cells, particularly with PCSK, increased P-gp activity. NK cells in ALL patients exhibit altered phenotypes and express TLR2, indicating this receptor as a potential therapeutic target. Modulating the TLR2/P-gp pathway could be a strategy to improve NK cell survival and efficacy during chemotherapy in ALL.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Discovery and Functional Characterization of Anti-TNFα Antibodies from Murine Immune Libraries

Álvarez-Fosado, Tomás Iván [1 2]; Gómez-Castellano, Keyla [1 2];
Rodríguez-Luna, Stefany [1 2]; Lujan-Torres, Carlos [1 2];
Vázquez-Leiva, Said [1 2]; Mendoza-Salazar, Ivette [1 2];
Elizarrarás-Rodríguez, Luis [1 2]; Pérez-Tapia, Sonia Mayra [1 2 3];
Almagro, Juan Carlos [1 4].

[1]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, México City, México. [2]Laboratorio Nacional para Servicios Especializados de Investigación, Desarrollo e Innovación (I+D+i) para Farmoquímicos y Biotecnológicos, LANSEIDI-FarBiotec-CONACyT. [3]Departamento de inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional. [4]Global Bio, Cambridge Massachusetts, United States.

E-mail: tomas.alvarez.fosado@gmail.com

Tumor Necrosis Factor alpha (TNFa) is a pivotal cytokine in inflammatory diseases like rheumatoid arthritis and Crohn's disease, where its overproduction drives tissue damage. Anti-TNF biologics (e.g., adalimumab) are clinically successful but face limitations in cost, immunogenicity, and variable patient response, necessitating novel antibodies with improved properties. To address this, we aimed to discover high affinity neutralizing antibodies using murine immune phage displayed libraries. BALB/c mice were immunized with recombinant human TNFa. From their spleens, RNA was isolated and used to construct immune murine library combining murine VH and VL regions. Following three rounds of phage display panning via semi-automated selection (kingfisher Flex) against decreasing biotinylated TNFa concentration (50 nM to 1 nM), a total 6 clones were identified and sequenced. One promising candidate (Clone F6) was converted into human IgG1 format and expressed in Expi293F cells. The converted IgG, D96E, showed strong binding to human TNFa with high specificity and efficiently blocked the TNFa/TNFR1 interaction in a competitive ELISA. In vitro, D96E neutralized TNFa induced cytotoxicity in L929 cells with EC50 in the same range as commercial anti-TNFa antibodies (adalimumab, infliximab). Additionally, D96E demonstrated strong binding to membrane bound TNFa. Biophysical characterization confirmed a high monomeric content (97%) by SEC and expected molecular weights of heavy and light chains by SDS-PAGE.

These results support the successfully developed and full characterization of a novel murine derived anti-TNFa antibody with comparable performance to established biologics. This work underscores the value of immunes libraries platforms for the discovery of antibody therapeutics targeting inflammatory cytokines.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Inflammation and endoplasmic reticulum stress association in bone marrow of pediatric acute lymphoblastic leukemia (B-ALL) patients

Ángeles-Floriano, Tania [1 2 3]; Román-Anguiano, Nadia [1 2 3];
Gómez-, Mauricio [4]; Rodríguez, Marco [4]; Martínez-Rodríguez,
Nancy Lucero [1]; Valle-Ríos, Ricardo [1 2 3].

[1] Federico Gomez Children’s Hospital, CDMX, Mexico. [2]Immunology and Proteomics Laboratory. Federico Gómez Children’s Hospital, CDMX, Mexico. [3]Research Division. School of Medicine, UNAM, CDMX, Mexico. [4]Science School, UNAM, CDMX, México.

E-mail: tania.angeles@yahoo.com.mx

In Mexico, B-cell acute lymphoblastic leukemia (B-ALL) is the most common cancer in pediatric patients. Treatment outcomes remain poor, with a 5-year disease-free survival rate of 61.4%, representing a national problem for the healthcare system. We previously observed that leukemic cells obtained from pediatric patients have active endoplasmic reticulum stress (ERS), as measured endoplasmic reticulum chaperone GRP78 to the cell membrane surface (csGRP78). Cellular stress has recently been observed to play an important role in inflammation. Although the response to ERS helps mitigate it, these pathways also promote inflammation in diseases such as cancer. Levels of the ISM1 ligand, as well as the cytokines IL-10, MIP-3α, IL-12(P70), IL-13, IL-17α, IL-2, IL-4, IL-5, IL-6, IL-7, IL-8, and TNFα, were analyzed in the bone marrow (BM) of n=72 patients with B-ALL at diagnosis. Data were analyzed using Prism V10 software (GraphPad). Decreased ISM1 levels were observed in individuals with B-ALL (374–627 pg/mL) compared to control individuals (673–845 pg/mL) (p=0.0007), and a negative correlation was observed between ISM1 levels and the percentage of csGRP78+ cells in B-ALL BM (r=-0.57, p=0.0223). Meanwhile, levels of IL-10, IL-13, IL-4, IL-5, IL-6, and IL-7 correlated positively and significantly with the presence of csGRP78+ cells. In addition to the observation of leukemic cells with endoplasmic reticulum stress, their correlation with interleukins IL-4, IL-5, IL-6, IL-7, IL-10, and IL-13 suggests an important role in blast proliferation, immune evasion, and modulation of the leukemic microenvironment, promoting a Th2 profile that is less effective against tumors. Furthermore, the ISM1-csGRP78 interaction is associated with inhibitory effects on cell proliferation and survival, and the negative correlation of ISM1 levels with csGRP78+ cells suggests a physiological role for ISM1. Grants: Secretaria de Salud Funding: FPIS2024-HIM-6904 to NLMR. Hospital Infantil de México Federico Góemez Grant HIM/2021/038 SSA 1781 to RVR. UNAM proyect 153/2017.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos B**

Myo1g controls B–T cell conjugate dynamics in antigen presentation.

Antonio-Buendia, Jesus Ivan [1]; Santos-Argumedo, Leopoldo [1 2].

[1]Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, Centro de Investigación sobre el Envejecimiento, CDMX, México. [2]Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, Departamento de Biomedicina Molecular, CDMX, México.

E-mail: antonioivan332@gmail.com

Myosin 1g (Myo1g) is an actin-dependent motor protein mainly expressed in immune system cells, including B cells. Previous studies have reported its involvement in processes such as migration, adhesion, and vesicle trafficking, which are crucial for the formation of the immunological synapse. However, its role in the interaction between B cells and CD4+ T cells during antigen presentation has not been thoroughly explored. In this work, we aimed to evaluate the role of Myo1g in the formation and stability of B–T cell conjugates. For this, we isolated B cells from the spleen of wild-type (WT) C57BL/6 mice and Myo1g-deficient (Myo1g-/-) mice. These B cells were activated with IL-4 and LPS and then loaded with ovalbumin (OVA). After that, they were co-cultured with CD4+ T cells obtained from OT-II transgenic mice, which are specifically reactive to the OVA 323–339 peptide. The formation of conjugates was analyzed by flow cytometry using antibodies against CD19 to identify B cells and CD4 to identify T cells. Through flow cytometry, we observed a decrease in the formation of T–B conjugates when B cells were derived from Myo1g-/- mice compared to those from WT mice. The decrease in conjugates is in agreement with previous results from our laboratory, which show a reduced proliferation of OT-II T cells when Myo1g-/- B lymphocytes perform antigen presentation. Although these results are preliminary, this information suggests that Myo1g plays a crucial role in mobilizing MHC-II molecules to the surface of APC cells; in its absence, defects occur in cytoskeleton organization and vesicle trafficking.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Characterization of the immunoglobulin lambda chain locus in native Mexican populations

Antonio-Mendoza, Kevin Jacob [1]; Martínez Barnetche, Jesus [2];
Godoy-Lozano, Elizabeth Ernestina [3].

[1]School of Public Health of México, Center for Infectious Diseases Research, Cuernavaca, Morelos, México. [2]National Institute of Public Health of México, Center for Infectious Diseases Research, Cuernavaca, Morelos, México. [3]National Institute of Public Health of México, Bioinformatics Department of Infectious Diseases, Cuernavaca, Morelos, México.

E-mail: kevinjacob11762@gmail.com

The immunoglobulin (IG) or B cell receptor is a cornerstone of the adaptive immune system and is responsible for antigen recognition and immune specificity. This specificity not only enables the neutralization of diverse pathogens and antigenic molecules but also facilitates the formation of immunological memory, a defining feature of adaptive immunity and the mechanistic basis of vaccination. IG molecules are encoded by three complex genomic loci, including the immunoglobulin lambda light chain locus (IGL) located on chromosome 22. However, the allelic diversity of these loci remains poorly characterized, largely due to their repetitive and structurally complex architecture, which poses persistent challenges for short-read sequencing technologies and reference-based genome assembly. Additionally, most existing data are derived from individuals of European ancestry, limiting our understanding of global immunogenomic diversity. To address this, we analyzed 95 whole-genome sequences from 31 native Mexican populations using a custom bioinformatic pipeline optimized for short-read data. Our analysis revealed a markedly elevated density of single-nucleotide variants (SNV) across the IGL locus relative to intergenic and genomic background averages, underscoring its high level of polymorphism. Additionally, we observed a high prevalence of the IGLV5-39 structural variant (SV), suggesting that this SV may be a common feature in the genetic landscape of native Mexican populations. These findings underscore the importance of including underrepresented populations in immunogenomic studies and demonstrate the utility of population-scale sequencing data in revealing hidden genetic diversity, thereby advancing our understanding of IG germline variation and its implications for health and disease.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Intratumoral injection EST of the human parasite T. canis increase lung metastasis

Aragón-Franco, Raúl; Ruíz-Manzano, Rocío; Nava-Castro, Karen Elizabeth; Del Río-Araiza , Víctor Hugo; Garay-Canales, Claudia Angélica; Chacón-Salinas, Rommel; Pérez-Torres, Armando; Girón-Pérez, Manuel Iván; Morales-Montor, Jorge.

[1]Laboratorio de Neuroinmunoendocrinología, Departamento de Inmunología, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, Ciudad de México, México. [2]Laboratorio de Biología y Química Atmosférica, Departamento de Ciencias Ambientales, Instituto de Ciencias de la Atmósfera y Cambio Climático, Universidad Nacional Autónoma de México, Ciudad de México, México. [3]Laboratorio de Interacciones Endocrinoinmunitarias en Enfermedades Parasitarias, Facultad de Medicina Veterinaria y Zootecnia, Departamento de Parasitología, Universidad Nacional Autónoma de México, Ciudad de México, México. [4]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional (ENCB-IPN), Ciudad de México, México. [5]Departamento de Biología Celular y Tisular, Facultad de Medicina, Universidad Nacional Autónoma de México, Ciudad de México, México. [6]Laboratorio de Inmunotoxicología, Secretaría de Investigación y Posgrado, Universidad Autónoma de Nayarit, Tepic, Nayarit, México.

E-mail: raulvir1982@gmail.com

Triple-negative breast cancer is a major health problem. Risk factors interact to increase its development. Biological agents such as helminth parasites and their EST could play an important role in tumor development. We previously demonstrated that chronic T. canis infection increases tumor size; the parasite does not invade the tumor. We decided to study whether EST affect tumor progression or metastasis. To determine the effect of intratumoral injection of purified T. cannis excreted/secreted antigens on tumor weight and size, the immune tumor microenvironment, lung metastasis, and systemic immune response in a murine model of mammary tumors. We used 8-week-old BALB/c mice, implanted 4T1 triple-negative mammary tumor cells orthotopically, divided them into three groups, 1 control without tumor, 1 control with tumor treated intratumorally with vehicle and 1 with tumor and intratumoral treatment with EST. We measured and weighed the tumors, analyzed the lungs, measured immune cells, cytokines and tumor cells, GLP, spleen and serum cytokines TNF-α, IFN-ɣ, IL-4, IL-5 and IL-10 and VEGF. Mice injected intratumorally with EST showed no changes in tumor size or weight; lung metastasis was increased. EST did not modulate tumor immune cells, spleen, or LPGs. Metastasis was increased in animals treated with TES compared with controls, accompanied by increas We found no modulation of tumor size, intratumoral immune population, spleen, or LPGs. The tumor microenvironment and lung metastasis were modified by EST, with VEGF increasing. Lung damage demonstrated that helminth infection and cancer increase tumor progression and metastasis.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Cytotoxic and Immunomodulatory Effects of the Immunopotentiator RTLV-1424 on HER2+ Breast Cancer Cells

Aranda-Nieto, Karol [1 2]; Cotés-Morales, Víctor Adrián [1]; Rocha-Zavaleta, Leticia [3]; López-Macías, Constantino [1].

[1]Instituto Mexicano del Seguro Social, Centro Médico Nacional Siglo XXI, UMAE Hospital de Especialidades, Unidad de Investigación Médica en Inmunoquímica, México City, México.
[2]Universidad Nacional Autónoma de México, Facultad de Química, México City, México.
[3]Universidad Nacional Autónoma de México, Instituto de Investigaciones Biomédicas, Departamento de Biología Molecular y Biotecnología, México City, México.

E-mail: karol.na2204@gmial.com

Breast cancer arises from the abnormal proliferation of mammary epithelial cells and remains the most prevalent and lethal cancer diagnosed among women worldwide. The HER2+ subtype (characterised by overexpression of the HER2 protein or amplification of the HER2 gene) exhibits increased proliferative and metastatic potential, accounting for 15–20% of invasive cases (19.3% at IMSS, Mexico). Although monoclonal antibody therapies have improved patient outcomes, the development of novel treatment strategies remains essential. RTLV1424, an immunopotentiator derived from Salmonella Typhi pathogen-associated molecular patterns, activates the innate immune system via TLR2/TLR4 and promotes Th1 and cytotoxic T-cell responses. However, its direct effects on tumour cells had not been previously characterised. This study evaluated the impact of RTLV-1424 on the viability, proliferation, and migration of MCF-7 breast cancer cells (a HER2+ subtype cell line) as well as its potential to modulate tumour-induced immunosuppression. MCF-7 cells were cultured in vitro and treated with RTLV1424 for 24, 48, and 72 hours. Trypan blue exclusion assays revealed that RTLV-1424 reduced cell viability and overall cell number. Furthermore, flow cytometry analysis using carboxyfluorescein succinimidyl ester (CFSE) demonstrated a marked decrease in cell proliferation. However, no significant effect was observed on cell migration. Notably, preliminary co-culture experiments indicated that RTLV-1424 reduced the tumour cells ability to suppress T-lymphocyte proliferation, as assessed by flow cytometry. In conclusion, RTLV-1424 exerts cytotoxic effects on HER2+ MCF-7 cells by reducing their viability and proliferation. Additionally, it appears to attenuate their immunosuppressive properties. These findings support the potential of RTLV-1424 as a novel immunotherapeutic candidate for the treatment of HER2+ breast cancer.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Immunomodulatory and Cytotoxic Effect of Immunostimulant RTLV-1424 over Luminal Breast Cancer Cells

Aranda-Nieto, Karol [1 2]; Cortés-Morales, Víctor Adrián [1];
Rocha-Zavaleta, Leticia [3]; López-Macías, Constantito [1].

[1]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México.
[2]Facultad de Química, Universidad Nacional Autónoma de México, México City, México.
[3]Departamento de Biología Molecular y Biotecnología, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, México City, México.

E-mail: karol.na2204@gmail.com

Breast cancer results from the anomalous proliferation of mammary cells; it is the cancer with the highest incidence and mortality diagnosed in women worldwide. The most prevalent subtype is Luminal A (50-70%), defined by being positive for estrogen receptor (ER+) and progesterone receptor (PR+) and a low proliferation index. It carries the best prognosis and responds favorably to hormonal therapy. However, a negative response to treatment in some patients has been associated with low immune infiltrate and an immunosuppressive cellular phenotype, highlighting the need for new therapeutic strategies.The immunopotentiator RTLV14-24, a Salmonella -derived pathogen-associated molecular pattern, activates innate immune response (via TLR2/TLR4) and induces a Th1 and cytotoxic T-lymphocyte activity; nevertheless, its immunomodulatory effect over tumor cells remained unknown. This study evaluated the effect of the RTLV14-24 on the viability, proliferation, and migration of the MCF-7 tumor cell line (ER+, PR+); as well as whether RTLV14-24 treatment modulates immunosuppressive response of these tumor cells. MCF-7 cells were cultured in vitro and treated with RTLV14-24 for 24, 48, and 72 hours. We found that RTLV14-24 decreases viability and cell number in culture, as assessed by trypan blue dye. The treatment also diminished the proliferation of tumor cells stained with carboxyfluorescein succinimidyl ester, evaluated by flow cytometry. However, the treatment did not affect their migration capacity. Surprisingly, treatment with the immunopotentiator diminishes the ability of tumor cells to inhibit T-lymphocyte (CD3+, CD4+ and CD8+) proliferation in an in vitro co-culture system, evaluated by flow cytometry. In this work, we preliminary conclude that RTLV-1424 exerts a cytotoxic effect on MCF-7 cells, because reduced their viability, proliferation, and migration. It also diminishes their immunosuppressive capacity over T cells. These findings suggest its potential as a new therapeutic strategy in breast cancer.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Association of SNVs in CTLA-4 and Galectin-3 in SLE in Southeastern Mexico.

Aranda -Uribe, Iván Sammir [1]; López-Briceño, Isaac Alberto [2];
Nakazawa-Ueji, Yumi Elena [3]; Angulo- Ramírez, Angélica Vanesa [4];
Gutiérrez-Pérez, Leticia [5]; López-Torres, Liliana Esther [6];
Valencia-Pacheco, Guillermo [7].

[1]Universidad Autónoma del Estado de Quintana Roo, Departamento de Ciencias Farmacéuticas, Laboratorio de Biología Celular y Molecular, Ciudad de Chetumal, Quintana Roo, México. [2]Universidad Autónoma de Yucatán, Centro de Investigaciones Regionales "Dr. Hideyo Noguchi", Mérida, Yucatán, México. [3]Universidad Autónoma de Yucatán, Centro de Investigaciones Regionales "Dr. Hideyo Noguchi", Mérida, Yucatán, México. [4]Hospital General Dr. Agustín O'Horán de Mérida, Mérida, Yucatán, México. [5]Centro Médico de Chetumal, Servicio de Reumatología, Othón P. Blanco, Quintana Roo, México . [6]Hospital General IMSS-Bienestar de Chetumal, Othón P. Blanco, Quintana Roo, México. [7]Universidad Autónoma de Yucatán, Centro de Investigaciones Regionales "Dr. Hideyo Noguchi", Mérida, Yucatán, México.

E-mail: ivan.aranda@uqroo.edu.mx

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by the interplay of environmental and genetic factors that lead to chronic inflammation and disruptions in peripheral immune tolerance. T cells play a crucial role in the disease's pathogenesis by secreting cytokines that activate B cells. These activated B cells, in turn, produce autoantibodies that can harm various tissues and organs. Additionally, both experimental models and patient studies have shown that abnormalities in the expression or function of the inhibitory receptor CTLA-4 can result in the polyclonal proliferation of T cells and contribute to the development of SLE. The CTLA-4 gene contains single nucleotide variants (SNVs) that can lead to receptor abnormalities. Specific SNVs, such as rs17268354, +49 A/G, and CT60 G/A, have been linked to an increased susceptibility to SLE in Asian populations. However, their impact on Latin American populations is not well understood. The objective of this study was to determine whether these three CTLA-4 SNVs are associated with an increased risk of developing SLE in populations from the Yucatan Peninsula, México. We recruited 120 patients with SLE and 120 control subjects for this purpose. In our study, DNA was extracted from blood samples, and genotyping was performed using TaqMan assays. We assessed Hardy-Weinberg equilibrium and analyzed genotypic associations with SLE using SNPStats software. Additionally, we conducted immunophenotyping via flow cytometry to identify lymphocyte populations and measure galectin-3 expression in peripheral blood. Preliminary data suggest that the rs17268354 variant and the GAG haplotype may be associated with an increased susceptibility to SLE in the population from the Yucatán Peninsula, Mexico. Furthermore, one patient carrying the risk allele exhibited decreased levels of galectin-3, although it remains uncertain whether this finding is related to the treatment effect.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Effect of a defensin (Def1.3) from Chagas disease vector against Trypanosomatids

Arce -Aceves, María Fernanda [1 2]; Santiago Santos, Marco Gael [1 3];
Martínez Martínez, Ignacio [1]; Espinoza Gutiérrez, Bertha Josefina [1].

[1]Instituto de Investigaciones Biomédicas, UNAM. [2]Posgrado en Ciencias Bioquímicas, UNAM.
[3]Facultad de Ciencias, UNAM.

E-mail: mariferarce@ciencias.unam.mx

The Trypanosomatidae family includes human pathogens such as Trypanosoma cruzi, the causative agent of Chagas disease, and Leishmania spp., responsible for leishmaniasis. These diseases are life-threatening, and their main route of transmission is through insect vectors. Although treatments exist for these diseases, they often have variable efficacy and toxic side effects. Therefore, alternative therapies are being explored, such as the use of antimicrobial peptides (AMPs), which are molecules of the innate immune response with a broad spectrum of activity due to their diverse mechanisms of action (disruption of the plasma membrane, binding to intracellular targets, and immunomodulation). Defensins represent a family of cysteine-rich AMPs. In our laboratory, we have studied the defensins from Triatoma pallidipennis, one of the vectors of T. cruzi. We obtained a recombinant defensin (Def1.3), which was found to alter the morphology of these parasites. Therefore, it is of interest to study the antiparasitic potential of Def1.3 and investigate the mechanisms of action involved. So far, it has been found that this defensin reduces the growth and viability (metabolic activity) of L. major and L. mexicana by approximately 60%, but not that of T. cruzi. It was also observed that Def1.3 alters the plasma membrane permeability of L. major and L. mexicana. Electron microscopy revealed that Def1.3 causes discontinuities in the plasma membrane and induces the formation of vacuoles, as well as the loss of nuclear integrity. Additionally, viability and hemolysis assays showed that Def1.3 is not cytotoxic. So far, Def1.3 has demonstrated significant therapeutic potential, and further studies on its mechanisms of action are needed.

We acknowledge the financial support of NUATEI-IIB and DGAPA 211723.

TRABAJO NO PRESENTADO

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

A novel methodology for NETs visualization under light microscopy

Arce-Mendoza, Alma Yolanda; Muñiz-Buenrostro, Antonio;
Montes-Zapata, Edgar.

[1]Servicio de Inmunología, Facultad de Medicina, Universidad Autónoma de Nuevo León, Monterrey, Nuevo León, México.

E-mail: aya.mayola@yahoo.com

Neutrophils are key players in innate immunity, deploying neutrophil extracellular traps (NETs) to ensnare and eliminate pathogens. While NETs are typically visualized using fluorescence or electron microscopy, these methods are costly and require specialized equipment, limiting their accessibility. This study presents a simple, economical alternative using safranin staining and light microscopy to detect NETs, enabling wider adoption in resource-limited settings.

Neutrophils were isolated from healthy donors, with purity (>98%) and viability (>95%) confirmed by trypan blue exclusion. Respiratory burst activity, assessed via nitroblue tetrazolium (NBT) reduction, validated functional integrity. NETs formation was induced by phorbol myristate acetate (PMA) or Staphylococcus aureus, followed by staining with safranin or DAPI for comparative analysis. Unstimulated neutrophils showed no NETs, whereas stimulated cells exhibited distinct NETs structures—diffuse clouds (PMA) or filamentous networks (S. aureus). Safranin effectively highlighted these structures under light microscopy, matching DAPI's fluorescence-based detection. A dual NBT-safranin stain further confirmed DNA specificity, with NETs emerging alongside formazan crystals after prolonged stimulation.

The safranin method produced stable, high-contrast images without the fading issues of fluorescent dyes, and slides remained analyzable for over two weeks. Though additional markers (e.g., myeloperoxidase, citrullinated histones) are needed for definitive NETs confirmation, this approach provides a reliable preliminary screening tool. By circumventing the need for expensive instrumentation, this technique expands NETs research capabilities, particularly in low-resource laboratories. Its simplicity and cost-effectiveness democratize the study of neutrophil function, fostering broader participation in immunological research and paving the way for innovative, affordable diagnostic applications.

This work underscores the potential of classical histochemical dyes in modern immunology, bridging the gap between advanced research and practical, scalable methodologies.

ISSN: 2007-3380

Revista Bio Ciencias

https://revistabiociencias.uan.edu.mx

Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y comparada**

Analysis of microplastic contamination in the grasshoper (Sphenarium purpurascens) and exploration of its effects on the immune response

Arias-Díaz, María Fernanda [1 2]; Aguilera -Corona, Margarita del Carmen [1];
Martínez -Jiménez, David [2]; Hernández-Villanueva, Hugo Abraham [2];
Vega-Muñoz, Frida [1]; Rodríguez -Solís, Mayra Cecilia [1];
Sánchez-Leyva, Ma. del Carmen [2]; Contreras-Garduño, Jorge [3];
Huchin -Mian, Juan Pablo [2]; Alva -Murillo, Patricia Nayeli [1].

[1]Laboratorio de Inmunomicrobiología Molecular, Departamento de Biología, División de Ciencias Naturales y Exactas, Campus Guanajuato, Universidad de Guanajuato, Noria Alta S/N; CP 36050, Guanajuato, Guanajuato.. [2]Laboratorio de Ecología, Departamento de Biología, División de Ciencias Naturales y Exactas, Campus Guanajuato, Universidad de Guanajuato, Noria Alta S/N; CP 36050, Guanajuato, Guanajuato.. [3]ENES, Unidad Morelia, UNAM, Antigua Carretera a Pátzcuaro No. 8701, Col. Ex Hacienda de San José de la Huerta; CP 58190, Morelia, Michoacán.

E-mail: mf.ariasdiaz@ugto.mx

Plastic pollution is a major environmental issue, with ubiquitous impacts across ecosystems. When plastics are exposed to biotic or abiotic factors, they degrade into smaller particles known as microplastics (MPs), defined as fragments less than 5 mm in size. Insects may ingest MPs while feeding, potentially compromising their immune function. In the commercial edible Mexican grasshopper (Sphenarium purpurascens), a species of high cultural and gastronomic relevance, MPs have already been detected, raising concerns about their potential biological effects. This study aimed to isolate, quantify, and characterize MPs present in S. purpurascens collected from three distinct environments, and to experimentally assess their impact on the insect's immune response. For this, grasshoppers were separated into four diet groups with 0, 2.5, 5, or 10% polyethylene terephthalate MP´s (w/w) for 14 days, and four immune-related variables were evaluated using hemolymph samples. The hemocyte count revealed a progressive increase, with statistically significant elevations beginning on day 3 post-ingestion (PI) and peaking on day 7 PI, suggesting cellular immune activation. Total protein concentration, determined via the Bradford assay, declined with prolonged exposure, and females exhibited significantly higher values than males. pro-Phenoloxidase activity, measured via an enzymatic reaction using alpha chymotrypsin, showed a marked reduction from day 3 PI, although a transient increase was observed on day 5 PI in the group exposed to 10% MPs. Lastly, lysozyme activity, assessed using Micrococcus lysodeikticus, showed a sustained decrease beginning on day 2 PI, after adjusting the data to meet the assumptions of the statistical analysis. The isolation, quantification, and characterization of MPs present in grasshoppers from distinct pollution environments remain to be determined. To date, these results suggest that the duration of microplastic exposure plays a key role in modulating immune responses in S. purpurascens.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

STAT6 Inhibition Promotes iTreg Stability and Therapeutic Efficacy in Inflammatory Bowel Disease

Arroyo-Olarte, Rubén D. [1 2]; Pérez-Noriega, Flaubert A. [1];
Correa-Pérez, María Fernanda [1]; Mejía-Muñoz, Aranza [1]; I.-
Terrazas, Luis [1 3]; León-Cabrera, Sonia [1 2].

[1]Unidad de Biomedicina. Facultad de Estudios Superiores-Iztacala. Universidad Nacional Autónoma de México. Av. De los Barrios 1, Los Reyes Iztacala, Tlalnepantla, Edo. De México. México 54090.ratorio de Inmunidad tumoral (L-16). [2]Carrera de Médico Cirujano, Facultad de Estudios Superiores Iztacala, Universidad Nacional Autónoma de México, Av. De los Barrios 1, Los Reyes Iztacala, Tlalnepantla, Edo. De México. México 54090.. [3]Laboratorio Nacional en Salud, Facultad de Estudios Superiores-Iztacala, Universidad Nacional Autónoma de México, Edo. De México, Tlalnepantla, México.

E-mail: rdao.1983@gmail.com

Induced regulatory T cells (iTregs)-based immunotherapy holds exciting potential against inflammatory diseases by restoring immune tolerance. However, clinical use of iTregs is hampered by their instability leading to diminished immunosuppressive potency. STAT6 signaling has been implicated in the downregulation of Foxp3, the key transcription factor defining Treg identity. In this study, we assessed how pharmacological inhibition of STAT6 phosphorylation influences iTreg differentiation, stability, and function in vitro and in vivo. Naïve CD4□ T cells were differentiated into iTregs under standard conditions followed by expansion with IL-2 in the absence or presence of the STAT6 inhibitor, AS1517499 (AS-iTregs). STAT6 inhibition promoted sustained expression of suppressive markers Foxp3, CD25, PD-1, and CTLA-4 for up to 10 days, even under inflammatory conditions. AS-iTregs also exhibited increased mRNA levels of Foxp3, IL-10, TGF-β, and PD-1, while showing reduced expression of inflammation and epigenetic instability-related genes IL-6, IL-1β, and DNMT1. Additionally, AS-iTregs displayed a higher suppressive capacity in vitro, suggesting enhanced function and epigenetic stability. In the AOM/DSS colitis model, the adoptive transfer of AS-iTregs alleviated disease severity, preserved mucosal architecture, and increased goblet cell numbers. Histological analysis showed reduced epithelial damage and inflammation relative to controls. Notably, AS-iTregs did not promote tumorigenesis in a colitis-associated cancer model. Moreover, systemic administration of AS1517499 during acute colitis enhanced Treg expansion, activation, and suppressive function in vivo. Altogether, these findings demonstrate that STAT6 inhibition stabilizes iTregs and enhances their therapeutic efficacy, offering a promising strategy to improve Treg-based therapies for inflammation-driven diseases.

Acknowledgements: UNAM-PAPIIT (grant number IN215421, IN201724 and IV200425).

Keywords: colitis, STAT6, Tregs, Foxp3, AS1517499.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

Molecular characterization of expanded allospecific Tr1 cells with therapeutic potential in transplantation

Arteaga-Cruz, Saúl; Cortés-Hernández, Arimelek; Alvarez-Salazar, Evelyn; García-Cruz, Axel; Soldevila-Melgarejo, Gloria.

[1]instituto De Investigaciones Biomédicas, UNAM, Departamento De Inmmunología, Ciudad De México, México. [2]instituto De Investigaciones Biomédicas, Unam, Laboratorio Nacional De Citometría De Flujo, Ciudad De México, México.

E-mail: saul.ac@iibiomedicas.unam.mx

Tr1 cells have emerged as promising tools to induce long-term tolerance, especially in allogeneic stem cell and solid organ transplantation. While commonly identified by co-expression of CD49b and LAG-3 along with high IL-10 production, recent evidence highlights the need for more detailed characterization—including co-inhibitory and chemokine receptor profiles—to ensure therapeutic efficacy. Moreover, the proinflammatory environment after transplantation may impair Treg function, making Tr1 stability a critical factor. Here, we present a novel protocol for the long-term in vitro expansion of highly purified allospecific Tr1 cells (Exp-allo Tr1). These cells produced IL-10 at levels above 90% and maintained expression of CD49b, LAG-3, and the co-inhibitory receptors PD-1, CTLA-4, TIM-3, TIGIT, and CD39. High-dimensional analysis revealed a distinct expression profile compared to conventional T cells, including overexpression of IL-10, CD39, CTLA-4, and LAG-3. Additionally, Exp-allo Tr1 cells expressed chemokine receptors relevant for allograft homing and tolerance induction (CCR2, CCR4, CCR5, and CXCR3), with reduced expression of CCR7. Functionally, they effectively suppressed allospecific but not third-party T cell responses, even after extended exposure to proinflammatory cytokines. Transcriptomic analysis revealed overexpression of BATF, a transcription factor critical for Tr1 differentiation. BATF promotes IL-10 expression both directly and indirectly through induction of c-MAF and IL-21, and regulates genes such as PRDM1 and HIF1A, contributing to Tr1 stability under inflammatory conditions. Although IRF1 was not significantly overexpressed, its cooperative role with BATF may support epigenetic regulation of the IL-10 locus. These findings demonstrate the successful expansion of functionally stable Exp-allo Tr1 cells with therapeutic potential. The RNA expression profile suggests that BATF is a central regulator of their identity and function. Future studies using BATF-deficient Exp-allo Tr1 cells will be crucial to confirm its role in maintaining their phenotype and suppressive capacity under inflammatory conditions and to advance Tr1-based immunotherapies in transplantation.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx

Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Development and validation of immunoassays for the characterization of IgG’s and IgA’s in dairy cattle

Autran-Martínez, Marcela; Ramírez-Andoney, Vianey;
Escobar-Chavarria, Omar; Acevedo -Jiménez, Gabriel Eduardo;
Iturbe-Requena, Sandra Lizeth; Akbarin-Akbarin, Mohammad
Mehdi; Hidalgo-Ruiz, Mario; García-López, Esperanza.

[1]Facultad de Estudios Superiores Cuautitlán, UNAM, Departamento de Ciencias Biológicas, Sección Salud Animal, México, Estado de México, México. [2]Facultad de Medicina Veterinaria y Zootecnia, UNAM, Departamento de Genética Molecular, Ciudad Universitaria, Ciudad de México, México. [3]Universidad Autónoma de Chiapas, Dirección General de Investigación y Posgrado, Chiapas, México.

E-mail: marcelaautranmartinez@cuautitlan.unam.mx

The determination of serum gamma globulins in newborn calves is essential for assessing the transfer of passive immunity through colostrum. Deficient immunity transfer increases the newborn’s vulnerability to infectious diseases, concomitantly increasing neonatal mortality. This study focused on the development and validation of serological immunoassays for quantifying IgGs using serum samples. A bank of 38 calf sera and colostrum from their mothers collected during February 2024-2025 was established. An innovative methodology was also developed for obtaining polyclonal antibodies directed against specific IgGs. Three quantification techniques were validated: two for total gamma globulins and one for specific IgGs using a protein micromethod. The purified IgGs were characterized by SDS-PAGE electrophoresis, confirming their identity by comparison with markers of known molecular weight. The results revealed significant differences between pre- and post-colostrum sera. The average gamma globulin concentration was 9.3 mg/mL, with the following distribution: 14 calves were identified with concentrations above 15 mg/mL, 4 calves between 10-15 mg/mL, 15 with 5 mg/mL, 2 with 10 mg/mL, and 3 calves without detectable gamma globulins. A low IgG concentration was observed at birth, increasing at 48 hours and reaching a maximum concentration at 72 hours post-colostrum feeding, confirming the direct impact of colostrum ingestion on serum gamma globulin levels. Statistical analysis yielded a Cohen’s Kappa coefficient of concordance of 0.67 for qualitative/semi-quantitative tests and a KI of 0.9 for assay effectiveness in the comparison between techniques. Surprisingly, no significant difference was found between low gamma globulin concentrations and the presence of concomitant diseases. This project validated valuable diagnostic tools for characterizing the bovine immune response, with practical applications aimed at improving the immune status of newborn calves and herd productivity indices.

This project was supported by PIAPIME 2.11.11.24. 1: “Characterization of the bovine immune response from samples obtained from the CEA-FESC”.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Modulation of immunological gene expression in sheep supplemented with a polyherbal additive

Ávila-Estrada, Mario Iván [1]; Martínez-Cortés, Ismael [2];
Mendoza-Martínez, Germán David[1]; Lizarazo-Chaparro,
Augusto César [3]; Campillo-Navarro, Marcia [4].

[1]Doctorado en Ciencias Agropecuarias, UAM-X. [2]Departamento de Producción Agrícola y Animal, UAM-X. [3]Centro de Enseñanza Práctica e Investigación en Producción y Salud Animal, FMVZ-UNAM. [4]Oficina de Apoyo Sistemático para la Investigación Superior, Instituto Nacional de Cardiología.

E-mail: mvzmarioavila@gmail.com

Polyherbal supplements have gained increasing importance due to their metabolite content with antioxidant, anti-inflammatory, or immunomodulatory effects. Supplementation with polyherbal products in cattle has been associated with a lower incidence of mastitis, abortions, and respiratory diseases; however, their effect on essential metabolic processes and the immune response in sheep is unknown. Therefore, the expression of genes involved in various metabolic and immunological processes was evaluated in postpartum sheep supplemented with a polyherbal additive formulated with Ocimum sanctum and Phyllanthus emblica (Power-C®). Nine pregnant East Friesian x Pelibuey ewes were divided into a control group (n=5) and a supplemented group (n=4), which received 3 g/day of Power-C® from 14 days before lambing to day 45 postpartum. Blood samples were collected every two weeks from lambing to day 42 postpartum for RNA extraction and microarray analysis. Differentially expressed genes were identified by calculating Z-scores and performing functional enrichment analysis. In the supplemented sheep, a decrease in the expression of genes involved in mitogen-activated protein kinase (MAPK) pathways (MAP2K1, MAPK14, ALK, GRB2, and MUC1) was detected, while an increase in the expression of STAT1, STAT3, IL2RA, IL2RG, SOCS2, and ROR genes was observed, compared to the control group. These results suggest that supplementation with Power-C® could exert anti-inflammatory activity through the decrease of IL-1β and TNF-α, in addition to promoting the activation of the JAK-STAT pathway and favoring the differentiation of Th1 and Th17 lymphocytes, promoting an effective immune response against pathogens in sheep.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Evaluation of B10 regulatory cells in idiopathic inflammatory myopathies.

Ayala-Rodríguez, Andrea Monserrat [1 2]; Reyes Huerta, Raúl
Fernando [2]; Velásquez-Ortiz, María Guadalupe [2];
Mandujano-López, Jorge Vladimir [1 2]; Bravo Ramírez, Bruno [4];
Gómez-Martín, Diana [5]; Torres-Ruíz, José Jiram [5];
Meza Sánchez, David Eduardo [6]; Sandoval Montes, Claudia [3];
Maravillas Montero, José Luis [2].

[1]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Posgrado en Inmunología, Ciudad de México, México. [2]Instituto de Biotecnología, Universidad Nacional Autónoma de México, Departamento de Medicina Molecular y Bioprocesos, Cuernavaca, Morelos, México. [3]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Departamento de Inmunología, Ciudad de México, México. [4]Centro de Investigación y Estudios Avanzados, Instituto Politécnico Nacional, Posgrado en Biomedicina Molecular, Ciudad de México, México. [5]Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Departamento de Inmunología y Reumatología, Ciudad de México, México. [6]Red de Apoyo a la Investigación, Coordinación de la Investigación Científica. Universidad Nacional Autónoma de México, Ciudad de México, México.

E-mail: andrea.arhb@gmail.com

Idiopathic inflammatory myopathies (IIM) are a group of autoimmune diseases that primarily affect skeletal muscle, as well as other organs such as the skin, lungs, and heart, leading to comorbidities such as cancer and interstitial lung disease. Multiple immune system cells contribute to the immunopathogenesis of these diseases, including B cells (CD19+), which play a crucial role in autoantibody production and immune response modulation. This is mainly due to regulatory B cells' ability to produce IL-10 (B10), a function essential for suppressing excessive inflammatory responses. This study aims to determine whether there is a reduction in the frequency and functionality of B10 cells compared to healthy individuals, which may contribute to the progression and severity of IIM. Blood samples from healthy individuals and IIM patients were analyzed using flow cytometry and IL-10 production assessment through cell culture to address this. Differences in B-cell frequencies were evaluated using the Mann-Whitney U test. An initial analysis revealed an increase in total B cells in IIM patients. However, when examining subpopulations, a decrease in memory B cells (CD27+CD38-), known for their IL-10 production capacity, was observed. Further analysis of B10 cells demonstrated a reduction in this population and decreased IL-10 production in IIM patients compared to healthy individuals. In conclusion, patients with idiopathic inflammatory myopathies exhibit a reduction in the frequency and functional capacity of peripheral blood regulatory B cells, particularly B10 cells. This deficiency may contribute to the chronic inflammation characteristic of the disease and the development of various complications.

Funded by PAPIIT-DGAPA-UNAM PAPIIT-IN215424.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Evaluation of candidate inducers of the trained immune response in bovine PBMC’s

Báez-Magaña, Marisol [1]; Zendejas Ramírez, Miriam [1];
López-Meza, Joel Edmundo [1]; Ochoa-Zarzosa, Alejandra [1].

[1]Universidad Michoacana de San Nicolás de Hidalgo, Centro Multidisciplinario de Estudios en Biotecnología-FMVZ, Morelia, Michoacán, México.

E-mail: mbaez@umich.mx

Trained immunity (TI) refers to the capacity of innate immune cells to develop a non-specific, long-lasting enhanced response following an initial exposure to a pathogen or stimulus. TI is mediated by epigenetic and metabolic reprogramming that offers broad-spectrum protection. TI has gained attention as a potential strategy to combat antimicrobial resistance (AMR), a significant global public health threat. The overuse of antibiotics in livestock, particularly for disease prevention and growth promotion, contributes significantly to the emergence and spread of resistant bacteria, which can be transmitted to humans. As an innovative alternative to reduce antibiotic dependency, TI-inducing agents could enhance the innate immune response in cattle. This study aimed to evaluate the potential of different TI inducers to improve immune function in calves. Peripheral blood mononuclear cells (PBMCs) were isolated from five calves aged 6 to 12 months. Two TI-inducing molecules were tested: Brucella abortus RB51® vaccine and laminarin (β-glucan derived from algae). To assess the induction of trained immunity, production of pro-inflammatory cytokines TNF-α, and IL-6, and reactive oxygen species (ROS) was measured at three time points: 24 hours after the first stimulus, on day 6 (after a resting phase), and 48 hours after a secondary challenge with lipopolysaccharide (LPS). Results showed that laminarin (5 μg/ml) increased IL-6, TNF-α, (~1.5 and 10-folds, respectively) and ROS production in PBMCs compared to controls. Similarly, the RB51 (MOI:1:2) vaccine enhanced IL-6, TNF-α (~16 and 4-fold, respectively) and ROS production after immune training. These findings suggest that both laminarin and RB51 vaccine can induce trained immunity in vitro in bovine PBMCs and may serve as promising alternatives to reduce antibiotic use in cattle by boosting innate immune defense mechanisms.

Área del artículo: **Alergias y autoinmunidad**

Potential Roles of FAM3A, SPG7, and MDH1 in Asthma Mitochondrial Regulation

Báez-Solano, Julisa Itzel [1]; Álvarez-González, Marisol [1 2].

[1]Facultad de Ciencias, UNAM, Ciudad de México, México. [2]Instituto Nacional de Enfermedades Respiratorias “Ismael Cosío Villegas”, Inmunofarmacología, Ciudad de México, México.

E-mail: itzelbaez@ciencias.unam.mx

Asthma is a chronic inflammatory disease of the airways characterized by airway hyperresponsiveness (AHR), reversible airflow obstruction, wheezing, and persistent inflammation. Although the immune cells involved have been extensively studied, there is growing interest in the role of airway smooth muscle (ASM) as an active effector cell. In this context, mitochondria have emerged as key organelles in ASM functional responses, particularly in processes such as migration, proliferation, and reactive oxygen species (ROS) production. This literature review explores the potential involvement of three mitochondrial proteins —FAM3A, SPG7, and MDH1— in asthma pathophysiology. FAM3A has been associated with calcium homeostasis regulation, ATP production, and redox signaling, which may directly influence ASM contractility and plasticity. SPG7 contributes to the balance between mitochondrial fusion and fission, as well as to the regulation of mitochondrial permeability and apoptosis. MDH1 plays a crucial role in the malate-aspartate shuttle, which is strongly involved in ATP synthesis. A database search was conducted in PubMed, Scopus, and ScienceDirect between 2010 and 2025, selecting relevant studies on cellular and animal models, as well as reviews focused on inflammatory airway diseases. Although no direct studies linking these proteins to asthma were found, the analyzed evidence supported the construction of a theoretical model proposing their potential involvement in airway remodeling through mitochondrial mechanisms. This model highlights new avenues for asthma research, emphasizing ASM as a therapeutic target through the lens of energy metabolism and oxidative stress biology.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Transcriptomic meta-analysis IL7R/STAT3 Axis Dysregulation in Pediatric Multiple Sclerosis

Banda-Morales, Gloria Edith [1 2]; Rojas-Gutiérrez, Santiago Emilio [1];
Ruiz-de la Cruz, María Luisa [1]; Montoya-Rosales, Alejandra [3];
Salinas-Carmona, Mario Cesar [1]; Macías-Segura, Noé.

[1]Servicio y departamento de Inmunología, Hospital Universitario "Dr. José Eleuterio González", Universidad Autónoma de Nuevo León, Monterrey, Nuevo León, México. [2]Departamento de Pediatría, Hospital Universitario "Dr. José Eleuterio González", Universidad Autónoma de Nuevo León, Monterrey, Nuevo León, México. [3]Laboratorio de Inmunomoduladores, Centro de Investigación y Desarrollo en Ciencias de la Salud, Universidad Autónoma de Nuevo León, Monterrey, Nuevo León, México.

E-mail: gloria.bandamo@gmail.com

Pediatric-onset multiple sclerosis (POMS) is an early and aggressive form of central nervous system autoimmunity. Despite its clinical importance, the peripheral immune mechanisms underlying POMS remain incompletely characterized. Transcriptomic studies offer an opportunity to uncover consistent immunopathological signatures and regulatory pathways involved in disease onset and progression.

We conducted a transcriptomic meta-analysis using two publicly available microarray datasets of peripheral blood mononuclear cells: GSE203241 and GSE146383. Differential expression analysis was performed separately for each dataset to identify significantly dysregulated genes (adjusted $p < 0.05$). Cell-type deconvolution was carried out to infer immune cell population differences using CIBERSORTx. Functional enrichment analyses were conducted on the differentially expressed genes (DEGs) using STRING database and functional enrichment with gene ontology and KEGG pathways. A consistent dysregulation of IL7R and IFI44L was observed across both datasets, implicating cytokine signaling and type I interferon responses in POMS. Immune cell deconvolution revealed a decrease in follicular helper T cells and resting NK cells in POMS samples, while regulatory T cells and gamma delta T cells were significantly increased compared to healthy controls. Enrichment analysis of downregulated genes in GSE203241 highlighted suppression of early response transcription factors including JUN, FOSL2, DDIT3, and EGR1. Upregulated genes in GSE146383 were enriched in pathways related to protein biosynthesis and cytosolic activity, with notable expression in CNS and hematopoietic tissues. Key signaling pathways altered in both datasets included IL-7 signaling (IL7R, STAT3, PIK3R1) and cytoplasmic stress response pathways.

Our findings highlight shared molecular and cellular signatures across independent POMS datasets, including altered IL7R/STAT3 signaling, type I interferon activation, and shifts in peripheral immune populations. These results suggest candidate biomarkers for early diagnosis and point to potential therapeutic targets in pediatric multiple sclerosis. Further validation in prospective cohorts is warranted.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Stage-specific immune remodeling in progressive cutaneous lupus

Barraza-Ramos, Brenda Alejandra [1 2]; Rojas-Gutiérrez, Santiago Emilio [1]; Martínez-Canales, Rodolfo [1]; Pérez-Barbosa, Lorena [3]; Skinner-Taylor, Cassandra Michele [3]; Ruiz-de la Cruz, María Luisa [1]; Montoya-Rosales, Alejandra [3]; Salinas-Carmona, Mario Cesar [1]; Macías-Segura, Noé [1].

[1]Servicio y Departamento de Inmunología, Hospital Universitario “Dr. José Eleuterio González”, Universidad Autónoma de Nuevo León. Monterrey, Nuevo León, México. [2]Departamento de Medicina Interna, Hospital Universitario “Dr. José Eleuterio González”, Universidad Autónoma de Nuevo León. Monterrey, Nuevo León, México. [3]Departamento de Reumatología, Hospital Universitario “Dr. José Eleuterio González”, Universidad Autónoma de Nuevo León. Monterrey, Nuevo León, México. [4]Laboratorio de Inmunomoduladores, Centro de Investigación y Desarrollo en Ciencias de la Salud, Universidad Autónoma de Nuevo León, Monterrey, Nuevo León, México.

E-mail: brenda_a.barraza@hotmail.com

Cutaneous lupus erythematosus (CLE) presents as a continuum from acute (ACLE) through subacute (SCLE) to chronic (CCLE) forms, yet the underlying immune dynamics remain poorly understood. To investigate this progression, we analyzed transcriptomic data from skin biopsies in dataset GSE109248 (healthy controls [HC], n=16; psoriasis [PSO], n=17; ACLE, n=6; SCLE, n=9; CCLE, n=13). We applied pathway enrichment (GSVA), exhaustion marker profiling, and immune cell deconvolution using CIBERSORTx to characterize stage-specific immunological remodeling. GSVA revealed a disregulation of type I interferon signaling, JAK–STAT activation, and immune checkpoint pathways (PD-1/CTLA-4) from HC to CCLE. A composite immune checkpoint gene signature (PDCD1, CD274, CTLA4, LAG3) showed low expression in healthy skin, moderate elevation in psoriasis, and marked induction across the lupus spectrum, peaking in CCLE. Expression of housekeeping genes (ACTB, GAPDH, RPL13A) remained stable, supporting biological validity over noise. CIBERSORTx analysis showed distinct shifts in immune cell composition through CLE stages: Early lesions were enriched in M1 macrophages, with lower relative levels of resting dendritic cells, mast cells, and neutrophils. M1 macrophages remained elevated in subacute, while γδ T cells began to increase, chronic lesions sustained high levels of plasma cells, γδ T cells, and M1 macrophages, along with reduced resting memory CD4+ T cells consistent with a persistently activated, pro-inflammatory microenvironment. All observed changes reached statistical significance after FDR correction.

Our findings delineate a trajectory of escalating immune activation and checkpoint engagement across CLE stages, providing insight into disease persistence and potential immunotherapeutic targets.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Area del artículo: **Inmunometabolismo**

Impact of Obesity on IgG and IgA Production in Colostrum of Female Mice

Barrera-Osorio, Mariel [1]; Baltierra-Uribe, Shantal Lizbeth [2];
Santos-Argumedo, Leopoldo [1]; Romero-Ramírez, Héctor [1].

[1]Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional (CINVESTAV), Departamento de Biomedicina Molecular, Ciudad de México, Ciudad de México, México. [2]Escuela Nacional de Ciencias Biológicas (ENCB), Departamento de Microbiología General, Ciudad de México, Ciudad de México, México.

E-mail: marielbarrera61@gmail.com

Introduction:

Colostrum is the first maternal milk secreted during the initial days after birth and is crucial for the development of the newborn's immune system due to its high antibody content, which provides passive immunity. Studies have shown that obesity can alter the immunological and nutritional characteristics of breast milk. Maternal obesity has been associated with a significant increase in IgG and a significant reduction in IgA levels in colostrum, which could impact the infant microbiota with potential long-term effects.

Objective:

To evaluate the impact of maternal obesity on the production of IgA and IgG antibodies in the colostrum of female mice.

Methods:

Two groups were established: a control group and an obesity group, each consisting of five six-week-old female BALB/c mice. The control group was fed a standard diet, while the obesity group received a diet containing 45% kcal from fat for 12 weeks. After this period, the females were mated. Colostrum was manually extracted 3 to 5 days after birth using an anesthesia system. Antibody quantification was conducted using a sandwich-type ELISA.

Results:

The obesity group showed a 24% increase in body weight. We detected IgA, IgG1, IgG2a, and IgG2b subtypes. A trend toward increased IgG1 and IgG2a levels was observed in the colostrum of obese mice compared to the control group.

Conclusions:

The results suggest that obesity may alter the immunological characteristics of colostrum, including antibody concentrations.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Transient Effects of Cry1Ac Toxin and Protoxin on Intestinal Barrier Integrity via MAPK Signaling in a Caco-2 Cell Monolayer

Bautista -Jacobo, Itzel Sarai [1]; Ilhuicatzi Alvarado, Damaris [1];
Hernández Almaraz, Karina Belén[2]; Schnoor, Michael [2];
Moreno-Fierros, Leticia [1].

[1]Universidad Nacional Autónoma de México, Facultad de Estudios Superiores Iztacala, Mucosal Immunity Laboratory, Biomedicine Research Unit, Tlalnepantla, Estado De México, México. [2] CINVESTAV-IPN, Department of Molecular Biomedicine, México City, México.

E-mail: biol.itzelbautista@gmail.com

Cry proteins from Bacillus thuringiensis, particularly Cry1Ac, are widely used as bioinsecticides due to their specificity against insect pests and safety in vertebrates. Beyond their insecticidal role, our research group has demonstrated immunomodulatory properties of Cry1Ac, highlighting its potential as a mucosal adjuvant capable of enhancing anti-tumor and anti-infective immunity. However, emerging evidence suggests Cry1Ac may also exert moderate allergenic effects when administered intragastrically, raising questions about its impact on intestinal physiology. In this study, we explored the effects of Cry1Ac protoxin and toxin (5–80 mg/ml) on intestinal epithelial integrity using a Caco-2 C2BBe1 monolayer model, which mimics polarized gut epithelium with tight junction expression. We assessed cell viability, activation of mitogen-activated protein kinase (MAPK) pathways (ERK, JNK, p38), barrier permeability, cytokine production, and tight junction protein expression.Our results show that neither Cry1Ac protoxin nor toxin affected mitochondrial activity at any tested dose. However, at concentrations ≥40 mg/ml, both forms differentially activated MAPK signaling and induced interleukin-8 production. Notably, Cry1Ac toxin had a more pronounced effect on barrier disruption, evidenced by altered permeability and decreased expression of the tight junction protein ZO-1. Additionally, we identified specific Cry1Ac-binding proteins within the intestinal epithelium, suggesting molecular mechanisms underpinning its epithelial interactions. These findings suggest that Cry1Ac toxin may have a more noticeable effect on epithelial barrier function compared to the protoxin, particularly at higher concentrations. While the overall impact is moderate, these differences highlight the importance of further evaluating the physiological interactions of Cry proteins with intestinal tissue. This work contributes to a nuanced understanding of Cry1Ac's immunological and epithelial effects, supporting future biosafety assessments and potential therapeutic applications.

ISSN: 2007-3380
Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Interaction between MSCs with tumor cells in cervical cancer favours macrophage polarization

Bautista-Sebastián, Eduardo [1 2]; Cortés-Morales, Víctor Adrián [3];
Fajardo-Orduña, Guadalupe Rosario [1]; Monroy-García, Alberto [4];
Castro-Manrreza, Marta Elena [5]; Saucedo-Campos, Alberto Daniel [6];
Gutiérrez-de la Barrera, Marcos [7]; Mayani-Viveros, Héctor [8];
Montesinos-Montesinos, Juan José [1].

[1]Laboratorio de Células Troncales Mesenquimales, Unidad de Investigación en Enfermedades Oncológicas, Hospital de Oncología, Centro Médico Nacional (IMSS), Ciudad de México, México. [2]Posgrado en Ciencias Biológicas, Universidad Nacional Autónoma de México, Coyoacán, Ciudad de México, México. [3]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México. [4]Laboratorio de Inmunología y Cáncer, Unidad de Investigación en Enfermedades Oncológicas, Hospital de Oncología, Centro Médico Nacional (IMSS), Ciudad de México, México. [5]Laboratorio de Inmunología y Células Madre, Unidad Multidisciplinaria de Investigación Experimental Zaragoza, FES Zaragoza, Universidad Nacional Autónoma de México, Ciudad de México, México. [6]Laboratorio 7 de Inmunología, Unidad de Morfofisiología y Función, FES Iztacala, Universidad Nacional Autónoma de México, Estado de México, México. [7]Facultad de Medicina, Universidad Nacional Autónoma de México, Ciudad de México, México. [8]Laboratorio de Células Troncales Hematopoyéticas, Unidad de Investigación en Enfermedades Oncológicas, Hospital de Oncología, Centro Médico Nacional (IMSS), Ciudad de México, México.

E-mail: e.bautista.sebastián@gmail.com

Macrophages exhibiting an immunosuppressive M2 phenotype have been associated with tumor development, and cervical cancer cells (CeCa-TC) are known to have the ability to polarize macrophages toward this phenotype. In previous studies, we have shown that mesenchymal stromal cells (MSCs) derived from CeCa (CeCa-MSCs), enhance the immunosuppressive activity of CeCa-TC against T lymphocytes. However, the effect of MSCs on CeCa-TC to promote the ability to polarize macrophages toward an M2 phenotype has not been studied. In this study, we analyzed the effect of MSCs from normal cervix (NCx) and CeCa on the ability of CeCa-TCs to polarize macrophages toward an M2 phenotype in a coculture system. Our results show that macrophages obtained from CeCa-TC/NCx-MSCs coculture decrease the expression of the CD163 marker compared to those from CeCa-TC/CeCa-MSCs coculture. Interestingly, we observed that macrophages from CeCa-TC/CeCa-MSCs coculture increased intracellular production of IDO, IL-4, and IL-10, decreased T lymphocyte proliferation, and increased soluble IL-10 in cocultures, compared to those from CeCa-TC/NCx-MSCs. In contrast, we observed that macrophages cocultured with CeCa-TC/NCx-MSCs decreased their ability to generate regulatory T lymphocyte populations, as well as their phagocytic capacity, and their presence increased soluble IL-6 in the cocultures, compared to those from CeCa-TC/CeCa-MSCs. Our results show that CeCa-TC/CeCa-MSCs cocultures, compared to CeCa-TC/NCx-MSCs, have a greater ability to polarize macrophages toward an M2 phenotype with increased immunosuppressive potential. This study suggests that cell communication between MSCs and CeCa-TCs present in cervical cancer could promote tumor growth through the polarization of macrophages with enhanced immunosuppressive capacity.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

Pathophysiological dynamics during neonatal murine neurocandidiasis

Ortiz-López, Pablo Armando [1]; Flores-Maldonado, Orlando E [1];
Villarreal-Rivota, Berenice [2]; Castelán-Jaime, Octavio [1];
Chacón-Salinas, Rommel 2; Andrade-Torres, Ángel [1];
Treviño-Rangel, Rogelio [1]; Garza-Rodríguez, María de Lourdes [3];
Molina-Morales, Ariel [1]; Becerril-García, Miguel Ángel [1].

[1]Department of Microbiology, School of Medicine, Universidad Autónoma de Nuevo León, Nuevo León, México. [2]Department of Immunology, National School of Biological Sciences, Instituto Politécnico Nacional, México City, México. [3]Oncology Service, Dr. José Eleuterio González University Hospital, Universidad Autónoma de Nuevo León, Nuevo León, México.
E-mail: miguel.becerrilgr@uanl.edu.mx

Systemic infection by Candida albicans is a leading cause of neurological injury and death in preterm neonates. To investigate the pathophysiological mechanisms underlying this condition, we established an intravenous infection model in neonatal BALB/c mice less than 24 hours old. This model allowed us to study the progression of fungal dissemination into the central nervous system (CNS) and the associated innate immune responses. Our results demonstrated that C. albicans reaches the neonatal brain within the first hour post-inoculation, initiating invasion at the meninges and subsequently spreading to deeper structures including the cortex, diencephalon, and brainstem. This process was accompanied by microglial activation and extensive cellular necrosis in susceptible regions. We further examined the role of dural mast cells as early immune effectors. Histological analyses revealed activation and recruitment of mast cells in infected neonates. Using sodium cromoglycate to pharmacologically stabilize mast cells, we observed a significant increase in cerebral burden and a reduction in survival, supporting the protective role of mast cell degranulation during early infection. In parallel, we analyzed the in vivo expression of virulence-related genes of C. albicans recovered directly from infected brain tissue. High expression levels of adhesins (ALS1-3), secreted aspartyl proteases (SAP4-7), and hyphal wall protein (HWP1) were detected under lethal conditions, suggesting fungal adaptation to the neonatal brain microenvironment.

In conclusion, our data indicate that the outcome of neonatal neurocandidiasis depends on both fungal virulence and the host innate immune response. Mast cells and microglia play pivotal roles in modulating early brain immunity. This neonatal murine model provides a physiologically relevant platform to study fungal–host interactions during early development and may inform future therapeutic strategies for managing fungal CNS infections in neonates.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

MSC protect leukaemia cells from elimination by PTL and DMAPT favouring inflammation.

Beltrán-Calderón, Yesica [1 2]; Ortiz-Reyes, Ana Elenka [2]; Chávez-González, María Antonieta [2].

[1]UNAM, Programa de maestría y doctorado en ciencias Bioquímicas, Ciudad de México, México. [2]IMSS, Unidad de Investigación Médica en Enfermedades Oncologicas del Centro Médico Nacional Siglo XXI, Cuidad de México, México.

E-mail: yesicabc@outlook.com

Acute lymphoid leukaemia is a haematological disease which mainly affects children and is characterized by abnormal and hyperproliferation of lymphoid progenitor and precursor cells. In Mexico, treatment protocols have between 60-70% remission rates and less than 50% survival for relapsed patients therefore, relapse is the main cause of death from this disease due to stem-like leukaemia cells that remain after treatment. These remaining cells survive mainly because of the bidirectional communication with bone marrow (BM) stromal cells like mesenchymal stem cells (MSC), which are characterized by their important immunomodulatory role in the BM microenvironment. To evaluate the role of MSC in chemoprotection of leukaemia cells from cytotoxic compounds, BM samples from paediatric ALL patients without treatment from Hospital de Pediatría del Centro Médico Nacional Siglo XXI (with informed consent), where processed to obtain MSC. These cells where co-incubated with the ALL-B cell lines: REH and RS4:11 with parthenolide (PTL) and diethylamino parthenolide (DMAPT). Both compounds have been reported to induce apoptosis in leukaemia cells through NF-kB signalling inhibition. Apoptosis and cytokine profile from conditioned media were measured by flow cytometry. The obtained results showed that PTL and DMAPT induce apoptosis in cell line monocultures however, when co-cultured with MSC from ALL patients, the apoptosis rate of leukaemia cells with either of the compounds was the same as co-cultures without treatment. A cytokine analysis of the conditioned culture media of both monocultures and co-cultures showed an increase in the production of pro-inflammatory cytokines and chemokines when MSC where present. The cytokine profile was characterized by the presence of IL-6, IL-8, INF-γ, IL-1β, CCL2, CXCL10, and CXCL1. These results indicate that interaction between the MSC and ALL-B cells activate inflammatory signals in MSC that protect ALL-B cells from the cytotoxic activity of both PTL and DMAPT.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Identification of immunogenic P. gingivalis proteins using a murine immunochemical approach

Beltrán-López, Evangelina [1]; Tiznado-Parra, Nanziuan Napaketzalli [1]; Luque-Ortega, Fred [1]; Arámbula-Meraz, Eliakym; Vega-Castillo, Ulises [1]; León-Sicarios, Claudia del Rosario [1]; López-Moreno, Héctor Samuel [1].

[1]Laboratorio de Biomedicina Molecular, CAC Biotecnología Biomédica UAS-264, Posgrado en Ciencias Biomédicas y Posgrado en Biotecnología, énfasis en Salud, Facultad de Ciencias Químico Biológicas, Universidad Autónoma de Sinaloa, Av. Las Américas S/N, Ciudad Universitaria, Culiacán, Sinaloa, México. E-mail: evabeltran@uas.edu.mx

Porphyromonas gingivalis is a key pathogen associated with chronic periodontitis. Its immunogenic proteins play critical roles in host-pathogen interactions and represent potential targets for diagnostic or therapeutic strategies. This study aimed to identify immunogenic proteins of P. gingivalis through immunochemical analysis using sera from BALB/c mice immunized with a bacterial protein extract. A total protein extract (ECPg) was prepared and used as the antigen in an intraperitoneal immunization protocol. Immune sera were collected, validated by ELISA, and analyzed by 12% polyacrylamide gel electrophoresis and Western blot to assess the protein profile and antigen-antibody recognition. Twelve protein bands were detected in the crude extract, of which four (43, 54, 62, and 72 kDa) were recognized by murine antibodies. In silico and literature analyses identified three of these as outer membrane protein 33 (PG33, p43), fimbriae Mfa1 (p54), and phosphotransacetylase (p72). The 62 kDa protein (p62) exhibited the highest immunoreactivity; however, its function remains unknown, highlighting the need for further immunoproteomic characterization. These findings advance the understanding of P. gingivalis antigenic determinants and support the use of murine models in immunogenicity research related to periodontal disease.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Bovine Viral Diarrhea Virus interaction prediction with caspase 1 support to viral replication

Benítez-Guzmán, Alejandro [1]; Ruelas -Mesa, Isabela [1];
Cantera-Bravo, Miroslava [1]; Gallegos-Rodarte, Claudia [1];
Gutiérrez-Guerrero, Yocelin [3]; Nájera-Rivera, Héctor [2].

[1]UNAM, Facultad de Medicina Veterinaria y Zootecnia, Departamento de Microbiología e Inmunología, Laboratorio de Inmunofisiología y proteómica, CDMX. [2]UNAM, Facultad de Medicina Veterinaria y Zootecnia, Departamento de Reproducción, CDMX. [3]Museum of Vertebrate Zoology, UC Berkeley Research, California, US.

E-mail: alebenitezg@fmvz.unam.mx

Flaviviridae viral family has worldwide impact due to economic, public and animal health affecting thousands of people and animals around the globe. This family is composed of 4 genera that have a ribonucleic acid (RNA) genome which encodes 10 to 12 proteins organized as a polyprotein and divided into structural and non-structural proteins with similar functions along the family. Bovine Viral Diarrhea Virus (BVDV) is part of the family and its presence around the world affects cattle due to loss in production and clinical manifestations. The inflammasome NLRP3 has been known to defend the body against infections by stimulating the excretion of IL-1β through caspase 1. It has been observed that, with the absence of caspase 1, the viral titer for BVDV decreases. We speculate that caspase 1 is able to interact with BVDV to cleave the viral proteins in charge of replication in key points to make them "activate" and start the viral replication process. In this study through in silico analysis we observe possible interactions of caspase 1 with viral proteins in which 3 possible cleavage sites were revealed. Proteins Erns, NS2-3 and NS5A have cleavage sites that were compared to other viral proteins from the viral family, similar cleavage sites were identified. Finally, a phylogenetic and structural analysis of the different proteins in the family showed similarities between the possible cleavage sites. This suggests similar behavior in the viral family that could help us identify critical steps in viral replication to prevent the spread of these diseases and, in the future, develop control mechanisms for diseases caused by this viral family.

Financial support: DGAPA-PAPIIT IN214724.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Intranasally administered SMPs change the immune landscape in a TNBC mouse model

Borges-García, Inti Atzayacatl; Rodríguez-Sanoja, Romina.

Instituto de Investigaciones Biomédicas, UNAM, Ciudad de México, México.

E-mail: inti.borges@iibiomedicas.unam.mx

Breast cancer is a neoplasia which arises from breast tissue. One of its subtypes, triple negative breast cancer (TNBC), is characterized by rapid growth of tumours and a high metastatic rate alongside a lack in expression of oestrogen, progesterone and human epidermal growth factor receptors. These characteristics make it difficult to develop an efficient treatment for the disease, especially vaccine design due to high antigenic variation and low infiltration of lymphocytes.

Variable epitope libraries (VEL) are new arising therapies in cancer research in which tumour antigens are mutated at strategic sites to mimic the tumours antigen variability and stimulate T cells against a broad repertoire of variations of the same antigen. In this study we used a VEL of the protein survivin as synthetic peptide in a TNBC model in Balb/c female mice; this strategy has showed promising results in reducing tumour growth and lung metastasis when administered in the spleen. Due to the low immunogenicity of peptides and the low translational potential of intrasplenic injections we use starch microparticles (SMPs) as adjuvants for the intranasal administration of the therapy. In previous studies it has been shown that these SMPs when administered via the nasal route with an antigen enhance both the local and systemic antigen specific immune response.

Results showed that when mice received SMPs alone and with the vaccine, tumours tend to grow less than the control group and to have less metastatic lesions in the lungs. To study the possible role of the immune system in these observations we performed flow cytometry to characterize cell populations in the spleen, tumour and lungs of the mice. The data obtained showed a differential immune landscape in the three tissues between the three groups of study along three independent experiments, denoting an increase in effector T-CD8 lymphocytes in the spleen.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Identify the SARS-CoV-2 viral load in rt-PCR could prevent death in COVID-19

Borrego-Moreno, Juan Carlos [1 4]; Canizalez-Roman, Vicente [2];
Leon-Sicairos, Nidia [2]; Angulo -Zamudio, Uriel Alberto [2];
Cardenas -De Luna, María Julieta [3]; Castañeda -Delgado, Julio Enrique [4].

[1]Hospital General de Zona IMSS Zacatecas. [2]Laboratorio de Biología Molecular de la Facultad de Medicina de la Universidad Autónoma de Sinaloa. [3]Unidad de Medicina Familiar # 57 De Zacatecas IMSS. [4]Laboratorio De Investigación Biomédica De Zacatecas IMSS.

E-mail: bogue6@hotmail.com

The epidemiological landscape of the SARS-CoV-2 pandemic has diminished, however, viral circulation in patients with risk factors continues to cause hospitalization and death. Currently, we do not have an indicator that allows us to classify which cases diagnosed with COVID-19 should be kept under close surveillance in order to avoid preventable deaths under critical medical care. The SARS-CoV-2 viral load identified through a real-time PCR study may represent this still necessary tool in the control of COVID-19 infection. In this cohort of 404 hospitalized patients, we evaluated the level of SARS-CoV-2 viral load and its follow-up until death or survival. We identified through regression analysis that a high viral load (RR 3.14 CI 1.466-6.761 p 0.003) was significantly associated with a higher risk of death. Observed survival at 10 days was only at 40% of the cases with high viral load (LogRank p 0.000). In Cox regression, hospitalized COVID-19 cases with high viral loads had a 6-times higher hazard ratio of dying compared to those with low viral loads (Cox RR 6.2 CI 4.23–9.12). A strong association was identified between high viral load and the risk of death in hospitalized COVID-19 patients, so we consider that viral load should be reported in the RT-PCR study to support medical judgment for clinical surveillance and timely hospitalization.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos B**

CD39 and CD73 are key enzymes in the adenosine (ADO) pathway

Bravo -Ramírez, Bruno [1 2]; Reyes-Huerta, Raúl Fernando [2]; Mandujano-López, Jorge Vladimir [2 3]; Ayala-Rodríguez, Andrea Monserrat [2 3]; Velásquez-Ortiz, María Guadalupe [2]; Torres-Ruiz, José Jiram [4]; Gómez-Martín, Diana [4]; Meza-Sánchez, David [5]; Ortiz-Navarrete, Vianney [1]; Maravillas-Montero, José Luis [2].

[1]Centro de Investigación y Estudios Avanzados del Intituto Politécnico Nacional, posgrado en biomedicina molecular, Ciudad de México, México. [2]Instituto de Biotecnología, Universidad Nacional Autónoma de México, Cuernavaca, Morelos, México. [3]Escuela Nacional de Ciencia Biológicas del Instituto Politécnico Nacional, Posgrado en Inmunología, Ciudad de México, México. [4]Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Departamento de Inmunología y Reumatología, Ciudad de México, Ciudad de México, México.. [5]Red de Apoyo a la Investigación, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán y Universidad Nacional Autónoma de México, Tlalpan, Ciudad de México , Mexico.

E-mail: brunobravo27@gmail.com

Double negative B cells (CD27- IgD-) are a small and poorly understand subset of cells first described in Systemic Lupus Erythematosus (SLE) due to its elevated frequency in patients. Recently multiple DN subsets have been described, one of them being DN1 (CD11c-CD21+). However, the origin and functional properties of DN1 B cells remain largely unknown. The frequency of DN1 subset in active SLE patients decreases in comparison with healthy and inactive (in remission) patients having a significant negative correlation with main parameters of the SLEDAI (SLE Activity Index) meaning that the low level of DN1 cells in SLE patients worsens the symptoms and parameters of the disease. So, these DN1 B cells may have a regulatory role in controlling inflammation by the yield of regulatory cytokines, the induction of other regulatory cells or by inhibiting inflammatory cells (by membrane suppressing proteins like PDL1 and CTL4 or adenosine production) thus explaining the active SLE symptoms and low DN1 B cell levels. B cells from healthy, active and inactive SLE patients were stimulated for future IL-10 production identification by flow-cytometry, finding higher levels of DN1 IL-10+ B cells in healthy and inactive SLE patients in comparison with the low levels of inactive SLE patients. B DN1 cells also underwent through a different flow cytometry panel were a high level of CD73 and CD39, both enzymes that yield extracellular adenosine suppressing T effector cells, was shown in healthy and inactive patients, compared with low levels in active SLE patients. These results show two different regulatory mechanisms by how DN1 B cells can control the inflammatory environment in autoimmune diseases such as SLE.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Standardization of an ELISA to identify anti-Actinomadura madurae IgM and IgG antibodies

Briones-Bautista, Benito [1]; Vázquez-Marmolejo, Anna Velia [1];
García-Vargas, Juan Ángel III [1]; Mejía-Torres, Manuel Guadalupe [1];
Molina-Torres, Carmen Amelia [2]; Bonifaz-Trujillo, José Alexandro [3];
Salinas-Carmona, Mario César [1].

[1]Servicio de Inmunología, Facultad de Medicina y Hospital Universitario "Dr. José Eleuterio González", Universidad Autónoma de Nuevo León. [2]Laboratorio Interdisciplinario y de Investigación Dermatológica, Servicio de Dermatología, Hospital Universitario "Dr. José Eleuterio González", Universidad Autónoma de Nuevo León. [3]Servicio de Dermatología, Hospital General de México "Dr. Eduardo Liceaga".

E-mail: beny.briones98@gmail.com

Actinomadura madurae is the second most common cause of Actinomycetoma in Mexico, yet no serological tests are available for a quick differential diagnosis. We propose standardizing an ELISA to identify specific antibodies during infection or immunization. An homogeneous bacterial suspension (50 mg/50 µL) was prepared for a plantar pad infection in a group of five BALB/c mice. Additionally, a crude cell extract (CCE) was obtained and adjusted to 50 µg/100 µL with incomplete Freund's adjuvant to immunize a second group of five mice on two occasions: day 0 and 30. Blood was collected from infected mice in days 5 and 45 and from the immunized group on day 15 after the last immunization and then centrifuged to obtain the serum. Actinomadura madurae CCE (5 µg/200 µL) as an acetate-buffered antigen was placed on an ELISA plate. Different dilutions of sera were used as the primary antibody. Rabbit-prepared mouse anti-IgM or IgG, labeled with HRP and revealed with OPD, was used as the second antibody. Absorbance readings were performed at 492 nm/630 nm. To rule out cross-reactivity of sera from mice infected or immunized with A. madurae, Nocardia brasiliensis CCE was used. To determine anti-A.madurae IgM antibodies in infected mice, optimal dilution was 1:20 for the first antibody and 1:1500 for the second antibody. For IgG, optimal dilution was 1:50 for the 1st antibody and 1:1000 for the 2nd antibody, with no cross-reactivity against CCE of Nocardia brasiliensis. For anti-A.madurae IgG antibodies in immunized mice, optimal dilution of the first and second antibodies was 1:1000, and no cross-reactivity was observed. An ELISA test was standardized to identify specific IgM and IgG anti-Actinomadura madurae antibodies in sera from infected mice, and specific IgG in sera from immunized mice; this test can be used for the differential diagnosis of Actinomadura madurae mycetoma patients.

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Design of Antimicrobial Peptides Using Artificial Intelligence and Their Characterization by Surface Plasmon Resonance

Cabral-Venegas, Valeria Areli [1 2]; Madrigal-Melchor, Jesús [2];
Zambrano -de la Torre, Misael [3]; García-Menchaca, Levi Esteban [3];
Rodríguez-Reyes, Daniel Alberto [2]; Santiago-Rivas, Bruno [1].

[1]Instituto Mexicano del Seguro Social, Zacatecas, Zacatecas, México. [2]Unidad Académica de Ciencia y Tecnología de la Luz y la Materia, Universidad Autónoma de Zacatecas, Zacatecas, Zacatecas, México. [3]Unidad Académica de Ingeniería Eléctrica, Universidad Autónoma de Zacatecas, Zacatecas, Zacatecas, México.

E-mail: vale.cabral.14@gmail.com

Introduction and Objective: Tuberculosis remains a major global public health concern, a situation further exacerbated by the rise of multidrug-resistant strains that undermine the efficacy of conventional treatments. In this context, the present project focuses on the rational design of antimicrobial peptides (AMPs) specifically targeting Mycobacterium tuberculosis, leveraging Artificial Intelligence (AI) tools. The main objective is to develop novel therapeutic alternatives through the identification and optimization of peptide sequences with antimicrobial activity using machine learning models. Methods: The methodology was structured in three stages. In Stage I, public AMP databases were collected, cleaned, and curated. Subsequently, machine learning algorithms, specifically Support Vector Machines (SVM) and Random Forest (RF), were employed to train a predictive model capable of classifying, designing, and characterizing peptides with antimicrobial potential. In Stage II, the selected peptides will be synthesized and evaluated through in vitro assays against M. tuberculosis. Finally, in Stage III, peptides demonstrating significant antimicrobial activity will be characterized using Surface Plasmon Resonance (SPR), an optical technique that enables the study of molecular interactions. Results: To date, data curation has been completed, and an initial classification model has been developed with promising performance, achieving an area under the ROC curve (AUC-ROC) of 97%, indicating high predictive power for identifying candidate peptides. Conclusion: These preliminary results support the potential of AI-driven approaches in the rational design of antimicrobial peptides targeting M. tuberculosis. The upcoming in vitro validation phase will allow for the assessment of their biological efficacy, representing a significant step toward the development of new therapies against resistant strains.

TRABAJO NO PRESENTADO

Área del artículo: **Inmunotecnología e inmunoterapia**

Generation and validation by ELISA of blocking the anti-ubiquitin scFv C1-Fc tandem

Calitl-Flores, Zeenen Margarita [1 2]; Mendoza-Salazar, Ivette [1 2]; Damián-Morales, Gabriela [1 2]; Álvarez-Fosado, Tomas [1 2]; Pérez-Vega, Daniel Jesús [1 2]; Gómez-Castellano, Keyla [1 2]; Almagro, Juan Carlos [1 2 3]; Pérez-Tapia, Sonia Mayra [1 2 4].

[1]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, México City, México. [2]Laboratorio Nacional Para Servicios Especializados de Investigación, Desarrollo e Innovación (I+D+i) Para Farmoquímicos y Biotecnológicos, LANSEIDI-FarBiotec-CONACyT, México City, México. [3]Global Bio, Cambridge Massachusetts, United States. [4]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, México City, México.

E-mail: margaritacalitlf@gmail.com

Ubiquitin (Ub) is a small protein of 76 amino acids that plays essential roles in multiple cellular processes in eukaryotic organisms. It has been identified as the main peptide component of the dialyzable leukocyte extract Transferon Oral®, and is also the most abundant protein in this product, which has sparked interest in its study as a potential biopharmaceutical. Previous studies have shown that Ub participates in the modulation of inflammatory responses and has therapeutic potential in diseases such as sepsis, multiple sclerosis, microbial infections, and tissue regeneration, including cardiac and brain tissue. Therefore, Ub has been proposed as a candidate for the development of therapies with anti-inflammatory, neuroprotective, and cardioprotective properties.

To quantify Ub in biological samples, we developed a blocking ELISA that requires high-affinity antibodies for optimal sensitivity. Recombinant Ub was produced at UDIBI and used to identify specific binders from ALTHEA Platinum VH:VL 4x3 Phage Library. From the screening, several scFv were obtained, among which scFv-C1 showed superior affinity compared to others. To improve its affinity, a tandem antibody was designed with three scFvs-C1 units in each Fab arm. Each scFv contains a VH and VL region joined by a 25-amino acid linker, and the three scFvs are connected by a 15-amino acid glycine-serine linker, fused to a human Fc region via a hinge. Assembly was performed using fragment fusion strategies and cloned for expression in Expi293F cells by lipotransfection. Purification was performed by protein A affinity chromatography, and functional evaluation is ongoing to confirm its potential for sensitive Ub quantification in biological samples.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Interaction between vitamin D SNVs with rheumatoid arthritis and hypovitaminosis D risk

Campos López, Bertha [1 2]; Rivera-Escoto, Melisa [1];
Ruiz-Ballesteros, Adolfo Isaac [1]; Pesqueda Cendejas, Karen [1 2];
Meza Meza, Mónica del Rosario [1 2]; Parra Rojas, Isela [1 3];
Mora -García, Paulina Esmeralda [1 2]; De la Cruz Mosso, Ulises [1 2].

[1]Red de Inmunonutrición y Genómica Nutricional en las Enfermedades Autoinmunes; Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud; Universidad de Guadalajara; Guadalajara, Jalisco 44340, México. [2]Instituto de Neurociencias Traslacionales, Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara 44340, Jalisco, México. [3]Laboratorio de Investigación en Obesidad y Diabetes; Facultad de Ciencias Químico-Biológicas; Universidad Autónoma de Guerrero; Chilpancingo de los Bravo 39087, Guerrero, México.

E-mail: berth.clampez@gmail.com

Rheumatoid arthritis (RA) is an autoimmune disease of multifactorial etiology. One environmental-nutritional factor associated with RA pathophysiology is serum vitamin D deficiency (hypovitaminosis D). Different genetic association studies have reported that around 65% of hypovitaminosis D can be partially explained by the presence of single nucleotide variants (SNV) in key genes of its metabolism. This study aimed to determine the association of genetic variants in (rs10741657) CYP2R1, (rs10877012) CYP27B1, (rs4809959) CYP24A1 and (rs731236 TaqI) VDR for the risk to RA and the hypovitaminosis D in the Mexican-Mestizo population. This study was conducted in 177 RA patients and 204 control subjects (CS), and allelic discrimination was performed with TaqMan probes. Vitamin D serum levels (calcidiol and calcitriol) were analyzed through ELISA commercial kits. SNVs were evaluated by multivariate dimensionality reduction (MDR) analysis. The rs731236 TaqI in VDR was presented in each of the models and this variant could have the most influence to RA. CT and CC TaqI genotypes were particularly associated with 1.8-fold more susceptibility to AR (OR=1.8; CI=1.2-2.7; p<0.01), as well as with 2.7-fold more susceptibility to activity of disease, according to DAS28-VSG (OR=2.7; CI=1.1-6.3; p=0.02). RA patients had higher calcitriol (47.83 vs. 36.85 pg/mL; p<0.001), and calcitriol/calcidiol ratio (2.07 vs. 1.48 pg/ng; p<0.001) compared to CS. Particularly, GG and TT genotypes on the rs10877012 CYP27B1 were associated with 1.7-fold more susceptibility to lower serum levels of calcidiol (OR=1.7; CI=1.2-2.7; p<0.01) in both study groups. In conclusion: the CT and CC genotypes on rs731236 TaqI VDR confer genetic susceptibility to RA and activity of disease and the GG and TT genotypes CYP27B1 was associated with hypovitaminosis D in the Mexican mestizo population.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Bovine Viral Diarrhea Virus Activates the NLRP3 Inflammasome Through Calcium Mobilization.

Cantera-Bravo, Meztli Miroslava [1]; Benítez-Guzmán, Alejandro [1];
Arellano-Reynoso, Beatriz [1]; Díaz-Muñoz, Mauricio [2].

[1]Universidad Nacional Autónoma de México, Facultad de Medicina Veterinaria y Zootecnia, Departamento de Microbiología e Inmunología, Ciudad de México, México. [2]Universidad Nacional Autónoma de México, Instituto de Neurobiología (INB) , Campus Juriquilla, Querétaro, México.

E-mail: 16mc05meztlicantera@gmail.com

Bovine viral diarrhea virus (BVDV) is a positive-sense RNA virus that belongs to the Flaviviridae family, genus Pestivirus. It infects both wild and domestic ruminants, with significant economic impact on the cattle industry due to its association with abortions, decreased milk production and immunosuppression.

It is currently known that BVDV induces activation of the NLRP3 inflammasome, a cytosolic multiprotein complex responsible for the proteolytic cleavage of pro-IL-1β into its active form. However, the mechanism by which BVDV triggers NLRP3 inflammasome assembly remains unclear. The aim of this study was to determine whether calcium mobilization—a process known to facilitate the replication of other Flaviviridae members—contributes to NLRP3 inflammasome activation and whether this is associated with viral replication.

Our results demostrate that during replication of NADL and New York strains of BVDV in MDBK cell line, calcium mobilization occurs, with significant differences observed over time post-infection. Additionally, we found that calcium release induced by ryanodine during NADL infection increases viral titers by one logarithmic unit, whereas calcium chelation with BAPTA-AM reduces viral replication by a similar magnitude. Our next step is to quantify IL-1β secretion and assess whether calcium inhibition reduces cytokine release during viral infection.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Effect of prostaglandin inhibition on DENV replication in Anopheles albimanus

Caraveo-Centeno, Luis [1 2]; Quezada -Ruiz, Edgar [1 2]; Lanz-Mendoza, Humberto [2]; Hernández-Hernández, Fidel de la Cruz [1].

[1]Departamento de Infectómica y Patogénesis Molecular, Centro de Investigación y de Estudios Avanzados del IPN, Ciudad de México, CDMX. [2]Centro de Investigaciones sobre Enfermedades Infecciosas, Instituto Nacional de Salud Pública, Secretaría de Salud, Cuernavaca, Morelos, México.

E-mail: alejan_cc3@hotmail.com

The immune system is modulated by various factors such amongst which are the prostaglandins (PG). These lipid molecules participate in various insects physiological processes such as reproduction, fluid secretion and immune response. Their involvement in pathogen-vector interactions has been documented due to their immunomodulatory role against bacteria, viruses, and parasites. It is known that various mosquito factors are required for the establishment and infection of DENV in the midgut. This first step leads to the mosquito systemic infection and to the transmission of the virus to the human population. Worldwide, the main DENV vectors are Aedes aegypti and Ae. albopictus. However, little is know about the factors of resistance to DENV infections in non vector mosquito species (Anopheles). The main objective of this work is to determine how the inhibition of PG by a nonsteroidal anti-inflammatory drug (NSAID) affects the susceptibility of An. albimanus to DENV infection. First, the effect of PG inhibition on mosquito physiology were assessed, measuring biological parameters such as lifespan and fecundity. The infection and dissemination of DENV in various mosquito tissues were evaluated using RT-qPCR. Additionally, the transcriptional expression of Dicer2, R2D2, and AGO2, components of the RNAi pathway (antiviral mechanism), was evaluated. Since the immune response of An. albimanus involves other factors, we measured the production of nitric oxide (NO) and the activity of phenoloxidase, upon PG inhibition by NSAID. The changes induced on the components of the immune response by PG synthesis alteration could alter the mosquito susceptibility to the pathogen infection.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Inmunophenotyping as a tool for classification and monitoring of MASLD in Veracruz population

Caraza-Sánchez, Yvanna [1]; García-Ponce, Fátima del Carmen [2];
González-Herrera, Sandra Luz [3]; Pérez-Lara, Jocelyn Carolina [4];
Palma -Jacinto, José Antonio [5]; Gallardo -Hernández, Carlos Arturo [6].

[1]Universidad Veracruzana, Facultad de Bioanálisis, Xalapa, Veracruz, México. [2]Universidad Veracruzana, Facultad de Bioanálisis, Xalapa, Veracruz, México. [3]Universidad Veracruzana, Facultad de Bioanálisis, Xalapa, Veracruz, México. [4]Centro de Investigaciones y Estudios Avanzados del Instituto Politécnico Nacional en el Departamento de Biomedicina Molecular, CDMX, México. [5]Universidad Veracruzana, Unidad de Servicios Analíticos en Salud Bioanálisis, Facultad de Bioanálisis, Xalapa, Veracruz, México. [6]Universidad Veracruzana, Unidad de Servicios Analíticos en Salud Bioanálisis, Facultad de Bioanálisis, Xalapa, Veracruz, México.

E-mail: YvannaCaraza@outlook.com

Metabolic dysfunction associated to steatosis liver disease (MASLD) is related to metabolic disorders and is one of the causes of morbidity associated to hepatic diseases in some countries, include Mexico. Interestingly, the physiopathology of MASLD is closely related to immunological response. In this sense, the immune cells play an important role in proinflammatory cytokines release and development of chronic inflammation, contributing to the conversion from steatosis stage to fibrosis and hepatocellular carcinoma. In this regard, an observational comparative cross-sectional study was conducted with patients diagnosed with MALSD from the Metabolism and Heart Society clinic in the city of Veracruz, Veracruz, Mexico for determinate the immunophenotype through flow cytometry to identify the proportion and absolute numbers of innate and adaptative immune cells from peripheral blood and correlate with the different stages of MALSD. The results demonstrate that CD4+, CD8+ T cells and total B cells increase during the fibrosis stage. Similarly, the proportion of innate immune cells that include granulocytes, natural killer cells and macrophages elevated and correlated with the severity of disease. Together, this evidence demonstrates that evaluating the immunophenotype in the difereten stages of the disease could be important to establish a specific diagnosis, prognosis and treatment of MALSD.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Neutralization dose-response in sera from mice immunized with the AVX/COVID-12 vectorized vaccine

Carballo-Uicab, Gregorio [1 2 3]; Mellado-Sánchez, Gabriela [1 2];
González-González, Edith [1 2]; Salinas-Trujano, Juana [1 2];
Mendoza-Salazar, Ivette [1 2 3]; Chagoya-Cortés, Héctor Elías [4];
Lozano-Dubernard, Bernardo [5]; Krammer, Florian [6 7];
López-Macías, Constantino [8]; Pérez-Tapia, Sonia Mayra [1 2 3];
Almagro-, Juan Carlos [9].

[1]Instituto Politécnico Nacional, Escuela Nacional de Ciencias Biológicas, Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), México City, México. [2]Laboratorio Nacional para Servicios Especializados de Investigación Desarrollo e Innovación (I+D+i) para Farmoquímicos y Biotecnológicos (LANSEIDI-FarBiotec-CONACyT), México City, México. [3]Instituto Politécnico Nacional, Escuela Nacional de Ciencias Biológicas, Departamento de Inmunología, México City, México. [4]Consultora Mextrategy, S.A.S. de C.V., México City, México. [5]Laboratorio Avi-Mex, S.A. de C.V. (Avimex), México City, México. [6]Icahn School of Medicine at Mount Sinai, Department of Microbiology, New York, NY, United States of América. [7]Icahn School of Medicine at Mount Sinai, Department of Pathology, Molecular and Cell-Based Medicine, New York, NY, United States of América. [8]Instituto Mexicano del Seguro Social (IMSS), Centro Médico Nacional Siglo XXI, Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, México City, México. [9]GlobalBio, Inc., Cambridge, MA, United States of América. [10]

* E-mail: gregorio.carballo@udibi.com.mx

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) remains a global health challenge, causing severe morbidity and mortality, particularly in vulnerable groups such as the elderly, immunocompromised individuals, and those with comorbidities. In low- and middle-income countries, vaccine access is hindered by high costs and inequitable distribution. To tackle these issues, Mexico developed the AVX/COVID-12 (V-Wu) vaccine, a recombinant Newcastle disease virus (NDV)-based platform expressing a stabilized ancestral Wuhan spike protein (HexaPro-S). Locally manufactured after rigorous testing and regulatory approval, V-Wu aims to enhance self-sufficiency and equity in immunization. This study evaluates an updated vaccine version, AVX/COVID-12 (V- BA), designed to combat Omicron subvariants by expressing the HexaPro-S protein of BA.2.75.2. Both vaccines were administered intramuscularly in BALB/c mouse model using a prime-boost regimen. Immunogenicity was analyzed by measuring antibodies against Omicron S proteins BA.2.75.2 and XBB.1.5, as well as neutralizing antibodies against Wuhan, BA.1, XBB.1.16, and JN.1 subvariants. Both vaccines were safe, eliciting robust antibody responses against Omicron S proteins and neutralizing antibodies against multiple emerging SARS- CoV-2 variants of concern (VOCs). V-BA demonstrated superior protection against current Omicron subvariants, while V-Wu offered broader coverage, including the ancestral Wuhan strain and emerging variants like JN.1 in a dose-response effect. These findings underscore the adaptability of NDV-based platforms in addressing the evolving SARS-CoV-2 landscape and reaffirm the ongoing utility of the ancestral Patria vaccine. Together, they demonstrate the potential of these platforms to drive the development of next-generation vaccines tailored to emerging viral threats.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Expression of FOXP3 in B-Cell Subtypes (CD19+CD39+, CD19+CD39-) and Their Regulatory Function

Cárdenas-Juárez, Abraham [1]; Portales-Pérez, Diana Patricia [2 3];
García-Hernández, Mariana Haydee [1].

[1]Unidad de Investigación Biomédica, Delegación Zacatecas, Instituto Mexicano del Seguro Social, IMSS, Zacatecas, México. [2]Facultad de Ciencias Químicas, Universidad Autónoma de San Luís Potosí, UASLP, San Luis Potosí, México. [3]Centro de Investigación en Ciencias de la Salud y Biomedicina, Universidad Autónoma de San Luis Potosí, San Luis Potosí, México.
E-mail: abrahamcardenas516@gmail.com

Aim. To evaluate FOXP3 expression in CD19+CD39+ and CD19+CD39- B cells, and to investigate its potential regulatory role. Methods. Peripheral blood B cells were obtained from 25 adult volunteers. B cell subsets were purified magnetically. FOXP3 expression at the mRNA and protein level was analyzed in CD19+CD39+ and CD19+CD39- B cells by FACS and RT-qPCR. Suppressive activity was assessed through co-cultures of PBMC with CD19+CD39+ and CD19+CD39- B cells stimulated with anti-CD3/anti-CD28, evaluating CD4+ T cell proliferation and the percentage of IFN-γ+ CD4+ T (Th1) cells. IL-10 mRNA expression was also assessed by RT-qPCR. Results. The percentage of CD19+CD39+ expressing FOXP3 B cells was higher compared to other phenotypes. There was a positive correlation between FOXP3 and CD39 in CD19+ B cells. FOXP3 mRNA was increased in CD19+CD39+ B cells compared to CD19+CD39- B cells. CD19+CD39- B cells reduced the proliferation, the percentage of Th1 cells, and expressed higher IL-10 mRNA compared to CD19+CD39+ B cells. B cell phenotypes were inversely associated with inflammatory markers (Th1 cells and CRP) whereas Th1 cells were directly correlated with CRP levels. CD19+CD39- was directly associated with β-cell functionality. Inverse association between HOMA-β and TyG index, HbA1c and FPG levels was observed. CD19+CD39+ expressing FOXP3 B cells were inversely associated with body mass index and waist circumference. Conclusions. FOXP3 is expressed on both CD19+CD39- and CD19+CD39+ B lymphocytes. CD19+CD39- cells showed high levels of IL-10 and low levels of FOXP3 mRNA. CD19+CD39- B cells decreased Th1 cells and were associated with β-cell function.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Non-canonical functions of Anopheles gambiae hemocytes during Plasmodium falciparum Infections.

Cardoso-Jaime, Víctor [1]; Dimopoulos-, George [1].

[1]W. Harry Feinstone Department of Molecular Microbiology and Immunology, Malaria Research Institute, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD, USA.
E-mail: v.cardoso.jaime@gmail.com

Mosquito hemocytes (insect´s macrophage) are well-known for their immune functions in killing pathogens. While most of the studies have been performed using murine models, mosquitoes’ immune responses against human and rodent malaria parasites present significant differences. Here, we show for the first time that hemocytes from the major malaria vector Anopheles gambiae play agonist roles during infections of the human malaria parasite Plasmodium falciparum through non-immune functions. By chemically depleting hemocytes, we found that they are essential in maintaining the mosquito midgut epithelium integrity through controlling intestinal stem cell proliferation producing basal lamina components, and modulating microbiota load. These processes are very important during the early stages of infection, because Plasmodium actively suppresses apoptosis in intestinal cells to avoid triggering damage and consequently the immune response. Upon reaching the basal lamina, the parasite exploits its components to form a capsule surrounding developing oocysts. Depleting hemocytes disrupts these processes, resulting in high mortality rates due to uncontrolled midgut damage caused by the blood meal, systemic infection by the midgut microbiota leaking into the hemolymph, and unfavorable conditions for Plasmodium to establish the infection, which is reflected in a reduction of oocysts per midgut. In conclusion, we provide evidence that Plasmodium falciparum exploits mosquito immune cells not only to support its own protection but also to promote its development. This mechanism may help explain why P. falciparum is the deadliest malaria parasite in the world.

We thanks to the Johns Hopkins Malaria Research Institute Parasite and Insectary Core Facilities and the Johns Hopkins Institute for Basic Biomedical Sciences microscope facility for their support. This work has been supported by NIH R01AI12274, R21AI131574, R01AI170692, R01AI158615, and the Bloomberg Philanthropies.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

New Insights into Antiviral Immune Response of Aedes aegypti Hemocytes (Macrophage-like cells)

Cardoso-Jaime, Víctor; Tikhe, Chinmay Vijay; Tavadia, Mihra;
Dimopoulos, George.

[1]W. Harry Feinstone Department of Molecular Microbiology and Immunology, Malaria Research Institute, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD, USA.

E-mail: v.cardoso.jaime@gmail.com

Although dengue has become one of the most important arboviral diseases worldwide, only a small fraction of mosquitoes become infectious and are capable of transmitting the virus. This is largely due to the robust immune system of these insects. Hemocytes (macrophage-like cells) are the professional immune cells of mosquitoes. They have been extensively studied for their roles in eliminating bacteria, fungi, and Plasmodium parasites; however, their function during viral infections remains poorly understood. Here, we present the first transcriptomic analysis of hemocytes and the fat body, the major immune tissues of Aedes aegypti. We found that both tissues exhibit a high abundance of immune-related genes; however, hemocytes show higher expression levels of genes involved in most antiviral immune pathways, particularly those related to the piRNA pathway. When comparing the transcriptomes of hemocytes from naïve and Dengue virus 2-infected mosquitoes (DENV2), we observed an upregulation of genes related to the piRNA pathway, suggesting that hemocytes may contribute to antiviral defense through this mechanism. Furthermore, after chemically depleting hemocytes, we evaluated both midgut and systemic infection and found that hemocyte-depleted mosquitoes exhibited higher systemic titers of DENV2. Finally, an assay using nanobead mimics virus size, showed that hemocytes and tracheas are the only tissues engulfed nano beads. Overall, our findings indicate that hemocytes help control systemic viral infection, most likely by up taking viral particles and eliminating them via the piRNA pathway following phagocytosis.

We thanks to the Johns Hopkins Malaria Research Institute Parasite and Insectary Core Facilities. This research was funded by the National Institutes of Health / National Institute of Allergy and Infectious Disease grant number R01AI141532 and the Bloomberg Philanthropies.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Implementation an efficient biobank in a research laboratory: ISO 9001:2015 certified

Carranza-Salazar, Claudia [1]; Juárez -Carvajal , Esmeralda [2];
Carreto-Binaghi, Laura E [1]; Chávez-Domínguez , Rodolfo L [1];
Viettri-Pinto, Mercedes [1] ; Zamudio-Meza, Horacio [1];
Corona -Galvan, Itzel A [1]; Reyna-Rosas, Edgar [1];
Palencia-Reyes, Andrea [1]; Nieto-Ponce, Milton [1];
Torres-Rojas, Martha [1].

[1]Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas. Laboratorio de Inmunobiología de la Tuberculosis. CDMX, México. [2]Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas. Laboratorio de alta contención biológica. CDMX, México.
E-mail: carranza.salazar.claudia@gmail.com

Despite the central role of biological samples in biomedical discovery, many research laboratories operate without robust sample storage systems. This compromises sample integrity, reproducibility, biosecurity, and the long-term usefulness of the data. Implementing structured sample storage policies and infrastructure is crucial to maintaining scientific quality, ensuring regulatory compliance, and promoting responsible conduct of research. It would be very beneficial for any research laboratory to move from sample storage to the implementation of a formal biobank. Biobanks constitute a critical infrastructure for biomedical research, as they systematically store biological specimens and databases, which are essential to maximize the utility of research and facilitate international collaborative efforts.

Most countries lack specific regulatory frameworks for biobanks, limiting the ability to implement simultaneous initiatives in line with international standards. Integration of quality management systems is essential to ensure sample quality and safety. In this paper, we analyze the importance of the storage conditions of biological samples and the steps necessary for the transition to a formal biobank. We proposed a guide to do so in the absence of a regulatory framework, based on the ISO 20387:2018 model, and included an example of successful implementation in a research science laboratory certified with ISO 9001:2015.

Transforming an internal sample storage system into a formal biobank is a strategic and scientific advance. It ensures that biological materials are managed with the rigor, transparency, and ethical responsibility that contemporary biomedical research demands. In this way, research institutions strengthen their capacity for innovation and collaboration.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

The effect of Mycobacterium tuberculosis-infected macrophages in the development of lung cancer

Carranza-Salazar, Claudia [1]; Chávez-Domínguez, Rodolfo Luis [1];
Viettri-Pinto, Mercedes Isabel [1]; Corona-Galván, Itzel Abigail [1];
Zamudio-Meza, Horacio [1]; Torres-Rojas, Martha [1].

[1]Instituto Nacional de Enfermedades Respiratorias, Departamento de Enfermedades Crónico-Degenerativas, Laboratorio de Inmunobiología de la Tuberculosis, Ciudad de México, México.
E-mail: carranza.salazar.claudia@gmail.com

Tuberculosis and lung cancer are two major public health issues worldwide and their relationship has been widely investigated. Multiple studies indicate that individuals with tuberculosis are at increased risk of developing lung cancer. Chronic inflammation triggered by Mycobacterium tuberculosis infection is thought to create a microenvironment for the emergence of incipient tumor cells. Previous research has reported that tuberculosis-infected macrophages are the source of inflammatory cytokines and other soluble factors implicated in epithelial cell transformation. However, the early participation of tuberculosis-infected macrophages in cancer development remains poorly understood. In this work, we studied the effect of the inflammatory microenvironment elicited by Mycobacterium tuberculosis infected-THP-1-derived macrophages in the proliferation rate of lung epithelial cells. THP-1 cells were differentiated into macrophages and were infected with the Mycobacterium tuberculosis H37Ra strain for 2 weeks. Cell culture supernatants were collected for cytokine quantification using Luminex. Infected macrophages were analyzed for the expression of surface markers of tumor-associated macrophages by flow cytometry. In addition, BEAS-2B immortalized lung epithelial cells were exposed to the cell culture supernatants, from THP-1 infected cells, for 6 weeks to assess alterations in proliferation. Infection of THP-1-derived macrophages induced the production of pro-inflammatory cytokines, including IL-6, TNF-alfa, IL-1b, and IFN-gamma. Interestingly, after two weeks of infection, CD14+ macrophages exhibited increased expression of CD163, HLA-DR, CD204, CD206, and CD73 which are molecules of M2 tumor-associated macrophages.

BEAS-2B cells exposed to the conditioned media showed a sustained increase in the proportion of cells in the G2/M phase, beginning at week two and persisted through week six. The chronic infection of macrophages with Mycobacterium tuberculosis promotes the emergence of cells expressing M2 tumor associated markers. These cells, may contribute to the establishment of a pro-tumorigenic microenvironment, potentially altering the proliferation dynamics of non-transformed epithelial cells.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Early Development of Cancer During Acute Terminal Infection in Experimental Malaria.

Carrasco-Ramírez, Elba [1]; Malagón-Gutiérrez, Filiberto [2];
Federico-Guerrero, Alfonso [2]; Carrasco-Ramírez, Liliana [3];
Flores-Guzmán, Fernando [4].

[1]UNAM. Unidad de Microscopía. Facultad de Medicina. Ciudad de México. México. [2]UNAM. Microbiología y Parasitología. Facultad de Medicina. Ciudad de México. México. [3]UNAM. Facultad de Ciencias Políticas y Sociales. Ciudad de México. México. [4]Universidad de Colorado. Denver. USA.

E-mail: elba.carrasco@facmed.unam.mx

Plasmodium yoelii yoelii (Pyy) infection in the murine model is widely used to study the pathophysiology of malaria. However, little is known about its possible relationship with malignant cellular transformation processes. A histopathological study was carried out to recognize the lesions that Pyy produces during the progress of the infection in the mouse, with the purpose of identifying the changes that suggest the beginning of the malignant transformation of the cells, and thus identify the mouse's risk of developing cancer, 6 groups of CD1 mice and a control group of 5 c/u mice were infected. Every 24 hr up to 6 days, a group of the problem subjects underwent an FDG-PET (18-fluoro-2-deoxyglucose-positron emission tomography) study. They were then bled white, blood smears were taken, 7 organs from each mouse were autopsied and dissected, which were histologically processed, to observe the pathological changes in organs over the course of the infection, from 24 hours post-infection until death. Radioactivity was measured in each organ. The blood was processed to measure plasma glucose and blood count. The smears were stained with Giemsa and parasitemia was estimated. The control group of healthy, uninfected mice was handled the same. Among the infected mice, two were detected with neoplasms, one with lymphoma of probable immunoblastic type, with lesions in the lung, spleen and mesenteric nodule and another with grade III astrocytoma in the cerebral cortex, both 5 days after infection. This study presents evidence of neoplastic lesions coinciding with the acute phase of Pyy infection in CD1 mice, suggesting a possible role of parasitic infection as a trigger or accelerator of latent oncogenic processes. Likewise, a limitation of FDG-PET to detect tumors in an acute systemic inflammatory context was observed. These results open new lines of research on the interaction between parasitic infections and tumor initiation.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

Effect of L-arginine and L-serine supplementation on neonatal T lymphocyte function

Carreón -Cortés, Ingrid Yaritzit [1 4]; Rodríguez -Jorge, Otoniel [1 4];
Santana-Calderón, María Angélica [1 4].

[1]Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México. [2]Instituto de Investigación en Ciencias Básicas y Aplicadas Cuernavaca, Morelos, México. [3]Centro de Investigación en Dinámica Celular, Cuernavaca, Morelos, México. [4]Laboratorio de Inmunología Celular y de Sistemas, Cuernavaca, Morelos, México.

E-mail: yaricaci@gmail.com

During the postnatal stage, the neonatal immune system slowly changes from a mainly innate response to more specific functions of the adaptive immune system. In this period, the innate immune system is the main defense, while the adaptive system is still developing. It responds more slowly, is more tolerant, and tends to develop a Th2-type profile. This helps newborns adapt to the outside microbial world and supports the formation of the microbiota. However, it also makes them more vulnerable to viral and bacterial infections. In fact, in 2019, 47% of deaths in children under five happened during the neonatal period, and about 24% of those were due to infections. This shows the need for strategies to regulate the neonatal immune response.

In this context, amino acids have been studied as possible immune system regulators. L-arginine and L-serine are important for the function and activation of CD4+ T cells. In our study, we analyzed the effect of these amino acids on neonatal CD4+ T cells by measuring the production of Th-type cytokines after stimulation with anti-CD3 and anti-CD28. Our results show that high concentrations of L-arginine reduce the production of effector Th cytokines, except for the Th1 profile, which stays active. This is important because the neonatal immune system usually favors a Th2 response, which is less effective against intracellular pathogens. The ability of L-arginine to keep the Th1 profile active suggests a possible positive effect in regulating the neonatal immune response.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Exploring the role of SPN-205: a nonsense-mediated decay targeted CD43 isoform

Carrillo-Carlos, Dulce Alejandra [1 2]; García-González, Carlos [1 2];
Chipres-Naranjo, Luis Eduardo [1]; Rosenstein-Azoulay, Yvonne Jane [1].

[1]Instituto de Biotecnología, Universidad Nacional Autónoma de México, Cuernavaca, Morelos, México. [2]Centro de Ciencias Genómicas, Universidad Nacional Autónoma de México, Cuernavaca, Morelos, México.

E-mail: alecarrillocarlos@gmail.com

CD43, encoded by the SPN gene, is a transmembrane sialomucin primarily studied in leukocytes. It plays an essential role in adhesion, migration, and the activation of immune cells. In cancer, CD43 plays a role in immune evasion, with its expression varying across different types of tumors. An important alteration in cancer is the dysregulation of nonsense-mediated decay (NMD), a highly conserved RNA quality control mechanism that degrades transcripts containing premature termination codons, preventing the production of potentially harmful truncated proteins. When NMD is dysregulated, it leads to the stabilization of aberrant isoforms and broad transcriptome remodeling. This remodeling can have dual outcomes: it may restore the expression of stress response factors that control the response to cancer, such as p53. Or on the contrary, promote the accumulation of transcripts that inhibit antitumor mechanisms, thereby promoting cancer development.

In this study, we analyzed publicly available transcriptomic datasets from The Cancer Genome Atlas (TCGA) to evaluate the differential expression of SPN and SPN-205, a predicted NMD-marked isoform of the SPN gene, in tumor versus normal samples. We found that SPN-205 is overexpressed in several tumor types, including acute myeloid leukemia (LAML), thymoma (THYM), tenosynovial giant cell tumor (TGCT), diffuse large B-cell lymphoma (DLBC), pancreatic adenocarcinoma (PAAD), and stomach adenocarcinoma (STAD). Notably, in LAML, the expression of SPN-205 reaches up to 32% of the expression level of canonical SPN. This suggests that SPN-205 may not be transcriptional noise, but could have yet unknown functions and interactions, potentially dysregulating the NMD pathway in certain types of cancer. To further explore its potential role, we will incorporate a functional analysis to assess the correlation between the expression of SPN and SPN-205 with gene expression signatures associated with cell proliferation, immune evasion, and apoptosis resistance.

Funded by DGPA/UNAM #IN22252.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Characterization of Immune Populations in Gastric Mucosa during HIV/ Helicobacter pylori Coinfection

Carvajal-Ruiz, Lady Laura [1 2]; Briceño-Cárdenas, Olivia [1]; Ordaz-Candelario, Karla Krystel [1]; Solís-Flores, Sebastián [1]; Godínez-Victoria, Marycarmen [2]; Soto-Nava, Maribel [1]; Carmona-Quirós, Aramis [1]; Ávila-Ríos, Santiago [1]; Torres-Ruiz, María Fernanda [1].

[1]Instituto Nacional de Enfermedades Respiratorias (INER), Centro de Investigación en Enfermedades Infecciosas (CIENI), Ciudad de México, México. [2]Sección de Estudios de Posgrado e Investigación, Escuela Superior de Medicina, Instituto Politécnico Nacional, Ciudad de México, México.

E-mail: ladylauracruiz@gmail.com

Helicobacter pylori (H.pylori) infection in people living with HIV (PLWHIV) has been associated with immunocompetence, characterized by higher CD4+ T cell counts and lower viral loads. However, the immunological mechanisms at the gastric mucosa remain poorly understood. This study aimed to characterize the immune populations present on the gastric mucosa of PLWHIV with or without H.pylori infection. Gastric biopsies from 15 PLWHIV were analyzed by multiparametric flow cytometry, including 8 individuals coinfected with asymptomatic H.pylori and 7 without H.pylori infection. Coinfected individuals showed a significantly higher frequency of leukocytes ($p = 0.014$), CD4+ T-cells ($p = 0.037$), CD19+ B-cells ($p = 0.035$), and CD16+ monocytes ($p = 0.004$) in the gastric mucosa compared to PLWHIV without H.pylori infection. Additionally, the frequency of CD4+/ HLA-DR+ T-cells was significantly lower on the coinfected group ($p=0.002$). These findings suggest a potentially protective immune profile in coinfected individuals, characterized by increased immune cell infiltration with reduced local activation. Further studies on gastric mucosa are needed to better understand the underlying mechanisms and the potential role of systemic immune regulation in PLWHIV with H. pylori coinfection. Exploring these localized interactions may provide key insights into mucosal immunity and reveal novel therapeutic strategies against H. pylori.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Study of the humoral response in patients with active pulmonary tuberculosis against mycobacterial antigens

Castañeda Casimiro, Jessica [1]; Diaz, Antonio Irving [1];
Hernández Solís, Alejandro [2 3]; Chacón Salinas, Rommel [1 4];
Wong Baeza, Isabel [1 4]; Serafín López, Jeanet [1 4].

[1]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas (ENCB), Instituto Politécnico Nacional (IPN), Ciudad de México, México. [2]Facultad de Medicina, Universidad Nacional Autónoma de México (UNAM), Ciudad de México, México. [3]Servicio de Neumología del Hospital General de México "Dr. Eduardo Liceaga", Secretaría de Salud, Ciudad de México, México. [4]Red de Salud del Instituto Politécnico Nacional. Ciudad de México, México.

E-mail: jcastanc@gmail.com

Tuberculosis (TB) is a disease considered a public health problem. Worldwide, there are an estimated 10 million cases and 1.6 million deaths annually. The etiological agent of this disease is Mycobacterium tuberculosis (M. tb) and it is considered that antibodies directed against this bacterium could help in the control of TB, since there are mechanisms such as opsonization, where macrophages through the Fc receptor bind the antibody, increasing their phagocytic capacity by more easily endocytosing the bacterium, in addition to inducing the formation of the phagolysosome. Antibodies also participate in the activation of the cytotoxic response of natural killer's cells. The aim of this study was evaluating the presence of different classes of Mtb-specific antibodies present in patients with active pulmonary TB (ATB) before and 6 months after treatment (TBT), as well as to analyze the different IgG subclasses in patients with ATB. Serum samples were obtained from patients with ATB, patients with TBT, and healthy donors, and the presence of IgA, IgM, and IgG antibodies against Mtb total soluble extract (MTSE) was assessed using ELISA. IgG1, IgG2, IgG3, and IgG4 subclasses directed against the same extract will also be determined in patients with ATB. In this study found that patients with ATB have increased levels of IgG, IgA, and IgM antibodies that recognize Mtb antigens compared to healthy individuals. Furthermore, the presence of IgG is significantly decreased in TBT patients, while IgM immunoglobulins tend to decrease, and IgA remains the same levels in patients with ATB and TBT.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Study of biological effect of extracellular vesicles from neutrophils during bacterial growth

Castañeda-Casimiro, Jessica [2]; Gordillo-Centeno, Ana Nadia [1];
Olvera-Medina, Saúl [1]; Serafín-López, Jeanet [2 3];
Wong-Baeza, Isabel [2 3]; Chacón-Salinas , Rommel [2 3];
Vázquez-Flores, Luis Francisco [1 3].

[1]Departamento de Bioquímica, Escuela Nacional de Ciencias Biológicas (ENCB), Instituto Politécnico Nacional (IPN), Ciudad de México, México. [2]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas (ENCB), Instituto Politécnico Nacional (IPN), Ciudad de México, México. [3]Red de Salud del Instituto Politécnico Nacional. Ciudad de México, México.

E-mail: jcastanc@gmail.com

Extracellular vesicles (EVs) are membrane structures with a lipid bilayer, which are released under physiological conditions or in response to a specific stimulus (e.g., the infection of a neutrophil by bacteria). Experimental evidence demonstrates the role of EVs in physiological processes, in addition to contributing to the exchange of genetic material between cells, the transport of oncogenic proteins, among others, making EVs potential candidates for use as biomarkers in diagnosis and in the targeted delivery of drugs. Previous results in our group confirmed the presence of molecules such as CD35, myeloperoxidase (MPO), elastase, and gp91phox (a subunit of NADPH oxidase) in EVs from neutrophils infected with Mycobacterium tuberculosis (EV-Mtb), which allows us to assume that these structures could behave as ecto-organelles with antimicrobial activity. The experiments of this study confirm that neutrophil-derived EVs are structures of heterogeneous size, which depend on the stimulus and the time after its purification. Furthermore, we demonstrated that Mtb-EVs modify the growth of pathogens such as Escherichia coli and Staphylococcus aureus when these bacteria are treated directly with EVs.

These results contribute to our understanding of the biological effects of EVs and suggest that these structures could act as biological agents with microbicidal action, which will inform future research into the treatment of bacterial infections

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Autophagy regulates Nakaseomyces glabrata clearance in human THP-1 macrophages

Castillo -Cruz, Juan [1]; Baltierra-Uribe, Shantal Lizbeth [2];
Castrejón-Jiménez, Nayeli Shantal [3]; López-Álvarez,
Xuxu Guadalupe [4]; Rojas-Guillen, Megan Zahori [5];
García-Pérez, Blanca Estela [6].

[1]Sección de Estudios de Posgrado e Investigación, Escuela Superior de Medicina, Instituto Politécnico Nacional. [2]Departamento de Microbiología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional. [3]Área Académica de Medicina Veterinaria y Zootecnia, Instituto de Ciencias Agropecuarias-Universidad.

E-mail: **juancast0508@gmail.com**

Nakaseomyces glabrata is an emerging opportunistic yeast with high resistance to azoles, biofilm-forming capacity, and tolerance to oxidative stress. Unlike other Candida species, N. glabrata can persist intracellularly in human macrophages by interfering with phagosome maturation and acidification. This study evaluated the role of autophagy as an immunological mechanism involved in the intracellular clearance of N. glabrata in THP-1-derived macrophages. Materials and Methods: An in vitro infection model was established using differentiated THP-1 human macrophages infected with N. glabrata. Autophagy was modulated pharmacologically using rapamycin (inducer) and wortmannin (inhibitor). Fungal intracellular burden was quantified through colony-forming units (CFU) at 1, 2, and 4 hours post-infection (hpi). The expression of LC3, p62, and MHC-II was assessed by Western blot, while intracellular trafficking was analyzed by confocal microscopy. Proinflammatory cytokine levels were measured by ELISA. All experiments were performed in triplicate in independent assays. The results showed that N. glabrata persists intracellularly and alters endosomal trafficking, preventing phagosomal acidification. Autophagy induction reduced fungal load, increased p62 expression, and partially restored macrophage phagocytic function. These findings suggest that autophagy actively contributes to the innate immune response against N. glabrata and may represent a targetable pathway to improve fungal clearance.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Evaluation of miRNA expression in macrophages infected in vitro with Nocardia brasiliensis

Castillo-Gálvez, Oscar Daniel [1]; Hernández-Armendáriz, Laura Karyme [1];
López-López, Nallely [1]; Macías-Segura, Noé [1]; Mejía-Torres, Manuel Guadalupe [1];
Rojas-Gutiérrez, Santiago Emilio [1]; Rosas-Taraco, Adrián Geovanni [1];
Salinas-Carmona, Mario César [1].

[1]Universidad Autónoma de Nuevo León, Servicio y Departamento de Inmunología, Monterrey,
Nuevo León, México.

E-mail: odcg97@outlook.com

MicroRNAs (miRNAs) are small non-coding RNAs that play regulatory physiological functions in processes such as the immune response. However, infection with certain pathogens affects their expression, potentially affecting the outcome of the infection. Although several studies have addressed the relevance of miRNA expression in Mycobacterium tuberculosis infections, information regarding intracellular infections with Nocardia brasiliensis is currently lacking. We evaluated the expression of miR-21, miR-26b, and miR-29c in human monocyte-derived macrophages infected in vitro with N. brasiliensis. In addition, an in silico analysis of target genes regulated by these miRNAs was performed using predictive databases, including TargetScan, ENCORI, miRDB, and MicroT, and experimentally validated databases, such as Tarbase and miRWalk. Finally, the identified genes were compared with profiles of differentially expressed genes reported in Mycobacterium tuberculosis infections to elucidate their role in regulating the immune response. An increased expression of miR-21, miR-26b, and miR-29c were observed in infected macrophages compared to non-infected controls. The in silico enrichment analysis revealed that miR-21-regulated genes, their involvement in the regulation of MAPK and ERK1/ERK2 pathways were found. MiR-26b is associated with protein modification processes, such as cytokines and chemokines, in addition to participating in cellular metabolism. In the case of miR-29c, it has a role in the regulation of the organization of the extracellular matrix, which is related to mycetoma formation. Therefore, alteration of these signaling pathways could contribute to a decrease in microbicidal activity and macrophage polarization during N. brasiliensis infection. Among the relevant genes regulated by each miRNA, the following were identified: CCL20, CCL1, FASLG, and STAT3 for miR-21, which are related to multiple inflammatory processes; PRKCD and PTEN for miR-26b, which are associated with the regulation of apoptosis; and TNFRSF1A and TRAF4 for miR-29c, both linked to TNF signaling, which is critical in M. tuberculosis infection.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Cytokines and Chemokines involved in the upregulation of CXCR4 in resting lymphocytes

Castillo-Sánchez, Marco Antonio [1]; Gamboa-Meraz, Uriel Alejandro [1]; Huerta-Hernández, Leonor [1].

[1]Instituto de Investigaciones Biomédicas, Departamento de Inmunología, UNAM, Ciudad de México.

E-mail: marcocassan98@gmail.com

CXCR4 is a chemokine receptor involved in multiple physiologic functions related to cell migration and also to pathogenic processes like metastatic cancer and HIV infection. CXCR4 is the coreceptor for aggressive HIV variants called X4 strains. During the natural HIV infection, the detection of X4 strains in the peripheral blood is associated with the transition from a partially asymptomatic stage to immunodeficiency (AIDS). Studies carried out in our laboratory have shown that minimum concentrations of a mixture of cytokines and chemokines released by activated PBMC (conditioned media) increase the expression of CXCR4 on the surface of resting memory T CD4 lymphocytes. Increased CXCR4 expression provoked an enhanced fusion of these lymphocytes with a cell line that expresses the HIV fusion protein from an X4 HIV strain.

To identify the specific factors promoting the CXCR4 expression, we tested the effect of twenty-six commercial cytokines and chemokines on the CXCR4 expression of resting memory T CD4 lymphocytes purified from healthy men blood donors. Lymphocytes were cultured in the absence of fetal bovine serum and in the presence of 5% human adult serum plus 1 ng/ml of the homeostatic cytokine IL-15, and then were exposed to individual cytokines and chemokines during 48 hours.

So far, we found that IL-1a, IL-4, IFN-gamma and GM-CSF upregulate the expression of CXCR4 at 1 ng/mL after 48 hrs. On the other hand, IL-9 and IL-10 reduce the expression of CXCR4 at the same concentration. So, it can be preliminarily concluded that the increase of CXCR4 is a dominant effect during the exposure of cells to conditioned media of activated cells. So, cytokines and chemokines may promote the infection of resting T CD4 lymphocytes by X4 HIV strains through the induction of CXCR4 expression.

The project is supported by grants IG200623 from PAPIIT-UNAM and CBF2023-2024-348 from SECIHTI, México.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Identification of microRNA drug targets for Alzheimer's and Diabetes Mellitus by network medicine.

Castillo-Velázquez, Ricardo; Lara-Ramírez, Edgar Eduardo;
Ruiz-Hernández, Sofia; Castañeda-Delgado, Julio Enrique;
López-Alvarenga, Juan.

[1]Centro de Investigación en Ciencias de la Salud y Biomedicina, Universidad Autónoma de San Luis, San Luis Potosí. [2]Unidad de Investigación Biomédica de Zacatecas, Instituto Mexicano del Seguro Social. [3]Laboratorio de Biotecnología Farmacéutica, Centro de Biotecnología Genómica, Instituto Politécnico Nacional, Reynosa.

E-mail: mcytq.ricardo@gmail.com

Type 2 diabetes mellitus (T2D) is a risk factor for the development of Alzheimer's disease (AD), and recent studies have revealed that both conditions share complex and interrelated pathophysiological mechanisms. In this context, network medicine offers a promising strategy to uncover common dysregulated processes between diseases such as AD and T2D. The present study aimed to identify differentially expressed genes (DEGs) shared by both conditions and to apply a network medicine approach to determine the microRNAs (miRNAs) involved in their association. For this purpose, gene expression microarray datasets comprising 384 control samples and 399 disease samples from individuals with AD and T2D were analyzed, revealing a small subset of 82 shared downregulated DEGs. These genes were significantly associated ($p < 0.01$) with biological processes related to chemical synaptic deregulation. The network analysis further identified 12 miRNAs linked to these DEGs, which are expressed in tissues relevant to both AD and T2D and are primarily associated with synaptic deregulation, cancer-related pathways, and AKT signaling. Additionally, small molecules such as steroidal anti-inflammatory agents, antineoplastic drugs, and glucose metabolites were predicted to be potential regulators of these miRNAs.

In conclusion, the application of network medicine enabled the identification of 12 miRNAs potentially involved in the connection between AD and T2D, suggesting they may serve as therapeutic targets for future treatment strategies; nonetheless, experimental validation of these findings is still required.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Drug repositioning of potential GPR15 inhibitors by molecular docking identifies effects on the signaling pathway and cell migration mediated by GPR15-GPR15L.

Castillo Velázquez, Ricardo [1,2]; Rangel, Yamili A. [1,2]; Macias
Pérez, Martha Edith [1]; Lara Ramírez, Edgar Eduardo [3];
Fernández Ruiz, Julio Cesar [1]; Portales Pérez, Diana Patricia [2];
Zarazúa Guzmán, Sergio [2]; Zapata Zúñiga, Martin [4,5];
Serrano Escobedo, Carmen Judith [1]; Rivas Santiago, Bruno [1];
Castañeda Delgado, Julio Enrique [1,6].

[1]Unidad de Investigación Biomédica de Zacatecas, Instituto Mexicano del Seguro Social (IMSS), Zacatecas, Zacatecas., México. [2]Posgrado en Ciencias Farmacobiológicas, Facultad de Ciencias Químicas, Universidad Autónoma de San Luis Potosí, San Luis Potosí, San Luis Potosí, México. [3]Centro de Biotecnología Genómica, Instituto Politécnico Nacional, Reynosa, Tamaulipas, México. [4]Hospital Rural No. 51 IMSS Bienestar, Villanueva, Zacatecas, México. [5]Facultad de Medicina y Ciencias de la Salud, Universidad Autónoma de Zacatecas, Zacatecas, Zacatecas., México. [6]Facultad de Medicina y Ciencias de la Salud, Universidad Autónoma de Zacatecas, Zacatecas, Zacatecas., México. [7]Investigadores por México, SECIHTI, Secretaría de Ciencias, Humanidades, Tecnología e Innovación.

E-mail: mcytq.ricardo@gmail.com

GPR15 is a chemokine receptor involved in lymphocyte migration to the colon, skin, and joints through its interaction with GPR15L. Several studies have linked this receptor to inflammatory diseases such as colitis, rheumatoid arthritis, and multiple sclerosis, as well as to an increased risk of myocardial infarction. Therefore, the study of GPR15 represents a potential target for understanding their biology and for the development of therapies. In this study, by means of bioinformatics approaches and molecular biology strategies and pharmacological inhibition assays, we identified compounds with potential antagonistic activity against GPR15. We performed analysis of PBMCs from healthy individuals and evaluated GPR15 signaling pathways in several competitive assays such as activation of beta-arrestin proteins by Western blot, Calcium flux analysis with Fluo4AM, expression by was also evaluated by flow cytometry, GPR15-mediated cell migration in response to GPR15L using Transwell chambers and blocking antibodies to both the receptor and the ligand. Based on the performed assays the compounds 1 and 2 tended to inhibit cell migration, at a higher rate compared to compound 3 promoted migrations, like that of the endogenous ligand. No significant differences were observed in the abundance of beta-arrestin-2 after stimulation with GPR15L, suggesting that GPR15 signaling is not dependent on these proteins. We confirmed that GPR15 mediated signaling is dependent on calcium and report on novel FDA approved molecules as potential inhibitors of GPR15. Taken together, our results indicate that two of the three compounds could act as potential GPR15 inhibitors.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Altered CD4+ and CD8+ T-cell Phenotype in HIV+ Individuals Convalescent from COVID-19

Castillo-Vera, Gonzalo Daniel [1 2]; Briceño-Cárdenas, Olivia [1]; de León-Rodríguez, Isabel [1]; Soto-Nava, Maribel [1];
Tapia-Trejo, Daniela [1]; Ávila-Ríos, Santiago [1].

[1]Instituto Nacional de Enfermedades Respiratorias (INER), Centro de Investigación en Enfermedades Infecciosas (CIENI), Ciudad de México, México. [2]Posgrado en Ciencias Biológicas, Facultad de Medicina, Universidad Nacional Autónoma de México (UNAM), Ciudad de México, México.

E-mail: gonzdan.cv@gmail.com

The long-term impact of convalescent COVID-19 on T-cell immunity in people living with HIV (PLWH) remains poorly characterized. In this study, we characterized the phenotype of T cells in PLWH previously infected with SARS-CoV-2. A total of 48 participants were divided into four groups: PLWH on antiretroviral therapy (ART) convalescent from COVID-19 at five months post-infection (H+C+, n=11), PLWH on ART without prior SARS-CoV-2 infection (H+C-, n=12), HIV-negative individuals convalescent from COVID-19 (H-C+, n=12), and HIV- and SARS-CoV-2-negative healthy controls (H-C-, n=13). Cryopreserved peripheral blood mononuclear cells (PBMCs) were analyzed by multiparametric flow cytometry to assess markers of proliferation (Ki67), co-activation (CD28), co-inhibition (CTLA-4), and cytotoxicity (perforin) on CD4+ and CD8+ T cells. H+C+ individuals exhibited a persistently higher frequency of CD4+Ki67+ T cells compared to H−C+ (p=0.0134), along with elevated CD28 expression on both CD4+ (vs. H−C−, p=0.0318) and CD8+ T cells (vs. H−C−, p=0.0239). Additionally, CD8+ T cells from the H+C+ group showed decreased CTLA-4 expression (vs. H−C−, p=0.005) and reduced perforin levels (vs. H−C+, p=0.0035), indicating compromised cytotoxic potential. At five months post-infection, PLWH convalescent from COVID-19 exhibit an altered T-cell profile marked by increased co-activation, reduced immune regulation, and diminished effector function. Our findings highlight the need for further long-term investigation into the mechanisms underlying these immune alterations, as well as the study of other T-cell subsets and markers.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Effect of Polysaccharide-Modified Hybrid Materials on the viability of macrophages

Castrejón -Jiménez, Isabel Amairani [1]; García-Pérez, Blanca Estela [1]; Castrejón-Jiménez, Nayeli Shantal [2]; Baltierra-Uribe, Shantal Lizbeth [1]; Téllez-Jurado, Lucía [3].

[1]Departamento de Microbiología, Instituto Politécnico Nacional, Escuela Nacional de Ciencias Biológicas Prolongación de Carpio y Plan de Ayala S/N, Col. Santo Tomás, Ciudad de México 11340, México. [2]Área Académica de Medicina Veterinaria y Zootecnia, Instituto de Ciencias Agropecuarias-Universidad Autónoma del Estado de Hidalgo, Av. Universidad No. 133. Col. San Miguel Huatengo, Santiago Tulantepec de Lugo Guerrero, Hidalgo, México, C.P. 43775. [3]Instituto Politécnico Nacional – ESIQIE, Departamento de Ingeniería en Metalurgia y Materiales, Unidad Profesional Adolfo López Mateos (UPALM), Av. Instituto Politécnico Nacional S/N, Zacatenco, 07738, Ciudad de México, México.

E-mail: icastrejonj2300@alumno.ipn.mx

This research is based on the high prevalence of bone defects worldwide. In Mexico, osteoporosis has been reported to affect more than 10 million people and contribute to between 5,000 and 7,000 fractures annually, more than 90% of which require surgical intervention. The development of hybrid biomaterials combining polydimethylsiloxane (PDMS) and tetraethylorthosilicate (TEOS) and modified with compatible elements has been explored. Hybrid materials modified with polysaccharides, such as starch and chitosan, support osteoblast adherence and proliferation. However, other cells are necessary to repair and contribute to cellular microarchitecture and the microenvironment for bone tissue regeneration. The macrophages regulate the inflammatory response in bone damage and promote successful repair. This study evaluates the regenerative potential of these hybrid materials in THP-1 macrophages in vitro, focusing on their impact on cell viability. The Alamar blue assay was used to evaluate metabolic activity and cellular viability of macrophages exposed to hybrid materials modified with 0, 5, and 7% starch and 0, 3, and 5% chitosan. The results demonstrated a significant increase in metabolic activity of macrophages in interaction with materials containing 5% and 7% starch, suggesting biocompatibility and the ability to enhance cellular activity. In contrast, chitosan-modified biomaterials decrease the metabolic activity of macrophages. These results highlight the influence of starch-based polysaccharides on THP-1-mediated metabolic activity in macrophages, suggesting the potential regenerative potential of these hybrid biomaterials. However, further research is required to characterize the dynamics of the immune response. This study provides crucial information for the design of advanced biomaterials with effective applications in bone tissue engineering.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Evaluation of the impact of culture media during DMSO neutrophilic differentiation of HL-60 cells

Cázares-Preciado, Jorge Andrés [1]; Cruz-Cárdenas, José Antonio [1];
López-Arredondo, Alejandra [1]; Brunck, Marion E. G. [3].

[1]Escuela de Ingenierías y Ciencias, Tecnológico de Monterrey, Monterrey, Nuevo León, México. [2]The Institute for Obesity Research, Tecnológico de Monterrey, Monterrey, México.

E-mail: marion.brunck@tec.mx

The promyelocytic HL-60 cell line can be differentiated toward neutrophil-like cells and has been historically used as a surrogate to study human neutrophil biology in vitro. Multiple differentiation protocols have been reported to generate neutrophil-like HL-60 cells, with limited consideration of how media metabolites might influence cell identity and functions. Here, we investigate the impact of the 3 most common culture media published for neutrophil-like cell differentiation with 1.3% DMSO over 5 and 7 days. We compared the impact of higher glucose in IMDM-20 cell differentiation over 7 days. From the 3 most common culture media, IMDM-20 led to the highest proliferation rate and cell yield during differentiation. Neutrophil-like cells produced in IMDM-20 and RPMI-10 exhibited significantly higher proportions of CD15+CD11b+ neutrophil-like cells, and significantly higher bacterial clearance compared to DMEM-10. Culture media did not affect phagocytosis, but using RPMI-10 over 5 days led to significantly higher ability to produce ROS. Using IMDM-20 correlated with significantly more IL-6 and IL-1β in culture supernatant following stimulation with opsonized UV-killed E. coli. Higher glucose supplementation in IMDM-20 correlated with an upregulation of CD11b and increased phagocytosis. Increased glucose concentrations correlated with features described in hyperglycemic donor neutrophils including increased CD11b and phagocytosis. Overall, these results invite for rational media choice when inducing HL-60 differentiation to study neutrophil biology *in vitro*.

TRABAJO NO PRESENTADO

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

Unlocking the Potential of Neonatal CD4+ T Cells with Cytokine Therapy

Cedillo -Baños, Alejandra [1]; Santana-Calderón, Angélica [1];
Rodríguez Jorge, Otoniel [1].

[1]Universidad Autónoma del Estado de Morelos, Instituto de Investigación en Ciencias Básicas y Aplicadas, Cuernavaca, Morelos, México.

E-mail: alejandra.cedilloban@uaem.edu.mx

Newborns are a population highly susceptible to infections, exhibiting a high morbidity and mortality rate. Their immune system differs from that of adults.

In response to stimulation, neonatal CD4+ T cells show a limited production of effector cytokines, particularly those associated with the Th1 phenotype. However, they respond with a higher proliferation rate than adult cells.

Notably, neonatal T cells have demonstrated the capacity to mount responses like their adult counterparts in specific conditions. For instance, in a murine model of influenza virus infection in the respiratory tract, neonatal CD4+ T cells responded even more robustly than adult cells. Additionally, the BCG vaccine in neonates has been shown to induce a Th1-type response.

An efficient effector response relies on antigen-presenting cells (APCs). APCs' higher antigen presentation capacity is associated with increased expression of co-stimulatory molecules and cytokines. We hypothesize that neonatal T-cell responses could show a more robust effector capacity in the presence of relevant cytokines, such as IL-12, IFN-β, IL-1β, and IL-6.

Therefore, in this work, we aimed to characterize the activation response of neonatal CD4+ T cells to stimulation through the TCR/CD28 in the presence of IL-12, IFN-β, IL-1β, or IL-6 cytokines. Cellular activation and phenotypes were assessed based on the expression of activation markers CD69 and CD25, effector cytokine production, expression of transcription factors, and cell proliferation as compared to the response of adult CD4+ T cells, all evaluated through flow cytometry.

Our preliminary results show that, in general, cytokine supplementation promotes increased activation and cytokine production. However, in neonatal CD4+ T cells, a greater additive effect was observed, even more with IL-6 supplementation. While in adult cells, this cytokine did not have the same effect. These data suggest that there is a differential response between neonatal and adult CD4+ T cells to the same stimulus.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Descubrimiento de un panel de anticuerpos de dominio único (SDAB) anti-PD-1

Ceja -Garcia, Erendira Itzel [1]; Almagro-Dominguez, Juan Carlos [1, 2, 4];
Perez-Tapia, Mayra Perez [1,2,3,5]; Castellaño-Gómez, Keyla Maria [1,2].

[1]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Ciudad de México, México. [2]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Alcaldía Miguel Hidalgo, Ciudad de México, México. [3]Universidad Nacional Autónoma de México (UNAM), Ciudad de México, México. [4]GlobalBio, Inc., Cambridge, MA, EE. U. [5]Instituto Politécnico Nacional (ENCB), Departamento de Inmunología Ciudad de México, México.

E-mail: erendiraitzelc@gmail.com

Cancer remains one of the leading global health issues, responsible for around 9.7 million deaths annually. This has driven the rapid development of therapeutic strategies, with immunotherapy standing out for its ability to enhance immune responses. Particularly, antibodies targeting immune checkpoints such as PD-1 have shown promise, as blocking its interaction with PD-L1 reactivates T cells to attack tumor cells. However, conventional antibodies face limitations due to their size and structural complexity.

To address these challenges, smaller antibody fragments like single-domain antibodies (sdAbs) have been developed, offering advantages in size, stability, and specificity. These sdAbs, with molecular weights of 12–15 kDa, are highly stable and can access hidden epitopes. This project aimed to obtain a panel of human sdAbs against PD-1 using a fully human semi-synthetic library and phage display technology.

The experimental design included four stages: selection, screening, characterization, and fusion to a human Fc domain. Phages specifically binding to hPD-1 were selected and amplified in E. coli TG1. After three selection rounds, clones with specific binding were identified, sequenced, and analyzed, revealing three unique sequences derived from the human IGHV3-23 gene. The sdAbs were expressed, purified via protein A affinity chromatography, and evaluated through binding assays.

Further characterization included concentration and purity analysis by UV spectrophotometry and SDS-PAGE. The selected sdAb was fused to a human Fc region via overlap PCR, purified, and tested for functionality using Jurkat NFAT cell binding assays.

Although we successfully obtained anti-PD-1 single-domain antibodies that bind to recombinant protein, they did not show binding to the native protein. These findings highlight the need for further optimization to improve their therapeutic potential.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Implementation of a platform for CAR-T cell manufacture and evaluation

Cenobio-Hernández, Luis A. [1 2]; Rubio-Robles, Rosa M. [2];
Aguilar-Fuentes, Rosa E. [2 3]; Madera-Salcedo, Iris K. [2];
Rosetti-Sciutto, Florencia [2]; Crispín-Acuña, José C. [2 3].

[1]Posgrado en Ciencias Bioquímicas, Universidad Nacional Autónoma de México, México City, México. [2]Departamento de Inmunología y Reumatología, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, México City, México. [3]Escuela de Medicina y Ciencias de la Salud, Tecnológico de Monterrey, Monterrey, México.

E-mail: cenobio.070100@gmail.com

Adoptive transfer of T cells engineered to express chimeric antigen receptors (CAR-T cells) has produced promising results in patients with hematological malignancies. CARs are composed of an extracellular tumor-associated-antigen (TAA) recognizing domain, typically derived from antibodies. This domain is anchored to the cell membrane by a flexible hinge region and fused to intracellular costimulatory and T cell activation domains. Binding of the CAR to the TAA activates the T cell, allowing it to exert effector functions. During the CAR T cell manufacturing process, T cells are isolated from the peripheral blood of a patient, they are edited to express the CAR, expanded, and reinfused. Before reinfusion, CAR T cells must be tested to demonstrate identity, purity, potency, and safety. Here, we describe a methodology to efficiently generate and evaluate CD19-directed CAR-T cells from healthy donors. We will describe the generation of CAR-encoding lentiviral particles, in vitro T cell activation, transduction and expansion, as well as the methods used to evaluate transduction efficiency, specific cytotoxicity assays, and a xenograft model for in vivo quantification of CAR-T cells killing capacity. These systems demonstrated identity by CAR surface expression with acceptable transduction efficiency, specificity (by absence of killing in control cell line not expressing CD19), and in vitro and in vivo potency, which led us to design our own CAR constructs and rationally tune their components.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Tamoxifen induces sexually dimorphic patterns in the immune response to malaria

Cervantes-Candelas, Luis Antonio; Legorreta-Herrera, Martha.

FES Zaragoza UNAM, Laboratorio de Inmunología Molecular, CDMX, México.

* E-mail: cervantescandelasluis@gmail.com

Malaria exhibits a clear sexual dimorphism, women develop lower parasitemia and mortality than men do; therefore, it has been proposed that estrogens that have immunopotentiating properties participate in this phenomenon because the cells of the immune response that eliminate the parasite have receptors for these hormones. Therefore, in this work, we studied the role of estrogens in the sexual dimorphism of the immune response in malaria. We blocked estrogen receptors with tamoxifen and evaluated their effects on parasitemia, the number of CD4+, CD8+, T lymphocytes and B220+ cells and the number of macrophages and NK cells in the spleen; and the serum concentrations of IL-2, IL-4, IL-6, IL-17, IFN-γ, TNF-α and IL-10 in male and female CBA/Ca mice infected with Plasmodium berghei ANKA. Tamoxifen was found to increase the parasitemia of male and female mice infected with Plasmodium berghei ANKA. This finding is likely a consequence of tamoxifen decreasing the number of B220+ cells in males and females. This findings suggest that estrogens modulate the increase in B220+ cell numbers in both sexes. Interestingly, tamoxifen increased the number of NK cells only in females and decreased the number of TCD4+ and TCD8+ lymphocytes only in males, suggesting that estrogens dimorphically regulate the maturation or fate of NK cells and T lymphocytes. However, tamoxifen did not affect the serum concentrations of cytokines. Our results demonstrate a clear estrogen-induced dimorphic response that impacts the number and function of immune response cells in Plasmodium berghei ANKA-infected CBA/Ca mice.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

BTK Inhibition by Valproic Acid: In Silico approach for Allergy Treatment

Martínez-Martínez, Ricardo Emmanuel [1]; Calva-Hernández, Carlos Iván [1]; Rodríguez-López, Gloria Mariana [2]; Soria-Castro , Rodolfo [1]; Ruiz-Sánchez, Bibiana Patricia [1]; Chávez-Blanco, Alma Delia [3]; Pérez-Tapia, Sonia Mayra [1 4]; Pavano-Rodríguez, Claudia G. [5]; Chacón-Salinas, Rommel [1].

[1]Instituto Politécnico Nacional, Escuela Nacional de Ciencias Biológicas, Departamento de Inmunología, CDMX, México. [2]Universidad Nacional Autónoma de México, Facultad de Veterinaria y Zootecnia, Departamento de Microbiología e Inmunología, CDMX, México. [3]Instituto Nacional de Cancerología, División de Ciencia Básica, CDMX, México. [4]Instituto Politécnico Nacional, Escuela Nacional de Ciencias Biológicas, Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), CDMX, México. [5]Instituto Politécnico Nacional, CECYT10, CDMX, México.

E-mail: rommelchacons@yahoo.com.mx

Mast cells drive type I hypersensitivity through activation via the FcεRI receptor, initiating a signaling cascade involving SyK, BTK, and PLCγ2 that culminates in cell degranulation. Valproic acid (VPA), a known antiepileptic drug, reduces PLCγ2 phosphorylation independently of SyK, suggesting a possible effect on BTK. To explore this, we conducted in silico molecular docking to assess VPA's interaction with BTK. Structural modeling and computational docking revealed favorable non-covalent binding of VPA near BTK's catalytic site, in a region distinct from that of irreversible inhibitors. These results propose that VPA may function as a reversible BTK inhibitor, highlighting its potential for repurposing in allergy treatment.

This project is funded by Secretaría de Investigación y Posgrado, IPN.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Antibodies responses against SARS-CoV-2 vaccines in teachers and students from Chihuahua, Mexico.

Chavarría-Bencomo, I. Valeria [1]; Chávez-Trillo, Carlos [2];
López-Quiñonez, Mónica G. [2]; Adame-Gallegos, Jaime R. [1];
Zurawski-, Sandra [3]; Espino-Solís, Gerardo P. [2];
Zurawski-Gerard [3].

[1]Universidad Autónoma de Chihuahua, Facultad de Ciencias Químicas, Chihuahua, Chihuahua, México. [2]Universidad Autónoma de Chihuahua, Laboratorio Nacional de Citometría de Flujo, Facultad de Medicina y Ciencias Biomédicas, Chihuahua, Chihuahua, México. [3]Universidad Autónoma de Chihuahua, Laboratorio Nacional de Citometría de Flujo, Facultad de Medicina y Ciencias Biomédicas, Chihuahua, Chihuahua, México. [4]Universidad Autónoma de Chihuahua, Facultad de Ciencias Químicas, Chihuahua, Chihuahua, México. [5]Biotechnology Center at Baylor Institute for Immunology Research, Dallas, TX, United States. [6]Universidad Autónoma de Chihuahua, Laboratorio Nacional de Citometría de Flujo, Facultad de Medicina y Ciencias Biomédicas, Chihuahua, Chihuahua, México. [7]Biotechnology Center at Baylor Institute for Immunology Research, Dallas, TX, United States.

E-mail: p185908@uach.mx

Coronavirus disease 2019 (COVID-19) was a very important pandemic for the entire world. The causative agent of this disease, Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), has spread worldwide since 2019. The vaccination was a effective method for control. Therefore, the determination of antibodies against SARS-CoV-2 was very important to identify the serological status of post-vaccinated individuals. We determined IgM and IgG concentrations in blood against 3C-like proteinase (3CLpro), nuclear protein (NP), and receptor binding domain (RBD) of SARS-CoV-2 in vaccinated teachers and students among a university population from Chihuahua, Mexico. Humoral response surveillance against the proteins of SARS-CoV-2 was carried out in 239 samples, of these, 172 belonged to students: 27.9% were vaccinated with AstraZeneca, 32.6% with Sinovac, 24.4% with Pfizer-BioNTech, 15.1% with other vaccines. 67 samples were from teachers who received CanSino vaccine. We found significant differences in the levels of IgG between preimmunization serum and post-immunization serum from teachers that were vaccinated with CanSino. Sera from asymptomatic individuals showed no differences before and after immunization. The students groups vaccinated with AstraZeneca, Pfizer and Sinovac did not show significant differences in anti-RBD IgG antibody titers compared to the positive control group, except for a Pfizer non-COVID-19 subgroup. No significant differences were observed between individuals previously diagnosed with COVID-19 and those who were not infected. These results provide a general idea about the immunological status in-person classes resumed.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Association of PD-1/PD-L Axis in cTfh, Tph, and atBm cells in SLE.

Chávez-Mireles, Ramón [1]; Salazar-Camarena, Diana Celeste [2 4];
Ortiz-Lazareno, Pablo Cesar [3]; Marín-Rosales, Miguel [4 5];
Espinoza-García, Noemí [4]; Reyes-Mata, María Paulina [4];
Palafox-Sánchez, Claudia Azucena [2 4].

[1]Doctorado en Ciencias Biomédicas (DCB), Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara 44340, Jalisco, México. [2]Centro Universitario de Ciencias de la Salud (CUCS), Guadalajara 44340, Jalisco. México. [3]División de Inmunología del Centro de Investigación Biomédica de Occidente (CIBO). [4]Grupo de Inmunología Molecular, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara 44340, Jalisco, México. [5]Hospital General de Occidente, Secretaría de Salud Jalisco, Guadalajara 45170, Jalisco, México.

E-mail: ramon.chavez9890@alumnos.udg.mx

Systemic lupus erythematosus (SLE) is a prototypic systemic autoimmune disease driven by a breach of immunological tolerance and sustained by dysregulated T–B cell interactions. Circulating T follicular helper (cTfh) and peripheral helper T cells (Tph) are implicated in extrafollicular B cell responses, particularly in inflamed tissues, and contribute to the differentiation of autoreactive atypical memory B cells (atBm). These subsets are characterized by elevated PD-1 expression, yet the functional implications of PD-1 and its ligands (PD-L1, PD-L2) in SLE pathogenesis remain elusive. We conducted a cross-sectional, observational study including 20 patients with SLE and 16 age- and sex-matched healthy controls (HCs). Peripheral blood mononuclear cells (PBMCs) were analyzed using full-spectrum flow cytometry to evaluate surface expression of PD-1, PD-L1, and PD-L2 on Tph, cTfh, and B cell subsets. Soluble forms (sPD-1, sPD-L1, sPD-L2) were quantified by ELISA. SLE patients exhibited increased frequencies of cTfh and Tph subsets. PD-1 expression was significantly upregulated in cTfh but not in Tph. PD-L1 and PD-L2 showed no overall membrane expression differences on T cells subpopulations, although altered ligand expression was observed in B cell subsets: increased PD-L2 in switched memory (SWM) and decreased in DN2 cells. Soluble PD-1, PD-L1, and PD-L2 levels were significantly elevated in SLE and positively correlated with disease activity (SLEDAI-2K). Notably, sPD-L1 levels negatively correlated with PD-L1□ cTfh and Tph frequencies. These data suggest that heightened PD-1 expression on helper T cell subsets in SLE may reflect a chronic activation state insufficiently counterbalanced by PD-L1/PD-L2 engagement, potentially due to ligand shedding or defective APC signaling. Moreover, impaired PD-1/PD-L regulation in B cell compartments may promote persistence of autoreactive clones. Our findings underscore the relevance of PD-1/PD-L axis imbalance as a contributing mechanism in SLE immunopathogenesis and as a potential immunoregulatory target.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

The CD43 sialomucin promotes GLUT-1 expression in T cells

Chipres-Naranjo, Luis Eduardo [1]; Melchy-Pérez, Erika [1];
García-González, Carlos [2]; Rodríguez-Jorge, Otoniel [3];
Rosenstein-Azoulay, Yvonne Jane [1].

[1]Instituto de Biotecnología, Universidad Nacional Autónoma de México, Cuernavaca, Morelos, México. [2]Licenciatura en Ciencias Genómicas, Universidad Nacional Autónoma de México, Cuernavaca, Morelos, México. [3]Centro de Investigación en Dinámica celular, Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México.

E-mail: luis.chipress@gmail.com

CD43 is a type I transmembrane sialomucin abundantly expressed in T lymphocytes. In normal T cells, CD43 delivers co-stimulatory signals involved in thymocyte selection, maturation, migration, adhesion, and the activation of mature T cells. Its expression in tumor cells is associated with a poor prognosis. We hypothesized that CD43 conveys environmental cues necessary for cell survival and proliferation. We specifically investigated whether CD43-mediated signals participate in the metabolic adaptations that T cells undergo in response to activation conditions where glycolysis supplies the bioenergetic demands of proliferation. We evaluated the expression levels of the glucose transporter GLUT-1 as well as the glycolysis and glutaminolysis rates in normal human peripheral blood CD4+ T cells and Jurkat cells (acute lymphocytic leukemia T lymphoblast) following stimulation with TCR+CD43, TCR+CD28, CD43, or CD28. At 48h post-stimulation, CD4+ T cells exhibited a ~10-fold increase in GLUT-1 membrane expression in response to the TCR+CD43 stimulus compared to unstimulated cells, outperforming the TCR+CD28 stimulus. In contrast, Jurkat cells showed increased total and membrane GLUT-1 expression only when stimulated with CD43 or CD28 alone, but not when co-stimulated with TCR+CD43, TCR+CD28. Interestingly, Jurkat cells showed no increase in glucose uptake but displayed decreased lactate levels, increased glutamine intake, and no change in glutamate release. We will present data on the role of CD43 in modulating cellular metabolism, specifically examining glycolytic activity, mitochondrial function, and key metabolic regulators, including Myc and HIF-1α, in response to CD43 co-stimulation in human T cells. Collectively, this data reveal an unforeseen role for CD43 as a regulator of T-cell and tumor cell metabolism.

Funded by DGAPA/UNAM IN222523.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Immunological profile of Leishmania mexicana and Brichromyia olmeca in inhabitants of Yucatan

Chiyean-Acosta, Angela Guadalupe [1]; Canché-Pool, Elsy Beatriz [1 2]; Reyes-Novelo, Enrique Alberto [1 2 3].

[1]Centro de Investigaciones Regionales Dr. Hideyo Noguchi (UADY), Laboratorio de Zoonosis, Mérida, Yucatán, México. [2]Centro de Investigaciones Regionales Dr. Hideyo Noguchi (UADY), Laboratorio de Zoonosis, Mérida, Yucatán, México. [3]Centro de Investigaciones Regionales Dr. Hideyo Noguchi (UADY), Laboratorio de Zoonosis, Mérida, Yucatán, México.

E-mail: angela.chiyean23@gmail.com

Introduction: The immune response evoked by an infection is divided into two groups (innate and adaptive). In leishmaniasis, the innate and adaptive cellular immune responses are known, but the role of the humoral adaptive response is unknown. A new approach currently exists that evaluates this response using vector saliva.

Objective: Evaluate the humoral immune response against L. mexicana and the saliva of Bi. olmeca olmeca in people living in localities with records of localized cutaneous leishmaniasis (LCL) in Yucatán.

Methodology: To evaluate this immune response, sera will be collected from people in localities with records of LCL cases in the state. Sandflies will also be collected in these locations, and the Leishmania mexicana strain will be cultured to obtain the antigens used to perform indirect ELISAs to detect IgG antibodies, as well as their subtypes (IgG1, IgG2, IgG3, and IgG4) present in the serum. A Western blot will then be performed to characterize the antigens recognized by the various antibodies through their molecular weight.

Preliminary results: 123 specimens belonging to the order Diptera were identified, of which 57 were female, while the remaining specimens were male. Five male species were identified (Brumptomyia hamata, Brumptomyia mesai, Lutzomyia cruciata, Psathyromyia shannoni and Dampfomyia deleoni), while based on females, two species have been identified (Dampfomyia deleoni and Bichromomyia olmeca olmeca).

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Transcriptional regulation of CCR6 by HIF-1 and its implication in pediatric Medulloblastoma

Cocoletzi -Bautista, Jonathan Erik [1, 2]; Luria -Pérez, Rosendo [1];
Baay-Guzmán, Guillermina Juliana [1].

[1]Children´s Hospital of México Federico Gómez, Unit of Investigative Research on Oncological Diseases, México City, México.. [2]National Polytechnic Institute, Graduate Studies Unit, México City, México.

E-mail: jonathan.cocoletzi.ba@gmail.com

Medulloblastoma (MB) is an aggressive embryonal tumor of the central nervous system (CNS) and one of the most common malignant brain tumors in the pediatric population. Chemokines and their receptors have been shown to play dual roles in tumorigenesis, acting either as pro-inflammatory or immunosuppressive agents within the tumor microenvironment. One of the key regulators in this context is the Hypoxia Inducible Factor 1 (HIF-1), a transcription factor activated under hypoxic conditions, which has been associated with poor prognosis, metastasis, low survival rates, and resistance to therapy in various cancers.

Previous studies have demonstrated that HIF-1 regulates the expression of the chemokine CCL20; however, its role in the transcriptional regulation of the corresponding receptor CCR6, remains unclear. The CCL20-CCR6 axis has been implicated in tumor progression and poor clinical outcomes in different malignancies. This study aimed to evaluate the transcriptional regulation of CCR6 by HIF-1 and its potential role in the pathophysiology of medulloblastoma.

Bioinformatic analysis of the CCR6 promoter identified three putative Hypoxia Response Elements (HREs) located at positions -1046 to -1053, -545 to -552, and +110 to +117 base pairs relative to the transcription start site. The activity of these HREs was confirmed through reporter plasmid assays and site-directed mutagenesis. Additionally, overexpression of HIF-1□ and CCR6 was observed in PC-3 cells and in 41 pediatric medulloblastoma biopsy samples analyzed via immunohistochemistry.

Overall, the results suggest that HIF-1 transcriptionally regulates the CCR6 receptor, potentially contributing to the development and progression of pediatric medulloblastoma. This regulatory pathway may serve as a promising therapeutic target and prognostic marker in this aggressive tumor.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Establishment of a Murine Model of B-ALL for the Preclinical Evaluation of CAR-T Cells

Consuegra-Sierra, Johan Sebastián; Rodríguez-Flores, Sofia Nohemí; Pedraza-Escalona, María Martha; Pérez-Tapia, Sonia Mayra.

[1]Estudiante del Programa de Posgrado en Ciencia y Tecnología de Vacunas y Bioterapéuticos, Instituto Politécnico Nacional, Ciudad de México, México. [2]Secretaría de Ciencia, Humanidades, Tecnología e Innovación (SECIHTI), Ciudad de México, México. [3]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Ciudad de México, México. [4]Laboratorio Nacional para Servicios Especializados de Investigación, Desarrollo e Innovación (I+D+i) para Farmoquímicos y Biotecnológicos (LANSEIDI-FarBiotec-CONAHCyT), Ciudad de México, México. [5]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Ciudad de México, México.

E-mail: jhoansierra51@gmail.com

B-cell acute lymphoblastic leukemia (B-ALL) is a hematological malignancy characterized by the malignant expansion of lymphoblasts in the bone marrow, predominantly affecting the pediatric population. Despite improvements in survival due to conventional therapies, a significant proportion of patients relapse or develop resistance. In this context, chimeric antigen receptor T cells (CAR-T) targeting CD19 have demonstrated remarkable efficacy. However, the implementation of this technology in Mexico is limited due to its high cost and lack of local availability for B-ALL treatment. This project aims to establish an immunodeficient murine model (NSG) to enable the preclinical evaluation of locally generated CAR-T cells, as a critical step before clinical application. Stable transduction of NALM6 leukemic cells with GFP and luciferase reporter genes was achieved, resulting in three unique clones with leukemogenic capacity confirmed in vivo through bioluminescence imaging. These clones allow monitoring of leukemia progression and engraftment in NSG mice. Preclinical validation in murine models is essential to ensure the efficacy and safety of therapies prior to clinical use in humans. Additionally, methodologies were standardized for the generation of CAR-T cells from human lymphocytes, including in vitro potency assays and in vivo functional evaluation protocols. This work represents a key advance toward developing accessible, locally produced CAR-T therapies in Mexico. The established preclinical model will permit validation of efficacy and safety prior to clinical phases, contributing to the strengthening of national technological sovereignty in biotherapeutics and the eventual implementation of these therapies within the healthcare system.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Elevated HIF-1α correlates with mitochondrial activity in CD14+IRF8+ monocytes and exhausted CD8+PD1+ T-lymphocyte phenotype in breast cancer patients

Contreras-Briones, Dalila del Socorro; Ibáñez-Méndez, Reyes de Jesús [1]; Turiján-Espinoza, Eneida [1,2]; Martínez-Leija, Miguel Ernesto [1,2]; Lara-Hernández, Ignacio [1]; González-Amaro, Roberto [1]; Gutiérrez-Gil, Mary Carmen [4]; Mendoza-Oliva, Dolores Leticia [4]; Guel-Pañola, Jaime Arturo [3]; Portales-Pérez, Diana Patricia [1,2].

[1]Research Center for Health Sciences and Biomedicine (CICSaB), Autonomous University of San Luis Potosí. San Luis Potosí, S.L.P. [2]Faculty of Chemical Sciences, Autonomous University of San Luis Potosí. San Luis Potosí, S.L.P. [3]Department of Oncology, "Dr. Ignacio Morones Prieto" High Specialty Regional Hospital, San Luis Potosí, S.L.P., México. [4]Faculty of Medicine, Universidad Autónoma de San Luis Potosí. San Luis Potosí, S.L.P.

E-mail: dalila.contreras@uaslp.mx

The tumor microenvironment, characterized by hypoxia, negatively impacts mitochondrial function and promotes immune cell exhaustion. Under these conditions, HIF-1α drives glycolytic metabolism, leading to increased exhaustion of CD8+PD-1+ T lymphocytes and CD14+IRF8+ monocytes, thereby favoring tumor growth. Understanding the regulation of HIF-1α is crucial as it influences the mitochondrial activity of these cells and the immune response.

The objective was to evaluate HIF-1α levels and mitochondrial activity in T lymphocytes and monocytes with exhaustion markers in patients diagnosed with breast cancer and healthy subjects. HIF-1α levels and mitochondrial mass were assessed in CD8+PD-1+ T lymphocytes and CD14+IRF8+ monocytes from peripheral blood of patients (n=13) and healthy subjects (n=13), and metabolic activity and reactive oxygen species (ROS) production were evaluated by flow cytometry.

An increase in CD8+PD1+ T lymphocytes from cancer patients stimulated with αCD3/αCD28 was observed; meanwhile, the exposure of LPS and CoCl2 showed an increase in the mean fluorescence intensity (MFI) of HIF-1α in CD14+IRF8+ monocytes. Under hypoxic conditions with CoCl2, metabolic activity and ROS levels increased in mononuclear cells from breast cancer patients. A trend was found for an increased population of HIF-1α-expressing monocytes from patients with different tumor stages.

This study shows that HIF-1α is stabilized in CD8+PD-1+ T lymphocytes and CD14+IRF8+ monocytes from breast cancer patients through oxygen-dependent and oxygen-independent mechanisms. This stabilization of HIF-1α is associated with poor prognosis tumors and mitochondrial defects, suggesting HIF-1α inhibition as a promising therapeutic strategy for breast cancer.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Production and isolation of porcine monoclonal antibodies from single B cells

Cordero-Ortiz, Maritza Alejandra; Melgoza-González, Edgar
Alonso; Vázquez-García, Verónica Alejandra; Hernández, Jesús.

[1]Centro de Investigación en Alimentación y Desarrollo. Nutrición. Hermosillo, Sonora, México.

E-mail: mcordero223@estudiantes.ciad.mx

TRABAJO NO PRESENTADO

Exploring new monoclonal antibodies is crucial for applications such as immunotherapy or diagnostics, particularly in recognizing viruses with pandemic potential, like the avian influenza virus (AIV) H5N1. An important step in the antibody discovery process is selecting an appropriate animal model. Several species have been used for monoclonal antibody development, but pigs are uncommon in this area. In the context of influenza, swine is not only considered a mixing vessel for influenza pandemic viruses but also shares immunological similarities with humans. Because of this, the objective of this work was to discover and produce antibodies from single B cells of pigs immunized with the hemagglutinin (H5) of the AIV H5N1. Pigs were immunized three times with the H5 protein, and blood samples were collected to evaluate antibody response by indirect ELISA. One week after the last dose, H5-specific B cells (CD8-CD172-CD3-IgG+H5+) were selected, and single-cell sorting was performed by FACS. Cells were captured, immediately frozen, and kept overnight. Then, cDNA was obtained from each cell using a reverse transcription reaction, and nested PCRs were performed to amplify the variable region of heavy (VH) and light (VL) chains. PCR products were sequenced to produce synthetic genes of the VH and VL or cloned in-frame in a vector containing the constant region. The VH and VL genes were co-transfected to express antibodies in an Expi293T system, purified by protein G chromatography, and evaluated by ELISA. The results showed a robust antibody response against H5 in the pig's serum. After sorting, 86 single B lymphocytes were obtained and 15 paired VH and VL chains were successfully amplified, some with kappa (n=9) or lambda (n=6) light chains. So far, one antibody has been produced in the Expi293T system, and another has been successfully cloned into specific vectors for the lambda and heavy chains.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Role of CCL28 in the development of colorectal cancer associated with colitis.

Correa Becerril, Diego Antonio [1]; Cabellos-Avelar, Tecilli [1];
Rodríguez Sosa, Miriam [1]; Pérez-López, Araceli [1].

[1]Facultad de Estudios Superiores Iztacala, UNAM, Unidad de Investigación en Biomedicina (UBIMED), Ciudad de México, México.

E-mail: diegocorrea.unam@gmail.com

Colorectal cancer (CRC) is a malignant neoplasm arising from the epithelium of the colon or rectum, within a microenvironment where tumor cells, immune cells, and soluble factors interact. Among these, the chemokine CCL28 is constitutively expressed in the intestinal mucosa and upregulated in inflammatory conditions such as ulcerative colitis and breast cancer. Its role in CRC remains controversial: while some studies report higher expression in healthy colon tissue, others suggest tumor-derived CCL28 recruits immunosuppressive cells.This study aimed to evaluate CCL28 production during colitis-associated CRC progression in a murine model induced by azoxymethane (AOM) and dextran sodium sulfate (DSS), and to assess its impact on immune cell recruitment and the expression of its receptors CCR3 and CCR10. Male and female C57BL/6 mice received intraperitoneal AOM (10 mg/kg, day 0) and DSS cycles (2%–1.5%) in drinking water for 11 weeks, with two-week rest periods between cycles. At weeks 5 (cycle 2), 8 (cycle 3), and 11 (cycle 4), fecal samples were collected to quantify IgA and IgG levels by ELISA. Colon tissue was analyzed for CCL28 production by immunofluorescence, while colon and blood samples were used to evaluate CCR3 and CCR10 expression in hematopoietic and epithelial cells. Results showed increased recruitment of neutrophils and B cells to the tumor, associated with elevated fecal IgA and IgG. Reduced CCR3 and CCR10 expression in epithelial and B cells suggested dysregulation of CCL28 signaling. Immunofluorescence confirmed decreased CCL28 expression in tumor tissue compared to adjacent and healthy colon. These findings suggest a role for CCL28 in shaping the inflammatory microenvironment of CRC through interactions with B cells, neutrophils, and epithelial cells.Funding: PAPIIT IA208222 and IV00425.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

nAChR-α7 in Nile tilapia leukocytes exposed to diazinon and Aeromonas hydrophila

Covantes-Rosales, Carlos Eduardo [2]; Anseno-Franco, Rebeca [1];
Toledo-Ibarra, Gladys Alejandra [2]; Díaz-Reséndiz, Karina Janice Guadalupe [2]; Barcelos-García, Rocío Guadalupe [2];
Girón-Pérez, Daniel Alberto [2]; Mercado-Salgado, Ulises [2];
Girón-Pérez, Manuel Iván [2].

[1]Universidad Autónoma de Occidente, Unidad Regional Los Mochis. Departamento Académico de Ciencias de la Salud. Los Mochis, Sinaloa, México. [2]El Laboratorio Nacional para la Investigación en Inocuidad Alimentaria (LANIIA)-Nayarit, Secretaría de Investigación y Posgrado, Universidad Autónoma de Nayarit, Tepic, Nayarit, México.

E-mail: Carlos.covantes@uan.edu.mx

Leukocytes possess a non-neuronal cholinergic system, meaning they can synthesize acetylcholine (ACh) de novo and respond to it. They also express enzymes for ACh degradation, such as acetylcholinesterase (AChE), as well as cholinergic receptors, including both muscarinic (mAChR) and nicotinic (nAChR) types. ACh plays a key role in regulating immune responses. However, this response capacity can be altered by exposure to organophosphate (OP) pesticides. One of them is diazinon (DZN), a potent AChE inhibitor that leads to increased ACh concentration and overstimulation of cholinergic receptors. Thus, the cholinergic system in leukocytes could be a molecular target of OPs and be involved in immunotoxicity. This immunological phenomenon may impair the immune response to bacterial antigens. Moreover, inflammatory pathophysiological processes, such as bacterial infection, also modulate the non-neuronal cholinergic system in leukocytes. Therefore, this study aimed to evaluate the presence of the nicotinic acetylcholine receptor α7 (nAChR-α7) in leukocytes of Nile tilapia fish (Oreochromis niloticus) exposed in vivo to DZN and challenged with Aeromonas hydrophila antigens. To this end, Nile tilapia were exposed in vivo to DZN and subsequently challenged with A. hydrophila antigens. Leukocytes from different treatment groups were isolated, cultured with AF-488-conjugated bungarotoxin (1 µg/mL), and incubated for 1 hour (28°C, 5% CO_2). After incubation, cells were analyzed by flow cytometry. The results showed the presence of nAChR-α7 in leukocytes. However, no differences in receptor expression were observed between DZN-treated groups at the tested concentrations, nor in the presence of A. hydrophila antigens. Nevertheless, higher nAChR-α7 expression was detected in polymorphonuclear cells compared to mononuclear cells.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Characterization of Peripheral Blood Leukocytes in the Mexican Elderly Population

Cristerna Ortega, Ángel Andrés [1]; Juárez Rivera, Zafiro Belén [1];
Tapia Sánchez, Wilfrido David [3]; Rosales García, Víctor Hugo [2 3];
Santos Argumedo, Leopoldo [1 4].

[1]Centro de Investigación y de Estudios Avanzados del Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, Departamento de Biomedicina Molecular, Cd. México, México. [2]Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, Unidad de Citometría, Laboratorio Nacional de Servicios Experimentales, Cd. México, México. [3]Diagnóstico Molecular de Leucemias y Terapia Celular, Cd. México, México. [4]Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, Centro de Investigación sobre el Envejecimiento, Cd. México Sede Sur, México.

E-mail: angel.cristerna@cinvestav.mx

In Mexico, adults over 60 constitute a significant and growing demographic, with approximately 15 million individuals. Aging is a complex process involving progressive functional decline, increasing vulnerability to disease and mortality. One key consequence is immunosenescence, a gradual deterioration of the immune system characterized by altered composition, reduced function of immune cells, and impaired responses.

In this study, we performed immune profiling of elderly Mexicans (60–70 years old) compared to younger adults (20–30 years old), recruiting a cohort of both sexes residing in the metropolitan area. We analyzed innate and adaptive immune cell populations to identify age-associated changes.

Major alterations were observed within the adaptive compartment. Total lymphocyte counts declined significantly with age, mainly within naïve populations. Naïve T cells, especially $CD4^+$ subsets, decreased drastically, while memory T cells increased, though statistical significance was reached only for memory $CD8^+$ T cells. Although the increase in regulatory T cells was not statistically significant, a trend toward higher levels was observed in the elderly group. B cell analysis revealed a decrease in naïve B cell numbers while maintaining the same proportions, whereas memory B cells declined in both number and percentage. Among innate subsets, monocyte counts decreased significantly with age. Although neutrophil numbers also declined, this change was not statistically significant, resulting in a slight increase in the neutrophil-to-lymphocyte ratio.

Our results highlight a marked reduction in naïve T and B cells, as well as a shift toward memory phenotypes for T cells. These changes may underlie diminished immune adaptability and contribute to increased susceptibility to infections in the elderly. These findings provide a detailed characterization of age-related immune alterations in older Mexicans. Further research is needed to elucidate the functional consequences of these changes, particularly how the decline of naïve populations impacts immune competence and vaccine responsiveness.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Immunoepigenetic and Molecular Impact of PAHs in B-Cell Acute Lymphoblastic Leukemia

Cruz-Quintá, Alfredo; Layseca-Espinosa, Esther; Martínez-Chío,
Elena Berenice.

[1]Centro de Investigación en Ciencias de la Salud y Biomedicina,
Universidad Autónoma de San Luis Potosí, México. [2]Facultad de
Medicina, Universidad Autónoma de San Luis Potosí, México.

E-mail: freddycq27@gmail.com

Polycyclic aromatic hydrocarbons (PAHs) are persistent environmental pollutants with recognized toxic effects, including DNA adduct formation, gene mutations, chromosomal abnormalities, and epigenetic modifications. Population-based studies have associated the presence of PAHs in household dust with an increased risk of B-cell acute lymphoblastic leukemia (B-ALL) in pediatric populations. Additionally, a PAH-triggered signaling pathway involving indenopyrene—one of the most abundant PAHs—has been described, which sustains transformation and proliferation in leukemic cell cultures.

The objective of this study is to investigate the association between PAH exposure and the presence of increased molecular and epigenetic alterations in pediatric patients with precursor B-cell acute lymphoblastic leukemia (B-ALL), compared to patients with no documented exposure. Furthermore, the study aims to evaluate the impact of PAH exposure on chemotherapy response.

The study includes patients aged 0 to 18 years with newly diagnosed B-ALL, treated at the Oncohematology Department of Hospital Central "Dr. Ignacio Morones Prieto." Patients already under treatment or surveillance are excluded. To date, 10 out of a projected 40 patients have been enrolled. Paired bone marrow and urine samples were collected. Detection of 1-hydroxypyrene, a biomarker of PAH exposure, is currently underway by high-performance liquid chromatography (HPLC), which will enable classification into exposed and non-exposed groups.

Bone marrow samples underwent flow cytometry immunophenotyping and epigenetic histone modification profiling (H3K4me3, H3K27me3, H3K27ac, H3K9ac). Additionally, common B-ALL-associated molecular translocations (BCR-ABL p190, TEL-AML1, TCF3-PBX1, and MLL-AF4) were evaluated via PCR. A second bone marrow sample is pending for minimal residual disease (MRD) assessment, to further analyze the influence of PAH exposure on therapeutic response.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Evaluation of Methods for Quantifying Cell Viability: Spectrophotometry, Microscopy, and Flow Cytometry

Cubillo-Cervantes, Danna [1]; Guzmán-García, Luz María [1 2];
Reyes-Pérez, Itzel Viridiana [2];
Sánchez-Hernández, Pedro Ernesto [1 4].

[1]Universidad de Guadalajara, Centro Universitario de Ciencias de la Salud (CUCS), Instituto de Investigación en Cáncer en la Infancia y Adolescencia, Departamento de Clínicas de la Reproducción Humana, Crecimiento y Desarrollo Infantil. Guadalajara, Jal. México. [2]Universidad de Guadalajara, CUCS. Doctorado en Ciencias Biomédicas, Departamento de Fisiología. Guadalajara, Jal. México. [3]Universidad de Guadalajara, CUCS, Departamento de Disciplinas Filosófico, Metodológicas e Instrumentales. [4]Universidad de Guadalajara, CUCS, Departamento de Fisiología. Guadalajara, Jal. México.

E-mail: danna.cubillo5276@alumnos.udg.mx

Cell viability assessment is essential in biomedical research and the development of antitumor drugs. Traditional methods such as trypan blue (TB) exclusion and the MTT assay have limitations in precision and scalability. Manual TB counting can be inconsistent, and MTT results may be influenced by optical interference or toxic solvents. Flow cytometry, using dyes like Zombie NIR or propidium iodide (PI), offers improved accuracy and high-throughput capabilities. The use of TB in flow cytometry is proposed as a low-cost alternative, maintaining the principle of plasma membrane integrity to distinguish viable from non-viable cells.

We evaluated three methods for cell viability quantification: microscopy with TB, spectrophotometry with MTT, and flow cytometry using Zombie NIR and TB. CEM cells (a T-cell leukemia line) were cultured in RPMI-1640 with 10% fetal bovine serum and 1% antibiotic-antimycotic, then treated with etoposide (6.2, 12.4, and 25 µg/mL) for 24 h or 3 h. TB and MTT assays were performed in 96-well plates with 20,000 cells/well, while flow cytometry used 24-well plates with 200,000 cells/well. MTT absorbance was measured at 570 nm. TB microscopy involved staining with 0.4% TB and manual counting using a Neubauer chamber. Zombie NIR and TB staining for flow cytometry included washing, staining, and acquisition on a cytometer using appropriate lasers and detectors. PI was used as a reference.

Results showed that traditional methods, although accessible, have limitations in accuracy and sample handling. Flow cytometry provided greater sensitivity and precision. TB, while cost-effective, required optimization of concentration and exposure time for cytometric analysis, but enabled the evaluation of a large number of samples. This comparative study highlights the importance of selecting and, when possible, combining appropriate methods for reliable and reproducible cell viability evaluation, based on study design, available resources, and sample number and characteristics.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Resistance mechanisms to immunotherapy in her2+ breast cancer: systematic review and meta-analysis resistance

De la Rosa-Salazar, Karina Daneyka [1]; Rojas-Gutiérrez, Santiago Emilio [1]; Salinas -Carmona, Mario Cesar [1]; Macias-Segura, Noé [1].

[1]Facultad de medicina UANL, Departamento de inmunología, Monterrey, Nuevo León, México.

E-mail: karina.delarosaslz@uanl.edu.mx

HER2-positive breast cancer comprises 20–25% of breast cancer cases and is associated with poor prognosis, although, thanks to the development of anti-HER2 therapies like trastuzumab, pertuzumab, T-DM1, and HER2-targeted tyrosine kinase inhibitors there have been significant improves in the outcome of many cases. However intrinsic and acquired resistance mechanisms still limit long-term treatment success. That's why is critical understanding the molecular basis and identifying predictive biomarkers to optimize therapy and treatment strategies.

We performed a systematic review and meta-analysis based on PubMed, EMBASE, and Scopus studies between the years 2015 to 2025 evaluating molecular resistance mechanisms to anti-HER2 therapies in women with HER2+ breast cancer. Eligible studies included clinical cohorts, translational analyses, and preclinical models comparing responders versus non-responders. Involving 68 studies that gather approximately 10,000 patients.

The present work identified key mechanisms of resistance to HER2-targeted therapies in HER2-positive breast cancer. These include loss or truncation of HER2 (e.g., p95HER2), activating mutations in HER2, and downstream alterations such as PIK3CA mutations or PTEN loss that sustain PI3K/AKT or MAPK signaling. Resistance is also promoted by compensatory pathways (HER3, IGF-1R, MET), immune evasion via HLA-G or MUC4 overexpression, and impaired internalization or trafficking of antibody-drug conjugates. These mechanisms highlight the complexity of resistance and the need for combination strategies.

The present work underscores the multifactorial nature of resistance to HER2-targeted therapies in HER2-positive breast cancer, involving both tumor-intrinsic and immune-related mechanisms. Understanding these pathways is essential for developing more effective treatment strategies, including combination therapies and novel agents aimed at overcoming resistance and improving patient outcomes.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Composition of Immune Niches Present in Tumor Microenvironment Associate with Disease Control or Progression in Melanoma

De León-Rodríguez, Saraí G. [1 2]. ; Aguilar-Flores, Cristina [1] ;
Sánchez-Contreras, Jair [3] ; Mantilla, Alejandra [4] ; Bonifaz, Laura [5]

[1]Unidad de Investigación Médica en Inmunología (UIMI). UMAE. Hospital de Pediatría. CMN SXXI.. [2]Posgrado en Ciencias Biológicas, Facultad de Medicina, Universidad Nacional Autónoma de México, Mexico City, México. [3]Leibniz-Institut fuer Analytische Wissenschaften, Alemania. [4]Servicio de Patología, Hospital de Oncología Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de Mexico, Mexico. [5]Coordinación de investigación en salud, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de Mexico, Mexico.

E-mail: sara.g.deleonr@gmail.com

Melanoma is a lethal skin cancer with low survival rates in advanced stages. Moreover, GLOBOCAN estimates that its incidence and mortality will continue to rise. This tumor has been a key model for advancing our comprehension of the interplay between cancer and the immune system. Recently, spatial techniques and analyses have revealed that immune cell organization within tumors plays a crucial role in disease progression across various cancer types. Understanding the tumor immune microenvironment (TIME) of melanoma could be crucial for offering prognosis and therapy selection. This study characterized immune clusters in melanoma at diagnosis and their association with disease control or progression. We analyzed multiplexed immunofluorescence whole-slide scans of melanoma biopsies using AI-driven image analysis and unsupervised clustering. Our results identified distinct immune cluster compositions linked to disease state. At diagnosis, patients who later achieved disease control exhibited immune clusters enriched in CD8+ T cells, dendritic cells (DCs), NK cells, CD4+ T cells, and mast cells. In contrast, metastatic patients showed macrophage-enriched along the tissue and immune clusters dominated by CD4+ T and CD66b+ cells, with fewer CD8+ T, DCs, and NK cells. These distinct compositions distinguished metastatic patients from those with disease control. These findings suggest that immune cluster composition at diagnosis is related to long-term disease control or metastasis. Understanding immune cell spatial organization and clustering may serve as a novel biomarker for patient stratification and prognosis.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Isolation and Characterization of a Neutralizing VNAR Fragment Against PD-L1 in cancer

Del Aguila -Velasco, Keily Naomi [1]; Licea-Navarro, Alexei
Fedorovish [2] [3]; Cabanillas-Bernal, Olivia [3].

[1]CICESE, Posgrado en Ciencias de la Vida, Ensenada, Baja California, México. [2]CICESE, Departamento de Innovación Biomédica (DIB), Ensenada, Baja California, México. [3]CICESE, Dirección de Impulso a la Innovación y el Desarrollo (DIID), Ensenada, Baja California, México.

E-mail: keily@cicese.edu.mx

Programmed death-ligand 1 (PD-L1) is an immune checkpoint protein expressed in various tumors, where it suppresses the antitumor immune response through interaction with PD-1. Blocking this pathway has demonstrated clinical success in several cancer types. However, conventional monoclonal antibodies present challenges such as high production costs, limited tumor penetration, and potential immunogenicity. To address these limitations, this project aims to develop an alternative immunotherapeutic tool based on variable new antigen receptor (vNAR) fragments derived from Heterodontus francisci shark. We used a synthetic phage display library to select vNARs that specifically bind to human PD-L1. After several rounds of biopanning, enriched phage clones were screened by ELISA to confirm binding activity. The most promising candidates will be evaluated in cancer cell lines to determine their ability to recognize human PD-L1 on the cell surface. These assays will represent the first step in validating their potential as neutralizing molecules. Due to their small size, high stability, low immunogenicity, the ability to bind hidden or recessed epitopes, and the rapid selection process from synthetic libraries, vNARs represent a valuable alternative to conventional antibodies. This strategy not only facilitates the development of targeted Immunotherapeutics with better tumor accessibility but also accelerates preclinical research and the discovery of novel checkpoint inhibitors. Overall, this work contributes to the advancement of biological tools for more effective and accessible cancer immunotherapy.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Oxidative stress and Nrf2 activation under an exercise protocol in women recovered from breast cancer.

Delgado-Benedey, David Humberto [1 2]; Quintana-Mendias, Estefanía [1]; Enríquez-Del castillo, Liliana Aracely [1]; Flores-Olivares, Luis Alberto [1]; Cervantes-Hernández, Natanael [1].

[1]Universidad Autónoma de Chihuahua, Facultad de Ciencias de la Cultura Física, Chihuahua, Chihuahua, México. . [2]Universidad Autónoma de Chihuahua, Facultad de Enfermería y Nutrición, Chihuahua, Chihuahua, México.

E-mail: david.delbenedey@gmail.com

Various breast cancer (BC) treatments have increased survival in recent decades, however, BC survivors may present high levels of oxidative stress (OS) due to chemotherapy or radiotherapy and this can last for years. OS is one of the main causes of cancer and the risk of recurrence, it is defined as a relative excess of reactive oxygen species (ROS) compared to antioxidants, therefore, redox homeostasis is crucial. Both aerobic and anaerobic exercise have been found to be key instruments in its decrease through the activation of nuclear factor erythroid-derived 2 (Nrf2). Nrf2 is a transcription factor found in the cytosol and functions as a key regulator of the antioxidant response to a variety of cellular stressors. This factor enters the cell nucleus and binds to an antioxidant response element (ARE), which is an enhancer sequence found in the promoter region of numerous antioxidant genes, and due to this binding, antioxidant proteins are produced. General physical activity guidelines and guidelines for BC survivors recommend concurrent exercise, which is defined as the combination of aerobic and anaerobic exercise. This suggests a different perspective for reducing oxidative stress in BC survivors. However, few studies utilize and analyze this type of exercise and its relationship with Nrf2 activation and the resulting antioxidant response. Therefore, the present study aims to evaluate the effect of concurrent exercise on oxidative stress markers and antioxidant enzymes through Nrf2 activation in women who have recovered from breast cancer.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

TGF-β3 Induces Oral Tolerance by Imprinting Gut-Homing Capacity in Regulatory T cells

Delgado-Zaldívar, Diego [1,2]; Tapia-Maltos, Marco [2,3]; Albarrán-Godínez, Adrián [1,2]; Ramírez-Vélez, Claudia [2]; De la Cruz-Rico, Abigail Estefanía; Monterrosa-Morales, Laura [2,4]; Cortez-Quezada, Diego [5]; Romero-Rodríguez, Dámaris Priscila [6]; Madera-Salcedo, Iris Karina [2]; Rosetti-Sciutto, Florencia [2]; Crispín-Acuña, José Carlos [2,4].

[1]Doctorado en Ciencias Biomédicas, Universidad Nacional Autónoma de México (UNAM). Ciudad de México. México. [2]Departamento de Inmunología y Reumatología, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán (INCMNSZ). Ciudad de México. México. [3]Plan de Estudios Combinados en Medicina (PECEM), Facultad de Medicina, UNAM, Ciudad de México. México. [4]Escuela de Medicina y Ciencias de la Salud, Tecnológico de Monterrey. Ciudad de México. México. [5]Laboratorio Nacional CONAHCYT de Investigación y Diagnóstico por Inmunocitofluorometría (LANCIDI), Instituto Nacional de Enfermedades Respiratorias (INER). Ciudad de México. México. [6]Centro de Ciencias Genómicas, UNAM, Cuernavaca. Meéxico.

E-mail: diegochpt@gmail.com

Immune responses against ingested proteins are avoided by a poorly understood mechanism known as oral tolerance (OT). In a previous study, we demonstrated that OT depends on TGF-β3 but occurs in the absence of TGF-β1. Here, we sought the cellular source of TFG-β3 during OT and analyzed the mechanism through which it induces the process. We used conditional knockout mice (cKO) to identify the cellular lineages that produce TGF-β3 during OT. We used ovalbumin (OVA) as a model antigen and OT-II FoxP3GFP cells to analyze Treg generation. OT was tested in a model of delayed-type hypersensitivity and anti-OVA antibodies were quantified. Conditional deletion of Tgfb3 in all cell lineages (Tamoxifen-Cre) abolished OT and enhanced anti-OVA antibody production. Deletion of Tgfb3 in hematopoietic cells (iVav-Cre) significantly impaired OT. Deletion of Tgfb3 in CD11c+, TCR-ɣδ+ (CD4-Cre), and TCR-ɣδ+ cells had no effect. Transcriptomic comparison of CD4 cells activated with TGF-β1 vs. TGF-β3 revealed that TGF-β3 promotes the expression of gut-homing molecules. This was confirmed using adoptively transferred OT-II cells, as OVA-specific Tregs were significantly less abundant in the small intestine lamina propria (siLP) in Tgfb3 cKO mice and expressed lower levels of CD103 and α4β7 integrin. Accordingly, during steady state, lack of TGF-β3 decreased the abundance of siLP Tregs and their expression levels of FoxP3, CD103, and α4β7 integrin. In conclusion, TGF-β3 produced mainly by hematopoietic cells is essential for oral tolerance. This cytokine imprints Tregs with an adhesion molecule expression profile necessary for their correct functionality in the gut.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e Inmunoterapia**

Anti-PD-1 therapy enhances progenitor-like and intermediate exhausted T cells in chronic leishmaniasis.

Mariana Diupotex [1 2] , Zamora-Chimal [1], Ingeborg Becker [1]

[1] Unidad de Investigación en Medicina Experimental, Facultad de Medicina, Universidad Nacional Autónoma de México, Ciudad de México, México. [2] Instituto de Biotecnología, Universidad Nacional Autónoma de México, Cuernavaca, Morelos, Mexico.

E-mail: diupotex@ciencias.unam.mx

Diffuse cutaneous leishmaniasis (DCL), caused by *Leishmania mexicana*, is a chronic, treatment-refractory form of leishmaniasis characterized by multiple nodular lesions and high parasite burden. In this context, T cells progressively acquire an exhausted phenotype, marked by high expression of inhibitory receptors such as programmed death 1 (PD-1) and impaired effector functions. Exhausted T (Tex) cells are heterogeneous and included progenitor-like (CXCR5+), intermediate (CXCR5+TIM-3+), and terminal differentiated (TIM-3+) subsets, which differ in proliferative and functional capacity. Their role in response to PD-1 checkpoint blockade in chronic cutaneous leishmaniasis (CL) remains unclear. Here, we show that treatment with a variable-dose anti-PD-1 regimen effectively reduced lesion progression and parasite load in footpad lesions and draining lymph nodes of C57BL/6 mice chronically infected with *L. mexicana*. This therapy enhanced Th1-protective immunity upon antigen recall and promoted T-cell reinvigoration in lymph nodes, as indicated by increased CD69, Ki-67, IFN-g, and granzyme B expression. Confocal microscopy of lesion sites further confirmed the enrichment of IFN-g _and TNF-a _coproducing T cells following immunotherapy. Importantly, we demonstrate that PD-1/PD-L1 blockade selectively enhances defined Tex subsets. We found a marked increase of PD-1+CXCR5+ and PD-1+TIM-3+ cells within lesions. An in-depth analysis of lymph node cells revealed preferential expansion of progenitor-like CXCR5+ Tex subsets in both CD4+ and CD8+ T-cell compartments, and of intermediate CXCR5+TIM-3+ Tex subsets within CD8+ T cells, suggesting that these exhausted subtypes mediate the immune control of CL. These findings provide novel evidence that PD-1/PD-L1 blockade promotes the expansion of progenitor and intermediate Tex subsets during chronic infection with *L. mexicana*. Our results underscore the therapeutic potential of PD-1/PD-L1 immune checkpoint inhibition in patients with DCL and support the design of novel immunotherapies that target responsive exhausted T-cell populations. This project was supported by: SECIHTI 6682, UNAM-PAPIIT IG2000924, and PAFRIM FM/DI/150/2024.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Antioxidant effect of grape pomace on murine macrophages

Domínguez-Gómez, Cuauhtli David [1]; Córdova-Dávalos, Laura E [1]; Salinas-Miralles, Eva [1]; Maldonado-Gonzaga, Andrea Yaxiri [1]; Casillas-Peñuelas, Rafael [2]; Cervantes-García, Daniel [1 3]; Jiménez-Vargas, Mariela [1].

[1]Department of Microbiology, Center for Basic Sciences, Autonomous University of Aguascalientes, Aguascalientes, Mexico. [2]Department Food Sciences, Center for Agricultural Sciences, Autonomous University of Aguascalientes, Aguascalientes, Mexico. [3]Secretariat of Science, Humanities, Technologies and Innovation, Mexico City, Mexico.

E-mail: cuauddg@gmail.com

There is a growing interest in identifying new sources of natural antioxidant compounds. Grape pomace is a solid by-product generated during the winemaking process consisting of peel, seed, stem, and pulp, which has potential applications for health. Macrophages are key immune cells in the regulation of inflammation through the oxidative burst, and alterations in their responses are linked to multiple pathologies. The aim of this study was to evaluate whether methanolic extracts (MEs) from grape pomace exhibit antioxidant activity on murine macrophages. Samples of pomace from Merlot grape, both "old world" and "new world" types, grown in two different vineyards in Aguascalientes (named A and B) were collected to obtain MEs. Antioxidant activity of MEs was determined using DPPH-Trolox reaction and phenolic compounds by the Folin-Ciocalteu reaction. The cytotoxicity of MEs was evaluated through the MTT assay. To analyze their effect on macrophages activation, RAW937 cells were pretreated with the MEs and subsequently activated with LPS to quantify nitric oxide (NO) production by Griess reaction. The results obtained show that all the MEs had antioxidant activity quantified by percentage of radical scavenging activity, ranging from 81.03% to 83.10%. Phenolic compounds were detected in all ME samples, with values from 1.4 to 2.1µg/mL. The MEs were not toxic at concentration from 0.005 to 0.5µg/mL. Only MEs from grapes grown in vineyard A significantly decreased 32.25% (old world) and 24.38% (new world) NO production when used at 0.05µg/mL of phenols. Samples of RNA were extracted from RAW937 cells treated with the ME that better reduced NO production, and the expression of genes related to antioxidants and anti-inflammatory pathways is being evaluated. In conclusion, MEs obtained from Merlot grape pomace presents antioxidant properties, which might depend on the soil type and agro-climatic conditions of the vineyard and not only on the grape variety.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Evaluation of the Cytotoxic Effect of Anticancer-Potential Nanopolymers on Human Peripheral Blood Mononuclear Cells

Donis -Maturano, Luis [1]; Olivas Sarabia, Amelia [2];
Sánchez-Martínez, Mayra Alejandra [1]; Arroyo-Sahagún, Eurydice
Carolina [3 4]; González-Ayala, Mauricio Enrique [1];
Espino-Solís, Gerardo Pavel [5].

[1]Facultad de Estudios Superiores Iztacala-UNAM, Tlalnepantla de Baz, Estado de México, México. [2]Centro de Nanociencias y Nanotecnología-UNAM, Ensenada, B.C., México. [3]Facultad de Ciencias Químicas e Ingeniería -UABC, Tijuana, B. C., México. [4]Instituto Tecnológico de Ensenada–TecNM, Ensenada, B.C., México. [5]Facultad de Medicina, UACH, Chihuahua, México.

E-mail: ludoma6@hotmail.com

Cancer remains one of the leading causes of mortality worldwide, which has driven significant efforts toward the development of novel treatments capable of preventing and combating this disease. Among these emerging strategies, the use of biodegradable nanoparticles as delivery systems for anticancer agents has gained increasing attention. These nanocarriers offer several advantages over conventional treatments, such as targeted and controlled drug release, enhanced bioavailability, and the potential to reduce adverse effects associated with chemotherapy and radiotherapy. Despite these benefits, it is crucial to evaluate their potential cytotoxicity on healthy, non-cancerous cells prior to clinical application, in order to ensure their safety and efficacy.

In this study, we assessed the cytotoxic effect of three experimental nanopolymers [in which Imiquimod was encapsulated within Polypropyleneglycol (P1), Polyethyleneglycol (P2), and Polyvinylpyrrolidone (P3)]. Each of these nanopolymer formulations, with their potential antitumor activity, was tested on human peripheral blood mononuclear cells (PBMCs) from healthy donors. The cells were exposed to concentrations of 1 µg/mL and 10 µg/mL for 24 and 48 hours, and cell viability was determined via flow cytometry. The results show that at 1 µg/mL, none of the nanopolymers significantly affected cell viability after 24 hours. However, nanopolymer P3 exhibited a significant cytotoxic effect at 48 hours of exposure. At the higher concentration of 10 µg/mL, a non-significant trend towards reduced viability was observed with nanopolymer P2 at 24 hours, while all three nanopolymers induced significant cell death at 48 hours of exposure.

These findings suggest that a lower concentration of 1 µg/mL may be a safer therapeutic window for the application of these nanopolymers in healthy human cells. Overall, this study contributes to the preclinical safety assessment of experimental anticancer nanomaterials and highlights the importance of dose optimization to minimize undesirable cytotoxic effects.

Área del artículo: **Inmunología de enfermedades infecciosas**

Dermaseptin DMS-DA6: a promising alternative to antibiotics for N. brasiliensis actinomycetoma

Duarte-Mata, Diana Ivonne [1]; Vázquez-Marmolejo, Anna Velia [1];
Alemán-Navarro, Estefanía [2]; Jiménez-Reyna, Antonio [1];
Sosa-Hernández, Paola Estefanía [1]; Rosenstein-, Yvonne [2];
Auvynet-, Constance [2]; Salinas-Carmona, Mario Cesar [1].

[1]Universidad Autónoma de Nuevo León, Departamento de Inmunología, Monterrey, Nuevo León, México.. [2]Universidad Nacional Autónoma de México, Instituto de Biotecnología, Cuernavaca, Morelos, México.

E-mail: dianaduarte23@hotmail.com

Actinomycetoma is a chronic infectious disease recognized by the World Health Organization as a neglected tropical disease. In the Americas, the most common etiologic agent is the Gram-positive bacterium, Nocardia brasiliensis. It is a facultative intracellular pathogen that can multiply and survive within macrophages, evading microbicidal mechanisms by inducing an immunosuppressive environment. Current antibiotic treatments are expensive, prolonged, and toxic, and bacterial resistance has been reported. Host defense peptides, known for their bactericidal and immunomodulatory effects, and their ability to induce poor bacterial resistance due to their direct effect on the bacterial membrane, represent a novel therapeutic approach. Recently, DMS-DA6, a dermaseptin isolated from the Mexican tree frog, Pachymedusa dacnicolor, has been shown to exert strong activity against Gram-positive bacteria, including multidrug-resistant strains. Our study evaluated the effects of DMS-DA6 in a N. brasiliensis-induced actinomycetoma mouse model in comparison with the ones observed with the conventional antibiotic linezolid. Infected mice were treated either twice a week with this peptide at a dose of 12.5mg/kg or every 12 hours with linezolid at a dose of 25 mg/kg over a three-week period. Our findings suggest that treatment with DMS-DA6 is more effective in resolving the disease than linezolid, as it induces a similar reduction in the volume of inflammation and bacterial load in the infected area at a lower dose and with fewer injections. These findings highlight the potential of DMS-DA6 as an innovative addition to current therapy.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Lyn Kinase as a Key Regulator in Replicative Senescence of Mast Cells

Esparza -Reyes, Diana Laura [1]; Ibarra -Sánchez, Alfredo [1];
Madera -Salcedo, Iris [3]; Gonzáles -Espinosa, Claudia [1 2].

[1]Departamento de Farmacobiología Cinvestav, Unidad Sur. CDMX. México . [2]Centro de Investigación en Envejecimiento. Cinvestav, Unidad Sur. CDMX. México. [3]Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán. CDMX. México.

E-mail: d.l.esparza76@gmail.com

Mast cells (MCs), key components of the innate immune system, perform primary effector functions by producing proinflammatory cytokines, histamine, and leukotrienes. These processes are regulated by tyrosine kinases such as Lyn. Cellular senescence is characterized by permanent cell cycle arrest mediated by the p21 and p16 proteins. Senescence in immune cells has been linked to chronic inflammation and disease development, but the molecular mechanisms behind MC senescence and its impact on MC-mediated responses remain uncharacterized. In this study, bone marrow-derived mast cells (BMMCs) from young and aged WT and Lyn KO mice were used to investigate the role of Lyn in MC senescence in vivo. Also, BMMCs were generated from young animals and cultured for 13 weeks to induce replication senescence in vitro. Senescence markers such as p21, along with the expression of specific cytokines associated with the senescence-associated secretory phenotype (SASP), were analyzed.

The two methods used to induce senescence in BMMCs from WT mice result in a similar phenotype, characterized by the expression of p21 and the appearance of a SASP composed by IL-6, 1IL-β, and TNF-α, among others. In contrast, in BMMCs derived from Lyn-/- mice, expression of p21 and cytokine secretion was higher than in WT BMMCs. Our findings are in line with those suggesting that Lyn kinase is a main regulator of MC function and add the characterization of the role of this kinase in MC senescence, suggesting that Lyn kinase is a negative regulator of senescence in MCs.

Supported by Grant Secihti CF3915.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Area del artículo: **Inmunidad tumoral**

Development of an immunotherapy-resistant human melanoma model to establish therapeutic response biomarkers

Esperante, Diego; Gajón, Julián A. [2 3]; Juárez-Flores, Ángel [4];
Fuentes-Panamá, Ezequiel M. [4]; Bonifaz, Laura C. [5].

[1]Plan de Estudios Combinados en Medicina (PECEM), Facultad de Medicina, UNAM, Ciudad de México, México. [2]Posgrado en Ciencias Bioquímicas, Universidad Nacional Autónoma de México, Ciudad de México, México. [3]Unidad de Investigación Médica en Inmunoquímica, Hospital de Especialidades “Dr. Bernardo Sepúlveda”, Centro Médico Nacional “Siglo XXI”, Instituto Mexicano del Seguro Social, Ciudad de México, México. [4]Unidad de Investigación en Virología y Cáncer, Hospital Infantil de México “Federico Gómez”, Ciudad de México, México. [5]Coordinación de Investigación en Salud (CIS), Centro Médico Nacional “Siglo XXI”, Instituto Mexicano del Seguro Social, Ciudad de México, México.

E-mail: diegoesperante523@gmail.com

Immunotherapy has revolutionized the treatment of patients with various types of cancers. Immune checkpoint blockade (ICB), a variant of immunotherapy, has proven particularly efficacious in the context of disseminated melanoma, where it now stands as a standard of care. In spite of its clinical benefit, ICB faces important limitations, one of the more significant being therapeutic resistance, whether primary of acquired (secondary). The mechanisms underlying the development of resistance to immunotherapy, particularly ICB, remain to be elucidated. Our group has previously established murine melanoma lines that are resistant to ICB through in vivo administration of anti-PD-1 monoclonal antibody. The resistant populations exhibited upregulation of stemness-related markers, such as Nanog, Oct-4, NGFR, etc. Stemness and, in particular, cancer stem cells (CSCs) have been proposed as significant barriers for antineoplastic therapy efficacy, including chemo-, radio- and targeted therapy. In this work, a similar approach is employed to generate therapeutic resistance in two human melanoma cells lines, one in situ, the other metastatic. These lines were submitted to immune pressure through co-culture with previously activated PBMCs, followed by treatment with anti-PD-1 antibody. Cells and spheroids isolated from consecutive co-cultures with PBMCs with or without ICB were then characterized: an important increase in the expression of stemness markers, including Wnt3a, Wnt5a, NGFR, Nanog, STAT3, among others, was observed when compared to non-pressed controls, suggesting an enrichment of the stemness profile in the context of ICB resistance. The main goal of this research is to better characterize human lines resistant to immunotherapy and identify biomarkers that allow us to define more precisely a resistant state.

Área del artículo: **Inmunología de enfermedades infecciosas**

Immune Profiling of Pediatric Patients Infected with Rickettsia and Ehrlichia

Manzanera-Ornelas, Diana [2]; Espinoza-Duarte, Mayela [1];
Baquera -Arteaga, Maribel [1,2]; Espino -Solís, Gerardo Pavel [1].

[1]Universidad Autónoma de Chihuahua; Facultad de Medicina y ciencias Biomédicas; Laboratorio de Investigación Traslacional; Chihuahua, Chihuahua, México. [2]Hospital Infantil de Especialidades de Chihuahua; Chihuahua, Chihuahua, México.

E-mail: gespinos@uach.mx

In 2021, 273 cases of Rocky Mountain spotted fever were reported in Mexico. In Chihuahua City, 14 pediatric patients with suspected rickettsial infection were evaluated. Molecular diagnosis revealed infection rates of 28.5 % for Rickettsia rickettsii, 43 % for Ehrlichia canis, and 28.5 % for coinfection. Clinical analysis identified marked alterations: 100 % of patients exhibited elevated hepatic enzymes and prolonged coagulation times; 64 % had leukocytosis with neutrophilia; 55 % presented thrombocytopenia, lymphopenia, and hypoalbuminemia; and 45 % showed normocytic normochromic anemia Immunological profiling revealed significant differences in chemokine expression. Notably, IL-8, RANTES, CXCL9/MIG, and CXCL10/IP-10 levels were altered in coinfected patients compared to controls. IP-10 was significantly elevated in R. rickettsii-infected individuals. Additionally, IL-1β, IL-6, IL-17, IFN-γ, and TNF-α were significantly upregulated in the R. rickettsii-positive group. Coinfected patients exhibited distinct cytokine profiles, including elevated IL-6, IL-8, and IL-10. Flow cytometry analysis further revealed significant differences in $CD8^+$ T cell subpopulations between R. rickettsii and E. canis-infected patients.

These findings suggest that pediatric rickettsial infections, particularly coinfections, induce a complex and differential immunological response, with potential implications for disease severity and prognosis.

Acknowledgments: Mas UACH, Mas Ciencia 2025 and Enrique Guevara.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Innovative Humanized NSG Mouse Platform for Cutting-Edge Immunotechnology and Biotherapeutic Evaluation

Espinoza-Hernández, Jeniffer [1]; Romero-Pérez, Maryfer [1];
López-Olvera, Karina; Valencia-Flores, Luis A. [1]; Pérez-Tapia,
Sonia M. [1 2]; Pedraza-Escalona, Martha [1 2 3].

[1]Unidad de Desarrollo e Investigación en Bioterapéuticos, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, CDMX, México. [2]Laboratorio Nacional para Servicios Especializados de Investigación, Desarrollo e Innovación (I+D+i) para Farmoquímicos y Biotecnológicos, LANSEIDI-FarBiotec-CONAHCyT, CDMX, México. [3]SECIHTI-Unidad de Desarrollo e Investigación en Bioterapéuticos, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, CDMX, México.

E-mail: jenifferespinozah@gmail.com

The development of robust and ethical models for evaluating biotherapeutics is essential to the advancement of immunotechnology. Here, we report the establishment and standardization of a humanized NSG mouse platform using a non-irradiative myeloablation protocol. This approach employs the alkylating agent busulfan to achieve hematopoietic niche clearance, thereby eliminating the need for radiation while maintaining effective myeloablation1. Female and male NSG mice received i.p. busulfan (30mg/kg) to deplete bone marrow niches, followed by intravenous transplantation of either 5×10^5 or 1×10^5 $CD34^+$ hematopoietic stem cells (HSCs) obtained from the bone marrow of healthy human donors (ATCC). To promote immune reconstitution, mice received estradiol (E2) starting at 14 weeks of age, prior to immunization. E2 modulates the hematopoietic and immunity by facilitating HSC differentiation into both lymphoid and myeloid lineages via activation of estrogen receptors ERα and ERβ. Furthermore, E2 upregulates AID and BLIMP-1 expression in B cells, promoting somatic hypermutation (SHM), class switch recombination (CSR), and plasma cell (PC) differentiation.

The engraftment process was monitored through serial peripheral blood sampling. Hematologic parameters confirmed the degree of myeloablation, while flow cytometry enabled longitudinal tracking of human hematopoietic cell engraftment. All critical steps—including conditioning, cell expansion, transplantation, and longitudinal monitoring—were successfully optimized. Notably, human $CD45^+$ leukocytes were detected in at least one implanted NSG mouse, confirming successful engraftment and suggesting the functional reconstitution of a human immune system3. This innovative platform represents a substantial refinement over traditional radiation-based models, reducing procedural complexity and animal stress while preserving efficacy. It enables a wide array of advanced applications, including the production of fully human antibodies, long-term studies human hematopoiesis, human tumor xenografts, infectious disease modeling, and evaluation of novel immunotherapies. Our findings validate this humanized NSG mouse model as a versatile and reliable in vivo system for next-generation immunobiological research.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

Piezo1 expression in reactive astrocytes in a murine model of Alzheimer’s disease

Esteban-Maldonado, Daniel [1]; Gevorkian-, Goar [1];
Acero-Galindo, Gonzalo Asariel [1].

[1]Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México (UNAM), departamento de inmunología, Apartado Postal 70228, Cuidad Universitaria, CDMX, CP 04510, México.

E-mail: danielesteban793@gmail.com

The presence of reactive astrocytes surrounding β-amyloid (Aβ) plaques is a defining characteristic of Alzheimer’s disease. These reactive astrocytes have been reported to release neurotoxic and pro-inflammatory mediators that exacerbate neuronal damage. Although the mechanisms by which astrocytes detect Aβ aggregates and transition into a reactive state remain largely unknown, mechanotransduction has recently emerged as a potential process linking changes in tissue stiffness associated with Aβ plaques to astrocyte reactivity. In this study, we evaluated the expression of the mechanosensitive ion channel Piezo1 in reactive astrocytes using the Tg-SwDI transgenic mouse model, which expresses human amyloid precursor protein (hAPP) carrying familial Alzheimer’s disease-associated mutations and recapitulates a broad spectrum of its neuropathological features. Integrated Fluorescence Density quantification using Regions of Interest (ROIs) in various brain regions of Tg-SwDI mice revealed that GFAP overexpression begins at six months of age, coinciding with the initial accumulation of Aβ. At this stage, astrocytes in the Tg-SwDI mice also exhibit morphological changes consistent with cellular hypertrophy, both key features of astrocyte reactivity. Furthermore, we found that reactive astrocytes surrounding Aβ aggregates in Tg-SwDI mice express the mechanosensitive ion channel Piezo1. In contrast, Piezo1 expression was absent in astrocytes from age-matched wild-type mice and from Tg-SwDI mice at three months of age, prior to the onset of pathology. These findings highlight a close relationship between mechanosensory components and Aβ pathology, emphasizing the therapeutic potential of modulating Piezo1 expression in astrocytes.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Altered expression of purinergic receptor genes in mast cells from IBD mice

Estepa-San Nicolás, Ana [1 2]; Guerrero-Alba, Raquel [2]; Salinas-Miralles, Eva [1]; Valdez-Morales, Eduardo [2]; Córdova -Dávalos, Laura E [1]; Cervantes-García, Daniel [1 3]; Jiménez-Vargas, Mariela[1].

[1]Department of Microbiology, Basic Science Center, Autonomous University of Aguascalientes, Aguascalientes, México. [2]Department of Physiology and Pharmacology, Basic Science Center, Autonomous University of Aguascalientes, Aguascalientes, Mexico. [3]Secretariat of Science, Humanities, Technologies and Innovation, México City, México.

E-mail: estepa8226@gmail.com

An excessive accumulation of mast cells (MCs) has been identified in the colonic mucosa of patients with inflammatory bowel disease (IBD). Elevated histamine and tryptase levels in the mucosa indicate MC degranulation, which may contribute to IBD pathogenesis. Emerging evidence suggests that an extracellular imbalance between ATP and adenosine is a key driver of immune dysregulation in IBD. Purinergic receptors, P1 (adenosine receptors) and P2 (P2X and P2Y), are recognized modulators of IBD onset and severity. This study aimed to examine the gene expression profile of P1 and P2 receptors in colonic mucosal MCs using a murine IBD model. IBD was induced in C57BL/6 mice by intrarectal administration of DNBS (200mg/kg in 50% ethanol), while control animals received the vehicle alone. Disease severity was assessed through weight loss and macroscopic damage scores of colonic tissues. Four days after IBD induction, distal colon samples were collected, rinsed with cold saline solution, and the mucosal layer dissected. MCs were isolated by enzymatic dissociation and discontinuous Percoll gradient. Cell viability and purity were 72% and 90%, respectively. MC identity was confirmed by mast cell protease-1 (Mctp1) expression using single-cell PCR, while purinergic receptor gene expression was assessed by qPCR. Results showed that DNBS-treated mice exhibited an 11% decrease in body weight, a significant 24.5% reduction in colon length, and higher macroscopic damage scores. Analysis of IBD-derived MCs revealed a significant decrease in P2Y6 and A1 receptor expression, alongside a marked increase in P2X1 expression compared to the control group. This differential expression suggests that P2Y6, P2X1 and A1 receptors may play functional roles in MC-mediated mechanisms contributing to IBD pathophysiology.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025
XXVI Congreso Nacional de Inmunología

Área del artículo: **Inmunología de enfermedades infecciosas**

Molecular characterization of a genetic variant in JAK3

Estevez-Mieres, Mercy; Cruz-Muñoz, Mario Ernesto [1];
Lugo-Reyes, Saul O. [2].

[1]Facultad de Medicina UAEM, Laboratorio de Inmunología Molecular, Cuernavaca, Morelos, México. [2]Instituto Nacional de Pediatría, Laboratorio de Inmuno Deficiencias, Ciudad de México, México, México.

E-mail: estevezmercy7@gmail.com

Cytokine receptor signaling represents a form of intercellular regulation that substantially impacts the development and function of immune cells. Activation of these specific receptors on the surface of these cells can mediate diverse cellular responses, including lineage commitment, differentiation, proliferation, survival, and functional activation. Protein tyrosine kinases, known as Janus kinases (JAKs), are crucial for transducing cytokine binding into appropriate intracellular signals. Among these, the JAK3 protein is unique to a specific family of cytokine receptors that play multiple pivotal roles in immunity. Mutations in JAK3 have been associated with a variety of immune cell disorders. Somatic gain-of-function mutations have been identified in various immune cell malignancies, while germline loss-of-function mutations have been linked to combined immunodeficiency. This study aimed to characterize the p.Arg799Cys variant in JAK3. The genome of a pediatric patient who presented with a diverse range of symptoms and subsequently developed natural killer (NK) cell lymphomas was sequenced. The p.Arg799Cys variant was detected in a heterozygous state in JAK3 and had not been previously reported in the literature. Subsequently, wild-type JAK3 was cloned into a pKS cloning vector. Site-directed mutagenesis was performed, followed by transfection into Baf3 cells. Finally, functional assays, including proliferation and Western blotting, were conducted. The p.Arg799Cys mutation is expected to result in a gain of function, which could explain the presence of NK cell lymphoma in the patient.

TRABAJO
NO
PRESENTADO

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

CRLF2 expression in pediatric Ph-like and Ph$^+$ B-cell acute lymphoblastic leukemia

Favela-Maldonado, Enver Tlali [1 2]; Núñez-Enríquez, Juan Carlos [3];
Moreno-Lafont, Martha Cecilia [1]; Robles-Ramírez, Roberto Joaquín [2];
Escobar-Sánchez, Aldo [2]; López-Santiago, Rubén [1];
Rodríguez-Martínez, Sandra [1]; Arriaga-Pizano, Lourdes Andrea [4].

[1]Escuela Nacional de Ciencias Biológicas IPN, Departamento de Inmunología, Alcaldía Miguel Hidalgo, Ciudad de México, México. [2]UMAE Hospital de Pediatría Centro Médico Nacional Siglo XXI IMSS, Laboratorio de Patología Clínica, Alcaldía Cuauhtémoc, Ciudad de México, México. [3]UMAE Hospital de Pediatría Centro Médico Nacional Siglo XXI IMSS, División de Investigación en Salud, Alcaldía Cuauhtémoc, Ciudad de México, México. [4]UMAE Hospital de Especialidades Centro Médico Nacional Siglo XXI IMSS, Unidad de Investigación Médica en Inmunoquímica, Alcaldía Cuauhtémoc, Ciudad de México, México.

E-mail: enverfavela@gmail.com

Pediatric B-cell acute lymphoblastic leukemia (B-ALL) includes several genetic subtypes, among which Philadelphia chromosome–positive (Ph$^+$) and Philadelphia-like (Ph-like) cases are associated with poorer outcomes. Ph$^+$ B-ALL is defined by the BCR-ABL1 fusion, while Ph-like B-ALL lacks that fusion but shares a similar gene-expression profile driven by other kinase-activating alterations. One such alteration involves the cytokine receptor–like factor 2 (CRLF2), which partners with the interleukin-7 receptor α chain to stimulate JAK/STAT signaling. This study set out to assess whether elevated CRLF2 expression marks Ph-like and Ph$^+$ B-ALL subtypes and to explore its usefulness in patient stratification. Bone marrow samples from children diagnosed between July 2024 and July 2025 underwent standard EuroFlow immunophenotyping to confirm B-ALL and exclude mixed-phenotype cases. We then measured CRLF2 transcript levels by multiplex qRT-PCR, normalizing to GAPDH. Of high-risk B-ALL patients, displayed markedly increased CRLF2 expression. Both belonged to the Ph-like category based on their clinical and immunophenotypic profiles. No Ph$^+$ cases were among this subset, suggesting that CRLF2 overexpression predominantly identifies Ph-like B-ALL. Our results indicate that assessing CRLF2 levels can reliably flag Ph-like B-ALL in pediatric patients. Incorporating CRLF2 detection into diagnostic workflows—first by flow cytometry, then by rapid qRT-PCR—could allow earlier risk assessment and inform the timely introduction of targeted therapies, such as JAK inhibitors or tyrosine kinase inhibitors. In this context, CRLF2 serves both as a driver of disease biology and as a practical biomarker for guiding treatment decisions.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

PD-L1 as prognostic predictor for treatment of non-small cell lung cancer.

Federico-Guerrero, Alfonso [1]; Pacheco-Zaragoza, Adriana Darina [1]; Carrasco-Ramírez, Liliana [2]; Flores-Guzmán, Fernando [3]; Carrasco-Ramírez, Elba [4].

[1]Facultad de Medicina, Universidad Nacional Autónoma de México, CDMX, México. [2]Facultad de Ciencias políticas y sociales, Universidad Nacional Autónoma de México, CDMX, México. [3]Escuela Médica, Universidad de Colorado, departamento de cirugía plástica, Denver, Estados Unidos de América. [4]Facultad de Medicina, Universidad Nacional Autónoma de México, departamento de microbiología y parasitología, CDMX, México.

E-mail: alf.110903p@gmail.com

Introduction: PD-1 is an inhibitory receptor that functions as an immunosuppressive checkpoint. It is primarily expressed on activated T lymphocytes exposed to chronic antigen stimulation. The PD-1/PD-L1 complex inhibits TCR, CD28, and ICOS signaling, leading to the suppression of CD8+ T-lymphocytes. In non-small cell lung cancer (NSCLC), PD-L1 expression in tumor tissue is the most widely used biomarker for predicting response to PD-1/PD-L1 inhibitor therapy.

Objective: To evaluate the utility of PD-L1 as a prognostic marker for initiating first-line immunotherapy over chemotherapy in patients with locally advanced NSCLC.

Methods: We conducted a comprehensive statistical analysis of clinical trials from the past 10 years using data obtained from the following databases and journals: Nature, Frontiers, The Lancet, and the IJC.

Result: The KEYNOTE clinical trial series revealed that initiating treatment with pembrolizumab as first-line therapy prolonged overall survival (OS), achieving a 5-year survival rate of 31.9% in patients with high PD-L1 expression levels. The KEYNOTE-042 trial showed that OS in the population with a TPS >1% was 20.0 months for pembrolizumab, compared to 12.2 months for chemotherapy, meanwhile, the KEYNOTE-024 trial reported a median OS of 26.3 months with pembrolizumab compared to 13.4 months with chemotherapy. A recent study by Chen-xing Jin *et al*. highlights the neutrophil to lymphocyte ratio (NLR), lymphocyte to monocyte ratio (LMR), and systemic immune-inflammation index (SII) as emerging biomarkers for predicting both the efficacy and prognosis of first-line ICI treatment in advanced NSCLC with negative driver gene status.

Conclusion: Pembrolizumab improves OS even in patients with low or absent PD-L1 expression, thus questioning its utility as a sole biomarker. Therefore, it is essential to incorporate novel inflammatory indicators such as NLR, LMR, and SII to optimize the selection of candidates for immunotherapy in advanced NSCLC.

ISSN: 2007-3380
Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Bioinformatic identification of immune receptors in tuberculosis targeted by progranulin

Félix-Arellano, Camelia [1 2]; Jacobo-Delgado, Yolanda [1];
Santos-Mena, Alan [1]; Rivas-Santiago, Bruno [1].

[1]Instituto Mexicano del Seguro Social, IMSS, Zacatecas, Zacatecas, México. [2]Universidad Autónoma de Aguascalientes, Centro de Ciencias Básicas, Aguascalientes, Aguascalientes, México.

E-mail: cameflx16@gmail.com

Progranulin (PGRN) is a pleiotropic protein with anti-inflammatory properties, acting as an antagonist of the tumor necrosis factor receptor. Few receptors have been described as PGRN targets in infectious diseases such as tuberculosis (TB), which is caused by the Mycobacterium tuberculosis (Mtb) complex and is considered the second deadliest infectious disease worldwide. Therefore, this study aims to evaluate, through molecular docking, the binding affinity of PGRN to immune response-associated receptors in TB, as well as to assess PGRN expression during infection with different Mtb strains.

Differentially expressed genes (DEGs) were identified from the Gene Expression Omnibus database by comparing individuals with TB to healthy controls. Fifteen selected receptors underwent molecular docking with PGRN fragments F, A, and C using Haddock 2.4. Binding affinity and interacting amino acids were assessed using the Haddock score and LigPlot+, respectively. Human monocyte-derived macrophages were infected in vitro for 2 hours with H37Rv and multidrug-resistant (MDR) Mtb strains. Total RNA was extracted at 1, 3-, 12-, 18-, and 24-hours post-infection, and relative PGRN expression was quantified by RT-qPCR from cDNA.

In silico analysis revealed that PGRN binds to 10 of the DEGs-identified receptors, several involving interactions at the receptor's active site. In vitro, infection with both H37Rv and MDR strains induced PGRN expression at 24 hours. However, no significant differences were observed in expression levels between the strains.

In conclusion, PGRN shows binding affinity to immune response receptors in TB, particularly the pattern recognition receptors CLEC4E and TLRs 1, 2, and 8. Additionally, Mtb infection of macrophages induces PGRN expression at 24 hours post-infection, regardless of whether the infecting strain is H37Rv or MDR.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Genotypic Variability of HEV Influences Renal Manifestations and CytokineProfile

Figueroa -Miranda, Addi Pamela [1]; Copado-Villagrana, Edgar Daniel [1]; Fierro-González, Nora Alma [2]; Du Pont -De Lara, Gissela [2]; Calderón-Flores, Arturo [2].

[1]Unidad de Medicina Familiar 5, Instituto Mexicano del Seguro Social, Nogales 84000, México. [2]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, Ciudad de México, 04510.

E-mail: addipamela.miranda@gmail.com

Background: Hepatitis E is a viral infection capable of causing extrahepatic manifestations, including renal complications. Although most infections are acute and self-limiting, in patients at risk of receiving immunosuppressive treatment—such as those undergoing hemodialysis (HD)—the infection may progress to chronic forms, mainly related to hepatitis E virus (HEV) genotype 3 (gt3). While cytokine profile alterations associated with hepatitis E have been described in the context of liver damage, the role of these proteins in the development of renal manifestations remains unknown. These proteins are essential for communication between immune cells. Objective: To identify the prevalence of HEV, the infectious genotype, and the serum cytokine profile associated with infection in a cohort of HD patients. Methods: A cohort of 67 HD patients was studied to determine HEV prevalence via ELISA (anti-HEV IgM/IgG antibodies) and seminested RT-PCR (viral RNA detection). Discrete regions of the viral genome were sequenced using the Sanger method to identify the genotype. The cytokine profile (IFN-γ, IL-10, IL-1β, and IL-12p70) was analyzed using multiplex assays in serum from RNA-positive and RNA-negative patients. Results: No anti-HEV IgM antibodies were detected. Anti-HEV IgG antibodies were found in 16% of samples. Among IgG-negative patients, 69% tested positive for viral RNA; 63% of IgG-positive patients were also RNA positive. Genotype 3 was identified in all cases. One patient developed chronic infection in the absence of immunosuppression; the others were classified as acute cases. No evidence of liver damage was found. Cytokine analysis showed increased IFN-γ in acutely infected patients, attributed to HD, and decreased IL-10 in infected vs noninfected patients. Conclusions: HEV genotype 3 circulates among HD patients. Antibody detection is insufficient; viral genome detection is essential. Infection can occur without liver dysfunction, and decreased IL-10 is associated with HEV infection.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Systemic inflammatory markers in ST-elevation myocardial infarction patients

Fisher-Bautista, Brandon Uriel [1 2]; Priego-Ranero, Ángel Alexis [3 4];
Amezcua-Guerra, Luis Manuel [1]; González-Pacheco, Héctor [5];
Fonseca-Camarillo, Gabriela [1].

[1]Instituto Nacional de Cardiología Ignacio Chávez, Departamento de Inmunología, Ciudad de México, México. [2]Instituto Politécnico Nacional, Programa de Maestría en Ciencias Químico Biológicas, Ciudad de México, México. [3]Instituto Nacional de Cardiología Ignacio Chávez, Departamento de Hemodinámica, Ciudad de México, México. [4]Universidad Nacional Autónoma de México, Programa de Maestría en Ciencias Médicas, Odontológicas y de la Salud, Ciudad de México, México. [5]Instituto Nacional de Cardiología Ignacio Chávez, Unidad Coronaria, Ciudad de México, México.

E-mail: brandoncamazotz@gmail.com

Inflammation is a physiological response against prejudicial agents such as bacteria, virus and protozoa. However, an exacerbated inflammatory process can lead to diseases and worse outcomes. It has been recognized that systemic inflammation has a major role in the pathogenesis and progression of ST-elevated myocardial infarction (STEMI). Inflammatory markers including neutrophile-lymphocyte ratio (NLR), platelet-lymphocyte ratio (PLR) and high sensitivity C reactive protein (hs-CRP) could be useful as prognostic markers in STEMI. The aim of the study was to observe the correlation between the NLR, PLR and hs-CRP in STEMI patients and clinical scores such as GRACE, TIMI and Killip-Kimball scores, risk factors and complications. For this, we conducted an observational, prospective and analytical study in STEMI patients from the Instituto Nacional de Cardiología Ignacio Chávez, prior to receiving reperfusion therapy. Patients with history of cardiomyopathy, stroke, sepsis, pacemaker, autoimmune diseases, neoplasia, or immunosuppression were excluded. All data were collected from the digital clinical record from the institute. Statistical analysis was performed in SPSS version 22. Statistical significance was considered with a p-value <0.05. We included a total of 57 STEMI patients and we observed that NLR, PLR and hs-CRP levels were significantly associated with high clinical scores (GRACE, TIMI, Killip-Kimball) [$p < 0.05$], which predict short- and long-term mortality. These results suggest that systemic inflammation increase the mortality risk. Additionally, prolonged ischemia and ventricular function were also associated with hs-CRP levels ($p < 0.01$). Meanwhile, NRL showed a significant association with mechanic complications and cardiogenic shock ($p < 0.01$). Intriguingly, only hs-CRP was associated with STEMI biomarkers such as troponin T and NT-proBNP ($p < 0.01$). Solely NLR showed a correlation with cardiovascular death ($p = 0.031$). In conclusion, systemic inflammatory markers are associated with STEMI prognostic scales, risk factors and complications. Inflammatory markers could be predictor factors and guide targeted therapies in STEMI patients.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

IL-37 against ferroptosis induced during ischemia/reperfusion in an *in vitro* model

Fisher-Bautista, Brandon Uriel [1 2]; Cruz-Gregorio, Alfredo [3];
Fonseca-Camarillo, Gabriela [1].

[1]Instituto Nacional de Cardiología Ignacio Chávez, Departamento de Inmunología, México City, México. [2]Instituto Politécnico Nacional, Programa de Maestría en Ciencias Químico Biológicas, México City, México. [3]Instituto Nacional de Cardiología Ignacio Chávez, Departamento de Fisiología, México City, México.

E-mail: brandoncamazotz@gmail.com

Although ferroptosis, a cell death process associated with increased reactive oxygen species and iron-induced oxidative stress, has been widely studied in diseases such as cancer, its development in diseases like ischemia-reperfusion injury (IRI) remains poorly understood. Therefore, little is known about the mechanisms that regulate inflammation in ferroptosis. IL-37, an immunoregulatory cytokine, has been shown to suppress ferroptosis and may play a crucial role during IRI, thereby significantly decreasing cardiomyocyte death. The aim of this study was to evaluate the effect of IL-37 as a cardioprotector against ferroptosis-induced death in an in vitro hypoxia/reoxygenation model. For this we employed an in vitro IRI model with the rat cardiomyoblast cell line H9c2, which was maintained in an anaerobic chamber with low levels of nutrients and oxygen, thereby simulating ischemia. Cells were subsequently provided with oxygen and nutrients to simulate reperfusion. We also exposed a group of cells to different concentrations of IL-37 before hypoxia/reoxygenation (H/R) treatment and, using MTT cell viability assays, sought to determine the cardioprotective concentration of IL-37 for H9c2 cells by increasing their viability. We observed that administration of IL-37 24 hours before H/R treatment did not produce a protective effect. Therefore, we hypothesized that administration of IL-37 before reoxygenation might reveal a cardioprotective effect. Consequently, we plan to perform these assays, these observations are preliminary results. If there is an increase in cell viability due to IL-37, we will proceed to analyze the cardioprotective effect of IL-37 against ferroptosis, focusing on the measurement of inflammatory markers.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Interaction among polymorphisms of cytokines reveals synergistic influence on susceptibility to RA

Flores-Castro, Ma. Natividad [1]; Falfán-Valencia, Ramcés [2];
Gutiérrez-Pérez, Ilse Adriana [1]; Zaragoza -García, Oscar [1];
Briceño Cárdenas, Olivia [3]; Guzmán-Guzmán, Iris Paola [1].

[1]Laboratorio de Investigación Multidisciplinaria e Innovación Biomédica, Facultad de Ciencias Químico-Biológicas, Universidad Autónoma de Guerrero, Chilpancingo, Guerrero, México. [2]Laboratorio de Neumogenómica, Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas, Ciudad de México, México. [3]Centro de Investigación en Enfermedades Infecciosas, Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas, Ciudad de México, México.

E-mail: natividadfloresc@icloud.com

The cytokines IL-6, IL-1β, and TNF-α play a crucial role in immune response, and inflammation, and contribute to joint damage and the pro-inflammatory state of rheumatoid arthritis (RA). Numerous genetic loci have been linked to susceptibility for RA, however, studies in the Mexican population have often shown limited association and not explore different gene-gene interactions to reveal new insights into RA susceptibility. Thus, the study aims to determine if specific combinations of genetic variations in IL-6, IL-1β, and TNF-α increase the susceptibility to developing RA. A case-control study was conducted in 250 subjects by each group. The SNPs of IL-1B -511A/G (rs16944), IL-6 3UTR'G/A (rs1818879) y TNF-a -308G/A (rs361525) were genotyped by allelic discrimination using commercial TaqMan® probes. The allelic distribution of the polymorphisms studied in the IL-1β, IL-6, and TNF-α genes was found in the Hardy-Weinberg genetic equilibrium ($p>0.05$). None of the polymorphisms were individually associated with susceptibility to RA. However, a haplogroup analysis reveals that the interaction of GGG alleles (rs16944, rs1818879, and rs361525) have a synergistic influence on susceptibility to RA (OR= 1.53, $p=0.025$). We conclude that interaction among the candidate cytokines genes may enhance susceptibility to RA in the Mexican population.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Combination therapy with doxorubicin and VLP-rME in a mouse model of TNBC

Flores-Correa, Edgar Emiliano [1]; Trejo-De la Mora, Sandra Sulibeth [1];
Moreno Fierros, Leticia [1]; Ilhuicatzi Alvarado, Damaris [1].

[1]Unidad de Biomedicina (UBIMED) Laboratorio de Inmunidad en Mucosas Facultad de Estudios Superiores Iztacala Avenida de los barrios Número 1, Los Reyes Iztacala Tlalnepantla, Estado de México, C.P. 54090.

E-mail: emilianoflorescorrea0@gmail.com

Globally and in Mexico, breast cancer ranks among the top three cancers in both incidence and mortality, making it a major public health concern. It is classified into four subtypes based on hormone receptor expression: Luminal A, Luminal B, HER2-positive, and triple-negative breast cancer (TNBC). TNBC, defined by the absence of estrogen receptor (ER), progesterone receptor (PR), and HER2 expression, is the most aggressive subtype, associated with poor prognosis and a lack of targeted therapies.

Immunotherapy has emerged as a promising strategy to enhance the immune system's ability to combat tumors. In previous work, our group developed a virus-like particle (VLP) prototype, termed rMe-VLP, designed as a delivery system for tumor neoepitopes. The selected neoepitopes included Tmtc2, Gprc5a, Qars, and survivin. These VLPs were evaluated in a TNBC mouse model using 4T1 cells inoculated into BALB/c mice. Treatment with rMe-VLP reduced tumor growth and macrometastases by inducing cytotoxic immune responses.

In the present project, a TNBC model was established by inoculating 4T1 cells into the right mammary gland to evaluate the enhanced therapeutic potential of rMe-VLP when combined with the chemotherapeutic agent doxorubicin, known to induce immunogenic cell death. Mice were immunized peritumorally with VLPs every seven days, and doxorubicin was administered intravenously. Subsequently, the spleen, inguinal lymph nodes, and tumor were harvested for analysis of myeloid-derived suppressor cell (MDSC) populations by flow cytometry, along with the quantification of proinflammatory cytokine production.

The combined treatment significantly reduced tumor volume and MDSC presence in the tumor, spleen, and inguinal lymph nodes. It also decreased lung metastasis formation and enhanced proinflammatory cytokine production. These findings highlight the potential of combining rMe-VLP with doxorubicin as an effective therapeutic strategy against TNBC.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Engineering a Safe Minicell-Producing Salmonella Typhimurium for Vaccine Delivery Vector purposes

Magaño-Bocanegra, Kevin Jorge [1,2]; Flores-Durand, Gary Jacsel [1,2]; Cedillo-Barrón, Leticia [2]; Rodriguez-Espino, Benjamín Antonio [3]; García-Nava, Yenny Elizabeth [1]; Flores-Martínez, Luis Fernando [1]; Cordoba-Trejo, Adonai [1]; Luria-Pérez, Rosendo [1*].

[1]Unit of Investigative Research on Oncological Diseases, Children's Hospital of México Federico Gómez, México City, México. *Correspondence: rluria@himfg.edu.mx. [2]Department of Molecular Biomedicine, Center for Research and Advanced Studies of the National Polytechnic Institute, México City, México. [3]Unit of investigative Research on Nephrology. Children's Hospital of México Federico Gómez, México City, México.

E-mail: gary.flores.durand@gmail.com

The development of safe and effective vaccine vectors is essential for optimizing immunization strategies. Live-attenuated Salmonella enterica has been extensively explored as a vaccine vector due to its intrinsic tropism for lymphoid cells, particularly Antigen-Presenting Cells, and its strong immunostimulatory capacity. However, its use remains limited due to biosafety concerns. To overcome this issue, bacterial minicells —non-replicative, chromosome-free particles —represent a promising alternative. Minicells retain the immunogenic components of the parental bacterium, can carry plasmids, and express recombinant proteins, and their inability to replicate eliminates the risk of infection, offering a safer platform for vaccine delivery. This study reports the generation of a minicell-producing Salmonella enterica serovar Typhimurium strain through targeted deletion of the minCD genes (bacterial cell division regulators) using a scarless genome editing system. This system includes: 1) the CAS9BAC1P plasmid (Sigma), which allows constitutive expression of the Cas9 endonuclease and arabinose-inducible expression of the lambda-red recombination system; 2) the pgRNAminD plasmid, encoding a gRNA targeting the minD gene; and 3) a donor dsDNA fragment carrying the ΔminCD deletion flanked by 300 bp of homology arms. The pgRNAminD plasmid was constructed by in vivo assembled from CRISPR30 (Sigma), and the donor dsDNA was generated by PCR from the pGEM-T-ΔminCD plasmid, which was constructed by deleting minCD from the minCDE operon cloned into pGEM-T (Promega). All plasmid constructs were analyzed by electrophoresis and restriction digestion and validated by Sanger sequencing. Salmonella Typhimurium ATCC 14028 was first transformed with CAS9BAC1P, induced with arabinose, and subsequently transformed with pgRNAminD and the ΔminCD donor fragment. PCR confirmed the ΔminCD deletion in 100% of the selected colonies. Finally, the production of minicells in Salmonella Typhimurium ΔminCD strains was verified by partial purification of the minicells and their analysis by optical microscopy and flow cytometry comparing them with the parental strain. Funding: HIM-2024-057.

Área del artículo: **Inmunidad tumoral**

Bone marrow dormant tumor cells maintain reactive memory CD8 T cells

Ramírez-Carrasco, Elba [1 2]; Federico-Guerrero, Alfonso [2];
Morales-Ramírez, Claudia Alejandra [3]; Carrasco-Ramírez,
Liliana [4]; Flores-Guzmán, Fernando [5].

[1]Unidad de Microscopia, Facultad de Medicina, UNAM, CDMX, México. [2]Departamento de Microbiologia, Facultad de Medicina, UNAM, CDMX, México. [3]Facultad de Ciencias Políticas y Sociales, UNAM, CDMX, México. [4]Hospital General Dr. Enrique Cabrera, CDMX, México. [5]Universidad de Colorado, Denver Colorado, EEUU.

E-mail: fernando.floresguzman@cuanschutz.edu

The hypothesis of the cancer stem cell (CSC) suggests that neoplastic clones are maintained by a fraction of tumor cells and represent disseminated dormant tumor cells. We used a ret transgenic mouse spontaneous melanoma model, in which 25% of transgenic mice develop skin tumors with metastases in lymph nodes (mLN), liver, lungs and bone marrow (BM). Mice older than 20 weeks without macroscopic tumors contain in the BM tyrosinase related protein (TRP)-2-specific effector memory CD8+T cells and show no further melanoma progression. This suggests a potential role of dormant tumor cells in the maintenance of memory CD8+T cells. TRP-2+CD133+ melanoma cells represent less than 1.5% of all cells in primary skin tumors and mLN. Most of these cells were Ki67-negative, thereby these cells exist in a dormant state. TRP-2+CD133+ melanoma cells were detected in 2 of 712(0.238%) and 4 of 1285(0.311%) disseminated melanoma cells in the BM of mice without and with macroscopic tumors, respectively. The dormant state of TRP-2+CD133+ melanoma cells was confirmed on the negative expression of Ki67 and PCNA. We found that TRP-2+Ki67-negative melanoma cells were co-localized with memory CD8+T cells both in mice without and with macroscopic tumors and the rate of memory CD8+ T cells interacting with TRP-2 +Ki67-negative melanoma cells was less than 15% in the BM. Certain IFN-gamma-producing CD8+T cells interacted either with single TRP-2+ melanoma cells or the smallest cluster of melanoma cells (2-5 TRP-2+ cells), which two TRP-2-specific CD8+T cells produced perforin, but none of them were co-localized either with TRP-2+ melanoma cells or TRP-2+CD133+ melanoma cells. In conclusion, our data demonstrate the existence of a subpopulation of CD133+ melanoma cells in ret transgenic mice and dormant TRP-2+ melanoma cells are able to interact with CD8+T cells in the BM of tumor-bearing mice.

Área del artículo: **Inmunotecnología e inmunoterapia**

Cell-permeable Bax BH3 peptide induces chemosensitization in Head Neck cancer cells

Flores -Martínez, Luis Fernando [1]; González Franco, José [1 2]; García Machorro, Jazmín [2]; Magaño Bocanegra, Kevin Jorge [1 3]; García Nava, Yenny Elizabeth [1 3]; Córdova Trejo, Adonai [1]; Valenzuela Maldonado, Altagracia [1]; Luria Pérez, Rosendo [1]*.

[1]Unit of Investigative Research on Oncological Diseases, Children's Hospital of México Federico Gómez, México City, México. *Correspondence: rluria@himfg.edu.mx. [2]Escuela Superior de Medicina, Instituto Politécnico Nacional, Ciudad de México, México. [3]Department of Molecular Biomedicine, Center for Research and Advanced Studies of the National Polytechnic Institute, México City, México.

E-mail: lfernandomvzfm@gmail.com

Squamous cell carcinoma of the head and neck (CCECC) comprises a group of neoplasms that are among the most common cancers in Mexico. Despite advances in treatment, drug resistance remains a significant obstacle to effective tumor eradication. One mechanism involved in this resistance is the overexpression of antiapoptotic proteins from the Bcl-2 family, such as Bcl-XL, Bcl-2, and Mcl-1. Peptides derived from the BH3 domain of the proapoptotic protein Bax have been reported to block the activity of Bcl-2 family antiapoptotic proteins and restore the apoptosis of tumor cells. Our previous work demonstrated that the cell-permeable Bax BH3 peptide, delivered by attenuated Salmonella enterica, induced apoptosis, reduced tumor volume, and improved survival of an in vivo model of non-Hodgkin lymphoma. In this work, we evaluate cell death, apoptosis restoration, and chemosensitization mediated by the cell-permeable Bax BH3 peptide (AntFBax) of CCECC cells. The three-dimensional structure of the AntFBax peptide (a construct comprising a fusogenic Antennapedia domain, Flag tag, and Bax-BH3 sequence) was obtained through bioinformatic analysis using AlphaFold 2.0. Western blot analysis confirmed the presence of proapoptotic (Bax, Bak) and antiapoptotic (Bcl-XL, Mcl-1) proteins in CCECC cells (FaDu cells). After the peptide synthesis, AntFBax induced cell death (MTT assay), restored apoptosis (active caspase-3 assay), and sensitized FaDu cells to cisplatin chemotherapy. Significantly, the peptide did not cause cytotoxic effects in peripheral blood mononuclear cells. The cell-permeable Bax BH3 peptide effectively induced apoptosis, promoted chemosensitization, and triggered cell death in CCECC cells without any damage to normal cells. These findings suggest that cell-permeable Bax BH3 peptides may represent a promising therapeutic strategy for overcoming drug resistance of Squamous cell carcinoma of the head and neck. Funding: CONACYT CB-2013-01-222446, Fondos Federales (HIM-2015-049 SSA 1217, HIM-2021-056 SSA 1756, HIM-2022-061 SSA 1826).

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Effect of Gold Nanoparticles on the Biological Function of Human Basophils

Gutiérrez-Cruz, Sinai Guadalupe [2]; Jiménez Vega, Ángel Adrián [1];
Llanos-González, Abdiel Nain [1]; Benítez-Arvizu, Gamaliel [3];
Rodríguez Cortés, Octavio [1]; Muñoz Diosdado, Alejandro [2];
Flores-Mejía, Raúl [1].

[1]Instituto Politécnico Nacional. Escuela Superior de Medicina. Laboratorio de Inflamación y Obesidad. Ciudad de México, México. [2]Instituto Politécnico Nacional. Unidad Profesional Interdisciplinaria de Biotecnología. Ciudad de México, México. [3]Instituto Mexicano del Seguro Social. Banco de Sangre del Centro Médico Nacional Siglo XXI. Ciudad de México, México.

E-mail: raflores@ipn.mx

Gold nanoparticles (AuNPs) stand out for their therapeutic and diagnostic potential due to their unique properties. Once in the body, regardless of their route of entry, these particles reach the bloodstream, interacting with immune cells and modulating responses that can activate or suppress immunity and cause inflammation. Basophils are essential in the regulation of inflammatory processes and allergic responses thanks to their ability to release inflammatory mediators such as histamine and cytokines. However, little is known about the direct impact of AuNPs on human basophils. Their interaction could alter key functions such as degranulation and cytokine production, with important implications for immune responses. This knowledge gap underscores the need to study the influence of AuNPs on these pivotal cells, both to explore promising biomedical applications and to identify potential associated risks. This work evaluated the effect of AuNPs on viability, degranulation, and cytokine production in primary cultured human basophils, as well as their cellular internalization. It began with the characterization of the AuNPs by TEM and Nanoparticle Tracking Analysis, confirming their size and dispersion. From a leukocyte concentrate, peripheral blood mononuclear cell (PBMC) cultures were prepared and treated with 5, 20, and 40 nm AuNPs at concentrations of 10, 20, and 30 µg/mL for 4, 24, and 48 hours. Cell viability, measured with 7-AAD, showed that none of the conditions evaluated generated cell death. Subsequently, degranulation was quantified in cultures treated for 48 hours at 30 µg/mL by flow cytometry, observing that AuNPs did not generate degranulation compared to fMLP. Finally, the internalization of AuNPs in basophils was analyzed by confocal microscopy. This last analysis revealed that the nanoparticles are mainly located in the cytoplasm, while a smaller fraction manages to enter the cell. Thanks to the IPN Research and Postgraduate Secretary (SIP) for funding.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Western Diet-Induced Obesity on the Response of Mast Cells from C57BL/6 Mice

Rodríguez-Cortés, Octavio [1]; Rechy-Ahumada, Miguel [2]; Meneses-Preza, Yatsiri [2]; Soria-Castro, Rodolfo [2]; Alfaro-Doblado, Ángel [2]; López-Aguilar, María Fernanda [1]; Chacón-Salinas, Rommel [2]; Flores-Mejía, Raúl [1].

[1]Instituto Politécnico Nacional. Escuela Superior de Medicina. Sección de Estudios de Posgrado e Investigación. Laboratorio de Inflamación y Obesidad. Ciudad de México, México.
[2]Instituto Politécnico Nacional. Escuela Nacional de Ciencias Biológicas. Departamento de Inmunología. Ciudad de México, México.

E-mail: raflores@ipn.mx

Obesity is a health problem, and one of the factors contributing to its development is the dietary changes that have occurred in recent decades. The Western diet is characterized by the consumption of high-calorie foods. Cases of obesity are projected to increase, and with it, the demand for health services for obesity-related conditions. Recent studies have shown that obese patients are susceptible to infections and at greater risk of developing complications. However, the immune mechanisms that are affected and promote infectious processes are unclear. Mast cells play a key role during the early immune response in infections; they are sentinel cells that detect the presence of pathogens and initiate the immune response that contributes to control. The present study was conducted using male C57BL/6 mice fed a high-calorie Western diet for 18 weeks. At the end of the diet, the somatometry, biochemistry, metabolism, and histology of the mice on the Western diet and mice on the standard diet were characterized. Bone marrow-derived mast cells (BMDMCs) were obtained from the bone marrow of mice and evaluated for differentiation purity, degranulation capacity, and IL-6 production using different bacterial stimuli. The Western diet group of mice showed statistically significant differences in total final weight and pelleted adipose tissue weight. They also presented alterations in cholesterol, SGPT, IL-6 expression in the liver, and histological changes. Regarding the BMDMCs, no differences were found in differentiation capacity or degranulation capacity. In contrast, a significant decrease in IL-6 production was found in BMDMCs from Western diet mice when stimulated with three different bacterial stimuli. The latter, since it is present in innate response cells, could be related to susceptibility to infections and the risk of developing complications. Thanks to the IPN Research and Postgraduate Secretary (SIP) for funding.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

B-1 lymphocytes and sepsis

Flores-Rabasa, Regina [1]; León-Pedroza, José Israel [1 2];
Correa-Beltrán, María Dolores [1].

[1]Universidad Anáhuac México, Centro de Investigación en Ciencias de la Salud, Huixquilucan, Estado de México, México. [2]Hospital General de México "Dr. Eduardo Liceaga", Laboratorio de Proteómica y Metabolómica, Ciudad de méxico, México.

E-mail: reginafloresrabasa337@gmail.com

Sepsis is a dysregulated immune response to an infectious process associated with high mortality. In 1983, B-1 lymphocytes were discovered in mice, which secrete natural antibodies, IL-10 and function as antigen presenting cells, protecting them from increased mortality in sepsis. In humans, they were recently redefined, so little is known about their function, including in sepsis. It is only known that the percentage of B-1 lymphocytes in peripheral blood of healthy people is 1%. Therefore, we consider that the relative abundance of B-1 lymphocytes influences sepsis in humans as well. Objective: To determine the prognostic value of B-1 lymphocyte count on 28-day mortality in adult patients with sepsis. Methods: Patients with sepsis were recruited from the General Hospital of Mexico. Peripheral blood samples were collected, stained with fluorescent antibodies (CD20, CD27, CD43, CD38 and CD69), red blood cells were lysed, centrifuged and decanted. Subsequently, they were taken to the flow cytometer with which the percentage of B-1 lymphocytes was determined Results: Thirty-two patients with sepsis were recruited, ten of whom died during the 28 days of follow-up. By flow cytometry, both groups of patients, survivors and non-survivors presented a percentage of B-1 lymphocytes of the total B lymphocytes lower than 1%: median 0.46% and 0.28 respectively, with a value of $p = 0.621$. The cut-off point established with the ROC curve was a median of 280 B-1 lymphocytes/mL, corresponding to 0.52% of B-1 lymphocytes, an ABC of 0.57, sensitivity of 77% and specificity of 47%. With the Kaplan-Meier curve, it was observed that patients with less than 280 lymphocytes (0.52%) B-1/mL had a tendency to die from day 15 of hospital admission.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Transcriptional analysis of Intestinal Wound Healing in patients with Ulcerative Colitis.

Fonseca-Camarillo, Gabriela [1 2] ; Yamamoto -Furusho, Jesús Kazuo [1].

[1]Inflammatory Bowel Disease Clinic. Department of Gastroenterology. Instituto Nacional de Ciencias Médicas y Nutrición, Salvador Zubirán.. [2]Department of Immunology, Instituto Nacional de Cardiología. Ignacio Chávez.

E-mail: gabrielafaster@gmail.com

Background and aim: Defects in the intestinal epithelial barrier function and intestinal wound healing have been associated in the pathogenesis of Ulcerative Colitis. However, genes involved in the epithelial barrier function and intestinal wound healing has not been yet described in patients with active and remission UC. The aim of the study was to study the transcriptome panel of genes (ATF2, ATF7, SIRT2, PRAP1, CDH1, LAMB1 and ECM1) in the colonic mucosa from UC patients. Patients and Methods: We studied a total of 40 patients with definitive diagnosis of UC (20 active and 20 remission) and non-inflamed control group (N=20) without endoscopic evidence of intestinal inflammation. In all groups, gene expression was measured by real- RT-PCR. Statistical analysis was performed using the SPSS 19 program by the Kruskal-Wallis One Way Analysis of Variance on Ranks Data were expressed as the median, range and mean ± SE. A P value ≤ 0.05 was considered as significant.

Results. Decreased gene expression of the ATF2, ATF7, SIRT2 and ECM1 were detected in the colonic mucosa from patients with active UC compared to the group in remission where an increase in messenger RNA levels was found ($p<0.05$). In contrast, the expression levels of the PRAP1, CDH1, and LAMB1 genes were found to be increased in the colonic mucosa of patients with active UC compared to controls ($p=0.002$, $p=0.000$, and $p=0.003$, respectively).

Conclusions: The gene expression analysis showed a decreased differential expression of the ATF2, ATF7, SIRT2 and ECM1 molecules under inflammatory conditions in patients with active UC compared to patients in remission and controls. This evidence suggests that in colonic mucosa of patients with active UC, there is a reduction in the tissue repair mechanisms of colonic epithelial cells, which favors inadequate remodeling of the epithelium due to degradation of extracellular matrix components.

Área del artículo: **Inmunología clínica y traslacional**

Participation of T Cell Factor -1 (TCF1) in Sjôgren’s Syndrome.

Fonseca -Camarillo, Gabriela [1]; Furuzawa -Carballeda, Janette [2];
Hernández-Molina, Gabriela [3]; Aguilar -León, Diana [4]; Fisher-Bautista,
Brandon Uriel [1]; Amezcua -Guerra, Luis Manuel [1]; Aranda -Fraustro,
Alberto; Loredo-, Maria Lilia [5].

[1]Department of Immunology, Instituto Nacional de Cardiología Ignacio Chávez, México, México.. [2]Department of Experimental Surgery. Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Mexico City, Mexico.. [3]Department of Immunology and Rheumatology, Instituto Nacional de Ciencias Médicas y Nutrición, Salvador Zubirán, Mexico City, Mexico.. [4]Department of Pathology, Instituto Nacional de Ciencias Médicas y Nutrición, Salvador Zubirán, Mexico City, Mexico.. [5]Department of Pathology, Instituto Nacional de Cardiología Ignacio Chávez, México, México.

E-mail: gabrielafaster@gmal.com

TCF1 is critical for the generation of the CD8+ T cell memory response. Sjögren’s Syndrome (SS), which is characterized by lymphocytic infiltration of the salivary and lacrimal glands, where the presence of CD4+ T cells (45-50%), CD8+ T cells (15-20%), B cells has been described. (10-20%). The role of the TCF1 factor in the regulation of the effector CD8 T cells response in the initiation, perpetuation and progression of the disease is unknown. The aim of this study was to evaluate the expression of T Cell Factor1 (TCF1) in minor salivary gland biopsies of patients with primary (pSS) and secondary (SSs) Sjögren’s Syndrome and its co-localization with subpopulations of T lymphocytes (CD8+) by double immunohistochemistry. We included serum samples and MSG biopsies from 24 pSS, 5 SSc/SS, 6 SSc, 6 SLE/SS, 6 SLE patients, 8 RA/SS, 4 RA and 2 non-SS sicca patients. To determine the subpopulation of TFC1+/CD8+ in situ, by double-staining procedure for IHQ. We estimated the mean percentage of positively staining cells in four fields per sample. The Holm-Sidak’s multiple comparisons test was performed in the GraphPad Prisma 6 program. The percentage of double positive cells (TCF1+/CD8+) were increased in the MSG tissue from patients with pSS in compared to control group (p< 0.001); SLE/SS vs Control (p=0.014); RA/SS vs control (p< 0.001); RA vs control (0.011), respectively. Serum levels of TCF1 were increased in patients with pSS (p=0.025) in compared with controls.

This is the first report regarding the increased expression and synthesis of TCF-1 in patients with Sjôgren’s Syndrome. Understanding the role of TCF1 and other transcription factors in T cell dysfunction could lead to insights into the pathogenesis of Sjögren’s syndrome and potentially inform therapeutic strategies.

ISSN: 2007-3380
Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Participation of the Extracellular Matrix Metalloproteinase Inducer (EMMPRIN) in dermatomyositis.

Fonseca-Camarillo, Gabriela [1]; Furuzawa-Carballeda, Janette [2];
Amezcua-Guerra, Luis Manuel [2]; Aranda-Frasutro, Alberto [3];
Loredo, María Lidia [3].

[1]Departamento de Inmunología. Instituto Nacional de Cardiología. Ignacio Chávez. [2]Departamento de Cirugía Experimental. Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán. [3]Departamento de Patología. Instituto Nacional de Cardiología. Ignacio Chávez.

E-mail: gabrielafaster@gmail.com

Extracellular matrix metalloproteinase inducer (EMMPRIN, CD147) is a member of the Ig superfamily, with various physiological roles including the induction of matrix metalloproteinases (MMPs), leukocyte activation, and tumor progression. In this study, the aim was to determinate the expression of EMMPRIN in muscular biopsies from patients with inflammatory myopathy. For this, we employed paraffin blocks of skeletal muscle biopsies from 5 patients with a confirmed diagnosis of dermatomyositis were obtained from the Pathology Department at Ignacio Chávez National Institute of Cardiology. Diagnosis and evaluation of disease activity in muscle tissue were based on and confirmed by histopathological criteria. Double immunohistochemistry was performed to detect EMMPRIN+/MMP10+ and EMMPRIN+/MMP23B+. Statistical analysis was performed using the Holm-Sidak multiple comparisons test in GraphPad Prisma 6. We enrolled 5 patients with active dermatomyositis and 2 controls without inflammation. Antinuclear antibodies (ANAs) were positive in all patients and capillaroscopy was abnormal. Pharmacological treatment for these patients was glucocorticoids (n=3, 60%), methotrexate (n=3, 60%), and azathioprine (n=1). Histopathological findings in muscle biopsies revealed a perivascular and perimysial inflammatory infiltrate with a predominance of CD4+ T cells and B cells, consistent with a diagnosis of dermatomyositis. An increased percentage of EMMPRIN+/MMP10+ double-positive cells (p=0.02) was found compared with the EMMPRIN+/MMP23B+ cell subpopulation in the perivascular and perimysial infiltrate areas of muscle biopsies from patients with dermatomyositis. The regulation of tissue synthesis of EMMPRIN is increased in patients with dermatomyositis, this induces local inflammatory response and the accumulation of metalloproteinases (MMP10 and MMP23B) that damage connective and muscle tissue. These findings suggest the role of EMMPRIN in the pathophysiology of inflammatory myopathies.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Participation of transcription factor NR4A and NDRG in patients with Inflammatory Bowel Disease.

Fonseca -Camarillo, Gabriela [1 2] ; Furuzawa-Carballeda, Janette [3] ;
Aguilar -León, Diana [4]; Barreto -Zuñiga, Rafael [5] ;
Yamamoto -Furusho, Jesús Kazuo [1].

[1]Inflammatory Bowel Disease Clinic. Department of Gastroenterology. Instituto Nacional de Ciencias Médicas y Nutrición, Salvador Zubirán. . [2]Department of Immunology. Instituto Nacional de Cardiología. Ignacio Chávez. . [3]Department of Experimental Surgery. Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán.. [4]Department of Pathology. Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán.. [5]Department of Endoscopy. Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán.

E-mail: gabrielafaster@gmail.com

Background: Ulcerative Colitis is characterized by an aberrant immune response. Recent studies have shown that NDRG1 protein interacts with the nuclear receptor NR4A1 and functionally inhibits the transcriptional activity of NF-κB (nuclear factor Kappa B) and modulates the inflammatory response. The role of NDRGs family members and NR4A transcription factors in modulating apoptosis in the context of the inflammatory response continues to be investigated. The role and expression profile of NDRGs via NR4A receptors in IBD patients is currently unknown. Aim: Characterize the gene expression of NDRGs family and NR4A transcription factors in patients with IBD.

Materials and methods: A cross-sectional and comparative study included 38 patients with UC, 10 CD, and 18 controls with no evidence of intestinal inflammation at the histological level. For the detection of NDRG1, NDRG2, NDRG3, NDRG4, NR4A1, NR4A2, and NR4A3 from colonic biopsies we performed real-time polymerase chain reaction (PCR) and IHQ analyses for protein detection of NDRGs members. Statistical analysis was performed with the prisma Graphpad program and the statistical package SPSS version 22. The distribution of the variables was determined with the Shapiro-Wilk test. The comparison of the quantitative variables was carried out using the Dunn test for multiple comparisons. Statistical significance was determined with a value of $p<0.05$.

Results: RNA levels of NDRG1 were increased in the colonic mucosa from control patients in compared to patients with active UC and remission ($p=0.014$ and $p=0.04$). NDRG2 gene expression was increased in patients with UC in remission compared to the control group ($p=0.014$). Gene expression of NDRG4 was increased in patients with active UC compared to controls ($p=0.035$). The expression of NR4A1, NR4A2, and NR4A3 genes were increased in colonic mucosa from patients with UC in remission compared to controls ($p=0.01$, $p=0.005$, and $p=0.013$, respectively). Decreased levels of the NR4A2 gene expression were associated with ultrasensitive PCR levels ($P=0.030$) and with ESR levels ($p=0.001$).

Conclusions: The gene expression of the transcription factors NDRG2, NDRG3, NR4A1, NR4A2, and NR4A3 is increased in the group of patients with remission and low levels of the NR4A2 gene were found to be associated with high levels of CRP and ESR. These findings suggest the immunomodulatory role of the set of NDRGs proteins and NR4A1 transcription factors in patients with UC.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Turning breast cancer bone metastases hot using 5-fluorouracil against MDSCs

Fournier Pierrick, GJ [1]; Esparza Palomares, Kiomy [2];
Almeida Luna, Paloma [3]; Jiménez, Samanta [1];
González-Sánchez, Ricardo [1]; Olvera, Felipe [4];
Internicola, Candice [5]; Duboeuf, François [5]; Juárez, Patricia [1].

[1]CICESE, Departamento de Innovación Biomédica, Ensenada, BC, México. [2]UNAM, Centro de Nanociencias y Nanotecnologia, Ensenada, BC, México. [3]CICESE, Posgrado en Ciencias de la Vida, Ensenada, BC, México. [4]IBT-UNAM, Departamento de Biología Molecular y Bioprocesos, Cuernavaca, MR, México. [5]Université de Lyon, INSERM UMR 1033, Lyon, France.

E-mail: fournier@cicese.edu.mx

Bone metastases are a frequent complication of advanced breast cancer, against which immune checkpoint inhibitors are not effective. We previously identified that PD-1+ T cells in a mouse model of breast cancer bone metastases are inactivated, potentially because of the expansion of PD-L1+ myeloid-derived suppressor cells (MDSCs) in bones. 5-Fluorouracil (5-FU) can decrease MDSCs in thymoma and colon cancer models. Thus, we aim to determine the efficacy of 5-FU in breast cancer bone metastases as well as its potential side effects. Mice with orthotopic tumors or bone metastases from 4T1 breast cancer cells received four inoculations of 5-FU (50 mg/kg), which prevented the expansion of polymorphonuclear- and monocytic-MDSCs, assessed by flow cytometry in both sites. The 10 and 25 mg/kg doses were not efficient against MDSCs. Also, while these lower doses tended to increase T cell infiltration, 50 mg/kg decreased it, suggesting toxicity. Using 50 mg/kg, two and three inoculations of 5-FU still prevented MDSC expansion while increasing the number of CD4+ and CD8+ T cells in bone metastasis. On radiographs, these 5-FU treatments also decreased the osteolytic lesions. However, there was also a sharp decrease in the total amount of bone marrow cells, including B and NK cells. Thus, we sought to assess the potential side effects. Three days after three inoculations of 5-FU (50 mg/kg) in healthy mice, neutrophils, monocytes, B, and NK cells were decreased in bones, while T cells were increased. However, these effects were transient since a week later, the mice had recovered, which could be due to an increase of hematopoietic stem and lymphocytic progenitor cells after 5-FU treatment. µCT also did not reveal changes in bone parameters. Thus, 5-FU could be used against MDSC with immunotherapy to turn bone metastases from a cold to a hot microenvironment.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Discovery and characterization of Anti-Ubiquitin scFvs via Phage Display Using ALTHEA Platinum VH:VL 4x3

Fragozo-Ortiz, Ana Laura [1,2]; Gómez-Castellano, Keyla María [1,2];
Mendoza-Salazar, Ivette [1,2,3]; Vallejo-Castillo, Luis Alberto [1,2];
Vázquez-Leyva, Said [1,2]; Almagro, Juan Carlos [1,2,4];
Pérez-Tapia, Sonia Mayra [1,2,3].

[1]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI). [2], Escuela Nacional de Ciencias Biológicas.

E-mail: analau2903@gmail.com

Extracellular ubiquitin, has been implicated in immune processes. In this study, single-chain variable fragments (scFvs) specific to wild-type ubiquitin (WT) were developed for potential application in ELISA-based assays and therapeutic strategies. Using semi-synthetic phage display libraries (ALTHEA Platinum VH:VL 4x3), a molecular screening campaign was conducted through solution-phase panning over three rounds, employing biotinylated ubiquitin as the target antigen. Enrichment of specific clones was evidenced by an increase in phage output titers across successive rounds. A total of 90 clones were evaluated via indirect ELISA, identifying 18 scFvs with specific binding to ubiquitin and no reactivity to BSA. Ten unique scFv sequences were obtained by PCR amplification and Sanger sequencing. Of these, six were expressed in E. coli and purified using protein L affinity chromatography. Physicochemical characterization by SE-UPLC and SDS-PAGE confirmed expected molecular weights (~30–35 kDa), monomeric states, and high purity and final concentrations exceeded 0.6 mg/mL. Functional evaluation through ELISA demonstrated differential binding capacities: scFv C1 exhibited the highest affinity, followed by H2, E4, and C11, while E10 and A7 showed lower binding responses. All clones bound ubiquitin specifically and displayed proper folding as evidenced by protein L interaction. This work demonstrates the successful generation of anti-ubiquitin scFvs through solution-phase panning, offering promising candidates for immunodetection and therapeutic development. The observed variability in binding profiles suggests differences in affinity or epitope recognition among clones. Notably, these scFvs show potential for use in ubiquitin detection systems in complex biological samples, such as serum and plasma from various species, supporting their application in preclinical and translational research settings.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Validation process in leukemia-specialized labs from collaborative network "Mexico in alliance with St. Jude"

Fuentes-Rodríguez, Anai [1]; Garay-Sánchez, Sergio [2];
Arce-Cabrera, Daniela [3]; Romo-Rubio, Hugo [4];
Ortiz-Ramírez, Mauricio [1]; Portillo-Zavala, Claudia [5];
Moreno-Serrano, Julio [2]; Mercado-, Nataly [6];
Soldevila-Melgarejo, Gloria [1]; Aguilar-Escobar, Dinora [2];
Friedrich-, Paola [7].

[1]Laboratorio Nacional de Citometría de Flujo, Instituto de Investigaciones Biomédicas-UNAM, México City, México. [2]Hospital Infantil Teletón de Oncología, Querétaro, México. [3]Hospital Pediátrico de Sinaloa, Culiacán, México. [4]Hospital Civil de Guadalajara "Dr. Juan I Menchaca", Guadalajara, México. [5]Hospital Infantil de Especialidades del Estado de Chihuahua, Chihuahua, México. [6]Casa de la Amistad para Niños con Cáncer, México City, México. [7]St. Jude Children's Research Hospital, Global Pediatric Medicine Department, Memphis, TN, United States of América.

E-mail: afuentes@iibiomedicas.unam.mx

Mexico in Alliance with St. Jude (MAS), launched the Bridge Project in 2018 to secure access to a consensus-derived diagnostic panel for children 0-18 with suspected acute lymphoblastic leukemia (ALL). Samples from 15 participating hospitals are centralized in a referral laboratory at Hospital Infantil Teleton de Oncología (HITO). We aimed to expand the number of externally validated laboratories able to perform the diagnostic panel: DNA index, diagnostic immunophenotype, and minimal residual disease (MRD) evaluation by flow cytometry.A quality improvement project was implemented in 2023 at Laboratorio Nacional de Citometría de Flujo (LabNalCit). The validation process was conducted in collaboration with HITO and the MAS network. The validation process consisted of identifying 20 consecutive patients aged 0-18 with suspected ALL, and shipping paired bone marrow (BM) samples for immunophenotype and DNA index evaluation at baseline, and MRD evaluation at the end of both induction and consolidation in LabNalCit and HITO simultaneously. HITO's results were used as the gold standard; both institutions conducted their processes independently and compared results, aiming for concordance in six consecutive ($p<0.05$) test results. We included 21 cases of suspected ALL in this validation process. Fourteen patients had B-cell ALL (66%), one T-cell ALL (4.7%), three Myeloblastic Leukemia (14.2%), and three were negative for cancer (14.2%). Immunophenotype was concordant in 21 cases (100%), MRD was concordant in 16/16 samples (100%) and is ongoing. DNA index was concordant in 18/21 cases (85%). More than six consecutive concordant results were achieved for each test. One introductory course and six feedback sessions were conducted following the model for improvement methodology. This project achieved immunophenotype concordance corroboration and expanded MRD and DNA index testing capacity. This project strengthened pre-existing laboratory tests, promoted the expansion of new specialized tests for ALL in Mexico, and encouraged intersectoral and interinstitutional collaboration.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

CRTAM is essential for the regulation of the innate immune cell during bacterial infections

Fuerte-Pérez, América Eréndira [1]; Hernández -Galicia, Gabriela [1];
Pérez -Lara, Jocelyn Carolina [1]; Pérez-Cedillo, Darina del Carmen [2]; Campos-Mojena, Rosario [1]; Ortiz-Navarrete, Vianney Francisco [1].

[1]Centro de Investigación y Estudios Avanzados del Instituto Politécnico Nacional, Departamento de Biomedicina Molecular, ciudad de México, México, México. [2]Universidad Veracruzana, Facultad de Ciencias Químicas, Orizaba, Veracruz, México.

E-mail: ae.fuerte11@gmail.com

Class I restricted T cell (CRTAM), also known as CD355, is an inducible molecule expressed on a variety of immune cells. It was initially identified in NKT cells and CD8 + T cells and later reported in double negative thymocytes, NK cells, CD4 + T cells, neutrophils, and eosinophils. CRTAM establishes homotypic and heterotypic interaction, with its ligand Necl-2. Therefore, CRTAM would participate in the adhesion and differentiation process. The function of CRTAM has been studied principally in CD4 + T cells from CRTAM knockout mice (CRTAM-KO), in these cells playing a significant role in the production of the cytokine IFN-γ. Furthermore, in an innate immune response model using Salmonella typhimurium infected with CRTAM-KO, reduced survival was observed compared to wild-type mice. These mice also showed a decreased frequency of CD3 IFN-γ– producing cells on day 5 after infection, suggesting that CRTAM is important in triggering an effective innate immune response against bacterial infections. The present study aimed to define the role of CRTAM during acute Salmonella infection. The frequency of innate immune cells was evaluated in CRTAM-KO mice infected with 100 CFU of Salmonella typhimurium. An increase in CD11b + populations was observed starting 48 hours after infection. A trend toward increased neutrophil frequencies was detected in the spleen from 48 to 72 hours after infection, which may suggest a role for CRTAM in neutrophil migration and/or retention. On the contrary, a lower frequency of NK cells was detected 24 hours after infection. These results show that the activity of phagocytic cells was inefficient, thus affecting the control of Salmonella infection, suggesting an important role for CRTAM for the function of innate immune cells.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Wnt3a-Expressing Cancer Stem Cells Impair CD8+ TRM Function, Contributing to Immunotherapy Resistance in Melanoma

Gajón, Julián A. [1,2]; Juárez-Flores, Ángel [3]; Hernández Acevez, Juan [4]; Muñoz-Cruz, Samira [2]; Fuentes-Panana, Ezequiel M. [3]; Alfonzo, Laura C. [2,5].

[1]Posgrado en Ciencias Bioquímicas, Universidad Nacional Autónoma de México, Ciudad de México, México. [2]Unidad de Investigación Médica en Inmunoquímica, Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México. [3]Unidad de Investigación en Virología y Cáncer, Hospital Infantil de México Federico Gómez, Ciudad de México, México. [4]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, Ciudad de México, México. [5]Coordinación de Investigación en Salud, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México.

E-mail: julian.argenis.23@gmail.com

Immune checkpoint blockade (ICB) has revolutionized the treatment of advanced malignancies such as melanoma. However, a significant proportion of patients fail to respond, owing to mechanisms that remain poorly understood. Here, we developed murine melanoma cell lines (R1 and R2) derived from MO4 tumors and that are resistant to anti-PD-1 therapy. These resistant cells exhibited increased tumor initiation capacity in vivo correlating with enhanced spheroid formation in vitro, and greater invasiveness and metastatic potential in a lung macrometastasis model. Mechanistically, both R1- and R2-derived tumors showed enrichment of a cancer stem cell (CSC)-like population overexpressing Wnt3a, which correlated with reduced infiltration and impaired function of CD8^{+} tissue-resident memory (TRMs) T cells. This suggested a functional link between Wnt-driven cancer stemness and TRMs dysfunction. Pharmacological inhibition of Wnt/β-catenin signaling in vitro enhanced CD8□ T cell proliferation and expression of functional markers. Notably, Wnt/β-catenin inhibition in vivo reversed anti-PD-1 resistance in resistant tumors, coinciding with reduced OCT4^{+}Wnt3a^{+} CSC-like cells and restoration of TNFα^{+}CD8^{+} TRMs. Finally, transcriptomic analysis of publicly available melanoma cohorts showed that enrichment of the Wnt pathway correlates with poor prognosis and ICB resistance, highlighting this pathway as a potential druggable negative regulator of ICB efficacy in melanoma.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

γδ T Cells Drive Anti-NPA Autoantibodies in Lupus Mouse Model

Galarce- Sosa, Edgar Iván [1]; Barrera Aveleida, Giovanna Berenice [1]; Ramos Monteagudo, Rodrigo [1]; Rundquist -Sánchez, Edgar José [1]; Trejo-Sánchez, Gabriel [1]; Baeza-Ramírez, María Isabel [1]; Wong-Baeza, Carlos [1]; Reséndiz-Mora, Claudia Albany [1].

[1]Laboratorios de Biomembranas, Departamento de Bioquímica, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, CDMX, México. Unidad Profesional Lázaro Cárdenas, Prolongación de Carpio y Plan de Ayala s/n, Col. Santo Tomás. Ciudad de México. México.

E-mail: ivan.gal27@hotmail.com

Systemic lupus erythematosus (SLE) is a multifactorial autoimmune disease characterized by the breakdown of immunological self-tolerance and the generation of pathogenic autoantibodies. While the immunogenic role of protein antigens has been well-characterized, the contribution of lipid-based antigens remains largely unexplored. We developed a novel lupus model using liposomes bearing stable non-bilayer phospholipid arrangements (NPA), which serve as effective lupus inducers and as a source of immunogenic lipid structures that mimic membrane perturbations observed in autoimmunity. BALB/c mice that were repeatedly exposed to NPA-bearing liposomes developed a lupus-like phenotype characterized by the generation of high-affinity autoantibodies. Our investigation focused on the function of T γδ cells, a distinct subset of T cells that can recognize lipid antigens. Utilizing multiparametric flow cytometry, we analyzed T γδ cells from both the spleen and mesenteric lymph nodes, assessing activation, proliferation, cell cycle distribution, mitochondrial dynamics, and cytokine production. Lupus-induced mice exhibited a notable expansion and activation of T γδ cells. These cells showed significant proliferation, with an increase in the S and G2/M phases of the cell cycle, and underwent mitochondrial fission as an indicator of metabolic reprogramming linked to effector function. Importantly, T γδ cells secreted IL-4 and IFNγ, cytokines known to drive class switching and promote the production of IgG autoantibodies against NPAs. These findings position T γδ cells as central players in the generation of anti-NPA autoantibodies, linking innate lipid recognition with adaptive immune activation. This lipid-based model not only recapitulates key features of SLE but also provides a unique platform for exploring novel immunotherapeutic approaches targeting lipid-reactive T cell populations.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Effect of Acute Intranasal Administration of Cry1Ac Proteins (Protoxin and Toxin) on Pulmonary Mucosa

Galindo Hernández, Ricardo Agustín [1]; Ilhuicatzi-Alvarado, Damaris [2]; Moreno-Fierros, Leticia [3].

[1]Universidad Nacional Autónoma de México FES-Iztacala, Unidad de Biomedicina UBIMED, Tlalnepantla, Estado de México, México.. [2]Universidad Nacional Autónoma de México FES-Iztacala, Unidad de Biomedicina UBIMED, Tlalnepantla, Estado de México, México.. [3]Universidad Nacional Autónoma de México FES-Iztacala, Unidad de Biomedicina UBIMED, Tlalnepantla, Estado de México, México.

E-mail: richi.galindo@yahoo.com.mx

Effect of Acute Intranasal Administration of Cry1Ac Proteins (Protoxin and Toxin) on Pulmonary Mucosa Galindo-Hernandez R. A., Ilhuicatzi-Alvarado D and Moreno-Fieros L. The Cry1Ac proteins (protoxin and toxin), derived from the bacterium Bacillus thuringiensis, are widely used as biopesticides in transgenic organisms that express Cry1Ac toxin. tCry1Ac and pCry1Ac have been recognized as immunogens, pCry1Ac is a highly effective adjuvant capable of enhancing immune protection against infections. This is due to its ability to stimulate immune response by activating leukocyte cells, especially macrophages, whose role in the lungs is to maintain homeostasis and keep the lungs free of foreign particles. Because of their inflammatory effects like LPS, their ability to activate macrophages through MAPK pathways, and their adjuvant potential, it´s important to evaluate the effect of these proteins on lung tissue. LPS has been used as a positive control in murine models of ARDS via the intranasal route. Since both LPS and Cry1Ac proteins show similar inflammatory effects, it´s important to determine whether Cry1Ac can induce ARDS when administered intranasally. Our results indicate that acute intranasal administration of tCry1Ac and pCry1Ac in a single dose does not produce inflammation at the same intensity as LPS. After 24 hours, neutrophil recruitment, alveolar macrophages, histopathological changes, and the production of pro-inflammatory cytokines were evaluated. LPS showed statistically significant recruitment of neutrophil populations, severe lung tissue damage, and didn't induce an increase in pro-inflammatory cytokines. On the other hand, both Cry1Ac proteins didn´t induce statistically significant neutrophil recruitment, although a trend toward increased populations was observed. They caused mild-moderate changes in lung tissue and likewise did not induce the production of pro-inflammatory cytokines. The absence of cytokine production with LPS may have been due to using a lower concentration than commonly reported. However, the concentration used has been previously employed. It is possible that evaluating the response at 48 or 72 hours may reveal changes in cytokines, increased cell recruitment, and severe histopathological alterations.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Bovine Viral Diarrhea Virus induces activation of IFI16 in bovine macrophages.

Gallegos Rodarte, Claudia Patricia [1]; Benítez Guzmán, Alejandro [1].

[1]Departamento de Microbiología e Inmunología, Facultad de Medicina Veterinaria y Zootecnia,
Universidad Nacional Autónoma de México.

E-mail: vetrodarte@gmail.com

Bovine viral diarrhea is a disease that causes significant economic losses in cattle due to its diverse clinical manifestation and its implication in the bovine respiratory complex. This disease is caused by the Bovine Viral Diarrhea Virus (BVDV) that is classified into two genotypes and two biotypes, depending on the effects they produce in in-vitro cultured cells, the cytopathic biotypes generate vacuolar lesions and cell death and the non-cytopathic biotypes has no apparent effects in cultured cells. BVDV infects macrophages and triggers differential expression of proinflammatory cytokines related to the biotype. In previous research made by our research group, we reported the caspase 1 implication in IL-1β secretion and a possible relation with viral replication during BVDVcp NADL infection in bovine macrophages. To evaluated for the first time the participation of IFI16 inflammasome in IL-1β secretion during infection with the cytophatic and non-cytophatic biotype in bovine macrophages, we evaluated the IFI16 activation by confocal microscopy with immunofluorecence, observing a colocalization between IFI16 and ASC, indicating a posible activation during BVDV infection in bovine macrophages, we also used the inhibitor A151 which is a competitive inhibitor of the IFI16 molecule finding a decreased in IL-1β secretion from 248 pg/mL to 128.3 pg/mL when we infected the macrophages with the non-cytophatic and a decreased from 4138.1pg/mL to 874.4 pg/mL with the cytophatic infection. We also evaluated the non-cytophatic titer at 72 hours post infection with A151 treatment finding a decrease in viral titer indicating a possible participation of IFI16 inflammasome during viral replication. Our results could suggest a participation of IFI16 inflammasome during BVDV infection in bovine macrophages.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

CXCR4 Upregulation by Soluble Factors Enhances HIV-1 Syncytia in Memory T Cells

Gamboa-Meraz, Uriel Alejandro; Ruelas-Galindo, Isabel [2];
Huerta-Hernández, Leonor [1].

[1]Instituto de Investigaciones Biomédicas, Departamento de Inmunología, Ciudad de México. México. [2]Instituto de Biotecnología, Chamilpa, Morelos, México.

E-mail: agamboa.meraz@gmail.com

According to the World Health Organization (WHO), approximately 20,000 new cases of HIV-1 infection are reported annually in Mexico. The progression to AIDS is associated with the emergence of X4 and X4R5 viral variants, which use the CXCR4 coreceptor alone or in combination with CCR5, respectively. In contrast, the asymptomatic phase is characterized by the predominance of R5 variants, which use only CCR5. In 2020, it was shown that increased levels of immune activation markers in peripheral blood precede the emergence of X4 and X4R5 variants. In our laboratory, we demonstrated that cytokines and chemokines released by activated immune cells induce CXCR4 upregulation on resting memory CD4+ T cells, without affecting the expression of other surface markers such as CD3, CCR5, CD54, or CD25. Considering that resting memory CD4+ T cells are a major reservoir for latent HIV-1, this finding suggests a mechanism by which inflammation may increase their susceptibility to infection. In the present study, we confirm that cytokine and chemokine induced CXCR4 upregulation and enhances the ability of these cells to fuse with cells expressing the HIV-1 envelope glycoprotein (Env). This fusion results in the formation of large syncytia containing more than 40 nuclei, a process known to facilitate viral replication and spread. Our results suggest that immune mediators present in an inflammatory microenvironment may enhance the permissiveness of resting memory CD4+ T cells to HIV-1 X4 variants by increasing CXCR4 expression. This mechanism could contribute to the progressive deterioration of the immune response observed during AIDS.

TRABAJO
NO
PRESENTADO

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad Tumoral**

Perinatal di-2-ethylhexyl-phthalate (DEHP) Exposure Increases Breast Cancer Metastasis to the Lung and Liver

Claudia A Garay Canales[1], Carmen T Gómez de Leon[1], Karen E. Nava Castro[2], Armando Pérez Torres[3] & Jorge Morales Montor[1]

[1] Immuniology department, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, Mexico city, MEXICO [2] Grupo de Biología y Química Atmosférica, Instituto de Ciencias de la Atmósfera y Cambio Climático, Universidad Nacional Autónoma de México, Mexico city, MEXICO [3]Facultad de Medicina, Universidad Nacional Autónoma de México, Mexico city, MEXICO

* jmontor66@iibiomedicas.unam.mx

Plastic pollution is a global challenge that is growing exponentially due to the excessive production of single-use products, driven by their low cost, malleability, and durability. Phthalates are chemical compounds known as endocrine disruptors. They are found in everyday products, from cosmetics to food packaging to single-use bags, and they exert their action by modulating various endocrine pathways. These plasticizers do not bind covalently to the plastic matrix, allowing them to easily leach out and cause high human exposure, with particularly harmful effects during critical periods of development, such as the perinatal stage. The chronic effects of DEHP on reproduction have been described, but the effect on the immune response when administered at critical stages of development has not been evaluated, especially its role in the development of mammary tumors. We decided to analyze the effect of perinatal administration of DEHP exposure in pregnant Balb/C mice. DEHP was provided in their drinking water. At weaning, we induced tumors using the 4T1 tumor line. We also evaluated the immune response, metastasis, and tumor microenvironment in a mammary cancer model using 4T1 cells inoculated into female offspring upon reaching sexual maturity. In these animals, we observed that the tumor microenvironment was different in animals exposed to DEHP, with a greater presence of neutrophils, macrophages, and mast cells. Importantly, there were also increased areas of lung metastasis in animals treated with DEHP, and particularly in the liver. Metastasis Cand the microenvironment showed a greater presence of tumor cells and immune cell infiltrates. Cytokines IL-1β, IFN-γ, TNF-α, CCL2, IL-6, IL-10, and particularly IL-5 were elevated compared to the control group. In conclusion, perinatal exposure to DEHP in pregnant mice modulates cellular and molecular components, resulting in increased susceptibility to developing breast cancer and metastasis in adulthood.

Keywords: breast cancer, DEHP, environmental pollutants, metastasis, murine model, tumor microenvironment.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Immunophenotype characterization of T-cell populations in a case of an orphan form of ichthyosis

García-Cruz, Axel [1 2]; Cortés-Hernández, Arimelek [1 2];
Olguin-Alor, Roxana [2]; Soldevila-Melgarejo, Gloria [1 2].

[1]Department of Immunology, Biomedical Research Institute, UNAM. [2]National Laboratory of Flow Cytometry, Biomedical Research Institute, UNAM.

E-mail: axel@iibiomedicas.unam.mx

Autosomal recessive congenital ichthyosis (ARCI) is a heterogeneous group of rare skin disorders (1:100,000), among which congenital ichthyosiform erythroderma (CIE) is one of the most prevalent subtypes. CIE is caused by mutations in genes essential for skin barrier formation and is characterized by scaling, widespread erythema, and hyperkeratosis. Although Th17-skewed immune profiles have been observed in CIE patients aged 10–45, data on early life are lacking. Here, we present a comprehensive phenotypic characterization of peripheral immune cells, with particular emphasis on conventional (Tconv) and regulatory (Treg) T-cell subsets, in an eleven-month-old CIE patient. Our findings revealed an expansion of a Th1/17 population ($CCR4^{+}CCR6^{+}CXCR3^{+}$) within the central memory (TCM) Tconv compartment compared to an age-matched control. Interestingly, we also observed a skew toward a Th1/17-like phenotype specifically in the TCM Treg subset ($CD25^{+}CD127^{-}$), while other memory Tconv and Treg subsets were comparable between the CIE and control groups. Analysis of additional leukocyte populations, including monocytes, dendritic cells , natural killer cells, granulocytes, B cells, and $CD8^{+}$ T cells, revealed no significant differences between the CIE patient and the controls. These findings suggest that an imbalance between the central memory Th1/17 and Treg compartments may contribute to the pathogenesis and severity of CIE. Longitudinal monitoring of these T-cell subsets will help determine their role in disease progression and may inform future therapies targeting the immune system. Given the current lack of effective treatments—restricted to topical emollients and oral retinoids with limited efficacy and significant adverse effects—our study supports the need to explore new immunomodulatory approaches for ARCI.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Monocytes from type 2 diabetic patients show metabolic and immune dysfunction

García-Gómez, Oliver Jair [1]; Jalomo-Urrutia, Ana Sofía [1]; Rosas-Taraco, Adrián Geovanni [1]; Salinas-Carmona, Mario César [1]; López-López, Nallely [1]; Castro-Corona, María de los Ángeles [1]; Mejía-Torres, Manuel [1].

[1]Universidad Autónoma de Nuevo León, Servicio y Departamento de Inmunología de la Facultad de Medicina, Monterrey, Nuevo León, México.

E-mail: oliverjgg22@gmail.com

Type 2 diabetes (T2D) is a metabolic disorder characterized by increased insulin resistance, insulin secretion deficiency or a combination of both. As a chronic disease it tends to increase the risk of vascular complications. A major component of these pathologies derives from a state of chronic inflammation, involving monocytes, which mediate the release of pro-inflammatory cytokines. Differential gene expression analysis is a widely used bioinformatics tool for transcriptomic studies, which allows the evaluation of the transcriptional status, and the regulatory transcriptional networks associated with cell function. The aim of the following study is to identify gene signatures associated to the pathophysiology of type 2 diabetes, using RNA-sequencing (RNA-seq) data analysis techniques from datasets obtained from GEO platform. The transcriptomic profiles from monocytes of T2D patients and control subjects were analyzed using the package DESeq2, and differentially expressed genes (DEG) were identified using a false discovery rate (FDR) < 0.05 and log2 fold-change threshold >0.5. Functional enrichment analysis was performed using KEGG, gene ontology (GO) and GSEA analysis. Our strategy identified 2659 upregulated and 3884 downregulated genes. Upregulated genes were enriched for glycolytic pathways, oxidative phosphorylation, and fatty acid oxidation. Downregulated genes included signaling pathways for NOD-like receptors, chemokines, inflammatory cytokines, NF-kB, and Toll-like receptors. In conclusion, these findings suggest a close relationship between metabolic alterations and immune dysfunction in monocytes from T2D patients.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Comprehensive RNA Sequencing Analysis of CD43 (Sialophorin) Expression in Human Cancers

García-González, Carlos [1 3]; Pérez-García, Erick [2 3];
Chipres-Naranjo, Luis Eduardo [3]; Ramírez-Vilchis, Gabriel [1 3];
Rosenstein-, Yvonne [3].

[1]Licenciatura en Ciencias Genómicas, Universidad Nacional Autónoma de México, Cuernavaca, Morelos, México. [2]Posgrado de Ciencias Bioquímicas, Universidad Nacional Autónoma de México, Cuernavaca, Morelos, México. [3]Instituto de Biotecnología, Universidad Nacional Autónoma de México, Cuernavaca, Morelos, México.

E-mail: carlosgg@lcg.unam.mx

CD43 is a type I transmembrane sialomucin expressed on hematopoietic and immune cells. Under physiological conditions, it regulates essential functions such as migration, adhesion, activation, survival, and pathogen recognition, contributing to immune, inflammatory, and oncological responses. In neoplastic contexts, CD43 has been linked to tumor progression by promoting the proliferation, migration, and survival of malignant cells. Its intracellular domain can activate pro-oncogenic genes, contributing to cancer development and progression. To assess the relevance of CD43 across human cancers, a transcriptomic analysis was conducted using RNA-seq data from 1,017 transcriptomes from 25 primary tumor types available in The Cancer Genome Atlas (TCGA). Raw reads were aligned to the human reference genome GRCh38 using STAR, and gene quantification was performed with FeatureCounts. Expression patterns and differential expression across tumor types were performed. Tumor stratification was visualized using t-distributed Stochastic Neighbor Embedding (t-SNE), revealing that hematologic malignancies formed distinct clusters, with high CD43 expression. Preliminary results showed variable, but consistently elevated, CD43 expression in leukemias and lymphomas, particularly in AML, CML, T-cell ALL, and B-cell ALL. These findings align with its immune cell-specific expression and its role in oncogenesis. This study provides a comprehensive transcriptomic overview of CD43 across various cancers, supporting its potential as a biomarker and therapeutic target, particularly in hematologic malignancies. Future steps include integrating these data with immune infiltration profiles, cell death signatures, and survival clinical outcomes to evaluate the functional and prognostic significance of CD43.

Supported by PAPIIT/UNAM, grant # IN222523

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Evaluation of fibrosis biomarkers in patients with Metabolic Dysfunction-Associated Steatotic Liver Disease

García-González, María Fernanda [1]; Priego-Parra, Bryan Adrián [2]; Cano-Contreras, Ana [2]; Remes-Troche, José María [2]; Hernández-Flores, Karina Guadalupe [1]; Vivanco-Cid, Héctor [1].

[1]Instituto de Investigaciones Medico-Biológicas UV, Laboratorio Multidisciplinario en Ciencias Biomédicas, Veracruz, 91700, México. [2]Instituto de Investigaciones Medico-Biológicas UV, Laboratorio de Motidilidad Gastrointestinal y Fisiología Digestiva, Veracruz, 91700, México.

E-mail: mafergg2@gmail.com

TRABAJO NO PRESENTADO

Metabolic dysfunction-associated steatotic liver disease (MASLD) has emerged as a major global health issue due to its high prevalence and progression to advanced hepatic fibrosis, cirrhosis and even to hepatocellular carcinoma. An early identification of fibrosis stage in MASLD remains challenging, relying primarily on invasive liver biopsy. Consequently, non-invasive biomarkers are urgently needed to improve clinical outcome. In our study, we evaluated plasma biomarkers in a group of 100 subjects (91 MASLD patients and 9 healthy subjects), all the biomarkers, indicative of hepatic fibrosis and autoantibodies in MASLD. We analyzed in the plasma samples, the levels of extracellular matrix proteins (laminin, type IV collagen, hyaluronic acid and cholylglycine), as well as autoantibodies (antinuclear antibodies or ANA, and antimitochondrial antibodies or AMA-M2), using chemiluminescence immunoassays (CLIA). Statistical analyses included Mann-Whitney U and Kruskal-Wallis tests for group comparisons, along with Spearman correlations to explore relationships between biomarkers and clinical parameters like hepatic stiffness measures by elastography and FIB-4 scores. Significant elevations in biomarker concentrations were found in MASLD group, correlating positively with advanced fibrosis stages and inflammatory states. Notably, laminin and hyaluronic acid displayed substantial diagnostic accuracy, confirming their utility in assessing hepatic fibrosis severity. These findings underscore the translational relevance of biomolecules as potential non-invasive biomarkers for early fibrosis detection in MASLD. Implementing these biomarkers clinically could facilitate timely therapeutic interventions, reducing the morbidity and mortality associated with advanced liver disease.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología Veterinaria y Evolutiva**

Serological Prevalence of Orthoflaviviruses in Wild Birds and Rodents from the Yucatán Peninsula

Montserrat Elemi García Hernández [1], Ana Laura Vigueras Galván [1], Benjamín Roche [1 2], Sergio Linares Caballero [1], Gerardo Susán Azpiri 3, Alicia Lizeth Alemán López 1, Rosa Elena Sarmiento Silva [1], Audrey Arnal[* 1 2]

[1] Departamento de Microbiología e Inmunología, Facultad de Medicina Veterinaria y Zootecnia, Universidad Nacional Autónoma de México (UNAM), Ciudad de México 04510, Mexico. [2] MIVEGEC, Université de Montpellier, Institut de Recherche pour le Développement (IRD), CNRS, 34394 Montpellier, France [3] Departamento de Etología, Fauna Silvestre y Animales de Laboratorio, Facultad de Medicina Veterinaria y Zootecnia, Universidad Nacional Autónoma de México (UNAM), Mexico.

* E-mail: Elemi.gh@gmail.com

Orthoflaviviruses are arthropod-borne viruses with zoonotic potential that circulate among a wide range of vertebrate hosts. Birds, especially migratory species, have been proposed as key amplifying hosts in the dissemination and establishment of these viruses, while rodents may act as sentinel or reservoir species. In this study, we evaluated the serological prevalence of orthoflaviviruses in wild birds and rodents from the Yucatán Peninsula, Mexico. Samples were collected from 2022 to 2023 across 12 locations in Campeche, Yucatán and Quintana Roo. A total of 891 bird blood samples and 236 rodent blood samples were preserved on Nobuto strips. Serum was extracted and tested using a commercial competitive ELISA (ID Screen® Flavivirus Competition) to detect anti-pr-E antibodies. Serological evidence of previous orthoflavivirus exposure was found in 55 birds (6.2%) and 9 rodents (3.8%). The low seroprevalence detected, particularly among birds, raises questions about the current circulation dynamics of orthoflaviviruses in the region, as well as the immunological vulnerability of wildlife populations. These findings show the importance of continuous ecological surveillance to better understand exposure patterns and to anticipate potential zoonotic spillover events in regions of high biodiversity and migratory flow such as the Yucatán Peninsula. This research was funded by the SEIHTI project PRONAII Virología 303002 and the French Institut de Recherche pour le Développement (IRD).

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Impaired expression of autophagic protein ATG16L1 attenuates intestinal pathology evoked by ZIKV

García-Huerta, Eduardo [1]; Bustos -Arriaga, José [2]; Castro-Jiménez, Karen [2]; González-González, Marisol [1]; Vannan-, Danielle [3]; Eksteen-, Bertus [4]; Reyes-Hernández, José [1].

[1]Laboratorio de Inmunología Experimental y Regulación de la Inflamación Hepato-Intestinal, UBIMED, FES Iztacala, UNAM, Tlalnepantla de Baz, Estado de México, México. . [2]Laboratorio de Biología Molecular e Inmunología de Arbovirus, UBIMED, FES Iztacala, UNAM, Tlalnepantla de Baz, Estado de México, México. . [3]Boston Scientific Corporation, Urology Division, 200 Boston Scientific Way, Marlborough, MA, USA. . [4]Aspen Woods Clinic, Calgary, AB, Canada.

E-mail: eduardo.gh1410@gmail.com

Zika virus (ZIKV) is an arbovirus member of Flaviviridae family and is transmitted through the bite of female mosquitoes from Aedes genus. Thus, ZIKV infection spreads in its host, which poses the risk to affect several organs not only the CNS. For our particular interest, it has been reported that ZIKV infection evokes gastrointestinal (GI) symptoms in both pediatric and adult patients. Therefore, gastrointestinal involvement during ZIKV infection is an additional component of the complex disease outcome in this infection. ZIKV exhibits several strategies to replicate within its host cell, autophagy is one of them. Amongst others, ATG16L1 has been shown to be involved in ZIKV-elicited pathology. ZIKV protease complex formed by NS-NS3, selectively degraded ATG16L1 and indeed, mutant mice unable to normally express ATG16L1 were more resistant to placental ZIKV infection and vertical transmission. In contrast, the participation of ATG16L1 in gastrointestinal ZIKV-related infection has not been explored. Herein, we explored the possibility of finding gastrointestinal pathology during mouse intraperitoneal (ip.) and subcutaneous (sc.) ZIKV infection with both reference and clinical isolates. Our results show decreased survival when the ZIKV is delivered via sc. in WT and ATG16L1 hypomorphic mice (ATG16L1HM) at 10 days post-infection. On the other hand, GI signs evoked by ip. inoculation in WT mice were severe, however, ATG16L1HM mice showed enhanced resistance. Additionally, a decrease in lymphocyte population and an increase in granulocytes population was observed in WT but not in ATG16L1HM mice. Our results suggest a potential protect role for ATG16L1 in ZIKV infection. Eduardo García received a postdoctoral fellowship from DGAPA, UNAM.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Cell-permeable Bax BH3 Peptide induces Apoptosis and Chemosensitization in Lymphoma cells

García -Nava, Yenny Elizabeth [1 2]; Juárez -Hernández, Uriel [1 2]; Hernández -Cueto, Daniel Dimitri [1 3]; Magaño -Bocanegra, Kevin Jorge [1 2]; González -Franco, José [1 3]; Flores -Martínez, Luis Fernando [1]; Córdova -Trejo, Adonai [1]; Luria -Pérez, Rosendo [1].

[1]Unit of Investigative Research on Oncological Diseases, Children's Hospital of México Federico Gómez, México City, México. [2]Department of Molecular Biomedicine, Center for Research and Advanced Studies of the National Polytechnic Institute, México City, México.. [3]Escuela Superior de Medicina, Instituto Politécnico Nacional, Ciudad de México, México.
E-mail: yenny.gn97@gmail.com

Non-Hodgkin lymphomas (NHL) are among the leading causes of cancer-related mortality in children and adolescents worldwide. Although conventional treatments have significantly improved survival, 10–20% of patients do not respond, which is associated with poor prognosis due to the lack of alternative therapies. One of the primary mechanisms contributing to relapse is chemoresistance, often associated with the overexpression of antiapoptotic proteins from the Bcl-2 family. Our previous studies have shown that the cell-permeable Bak BH3 peptide can reverse this resistance by inducing apoptosis and sensitizing tumor cells. This study evaluated the ability of the cell-permeable Bax BH3 peptide (AntFBax) to restore apoptosis and promote chemosensitization in a human B-cell non-Hodgkin´s Lymphoma cell line (Ramos). The bioinformatic analysis confirmed the correct folding of the AntFBax peptide, which consists of the BH3 domain of the Bax protein fused to a FLAG epitope and a fusogenic peptide derived from the Antennapedia protein. Western blot confirmed the expression of antiapoptotic (Bcl-XL and Mcl-1) and proapoptotic molecules (Bax and Bak) in Ramos cells. After peptide synthesis, we showed that AntFBax reduced cell viability (MTT assay), increased levels of apoptosis (active caspase-3 assay), and enhanced the cytotoxic effect of vincristine in Ramos cells. Additionally, the cell-permeable AntFBax peptide did not affect peripheral blood mononuclear cells. These findings suggest that permeable Bax BH3 peptides can restore the apoptotic pathway and sensitize NHL cells to chemotherapy, positioning them as a promising therapeutic strategy for patients with refractory disease. Funding: CONACYT CB-2013-01-222446, Fondos Federales (HIM-2015-049 SSA 1217, HIM-2021-056 SSA 1756, HIM-2022-061 SSA1826).

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

Smad7 deletion as a strategy to enhance immune regulation via TGF-β in $CD8^+$ T cells

García-Rasilla, Verónica Yutsil [1]; Zambrano Romero, Jesús Daniel [1]; Contreras Castillo, Eugenio [1]; Ramos Balderas, José Luis [1]; Licona Limón, Paula [1].

[1]Departamento de Biología Celular y del Desarrollo, Instituto de Fisiología Celular, Universidad Nacional Autónoma de México, México City, México.

E-mail: veronicag@ifc.unam.mx

Transforming growth factor-beta (TGF-β) is a pleiotropic cytokine that plays a critical role in immune regulation. In T cells, TGF-β influences thymic development, maintains peripheral homeostasis, and directs differentiation upon activation (Sanjabi *et al*., 2017). Understanding how this pathway is regulated is crucial for designing new immunotherapeutic strategies. $CD8^+$ T lymphocytes are essential for the adaptive immune response, eliminating intracellular pathogens and tumor cells (Turner *et al*., 2021). TGF-β has been shown to regulate their activation, proliferation, differentiation, and effector functions (Chen, 2023). However, the precise molecular mechanisms underlying these effects remain incompletely understood (Thomas & Massagué, 2005; Gunderson *et al*., 2020). Smad7 is an inducible antagonist of TGF-β signaling that can inhibit the pathway at multiple levels. While its role has been described in $CD4^+$ T cell-mediated inflammation and immune regulation (Haupeltshofer *et al*., 2019; Garo *et al*., 2019; Rizzo *et al*., 2014), its function in $CD8^+$ T cells is poorly defined. In our lab, we investigated the impact of conditional Smad7 deletion (Smad7fl/fl-cd4-cre) on $CD8^+$ T cell responses. In a murine melanoma model, Smad7-deficient $CD8^+$ T cells showed reduced tumor infiltration, impaired cytotoxic molecule production, and an increased exhaustion phenotype. Similarly, during acute bacterial infection, these cells exhibited defective expansion, impaired activation, reduced differentiation into short-lived effector cells (SLECs), and diminished effector molecule expression. In vitro, Smad7-deficient $CD8^+$ T cells cultured with TGF-β displayed reduced proliferation, increased apoptosis, and decreased expression of IFN-γ, granzyme B, and perforin. These findings suggest that Smad7 counteracts TGF-β–mediated inhibition in $CD8^+$ T cells and is essential for maintaining their effector functions during immune responses.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Evaluation of T-cell responses induced by AVX/COVID-12 ‘Patria’ COVID-19 vaccine in pigs

García-Valeriano, María Guadalupe, [1 2]; Tepale-Segura, Araceli, [2]; López-Macías, Constantino, [2].

[1]Unidad Interdisciplinaria de Biotecnología, Posgrado en Ciencia y Tecnología de Vacunas y Bioterapéuticos del Instituto Politécnico Nacional México City, México. [2]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México.

E-mail: magvalina@gmail.com

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) continues to cause significant morbidity and mortality, especially among vulnerable populations such as those with comorbidities, immunocompromised patients, and the elderly. Despite global vaccination efforts, distribution remains uneven, particularly in low- and middle-income countries (LMICs), where vaccines often reached the general population months later than in nations with established manufacturing capabilities. This disparity highlights the urgent need for locally produced vaccine platforms. In response, the Mexican company Avimex developed AVX/COVID-12 “Patria,” a Newcastle disease virus (NDV)-vectored vaccine expressing the SARS-CoV-2 spike (S) protein. Patria showed safety, tolerability, and immunogenicity in preclinical studies (mice, hamsters, pigs) and Phase I–III clinical trials in humans, where it was non-inferior to the ChAdOx1 (AstraZeneca) vaccine. It has received regulatory approval for adult use in Mexico. These advances underscore the need to investigate the immunological mechanisms of protection and long-term immunity elicited by this platform. In particular, understanding T-cell–mediated responses is crucial for the continued development of Patria and its future iterations. We evaluated T-cell responses induced by Patria in pigs. Animals were immunized with $1x10^8$ viral particles via intramuscular (IM), intranasal (IN), or combined IM/IN routes. Peripheral blood mononuclear cells (PBMCs) were analyzed post-immunization. Both strategies, with a booster on day 21, induced distinct $CD3^+$ T-cell responses. Strong IFN-γ expression in $CD4^+$, $CD8^+$, and $CD4^+CD8^+$ subsets was observed upon in vitro restimulation at days 7 and 14 post-boost. Heterologous IN/IM immunization increased central memory ($CD8^+CD27^+$) and effector memory ($CD8^+CD27^-$) T-cell subsets, while IM-only immunization mainly promoted effector memory responses from day 21 to 35. In conclusion, Patria vaccination induces durable memory T-cell responses, complementing its humoral immunogenicity. These findings support rational vaccine strategies leveraging immunization routes aligned with pathogen entry pathways to enhance protective efficacy.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Systematic Review and Meta-Analysis of Predictive Biomarkers for Rheumatoid Arthritis

García-Vargas, Juan Ángel III [1]; Rojas-Gutiérrez, Santiago Emilio [1]; Martínez-Canales, Rodolfo [1]; Salinas-Carmona, Mario César [1]; Ruiz-De La Cruz, María Luisa [1]; Macías-Segura, Noé [1].

[1]Universidad Autónoma de Nuevo León, Facultad de Medicina, Departamento de Inmunología, Monterrey, Nuevo León, México.

E-mail: angeljunior2003@outlook.com

Rheumatoid arthritis (RA) is a chronic autoimmune disease preceded by a preclinical phase in genetically or serologically at-risk individuals. Early identification of those who will transition to clinical RA remains a key challenge in precision immunology. This study aimed to evaluate and compare the predictive performance of individual biomarkers and multi-analyte panels for RA development in at-risk populations through a systematic review and meta-analysis. Following PRISMA 2020 guidelines and a PICO framework, we screened studies from 2005 to 2025 evaluating predictive biomarkers in first-degree relatives, seropositive individuals without arthritis, and patients with preclinical RA symptoms. A total of 74 studies were included. Data on odds ratios (OR), hazard ratios (HR), area under the ROC curve (AUC), sensitivity, and specificity were extracted. Forest plots and heatmaps were generated to visualize effect sizes and performance by biomarker type. Individual biomarkers such as ACPA, anti-CarP, and RF showed variable predictive strength (OR range: 1.18–94.1; HR: 1.56–14.86; AUC: 0.65–0.82). Combined panels integrating serological, genetic, and transcriptomic features achieved superior performance (OR up to >132.03, AUC up to 0.935). Proteomic signatures and transcriptomic panels demonstrated high AUCs (0.785–0.935), especially in studies with multi-marker integration. However, confidence intervals were inconsistently reported across studies, limiting quantitative synthesis. Multi-biomarker panels consistently outperform single biomarkers in predicting RA development. ACPA remains a robust marker, but its predictive power increases when combined with genetic and proteomic indicators. The use of transcriptomic and proteomic signatures shows promise for improving early risk stratification. Future research should prioritize standardized reporting, consistent use of confidence intervals, and prospective validation of multibiomarker models across diverse clinical populations. These efforts will be critical to building clinically applicable tools that support early intervention in rheumatoid arthritis.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Multibiomarker Model to Predict Rheumatoid Arthritis Risk in At-Risk Individuals

García-Vargas, Juan Ángel III [1]; Rojas-Gutiérrez, Santiago Emilio [1]; Martínez-Canales, Rodolfo[1]; Ruiz-De la Cruz, María Luisa [1]; Salinas-Carmona, Mario Cesar [1]; Macías-Segura, Noé [1].

[1]Universidad Autónoma de Nuevo León, Facultad de Medicina, Servicio de inmunología, Monterrey, Nuevo León, México.

E-mail: juan.garciavargas@uanl.edu.mx

Rheumatoid arthritis (RA) is a systemic autoimmune disease with a silent preclinical phase. Biomarkers in high-risk individuals, especially first-degree relatives (FDRs), could enable preventive strategies and early monitoring, for these and other diseases. We designed a systematic review aimed to identify molecular biomarkers and build a clinically useful predictive model. A comprehensive search was performed across PubMed, EMBASE, Scopus, Web of Science, Cochrane, Google Scholar, medRxiv, and bioRxiv (2005–2025), following PRISMA 2020 and PICO framework. Human studies reporting quantitative measures as follows: Odds Ratio (OR), Risk Ratio (RR), Area Under the Curve (AUC), and the RA at-risk populations biomarkers. Fifty-three eligible studies were identified. We include the most predictive known biomarkers: Anti-Citrullinated Protein Antibodies (ACPA), Rheumatoid Factor (RF) (AUC = 0.88, OR >10), IL-6 (AUC = 0.75), Anti-14-3-3 eta protein (14-3-3η) (AUC = 0.73), and HLA-DRB1 shared epitope (OR ~3.8). We used the combined model of ACPA, RF, IL-6, 14-3-3η, HLA-DRB1 to achieve an AUC of 0.90, accuracy of 0.86, sensitivity of 0.82, and specificity of 0.91. We used the graphical tool Receiver Operating Characteristic Curve (ROC) curves, radar charts, net reclassification improvement (NRI), and decision curve analysis (DCA) to demonstrate our model superiority over individual biomarkers. We found that the use of systematic metanalysis of previous publications data with the approach used here, resulted very helpful to construct a predictive model to enhance clinical surveillance, early diagnosis, and selection for preventive interventions on RA risk in at-risk individuals. Our approach combining the used RA of biomarkers provides high discriminatory power for assessing risk of developing RA. Additional studies are necessary to support the future use of these bioinformatics tools for other diseases.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Anticancer activity of antimicrobial peptides from Bacteria Lactic-Acid on epithelial cancer cells

Garduño-Espinoza, Maried [1]; Monroy-Torres, Edna Michelle [1]; Reyes -Martínez, Cyntia Denisse[1]; Baltierra -Uribe, Shantal Lizbeth [2]; García-Pérez, Blanca Estela [2]; Hernández -González, Juan Carlos [1]; Castrejón-Jiménez, Nayeli Shantal [1].

[1]Área Académica de Medicina Veterinaria y Zootecnia, Instituto de Ciencias Agropecuarias-Universidad Autónoma del Estado de Hidalgo, Av. Universidad km. 1. Exhacienda de Aquetzalpa A.P. 32, Tulancingo 43600, Hidalgo, México. [2]Departamento de Microbiología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Prolongación de Carpio y Plan de Ayala s/n, Ciudad de México 11340, México.

E-mail: maried.g.0406@gmail.com

Cancer is a disease that causes thousands of deaths globally. According to the WHO, in 2022, there were approximately 20,000,000 new cancer cases worldwide. Lung cancer is the most common and leading cause of death per year (approximately 1.8 million deaths), followed by colon rectal cancer (900,000 deaths) and liver cancer (760,000 deaths, sixth in frequency). Given the growing problems and limitations of conventional treatments, such as chemoresistance and the high rate of tumor recurrence, there is a need to explore therapeutic alternatives, one of which is the bacteriocins from lactic acid bacteria (LAB). Bacteriocins are antimicrobial peptides (AMP) of cationic nature that have been shown to have bioactive properties such as immunomodulators, antimicrobials, biopreservatives, and anticancer activity. The main mechanism of action through the interaction of cationic peptides towards negatively charged cell membranes, a condition that has been described in cancer cells due to changes in their lipid composition due to high concentrations of phosphatidylserine, glycosylated O- mucin, and heparin sulfates. This allows cell death by forming pores in the membrane or activating proapoptotic pathways as the intrinsic pathway of apoptosis, favoring the production of reactive oxygen species (ROS). This study aimed to evaluate the anticancer effect of AMP from LAB on lung (A549) and liver (HepG2) cell lines. Cell monolayers were performed and co-cultured with different concentrations of bacteriocins (0.1, 0.2, 0.45, 0.9, 1.8, 3.6, 7.3 µg/ml), and viability tests and ROS expression was determined. Results showed that bacteriocins induce a decrease in the viability of cancer cells and increase ROS production, reaching levels almost twice at 3.6 and 7.3 µg/mL concentration. This suggests that bacteriocins contribute to cell death by forming pores and activating ROS-mediated mechanisms.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Analysis of -2518 A>G (rs1024611) CCL2 with cardiometabolic variables in rheumatoid arthritis

Garza-García, Leslie; Campos-López, Bertha; Mora-García,
Paulina E; Pesqueda-Cendejas, Karen; Meza-Meza, Mónica R;
De la Cruz-Mosso, Ulises.

[1]Red de Inmunonutrición y Genómica Nutricional en las Enfermedades Autoinmunes; Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud; Universidad de Guadalajara; Guadalajara, Jalisco 44340, México. . [2]Instituto de Neurociencias Traslacionales, Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara 44340, Jalisco, México.

E-mail: lesliegarza30042000@gmail.com

Rheumatoid arthritis (RA) is a chronic inflammatory disease associated with increased cardiometabolic comorbidity. The Chemokine (C-C motif) ligand 2 (CCL2) gene encode the chemokine monocyte chemoattractant protein-1 (MCP-1), which recruits monocytes and amplifies inflammation. Previous studies have suggested that the −2518 A>G (rs1024611) single nucleotide variant (SNV) in CCL2 may influence both RA susceptibility and the intensity of the inflammatory response. This study aimed to determine the association of the −2518 A>G (rs1024611) SNV in CCL2 with RA susceptibility and with clinical, biochemical, and metabolic parameters in Mexican-Mestizo RA patients. This study was conducted in 199 RA patients and 206 control subjects (CS), all females. Genotyping was performed using TaqMan probes by allelic discrimination. Inflammatory markers (ESR, hs-CRP, DAS28), lipid profile (total cholesterol, HDL-C, LDL-C, triglycerides), body composition (BMI, circumferences, lean and fat mass), and cardiometabolic indices (TG/HDL ratio, LAP, waist-to-height ratio, etc.) were assessed. Allelic and genotypic frequencies, genetic models (dominant, recessive), and Hardy–Weinberg equilibrium were calculated. Compared to SC, RA patients showed an inflammatory dyslipidemia profile (lower HDL, higher triglycerides), sarcopenic obesity, and deranged cardiometabolic indices (TG/HDL, LAP, waist-to-height ratio; all $p < 0.001$). No significant association was found between rs1024611 and RA risk (G allele: 51 % in RA patients vs. 49 % in CS; $p = 0.50$). However, carriers of AG+GG genotypes showed significantly higher hs-CRP, CRP/albumin ratio, DAS28 scores, and swollen joint counts during disease flares ($p < 0.01$). According to our findings, RA patients exhibit an adverse cardiometabolic profile. Moreover, the −2518 A>G SNV in CCL2 was not associated with RA susceptibility; nevertheless, the G allele modulates the severity of systemic and clinical inflammation during active RA.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Salmonella typhimurium infection increased the necrosis of diazoxon-exposed mononuclear cells

Girón- Pérez, Daniel Alberto [1]; Covantes-Rosales, Carlos Eduardo [1]; Toledo-Ibarra, Gladys Alejandra [1]; Díaz-Resendiz, Karina Janice Guadalupe [1]; Bueno-Duran, Adela Yolanda [1]; Benítez-Trinidad, Alma Betsaida [1]; Girón-Pérez, Manuel Iván [1].

[1]Universidad Autónoma de Nayarit, secretaria de Investigación Posgrado, LANIIA, Tepic, Nayarit. México.

E-mail: daniel.giron@uan.edu.mx

The Salmonella typhimurium is a Gram-negative bacterium that causes salmonellosis. It has been reported that this bacterium can infect immune system cells and spread throughout the body. This infection process can be influenced by pesticide exposure, and this phenomenon may also cause alterations in cell death. This could be beneficial for the bacterium, as it allows it to infect other cells, evade the immune system, and acquire nutrients. In this study, apoptosis and necrosis were evaluated in human mononuclear cells previously exposed to diazoxon (DXN) (1 µM) and infected with S. typhimurium for 1 h. It was observed that in mononuclear cells exposed only to the pesticide or infected with the bacteria, cell death occurred mainly through apoptosis. However, the combination of both factors led to necrosis, suggesting that the pesticide enabled the activation of other signaling mechanisms. Additionally, actin polymerization activity increases when cells are exposed to bacteria and pesticides, suggesting the activation of mechanisms related to the actin cytoskeleton. Conversely, when colonies exposed to both DXN and S. typhimurium harbored more intracellular bacteria. These findings suggest that diazoxon exposure combined with S. typhimurium infection induces an immunotoxic process in which the predominant mechanism is necrosis. Moreover, the shift from apoptosis to necrosis may impair the host's ability to control bacterial proliferation, facilitating systemic dissemination. The modulation of cell death pathways by pesticide exposure could therefore play a significant role in the pathogenesis of Salmonella infections.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Immunopotentiator “RTLV-1424” Modulates Mesenchymal Stem Cells Immunosuppressive Capacity Over T Cells.

Godínez-Mejía, Laura Angélica [1,2]; López-Macías, Constantino [1];
Montesinos-, Juan José [3]; Pastelín-Ruíz, Rodolfo Eduardo [4];
Lozano-Cisneros, Diego [1]; Pérez-Vergara, Ana Delia [1]; Fajardo-Orduña,
Guadalupe Rosario [3]; Cortés-Morales, Víctor Adrián [1,5].

[1]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México. [2]Facultad de Química, Universidad Nacional Autónoma de México, México City, México. [3]Unidad de Investigación Médica en Enfermedades Oncológicas, UMAE Hospital de Oncología, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México. [4]Unidad de Cirugía, UMAE Traumatología, Ortopedia y Rehabilitación “Dr. Victorio de la Fuente Narvaez, Instituto Mexicano del Seguro Social, México City, México. [5]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México

Corresponding: v.adrian.cortes@gmail.com. E-mail: lagm2731@gmail.com

Mesenchymal stem cells (MSC) are part of the tissue microenvironment. These cells have demonstrated immunosuppressive potential by inhibiting T cell proliferation, promoting the differentiation of regulatory T cells, and expressing PD-L1, which facilitates tumor progression. These cells can acquire a proinflammatory or antiinflammatory phenotype when activated through Toll-like receptors (TLR), where TLR3 is associated with an anti-inflammatory phenotype and TLR4 with a proinflammatory one. Bacteria-derived immunopotentiator agents have the ability to induce an inflammatory phenotype by stimulating TLR in immune cell populations. In our study, we used bone marrow-derived MSC and aimed to evaluate whether the molecule RTLV-1424, derived from Salmonella Typhi, can modulate the immunosuppressive capacity of MSC over phytohemagglutinin (PHA)-stimulated T cells using an in vitro coculture system. Membrane markers, intracellular molecules, and soluble cytokines were assessed using flow cytometry. We found that treatment with RTLV-1424 during 48 hours reduced the ability of MSC to inhibit T cell proliferation (CD3+, CD4+ or CD8+) and the generation of $CD4^{+}CD25^{+}FOXP3^{+}$ regulatory T cells. Surprisingly, this treatment favored the generation of $CD4^{+}IFN\text{-}\gamma^{+}$ and $CD8^{+}IFN\text{-}\gamma^{+}$ T cells, and decreased the concentration of soluble IL-10 in coculture systems. Additionally, the presence of RTLV-1424 decreased PD-L1 expression and promoted TLR4 expression in MSC, which is associated with an inflammatory phenotype in these cells. These results suggest that the immunostimulant RTLV-1424 has the capacity to induce an inflammatory phenotype in MSC, which could be useful in pathologies where enhancing the inflammatory response of the microenvironment is desirable, such as in cancer.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Analysis of gut barrier markers in mice fed high-fat diet plus butyrate

Godínez-Victoria, Marycarmen [1]; Drago-Serrano, María Elisa [2];
Oros-Pantoja, Rigoberto [3]; Bernal-Rojas, Jazmín [3]; Tamay-Cach,
Feliciano [1]; Aguilar-González, Joab Elí [1]; Martínez-Gopar, Pablo
Eliasib [2]; Guzmán-Mejía, Fabiola [2].

[1]Sección de Estudios de Posgrado e Investigación, Escuela Superior de Medicina, Instituto Politécnico Nacional, CP 11340 CdMx México. [2]Laboratorio de Homeostasis Intestinal, Depto. Sistemas Biológicos UAM-Xochimilco, Calzada del Hueso No. 1100 CP 04960 CdMx México. [3]3Laboratorio de Neuroinmunoendocrinología Facultad de Medicina, Universidad Autónoma del Estado de México (UAEMEX) CP 58103 Toluca, México.

E-mail: maric_27@yahoo.com

Gut-barrier function entails selective transport through the intestinal epithelium of harmless molecules present in the lumen to the inner milieu, and at the same time, hampering the entry of noxious agents. Gut-barrier function is under the control of components with divergent distribution in the proximal small gut and colon including the i) mucus-layer containing mucopolysaccharides, ii) epithelial-cell monolayer joined adjacently by protein-complexes that regulate the paracellular-permeability which can be measured by quantifying the luminal levels of serum albumin and iii) intestinal-immunity effectors like immunoglobulin A (IgA). Gut-barrier function can be disrupted by high-fat-diet while the presumable role of butyrate in restoring it is not fully known. This study aimed to analyze the impact of butyrate in high-fat-diet fed mice on some markers of the gut-barrier in the proximal small intestine and colon. For that, eight-week-old male CD1 mice were divided in groups of six: i) standard-diet plus water control (Ctrl) ii) high-fat-diet (HFD) iii) standard-diet plus butyrate (But) and iv) HFD plus butyrate (HFD+But). After four-weeks body weight of mice was computed, and then mice were euthanized to collect intestinal secretions to analyze albumin and IgA by ELISA and intestinal segments to analyze mucopolysaccharide content by alcian blue test. One way ANOVA and the post-hoc Tukey test were used for data comparison and significant differences were set-up at $p\leq 0.05$ value. Body weight was either greater in HFD group versus the Ctrl and HFD+But groups or lower in HFD+But versus But group (P=0.01 all cases). Albumin concentration in both segments was greater in HFD versus the Ctrl and HFD+But (P=0.001) and lower in HFD+But versus But (P=0.01 proximal; P=0.001 colon) and But versus Ctrl (P=0.05 proximal; P=0.001 colon). Additional significant differences were unseen. In conclusion, butyrate may counteract the effect of HFD on decreasing the permeability in both intestinal segments.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Discovery and characterization of two anti-PD-1 antibodies with a unique PD-1 binding

Gómez-Castellano, Keyla María [1 2]; Montes-Luna, Alejandra [1 2];
Carballo-Uicab, Gregorio [1 2]; Ramírez-Villedas, Frida Daniela [1 2];
Elizarrarás-Rodríguez, Luis Javier [1 2]; Vázquez-Leiva, Said [1 2];
Holland, Ben [3]; Della, Cristina P. [3]; Barrera-Saldaña, Hugo Alberto [4];
Pérez-Tapia, Sonia Mayra [1 2 5]; Almagro, Juan Carlos [1 6].

[1]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Prolongación de Carpio y Plan de Ayala S/N, Colonia Santo Tomás, Alcaldía Miguel Hidalgo, CDMX 11340, México. [2]Laboratorio Nacional Para Servicios Especializados de Investigación, Desarrollo e Innovación (I + D + I) Para Farmoquímicos y Biotecnológicos, LANSEIDI-FarBiotec-CONACyT, Prolongación de Carpio y Plan de Ayala S/N, Colonia Santo Tomás, Alcaldía Miguel Hidalgo, CDMX 11340, México. [3]Antiverse Ltd., 34 Roath Court Road, Cardiff, Wales, CF24 3SD. [4]Laboratorios Columbia SA de CV. Dirección de Investigación Científica. Panzacola 62, Colonia Villa Coyoacán. Alcaldía Coyoacán. CDMX. C.P. 04000. [5]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Prolongación de Carpio y Plan de Ayala S/N, Colonia Santo Tomás, Alcaldía Miguel Hidalgo, CDMX 11340, México. [6]GlobalBio, Inc., 320 Concord Ave, Cambridge, MA 02138, USA.

E-mail: Keyla.gomez@udibi.com.mx

Targeting checkpoint inhibitors is an effective therapy for treating cancer, with human programmed cell death protein 1 (hPD-1) being one of the most successful targets for developing antibody-based drugs. This work isolated a panel of anti-PD-1 scFvs with different binding and functional profiles from a fully synthetic human phage display library. Conversion of the best clone to hIgG1LALA and hIgG4PE, called UDIZ-007 and UDIZ-008, respectively, resulted in antibodies that effectively blocked the PD-1:PD-L1/L2 interaction, were selective - didn't cross-reacted with CD28 receptor family members. Administration of UDIZ-007 or UDIZ-008 at 10 mg/kg, every three days in six doses, eradicated MC38-hPD-L1 colon tumors in B-hPD-1 transgenic mice for hPD-1 at day 17, with no relapse until the end of the study at day 56. Importantly, these antibodies bind hPD-1 in a unique region when compared to the anti-PD-1 antibodies of known structure, which might have an impact on novel oncology indications as a standalone therapy or in combination with currently approved anti-PD-1 therapeutic antibodies. Therefore, UDIZ-007 and UDIZ-008 seem to be promising candidates for the development of antibody-based drugs targeting checkpoint inhibitors as a threat to cancer.

Keywords: Programmed cell death protein 1; Anti-PD-1 antibody; Antitumor activity; Cancer Immunotherapy.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación Inmune**

Vitiligo: association between physical activity, strength, and body composition with oxidative stress

Jesús Ricardo Gómez Frías, Liliana Araceli Enríquez del castillo[1], Sandra reza López[2], Jorge Octavio Acosta Montes[3], Claudia Esther Carrasco Legleu[1], Estefanía Quintana Mendías[1]

[1]Facultad de Ciencias de la Cultura Física, Universidad Autónoma de Chihuahua. [2]Facultad de Medicina y Ciencias Biomédicas, Universidad Autónoma de Chihuahua. [3]Facultad de Enfermería y Nutriología, Universidad Autónoma de Chihuahua.

E-mail: ana.vigueras09@gmail.com

Vitiligo is an autoimmune disease characterized by physiological alterations that exacerbate oxidative stress. Factors such as physical activity, strength levels, and body composition involve mechanisms that may modulate this redox balance. This study analyzed the relationship between physical activity, strength, and body composition with oxidative stress in patients with vitiligo. 39 patients were recruited in Chihuahua city. Physical activity was assessed using the International Physical Activity Questionnaire, grip strength and body composition evaluated through dynamometer and bioelectrical impedance analysis, respectively. Malondialdehyde levels and total antioxidant capacity were also analyzed. A linear regression analysis was performed to establish the relationship between these oxidative stress markers and levels of physical activity, strength, and body composition. A positive relationship was found between malondialdehyde levels and physical activity measured by METS ($\boldsymbol{\beta}$=0.003, 95% CI: 0.000 – 0.005, p=0.048), as well as an inverse relationship with left-hand grip strength ($\boldsymbol{\beta}$=–0.006, 95% CI: –0.011 – –0.001, p=0.029). Additionally, a positive relationship was observed between grip strength and total antioxidant capacity in men for the right hand ($\boldsymbol{\beta}$=0.64, 95% CI: 0.19 – 1.09, p=0.010) and the left hand ($\boldsymbol{\beta}$=0.58, 95% CI: 0.03 – 1.14, p=0.014), and body fat in women ($\boldsymbol{\beta}$=0.30, 95% CI: 0.03 – 0.56, p=0.03). The findings suggest a relationship between physical activity and the modulation of oxidative stress in patients with vitiligo, as well as a potential to improve the course of the disease through these mechanisms.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Molecular mimicry between gut microbiome and ghrelin: in-silico exploration of anti-GRL antibodies-origin

Gómez-Medina, Daniela [1]; Reyes-Castillo, Zyanya [1];
Magaña-Cuevas, Elsa Patricia [2]; Vázquez-Solorzano, Rafael [1];
Gómez-Márquez, Carolina Elizabeth [2]; Juárez-Valderrama, Hugo
Adrián [1]; Mendoza-Hernández, Victor Ignacio [1]; Enciso-Ramírez,
Mayra Alejandra [1].

[1]Laboratorio de Biomedicina y Biotecnología para la Salud, Centro Universitario del Sur, Universidad de Guadalajara, Ciudad Guzmán, Jalisco, México. [2]Laboratorio de Innovación Biodigital, Centro Universitario de Ciencias Exactas e Ingenierías, Universidad de Guadalajara, Guadalajara, Jalisco, México.

E-mail: daniela.gomez9171@alumnos.udg.mx

The gut microbiota (GM) plays a key immunomodulatory role and has been implicated in the production of low-affinity antibodies directed against metabolic hormones such as ghrelin (anti-GRL), through the phenomenon of molecular mimicry. Anti-GRL antibodies are associated with behavioral disorders and hyperphagic obesity; however, the phenomenon of molecular mimicry has not been thoroughly explored at the sequential and structural level, especially considering the exponential growth of gut microbiome databases. This research aimed to identify microorganisms and proteins from the human GM that present molecular mimicry with the hormone ghrelin, through sequence and structural alignments using bioinformatics tools. A total of 224 bacterial and 125 fungal species from the GM were compiled from scientific literature and NCBI bioprojects. Their protein sequences were retrieved via UniProt to create databases. Sequence alignments were performed using Local BLAST, and structural alignments using CCP4MG. The workflow was automated through a Python-developed pipeline. Among the analyzed microorganisms, 441 mimetic proteins were identified in bacteria (95 shared among two or more species) and 324 in fungi (58 shared among species). In bacteria, most mimetic proteins were related to energy, transport, and metabolism; while in fungi, they were mainly associated with gene regulation, post-translational modifications, lipid and sugar processing, and energy generation. Structural-level analyses of mimetic sequences are currently ongoing. These results support the hypothesis that molecular mimicry plays a role in the modulation of anti-GRL antibodies and provide an updated and robust analysis identifying new microorganisms and mimetic proteins with GRL hormone.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Identification of Cry1Ac Toxin Receptors in Plasma Membranes of Macrophages

Gómez-Méndez, Eduardo [1]; Raya Malvaez, Bryan [1]; Ilhuicatzi
Alvarado, Damaris [1]; Moreno-Fierros, Leticia [1].

[1]Facultad de Estudios Superiores Iztacala UNAM.

E-mail: egome22@yahoo.com

Cry proteins are toxins with insecticidal properties produced by Bacillus thuringiensis bacteria. This is why they are used to control pests in the agronomy industry. The proteins Cry that targets Lepidoptera are known as Cry1Ac. This is obtained in crystal form; it's solubilized and is obtained like protoxin which has a molecular weight of 130 kDa. To obtain Cry1Ac toxin (tCry1Ac), protoxin must undergo proteolysis, reducing its weight to 65 kDa. Due to their high specificity, tCry1Ac proteins are not toxic to humans. However, tCry1Ac has been shown to have immunostimulatory properties, like the ability to activate macrophages and dendritic cells, inducing proinflammatory cytokines and costimulatory molecules; making it a potential vaccine adjuvant. Although the immune response induced by tCry1Ac is known, the specific receptors in macrophages remain unidentified. In previous attempts, protein candidates to receptor precipitations using tCry1Ac conjugated to Sepharose beads were performed, revealing numerous proteins and only demonstrating the partial involvement of HSP70. To address this, we developed a new methodology and more specific. Concentrated plasma membrane proteins were obtained from murine macrophages of the RAW 264.7 cell line and incubated with biotin-conjugated tCry1Ac. Afterward, we performed a positive selection using a Biotin/Streptavidin assay with magnetic bead columns (MACs) to purify potential receptors. The resulting proteins were separated by electrophoresis on a gel stained with Coomassie blue and sent for sequencing at IBT-UNAM using MALDI-TOF to identify possible receptor candidates. We performed a ligand blot assay to confirm that the precipitated bands corresponded to tCry1Ac receptors and conducted flow cytometry assays to verify protein binding at different times and concentrations. The procedure was standardized, and the best receptor candidates identified so far are HSP70 and Vimentin.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Role of the Complement System in Inflammation and Autoimmune Diseases: Perspectives for Future Therapies

Gómez-Montañez, Emiliano; Rojas-Salazar, Yareli Lizbeth;
Rojas-Salazar, Jorge Gustavo.

[1]Instituto Nacional de Medicina Genómica, Ciudad de México, México. [2]Instituto Nacional de Medicina Genómica, Ciudad de México, México. [3]Universidad Autónoma de Ciudad Juárez, Ciudad Juárez, Chihuahua, México.

E-mail: emgomez177566@gmail.com

The complement system is a central component of innate immunity, consisting of over 30 proteins that, once activated through the classical, alternative, or lectin pathways, generate inflammatory mediators and membrane attack complexes; and while crucial for eliminating pathogens, excessive or uncontrolled complement activation can lead to tissue damage and contribute to the pathogenesis of autoimmune diseases such as systemic lupus erythematosus (SLE) and myasthenia gravis.

This study aims to review the dual role of the complement system in host defense and autoimmunity, and to explore current and future therapeutic strategies aimed at modulating complement activity in a targeted and safe manner. A literature review was conducted using recent publications from peer-reviewed databases, focusing on the mechanisms of complement activation, its role in autoimmune diseases, and emerging therapeutic approaches targeting complement components or regulatory proteins.

Research has identified several key proteins involved in complement regulation, such as factor H and C5; in which therapies like eculizumab, a monoclonal antibody that inhibits complement component C5, have shown clinical benefit in conditions like paroxysmal nocturnal hemoglobinuria and myasthenia gravis; and ongoing studies aim to identify novel targets and biomarkers to enhance diagnosis, predict disease activity, and develop safer interventions that modulate the complement cascade without impairing antimicrobial defense.

The complement system is both a protector and a potential aggressor in immune responses. Advancing our understanding of its regulation and dysfunction opens new therapeutic avenues for autoimmune diseases; and in the future, therapies will likely focus on precision modulation of complement activity, offering effective treatment while minimizing immune compromise.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

The Role of Dendritic Cells in Antigen Presentation and Immune Tolerance

Gómez-Montañez, Emiliano; Rojas-Salazar, Yareli Lizbeth;
Rojas-Salazar, Jorge Gustavo.

[1]Instituto Nacional de Medicina Genómica, Ciudad de México, México. [2]Instituto Nacional de Medicina Genómica, Ciudad de México, México. [3]Universidad Autónoma de Ciudad Juárez, Ciudad Juárez, Chihuahua, México.

E-mail: emgomez177566@gmail.com

Dendritic cells (DCs) are professional antigen-presenting cells critical for initiating adaptive immune responses. They bridge innate and adaptive immunity by capturing, processing, and presenting antigens to naïve T cells; and beyond activating immunity, DCs are also central to maintaining immune tolerance and preventing autoimmunity. This study aims to analyze the dual role of dendritic cells in immune activation and tolerance, and to evaluate their therapeutic potential in autoimmune diseases and cancer immunotherapy. Recent peer-reviewed articles on dendritic cell subsets, antigen processing pathways, and immune regulation were reviewed, with special focus on clinical trials involving DC-based vaccines and tolerogenic DCs in autoimmune disorders. DCs induce either immunogenic or tolerogenic responses depending on the context; for example in cancer, ex vivo–generated DCs are used to stimulate antitumor immunity. Conversely, tolerogenic DCs can suppress autoreactive T cells and are under investigation for conditions such as type 1 diabetes and multiple sclerosis.

To conclude, dendritic cells are versatile regulators of the immune system, and manipulating their function offers promising avenues for developing tailored immunotherapies, either to enhance immune response or to restore tolerance in autoimmunity.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Evaluation of Piezo2 as a potential therapeutic target in chronic myeloid leukemia

González-Barragán, Jaqueline; González-Chávez, María Antonieta; Pedraza-Escalona, María Martha; Almagro-Domínguez, Juan Carlos; Pérez-Tapia, Sonia Mayra.

[1]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, CDMX, México. [2]Laboratorio Nacional para Servicios Especializados de Investigación, Desarrollo e Innovación (I+D+I) para Farmoquímicos y Biotecnológicos, LANSEIDI-FarBiotec-CONAHCyT, CDMX, México. [3]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Departamento de Inmunología, CDMX, México. [4]Unidad de Investigación Médica en Enfermedades Onológicas, CDMX, México.

E-mail: jaquelineglezb@gmail.com

Chronic Myeloid Leukemia (CML) is a hematologic neoplasm characterized by the uncontrolled proliferation of immature myeloid cells in the bone marrow and peripheral blood. This condition is associated with a genetic abnormality known as the Philadelphia chromosome, which triggers the overproduction of the BCR-ABL fusion protein with tyrosine kinase activity. The primary treatment for CML involves tyrosine kinase inhibitors (TKIs), such as Imatinib™. However, a significant proportion of patients fail to achieve a durable response or develop resistance to TKIs, often due to the persistence of quiescent leukemic cells within the leukemic niche. These are non-replicating leukemic stem cells that maintain a silent transcriptional activity. Gene expression profiling of stem and progenitor cells from CML patients has revealed a 13-fold overexpression of tRNA encoding for PIEZO2 compared to normal cells[1]. In addition, in silico studies have analyzed the relationship between PIEZO2, cancer stem cells, and genetic mutations, finding that PIEZO2 expression is positively correlated with the gene expression associated with tumor progression and metastasis in gastric cancer[2]. PIEZO2 has been identified as a transmembrane protein consisting of 2,752 amino acids, with a homotrimeric structure and 38 transmembrane segments. It functions as a force-activated, mechanosensitive ion channel, essential for touch, proprioception, and mechanical pain detection. To date, the Ca^{2+} permeability of PIEZO2 has been implicated in cancer metastasis, positioning it not only as a biomarker for CML but also as a potential therapeutic target, offering a unique opportunity for the development of therapies specifically directed against CML. Therefore, in this study, we aim to evaluate whether PIEZO2 can serve as an effective therapeutic target for CML, with the ultimate goal of developing biotherapeutics capable of eliminating the leukemic stem cells responsible for driving the disease.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Joint Microbiota Dysbiosis Contributes to Immune Responses and Inflammation in Spondyloarthritis

González-Chávez, Susana Aideé [1]; Alvarado-Jáquez, María Fernanda [1]; Salas-Leiva, Joan Sebastián [2]; Mohl, Jonathon E. [3]; Chaparro-Barrera, Eduardo [1]; Prieto-Carrasco, Rodrigo [1]; Loya-Rivera, Mario [1]; Pacheco-Silva, César [1]; Pacheco-Tena, César [1].

[1]Universidad Autónoma de Chihuahua, Facultad de Medicina y Ciencias Biomédicas, Laboratorio PABIOM. Chihuahua, Chihuahua, México. [2]Centro de Investigación en Materiales Avanzados (CIMAV), Chihuahua, México. [3]University of Texas at El Paso, El Paso Tx., USA.

E-mail: sagonzalez@uach.mx

Recent evidence suggests the existence of a commensal joint microbiome; however, its immunological relevance in the pathogenesis of spondyloarthritis (SpA) remains poorly understood. This study investigates the immunoinflammatory consequences of joint-associated bacteria in a spontaneous murine model of arthritis (SpAD) and in human SpA biopsies. Joint, gut, liver, and heart tissues from SpAD (DBA/1) and healthy (BALB/c) mice were analyzed using Gram staining, immunohistochemistry, 16S rRNA gene amplicon sequencing, and transcriptomic profiling via microarray analysis. In parallel, synovial biopsies from SpA patients were examined for the presence of microbes and immune activation using Gram staining and proinflammatory immune markers. The spatial interaction between bacterial components and cytokines (IL-6, TNF-α, IL-1β) was evaluated by double indirect immunofluorescence (IIF) in joint tissues. SpAD mice exhibited a distinct intra-articular dysbiosis, characterized by the enrichment of genera such as Pelomonas and Aerococcus. Mucosa-associated species, including Prevotella sp., R. gnavus, L. johnsonii, and L. reuteri, were detected in both gut and joint samples. Bacterial DNA from these taxa was also amplified from liver and heart tissues, indicating systemic dissemination. Transcriptomic profiling showed that, despite bacterial overlap, gut and joint tissues triggered distinct immune responses. Gut samples upregulated defensins, inflammasome components, gasdermin-mediated pyroptosis, and phospholipase activity. Conversely, joint tissues showed enrichment of TLR/MyD88 signaling, complement activation, and antigen presentation. These results underscore the existence of compartmentalized immune programs in response to microbial presence. Co-localization of bacterial structures with IL-1β and TNF-α further confirmed local immune activation. In human SpA biopsies, Gram staining and IHC revealed bacterial structures and the expression of proinflammatory markers in sacroiliac and tarsal joints.

This study demonstrates that joint dysbiosis and microbial immune sensing contribute to inflammation in SpA. The joint microbiota emerges as an immunologically active component and a potential therapeutic target in immune-mediated arthritis.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

NPY Links Inflammation and Senescence in Experimental Rheumatoid Arthritis via mTOR Modulation

González-Chávez, Susana Aideé [1]; Chaparro-Barrera, Eduardo [1]; Rivera-Loya, Mario [1]; Rodríguez-Castillo, Alejandra Jazmín [1]; Prieto-Carrasco, Rodrigo [1]; Aguilera-, Renato J. [2]; Betancourt-, Ana P. [2]; Mohl-, Jonathon E. [2]; Ruizesparza-Hinojos, Daniel Alberto [1]; Pacheco-Tena, César [1].

[1]Universidad Autónoma de Chihuahua, Facultad de Medicina y Ciencias Biomédicas, Laboratorio PABIOM. Chihuahua, Chihuahua, México. [2]University of Texas at El Paso, Border Biomedical Research Center (BBRC). El Paso, Texas, United States.

E-mail: sagonzalez@uach.mx

Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by persistent joint inflammation, immune dysregulation, and progressive destruction of articular structures. Cellular senescence has been implicated in sustaining inflammation through the senescence-associated secretory phenotype (SASP), yet the molecular mechanisms linking senescence and immune activation in RA remain incompletely understood. To explore this connection, mTOR was pharmacologically inhibited using rapamycin in the collagen-induced arthritis (CIA) model. DBA/1 mice with established CIA received rapamycin for 40 days, resulting in a significant reduction in joint inflammation, clinical arthritis scores, and tissue damage. Transcriptomic analysis of inflamed joints revealed differentially expressed genes involved in inflammatory, autophagy, and senescence pathways. Among these, neuropeptide Y (Npy) was identified as a key immunomodulatory gene downregulated by rapamycin. Immunohistochemical staining confirmed decreased protein expression of NPY, TNF-α, and β-galactosidase in synovial tissues following rapamycin treatment, indicating suppression of both inflammatory and senescent phenotypes. Additionally, rapamycin altered the expression of Npy1r and Npy2r receptors and upregulated autophagy-associated genes Sirt1, Sirt6, and Lc3b. In vitro, silencing of Npy in fibroblast-like synoviocytes led to a marked reduction in the SASP-associated cytokines Tnfa, Il1b, and Il6, as well as in Npy1r/2r expression, while significantly increasing Sirt1 levels. These effects highlight NPY's role in coordinating inflammatory and senescence-related signaling in RA. Collectively, these findings identify NPY as a molecular link between chronic inflammation and cellular senescence in autoimmune arthritis. Regulation of this pathway by rapamycin not only reduced local and systemic inflammatory responses, but also restored immune balance. Targeting the mTOR–NPY–sirtuin axis may offer a novel immunotherapeutic approach to limit joint destruction and inflammatory persistence in RA.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Cytotoxic effect of an anti-CD44v6/CD54 CAR-T against gastric cancer

González -Domínguez, Gonzalo [1]; Ortiz-Sánchez, Elizabeth [2];
Becerril-Rico, Jared [2]; Kim-, Yoo-Shin [3]; Vedvyas-, Yogindra [3] ;
Yang-, Yanping [3] ; Jin-, Moonsoo M. [3].

[1]Universidad Nacional Autónoma de México, Facultad de Química, Ciudad de México, México. [2]Instituto Nacional de Cancerología, Laboratorio de Células Troncales y Desarrollo Terapéutico Antineoplásico, Ciudad de México, México. [3]Houston Methodist Research Institute, Radiology Research Department, Houston, Texas, USA.

E-mail: gonzalo030793@gmail.com

Background Gastric cancer ranks among the top 5 cancers and has a 5-year overall survival rate of only 29%. CD44 variant 6 (CD44v6), an isoform of CD44, is identified in 70% of gastric cancer cases and considered a marker of poor prognosis. CD44v6 expression in normal tissue is very low and is restricted to certain cell types. Therefore, CD44v6 is a promising target for targeted therapies against gastric cancer. CD54 or ICAM-1 is overexpressed in different kinds of tumors, forming part of a cancerous stem phenotype in gastric cancer, but is absent in non-cancerous tissue in the stomach. In this study, we developed a chimeric antigen receptor (CAR) T cell therapy targeting CD44v6 and CD54 in vitro and in vivo. Methods: First, we generated two CAR-Ts anti-CD44v6 with scFvs derived from clones BIWA-4 and BIWA. Lentivirus were generated, and then, the Jurkat cell line and primary human T cells were transduced. Binding and activation of the CAR-Ts anti-CD44v6 were carried out, and surface activation markers and soluble cytokines were measured. Co-cultures with target cells expressing luciferase at different effector: target ratios were carried out for specific killing. Finally, antitumoral activity was measured by engrafting NSG mice subcutaneously. To increase the potency, the addition of CD54 as the target was measured using a BiTE anti-CD3/CD54 and with a bispecific-polycistronic CAR.

Results: Jurkat cells expressing the CAR demonstrated specific binding to the recombinant monomeric CD44v6. The transduction efficiency of primary T cells reached □70% after 10 days of expansion. CAR-Ts expressed CD69 and CD25 at 16 and 72 hours of antigen exposure, respectively, and in vivo data demonstrated that BIWA 8 was able to delay the tumor growth more in comparison to BIWA 4. The addition of CD54 as a second target increased CAR-T potency.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Purinergic Receptors and Exhaustion Marker Levels in T Lymphocytes in Breast Cancer

González -Hernández, Osiel [1]; Layseca-Espinosa, Esther [1];
Guel-Pañola, Arturo [3]; Morquecho-Robledo, Cristina [1];
Zavala-Reyes, Daniel [1]; Estrada-Sánchez, Ana María [2];
Portales-Pérez, Diana Patricia [1].

[1]Centro de Investigación en Ciencias de la Salud y Biomedicina (CICSaB) Universidad Autónoma de San Luis Potosí, Laboratorio de Medicina Molecular y Traslacional, San Luis Potosí, México. [2]Instituto Potosino de Investigación Científica y Tecnológica (IPICYT), Laboratorio de neurobiología, División de Biología Molecular, San Luis Potosí, México. [3]Hospital Regional de Alta Especialidad "Dr. Ignacio Morones Prieto", Oncología Médica, San Luis Potosí, México.

E-mail: osielgonzher@gmail.com

Breast cancer is an inflammatory disease in which danger-associated molecular patterns (DAMPs), such as ATP, are released and bind to purinergic receptors. P2X7 and P2X4 receptors are expressed in T cells and contribute to their activation and proliferation. Meanwhile, PD-1, CD39, and A2A are negative regulatory markers that help control immune responses. While the expression of these molecules in T cells from the tumor microenvironment is well known, their expression in circulating T cells is still unknown. This study aimed to assess the expression levels and function of P2X7, A2A, PD-1, and CD39 in T cells from breast cancer patients. Sixty patients were recruited with an average age of 50±10 years and an average Body Mass Index (BMI) of 30±6.7. A control group of thirty healthy women was also recruited. Blood samples were obtained, and surface staining of P2X7, A2A, PD-1, and CD39 was performed in CD8+ and CD4+ T cells; samples were then analyzed by flow cytometry. The results showed a significant increase in P2X7+PD1+CD8+ cells in Luminal B, HER2, and triple-negative patients compared to the control group. Meanwhile, in the P2X7+PD1+CD4+ subset, this difference was only observed in Luminal B patients compared to the control group. P2X7+CD39+ cells were significantly higher in the CD8+ and CD4+ cell subsets of Luminal B and HER2 patients. Conversely, P2X7+A2A+CD4+ cells were higher in HER2 patients than in the control group. Furthermore, patients with larger tumors showed a significant increase in P2X7+PD1+CD8+, P2X7+CD39+CD8+, and P2X7+CD39+CD4+ cells compared to the control group. Regarding lymph node involvement, high levels of P2X7+PD1+CD8+ cells were observed in N1, N2, and N2A patients. P2X7 functions were evaluated by proliferation, granzyme B, and calcium levels in cultured cells. Therefore, the expression of purinergic receptors and exhaustion markers in T cells may predict prognosis or response to treatment.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Development of a 3D Co-Culture Platform for Chemoresistance Prediction in B-ALL.

González-Reyes, Nelly Carmina [1]; Ángeles-Arvizu, Adriana [1];
Herrera-Torres, Estefanía [1]; Amador-Cardoso, Jiovanni [2];
Balandran-Juárez, Juan Carlos [3]; Casique-Aguirre, Diana [2];
Pelayo-Camacho, Rosana [2]; Pérez-Tapia, Sonia Mayra [1].

[1]Unidad de desarrollo e investigación en bioterapéuticos, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Ciudad de México, CDMX, México. [2]Centro de Investigación Biomédica de Oriente, Instituto Mexicano del Seguro Social, Delegación Puebla. Atlixco-Metepec. 74360. Pue. México. [3]Laboratorio Juárez, Oaxaca, México.

E-mail: nellycarmina19@gmail.com

B-cell acute lymphoblastic leukemia (B-ALL) is a multifactorial hematological disease that predominantly affects the pediatric population. It is characterized by the uncontrolled proliferation of lymphoblasts, which progressively displace mature cell lineages. Despite the use of chemotherapy as the gold standard treatment, Mexico continues to experience high rates of mortality and morbidity. Therefore, there is a need to implement in vitro culture systems that can predict the behavior of leukemia-initiating cells (LICs). Initially, 2D cultures were attempted; however, their structure fails to simulate the physiological conditions required to evaluate the leukemic niche. As a result, the approach shifted to 3D co-cultures, which provide a more suitable environment for assessing both the microenvironment and leukemia-initiating cells. This study utilized leukemic cell lines (Nalm-6, REH, RS4;11) and a murine mesenchymal stromal cell line (OP9) to establish a 3D co-culture system. The resistance or sensitivity of these cells was evaluated against vincristine, prednisolone, and daunorubicin at different concentrations. Additionally, a combined treatment was tested using the highest concentrations of these drugs along with 5 mM methotrexate. Following standardization with cell lines, patient-derived cells were assessed and compared with the Nalm-6 control, revealing similar behavior. These findings highlight the importance of using physiologically relevant platforms to develop personalized treatments, potentially reducing relapse rates in B-ALL patients.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Functional Role and Molecular Mechanisms of NTB-A (SLAMF6) in Human Natural Killer Cells

González-Sánchez, Mireille [1]; Cruz-Muñoz, Mario Ernesto [1].

[1]Facultad de Medicina, Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México. [2]Unidad de Investigación en Inmunología e Infectología, Facultad de Medicina, Universidad Autónoma del Estado de Morelos (UAEM).

E-mail: mireillegonz@gmail.com

targeting virus-infected and tumor cells. NTB-A (SLAMF6) is a transmembrane receptor of the SLAM family that modulates NK cell responses through interactions with adaptor proteins such as SAP and EAT-2. Depending on the intracellular signaling context, NTB-A can promote or inhibit NK cell activation, making it a potential therapeutic target in cancer and immune regulation. This project aims to elucidate the molecular mechanisms by which NTB-A regulates the effector functions of human NK cells. We cloned the human NTB-A gene from activated PBMCs and successfully inserted it into the pSK vector. The recombinant plasmid (pSK-NTBA) was verified by restriction digestion and sequencing. Subcloning into expression vectors (pSRalpha and PGB) is currently in progress to assess NTB-A function in effector (YT-S) and target cells. Our strategy includes evaluating NTB-A expression in NK cells via flow cytometry, analyzing cytotoxic activity against target cells (K562 and YTS), and studying interactions with other receptors (e.g., NKG2D, DNAM-1). We will also investigate downstream signaling pathways such as VAV1 and NF-B activation by Western blot and cytokine production (IFN-, TNF-alpha) by ELISA. Understanding how NTB-A signaling shapes NK cell responses could lead to new strategies for enhancing anti-tumor immunity or mitigating autoimmune responses. Our preliminary results support NTB-A as a dual-function receptor with context-dependent effects on NK cell cytotoxicity and cytokine production.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Acute Myeloid Leukemia and Immunodeficiency With Multisystemic Involvement in Fanconi Anemia

Gorgonio-Meza, Diana Ximena; Ríos-Delgado, José Concepción [2];
Correa-Serrano, Carlos Alberto [3]; Muñoz-Plascencia, Sandra [4].

[1]ISSSTE, Medicina Interna, Zapopan, Jalisco, México. [2]ISSSTE, Neumología, Zapopan, Jalisco, México. [3]ISSSTE, Alergología, Zapopan, Jalisco, México. [4]ISSSTE, Medicina Interna, Zapopan, Jalisco, México.

E-mail: dianagorgonio@gmail.com

We present the case of a 54-year-old woman with a known diagnosis of Fanconi anemia (FA), who developed acute myeloid leukemia (AML) subtype M5, along with multiple systemic complications including immunodeficiency, pulmonary nodular disease, pleural effusion, and central nervous system involvement. The diagnostic complexity was heightened by the coexistence of infectious, hematologic, and microangiopathic features. The patient was initially admitted to a private hospital due to severe anemia and was transfused with three units of packed red blood cells. A peripheral blood smear raised suspicion for AML-M5, prompting referral to our center for further evaluation. Upon admission, she presented with normochromic anemia, severe thrombocytopenia, and leukocytosis with monocytosis and neutrophilia. Diagnostic workup included bone marrow aspiration and biopsy, immunophenotyping, serologic and biochemical panels, and whole-body imaging to investigate potential lymphoproliferative processes. Past medical history was significant for cryptococcal meningitis and common variable immunodeficiency (CVID), for which she had been receiving intravenous immunoglobulin therapy. During hospitalization, she developed progressive dyspnea and pleuritic symptoms. Imaging revealed bilateral cavitary pulmonary nodules and a left-sided exudative pleural effusion; thoracentesis was performed, but cytology and cultures were inconclusive. Echocardiography showed a moderate pericardial effusion. Cranial MRI identified gliotic foci in periventricular and subcortical white matter, cortical-subcortical atrophy, and leukoaraiosis, consistent with chronic microangiopathy. Additionally, sinus imaging revealed septal deviation and mucosal thickening. Given the patient's immunosuppressed state, prophylactic treatment with trimethoprim-sulfamethoxazole, fluconazole, and nystatin was initiated. This case exemplifies the complex interplay between FA, hematologic malignancy, immunodeficiency, and multisystemic involvement. Comprehensive, multidisciplinary evaluation was essential to address diagnostic uncertainty and guide clinical management.

TRABAJO NO PRESENTADO

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Epicatechin regulates autophagy and PD-L1 expression in hepatocellular carcinoma cell line

Gress-Mogica, Karla Cecilia [1]; Hernández-González, Juan Carlos [1]; Cariño-Cortés, Raquel [3]; Fernández-Martínez, Tomás Eduardo [3]; Castillo-Cruz, Juan [2]; García-Pérez, Blanca Estela [2]; Castrejón-Jiménez, Nayeli Shantal [1].

[1]Área Académica de Medicina Veterinaria y Zootecnia, Instituto de Ciencias Agropecuarias-Universidad Autónoma del Estado de Hidalgo, Av. Universidad No. 133. Col. San Miguel Huatengo, Santiago Tulantepec de Lugo Guerrero, Hidalgo, México, C.P. 43775. [2]Departamento de Microbiología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Prolongación de Carpio y Plan de Ayala s/n, Ciudad de México 11340, México. [3]Área Académica de Nutrición, Instituto de Ciencias de la Salud, Universidad Autónoma del Estado de Hidalgo Circuito Ex Hacienda, La Concepción S/Carretera Pachuca Actopan, San Agustín Tlaxiaca, Hidalgo.

E-mail: karlacgressm@gmail.com

Hepatocellular carcinoma is the most common primary neoplasm of the liver. It has become a public health problem, which has encouraged the research of new drugs derived from natural products. The bioactive compounds that have garnered significant interest due to their various biological and functional activities are flavonoids, among which is epicatechin, found in a wide variety of fruits and sedes and which has been shown to possess antioxidant, anti-inflammatory, anticancer, and immunomodulatory properties. The induction of the autophagic process is demonstrated by increasing the expression of essential proteins such as Beclin-1 and LC3, which are fundamental for the formation of autophagosomes. On the other hand, the PD-L1 protein, expressed in tumor cells, interacts with PD-1 on T cells, suppressing the immune response and facilitating tumor immune evasion. In this context, it has been reported that epicatechin, like other catechins, can inhibit the expression of PD-L1, favoring a more efficient immune response against cancer cells. Consequently, this research aims to demonstrate the effect of epicatechin (50, 100, 250, 500, 750, 1000, and 1500 µM) on a HepG2 hepatocarcinoma cell line, focusing on the modulation of autophagy and immune evasion mechanisms. For this purpose, cell viability was assessed using the MTT assay. The preliminary results indicated a decrease in cell viability, with a median inhibitory concentration (IC_{50}) of 750 µM for epicatechin and 50 µM for cisplatin. The expression of PD-L1 and autophagy markers (LC3-I, LC3-II, and p62) was determined by Western Blot, indicating that epicatechin promotes autophagy in a dose-dependent manner, although not as efficiently as rapamycin. On the other hand, it could contribute to a more effective immune response by inhibiting PD-L1 in cancerous liver cells at a concentration of 500 µM. Therefore, it's suggested that epicatechin could be involved in mechanisms of immune modulation in cancerous liver cells.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Phenotypic analysis of T lymphocytes and monocytes in patients with acute pancreatitis

Güemes-González, Azmavet Maharai [1]; León-Pedroza, José Israel [2 3]; Rodríguez-Negrete, Elda [4]; Arriaga-Pizano, Lourdes Andrea [5]; Chacón-Salinas, Rommel [1 6]; Ferat-Osorio, Eduardo [5 7]; Wong-Baeza, Isabel [1 6].

[1]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Ciudad de México, Mexico. [2]Coordinación de Investigación, Unidad 401-C, Urgencias Médicas, Hospital General de México “Dr. Eduardo Liceaga”, Ciudad de México, Mexico. [3]Coordinación de Ciclos Básicos, Universidad Anáhuac, Ciudad de México, Mexico. [4]Servicio de Gastroenterología, Clínica de Páncreas, Hospital de Especialidades, Centro Médico Nacional “Siglo XXI”, Instituto Mexicano del Seguro Social (IMSS), Ciudad de México, Mexico. [5]Unidad de Investigación Médica en Inmunoquímica. Centro Médico Nacional “Siglo XXI”, Instituto Mexicano del Seguro Social (IMSS), Ciudad de México, Mexico. [6]Red de Salud del Instituto Politécnico Nacional, Ciudad de México, Mexico. [7]División de Investigación Clínica, Coordinación de Investigación en Salud, Instituto Mexicano del Seguro Social, Ciudad de México, Mexico.

E-mail: maha.glez.bios@gmail.com

Acute pancreatitis is a type of inflammation of the pancreatic tissue considered sterile, associated with the premature activation of digestive enzymes within the pancreatic acini, which leads to a process of self-digestion and the generation of an inflammatory response. Most patients present with the mild or moderately severe form of the disease; however, 20% of patients may develop the severe form, which is frequently complicated by sepsis.

Sepsis is a dysregulated immune response to an infection, leading to organ failure and its severity is potentially life-threatening. Among the immunological features of sepsis are the exhaustion of T lymphocytes, evidenced by increased PD-1 expression, the decrease of HLA-DR in monocytes, and the reduced response of these cells to lipopolysaccharide (LPS). In this study, peripheral blood from patients with acute pancreatitis in the early stages of the disease was analyzed to determine whether these patients exhibited immunological features characteristic of sepsis. The results showed that T lymphocytes from patients with acute pancreatitis had lower levels of PD-1 expression than lymphocytes from patients with sepsis at the onset of pancreatitis, although PD-1 levels increased in CD4+ T lymphocytes from patients with moderately severe pancreatitis 3 or 7 days after onset. Classical monocytes from patients with acute pancreatitis and from patients with sepsis showed lower HLA-DR expression than monocytes from healthy individuals. Finally, classical monocytes from patients with acute pancreatitis produced higher levels of TNF-alpha in response to LPS, like those of monocytes from healthy individuals. These findings suggest that patients with acute pancreatitis present certain immunological features associated with sepsis.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Compatibility of NK cell immunotherapy with cannabidiol in leukemia treatment

Gutiérrez-Iñiguez, Cecilia Lizette [1]; Olivas-Aguirre, Miguel Ángel [2 3]; Pottosin-, Igor [1]; Dobrovinskaya-, Oxana [1].

[1]Laboratory of Immunobiology and Ionic Transport Regulation, University Center for Biomedical research, University of Colima, Colima 28040, Mexico. [2]Secretaria de Ciencia, Humanidades, Tecnología e innovación (Secihti), Programa de Investigadores e Investigadoras por México, México City 03940, México. [3] Laboratory of Cancer Pathophysiology, University Center for Biomedical Research, University of Colima, Colima 28040, Mexico.

E-mail: cgtziniguez@gmail.com

Cannabidiol (CBD), a non-psychoactive compound from cannabis, has shown anticancer properties in preclinical studies. Our previous study indicated that acute lymphoblastic leukemia (ALL) cells were the most sensitive to CBD, as compared to resting lymphocytes and other tissues or cancer types. This positions CBD as a potential non-toxic agent for treating ALL. Additionally, CBD offers clinical benefits in managing cancer-related symptoms. Concurrently, Natural Killer (NK) cell-based immunotherapy is a promising strategy for combating various cancers, including leukemia, lymphoma, and solid tumors. This approach is well-tolerated by patients, minimizing the severe side effects often caused by conventional chemotherapies. Yet it was unknown to which extent CBD exerts an immunosuppressive action on intrinsic or introduced NK cells. We assessed CBD's toxicity in NK cells using trypan blue exclusion, metabolism, and cell death assays. To evaluate NK cell functionality, we employed co-culture of NK cells with leukemic target cells (Jurkat) and evaluated their migration, degranulation, and target-directed cytotoxicity. Pharmacological tools were used to identify endocannabinoid system receptor involvement. At lower concentrations (0.5 – 10 μM), CBD neither promoted NK cell proliferation nor negatively impacted their cytotoxic capacity and migratory potential. Intermediate CBD doses (10 – 25 μM) partly reduced NK cells' cytotoxic efficacy against leukemic cells. Higher CBD concentrations (30-100 μM) induced NK cell death, dependent on the mitochondrial permeability transition pore (mPTP) formation. These findings collectively suggest that NK cells are relatively resistant to CBD as compared to ALL cells, which opens a therapeutic window for a combined use of CBD and NK cells-based therapy. Further preclinical studies should define an optimal combination of CBD treatment and NK cell-based immunotherapy, to design an innovative strategy to overcome the ALL resistant for conventional drugs.

This work was supported by CONAHCYT grants PRONACES #303072, CF-2019 #21887 and FOP02-2022-02 #321696.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Targeting HPV16-E5 to DCs by prophylactic vaccination reduces tumor growth and metastasis.

Pérez-Morales, Aislinn C [1 2]; Badillo-Godínez, Oscar [3];
Maldonado-Gama, Minerva [1]; Bonifaz-Alfonzo, Laura [4];
Esquivel-Guadarrama, Fernando R [5]; Gutiérrez-Xicotencatl, Lourdes [1].

[1]Centro de Investigación Sobre Enfermedades Infecciosas, Instituto Nacional de Salud Pública, Cuernavaca, Morelos, México. [2]Facultad de Ciencias, Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México. [3]Uppsala University, Uppsala, Sweden. [4]Hospital de Especialidades, Centro Médico Nacional Siglo XXI, IMSS, México. [5]Facultad de Medicina, Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México.
E-mail: mlxico@insp.mx

Human papillomavirus (HPV) is the main etiological agent associated with cervical cancer (CC), and type 16 is the most prevalent in this type of cancer. The high-risk HPVs express the oncoproteins E5, E6, and E7 that favor cellular transformation and progression to cancer. HPV16-E5 oncoprotein is expressed early during the viral cycle, interfering with the endocytic traffic, endosome acidification, and reducing MHCI and II expression. Thus, as E5 interferes with the immune response and is expressed early during the transformation process, it is considered an ideal therapeutic target for HPV associated cancers. New approaches to potentiate the immune response, such as targeting antigens to dendritic cells (DCs) receptors to increase antigen presentation. In this sense, the anti-tumoral immune response generated by targeting E5 to the DCs receptor DEC-205 has been evaluated in a mouse tumor model for HPV16. Balb/c female mice (6-8 weeks old) were immunized on days 0 and 21 with 5μg of HPV16-E5 cross-linked to anti-DEC-205 monoclonal antibody or with control treatments (PBS or E5 alone). Three weeks later, mice were inoculated on the back with BMK16-myc cells (expressing the HPV16 full genome), and tumor growth, survival, and the immune response were evaluated. Prophylactic immunization with anti-DEC-205:E5 slows down the tumor growth for more than 10 days and increases 50% the mice's survival by day 45, as compared to the PBS and E5-alone control treatments. The immune response associated with the anti-DEC-205:E5 treatment was predominantly CD8+ and Th1-type memory response. Additionally, only the anti-DEC-205:E5 treatment showed inhibition of lung metastasis. Targeting HPV16-E5 to DCs by prophylactic immunization of mice slows down tumor growth, increases survival, and keeps the mice metastasis-free. This suggests that prophylactic immunization of HPV16-E5 by targeting DCs allows a strong CD8+ memory immune response, which controls tumor growth, increases survival, and maintains mice metastasis-free.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

The neutrophil-to-hemoglobin and lymphocyte score and clinical profile of Rheumatoid Arthritis

Gutiérrez-Pérez, Ilse Adriana [1]; Flores-Castro, Ma. Natividad [1];
Zaragoza-García, Oscar [1]; Briceño-Cárdenas, Olivia [2];
Guzmán-Guzmán, Iris Paola [1].

[1]Laboratorio de Investigación Multidisciplinaria e Innovación Biomédica, Facultad de Ciencias Químico-Biológicas, Universidad Autónoma de Guerrero, Chilpancingo, Guerrero, México.
[2]Centro de Investigación en Enfermedades Infecciosas, Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas, Ciudad de México, México.

E-mail: ilsegtz@yahoo.com

Rheumatoid Arthritis (RA) is a pathology that primarily affects synovial joints. The persistence of the inflammatory process leads to a degradation of the underlying cartilage and bone, promoting the development of erosions, joint deformity, functional disability, impaired quality of life, and premature death related to other comorbidities. On the other hand, neutrophils and lymphocytes are key effector cells in the inflammatory response, and iron deficiency anemia is a common condition in patients with RA. The neutrophil-to-hemoglobin and lymphocyte (NHL) score has been little evaluated in RA. This study aims to assess the association between the NHL score and the clinical profile related to clinical activity and severity of RA. This study included 150 patients diagnosed with RA according to the 2010 ACR/EULAR criteria. The clinical parameters evaluated were the functional disability index as measured by the HAQ-DI (Health Assessment Questionnaire) scale; radiological joint damage, using the SHS (Sharp-van der Heijde) score, and the clinical activity determined by the DAS28-ESR (Disease Activity Score-28, erythrocyte sedimentation rate). C-reactive protein (CRP) levels were determined by immunoturbidimetry. The NHL score was derived from hematological parameters and estimated as follows: N/(Hb*L), where N is the baseline blood neutrophil count, Hb is hemoglobin, and L is the lymphocyte count. Statistical analysis was performed using Stata software, v. 16. The NHL score correlated positively with CRP levels ($p= 0.001$) and the HAQ-DI index ($p= 0.03$). Furthermore, an increased NHL score was associated with a higher clinical activity index ($p= 0.008$), as well as with loss of joint space narrowing and bone deformity ($p= 0.04$). In conclusion, these results suggest that NHL score values are related to the clinical profile of rheumatoid arthritis and could be used in clinical assessments in healthcare settings.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Integrated Epigenomic and Transcriptomic Profiling of TNF-α-Mediated Dysregulation in CD4+ T Cells

Guzmán-Barrenechea, Karla Paulina [1,2]; Malvaez-Luis, Dulce Rosario [1]; Bejar-Jiménez, Perla Aimeé [1]; Madera-Salcedo, Iris Karina [1].

[1]Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán. Departamento de Inmunología y Reumatología. Tlalpan, CDMX. México. [2]Universidad Nacional Autónoma de México. Posgrado en Ciencias Biológicas. Ciudad Universitaria, CDMX. México.

E-mail: karlabarrenech@gmail.com

Tumor necrosis factor-α (TNF-α) is a key inflammatory cytokine that shapes immune responses. While transient TNF-α signaling supports host defense, chronic exposure leads to aberrant T cell activation, a hallmark of several autoimmune and inflammatory diseases. CD4$^+$ T cells, which differentiate into helper T cell (Th) subsets, are sensitive to inflammatory cues. TNF-α influences their gene regulation, but its long-term impact on epigenomic and transcriptomic programming remains unclear. In this study, we sought to elucidate how chronic TNF-α signaling reprograms the epigenomic and transcriptomic landscape of CD4$^+$ T cells. Using an in vitro model of T cell activation in the presence of TNF-α, we combined flow cytometry with ATAC-seq and RNA-seq to profile changes in Th subset distribution, chromatin accessibility, and gene expression. Flow cytometry revealed that chronic TNF-α exposure skewed CD4$^+$ T cell differentiation by promoting Th2 polarization while suppressing Th17 and Th9 subsets. RNA-seq analysis identified 1,563 differentially expressed genes (DEGs), including reduced expression of cytokines associated with Th9 and Th17 identity. Concurrently, 1,400 differentially accessible regions (DARs) were detected, enriched at putative enhancer elements. Strikingly, several TNF-α-induced accessible regions were associated with reduced transcription of nearby genes, suggesting noncanonical regulatory effects. Within this reprogrammed epigenetic landscape, noncoding RNAs emerged as critical modulators of Th cell fate. Among them, MIR155HG, the host transcript of miR-155, was downregulated following TNF-α treatment. miR-155 is a key regulator of CD4$^+$ T cells polarization, and function. Its downregulation may underlie the reduced Th9 population, likely through disruption of the MIR155HG–OX40 signaling axis. These findings demonstrate that chronic TNF-α signaling reshapes the chromatin and transcriptional landscape of CD4$^+$ T cells, linking persistent inflammation to durable changes in Th subset composition and effector function.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Melittin and methotrexate: Combined effect on leukemic cells

Guzmán -García, Luz María [1 2]; García-Iglesias, Trinidad [1 3]; Ruvalcaba-Plascencia, Ariel Humberto [1]; Cubillo -Cervantes, Danna [1]; Aguirre-León, Jovani Guadalupe [1]; Reyes-Pérez, Itzel Viridiana [4]; Machado-Sulbaran, Andrea [1]; Sánchez-Hernández, Pedro Ernesto [1 3].

[1]Universidad de Guadalajara, Centro Universitario de Ciencias de la Salud (CUCS), Instituto de Investigación en Cáncer en la Infancia y Adolescencia, Departamento de Clínicas de la Reproducción Humana, Crecimiento y Desarrollo Infantil. Guadalajara, Jal. México. [2]Universidad de Guadalajara, CUCS. Doctorado en Ciencias Biomédicas, Departamento de Fisiología. Guadalajara, Jal. México. [3]Universidad de Guadalajara, CUCS, Departamento de Fisiología. Guadalajara, Jal. México. [4]Universidad de Guadalajara, CUCS, Departamento de Disciplinas Filosófico, Metodológicas e Instrumentales.

E-mail: luz.guzman8315@alumnos.udg.mx

Acute lymphoblastic leukemia (ALL) is the most common childhood cancer, with T-cell ALL associated with a poorer prognosis requiring intensive treatment. In Mexico, it is the leading cause of disease-related death among children aged 5-14 years, partly due to the therapies toxicity. Therefore, there is an urgent need for more specific and less toxic adjuvant treatments. Melittin (MEL), the main component of bee venom, can cross cell membranes and has been shown to induce cell death in various cancer cell lines. However, its combination with standard ALL drugs, as methotrexate (MTX) remains unstudied. This study aims to assess the cytotoxic activity of MEL and its potential synergistic effect with MTX on the T-ALL cell line CEM. In vitro experimental study. CEM cells were treated with MEL, MTX, and their combination, along with negative (no treatment) and positive (etoposide treatment) controls. Cytotoxicity was evaluated using MTT and trypan blue assays; cell death was analyzed by flow cytometry with annexin V and propidium iodide. FlowJo was used for flow data analysis, and GraphPad Prism and R for statistical processing. The IC50 of MEL at 24 h was 2.036 µM; concentrations of 0.5µM, 1µM, and 2µM were selected. Dose-time curves were used to determine exposure times: 3 and 24 hours. For MTX, concentration 1µM and time 24 h were established. Combined treatments were tested under two schemes: simultaneous (both drugs together) and sequential (MEL followed by MTX). The combination showed greater reduction in cell viability compared to individual treatments, with no differences between schemes. Necrosis was the main type of cell death. MEL showed cytotoxic activity in CEM cells and enhanced the viability reduction induced by MTX, suggesting a potential adjuvant effect. Necrosis was the predominant type of cell death, indicating the need to further study its underlying mechanisms.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Interleukin 27 serum levels are associated with radiographic progression in rheumatoid arthritis

Guzmán-Guzmán, Iris Paola [1]; Briceño-Cárdenas, Olivia [2];
Falfán-Valencia, Ramcés [3]; Gutiérrez -Pérez, Ilse Adriana [1];
Zaragoza-García, Oscar [1].

[1]Laboratorio de Investigación Multidisciplinaria e Innovación Biomédica, Facultad de Ciencias Químico-Biológicas, Universidad Autónoma de Guerrero, Chilpancingo, Guerrero, México.. [2]Centro de Investigación en Enfermedades Infecciosas, Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas, Ciudad de México, México.. [3]Laboratorio de Neumogenómica, Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas, Ciudad de México, México.

E-mail: pao_nkiller@yahoo.com.mx

Interleukin 27 (IL-27) is a soluble cytokine known for its pleiotropic roles in modulating inflammatory responses in rheumatoid arthritis (RA). This study aims to evaluate the role of serum IL-27 levels according to radiographic damage categories, clinical characteristics, and inflammatory markers. Here, we report a cross-sectional study of 60 AR women from southern Mexico. RA patients with erosions (presenting a Sharp Van der Heijde score >III) had longer durations of illness, higher morning stiffness duration, higher HAQ-DI scores, and higher serum levels of IL-27 than patients without erosions. Moreover, serum levels of IL-27 positively correlated with rheumatoid factor (RF). Circulating IL-27 has been proposed as a biomarker of clinical activity in RA, but it could also be considered a surrogate biomarker of radiographic progression. In conclusion, these findings suggest a possible role of IL-27 in joint destruction and a potential role as a predictive marker of destructive disease. Further studies are needed to clarify the pathophysiological role of IL-27 in larger cohorts of RA patients.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Optimizing Methotrexate and L-Asparaginase Therapy for B-Cell Acute Lymphoblastic Leukemia.

Hasing-Macías, Camila Lorena [1]; Ratkovich-González, Sarah [1]; Barba Barba, Cesar Cenobio [2]; Ávila Ramírez, David [1]; Cáceres-Espíndola, Keila Saraí [1]; Nieto Hernández, Alejandro [1]; Bustamante Martínez, Juan Andrés [1]; Medina-Escobar, Rina Alejandra [2]; Núñez-Marrero, Judith Alexandra [2]; Rosas González, Vida Celeste [1].

[1]Tecnologico de Monterrey, Escuela de Medicina y Ciencias de la Salud, Zapopan, Jal. México.
[2]Hospital Civil de Guadalajara “Dr. Juan I. Menchaca”, Departamento de Pediatría, División de Hemato-Oncología Pediátrica, Guadalajara, Jal. México.

E-mail: camila.hasing@gmail.com

Acute lymphoblastic leukemia (ALL) represents the most prevalent hematological malignancy in children, with the B cell subtype being the most common. Standard first-line therapy incorporates methotrexate (MTX) and L-asparaginase (L-asp); however, prior administration of L-asp has been shown to reduce MTX efficacy by inducing G1 phase arrest, preventing cells from entering the S phase, where MTX exerts its effects. Consequently, evaluating the combined effect of these agents on primary cells from pediatric B-ALL patients is of clinical significance. Bone marrow samples were obtained from patients diagnosed with B-ALL, and mononuclear cells were isolated via density gradient centrifugation. Pre-B lymphoblasts were purified through negative magnetic selection and treated with MTX (15, 30, and 45 nM), L-asp (0.1, 2.5, and 5 U/mL), or a combination (2.5 U/mL + 15 nM). Cell viability was assessed using the WST-8 assay; apoptosis and cell cycle were analyzed by flow cytometry using Annexin V/PI and propidium iodide staining. In terms of cell cycle dynamics, treatment with 2.5 U L-asp resulted in elevated levels in the G0/G1 phase, accompanied by an S-phase arrest; higher doses further increased G0/G1 arrest. MTX treatment alone induced S phase arrest even at low doses. The concurrent application of L-asp and MTX nearly doubled S phase arrest compared to MTX alone. Clinically, the sequential administration of L-asp followed by MTX showed a lower percentage of arrest compared to simultaneous treatment. In addition, MTX monotherapy significantly reduced cell viability in a dose-independent manner, whereas L-asp demonstrated a dose-dependent effect. Preliminary data indicate that L-asp and MTX combination more effectively reduces viability than subsequent MTX treatment following L-asp administration. These findings suggest that the sequence and concentration of MTX and L-asp critically influence leukemic cell response, and their combined application may present a more efficacious strategy for inducing apoptosis in pre-B lymphoblasts.

Funding: FODECIJAL-11130-2024.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

IL-36 and VHL as possible mechanisms of VEGF production in mesangial cells

Heredia-Murillo, Marcelo Wilfredo [1]; Sánchez -Torres, María Marcela [1]; Cancino -Diaz, Juan Carlos [1]; Cancino -Diaz, Mario Eugenio [1].

[1]Instituto Politécnico Nacional, Escuela Nacional de Ciencias Biológicas, Departamento de Inmunología, Ciudad de México, México.

E-mail: marceloheredia80@gmail.com

Diabetic nephropathy is one of the main complications of diabetes mellitus, associated with alterations in the function of glomerular cells such as podocytes, endothelial cells and mesangial cells. In this context, interleukin 36 (IL-36) and Von Hippel-Lindau protein (VHL) could play a key role in the regulation of vascular endothelial growth factor (VEGF), a key protein in the regulation of endothelial cells. Increased VEGF has been associated with abnormal angiogenesis in the glomerulus, which could contribute to the progression of renal damage. The aim of this work was “To study the possible molecular pathways used by mesangial cells for VEGF production” in order to better understand their involvement in the pathogenesis of diabetic nephropathy. For this purpose, mesangial cells (MES SV40) were cultured in media with high glucose concentrations. The expression of IL-36, VHL, TGF-β and VEGF was assessed by RT-PCR. Preliminary results indicate that high glucose concentration induces IL-36 expression at 24 hours and at 25 hours time at which time high VEGF expression begins in MES SV40 cells. At the same time it was also observed that glucose induces TGF-β and VEGF expression, while VHL expression decreases. These preliminary findings suggest that IL-36 and VHL play a role in the regulation of VEGF in SV40 MES cells at high glucose concentrations.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Reactive Oxygen Species mediate the antibabesial effect of Aquiluscidin

Hernández-Arvizu, Edwin Esaú [1]; Rodríguez-Torres, Angelina [2];
León-Ávila, Gloria de la Luz [3]; Vega y Murguía, Carlos Agustín [2];
Rivas-Santiago, Bruno Tonatiuh [4]; Mosqueda-, Juan [1].

[1]Immunology and Vaccine Research Laboratory, Natural Sciences College, Autonomous University of Querétaro, 76230 Querétaro, México. [2]Natural Sciences College, Autonomous University of Querétaro, 76230 Querétaro, México. [3]Department of Zoology, National School of Biological Sciences, National Polytechnic Institute, Carpio y Plan de Ayala S/N, C.P. 11340, Casco de Santo Tomas, México City, México. [4]Medical Research Unit Zacatecas-IMSS, Zacatecas, 98053 Zacatecas, México.

E-mail: esau.hernandez@uaq.mx

Babesiosis is a zoonotic disease caused by Babesia spp., an intraerythrocytic protozoan that affects different mammalian species. In recent years, the search for new antibabesial compounds has become increasingly important due to the limitations of current chemotherapeutics, including adverse side effects and prolonged withdrawal periods. To develop effective therapeutic strategies, it is essential to elucidate the mechanisms of action of new candidate molecules. Cathelicidins, a family of host defense peptides, are essential components of the innate immune system, contributing to both direct antimicrobial activity and immunomodulation. They have demonstrated antiprotozoal activity by disrupting parasite structures and inducing cell death. Previously, Aquiluscidin, a cathelicidin identified in Crotalus aquilus, exhibited in vitro activity against three Babesia species infecting cattle (B. bigemina, B. bovis and B. ovata). Nevertheless, the action mechanism was not determined. The objective of this work was to investigate the mechanism underlying the antiparasitic activity of Aquiluscidin against B. bigemina. Infected red blood cells (*in vitro*) were treated with 14.48 μM and 20.70 μM of Aquiluscidin for 24 hours. The production of reactive oxygen species (ROS) was evaluated using fluorescence microscopy. Water and hydrogen peroxide (H_2O_2) served as negative and positive controls, respectively. Fluorescence images were analyzed, and statistical analyses were conducted. Both concentrations of Aquiluscidin induced a significant increase in ROS production compared to the negative control ($p < 0.0001$). These results suggest that the antiparasitic activity of Aquiluscidin involves oxidative stress mediated by ROS overproduction. In conclusion, Aquiluscidin exerts an antimicrobial effect against the intraerythrocytic stages of B. bigemina by increasing ROS generation, which would contribute to the subsequent parasite cell death.

This work was funded by UAQ-FOPER (FOPER2021-FCN02411).

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Immunomodulatory Effects of Hydroxycinnamic Acid and Epicatechin on NK Cells in a Breast Cancer Co-Culture Model

Hernández-Benítez, José Luis [1]; Gutiérrez-Silerio, Gloria Yareli [1];
García-Solís, Pablo [1].

[1]Autonomous University of Queretaro, Interdisciplinary Center of Applied Biomedicine, Santiago de Querétaro, Querétaro, México.

E-mail: jluishebez@gmail.com

Breast cancer is the most frequently diagnosed cancer and the leading cause of cancer-related death among women in Mexico and worldwide. Hydroxycinnamic acid (HCA) and epicatechin (EC) have been investigated for their antitumor properties in various cancers. Recent studies suggest that these polyphenols may also modulate the immune system, particularly by restoring the cytotoxic function of natural killer (NK) cells. Oxidative stress in the tumor microenvironment is known to suppress NK cell activity in both in vitro and in vivo models, we hypothesized by restoring redox balance with HCA and EC, NK cells may recover their cytotoxic potential. To evaluate the modulatory effect of HCA and EC on NK cells cultured with breast cancer cells a co-culture system of MCF-7 breast cancer cells and NK-92 cells will be established using non-cytotoxic concentrations of HCA and EC. Expression levels of activating (NKG2D) and inhibitory (NKG2A) receptors on NK cells will be measured. In MCF-7 cells, Annexin V will be assessed as an apoptosis marker. Additionally, cytotoxic granules including Granzyme B and Perforin will be quantified to confirm NK cell cytotoxic function. Preliminary data confirm the antitumor effect of HCA (IC_{50}: 30 µg/mL) and EC (IC_{50}: 520 µg/mL) on MCF-7 cells. However, the safe working concentration for NK-92 cells in co-culture is still being optimized. An increase in NKG2A and a decrease in NKG2D expression on NK cells could indicate a molecular mechanism underlying the immunomodulatory effects of these polyphenols and an alternative treatment for breast cancer.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Quality of life in patients with Guillain-Barre syndrome and dysautonomia

Hernández-Campos, María Elena [1]; Solís-Gómez, Raymundo [1 2];
Martínez-Piña, Daniel Arturo [2]; López-Hernández, Juan Carlos [2].

[1]Sección de Estudios de Posgrado e Investigación, Escuela Superior de Medicina del Instituto Politécnico Nacional. Plan de San Luis y Díaz Mirón s/n, Casco de Sto. Tomás, Ciudad de México, México. [2]Clínica de Enfermedades Neuromusculares, Instituto Nacional de Neurología y Neurocirugía "Manuel Velasco Suárez". Ciudad de México, México.

E-mail: mehernandezc@ipn.mx

Guillain-Barre syndrome (GBS) is an acute polyradiculoneuropathy. Between 30% and 66% of patients present dysautonomia during hospitalization, which is associated with impaired quality of life (QoL) up to 6 months after symptom onset. This cohort aim to determine global QoL and the subdivisions the Physical Component Summary (PCS) and Mental Component Summary (MCS), as well as factors related to QoL in patients with GBS and dysautonomia at 3 months. Materials and Prospective cohort of patients with GBS conducted in 2024. Clinical and paraclinical characteristics were collected at admission. At 3-month follow-up, the SF-36 questionnaire was applied to assess global, physical, and mental QoL. Univariate correlations were performed with clinical and paraclinical variables; significant variables were further analyzed by multivariate analysis. Thirty-five patients (mean age 47.09±5.08 years), 57.1% male. At admission, mean Hughes score was 3.86±0.64, mEGOS 7.11±2.84, EGRIS 4±1.66, MRC 30.69±15.26, with 74.3% having axonal variant. At 3 months, global QoL score was 53.71±22.23, PF 49.16±22.16, and MF 58.31±25.47. Positive correlations were found between PCS and MRC at admission and length of hospitalization; MCS correlated with hospitalization days; global QoL correlated with MRC and mEGOS at admission. Axonal variant was associated with worse global QoL, PCS, and MCS scores; male sex was associated with worse global and MCS scores. In conclusion patients with GBS and dysautonomia, global, physical, and mental QoL are markedly impaired at 3 months after symptom onset, with greater impairment in males and axonal variants, independent of functionality.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Modified Rankin scale as a tool for functional follow-up in Guillain-Barre syndrome

Hernández-Campos, María Elena [1]; Solís-Gómez, Raymundo [1 2];
Martínez-Piña, Daniel Arturo [2]; López-Hernández, Juan Carlos [2].

[1]Sección de Estudios de Posgrado e Investigación, Escuela Superior de Medicina del Instituto Politécnico Nacional. Plan de San Luis y Díaz Mirón s/n, Casco de Sto. Tomás, Ciudad de México, México. [2]Clínica de Enfermedades Neuromusculares, Instituto Nacional de Neurología y Neurocirugía "Manuel Velasco Suárez". Ciudad de México, México.

E-mail: mehernandezc@ipn.mx

Guillain-Barre syndrome (GBS) affects patient functionality, primarily impairing gait. Therefore, gait recovery is commonly evaluated using the disability scale (Hughes scale); however, this scale does not always reflect overall disability and functional independence. The modified Rankin scale (mRS) could offer a better approximation of global functional status during follow-up. This cohort aim determines whether the mRS allows identification of very good functional outcome at 1 and 3 months in patients with GBS, as well as to identify predictive factors associated with this outcome. Prospective cohort of patients with GBS from January to November 2024. Baseline demographic data, prognostic scales, and nerve conduction studies at admission were collected. Informed consent was obtained. Prognostic scales and clinical evaluations were performed at 1 and 3 months. Bivariate and multivariate analyses were conducted for very good outcome defined by mRS 0-1, Hughes scale ≥2, and Barthel scale at 1 and 3 months to determine the best scale. Forty-three patients (mean age 46±16 years), 61% male. At admission, 72% had Hughes scale ≥4. At 1 month, 20 patients recovered gait, 9 achieved very good functionality, and 3 returned to work. Predictors associated were primary caregiver (children or partner) and lower score on the mEGOS gait recovery scale ($R^2=0.565$). At 3 months, 36 patients recovered gait, 17 reached very good functionality, and 24 returned to work. Predictors were deltoid strength <3 by MRC and amplitudes ≥20% of the minimum value of the median nerve ($R^2=0.306$). In conclusion the mRS is useful for assessing functionality beyond gait, providing a more comprehensive view. Recovery of upper limb function is an Important factor.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Administration of Anti-inflammatory Peptides in EAE Mice and Disease Progression Analysis

Hernández -Doroteo, Adriana [1]; Cabrera-Rivera, Graciela Libier [2];
González-Mireles, Verónica [1]; Rodríguez-Martínez, Sandra [1].

[1]Departamento de Inmunología, Laboratorio de Inmunología Aplicada, Escuela Nacional de Ciencias Biológicas del Instituto Politécnico Nacional. Campus Santo Tomás, Ciudad de México. [2]Investigador en ciencias médicas “C”, Departamento de infectología e inmunología, Instituto Nacional de Perinatología, Ciudad de México.

E-mail: anairda8520@gmail.com

Multiple sclerosis (MS) is an autoimmune disease characterized by the infiltration of immune system cells into the central nervous system (CNS), leading to an inflammatory reaction and, consequently, demyelination and variable damage to axons and neurons. The purpose of this study is to develop a passive model of experimental autoimmune encephalomyelitis (EAE), to test the peptides pCCR2 and pTNF, and to analyze disease progression. Female C57BL/6 mice were used and immunized with an emulsion containing MOG_{35-55} (100 µg) and CFA (100 µg); in addition, pertussis toxin (250 ng) was administered. Subsequently, lymph nodes and spleen were collected, and a cell suspension was obtained and stimulated in vitro with MOG_{35-55} (64 µg/µL) and PMA (50 ng/µL) for 3 hr. A total of 5×10^5 activated cells were transferred into C57BL/6 recipient mice, which also received the emulsion and pertussis toxin. Daily clinical follow-up was performed, and the presence of $CD4^{+}IL\text{-}17^{+}$ cells in peripheral blood samples was assessed by immunofluorescence on days 0, 6, 12, and 21. Brain, bone marrow, and spleen were collected, from which histological sections will be prepared to observe cellular infiltration and demyelinated areas. An early expansion of Th17 cells was observed on day 6, with high individual variability, reflecting the heterogeneity of the model. This activation was not associated with weight loss but did correlate with an increase in clinical score. On day 12, an increase in $IL\text{-}17^{+}$ neutrophils was detected, again without weight loss but with worsening clinical signs, suggesting their role in inflammatory progression. In conjunction, these findings reinforce the key role of Th17 cells in MS pathophysiology and open the door to testing pCCR2 and pTNF peptides in this model.

TRABAJO NO PRESENTADO

Área del artículo: **Inmunología de enfermedades infecciosas**

Impact of CRR3 Inhibition on Neutrophil Extracellular Trap Formation and Proinflammatory Cytokine Release in Influenza Infection

Hernández -González, David [1]; López-Leal, Fátima [1]; Medina-Reyes, Estefany Ingrid [2]; Cabellos-Avelar, Tecilli [1]; Pérez -López, Araceli [1].

[1]Universidad Nacional Autónoma de México, Facultad de Estudios Superiores Iztacala, Laboratorio de Inmunología e Interacciones Microorganismo-Hospedero, UBIMED, Estado de México, Tlanepantla, México. [2]Universidad Nacional Autónoma de México, Facultad de Estudios Superiores Iztacala, Laboratorio de Carcinogénesis y Toxicología, UBIMED, Estado de México, Tlanepantla, México.

E-mail: greenterror.dhg@gmail.com

The production of neutrophil extracellular traps (NETs) is a key mechanism of the innate immune response, particularly in defending against infections in lung tissue, including influenza A virus (IAV). Our research group has shown that neutrophils recruited to the lung express CCR3, and its blockade reduces their translocation to the alveoli. This study aimed to evaluate the role of CCR3 in NET formation and proinflammatory cytokine release during IAV infection. We used two pharmacological strategies to block CCR3 in a murine model: systemic administration of a specific CCR3 antagonist (SB328437) or a CCR3-blocking antibody (anti-CCR3). C57BL/6 mice were intranasally infected with IAV, and SB328437 or anti-CCR3 was administered intraperitoneally at 36 and 72 hours post-infection. Lung tissue and bronchoalveolar lavage (BAL) were collected at 96 hours. Lung tissue was processed to assess damage using hematoxylin and eosin staining, and NET formation was evaluated by immunofluorescence using citrullinated histone and myeloperoxidase as markers. The BAL was analyzed to determine IL-6, TNFα, and IL-1β concentrations by ELISA. Our results revealed that administration of SB328437 or anti-CCR3 reduced tissue damage in mice infected with IAV. Blocking CCR3 using either strategy reduced NET production in the lungs during IAV infection by approximately 2.5-fold. In line with these findings, we observed a reduction in pro-inflammatory cytokine concentrations when CCR3 was blocked, with a significant decrease in TNFα levels in mice treated with anti-CCR3. Additionally, there was a significant positive correlation between NET formation and IL-6 and TNFα levels. In conclusion, CCR3 blockade reduces NET formation and pro-inflammatory cytokine production during acute lung inflammation, highlighting its potential as a therapeutic target in respiratory diseases that involve inflammation.

Acknowledgments: This project was funded by CONAHCYT (now SECIHTY), project CBF2023-2024-1378, and COMECYT, project EDOMÉX-FICDTEM-2021-054.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Prevalent HLA-DRB1 alleles in México confer protection against symptomatic SARS-CoV-2 infection.

Hernández-Hernández, Eric Gregorio [1]; Daza-Mendoza, Diego Antonio [1]; González-Rivas, Enrique [1]; Serrano-Vázquez, Angélica [1]; Pérez-Juárez, Horacio [1]; Uribe-García, Alina [2]; Figueroa-González, Gabriela [3]; Reyes-Hernández, Octavio Daniel [3]; Morán-Silva, Patricia Belem [1]; Rojas-Velázquez, Liliana [1]; Ximénez-García, Cecilia [1].

[1]Laboratorio de Inmunología, Unidad de Investigación en Medicina Experimental "Dr. Ruy Pérez-Tamayo", Facultad de Medicina, UNAM. [2]Unidad de Biotecnología y Prototipos (UBIPRO), FES-Iztacala, UNAM. [3]Laboratorio de Farmacogenética, Unidad Multidisciplinaria de Investigación Experimental Zaragoza (UMIEZ), FES-Zaragoza, UNAM.

E-mail: ericghdz@yahoo.com.mx

The COVID-19 global pandemic, caused by the SARS-CoV-2 virus, left numerous damages around the world. Mexico was one of the most affected countries, with a high number of infections, hospitalizations and deaths. In Mexico, the clinical manifestation of the disease varied significantly between individuals. Several factors can influence susceptibility to SARS-CoV-2 and disease progression. In particular, the highly polymorphic genes of the HLA system play a fundamental role in the presentation of peptide antigens to T-lymphocytes, modulating the immune response and consequently, the course of various infectious diseases such as COVID-19. Previous studies have identified associations between alleles of the HLA-DRB1 locus and SARS-CoV-2 infection in the Mexican population; however, these studies remain scarce and do not encompass the country's broad genetic diversity. Therefore, the objective of this study was to analyze the association between HLA-DRB1 genetic polymorphism and susceptibility and/or resistance to symptomatic SARS-CoV-2 infection in patients residing in the metropolitan area of the Valley of Mexico. Approved protocol number by UNAM: FM/DI/059/2023. Individuals with a confirmed diagnosis of COVID-19 were recruited (n= 90), as well as individuals with no history of infection and/or symptoms (n= 60). The HLA-DRB1 locus was typed using next-generation sequencing (NGS). We found a significant increase in the frequency of the HLA-DRB1*08:02 allele in the control group (OR: 0.5; 95% CI: 0.28–0.91; p = 0.0236). However, this association did not remain significant after Bonferroni correction. On the other hand, multivariate analysis showed that the HLA-DRB1*14 allele group was significantly associated with protection against symptomatic infection (OR = 0.35; 95% CI: 0.16–0.76; p = 0.008). These data suggested that HLA-DRB1*08:02 and HLA-DRB1*14, prevalent alleles in the Mexican population, could confer a protective effect against symptomatic SARS-CoV-2 infection in individuals from the metropolitan area of the Valley of Mexico. Supported by DGAPA-PAPIIT IV200420, IN219624.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Immunological Evaluation of an mRNA Vaccine Against Salmonella Typhimurium in Mice

Hernández-Jiménez, Daniela [1 2]; Rivera-Hernández, Tania [1 3]; García-Valeriano, María Guadalupe [1]; Jiménez-Rosas, Alfonso Enrique [1 4]; Lozano -Cisneros, Diego [1]; Pérez-Vergara, Ana Delia [1]; Liu-, Jun [5]; Cunningham, Adam [5]; Martín-Orozco, Natalia [6]; López-Macías, Constantino [1].

[1]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, del Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México. [2]Facultad de Química, Universidad Nacional Autónoma de México, Ciudad de México, México. [3]Programa Investigadores por México, Secretaría de Ciencia, Humanidades, Tecnología e Innovación. Ciudad de México, México. [4]Unidad Profesional Interdisciplinaria de Biotecnología, Instituto Politécnico Nacional, Ciudad de México, México. [5]Bacterial Vaccines Network, Immunology and Immunotherapy, University of Birmingham, Birmingham, United Kingdom.. [6]Providence Therapeutics Holdings Inc. Toronto, Canadá.

E-mail: danielah2412z@gmail.com

Gastroenteritis, one of the clinical syndromes associated with salmonellosis, is primarily caused by non-typhoidal Salmonella (NTS), with Salmonella Typhimurium being among the most prevalent serotypes. Vulnerable populations, such as children and immunocompromised individuals, are particularly at risk. The rise of antibiotic-resistant strains in both animal reservoirs and clinical isolates has led to an alarming increase in NTS cases, underscoring the urgent need for preventive strategies, including vaccine development. This study presents two mRNA-based vaccine candidates encoding the protective antigen DRT831424, with the aim of evaluating their immunogenic potential in a murine model. Specific IgG antibody titters were assessed by ELISA following two immunizations. The results showed that neither mRNA vaccine candidate elicited a significant antibody response compared to the group immunized with purified DRT831424 protein, which induced a robust antibody response. The protective efficacy of the vaccines was evaluated through an intraperitoneal challenge with a known dose of S. Typhimurium, by quantifying bacterial dissemination in spleen, liver, and blood samples. No protection was observed in the mRNA-immunized groups, as bacterial loads in the liver and spleen were comparable to those in the negative control group. In contrast, mice immunized with purified DRT831424 protein or inactivated bacteria exhibited a significant reduction in bacterial burden in the liver and spleen. These findings suggest that the current mRNA vaccine formulations did not elicit sufficient humoral protective immune responses. We further assessed vaccine protection using an intragastric infection model, where cellular immune responses can play a key role in protection. Survival of mRNA immunized groups was comparable to DRT831424 protein and higher compared to the negative control group, although this difference did not reach statistical significance. Subsequent studies are needed to confirm these observations and determine whether the mRNA vaccine formulations are eliciting a functional cellular response.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

The immune response modifies the allatotropin levels in Aedes aegypti

Hernández-Martínez, Salvador [1]; Noriega-, Fernando Gabriel [2];
Lozano-Moreno, José Manuel [3]; Guzmán-Rabadán, Karla Karina [1].

[1]Research Center for Infectious Diseases. National Institute of Public Health. Cuernavaca, Morelos, México.. [2]Florida International University, Miami, FL, USA.. [3]Pharmacy Department, National University of Colombia, Bogotá, Colombia.

E-mail: shernandez@insp.mx

Recently, ex vivo studies indicate that allatotropic neuropeptide allatoropin (AT) modulates immune responses in two important mosquito disease vectors, Anopheles albimanus and Aedes aegypti. Here we evaluate changes in AT levels in ventral nerve ganglia (VNG), hemolymph and brain of Ae. aegypti adult females after different immune challenges and during a Dengue virus infection. Samples were obtained from sugar- or blood-fed mosquitoes at different ages or times post-injection with different microorganisms, including Dengue virus (DV) infections. The evaluation of AT was performed by an indirect ELISA. The highest AT level was observed in VNG and hemolymph at 0h post-emergence (28.9±7 and 95.8±6 fmols/mosquito respectively). In sugar-fed, the lowest values were at 6-7 days-old (4.3±2 and 50.2±12 fmols/mosquito respectively). In blood-fed mosquitoes of 5-days-old, the highest AT values in VNG and hemolymph were at 6h post-blood-fed (55.8±5 and 148.8±5 fmol respectively) and the lowest at 72h (3.8±0.5 and 46.9±11 fmols respectively). In brains, from 0 to 7-days-old, the highest AT values were at 4 days-old (19.8±3.7 fmol/brain). The immune challenge was performed in sugar-fed females of 5 days-old. In VNG and hemolymph important changes were observed at 2h post-challenge with different inactivated microorganisms (including inactivated DV) and during DV infection. Interestingly, no changes in AT levels from brains were observed in the immune challenge assays. In conclusion, AT levels in VNG and hemolymph change with age, nutritional status and during the mosquito immune response; these changes were directly proportional in both tissues, suggesting that the AT in hemolymph comes from VNG.

This work was supported by a Conahcyt Grant CBF2023-2024-827 to S.H.M.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Effects of fermentation products from Agave tequilana inulin on allergic inflammation

Hernández-Mercado, Alicia [1]; Ibarra-Sánchez, Alfredo [2];
Salinas-Miralles, Eva [3]; González-Espinosa, Claudia [1 2].

[1]Centro de Investigación y de Estudios Avanzados (Cinvestav) del Instituto Politécnico Nacional (IPN) Sede Sur, Departamento de Farmacobiología, Ciudad de México, CDMX, México.. [2]Centro de Investigación y de Estudios Avanzados (Cinvestav) del Instituto Politécnico Nacional (IPN) Sede Sur, Centro de Investigación sobre el Envejecimiento (CIE), Ciudad de México, CDMX, México.. [3]Universidad Autónoma de Aguascalientes, Departamento de Microbiología, Centro de Ciencias Básicas, Aguascalientes, Aguascalientes, México.

E-mail: alicia.hernandez@cinvestav.mx

Type I hypersensitivity reactions (allergies) are initiated by allergen-induced immunoglobulin E (IgE), which binds to the high-affinity IgE receptor (FcεRI) at the surface of mast cells (MCs). Upon subsequent exposure to the allergen (antigen, Ag), complexes formed by IgE/Ag promote FcεRI crosslinking and activate a complex signaling cascade leading to MCs degranulation and the release of preformed mediators such as histamine, TNF, and β-hexosaminidase, inducing inflammation. Fructose-based dietary fibers, such as inulin, are indigestible by human enzymes but are metabolized by gut microbiota, producing fermentation products (FPs) like vitamins, neurotransmitters, and short-chain fatty acids (SCFAs). SCFAs travel from the gut to the bloodstream, exerting multiple effects on the immune system. Notably, they inhibit histone deacetylases (HDACs), suggesting that epigenetic mechanisms are involved in some of their actions. In this project, we administered Agave tequilana inulin as an oral pretreatment to C57BL6/J mice and determined its effect in a local inflammatory reaction using a passive cutaneous anaphylaxis (PCA) model. Inulin consumption reduced inflammation in a dose-dependent manner, except at the highest concentration. To evaluate the participation of FPs in those effects, we harvested mouse feces to obtain their microbiota and fermented them with inulin in vitro under anaerobic conditions. FPs were then purified and used to pretreat bone marrow mast cells (BMMCs) for 24h. FPs treatment reduced the FcεRI-induced release of β-hexosaminidase. The inhibitory effects of one of the FPs were analyzed by performing long and short-term exposure of BMMCs to sodium Butyrate (Bt). A 24h-long treatment decreased FcεRI-induced and phorbol-myristate acetate/A23187-induced degranulation of BMMCs. Bt treatment reduced FcεRI γ subunit protein expression and blunted calcium mobilization. Fifteen min-long exposure to Bt also reduced BMMCs degranulation, affected calcium mobilization and increased tubulin acetylation. This result indicates that FPs, like butyrate, alter signaling pathways involved in MC activation.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

Combinatorial neuroprotective therapy in a rat model of traumatic spinal cord injury

Hernández-Moran, David [1 2]; Toscano-Zapien, Alberto [4]; Blancas-Espinoza, Liliana [1]; Flores-Romero, Adrián [4]; Sánchez-Torres, Luvia Enid [3]; Meza-Calzada, Claudia Ivette [1 5]; Cruz-Cruz, Daniel [1 6]; Troncoso-Avilés, Ilse Pamela [1 6]; Silva -García, Raúl [1].

[1]Instituto Mexicano del Seguro Social, Unidad de Investigación Médica en Inmunología, Hospital de Pediatría, Centro Médico Nacional Siglo XXI, Ciudad de México, México. [2]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Departamento de Inmunología, Ciudad de México, México. [3]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Laboratorio de Inmunología de los Microorganismos, Ciudad de México, México. [4]Universidad Anáhuac Norte, Facultad de Ciencias de la Salud, Ciudad de México, México. [5]Universidad Nacional Autónoma de México, Facultad de Ciencias, Ciudad de México, México. [6]Universidad Nacional Autónoma de México, Facultad de Estudios Superiores Zaragoza, Ciudad de México, México.

E-mail: hrnzmorandavid@gmail.com

Traumatic spinal cord injury (TSCI) is a catastrophic event leading to motor/sensory impairments and autonomic nervous system damage. It triggers primary neuronal damage from physical impact, followed by a secondary phase characterized by ischemic necrosis, oxidative stress, and dysregulated inflammation. These mechanisms hinder neuroregeneration chronically, with no current safe and effective pharmacological therapies available. Therefore, we evaluated a combinatorial approach using Monocyte Locomotion Inhibitory Factor (MLIF) plus the antioxidant Q832 as neuroprotective agents, delivered via a hydrogel-based polymeric scaffold. This strategy aimed to enhance neuroprotection in TSCI rats by modulating the neurotoxic microenvironment and inflammatory response to promote functional recovery. Sprague Dawley rats underwent TSCI and received different treatment combinations. At 4 weeks post-injury, glial scar formation and neuroregeneration-associated markers were assessed via digital PCR, with translation products analyzed by immunofluorescence. Motor recovery was evaluated over 8 weeks. Treated groups showed improved motor recovery scores versus untreated controls. Differential expression was observed in glial scar-associated genes (GFAP, Iba1), along with variations in the neuroregeneration marker GAP43 and the neurodegeneration marker Nogo-A.

Área del artículo: **Inmunidad innata**

Unmasking a Tumor Suppressor Role: mMGL1 Loss Enhances Anti-Tumor Inflammation in Colorectal-Cancer

Hernández- Navia, Sonia Elizabeth [1]; Flores-Cortez, Layla Sabrina [1]; Martínez-García, Brandon Alexander [1]; Rodríguez-Sosa, Miriam [1].

[1]Laboratorio de inmunidad innata, UBIMED, Av. De los Barrios 1, Los Reyes Iztacala, Tlalnepantla, Edo. de México, 54090, México.

E-mail: so.navia@ciencias.unam.mx

Colorectal cancer is the third most prevalent cancer worldwide. It develops in the colon and rectum, leading to clinical manifestations such as bleeding, diarrhea, and weight loss, along with the consequent development of polyps and tumors which exhibit cancer-associated aberrant glycosylation. This glycosylation pattern can be recognized by the galactose-type C-type lectin 1 (mMGL1) receptor. However, whether this interaction modulates the anti-tumor immune response remains unclear. The aim of our study was to evaluate the role of mMGL1 in the immune response during colitis-associated colorectal cancer (CAC). The development of azoxymethane/dextran sodium sulfate-induced tumor was compared between Mgl1 knockout (Mgl1-/-) and wild-type (WT) C57BL/6 mice. Our results show that the Mgl1-/- mice with CAC exhibited a lower disease activity index and developed fewer tumors compared to WT CAC mice. Western blot analysis revealed increased activation of NF-κB and decreased ERK1/2 phosphorylation in the colon tissue of Mgl1-/- mice with CAC. Furthermore, macrophages (Mφ) from WT mice stimulated with tumor antigen produced higher levels of IL-12 and TNF-α compared to unstimulated WT macrophages. These findings suggest that the absence of mMGL1 partially suppresses colorectal tumorigenesis, likely through enhanced pro-inflammatory and anti-tumor immune responses. This highlights the potential immunomodulatory role of mMGL1 in the tumor microenvironment and highlighting the importance of further investigation into its therapeutic implications.

FUNDING: Sonia Elizabeth Hernández Navia received a scholarship from Estancias Posdoctorales por México, 2024, SECIHTI), CVU 537911.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

Comparative efficacy of Tocilizumab versus Azathioprine and other empirical disease - modifying drugs.

Hernández-Villegas, Aldo [1].

[1]Estudiante de octavo semestre de Medicina General en la Universidad de Durango, campus Chihuahua, Chihuahua, México.

E-mail: aldohdz7113@gmail.com

Neuromyelitis optica spectrum disorder (NMOSD) is a severe autoimmune demyelinating disease of the central nervous system characterized by recurrent attacks predominantly targeting the optic nerves and spinal cord, often leading to significant disability. The pathogenic hallmark involves IgG autoantibodies against aquaporin-4 (AQP4-IgG), with interleukin-6 (IL-6) playing a central role in disease pathogenesis by promoting plasmablast-mediated antibody production and sustaining neuroinflammation. Tocilizumab, a humanized monoclonal antibody targeting the IL-6 receptor, has been proposed as a targeted immunotherapeutic option. This systematic review identified two randomized controlled trials (RCTs) encompassing 159 patients that evaluated the efficacy and safety of tocilizumab in NMOSD compared to conventional immunosuppressive therapies. The methodological quality was assessed using the Cochrane Risk of Bias 2.0 tool, revealing high risk in key domains such as randomization and outcome measurement. Despite these limitations, both RCTs consistently reported a reduction in relapse rates and an acceptable safety profile associated with tocilizumab treatment. The limited number of RCTs and heterogeneity in study design, population, and comparators precluded meta-analytic synthesis. These findings highlight the need for further large-scale, multicenter RCTs with rigorous methodology, including stratification by antibody serostatus (AQP4-IgG versus MOG-IgG), optimization of dosing regimens, and evaluation of biomarker correlates. This review consolidates current evidence supporting IL-6 receptor blockade as a promising therapeutic strategy in NMOSD and delineates critical knowledge gaps for future research.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Extracellular Ubiquitin induces monocyte activation in a CXCR4-independent manner

Herrera-Torres, Estefanía [1 2 3]; Mendoza-Salazar, Ivette [1 2 3]; Fragozo, Ana [1 2]; Aguilar-Alonso, Francisco Antonio [1 2]; González, Aneth [1 2]; Pavón, Lenin [4]; Vallejo-Castillo, Luis [1 2]; Almagro, Juan Carlos [1 2 5]; Pérez-Tapia, Sonia Mayra [1 2 3].

[1]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, México City, México. [2]Laboratorio Nacional Para Servicios Especializados de Investigación, Desarrollo e Innovación (I + D + I) Para Farmoquímicos y Biotecnológicos, LANSEIDI-FarBiotec-CONACyT, México City, México. [3]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, México City, México. [4]Laboratorio de Psicoinmunología, Dirección de Investigaciones en Neurociencias, Instituto Nacional de Psiquiatría Ramón de la Fuente Muñiz, México city, México. [5]Global Bio, Cambridge Massachusetts, United States.

E-mail: estefa_h@hotmail.com

Ubiquitin (Ub) is a 76 amino acids protein that is widely present in eukaryotes and shows a significant level of similarity across different species. The first reported biological function of Ub was found to be extracellular, but its significance rapidly increased due to its intracellular role in post-translational modification of intracellular proteins (ubiquitination). In subsequent years, the extracellular function of Ubiquitin (Ub) was given less attention, until an association was noticed between a greater rate of survival and elevated levels of Ub in the bloodstream of patients with sepsis and burns. CXCR4 has been suggested as a receptor for extracellular ubiquitin (eUb), but this idea is still controversial. It has been reported that eUb is internalized in monocytes, however its biological effects on these type cells remain to be elucidated. In this study, flow cytometry and confocal imaging assays revealed that eUb binding and internalization to cells is CXCR4 independent. Moreover, eUb activates THP-1 monocytes in a CXCR4 independent manner, which results in the upregulation of proinflammatory (TNF-α, IL-6, and CCL3) and anti-inflammatory (IL-10) cytokines gene expression (determined by qRT-PCR), and promoted an increase of secreted TNF-α, IL-6, and IL-10 determined by CBA. These findings contradict prior research showing that eUb stimulates CXCR4 and suggests that CXCR4 is not the solely receptor for eUb.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Modelling the pro- and anti-inflammatory macrophage differentiation: role of NFkB and CREB

Huerta-Hernández, Leonor [1]; Martínez-Méndez, David [1 3];
Aguilar-Elguea, Ilean Z. [2]; Castelán-Pacheco, Lilian Sofi [4];
Jiménez Alvares, Luis [5]; Cruz-Lagunas, Alfredo [5]; Zúñiga-Ramos, Joaquín [5 6]; Villarreal, Carlos [2].

[1]Instituto de Investigaciones Biomédicas, Departamento de Inmunología, UNAM, CDMX, México. [2]Instituto de Física, Departamento de Física Cuántica y Fotónica, UNAM, CDMX, México. [3]Secretaría de Ciencia, Humanidades, Tecnología e Innovación. [4]Universidad Autónoma Metropolitana, Unidad Cuajimalpa, CDMX, México. [5]Instituto Nacional de Enfermedades Respiratorias Daniel Cosío Villegas, Unidad de Investigación, Secretaría de Salud, CDMX, México . [6]Tecnológico de Monterrey, Escuela de Medicina y Ciencias de la Salud, CCM, CDMX, México.

E-mail: leonorhh@iibiomedicas.unam.mx

Integrating the complexity of monocyte intracellular signalling into a mathematical model can support the understanding of dynamic transitions that are crucial for immune regulation. We formulated a comprehensive mathematical model of monocyte activation, differentiation, and metabolic adaptation to simulate its response to different stimuli and cytokine microenvironments. The model comprises a 128-node complex regulatory network composed on the base of a review of experimental literature. Node interactions were described by logic rules which were translated to a set of ordinary differential equations (ODEs) system. The network includes signalling events induced by LPS, ssRNA, activating IgG immune complexes, and the IFN-gamma, IL-4 and IL-10 cytokines. Autocrine feedback loops for IL-10 and TNF-alfa, and a metabolism subnetwork were included. The system outputs describe the dynamics of cell metabolic activity, activation of transcription factors, cytokine production and phagocytosis.

Results: States corresponding to the M1, M2, M2b and M2c macrophage profiles were obtained. The predominance of glycolysis and oxidative phosphorylation in the M1 and M2 responses, respectively, was reproduced. Modelling highlights the convergence to the activation of the NF-kB transcription factor in the pro-inflammatory response, while anti-inflammatory profiles are related to the induction of CREB, a NF-kB inhibitor and promoter of IL-10 synthesis. Modelling agrees with the activation of the CREB inhibitor GSK3-beta by IFN-gamma induced signals, which then enables the pro-inflammatory response. The inhibition of NF-kB by IL-4 signalling can turn the response into an M2 profile. Results align closely with experimental observations reported in the literature. Conclusion: The model constitutes an integrative framework to examine how signals from diverse cytokine and antigenic environments shape macrophage pro-inflammatory or anti-inflammatory responses. An interactive program of the model is available online upon request.

Supported by grants IG200623 from PAPIIT-UNAM and CBF2023-2024-348 from SECIHTI, México.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Effects of the oral administration of Cry1Ac toxin in gut mucosal tissue

Ilhuicatzi -Alvarado, Damaris [1]; Hernández-Rivera, Leonel [1];
Bautista-Jacobo, Itzel Saraí [1]; Moreno-Fierros, Leticia [1].

[1]Facultad de Estudios Superiores Iztacala, UNAM, Unidad de Biomedicina, Tlalnepantla Edo. de México, México.

E-mail: damarisialvarado4@gmail.com

The Bacillus thuringiensis (BT) Cry1Ac toxin are widely used as biopesticides and can be expressed by genetically modified plants for human and animal consumption like BT crops. Cry1Ac is not toxic for vertebrates; however, previous studies from our group have shown that recombinant Cry1Ac toxin is able to activate macrophages and B lymphocytes. Besides Cry1Ac toxin was shown to be moderately allergenic when it was intragastrically administrated for two months, once a week, provoked hyperplasia in the large intestine. Here we evaluated the intestinal inflammatory potential of Cry1Ac toxin. For these experiments we used as positive control a model of acute intestinal inflammation induced with DSS 3% for 7 days. Mice were administered intragastrically with i) vehicle phosphate-buffered saline, ii) Cry1Ac toxin 2mg/ml iii) Cry1Ac toxin 20 mg/ml iv) Cry1Ac toxin 50 mg/ml v) DSS 3% for 7 days. After 7 days we evaluated the characteristics like blood in feces, large intestine size, weight loss, inflammatory production of cytokines in explants like IL-6, TNF-a, CXCL2, IFN-g, IL-10 and TGF-b and histopathologic changes were evaluated at intestinal and systemic level. The Cry1Ac administered experimental groups; developed poor inflammation related reactions. While only the positive group DSS 3% presented severe inflammatory features such as blood in feces, weight loss, and reduction of intestinal size with high inflammation score of inflammation while with Cry1Ac treatments the inflammation score was null. The measure of cytokine levels in the Cry1Ac administered groups in explants showed significant production of CXCL2 and reduction of IL10 levels with respect to the vehicle group. The histopathological analysis in these Cry1Ac administered groups revealed edema with apparent recruitment of neutrophils. The outcomes sustain that via intragastric administration Cry1Ac toxin does not provoke severe inflammation, but it is not innocuous.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Evaluation of MAMDC2 protein as a potential therapeutic target in Chronic Myeloid Leukemia

Islas-Saldívar, Guillermo Alejandro [1 2]; Aguilar-Serafín, Gloria Josceline [1]; Damián-Morales, Gabriela [1 2]; Ángeles-Arvizu, Adriana [1 2]; González-Chávez, María Antonieta [3]; Almagro, Juan Carlos [1 2 4]; Pérez-Tapia, Sonia Mayra [1 2 5].

[1]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, México City, México. [2]Laboratorio Nacional Para Servicios Especializados de Investigación, Desarrollo e Innovación (I+D+i) Para Farmoquímicos y Biotecnológicos, LANSEIDI-FarBiotec-CONACyT, Mexico City, Mexico. [3]Unidad de Investigación Médica en Enfermedades Oncológicas. CMN Siglo XXI. IMSS. [4]Global Bio, Cambridge Massachusetts, United States. [5]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, México City, México.

E-mail: a1606.alejandro@gmail.com

Chronic myeloid leukemia (CML) is a myeloproliferative disease characterized by the uncontrolled growth of myeloid cells at all stages of their development. In Mexico it accounts for about 10% of all cases of leukemia. Current treatments include tyrosine kinase inhibitors (TKIs), allogenic bone marrow transplants, and the use of interferon-α and hydroxyurea. Biomarkers can also be used as therapy targets because they identify abnormal cells or cellular processes which characterize the disease. MAMDC2 has been associated with many types of cancer and proposed as a secreted protein with tumor growth inhibiting functions like decreasing MAPK signaling. However, in the case of CML its role is unknown and to date its expression has only been evaluated at RNA level. In this study, we aimed to evaluate MAMDC2 as therapeutic target for CML. Our results show that MAMDC2 is expressed in CML cells, particularly in the K562 cell line, which is commonly used to study CML. Moreover, we demonstrated that opsonization with anti-MAMDC2 antibody induces phagocytosis of CML cells by immune cells. Additionally, we found that MAMDC2 is highly expressed in bone marrow mononuclear cells from CML patients, but not in those with Acute Lymphoblastic Leukemia (ALL) or in healthy controls. Our findings suggest that MAMDC2 could be a potential therapeutic target for CML stem cells and a biomarker for distinguishing LSCs from hematopoietic stem cells. Further studies are needed to explore the role of MAMDC2 in CML and its potential applications in the development of targeted therapies.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Isthmin1 Upregulation in the Intestinal Microenvironment During Salmonella Typhimurium Infection

Jaimes-Ortega, Gustavo Alberto [1 2 3]; Angeles-Floriano, Tania [4]; Rivera-Torruco, Guadalupe [5]; Almanza-Pérez, Julio Cesar [6]; Xicohtencatl-Cortes, Juan [7]; Hernández-Cuellar, Eduardo [8]; Medina-Contreras, Oscar [9]; Cruz-Córdoba, Ariadnna [10]; Valle-Ríos, Ricardo [1 3].

[1]Immunology and Proteomics Research Laboratory, Children's Hospital of México "Federico Gómez" (HIMFG), México City 06720, México. [2]Experimental Biology Postgraduate Program, Department of Biological and Health Sciences, Metropolitan Autonomous University (UAM), México City 09310, México. [3] University Research Unit UNAM-HIMFG, Research Division, School of Medicine, National Autonomous University of México, México City 04510, México. [4]Subdirección de Diagnóstico Clínico y Departamento de Laboratorio Clínico, Hospital Infantil de México Federico Gómez, México City 06720, México. [5] Department of Laboratory Medicine, Medical Center, University of California, San Francisco, CA 94102, USA. [6]Departamento de Ciencias de la Salud, Universidad Autónoma Metropolitana Unidad Iztapalapa, Av. San Rafael Atlixco 186, Col. Leyes de Reforma 1A Sección, Alcaldía Iztapalapa, Ciudad de México 09310, México. [7]Laboratorio de Investigación en Bacteriología Intestinal, Unidad de Investigación en Enfermedades Infecciosas, Hospital Infantil de México Federico Gómez, Ciudad de México 06720, México. [8]Laboratorio de Biología Celular y Tisular, Departamento de Morfología, Universidad Autónoma de Aguascalientes, Aguascalientes 20100, México. [9]Epidemiology, Endocrinology & Nutrition Research Unit, Children's Hospital of México "Federico Gómez", México City 06720, México. [10]Laboratorio de Investigación en Inmunoquímica, Hospital Infantil de México Federico Gómez, México City 06720, México.

E-mail: gelet321@gmail.com

Isthmin1 (ISM1) is a constitutively secreted cytokine produced by barrier tissues and different immune cell types. Importantly, ISM1 is also expressed by cells with a hematopoietic stem cell phenotype in the lung and plays a role during hematopoiesis. Under inflammatory conditions, ISM1 levels are also altered. Given that the intestine is one of the tissues with the highest ISM1 gene expression, in this work, we characterized the immunophenotype of ISM1-producing cells in the small intestine of uninfected or Salmonella Typhimurium-infected mice. We found a variety of ISM1+-expressing cells, including CD45+ cells, EpCAM+ cells, and, importantly, we also found different subsets of cells carrying hematopoietic stem cell markers (LSKs) expressing ISM1, and their frequency was perturbed during infection. Finally, we also found that isthmin1 is secreted into the intestinal lumen, and its concentration was increased during S. Typhimurium infection. Our findings suggest that increased luminal ISM1 secretion during mucosal infection may serve as a potential novel biomarker of pathogen-mediated inflammation.

This research was funded by Fondos Federales, grant numbers HIM/2019/044 SSA 1598 and HIM/2022/056 SSA1839.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Evaluation of a vaccine prototype against Salmonella Typhimurium in mice

Jiménez -Rosas, Alfonso Enrique [1 2]; García-Valeriano, María Guadalupe [1]; Castrejón-Flores, José Luis [2]; Pérez-Vergara, Ana Delia [1]; Hernández-Jiménez, Daniela [1]; Vicencio-Hernández, Erick Raúl [1]; López-Macías, Constantino [1]; Rivera-Hernández, Tania [1 3].

[1]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades del Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México. [2]Unidad Profesional Interdisciplinaria de Biotecnología, Instituto Politécnico Nacional. Ciudad de México, México. [3]Investigadores por México, Secretaría de Ciencia, Humanidades, Tecnología e Innovación. Ciudad de México, México.

E-mail: alfonsojmz14@gmail.com

Non-typhoidal salmonellosis (NTS) caused by Salmonella Typhimurium remains a major public health concern, particularly in low- and middle-income countries. In Mexico, the National Epidemiological Bulletin reported over 30,000 cases of NTS in 2024; however, this figure likely underestimates the true burden of disease. The increasing emergence of multidrug-resistant strains further highlights the urgent need for effective preventive strategies. Currently, no licensed vaccine is available for NTS, underscoring the importance of exploring alternative approaches. In this context, Salmonella porins have shown immunogenic potential and represent a promising target for vaccine development.This study aimed to evaluate the immunogenicity and protective efficacy of a vaccine prototype based on S. Typhimurium porins in a murine model. Specific antibody titres were quantified following primary and booster immunisations using ELISA, and the protective effect of the vaccine was assessed through a lethal intragastric challenge with S. Typhimurium. The immunisation protocol induced porin-specific antibodies, with a significant increase observed after the booster dose. A statistically significant difference in antibody titres was found between the porin-immunised and saline control groups. Moreover, vaccinated mice exhibited increased survival upon intragastric challenge compared to controls, a finding of particular relevance given that this route mimics the natural portal of entry for the pathogen.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Dioscorea remotiflora reduces the inflammatory response in experimental allergic urticaria

Jiménez-Vargas, Mariela [1]; Hernández-Villalobos, Martín de Jesús [1]; De los Santos-Jaramillo, Itmar Yaritzi [1]; Pinacho-Alvarado, Ana Victoria [1]; Córdova-Dávalos, Laura E. [1]; Gómez-Aguirre, Yenny Adriana [2]; Cervantes-García, Daniel [1 3]; Santacruz-Ruvalcaba, Fernando [4]; Salinas-Miralles, Eva [1].

[1]Department of Microbiology, Basic Science Center, Autonomous University of Aguascalientes, México. [2]Department of Chemestry, Basic Science Center, Autonomous University of Aguascalientes, México. [3]Secretariat of Science, Humanities, Technologies and Innovation, México City, México. [4]University Center for Biological and Agricultural Sciences (CUCBA), University of Guadalajara.

E-mail: mayojv@hotmail.com

Allergic urticaria is an adverse cutaneous reaction to common harmless substances known as allergens, mediated by allergen-specific IgE and mast cells. The onset of the disease is influenced by both genetic and environmental factors, highlighting the need for effective prophylactic strategies. Most current treatments are palliative, while preventive approaches tend to be expensive, time-consuming, and not suitable for all patients. Tubers from Dioscorea spp. have shown immunomodulatory, anti-inflammatory, and antioxidant properties, which vary depending on the species and environmental growing conditions. Dioscorea remotiflora is a poor-studied species endemic to Mexico. This study aimed to evaluate the prophylactic effect of D. remotiflora on experimental allergic urticaria. Tubers of D. remotiflora cultivated in Jalisco, Mexico, were washed, peeled, blanched, dehydrated, and pulverized. Two extracts were obtained from the resulting flour: alcoholic (ALD) and aqueous (ACD). Male Wistar rats weighing 150 g were divided into 5 groups: one group without allergy and treated with water, and four groups with allergy to ovalbumin (OVA) and treated with: water, ALD (20 mg/kg/day), ACD (20 mg/kg/day) and cetirizine (0.2 mg/kg). Treatments were administered daily and orally, starting 3 days before the first immunization and continuing until the day of urticaria evaluation. Blood samples were collected, and wheals were induced by intradermal OVA injection. Serum anti-OVA IgE levels were measured using passive cutaneous anaphylaxis assay, and cutaneous extravasation was assessed by the Evans blue dye method. Oral administration of ACD and ALD reduced the allergen-specific inflammatory response by 26% ($p<0.05$) and 37% ($p<0.001$), respectively, compared to untreated animals. Neither extract altered serum anti-OVA IgE levels. These findings suggest that prophylactic intake of D. remotiflora exerts an anti-inflammatory effect on allergic wheals, supporting its potential application in urticaria. Further studies are needed to elucidate the underlying mechanisms of action. Financing by Autonomous University of Aguascalientes (PIBB24-10).

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Leukocytes in human breastmilk: a scoping review

Jiménez-López, Brenda [1,2]; García-Alonso, Claudia Angélica [1,2];
Castaño-Duque, Sebastián [3]; Sánchez-Salguero, Erick [4];
Lampousi, Anna-María [5]; Brunck, Marion E. G. [2].

[1]Tecnologico de Monterrey, Escuela de Medicina y Ciencias de la Salud, Av. Eugenio Garza Sada 2501 Sur, Tecnológico, 64849 Monterrey, Nuevo León, México. [2]The Institute for Obesity Research, Tecnológico de Monterrey, Av. Eugenio Garza Sada 2501 Sur, Tecnológico, 64700, Monterrey, Nuevo León, México. [3]Hospital Universitario San Ignacio, Bogotá, D.C., Colombia. [4]Sir William Dunn School of Pathology, University of Oxford, Oxford U.K. [5]Institute of Environmental Medicine, Karolinska Institute, Stockholm, Sweden.

E-mail: marion.brunck@tec.mx

The immunological composition of breastmilk has gained interest as breastfeeding correlates with improved health outcomes for infants, short and long term. Within this field, breastmilk leukocytes (BreLeuk) are currently underexplored. In this scoping review, we collected, mapped and synthesized all available published literature on human BreLeuk to establish a State-of-the-Art. A systematic search across 4 databases from inception until February 10th 2025, yielded 216 relevant peer-reviewed research articles from 4336 collected abstracts. Overall, BreLeuk research has been dominated by high-income countries (80%), with minimal representation from low-income regions (1.7%). Colostrum has been the most studied milk type. Macrophages were the most reported BreLeuk with 110 associated studies, followed by T lymphocytes. Conversely, eosinophils and basophils were rarely studied and mast cells were investigated, but not found in human milk. Research has predominantly focused on analyzing BreLeuk relative proportions, immunophenotype, morphology, and phagocytosis/killing activity, and comparing to paired blood circulating leukocytes. Maternal demographics have been historically underreported, hampering correlations with BreLeuk composition, although recent studies have more consistently reported these variables. Over 63 % of studies evaluated BreLeuk characteristics in healthy mothers, versus conditions such as HIV (14 studies, 6.5%), allergies or obesity. We provide a comprehensive database of human BreLeuk identification and reported phenotypes and functions and propose a guide for systematic reporting of relevant variables, to facilitate future research.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Immunomodulation by bacterial products promotes innate signatures favorable to tuberculosis host response

Juárez-Carvajal, Esmeralda [1]; Romero-Rodríguez, Dámaris P [2]; Montes-Martínez, Eduardo [3]; Rocha-González, Héctor I [4]; Zúñiga-Ramos, Joaquín [5]; Torres-Rojas, Martha [6].

[1]Laboratorio de Alta Contención Biológica (LACBio), Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas. CDMX, México. [2]Laboratorio Nacional Conahcyt de Investigación y Diagnóstico por Inmunocitofluorometría (LANCIDI). Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas. CDMX, México. [3]Laboratorio de Biología Molecular. Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas. CDMX, México. [4]Sección de Estudios de Posgrado e Investigación, Escuela Superior de Medicina, Instituto Politécnico Nacional, CDMX, México. [5]Laboratorio de Inmunología y Genética, Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas, CDMX, México. [6]Laboratorio de Inmunobiología de la Tuberculosis, Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas, CDMX, México.

E-mail: esmeraldajc@yahoo.com

Despite widespread use of antibiotics and the BCG vaccine, tuberculosis remains a leading cause of death from infectious diseases globally, underscoring the need to boost innate responses in monocytes and macrophages to enhance early control of Mycobacterium tuberculosis infection. Trained immunity, a form of innate immune memory, enhances macrophage responsiveness through epigenetic and metabolic reprogramming, offering a promising approach to strengthen host defenses against M. tuberculosis. This study evaluated the immunomodulatory potential of pharmaceutical-grade bacterial suspension (BS, IPI bacterial suspension) and bacterial lysates (BL, Pulmonarom) in human monocyte-derived macrophages (MDM) and their role in innate response to M. tuberculosis infection. MDMs were stimulated with M. bovis BCG, BS, and BL following a training protocol described for BCG-dependent trained immunity. We observed that BS and BL induced sustained cytokine responses and a metabolic transcriptional profile upon secondary stimulation with M. tuberculosis. BS and BL promoted increased IL-1β and TNFα production in M. tuberculosis-infected macrophages. Also, the expression of surface markers was shifted to high HLA-DR, CD80, CD86, CD14, TLR2, and TLR9 expression consistent with an M1 phenotype. Moreover, BS and BL upregulated the expression of antimicrobial transcriptional signatures, including autophagy-related LC3 and ATG16L1 expression. These findings indicate that BS and BL engage immunomodulatory pathways, suggesting potential for tailored immune activation strategies in trained immunity. Given the central role of trained macrophages in the early containment of Mycobacterium tuberculosis, these immunological signatures may provide new adjunctive strategies to boost innate responses in tuberculosis and other chronic infections.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Evaluation of the inflammatory profile in a population of older adults with no clinical history of disease

Juárez-Rivera, Zafiro Belén [1]; Cristerna -Ortega, Ángel Andrés [1];
Tapia-Sánchez, Wilfrido [3]; Rosales-García, Víctor Hugo [2 3];
Santos-Argumedo, Leopoldo [1 4].

[1] Biomedicina Molecular, CINVESTAV-IPN, Ciudad de México, México.. [2]Unidad de Citometría, Laboratorio Nacional de Servicios Experimentales, CINVESTAV-IPN, Ciudad de México, México. [3]Unidad de Citometría de Flujo, Diagnóstico Molecular de Leucemias y Terapia Celular (DILETEC), Ciudad de México, México. [4]Centro de Investigación sobre el Envejecimiento, CINVESTAV-IPN, Ciudad de México, México.

E-mail: zafiro.belen22@gmail.com

The global population is aging at an accelerated rate. It is estimated that there are over 1 billion people aged 60 years or older worldwide, and this number is projected to increase by 16% by the year 2050. According to INEGI, 12% of the population in Mexico falls within this age group. In Mexico, at least fifty percent of individuals older than 60 years present hypertension and diabetes, among other chronic diseases. One of the most relevant physiological processes associated with aging is inflammaging, a term that describes a state of low-grade chronic inflammation that progressively develops with age. To evaluate the concentration of inflammatory markers in healthy older adults and compare them with healthy young adults with no prior disease diagnosis. Following approval by the Cinvestav ethical committee, clinically healthy volunteers were recruited and divided into two groups: young adults (20–30 years old) and older adults (60–70 years old). Serum concentrations of IL-6, IL-1β, IL-2, IL-12p70, IL-10, IL-4, TNF-α, TGF-β, IFN-γ, CXCL10, and CCL2 were quantified using a Multiplex assay. Individuals aged 60–70 showed a statistically significant increase in the concentrations of IL-6, IL-12p70, IL-2, IL-10, and IFN-γ compared to the younger group. When analyzing by sex, an increase of IL-12p70, IL-10, and IFN-γ levels was observed in older men. These findings suggest that, even in the absence of diagnosed diseases, the concentrations of certain pro- and anti-inflammatory cytokines increase in older adults. Elevated serum levels of IL-6, IL-12p70, IL-2, IL-10, and IFN-γ in older adults without previous disease diagnosis may be associated with the development of "inflammaging", supporting the hypothesis of subclinical chronic inflammation linked to aging. DILETEC and Cinvestav supported this work.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Molecular Mimicry Between Food Antigens and Metabolic Hormones: Role on Anti-LEP/Anti-GRL Antibodies

Juárez-Valderrama, Hugo Adrián 1; Mendoza-Hernández, Victor Ignacio [1]; Reyes-Castillo, Zyanya; Gómez-Medina, Daniela [1]; Magaña- Cuevas, Elsa Patricia [1]; Gómez-Márquez, Carolina [1 2].

[1]Laboratorio de Biomedicina y Biotecnología para la Salud, Departamento de Ciencias Clínicas, Centro Universitario del Sur, Universidad de Guadalajara, Zapotlán el Grande, Jalisco, México.
[2]Laboratorio de Innovación Biodigital, Departamento de Bioingeniería Traslacional, Centro Universitario de Ciencias Exactas e Ingenierías, Universidad de Guadalajara, Guadalajara, Jalisco, México.

E-mail: hugo.juarez9374@alumnos.udg.mx

Molecular mimicry is defined as the sequential or structural similarity between self and foreign antigens. Leptin and ghrelin participate in appetite regulation, and a fraction of these hormones circulate bound to low-affinity antibodies (Abs) (anti-LEP and anti-GRL) in healthy subjects, while changes in their affinity have been reported in metabolic diseases. It has been proposed that the origin of these antibodies is due to molecular mimicry between leptin/ghrelin and proteins in the gut microbiota. However, the role of food antigens in this phenomenon is unknown, so this exploratory study aims to identify the presence of food antigens with sequential or structural similarity to leptin/ghrelin. Food lists were created based on the SMAE food classification for foods that are consumed raw or frequently. Food protein databases were created from UniProt, and protein-hormone alignments were performed using local Blast. The results were then filtered, and the description and function of the proteins were sought. The process was automated using Python. Anti-GRL and anti-LEP Abs and immune complexes (ICs) were analyzed in the serum of healthy adults with a characterized diet. Out of a total of 136 foods evaluated, 421 proteins with sequential mimicry with leptin and 455 with ghrelin were identified. Seven foods had more than 10 leptin-mimetic proteins, five of which were frequently consumed by the participants. However, anti-LEP Abs levels were not related to the frequency of consumption of these foods. For ghrelin, four frequently consumed foods with more than 10 mimetic proteins were identified, and participants who consumed these foods most frequently showed a higher percentage of anti-GRL ICs. These results support the hypothesis that some food antigens exhibit molecular mimicry with appetite-regulating peptides such as ghrelin and leptin. However, it will be important to confirm the cross-reaction between Abs and the identified food antigens.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Impact of Delivery Mode on Neonatal CD4+ T Cell Gene Expression Profiles.

Kempis -Calanis, Linda Aimara [1]; Ventura-Martínez, Carlos [1];
Spicuglia, Salvatore [2]; Rodríguez-Jorge, Otoniel [1].

[1]Laboratorio de Inmunología Celular y de Sistemas. Centro de Investigación en Dinámica Celular. Universidad Autónoma del Estado de Morelos. [2]Aix-Marseille University, Inserm, TAGC, UMR1090, 13288 Marseille, France.

E-mail: aimara.kempiscal@gmail.com

Prematurity remains the leading cause of neonatal morbidity and mortality worldwide. Most premature births occur by cesarean section, a medical proccedure that is important to save lives when indicated. In Mexico, cesarean deliveries account for over 50% of all births, significantly exceeding the World Health Organization's recommendation that cesarean sections should not exceed 10–15% of total births. This study aimed to investigate whether the mode of delivery and gestational age influence the gene expression profile and functionality of neonatal CD4+ T cells. We performed transcriptomic (RNA-seq) analysis on naïve CD4+ T cells isolated from neonates born via cesarean section or vaginal delivery, both at term and preterm, and performed functional experiments to validate some of our findings. Our results reveal that the mode of delivery significantly determines the neonatal CD4+ T cell gene expression profile. Specifically, CD4+ T cells from neonates delivered by cesarean section at term exhibited a distinct gene expression profile compared to those delivered vaginally, with 600 differentially expressed genes. Overexpressed genes in CD4+ T cells from neonates born by natural birth were enriched in pathways related to immune response (e.g., cytokine-cytokine receptor interaction, Th17 signaling), activation of various signaling pathways (e.g., NF-kB, TNF, NOD, MAPK), and several diseases. These results suggest that vaginal delivery may induce the immune priming of CD4+ T cells. To validate this result, CD4+ T cells were stimulated (CD3/CD28) in vitro and Th cytokine production was measure, which confirmed our results. In contrast, preterm neonates did not exhibit significant changes in their gene expression patterns compared to term neonates born via cesarean section. These findings indicate that the mode of delivery plays a crucial role in establishing the neonatal CD4+ T cell gene expression profile and functionality, with a greater impact than the final weeks of gestation.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

In vitro–generated TRM T cells show enhanced effector function and ICB response in human melanoma

Ko, Nahuel Matías [1 6]; Gajón-Martínez, Julián Argenis [2 6];
Esperante, Diego [3 6]; Fuentes-Panana, Ezequiel M. [4];
Bonifaz-Alfonzo, Laura Cecilia [5 6].

[1]Facultad de Química, Universidad Nacional Autónoma de México, Ciudad de México, México. [2]Posgrado en Ciencias Bioquímicas, Universidad Nacional Autónoma de México, México. [3]Plan de Estudios Combinados en Medicina, Universidad Nacional Autónoma de México, Ciudad de México, México. [4]Unidad de Investigación en Virología y Cáncer, Hospital Infantil de México Federico Gómez, Ciudad de México, México. [5]Coordinación de Investigación en Salud, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México. [6]Unidad de Investigación Médica en Inmunoquímica, Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México.

E-mail: nahuelko2000@gmail.com

Tissue resident memory (TRM) CD8 T cells are a special subset of memory T cells that reside in non-lymphoid tissues and offer long-lasting immune responses against specific and persistent antigens. It has been demonstrated that TRMs are essential in the clearance of chronic viral and bacterial infections and play a key role in tumor infiltration and elimination. The study of TRM biology has become stagnant due to a lack of stimuli that can generate in vitro TRMs in a reproducible manner. Previous studies have shown that TRMs can be produced in vitro through a combination of IL-7, IL-15 and transforming growth factor (TGF) β. However, this method has proven to be rather expensive and possesses limited reproducibility. In this study, we demonstrate that the activation of peripheral blood mononuclear cells (PBMCs) with phytohemagglutinin (PHA) robustly generates a TRM-like cell subpopulation. These TRM-like cells express the canonical tissue residency markers (CD69 and CD103) and transcription factors associated with TRMs (RUNX3 and HOBIT). Additionally, when co-cultured with human melanoma spheroids, these TRM-like cells are more functional and cytotoxic, which is demonstrated by a heightened IFNγ, TNFα and perforin expression. Furthermore, these TRM-like cells respond to immune checkpoint blockers (ICB) by increasing the expression of the previously mentioned effector and cytotoxic molecules. Finally, this study provides the basis for the reproducible generation of TRM-like cells that possess heightened effector capabilities. This will enable the study of TRM biology in the context of cancer and immunotherapy.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

New Evidence of Emerging Arboviruses Circulating in Mexico

Laredo-Tiscareño, S. Viridiana; Adame-Gallegos, Jaime R.; Rodríguez-Alarcón, Carlos A.; González -Peña, Rodolfo; Rubio-Tabares, Ezequiel; De Luna-Santillana, Erick de Jesús; Garza-Hernández, Javier Alfonso.

[1]Universidad Autónoma de Ciudad Juárez. Departamento de Ciencias Químico Biológicas. Ciudad Juárez, Chihuahua. México. [2]Universidad Autónoma de Chihuahua. Facultad de Ciencias Químicas. Chihuahua. Chihuahua. México. [3]Universidad Autónoma de Yucatán. Centro de Investigación Regional Dr. Hideyo Noguchi Mérida, Yucatán. México. [4]Instituto Politécnico Nacional. Centro de Biotecnología Genómica. Reynosa, Tamaulipas. México.

E-mail: viridiana.laredo@gmail.com

We conducted serologic surveillance for flaviviruses and orthobunyaviruses in vertebrate animals in Mexico in 2018-2019. Sera were collected from 856 vertebrate animals, including 323 dogs, 223 horses, and 121 cows, from 16 species. The animals were from 3 states: Chihuahua in northwest Mexico (704 animals) and Guerrero and Michoacán on the Pacific Coast (27 and 125 animals, respectively). Sera were assayed by plaque reduction neutralization test using four flaviviruses (dengue type 2, St. Louis encephalitis, West Nile, and Zika viruses) and six orthobunyaviruses from the Bunyamwera (BUN) serogroup (Cache Valley, Lokern, Main Drain, Northway, Potosi, and Tensaw viruses). Antibodies to West Nile virus (WNV) were detected in 154 animals of 9 species, including 89 (39.9%) horses, 3 (21.4%) Indian peafowl, and 41 (12.7%) dogs. Antibodies to St. Louis encephalitis virus (SLEV) were detected in seven animals, including three (0.9%) dogs. Antibodies to Lokern virus (LOKV) were detected in 22 animals: 19 (8.5%) horses, 2 (1.7%) cows, and a dog (0.3%). Antibodies to Main Drain virus (MDV) were detected in three (1.3%) horses. WNV and LOKV activity was detected in all three states, SLEV activity was detected in Chihuahua and Michoacán, and MDV activity was detected in Chihuahua. None of the animals was seropositive for Cache Valley virus, the most common and widely distributed BUN serogroup virus in North America. In conclusion, we provide serologic evidence that select flaviviruses and BUN serogroup viruses infect vertebrate animals in Chihuahua, Guerrero, and Michoacán. We also provide the first evidence of LOKV and MDV activity in Mexico.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Testosterone differentially regulates Bmal1 and Clock expression and the malaria immune response

Legorreta-Herrera, Martha; Nolasco-Pérez, Teresita de Jesús [1];
Nolasco-Pérez, Valery Samantha [1]; Franco-Varela.

[1]Universidad Nacional Autónoma de México, Unidad de Investigación Química Computacional, Síntesis y Farmacología en Moléculas de Interés Biológico, Facultad de Estudios Superiores Zaragoza, Ciudad de México, Ciudad de México, México.

E-mail: marthal@unam.mx

Malaria is the most lethal parasitic disease in the world, causing higher mortality in men than in women. Testosterone is the male sex hormone responsible for the main differences between the sexes; it has immunosuppressive properties. In addition, testosterone levels change throughout the day in a circadian rhythm. In the early hours of the morning, its levels are higher than those at night. Therefore, in this work, we studied whether increasing testosterone levels modify the hypothalamic expression of the Clock and Bmal1 genes (which regulate the circadian rhythm in the suprachiasmatic nucleus) in male CBA/Ca mice infected with Plasmodium berghei ANKA and whether this affects the immune response differently during the day (ZT3) than at night (ZT15). We increased the testosterone concentration and simultaneously inhibited aromatase to avoid estrogen interference. Then, we measured testosterone levels, parasitemia, cell populations, and the expression of the Clock and Bmal1 genes at ZT3, and ZT15. An increasing testosterone concentration led to an increase in parasitemia, particularly at ZT3, which corresponded with a reduction in CD4+ and CD8+ T cells (which are essential for Plasmodium elimination), as well as decreased Bmal1 expression and increased Clock expression. This study provides knowledge on the regulatory effects of testosterone on genes involved in the circadian cycle and explains, at least in part, the immunosuppressive role of testosterone in malaria.

This work was supported by PAPIIT, IN223524.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

STAT6 Deficiency Accelerates Skin Carcinogenesis and Promotes Immunosuppressive Responses in a Murine Model

León-Cabrera, Sonia Andrea [1,2]; Galindo -Santamaría, Tania [1]; Moreno-Tovar, Valeria [1]; Mejía-Muñoz, Aranza [1,2]; Arroyo-Olarte, Rubén [1,2].

[1]Unidad de Investigación en Biomedicina, Facultad de Estudios Superiores Iztacala. Universidad Nacional Autónoma de México. Tlalnepantla, México. México. [2]Unidad de Investigación en Biomedicina, Facultad de Estudios Superiores Iztacala. Universidad Nacional Autónoma de México. Tlalnepantla, México. México. [3]Carrera de Médico Cirujano. Facultad de Estudios Superiores Iztacala. Universidad Nacional Autónoma de México. Tlalnepantla, México. México.

E-mail: soleon81@gmail.com

Basal cell carcinoma (BCC) and squamous cell carcinoma (SCC)—collectively known as non-melanoma skin cancer (NMSC)—are the most common skin cancers in humans. Targeted inhibitors of STAT (Signal Transducer and Activator of Transcription) proteins are currently under investigation, with some demonstrating efficacy in solid tumors. STAT6, in particular, plays a critical role in various cancers, including breast, prostate, melanoma, and colon; however, its involvement in NMSC remains largely unexplored. To investigate the role of STAT6 in skin carcinogenesis, we established a murine model of chemically induced skin cancer. Wild-type (WT) and STAT6-deficient (STAT6$^{-/-}$) mice were treated with DMBA (initiator) and TPA (promoter) over 20 weeks. Body weight, clinical condition, lesion appearance, and papilloma size were monitored twice weekly.

STAT6 deficiency led to a significantly earlier onset of papilloma formation—up to four weeks earlier than in WT mice. Additionally, STAT6$^{-/-}$ mice developed a markedly higher number of papilloma (23 ± 6) compared to WT controls (2 ± 1.8). While papilloma size did not differ significantly between groups, STAT6$^{-/-}$ mice displayed more severe histological damage and increased epidermal thickness. Ki-67 staining revealed elevated cell proliferation in all treated mice, though levels were higher in WT animals, possibly due to delayed tumor progression. Although CD11b^{+} myeloid cell infiltration was similar across groups, STAT6$^{-/-}$ mice exhibited a significant increase in regulatory T cells (Tregs) in both axillary and inguinal lymph nodes and within papillomas. Furthermore, IFN-γ expression was reduced, and FOXP3 expression was elevated in papilloma from STAT6$^{-/-}$ mice, indicating a potential suppression of antitumor immunity.

These findings suggest that STAT6 plays a regulatory role in SCC development, and its deficiency may favor tumor progression by altering the local immune environment. Further research is warranted to explore its therapeutic potential.

Funding Information: UNAM-PAPIIT IN201724

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y comparada**

Interspecies cross-immunity as treatment for canine distemper virus infection

Lopez -Pantoja, Jose Alberto [1]; Arteaga -Tinoco, Anelle Siboney [1]; Secundino-Velázquez, Ismael [1].

[1]Universidad La Salle Bajío, Facultad de Veterinaria, León Guanajuato, México.

E-mail: chivotinoco@outlook.com

Canine distemper virus (CDV) infection is one of the most feared diseases in veterinary medicine with a high morbidity and mortality rate, despite the availability of vaccines. It mostly affects domestic dogs, but has the ability to infect many different species including various wildlife animals which can also act as reservoirs. Canine distemper virus is considered polytropic, as it infects many different tissues, but the prognosis becomes grave when the patient begins to show neurological damage. Avian Newcastle virus shares the same ancestry with canine distemper virus, both are viruses of the family Paramyxoviridae and have similar structural proteins. Provided that Newcastle virus only infects poultry and limited wild birds, it is not capable of proliferating inside mammalian cells. Evidence suggests that anti-newcastle serum in combination with a palliative treatment in dogs could protect against lethal canine distemper virus disease. However, the mechanism underlying this protection is not known. The aim of this study is to define the mechanism by which anti-newcastle serum induces immunity in infected dogs with canine distemper virus. Here, two healthy mixed breed dogs were immunized with Newcastle vaccine La Sota Strain, B1 type. After 10-12 hours of administration of Newcastle vaccine, the dogs were anesthetized and blood was collected. Serum was recovered and stored at -20°C until analysis. The presence of IFN-α by ELISA assay was found in anti-newcastle serum, with concentrations of 2.472 ng/ml and 2.068 ng/ml in both dogs, respectively. Certainly, interferon-α is not the only component in the innate immune response induced by Newcastle vaccine and more investigation is required to understand what other chemokines or cytokines might be involved in anti-distemper immunity and why the serum works so well against a disease that has such a poor prognosis.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Base deficit and leukocyte count as predictors of mortality during sepsis

Lopez -Alvarez, Xuxu Guadalupe [1]; Rojas-Guillen, Megan Zahori [2]; Plata-Ramos, Ivonne Mónica [3]; Castillo-Cruz, Juan [1].

[1]Instituto Politécnico Nacional, Escuela Superior de Medicina, Ciudad de México, CDMX, México. [2]Instituto Politécnico Nacional, Escuela Superior de Enfermería y Obstetricia, Ciudad de México, CDMX, México. [3]IMSS, Hospital General Regional No.72 " Lic. Vicente Santos Guajardo", Estado de México, México.

E-mail: xuxulopez39@gmail.com

Sepsis is a clinical syndrome characterized by a dysregulated immune response to infection, leading to organ dysfunction and high mortality. In this context, base deficit (BD), as a marker of metabolic acidosis, and leukocyte count (LC), as an indicator of immune activity, may serve as useful prognostic biomarkers. Objective: To determine the prognostic value of BD and LC in relation to mortality in patients diagnosed with sepsis. Materials and Methods: An observational, analytical, retrospective cohort study was conducted using primary source clinical records. Patients of both sexes, aged 20 to 75 years, admitted to the emergency department with a diagnosis of sepsis, were included through systematic random sampling. Serum values of BD, LC, and platelet count were collected the first 72 hours, with a sample size of 164 subjects. The main outcome was in-hospital mortality. A comparative statistical analysis was performed. Results: BD was significantly more negative in deceased patients (-10.5 ± 8.0) compared to survivors (-8.2 ± 5.8), with p = 0.05. LC was higher in survivors (18,919.3 ± 19,442.8) than in deceased patients (16,136.5 ± 15,782.0), though not statistically significant (p = 0.31). Platelet counts were also lower in deceased individuals (194,428 ± 136,610.7) compared to survivors (230,975.8 ± 120,678.3), with p = 0.95. During hospitalization, BD values improved significantly over time, LC remained stable, and platelet counts showed a marked decrease in patients who died. Conclusions: Base deficit was significantly associated with mortality, suggesting its potential as a prognostic biomarker in septic patients. Although leukocyte and platelet counts did not show statistical significance at admission, their temporal behavior may contribute to the clinical assessment of immune system status and disease progression.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

HIF-1 activation modulates CCR6 expression in vitro and in vivo models.

López-Ávila, Brandon Arath [1]; Baay-Guzmán, Guillermina Juliana [1];
Arenas-Huertero, Francisco [2]; Vargas-Hernández, Jacob Josman [2];
Torres-Ruíz, Sarahí [1].

[1]Hospital Infantil de México Federico Gómez, Unidad de Investigación por enfermedades oncológicas, Ciudad de México, México. [2]Hospital Infantil de México Federico Gómez, Unidad de Investigación en patología experimental, Ciudad de México, México.

E-mail: lopezavilaarath@gmail.com

Asthma is a chronic inflammatory disease of the airways that leads to lung remodeling and a hypoxic and inflammatory microenvironment, promoting the activation of the transcription factor HIF-1, which regulates nearly 200 genes involved in this condition. Previous studies suggest that the CCR6 receptor, present in Th17 lymphocytes and neutrophils—both implicated in severe forms of asthma by being recruited by their ligand produced by epithelial cells and macrophages—is regulated by HIF-1. This study evaluated HIF-1-mediated CCR6 expression in in vitro and in vivo models. For this purpose, bronchial epithelial cells (NL-20) were exposed to normoxia or hypoxia for different periods (0h, 3h, 6h, 9h, 12h, 18h, and 24h), and immunofluorescence staining was performed to detect HIF-1α and CCR6. In parallel, Balb/c mice were sensitized with albumin and aluminum hydroxide and later challenged three times with the allergen to induce allergic pulmonary inflammation (API) with varying degrees of severity. Additionally, the mice were treated with either a HIF-1α inducer (ethyl-3,4-dihydroxybenzoate) or an inhibitor (2-methoxyestradiol). Subsequently, immunohistochemical staining was performed on lung tissue to analyze CCR6 expression.

The results showed that HIF-1α localizes in the cytoplasm after 6 hours of hypoxia exposure and translocates to the nucleus after 9 hours, coinciding with CCR6 overexpression at 9 hours under hypoxia. Likewise, mice with severe asthma or those treated with the HIF-1α inducer exhibited higher CCR6 expression, whereas those treated with the inhibitor showed a *decrease compared to the other two groups.

In conclusion, HIF-1 activation induces CCR6 overexpression both in vitro and in vivo, contributing to asthma severity.

Área del artículo: **Inmunología clínica y traslacional**

Validation of a serological ELISA method to detect anti-SARS-CoV-2 IgG and IgM antibodies in Mexican population.

Rodríguez-González , Mabel ; Losoya -Uribe, Luis Fernando [1];
Barreto -Cabrera, Daniel [2] ; Rivera -Castro , Juan Carlos [3] ;
Hernández -Ortiz , Luis Eduardo [4] ; Calderón -Corona, Arlene [5] ; Olvera
-Rodriguez , Maricela [6] ; López-Camacho , Andrea [7] ;
Gutiérrez -Mayret, Michelle [8] ; Ramírez -Reivich, Octavio Tonatiuh [9] ;
Palomares-Aguilera , Laura Alicia [10] .

[1]Instituto de Biotecnología, LAMMB, Cuernavaca, Morelos, México . [2]Instituto de Biotecnología, LAMMB, Cuernavaca, Morelos, México . [3]Instituto de Biotecnología, LAMMB, Cuernavaca, Morelos, México . [4]Instituto de Biotecnología, LAMMB, Cuernavaca, Morelos, México . [5]Instituto de Biotecnología, LAMMB, Cuernavaca, Morelos, México . [6]Instituto de Biotecnología, LAMMB, Cuernavaca, Morelos, México . [7]Instituto de Biotecnología, LAMMB, Cuernavaca, Morelos, México . [8]Instituto de Biotecnología, LAMMB, Cuernavaca, Morelos, México . [9]Instituto de Biotecnología, LAMMB, Cuernavaca, Morelos, México . [10]Instituto de Biotecnología, LAMMB, Cuernavaca, Morelos, México

* E-mail: andrea.lopez@lammb.unam.mx

The COVID-19 pandemic was a significant challenge to the medical and scientific community due to the urgent need for action. The Laboratory for the Analysis of Biotechnological Molecules and Medicines (LAMMB), an analytical control laboratory certified by COFEPRIS, was urged to validate a serological assay to detect anti-SARS-CoV-2 antibodies in the Mexican population, for that reason LAMMB validated two ELISA assays to detect IgG and IgM antibodies directed against the RBD domain of the S protein of the SARS-CoV-2 virus. RBD was produced and purified in the facilities of the Biotechnology Institute of the UNAM. For validation, it was classified as an Identity test, based on the Mexican Pharmacopoeia and the performance parameters were evaluated during the assay validation: 1) System suitability, 2) Specificity/ Selectivity, and 3) Robustness. The two serological methodologies successfully passed the scanning and confirmatory tests with the system verification criteria established (CV% of positive serum, corrected absorbance of positive, negative, and blank serum), as well as the robustness (measurement of AU at 0 and 30 minutes with liquid and lyophilized RBD) and specificity/selectivity of each method. The serological assays to detect anti-SARS-CoV-2 IgM and IgG antibodies were succesfully validated. It met the following parameters: 1) System suitability, 2) Specificity/Selectivity, and 3) Robustness. Subsequently the ELISA tests were transferred to the Laboratorio Estatal de Salud Pública (LESP, Government of the State of Hidalgo) and to the National Institute of Public Health, México.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

Neuroinflammation sex-differences in the mouse striatum induced by aging and excitotoxic damage

López-Castillo, Jesús [1]; González-Espinosa, Claudia [1];
Pérez -Severiano, Francisca [1].

[1]CINVESTAV, Departamento de Neurofarmacología, Ciudad de México, México. [2]CINVESTAV, Centro de Investigación sobre el envejecimiento, Ciudad de México, México. [3]Instituto Nacional de Neurobiología y Neurocirugía Manuel Velazco Suárez, Departamento de Investigación, Ciudad de México, México.

E-mail: lopezjesus.ca@gmail.com

Aging is linked to the development of chronic diseases, and the changes associated with it have detrimental repercussions on different tissues. Specifically, inflammatory state associated with aging known as “imflamaging” has been identified. This is a product of changes in the functioning of the immune system, as well as an increase in the number of senescent cells. These cells acquire a particular phenotype, such as the development of a senescence-associated secretory phenotype (SASP), which consists of an increased expression of immune response regulatory molecules. Neuroinflammation is a key factor in neurodegenerative diseases, which are more common in the elderly. However, the immune response in the aging brain remains poorly understood. The striatum a critical region for motor control, memory, and reward is notably affected in diseases like Huntington’s and Parkinson’s. Despite its importance, little is known about immune changes in this area, including potential sex-specific differences in its inflammatory response. This study seeks to identify changes in the acute inflammatory response during aging, evaluating senescence markers, inflammatory cytokines related to the SASP and the S100B protein and the RAGE receptor, through the implementation of a murine model of excitotoxicity, allowing us to evaluate neurodegeneration at a behavioral level, as well as the expression of the previously mentioned markers after acute damage in young and aged male and female mice. By now, we have been able to verify that there is a significant change in the expression of these markers associated with inflammation and cellular damage in the striatum of young and aged animals. The results so far indicate a reduction in the response capacity to excitotoxic damage by aged animals compared to young ones. Regarding the difference between sexes, it was more marked in young animals, while with age, both males and females presented a more attenuated response.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Autophagy-dependent cytotoxicity induced by oleanolic and ursolic acids in lung cancer cells

López-García, Alejandra [1 2]; Hernández-González, Juan Carlos [2]; Castillo-Cruz, Juan [3]; Baltierra-Uribe, Shantal Lizbeth [4]; Cariño-Cortés, Raquel [5]; Piloni-Martini, Javier [2]; García-Pérez, Blanca Estela [4]; Castrejón-Jiménez, Nayeli Shantal [2].

[1]Área Académica de Nutrición, Instituto de Ciencias de la Salud, Universidad Autónoma del Estado de Hidalgo, San Agustín Tlaxiaca, Hidalgo, México. [2]Área Académica de Medicina Veterinaria y Zootecnia, Instituto de Ciencias Agropecuarias, Universidad Autónoma del Estado de Hidalgo, Santiago Tulantepec de Lugo Guerrero, Hidalgo, México. [3]Escuela Superior de Medicina, Sección de Estudios de Posgrado e Investigación, Instituto Politécnico Nacional, Alcaldía Miguel Hidalgo, Ciudad de México, México. [4]Departamento de Microbiología General, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Alcaldía Miguel Hidalgo, Ciudad de México, México. [5]Área Académica de Medicina, Campus Dr. Eliseo Ramírez Ulloa, Instituto de Ciencias de la Salud. Universidad Autónoma del Estado de Hidalgo, Pachuca de Soto, Hidalgo, México.

E-mail: lo294624@uaeh.edu.mx

Lung cancer remains one of the leading causes of mortality globally. Largely due to its early-stage asymptomatic nature, making it difficult to diagnose early. The effectiveness of chemotherapeutic agents is compromised by the development of drug resistance by tumor cells. This therapeutic limitation has led to the search for new antineoplastic strategies, among which stand out the secondary metabolites derived from aromatic plants and fruits, such as oleanolic acid (OA) and ursolic acid (UA), which have demonstrated antitumor activities and the ability to induce autophagy, positioning themselves as potential adjuvants in the treatment of lung cancer. Autophagy is a cellular self-digestion process that plays a key role in maintaining homeostasis and has been widely associated with cancer regulation. Several studies have shown that cancer cells can take advantage of this mechanism to survive under stressful conditions such as chemotherapy. Therefore, this work aimed to demonstrate the involvement of the autophagy process in the A549 lung cancer cell model stimulated with OA and UA. Cell monolayers were performed to evaluate morphological changes, cell viability, production of reactive oxygen species (ROS), and inhibition of autophagy in A549 cells stimulated with OA and UA. The results demonstrated changes in the integrity of the monolayer at different stimulus times (2, 4 and 24 h). The decrease in cell viability after stimulation with OA and UA and treatment with cisplatin at 24 h. Also, increased ROS production. Interestingly, a greater amount of OA and UA are required to decrease cell viability when autophagy is inhibited. In conclusion, UA showed greater cytotoxicity in A549 cancer cells than OA. The increase in ROS with both acids was demonstrated at short times, and it was also evidenced that the autophagy promotes cell death.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Single-cell analysis identifies inflammatory and tissue-remodeling tumor macrophages distinct from M1/M2

López-Huerta, Eric [1 2]; Portales-Pérez, Diana Patricia [3 4];
Martinez-Leija, Miguel Ernesto [3 4]; Ortiz-Navarrete, Vianney [1];
Fuentes-Pananá, Ezequiel Moisés [2].

[1]Department of Molecular Biomedicine, Center for Research and Advanced Studies (CINVESTAV), National Polytechnic Institute, 07360. Mexico City, México. [2]Research Unit in Virology and Cancer, Children´s Hospital of México Federico Gómez, 06720, México City, México. [3]Laboratory of Immunology and Cellular and Molecular Biology, Faculty of Chemical Sciences, Autonomous University of San Luis Potosí, 78210, San Luis Potosí, San Luis Potosi, México. [4]Translational and Molecular Medicine Department, Research Center for Health Sciences and Biomedicine (CICSaB), Autonomous University of San Luis Potosí, 78210, San Luis Potosí, San Luis Potosí, México.

E-mail: eric.lopez@cinvestav.mx

Tumor-associated macrophages (TAM) are a central component of the tumor microenvironment, exerting both pro- and anti-tumoral functions that influence cancer progression and patient prognosis. However, the heterogeneity revealed by advanced technologies like single-cell RNA sequencing (scRNA-seq) remains incompletely characterized. Our study aims to explore TAM heterogeneity and its clinical implications implementing unsupervised analysis. With R programming language we conducted weighted gene co-expression network analysis to identify transcriptional gene signatures expressed by TAMs. We applied this analysis to publicly available scRNA-seq datasets, comprising samples from blood, tumor, and non-tumoral mammary tissue (NT tissue) from both cancer patients and healthy individuals. We identified seven distinct gene signatures corresponding to different TAM subsets, each exhibiting unique functional profiles, including high interferon response, inflammation, scavenging, and matrix remodeling, with the latter two being characteristic of tissue repair processes. Notably, none of these subsets aligned perfectly with the conventional M1/M2 macrophage classification. Signatures associated with inflammation and interferon-related genes were predominantly enriched in blood monocytes, whereas tissue-repairing-related signatures were more abundant in macrophages from NT tissue. This suggests that TAMs with these signatures resemble monocytes and tissue-resident macrophages, respectively. These findings were consistently observed across multiple scRNA-seq datasets from various cancer types. Additionally, one signature was differentially expressed in peripheral monocytes from BRCA patients compared to healthy individuals, and its genes were experimentally validated in a cohort of Mexican BRCA patients using RT-PCR. Furthermore, in an independent cohort of 1,100 BRCA patients from TCGA, we estimated the abundance of these TAM subsets and found that TAMs with high interferon gene expression were associated with a favorable prognosis, whereas tissue-repairing TAMs correlated with poor prognosis. These findings suggest that TAMs resembling tissue-resident macrophages exhibit a tissue-repairing phenotype and are associated with poor survival, whereas monocyte-like TAMs display an inflammatory/interferon response and are linked to better prognosis.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

CCR3 blockade reduces neutrophil lung recruitment during acute influenza A H1N1 infection

López-Leal, Fátima [1]; Cabellos-Avelar, Tecilli [1]; Hernández-González, David [1]; Reyes-Huerta, Raúl F. [2]; Castro-Jiménez, Tannya Karen [1]; Bustos-Arriaga, José [1]; Maravillas-Montero, José Luis [2]; Pérez-López, Araceli [1].

[1]Unidad de Investigación en Biomedicina, Facultad de Estudios Superiores Iztacala, UNAM, Tlalnepantla, Estado de México 54090, México. [2]Red de Apoyo a la Investigación, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Ciudad de México 14080, México.

E-mail: fatimalp198@gmail.com

During influenza infection, neutrophils (NPs) constitute most cells infiltrating the lung and are essential for virus elimination. However, uncontrolled activation of these cells contributes to acute tissue damage. We have shown that NPs acquire CCR3 expression at the site of infection, and its activation boosts their effector functions. Thus, we evaluated whether CCR3 blockade using the specific antagonist SB-328437 and a monoclonal antibody could reduce NP recruitment to the lung in a murine model of H1N1 influenza infection. C57BL/6 mice were infected intranasally with H1N1 influenza (10^6 PFU), and SB-328437 (5 µg/kg) or anti-CCR3 antibody (clone 6S2-19-4, 25 µg) was administered intraperitoneally at 32 and 72 hours post-infection. At 4 days post-infection (dpi), bronchoalveolar lavage fluid (BALF), lung tissue, blood, and bone marrow were collected. The total number of cells in BALF was quantified, and the presence of NPs was evaluated by flow cytometry. Our results showed an increase in total cells in BAL compared to control groups, and ~70% of these cells correspond to NPs. When CCR3 was blocked, there was a ~4- to 5-fold reduction in total BALF cells compared to influenza-infected mice alone. Flow cytometry analysis revealed a ~5- to 6-fold reduction of NPs in CCR3-blocked groups relative to infected but untreated animals. No significant changes were observed in blood, lung, or bone marrow NP proportions. Additionally, a trend toward reduced viral load was observed in treated animals. These findings support using CCR3 blockade to modulate NP recruitment to the lung during influenza infection. This project was funded by CONAHCYT (now SECIHTY), project CBF2023-2024-1378, and COMECYT, project EDOMÉX-FICDTEM-2021-05.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Potential association of B regulatory cells in bovine leukemia virus infection

López-Montaño, Maresa [1]; Marín-Flamand, Ernesto [1]; González-Díaz, Francisco Rodolfo [1]; Vargas-Ruíz, Alejandro [1]; Araiza-Hernández, Diana Michele [1]; Hernández-Balderas, Ruperto Javier [2]; Rangel-Rodríguez, Ignacio Carlos [1]; García-Camacho, Lucía Angélica [1].

[1]Facultad de Estudios Superiores Cuautitlán, Universidad Nacional Autónoma de México, departamento de ciencias biológicas, Cuautitlán Izcalli, Estado de México, México. [2]Facultad de Estudios Superiores Cuautitlán, Universidad Nacional Autónoma de México, departamento de ciencias pecuarias, Cuautitlán Izcalli, Estado de México, México.

E-mail: maresa_6273@hotmail.com

Persistent lymphocytosis (PL) is an outcome of bovine leukemia virus (BLV) infection used as a disease susceptibility marker and related to BLV-induced immune suppression in dairy cows. Previously, significantly higher expression of IL10 and TGFB along with IL12, IFN gamma, and IL4 lower levels have been found in BLV+/PL+ cows. Development of PL comprises a polyclonal proliferation of BLV target cells (IgM+, CD5+ B cells). Such expansion may enrich for B regulatory cells (Bregs), whose phenotype is similar to that of the target cells. To identify potential Breg subpopulations, 38 cows were sampled for six months to perform routine lymphocyte counts, and PBMC phenotyping (IgM, CD5, and IL10 or TGFB) by flow cytometry. The animals were grouped as follows: BLV-/PL- (n= 10), BLV+/PL- (n= 14) BLV+/PL+ (n=14). CD5+, and CD5- B cells secreting IL10-, or TGFB were found in the experimental population during the experiment, being significantly higher in the BLV+PL+ group compared to the other groups. In both CD5+, and CD5- populations, the TGFB-secreting B cells were significantly more frequent than the IL10 counterparts. This finding was more striking in CD5+ B cells. The data herein presented suggest that IL10-, and TGFB-secreting B cells might contribute to immune suppression described in BLV infected cows with PL. Likely, TGFB have a key role in BLV infection given that a preponderance of TGFB-secreting B cells was observed. Further characterization of the putative Breg populations in BLV infected cows must be performed.

Funded by: DGAPA-PAPIIT IN202023, and FESC-PIAPI CI2465.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Vacunas**

In silico design of DNA vaccine against T. gondii targeting ROP35 epitopes

López-Moreno, Héctor Samuel [1]; Velázquez-Escobar, Frida Monserrat [1]; Beltrán-López, Evangelina [1]; Vega-Castillo, Ulises [1]; Chávez-Ontiveros, Jeanett [1].

[1]Laboratorio de Biomedicina Molecular, CAC Biotecnología Biomédica UAS-264, Posgrado en Ciencias Biomédicas y Posgrado en Biotecnología, énfasis en Salud, Facultad de Ciencias Químico Biológicas, Universidad Autónoma de Sinaloa, Av. Las Américas S/N, Ciudad Universitaria, Culiacán, Sinaloa, México.

E-mail: hslmoreno@ms.uas.edu.mx

Toxoplasmosis is a globally prevalent zoonosis caused by Toxoplasma gondii (T. gondii), with an incidence ranging from 30 % to 70 %. It primarily affects immunocompromised individuals and pregnant women, potentially leading to severe complications, including death. ROP proteins, which are located in the T. gondii rhoptries, are crucial for parasite invasion and persistence. Among them, ROP35 has been identified as a relevant antigen, making it a suitable target for the in silico development of a DNA vaccine to prevent T. gondii infection. Using the ROP35 sequence, B and T cell epitopes restricted to H2-IAd in BALB/c mice were predicted with MHC-II Binding and ElliPro software and further evaluated using VaxiJen 2.0 and MHCPred. The epitopes MPEQDLASGFL and WDDAHLVQVSTSHPD exhibited the highest antigenic properties for B and T CD4+ cells respectively. The corresponding genetic sequences were cloned into the pVAX1 expression vector, incorporating a start codon and the GSGGSG spacer sequence to ensure optimal expression. This construct, named pR35BT, enables the simultaneous synthesis of both epitopes, representing a significant step toward the development of a genetic vaccine against toxoplasmosis. It holds potential for in vitro and in vivo validation in murine models, contributing to the establishment of molecular foundations for toxoplasmosis prevention in humans.

TRABAJO NO PRESENTADO

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Vacunas**

In silico design of a DNA vaccine against Toxoplasma gondii using GRA6

López-Moreno, Héctor Samuel [1]; Guzmán-Sánchez, María Fernanda [1]; Beltrán-López, Evangelina [1]; Vega-Castillo, Ulises [1]; Murúa-López, Carolina del Carmen [1].

[1]Laboratorio de Biomedicina Molecular, CAC Biotecnología Biomédica UAS-264, Posgrado en Ciencias Biomédicas y Posgrado en Biotecnología, énfasis en Salud, Facultad de Ciencias Químico Biológicas, Universidad Autónoma de Sinaloa, Av. Las Américas S/N, Ciudad Universitaria, Culiacán, Sinaloa, México.

E-mail: hslmoreno@ms.uas.edu.mx

Toxoplasma gondii is a ubiquitous intracellular protozoan capable of infecting a wide range of warm-blooded hosts, including humans. While toxoplasmosis is typically asymptomatic in immunocompetent individuals, it poses serious health risks in immunocompromised patients and during pregnancy due to the potential for fetal complications. Despite its global prevalence, no approved vaccine is currently available for human use, underscoring the urgent need for novel vaccine strategies. Building on the advances in genetic vaccines during the COVID-19 pandemic—such as ZyCoV-D, which offers production and storage advantages—we aimed to design a DNA vaccine against toxoplasmosis. This design focused on the Dense Granule Antigen 6 (GRA6, GenBank AKR54212.1) using an in silico approach to identify immunogenic epitopes specific to BALB/c mice. The ABCpred and MHC-II Binding Predictions tools were used to predict B and CD4+ T cell epitopes, respectively. The epitope "DVDPFPVLANEGKSE" (DE15) was selected as the top vaccine candidate based on its predicted immunogenicity. The codifying nucleotide sequence of DE15—GGC GAT GTG GAT CCG TTT CCG GTG CTG GCG AAC GAA GGC AAA AGC—was synthesized with the addition of an initiation codon (ATG) to ensure proper transcription in murine cells. This sequence was cloned into the pDE15 plasmid vector for future in vitro expression studies. This study presents a cost-effective and innovative pipeline for developing vaccines against T. gondii, particularly relevant for vulnerable populations such as pregnant women and immunocompromised individuals. In silico epitope prediction and validation may expedite the development of effective vaccine candidates.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Effect of premature birth on the Gene Expression Profile of Neonatal CD8+ T Lymphocytes

López-Valencia, Paulina [1]; Kempis-Calanis, Linda Aimara [1]; Hernández-Mendoza, Armando [2]; Santana-Calderón, María Angélica [1]; Rodríguez-Jorge, Otoniel [1].

[1]Laboratorio de Inmunología Celular y de Sistemas, Centro de Investigación en Dinámica Celular, Instituto de Investigación en Ciencias Básicas y Aplicadas, Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México. [2]Laboratorio de Bioinformática y Genómica Evolutiva, Centro de Investigación en Dinámica Celular, Instituto de Investigación en Ciencias Básicas y Aplicadas, Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México.

E-mail: paulina.lopezval@uaem.edu.mx

The neonatal immune system is adapted to the intrauterine environment, characterized by its immunotolerance. However, premature newborns (<37 weeks of gestation) are more vulnerable to infections and inflammatory diseases. Gestational age and the mode of delivery (cesarean section or vaginal delivery) influence the gene expression of CD8+ T lymphocytes. This study aimed to compare the gene expression of CD8+ T lymphocytes in neonates born at term and those born prematurely by cesarean section and vaginal delivery, using RNA-Seq and to validate the results experimentally with RT-qPCR and flow cytometry. The transcriptomic profile of CD8+ T cells of newborns was analyzed using bioinformatics tools. We identified 358 genes that were differentially expressed in CD8+ T lymphocytes, of which 351 genes were overexpressed in CD8+ T cells of premature neonates and 7 were overexpressed in CD8+ T cells of neonates born at term. The genes overexpressed in CD8+ T lymphocytes of premature neonates were significantly enriched in the cell cycle pathway. A high level of expression of some components involved in the glycolysis and fatty acid synthesis pathway were identified too. In contrast, when analyzing the differential expression based on the mode of delivery (cesarean section or vaginal delivery) born at term, 29 differentially expressed genes were found, of which 14 genes are overexpressed in CD8+ T cells of neonates born by cesarean section and 15 genes are overexpressed in CD8+ T cells of neonates born by vaginal delivery, however, they were not significantly enriched in any functional pathway. Taken together, these results suggest that CD8+ T cells from premature neonates have a distinct transcriptomic profile, characterized by the activation of pathways related to proliferation, glycolytic metabolism, and fatty acid synthesis. Currently, this project is undergoing experimental validation, which will confirm its biological relevance and potential use as biomarkers.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Comprehensive profiling of breast cancer-derived extracellular vesicles associated with metastasis and immunomodulation

López-Pacheco, Cynthia Paola [1]; Olivo-Escalante, Karen Donají [1];
Ortega-Soto, Enrique [1]; Soldevila-Melgarejo, Gloria [1 2].

[1]Instituto de Investigaciones Biomédicas, Departamento de Inmunología, CDMX, México.
[2]Instituto de Investigaciones Biomédicas, Laboratorio Nacional de Citometría de Flujo, CDMX, México.

E-mail: cyntlop@iibiomedicas.unam.mx

Extracellular vesicles (EVs) are lipid bilayer-enclosed nano-sized particles released by all cells, essential for intercellular communication. Exosomes, a subtype of EVs, have gained attention as diagnostic and prognostic tools in cancer due to their ability to transfer bioactive molecules. EV cargo including proteins, lipids, and nucleic acids can modulate immune responses within the tumor microenvironment, facilitating immune evasion and promoting an immunosuppressive niche that supports tumor progression. In breast cancer (BCa), EVs play a critical role in remodeling the tumor microenvironment and modulating immune function. Integrins, membrane receptors involved in cell adhesion and extracellular matrix interactions, are EV cargo that contribute to tumor invasion and immune modulation. EV-associated integrins influence immune cell recruitment, polarization, and suppression, thereby aiding pre-metastatic niche formation. This study aimed to characterize integrin profiles of EVs derived from BCa cells with different metastatic potential, to identify novel immunological biomarkers of tumor progression. EVs were isolated from BCa spheroids and analyzed by nano flow cytometry using tetraspanin- and integrin-specific antibodies. EVs isolated from 3D-culture conditioned media seem to be enriched in small particles. Moreover, exosome biomarkers were identified in EVs, suggesting an exosome enrichment. Our data revealed distinct integrin and tetraspanin profiles in EVs from different BCa cell lines, correlating with aggressiveness and metastatic potential. EVs from MDA-MB-231 cells showed increased levels of CD63 and integrins β1, αv, and β5 compared to EVs from MCF7 cells. EVs from non-tumorigenic MCF10A cells lacked detectable β1 integrin expression, highlighting its possible role in cancer progression and immune modulation. Additionally, differential expression of α3 integrin among EVs from MDA-MB-231, MCF7, and MCF10A cells was observed, linked to invasiveness. These findings support the potential of selective integrin profiles in EVs as immunological biomarkers for BCa progression, offering new avenues for therapies targeting tumor-immune interactions to enhance clinical outcomes.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Therapeutic Potential of Nanodiamonds as Nanocarriers of Tofacitinib in Cellular Models of Arthritis

Loya-Rivera, Mario [1]; Gonzáles-Chávez, Susana Aideé [1]; Prieto-Carrasco, Rodrigo [1]; Chaparro-Barrera, Eduardo; Esparza-Ponce, Hilda Esperanza [2]; Pacheco-Tena, César [1].

[1]PABIOM Laboratory, Autonomous University of Chihuahua, Faculty of Medicine and Biomedical Sciences, Chihuahua, Mexico.. [2]Centro de Investigación en Materiales Avanzados (CIMAV), Chihuahua, México.

E-mail: mloyar@uach.mx

Nanodiamonds (ND) can be used as nanocarriers for drug delivery, as they exhibit high biocompatibility and facilitate the drug's passage through the cell membrane, increasing treatment efficacy and reducing cellular toxicity. Tofacitinib (TOFA) a JAK pathway inhibitor is used in the treatment of autoimmune diseases, including arthritis. Despite its clinical indication, TOFA may cause adverse effects; therefore, lower concentrations administered in a targeted manner could help minimize these effects. This project evaluates the effect of a nanodiamond–tofacitinib (ND–TOFA) nanocarrier on the viability of synovial fibroblasts from murine models of rheumatoid arthritis. Nanodiamonds were successfully loaded with TOFA and characterized by transmission electron microscopy (TEM), Fourier-transform infrared spectroscopy (FTIR), hydrodynamic diameter and zeta potential. Cell viability and toxicity were measured using MTT assays in 96-well microplates, with 15,000 cells per well in a volume of 200 µL. Cells were cultured in the presence of ND–TOFA and free TOFA at concentrations of 0.5, 1.0, and 1.5 µM, with a 24-hour exposure, and pristine ND as control. While the viability of fibroblasts was not significantly affected by any concentration of the free drug, treatment with the nanocarrier at 1.5 µM caused a 42% decrease in viability. Additionally, the expression of cytokines IL-1, IL-6, IL-17, IL-23, and TNF were quantified for both the free drug and the nanocarrier.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Intratumoral Administration of RTLV-1424 Modulates Immune Infiltrate in Breast cancer Mouse Model

Lozano-Cisneros, Diego [1,2]; Cortés-Morales, Víctor Adrián [1];
Solorio-Cárdenas, Manuel [1,3]; Torres-Flores, Alejandro [1];
Hernández Aceves, Juan Alberto [4]; Zentella-Dehesa, Alejandro [5,6];
Fragoso-González, Gladis del Carmen [4]; López-Macías, Constantino [1,7].

[1]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México. [2]Posgrado en Ciencias Biológicas, Facultad de Medicina, Universidad Nacional Autónoma de México, México City, México. [3]Facultad de Química, Universidad Nacional Autónoma de México, México City, México. [4]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, México City, México. . [5]Unidad de Bioquímica, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, México City, México. [6]Departamento de Medicina Genómica y Toxicología Ambiental, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, México City, México. [7]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México.
Corresponding: constantino.lopez135@gmail.com.

E-mail: dlc99236z@gmail.com

Breast cancer is the most prevalent and deadly cancer among women globally. Accumulating evidence highlights the pivotal role of the immune system in tumour development and progression, motivating the development of immunomodulatory therapies such as immune checkpoint inhibitors. RTLV-1424, a protein TLR agonist derived from Salmonella Typhi, has demonstrated immunostimulatory activity by promoting the maturation of $CD11c^+$ dendritic cells and inducing robust humoral and cellular immune responses, including the generation of effector and memory T cells. While its adjuvant effects have been documented in melanoma models in combination with anti-PD-1 therapy, its immunomodulatory potential in breast cancer has not been previously explored. In this study, we evaluated the effect of intratumoral RTLV-1424 administration on tumour progression and the composition of the intratumoral immune infiltrate in a murine model of breast cancer generated with 4T1 tumor cell line. After tumour establishment, RTLV-1424 was administered directly into the tumour, and 28 days post-treatment, tumours were harvested for immunophenotypic analysis with flow cytometry. Although RTLV-1424 treatment did not result in a significant reduction in tumour volume. We found an increase in tumor infiltranting leucocyte number. No notable changes were observed in the frequencies of $CD11c^+$ dendritic cells, CD19+ B cells or $NK1.1^+$ natural killer cells. Interestely total T lymphocytes and $CD8^+$ T cells Increased their frequency, also we found that T cells improve INFgamma and TNFalpha expression after RTLV-1424 treatment. These findings suggest that RTLV-1424 exerts immunomodulatory effects that may reshape the tumour microenvironment, thereby supporting its potential use in combination with other immunotherapeutic approaches, particularly immune checkpoint blockade.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

The protein ATG16L1 regulates myeloid cells in the peritoneal cavity

Macedo -Hernández, Mariano I [1]; González, Marisol I [1];
Saucedo, Alberto [4]; Eksteen, Bertus [2]; Vannan, Danielle [3];
Mendiola, Daniela [1]; Quintero Hernández, Jorge [1]; Ramírez-López, Berenice Elizabeth [4]; García Huerta, Eduardo [1];
Reyes, José L [1].

[1]Laboratorio de Inmunología Experimental y Regulación de la Inflamación Hepato-Intestinal, UBIMED, FES Iztacala, UNAM, Tlalnepantla de Baz, Estado de México, México. [2]Aspen Woods Clinic, Calgary, AB, Canada. [3]Boston Scientific Corporation, Urology Division, 200 Boston Scientific Way, Marlborough, MA, USA. [4]Carrera Médico Cirujano, FES Iztacala, UNAM, Tlalnepantla de Baz, Estado de México, México.

E-mail: mariano316ma@gmail.com

The ATG16L1 protein is part of a group of proteins known as autophagy-related proteins. Autophagy is a cellular mechanism responsible for eliminating damaged components (such as misfolded proteins, defective organelles, or pathogens) and recycling them to maintain cellular homeostasis. In addition to its role in autophagy, ATG16L1 has been associated with several immunological processes. Our research group also observed that, under suboptimal expression conditions of ATG16L1, the immunobiology of B cells was altered in a murine model of colitis-associated colorectal cancer. These findings led us to investigate whether ATG16L1 deficiency affects the biology of mononuclear and polymorphonuclear phagocytes in models of both sterile and bacteria-induced peritonitis. For this study, we compared the myeloid cell infiltration kinetics between wild type (WT) and hypomorphic mice for ATG16L1 (ATG16L1HM) during peritonitis induction. To induce sterile peritonitis, mice were intraperitoneally injected with thioglycolate broth and sacrificed at 24, 48, and 72 hours post-injection. For bacterial peritonitis, two different bacterial isolates from patients with peritoneal dialysis-associated peritonitis were used. These mice were euthanized at 4 and 24 hr post-injection. Flow cytometry was employed to immunophenotyping the cells in the peritoneal cavity and revealed that ATG16L1HM mice presented larger populations of neutrophils (CD11b+Ly6G+F480- cells) at 24 hr. Interestingly, most of the neutrophils present in ATG16L1HM mice showed a CD11bhi phenotype as compared to neutrophils from WT animals. Additionally, in terms of macrophages, ATG16L1HM mice exhibited macrophages with a differencial expression of Ly6C. Therefore, ATG16L1 is central regulator of myeloid cells in the peritoneal cavity.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Antileukemic potential of Ganoderma lucidum: Cytotoxicity on THP-1 and K562 cell lines

Machucho-Guzmán, Miguel Ángel [1]; Domínguez-Chávez, Jorge Guillermo [3]; Luria-Pérez, Rosendo [4]; Silva-Cañetas, Carmen Sofía [2]; Sumoza-Toledo, Adriana [2].

[1]Veracruzana University. Master's Program in Translational Biomedicine. Institute of Medical-Biological Research. Veracruz, Veracruz. México. [2]Veracruzana University. Institute of Medical-Biological Research. Veracruz, Veracruz. México. [3]Veracruzana University. Faculty of Bioanalysis. Veracruz, Veracruz. México. [4]Federico Gomez Children's Hospital of México. CDMX. México.

E-mail: Miguelguzmamac@gmail.com

Leukemias encompass a group of hematological malignancies that significantly contribute to mortality among Mexican children, with acute leukemia being particularly prevalent in the 1 to 14-year age group. Ganoderma lucidum is a medicinal fungus known to exhibit anti-tumor activity in several cancer types; however, its effects on leukemias remain unclear. In this study, we evaluated the cytotoxic activity of Ganoderma lucidum extracts on leukemic cell lines THP-1 and K-562. Specimens of Ganoderma lucidum were collected from Cofre de Perote National Park in Veracruz México. The fungus was then dried, pulverized, and subjected to extraction of secondary metabolites using a 1:1 mixture of dichloromethane/methanol. This procedure was carry out twice, yielding four distinct phases, designated as FS1-2, FL1-2, FSAE1-2 and FLNC1-2. Leukemic cell lines and peripheral blood mononuclear cells (PBMCs), used as control for healthy cells, were treated in vitro with 1 μg/mL or 10 μg/mL of each phase for 24 hours. Cytotoxicity was then assessed using the MTT (tetrazolium bromide) assay. The data showed that the FS and FL phases induced cell death in THP-1 and K-562 cells in a dose-dependent manner, while having not cytotoxic effect on PBMCs. Chemical screening indicated the presence of terpenoids in both biological active phases. In conclusion, the compounds derived from the fungus Ganoderma lucidum may have potential as anti-cancer agents because of their selective effects on leukemic cell lines.

Funding for carrying out this research was provided by Institute of Medical-Biological Research. Veracruzana University.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Methyl-donor nutrient intake and global leukocyte DNA methylation in rheumatoid arthritis patients

Macias -Almanza, Gerardo Axel [1 2]; Campos-López, Bertha [1 2];
Pesqueda-Cendejas, Karen [1 2]; Mora-García, Paulina Esmeralda [1 2];
Ruiz -Ballesteros, Adolfo [1 2]; Rivera -Escoto, Melissa [1 2];
De la Cruz-Mosso , Ulises [1 2].

[1]Red de Inmunonutrición y Genómica Nutricional en las Enfermedades Autoinmunes; Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud; Universidad de Guadalajara; Guadalajara, Jalisco 44340, México. [2]Instituto de Neurociencias Traslacionales, Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara 44340, Jalisco, México.

E-mail: gerardo.macias9460@alumnos.udg.mx

Rheumatoid arthritis (RA) is an autoimmune disease influenced by epigenetic factors such as DNA methylation. Hypomethylation could be related to pro-inflammatory genes expression, contributing to disease severity. This process may be influenced by diet; particularly, methyl-donor nutrients like methionine, choline, and B vitamins. This study aimed to assess the association between methyl-donor nutrient intake, global leukocyte DNA methylation with cardiometabolic and clinical variables in RA patients. A cross-sectional study was conducted with 82 female RA patients classified by 2010 ACR-EULAR criteria, and 89 control subjects (CS). Disease activity was assessed by DAS28-C-reactive protein (CRP) , and DNA methylation status with the 5-mC DNA ELISA Kit (Zymo). RA patients showed significantly lower methylation levels compared to controls (2.7 vs. 5.72%; $p < 0.001$). Hypomethylation was more frequent in RA patients (99% ; $p < 0.001$), while high methylation was predominant in controls (62%; $p < 0.001$). Methylation status correlated positively with cobalamin ($r=0.24$; $p = 0.003$), choline ($r=0.17$; $p = 0.03$), and methionine intake ($r=0.23$; $p = 0.004$), and negatively with CRP ($r=-0.24$; $p = 0.001$), body fat ($r=-0.31$; $p < 0.001$), visceral fat ($r=-0.33$; $p < 0.001$), glucose ($r=-0.16$; $p = 0.032$), and triglycerides ($r=-0.25$; $p < 0.001$) serum levels. Notably, hypomethylation status was associated with cardiometabolic risk assessed by hs-CRP levels (OR =2.8; CI: 1.2–6.8; $p = 0.008$), excess weight (OR =10.2; CI: 3.2–41.6; $p < 0.001$), body fat (OR =4.2; CI: 1.5–13.7; $p = 0.003$), cardiometabolic index (OR = 6.7; CI: 2.8–17.1), CRP/albumin ratio (OR =3.06; CI: 1.4–7.0; $p = 0.003$), TyG index (OR = 3.5; CI: 1.5–8.2; $p = 0.001$), and waist-to-height ratio (OR =10.9; CI: 4.1–33.2; $p < 0.001$). In conclusion, a global hypomethylation state of leukocyte DNA was associated with a cardiometabolic risk profile.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Senescence Enhances Pro-Inflammatory Signaling in Mast Cells

Madera-Salcedo, Iris K. [1]; Ibarra-Sánchez, Alfredo [2,4];
Esparza-Reyes, Diana [2,4]; Mendoza-Montiel, Paola [2,4];
Padilla-J, Claudia [3]; González-Espinosa [2,4].

[1]Immunology and Rheumatology Department, National Institute of Medical Sciences and Nutrition Salvador Zubirán. México City, México. [2]Pharmacobiology Department, Center for Research and Advanced Studies (Cinvestav) of the Polytechnic Institute (IPN), South Campus. México City, México. [3]Microbiology and Parasitology Department, Faculty of Medicine, National Autonomous University of México. México City, México. [4]Center for Research in Aging, Cinvestav, South Campus. México City, México.

E-mail: iris.maderas@incmnsz.mx

Mast cells (MC) are long-lived immune effectors involved in inflammation, allergy, and innate immunity. However, senescence-associated changes in their phenotype remain poorly defined. We investigated replicative and stress-induced senescence in bone marrow-derived MCs (BMMCs) from young C57BL/6J mice, using long-term culture and lipopolysaccharide (LPS) exposure, respectively. Both conditions induced hallmarks of senescence, including increased p16INK4A and p21CIP1 expression, SA-β-Gal activity, cell cycle arrest, and metabolic changes, without significant loss of viability. Senescent MCs exhibited a distinctive secretory profile (SASP) marked by elevated IL-6, IL-23, and VEGF, alongside with reduced activation through FcεRI and TLR4. Using aged MC-deficient (c-Kit Wsh/Wsh) mice and c-Kit Wsh/Wsh mice reconstituted with MC, we confirmed the exaggerated cytokine production seen in senescent cells during the acute, canonical MC-dependent response to intraperitoneal LPS administration. Furthermore, MC isolated from chronically LPS-treated animals exhibited elevated basal cytokine levels. Our findings demonstrate that: 1) MC undergo replicative and stress-induced senescence characterized by distinct molecular markers; 2) senescence alters both basal and activated effector functions of MC; and 3) aging is associated with heightened MC-dependent inflammatory responses. Overall, our results indicate that senescence profoundly impacts MC function and may contribute to age-related chronic inflammation, or “inflammaging.” Supported by Secihti.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

Peripheral Neutrophil NFAT5-Aldose Reductase Pathway: A Contributor to Inflammation in Diabetic Retinopathy

Magaña -Guerrero, Fátima Sofía [1]; Buentello-Volante, Beatriz [1];
Magaña -Guerrero, Norma Angélica [1 2]; Domínguez -López, Alfredo [2];
Vivanco -Rojas, Oscar [2]; Garfias -Becerra, Yonathan Omar [1 2].

[1]Instituto de Oftalmología Fundación Conde de Valenciana, IAP, Department of Celular and Tisular Biology, Research Unit, Mexico City, México.. [2]Universidad Nacional Autónoma de México, Department of Biochemistry, Faculty of Medicine, Mexico City, Mexico.

E-mail: fatima.magana@institutodeoftalmologia.org

Diabetic retinopathy (DR), a prevalent microvascular complication of type 2 diabetes (T2D), develops alongside exacerbated chronic inflammation. Neutrophils, key immune cells, contribute to this inflammation through functions like extracellular trap release (NETs) and the secretion of granular compounds and cytokines. The activation of these inflammatory pathways is strongly linked to a poorer prognosis for T2D complications, including diabetic neuropathy, retinopathy, and nephropathy. The nuclear factor of activated T-cells-5 (NFAT5) activates under hyperosmolar and cellular stress conditions. NFAT5 then activates aldose reductase (AR), an enzyme that converts glucose into sorbitol. Unfortunately, excess sorbitol harms retinal endothelial cells, contributing to DR. Given NFAT5's known associations with T2D, obesity, and insulin resistance, we hypothesize that the NFAT5-AR pathway may be altered in neutrophils and contribute to DR progression. This study aimed to determine the expression of NFAT5 and AR in peripheral neutrophils from T2D, DR, and non-T2D subjects, and to evaluate their role in DR pathophysiology. We conducted a case-control study, enrolling individuals with type 2 diabetes (both with and without DR) and non-T2D controls. Each participant underwent clinical, ophthalmological, and metabolic evaluations and peripheral blood neutrophils (PBN) were isolated. We realize the analysis of NFAT5 and AR expression using RT-qPCR and ELISA, respectively. Finally, we performed clinical correlations between relevant variables and inflammatory scores. We observed significantly ($p<0.05$) higher NFAT5 expression in DR subjects compared to T2D subjects. Interestingly, NFAT5 protein concentration was significantly ($p<0.05$) lower in T2D subjects than in controls, though no significant difference emerged in the DR group. Conversely, the AR enzyme concentration was significantly ($p<0.05$) elevated in the DR group compared to T2D and control groups. Our findings indicate an association between DR and elevated AR levels, alongside NFAT5 overexpression, especially on T2D subjects.

Funded by Secretaria de Educación, Ciencia, Tecnologia e Innovacion SECTEI/159/2023, UNAM-PAPIIT-DGAPA:IN210224; Conde de Valenciana Foundation.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

Anti-inflammatory and corneal repair properties of amniotic-membrane extract (AMEx) in a chemical de-epithelization model.

Magaña -Guerrero, Norma Angelica [1 2]; Haro-Morlett, Luis
Andrés [1]; Magaña-Guerrero, Fátima Sofia [1]; Buentello-Volante,
Beatriz [1]; Vivanco -Rojas, Oscar [2]; Domínguez -López , Alfredo [2];
Garfias -Becerra, Yonathan Omar [1 2].

[1]Department of Celular and Tisular Biology Research Unit, Instituto de Oftalmología Fundación Conde de Valenciana, IAP, Mexico City, México. [2]Department of Biochemistry, Faculty of Medicine, Universidad Nacional Autónoma de México. Mexico City, Mexico.

E-mail: namg6398@gmail.com

While the cornea enjoys immune privilege, viral infections and inflammatory injuries can trigger immune responses, causing keratitis and vision loss. Crucially, promoting epithelial cell proliferation and maturation drives effective corneal wound healing. Amniotic membrane (AM) transplantation effectively treats keratitis by suppressing immune cell activation, inducing apoptosis, and down-regulating pro-inflammatory cytokines. AM's therapeutic benefits stem from its stromal components: hyaluronic acid (HA), proteoglycans, fibronectin, laminin, and growth factors. This study determined the impact of soluble factors from amniotic membrane extract (AMEx) on corneal wound healing and their anti-inflammatory properties, specifically examining IL-6 and IL-1β expression within a chemical de-epithelization model. We obtained AMEx from 100 mg of AM. We induced corneal chemical de-epithelization in the right eyes of anesthetized C57BL/6 mice using 99% ethanol for 30 seconds. After de-epithelization, we treated mice topically every 4 hours for 4 days with AMEx 100 ng/mL, vehicle (V), sodium hyaluronate (SH), or saline solution (SSI). We evaluated daily wound closure using in vivo corneal fluorescein staining. We collected tissue samples on days 3 and 4 for Ki67 and keratin 19 analysis by means of immunofluorescence while IL-6 and IL-1β transcripts were measured by RT-qPCR Corneal healing in the AMEx group mirrored the SH group, commencing on day 1 and achieving full epithelialization by days 3 and 4. On day 3, AMEx promoted epithelialization, evidenced by keratin and Ki67 expression. By day 4, AMEx demonstrated near-complete maturation. Interestingly, while SH showed keratin expression and proliferative activity, it resulted in slower epithelial maturation compared to AMEx. AMEx reduced IL-1β expression and increased IL-6 respect to SSI group. In summary, the results suggest that AMEx significantly inhibits the inflammatory environment and enhances epithelialization during corneal wound healing, thus proposing a novel mechanism of action for AM transplantation.

Funded: UNAM-PAPIIT-DGAPA: IN210224; SECTEI: 159/2023; Conde de Valenciana Foundation.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Surface display of scFv-TfR1 enhances bacterial adhesion for bacteria-based anti-lymphoma therapy purposes

Magaño -Bocanegra, Kevin Jorge [1,2]; Cedillo-Barrón, Leticia [2]; Rodríguez-Espino, Benjamín Antonio [3]; García-Nava, Yenny Elizabeth [1,2]; Flores -Martínez, Luis Fernando [1]; Córdoba -Trejo, Adonaí [1]; Luria -Pérez, Rosendo [1].

[1]Unit of Investigative Research on Oncological Diseases, Children’s Hospital of México Federico Gómez, México City, México *Correspondence: rluria@himfg.edu.mx. [2]Department of Molecular Biomedicine, Center for Research and Advanced Studies of the National Polytechnic Institute, México City, México. [3]Unit of investigative Research on Nephrology. Hospital Infantil de México Federico Gómez. Ciudad de México, México.

E-mail: kevin.magano@cinvestav.mx

In recent decades, advances in the treatment of non-Hodgkin lymphoma (NHL) have significantly improved patient survival. However, approximately 20% of patients do not respond to current therapies, highlighting the need for more effective alternatives. In recent years, attenuated strains of bacterial species such as Escherichia coli and Salmonella enterica have been reported to selectively proliferate in tumors, including NHL, promoting an antitumor response. Nevertheless, limited tumor colonization observed in clinical trials has hindered their application. This study proposes enhancing the adhesion of Escherichia coli to human NHL cells that overexpress transferrin receptor 1 (TfR1) by expressing a single-chain variable fragment targeting TfR1 (scFv-TfR1) on the bacterial surface. To achieve this aim, plasmids were constructed to express scFv-TfR1 on the bacterial surface via the ShdA autotransporter system (scFv-TfR1/ShdA30 and scFv-TfR1/ShdA100), along with controls (ShdA30 and ShdA100). In silico structural analyses confirmed the proper folding of the recombinant proteins. Additionally, molecular docking analysis demonstrated the specific interaction between scFv-TfR1 and TfR1. Expression and surface localization of the chimeric proteins in Escherichia coli were validated by dot blot, flow cytometry, and fluorescence microscopy. Bacterial adhesion was assessed using immobilized protein extracts from Ramos cells (NHL), which overexpress TfR1, showing increased binding in strains expressing surface-displayed scFv-TfR1. Finally, enhanced adhesion of scFv-TfR1-expressing Escherichia coli to Ramos cells was confirmed by flow cytometry after direct interaction. These findings demonstrate that surface display of scFv-TfR1 improves bacterial adhesion to NHL cells, representing a step forward in the development of more effective bacterial vectors for targeted immunotherapy.

Funding: Fondos Federales (HIM-2019-061 SSA 1594, HIM-2021-056 SSA 1756, HIM-2022-061 SSA1826, HIM-2024-057).

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Association of number and proportion of B1 lymphocytes with emergency abdominal surgery outcomes

Magdaleno-Villanueva, Juan Alejandro [2 1]; León-Pedroza, José Israel [1]; Rodríguez-Cortés, Octavio [2]; Gutiérrez-Uvalle, Gabriela Elaine [1]; Prieto-Chávez, Jessica Lakshmi [3].

[1]Hospital General de México “Dr. Eduardo Liceaga”, Laboratorio de Metabolómica de la Dirección de Investigación (Unidad 501), Ciudad de México, CDMX, México.. [2]Escuela Superior de Medicina-IPN, Laboratorio de Inflamación y Obesidad, Ciudad de México, CDMX, México.. [3]Instituto Mexicano del Seguro Social (IMSS), Laboratorio de Citometría de la División de Desarrollo de la Investigación en Salud, Ciudad de México, CDMX, México.

E-mail: jam9807@gmail.com

Emergency abdominal surgery (EAS) is used to treat conditions that cause abdominal sepsis. B1 lymphocytes (CD20+CD43+CD38-/lo/intCD27+) are scarce in blood but prevalent in cavities such as the peritoneal cavity. In murine models of abdominal sepsis, B1 deficiency increased mortality. Our objective was to determine whether, in patients undergoing EAS, there is an association between the proportion of B1 lymphocytes (in blood and peritoneal cavity) and progression to a composite outcome consisting of any of the following events: evolution to sepsis, septic shock, death, or the need for reintervention within 28 days.

In a cohort of patients undergoing EAS, biological samples of peripheral blood and peritoneal lavage were obtained. They were stained with a pre-designed panel of antibodies and fluorochromes and acquired on an Aurora cytometer (Cytek ®). Differences in the proportion of B1 lymphocytes according to the composite outcome were compared using Student’s t-test for normal distributions and Mann-Whitney’s U-test for non-normal distributions.

Nineteen patients with a mean age of 50.0 (SD 15.3) years were included. The percentage of B1 lymphocytes relative to total B lymphocytes in blood was on average 4.3 (SD 2.1) in patients without a composite outcome vs. 2.3 (SD 1.1) in those with the outcome ($p = 0.029$). No statistically significant associations were found for B1 lymphocytes in the peritoneal cavity (5.8 SD 5.4 vs. 3.3 SD 2.5; $p = 0.630$). There is an inverse association between the proportion of B1 lymphocytes in peripheral blood and the development of an adverse outcome (sepsis, septic shock, need for reintervention, or death) in patients undergoing EAS, making it a potential prognostic biomarker in this context.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

B55β: Critical mediator of arthritis pathogenesis and modulator of TNF-α inhibitor efficacy

Malvaez-Luis, Dulce R. [1 2]; Gutiérrez-Hernández, Jesús E. [2];
López-Hernández, Laura Q. [2]; Bejar, Perla A. [2]; Guzmán-
Barrenechea, Karla P. [2]; Rosetti, Florencia [2]; Crispín, José C. [1 2];
Madera-Salcedo, Iris K. [2].

[1]Universidad Autónoma del Estado de México, Faculty of Chemistry, Toluca, State of México, México. [2]Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Department of Immunology and Rheumatology, México City, México.

E-mail: dulmalvaezp@gmail.com

Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by persistent inflammation of the synovial membrane, driven by the infiltration of immune cells that secrete pro-inflammatory mediators. Among these, tumor necrosis factor-alpha (TNF-α) plays a central role in the initiation and progression of the disease. Although anti-TNF-α therapies have significantly improved clinical outcomes, approximately 40% of patients fail to respond, underscoring an incomplete understanding of the molecular pathways involved in RA pathogenesis and therapeutic resistance. B55β, a regulatory molecule essential for terminating immune responses, is aberrantly expressed in T cells from RA patients, and its expression and function are modulated by TNF-α. Based on these observations, we hypothesize that dysregulated B55β expression in T cells contributes to immune dysfunction in RA and may impair the therapeutic efficacy of TNF-α inhibitors. To test this, we induced collagen-induced arthritis (CIA) in Ppp2r2bfl/fl CD4.Cre (T cell-specific B55β KO) and Ppp2r2+/+ CD4.Cre (WT) mice, with or without anti-TNF-α treatment (adalimumab). We evaluated disease progression, CD4 T cells infiltration in the synovial tissue (ST) and serum IgG titers. We found that B55β KO mice developed arthritis earlier and exhibited more severe inflammation and cellular infiltration than WT. Furthermore, B55β KO mice showed an increased frequency of follicular helper T cells (Tfh) and a reduction in regulatory T cells (Treg) within the ST, accompanied by elevated levels of anti-collagen IgG. Following anti-TNF-α treatment, both groups showed clinical improvement; however, B55β KO mice exhibited a more pronounced response, marked by normalization of Tfh and Treg cell proportions in the ST. These findings suggest that B55β plays a pivotal role in RA pathogenesis, particularly in regulating Tfh cell function and modulating the therapeutic response to anti-TNF-α treatment.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Evaluation of the biomarker potential of the tetraspanin33 expression in B-cell lymphomas

Mandujano-López, Jorge Vladimir [1 2]; Ayala-Rodríguez, Andrea Monserrat [1 2]; Reyes-Huerta, Raúl Fernando [1]; Velásquez-Ortiz, María Guadalupe [1]; Ríos-Valencia, Javier [6]; López-Ávila, Arath Brandon [3 7]; Baay-Guzmán, Guillermina Juliana [6]; Luria-Pérez, Rosendo [6]; Sandoval-Montes, Claudia [5]; Torres-Ruíz, José Jiram [4]; Maravillas-Montero, José Luis [1].

[1]Instituto de Biotecnología, Universidad Nacional Autónoma de México, Departamento de Medicina Molecular y Bioprocesos, Cuernavaca, Morelos, México. [2]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Posgrado en Inmunología, Ciudad de México, México. [3]Hospital Infantil de México Federico Gómez, Unidad de Investigación en Enfermedades Oncológicas, Ciudad de México, Ciudad de México, México. [4]Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Departamento de Inmunología y Reumatología, Ciudad de México, México. [5]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Departamento de Inmunología, Ciudad de México, México. [6]Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Departamento de Patología, Ciudad de México, Ciudad de México, México. [7]Escuela Superior de Medicina, Instituto Politécnico Nacional, Posgrado en Ciencias de la Salud, Ciudad de México, México.

E-mail: inmunotobi@gmail.com

Many molecules have been identified in the development of different types of cancer, which are involved in migration, invasion, extracellular vesicles production, stemness and drug resistance. Thus, many therapeutic targets have been proposed for cancer treatment, such as tetraspanins. These proteins are a group of transmembrane proteins involved in cellular processes like signal transduction, motility, migration, membrane fusion and phagocytosis. Tetraspanin 33 (TSPAN33) is the most recently reported tetraspanin which expression has been characterized in some types of lymphoma and lymphoma-derived cell lines. “Lymphoma” is a term that comprehends a heterogenic group of neoplasms derived from cells of the lymphoid lineage: B, T or NK cells, and can be classified in Hodgkin lymphomas (HL) or non-Hodgkin Lymphomas (NHL) which can bear up to 50 different entities, varying in cell phenotype, prognostic, treatment and tetraspanin expression. In order to determine the link between the expression of TSPAN33 and clinical data, immunohistochemistry was performed on HL and NHL tissues and therefore correlated with clinical criteria such as treatment response, metastasis and outcome. Here we discovered that the expression of TSPAN33 has a negative correlation with treatment response and a positive correlation with metastasis. In conclusion, the TSPAN33 expression is a biomarker that can be used to predict the lymphoma progression and can be used as a therapeutic target.

Área del artículo: **Linfocitos B**

Novel B Cell Subsets as Potential Biomarkers in Idiopathic Inflammatory Myopathies: Insights into Disease Pathogenesis and Disease Activity

Mandujano-López, Vladimir [1], Reyes-Huerta, Raúl [1],
Alcalá-Carmona Beatriz, [3], Ostos-Prado María J. [3],
Velásquez-Ortiz, Guadalupe [1], Torres-Ruiz, Jiram [3],
Gómez-Martín, Diana [3] and Maravillas-Montero José L.[1]

1 Instituto de Biotecnología, Universidad Nacional Autónoma de México, 2 Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, 3 Departamento de Inmunología y Reumatología, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán

* E-mail: maravillas@unam.mx

Idiopathic inflammatory myopathies are a heterogeneous group of rare autoimmune disorders characterized by progressive muscle weakness and the histopathologic findings of inflammatory infiltrates in muscle tissue. The diagnosis of IIM is based on clinical manifestations and is complicated due to the lack of reliable clinical biomarkers. Although their pathogenesis remains indefinite, the detection of specific autoantibodies and the evidence of the high effectiveness of depleting therapies suggest that B cells could be implicated. However, little is known about changes in peripheral blood B cell subsets in IIM patients and how these changes are related to disease activity. Therefore, we isolated PBMCs from 27 patients and 24 healthy controls and explored the landscape of peripheral B cells in this disease by multiparametric flow cytometry. We found significant numerical decreases in memory and double negative subsets, as well as an expansion of the naïve compartment relative to healthy controls, that contribute to defining disease-associated B cell subset signatures and correlating with different clinical features of patients such as the Visual Analog Scale (VAS) for disease activity and the Manual Muscle Testing (MMT8) muscle strength scale. Additionally, we determined the potential value of these subsets as diagnostic biomarkers, thus positioning B cells as neglected key elements possibly participating in the onset or development of idiopathic inflammatory myopathies. Funding: SECIHTI CBF-2025-I-3132 and DGAPA-PAPIIT-UNAM IN215424.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Dissecting TGF-β Pathway in Melanoma Immunity Using CRISPR/Cas9 Gene Editing

Marcial -Medina, Ma. Cleofas [1]; Ramos-Balderas, José Luis [1];
Licona-Limón, Paula [1].

[1]Instituto de Fisiología Celular, Departamento de Biología Celular y del Desarrollo, Universidad
Nacional Autónoma de México, México City, México.

E-mail: cmarcialm@hotmail.com

The transforming Growth Factor beta (TGF-β) pathway is a crucial immunosuppressive regulator within the tumor microenvironment, particularly in suppressing cytotoxic T cell-mediated anti-tumor immunity. Understanding the molecular mechanisms that enable the diverse effects induced by this cytokine in various lymphocyte populations could help identify molecular targets for cancer treatment. Clear evidence suggests that inhibiting TGF-β may be a potentially effective therapeutic strategy. Therefore, the objective of this project is to evaluate the therapeutic effects of blocking the activity of different molecular components of the TGF-β pathway in CD8+ lymphocytes and the tumor microenvironment. Current immunotherapies are also being evaluated in combination with the inhibition of TGF-β signaling. Uncovering the molecular mechanisms that promote or impede an effective antitumor response will provide guidelines for developing new therapeutic strategies with greater efficacy in cancer treatment. Our results indicate that deleting SMAD4 or TIF1γ in OT-1 cells using CRISPR/Cas9 can increase the percentage of OT-1 cells exhibiting a cytotoxic phenotype. Similarly, the deletion of SMAD3 or TIF1γ may enhance NK cell infiltration into the tumor. Additionally, chemical inhibition of TGFBR1 in the tumor demonstrated strong therapeutic potential by improving tumor growth control, increasing IFNγ expression in CD8+ lymphocytes, enhancing cytotoxic activity in NKT cells, and reducing PD1 expression in CD4+ lymphocytes. Furthermore, the combination of the inhibitor SIS3 with SB-505124 exhibited strong therapeutic potential by promoting better tumor control, increasing GzB and IFNγ expression in CD8+ lymphocytes, enhancing cytotoxic activity in NKT cells, and decreasing PD1 expression in CD4+ lymphocytes. Overall, these results indicate that inhibiting TGF-β signaling in CD8+ cells and within the tumor microenvironment shows promise as a therapeutic strategy for melanoma treatment.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos B**

Human B-1-like lymphocytes: truly similar to murine B-1 cells?

Martínez Gutiérrez, Mónica Itzel [1]; Correa Beltrán, María Dolores [2];
Gómez Chávez, Fernando [3]; Cañedo Solares, Irma [4];
León Pedroza, José Israel [2 5]; Flores Rabasa, Regina [2];
Magdaleno Villanueva, Juan Alejandro [5].

[1]Plan de Estudios Combinados en Medicina. Facultad de Medicina, Universidad Nacional Autónoma de México. Ciudad de México, México. [2]Centro de Investigación en Ciencias de la Salud. Facultad de Ciencias de la Salud, Universidad Anáhuac México. Huixquilucan, Estado de México, México. [3]Laboratorio de Enfermedades Osteoarticulares e Inmunológicas, Sección de Estudios de Posgrado e Investigación, Escuela Nacional de Medicina y Homeopatía– Instituto Politécnico Nacional. Ciudad de México, México. [4]Laboratorio de Inmunología Experimental. Subdirección de Medicina Experimental. Instituto Nacional de Pediatría, Secretaría de Salud. Ciudad de México, México. [5]Hospital General de México Eduardo Liceaga, Ciudad de México, México.

E-mail: monica.itzel.martinez17@gmail.com

B-1 cells are a subset of B cells with innate-like properties discovered in the early 1980s in mice. They are important actors in homeostasis, partially because they secrete natural antibodies, which have scavenging-helping functions. They mainly reside in pleural and peritoneal cavities, as well as in the spleen and lamina propria of the intestinal mucosa. They differ from conventional (B-2) cells in their ontogeny, phenotype and functions. In humans, the search for a B-1 cell counterpart began shortly after their discovery in mice, but there were several problems to identify them, due to inaccurate phenotypic markers and inaccessibility to sites in where they reside. Three decades later, it was proposed that B-1-like cells in humans can be identified by functional features, which lead to the phenotype CD19+ CD20+ CD27+ CD38low/int CD43+. Such discovery was vigorously debated, but their existence and equivalence with murine B-1 cells has been gaining acceptance in recent years. These human B-1-like cells are present in blood, skin, peritoneum and spleen, as well as in fetal tissues. They present tonic intracellular signaling and secrete antibodies in a spontaneous manner that share some specificities with classical natural antibodies (i.e. phosphorylcholine, polysaccharides). However, they present several differences with the murine B-1 cells, like the presence of somatic hypermutation and the spontaneous secretion of IgG. Also, there are unresolved questions regarding their residence sites, origin during fetal life and role in homeostasis maintenance and infection control. In this narrative review we discuss the current findings about B-1 like cells in humans and compare them with murine B-1 cells, in order to establish equivalencies and differences. These cells may have important functions in aging, autoimmunity, and regulation of other diseases.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Cytotoxic Molecules as Potential Biomarkers for Active and Inactive Systemic Lupus Erythematosus

Martínez -Iturbe, Iyari Iletse [1 2]; Santana-Sánchez, Paola [1 2];
Ramírez -Pérez, Astrid Asminda [3]; Alberti-Minuti, Paolo [4];
Gajon, Julián A [5 6]; Bonifaz, Laura C [6 7]; Sánchez Escobar,
Norberto [1]; Legorreta -Haquet, María Victoria [1];
Chávez -Sánchez, Luis [1]; Chávez Rueda, Adriana Karina [1].

[1]Unidad de Investigación Médica en Inmunología, Unidad Médica de Alta Especialidad (UMAE) Hospital de Pediatría, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México. [2]Doctorado en Ciencias Biológicas y de la Salud, Universidad Autónoma Metropolitana, Ciudad de México, México. [3]Servicio de Reumatología, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México. [4]Servicio de Medicina Interna, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México. [5]Posgrado en Ciencias Bioquímicas, Universidad Nacional Autonoma de México, Ciudad de México, México. [6]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México. [7]Coordinación de investigación en salud, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México.

E-mail: Iyari689@gmail.com

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease. Although their prognosis has improved over the last decades, the mortality rate in these patients is 4.5 times higher than in the general population, so it is important to know the molecular, cellular, and immunological mechanisms involved in the development of the disease. CD4 and CD8 T cells play a relevant role in SLE; they present greater hyperreactivity and secretion of proinflammatory cytokines, contributing to tissue damage and exacerbation of the disease. The activation of CD4 T cells promotes their differentiation into cell populations such as Th1, Th2, Th17, producers of IFNγ, IL4 and IL-17 respectively. While CD8 mainly secrete IFNγ, granzyme B (GZMB), perforins and granulysin. Deregulation of the secretion of these cytokines in SLE has been explored in patients and animal models. However, studying their prevalence together and in relation to disease activity may result in the differentiation of pathological subpopulations of T cells, such as exhausted T cells. Peripheral blood samples were collected from patients with active and inactive SLE. For serum determination of cytokine concentration, the commercial LEGENDplex™ Human CD8/NK Panel (13-plex) kit was used. Cellular events were acquired using the MACSQuant Analyzer 10 cytometer (Miltenyi Biotec, Germany), and the results obtained were analyzed with LEGENDplex software. As a control group, samples were obtained from healthy donors. In this study we investigated the concentration of 13 cytokines: IL-17A, IL-2, IL-4, IL-10, IL-6, TNF-α, Fas, FasL, IFN-γ, Granzyme A, Granzyme B, Perforin, Granulysin. We studied the behavior of cytokines in patients with active and inactive SLE and identified a subset of SLE patients with increased levels of cytotoxic molecules. The study of molecules addressed here could be important for monitoring the possible cell populations involved in the production of these, as well as for patient monitoring.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Low levels of miRNA-125b are associated with a poor response to neoadjuvant treatment in patients with breast cancer

Martínez-Leija, Miguel Ernesto [1 2]; Hernández-Hernández, Gloria Estela [1]; Aranda-Herrera, Zulema [2]; Paz-Rodríguez, Víctor Alejandro [1 2]; Turijan -Espinoza, Eneida [2]; González-Hernández, Osiel [1]; Ibáñez-Méndez, Reyes de Jesús [1]; Guel-Pañola, Arturo [3]; Portales -Pérez, Diana Patricia [1 2].

[1]Laboratorio de Medicina Molecular y Traslacional, Centro de investigación en Ciencias de la Salud y Biomedicina (CICSaB), Universidad Autónoma de San Luis Potosí, S.L.P., México. [2]Laboratorio de Inmunología y Biología Celular y Molecular (IBCM), Facultad de Ciencias Químicas, Universidad Autónoma de San Luis Potosí. [3]Hospital Central "Dr. Ignacio Morones Prieto", San Luis Potosí, SLP, México.

E-mail: mernesto.dfs20@gmail.com

The molecular mechanisms of chemoresistance development during neoadjuvant therapy in breast cancer patients are unknown. A previous microarray analysis of tumor tissue and serum, combined with bioinformatic analysis by our research group, identified miR-125b and miR-145 as potential biomarkers associated with response to neoadjuvant treatment. The goal of this project was to evaluate the serum expression levels of miR-125b and miR-145 as potential prognostic biomarkers for disease progression and treatment response in breast cancer patients. Serum levels of miR-125b and miR-145 were measured in breast cancer patients before therapy (T0) and five months (T5) afterward. We evaluated the results from PCR, along with clinical, anthropometric, diagnostic, and treatment response variables from patients at both T0 and T5 time points using Principal Component Analysis (PCA). This analysis allowed us to identify correlation patterns between the variables associated with miRNA expression in either chemoresistant or chemosensitive patients, as well as in the four molecular subtypes (Luminal A, Luminal B, HER2+, and Triple Negative (TN)). The PCA results indicate that, prior to receiving neoadjuvant therapy, higher levels of miR-125b were associated with patients who had the HER2+ or TN subtypes and who responded well to treatment. In contrast, miR-145 was exclusively associated with the TN subtype. Real-time PCR data analysis revealed a significant decrease in serum miR-125b expression levels in patients who were chemoresistant or had the HER2+ or TN subtypes after five months of therapy. During this same period, miR-145 expression levels increased only in patients with stable disease (SD) or with the Luminal A or B subtypes. Our results suggest that serum expression of miRNA-125b serves as a biomarker for monitoring resistance to neoadjuvant therapy in breast cancer patients. Meanwhile, expression of miRNA-145 may predict a favorable treatment response in patients with Luminal A and B subtypes.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Valproic Acid: Novel antitumor strategy for the treatment of canine mastocytoma

Martínez Martínez, Ricardo E [1]; Nava Santos, José A [1];
Modesto Camacho, Ingrid T [1]; Rodríguez López, Gloria M [2];
Ruiz Sánchez, Bibiana P [1]; Muñoz Cruz, Samira [3];
Chávez Blanco, Alma D [4]; Pérez Tapia, Sonia M 1 [5];
Pavano Rodríguez, Claudia G [6]; Chacón Salinas, Rommel [1].

[1]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, México City, México. [2]Departamento de Microbiología e Inmunología, Facultad de Medicina Veterinaria y Zootecnia, Universidad Nacional Autónoma de México, México City, México. [3]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México. [4]División de Ciencia Básica, Instituto Nacional de Cancerología, Subdirección de Investigación Básica, México City, México. [5]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, México City, México. [6]CECYT 10, Instituto Politécnico Nacional, México City, México.

E-mail: ricard.martinezmartinez@gmail.com

Canine mastocytoma is the most common cutaneous tumor in dogs, characterized by aggressive behavior, metastatic potential, and high rates of relapse and mortality. Valproic acid (VPA), a histone deacetylase inhibitor, has demonstrated antitumor activity in various human and murine models, but its effect on canine mastocytoma remains largely unexplored. The aim of this study was to characterize the potential antitumor effect of VPA on the HRMC cell line, which is derived from a canine mastocytoma. To conduct this study, an MTT assay was performed to assess cell viability and calculate inhibitory concentrations (IC50 and IC75). Additionally, Annexin V/Propidium Iodide staining was used to identify the type of cell death induced, and Propidium Iodide alone was used to evaluate cell cycle effects. We observed a consistent reduction in cell viability, with IC50 values around 4 mM at 24, 48, and 72 hours of exposure. Furthermore, an important pro-apoptotic effect was observed in cells treated with the IC50, with secondary apoptosis predominating at 24 hours post-exposure and primary apoptosis becoming more prominent at 48 and 72 hours. Moreover, alterations in the cell cycle were observed, suggesting polyploidy in VPA-treated cells, although this effect requires further studies for confirmation. In summary, VPA exhibits significant antitumor activity against the HRMC cell line, offering a novel therapeutic approach for canine mastocytoma and opening new research opportunities into its effects on these tumors.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Immunophenotypic alterations in people occupationally exposed to pesticides in Monte Blanco, Veracruz

Martínez-Meza, Perla Susana [1]; Zavaleta-Reyes, Brenda Lizeth [1]; Montero-Ruíz, Oreth [1]; Santiago-Roque, Isela [2]; Pérez-Lara, Jocelyn Carolina [3]; Palma-Jacinto, José Antonio [4]; Gallardo-Hernández, Carlos Arturo [4].

[1]Universidad Veracruzana, Facultad de Bioanálisis, Xalapa, Veracruz, México. [2]Universidad Veracruzana, Laboratorio de Investigación en Neurotoxicología y Bioquímica, Xalapa, Veracruz, México. [3]Centro de Investigación y Estudios Avanzados del IPN, Departamento de Biomedicina Molecular, CDMX, México. [4]Universidad Veracruzana, Unidad de Servicios Analíticos en Salud Bioanálisis, Xalapa, Veracruz, México.

E-mail: perlasusanaxm@gmail.com

Pesticides are chemical constituents used to prevent or control pests, including insects, rodents, fungi, weeds, and other unwanted organisms. These substances are easily accessible to agricultural workers and widely use in diverse country include Mexico. Despite their advantages in crop production and disease management, the use indiscriminate of pesticides poses significant hazards to the environment and public health. Regular pesticide use has been observed among agricultural workers in the community of Monte Blanco, in Teocelo, Veracruz. To determine whether pesticides influence immunity, an observational comparative cross-sectional study was conducted with a population of farmers exposed to pesticides in this community. Whole blood samples were isolated to identify B and T lymphocytes, as well as analyzing CD4+ and CD8+ T cells subpopulations through flow cytometry. The results show an increase in proportion of leukocytes; however, the lymphocyte subpopulations showed a notable decrease in people exposed to pesticides, specifically total B cells, CD4 and CD8 T cells. In this context, the decrease in these cells suggests the inhibition of anti-apoptotic factors that could promote pro apoptotic signaling pathway. Together, these results demonstrate the impact to pesticides exposure in the immune system and highlight the importance of regulating its use and using appropriate protective equipment.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Effect of acute stress on mast cells and ileum epithelium of mice.

Martínez-Morales, Javier Alejandro [1]; Díaz-Ayala,
Patricia Fabiola [1]; Arciniega-Martínez, Ivonne Maciel [2];
Reséndiz-Albor , Aldo Arturo [1].

[1]Laboratorio de Inmunidad de Mucosas, Sección de Estudios de Posgrado e Investigación, Escuela Superior de Medicina del Instituto Politécnico Nacional. [2]Laboratorio de Inmunonutrición, Sección de Estudios de Posgrado e Investigación, Escuela Superior de Medicina del Instituto Politécnico Nacional.

E-mail: jalejandro.mtz.unam@gmail.com

It has been reported that stress can alter several physiological systems, altering the immune response in the intestine. These alterations involve an increase in proinflammatory cytokines and tight junction proteins. However, there exists a lack of studies focusing on the effects of acutes stress addressing histological changes, inflammatory response, mast cells and T lymphocytes in the distal portion of the small intestine. The objective of this study is to determine the effect acute stress on mast cells and ileum epithelium. We used two study groups: 1) control group was maintained under physiological conditions. The stress group was a model of immobilization for 4 hours. Serum was recollected for corticosterone determination, also small intestine samples were recollected for histological analysis, and proinflammatory cytokines and T regs cells were measured by flow cytometry. Results showed modifications of the ileum epithelium, with increased villi length and intervillous cellularity, the number and size of goblet cells and an increase in the number of mast cells in the stressed group. Proinflammatory cytokines (IL-1β, IL-6, TNF-α, IL-12, IFN-g) and T regs cells also showed an increase in stress group compared to control group. We conclude that acute stress causes various modifications at the histological and cellular level, in BALB / c mice, due to the action of the corticosterone released during this condition.

This work was supported by SIP 20250872 and COFFA-IPN.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Diazinon-induced immunotoxicity on Oreochromis niloticus challenged with Aeromonas hydrophila

Martínez-Pérez, Vasti Elena [1]; Mercado-Salgado, Ulises [2];
Ulloa-Ramírez, Brandon Bladimir [1]; Barcelos García, Rocío
Guadalupe [2]; Girón Pérez, Daniel Alberto [2]; Covantes Rosales,
Carlos Eduardo [2]; Girón -Pérez, Manuel Iván [2];
Toledo-Ibarra, Gladys Alejandra.

[1]Unidad Académica de Ciencias Químico Biológicas y Farmacéuticas, Universidad Autónoma de Nayarit. Tepic, Nayarit, México. [2]Laboratorio Nacional para la Investigación en Inocuidad Alimentaria (LANIIA-Unidad Nayarit). Universidad Autónoma de Nayarit. Tepic, Nayarit, México.

E-mail: 19008330@uan.edu.mx

The growth of the global population has made pesticide use essential for maintaining or increasing crop yields. Pesticides are any substances used to prevent, destroy, or control pests. The most widely used pesticides worldwide are organophosphates. In Mexico, there is a lack of detailed information regarding their use, regulation, or monitoring, which may pose an environmental risk since these compounds exhibit neurotoxic and immunotoxic effects on exposed organisms. This study proposes a mechanism of immunotoxicity caused by the deregulation of cell communication mediated by cytokines. It evaluates the expression of pro-inflammatory and anti-inflammatory cytokines, as well as transcription factors, in response to an antigenic challenge with Aeromonas hydrophila and co-exposure to diazinon, using Nile tilapia fish (Oreochromis niloticus) as the study model. Adult male fish (O. niloticus) were used. For the antigenic challenge (72 h), a bacterial inoculum of A. hydrophila (9.0 × 108 CFU) was prepared, and commercial diazinon was applied in 30 L fish tanks (0.97 mg/L/24 h). Subsequently, spleen mononuclear cells (SMC) were isolated, RNA was extracted using the TRIzol method, and cDNA synthesis was performed. Finally, a quantitative PCR (qPCR) was performed. Relative gene expression was calculated using the comparative CT method (2-ΔΔCT). The results show a significant decrease ($p < 0.05$) in the expression of IL-6 (a pro-inflammatory cytokine) and IL-10 (an anti-inflammatory cytokine) in SMC, indicating the deregulation of communication mechanisms caused by organophosphate pesticides. This impairs the organism's ability to respond to antigenic challenges due to an altered inflammatory response. Therefore, the indiscriminate pesticide use poses a risk of incidental exposure to economically important organisms such as tilapia, leading to losses in aquaculture-related economic activities.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

CD4⁺ T Cell Phenotypes May Distinguish ICB Responders from Non-Responders in Melanoma

Martínez-Sarabia, Joahan K.; De León Rodríguez, Saraí G.;
Aguilar Flores, Cristina; Bonifaz-Alfonzo, Laura.

[1]Universidad Nacional Autónoma de México, Facultad de Química, CDMX., CDMX., México. [2]Instituto Mexicano del Seguro Social, UIMI, CDMX., CDMX., México. [3]Instituto Mexicano del Seguro Social, UIMI, CDMX., CDMX., México. [4]Instituto Mexicano del Seguro Social, Coordinación de Investigación en Salud, CDMX., CDMX., México.

E-mail: 318033646@quimica.unam.mx

Melanoma is a type of skin cancer that arises from melanocytes, the melanin-producing cells of the skin. This disease is the deadliest form of skin cancer, and its incidence and mortality are rising worldwide. Immune checkpoint blockade (ICB) reinvigorates the immune response against the tumor and achieves response rates of up to 60%, increasing patient survival about 72 moths. However, 40% of individuals who receive ICB are considered non-responders. Therefore, it is important to develop strategies to improve treatment selection. $CD4^+$ T cells have been observed as an expanding population after ICB, suggesting that they may be a direct target of the therapy. Higher progression-free survival has been observed in patients with a greater density of $CD4^+$ T cells. However, transcriptomic signatures associated with proliferating $CD4^+$ T cells have also been linked to non-responder patients. This contrasting evidence may be attributed to the different helper phenotypes that $CD4^+$ T cells can develop. Based on their profiles, these cells can exert either pro-tumoral or anti-tumoral effects. This study aims to identify which $CD4^+$ T-cell profiles are associated with response to ICB therapy. To achieve this, we performed immunofluorescence analysis of melanoma biopsies (stage III or IV) taken before the initiation of ICB therapy. Our results suggest that the Th1 subpopulation is associated with a positive response to therapy. In contrast, Th2 and Treg cells were present at higher levels in non-responder patients compared to responders. Th17 profile did not show a statistically significant difference. Although the precise mechanisms by which these subpopulations influence the response to ICB remain unclear, we propose that the cytokines released by different Th subsets shape the tumor microenvironment, thereby influencing patient outcomes. These preliminary findings may serve in the future as a clinical tool to help therapy selection, improving precision medicine.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

The peritoneal cestode Taenia crassiceps restructures gut bacterial communities in the mouse host

Mateos -Arenas, Diego [1]; Ruiz -De la Cruz, Miguel [2];
Martínez-Gregorio, Héctor [2]; González-González, Marisol Ibet [1];
Vaca -Paniagua, Felipe [2]; Díaz-Velázquez, Clara E. [2];
Eksteen-, Bertus [3]; Vannan, Danielle [4]; Terrazas-Valdés, Luis I. [5];
Reyes-Hernández, José Luis [1].

[1]Laboratorio de Inmunología Experimental y Regulación de la Inflamación Hepato-Intestinal, UBIMED, FES Iztacala, UNAM, Tlalnepantla de Baz, Estado de México, México. [2]Laboratorio de Genómica Integrativa del Cáncer y Enfermedades Infecciosas, UBIMED, FES Iztacala, UNAM, Tlalnepantla de Baz, Estado de México, México. [3]Aspen Woods Clinic, Calgary, AB, Canada.. [4]Boston Scientific Corporation, Urology Division, 200 Boston Scientific Way, Marlborough, MA, USA. [5]Laboratorio de Inmunoparasitología, UBIMED, FES Iztacala, UNAM, Tlalnepantla de Baz, Estado de México, México.

E-mail: diegomateos2002@gmail.com

The NLRP3 inflammasome is a crucial protein complex in the innate immune response assembled in response to pathogens. It has been reported that the NLRP3 receptor is involved in both Th1 and Th2 immune responses, with the latter observed in helminth infections, where NLRP3 can play a regulatory role depending on the type of helminth involved in the infection. One example of such infections is the experimental infection with Taenia crassiceps, where this parasite induces a Th2 response and suppressive cells, mainly macrophages. Another important factor related to the modulation of the immune response and helminth infection is the microbiota, as its composition can positively and/or negatively affect the survival and proliferation of helminth parasites. However, it is unknown whether infection with T. crassiceps can modify this component. Additionally, it is known that NLRP3-deficient mice show greater resistance to T. crassiceps, Thus, the aim of this work is to analyze the composition of the intestinal bacterial microbiota in NLRP3-deficient mice and WT mice infected with T. crassiceps to identify potential bacteria influencing resistance to this parasite. To achieve this, massive sequencing of the V3 and V4 subunits of the 16s ribosomal RNA gene extracted from mouse feces at different infection times was performed. A change in microbial composition was observed across the different study groups, with notable changes in the relative abundance of taxa from the genera Lactobacillus, Dubosiella, Prevotella, Muribaculum, Ileibacterium, Lachnospira, Akkermansia, Bifidobacterium, and Clostridium across different strains and infection times. This indicates that both NLRP3 and T. crassiceps influence the diversity and composition of the intestinal microbiota. However, the specific relationship between these bacteria and the aforementioned factors remains unknown, and further studies focused on this relationship are suggested.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

LITAF-like Transcription Factor, a new regulator of the immune response in Drosophila

Maya-Maldonado, Krystal [1 2]; Rowe-, Bella [2]; Kurien-, Natania [2];
Chou- , Amber [2]; Broderick-, Nichole [2].

[1]Center for Research and Advanced Studies of the National Polytechnic Institute (CINVESTAV), Biochemistry Department, Mexico City, Mexico. [2]Johns Hopkins University, Department of Biology, Baltimore, Maryland, USA.

E-mail: kmaya@cinvestav.mx

Immune responses are critical for the host to maintain a normal microbiome and prevent the establishment of pathogens. Thus, alterations in basal immune gene expression influenced by disequilibrium in microbiome homeostasis may have subsequent effects on infection susceptibility. Historically, Drosophila melanogaster has been a useful tool to study the immune response and host-microbe interactions due to the genetic tractability of the model as well as the simplicity of associating it with specific bacterial communities. Most research regarding the maintenance of microbes in the insect gut is focused on the classic immune pathways, meaning that additional pathways or components of these pathways remain less understood or unexplored. In this work, we characterized a sequence that putatively encodes the Lipopolysaccharide-induced tumor necrosis factor-α-like (LITAF-like) Transcription Factor (TF) as a potential new immune regulator of the innate immune response. By using LITAF-like TF loss-of-function mutant flies, we evaluated impacts on the microbiome and found an increase in microbiome density in females, indicating dysregulation of the microbial community in the gut. In addition, we exposed these mutant fly lines to a solution of Pseudomonas entomophila (Pe), one of the most common pathogens used to study immune responses in Drosophila. We found that survival in infected LITAF-like TF mutant flies, both females and males, it is similar to the survival of immune-deficient (REL less) infected flies, indicating that the loss of the LITAF-like TF gene increases susceptibility to Pe infection. In addition, we observed that the loss of function of LITAF-like TF alters immune gene expression, with effects differing between females and males, suggesting sex-specific effects mediated by LITAF-like TF. Our results identify LITAF-like TF as a new regulator of the immune response in the Drosophila gut. In addition, our work provides insights into sex-specific regulation of immune responses.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

Systematic review for GPR15 / GPR15/L as a biomarker or drug target in inflammatory diseases.

Medina-Sosa, Kenia [1,2]; Rivas-Saucedo, Estefanía [1,2];
Rivas-Santiago, Bruno [2]; Macias-Segura, Noe [3]; Castillo-Torres,
Andrea [1,2]; Castañeda-Delgado, Julio Enrique [2 4].

[1]Unidad Académica de Ciencias Químicas, Universidad Autónoma de Zacatecas. [2]Unidad de Investigación Biomédica de Zacatecas, Instituto Mexicano del Seguro Social, Zacatecas 98000, México. [3]Universidad Autónoma de Nuevo León, Facultad de Medicina, Departamento de Inmunología, Monterrey, Nuevo León, México. [4]Investigadores por México, Secihti, Secretaría de ciencias, humanidades, tecnología e innovación. Ciudad de México, México.

E-mail: 37184147@uaz.edu.mx

This systematic review focuses on GPR15, a G protein-coupled receptor involved in the pathophysiology of various inflammatory diseases. Initially identified as a co-receptor for HIV and SIV, GPR15 shares structural features with chemoattractant receptors. Its natural ligand, C10orf99—a CC chemokine-like peptide—forms a signaling axis with GPR15 that regulates immune cell migration, homeostasis, and inflammation. Additionally, GPR15 has C10orf99-independent roles in disease processes. It is highly expressed in barrier tissues such as skin and mucosal epithelium, where it supports immune cell localization and barrier function. These properties make GPR15 and its ligands promising targets for future therapeutic development.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

Improving fibromyalgia diagnosis: optimizing tests

Meester-, Irene [1]; Moreno-Ramírez, José Alfredo [1];
Nakagoshi-Cepeda, María Argelia Akemi [2]; Solís -Soto, Juan Manuel [2].

[1]Universidad de Monterrey, Escuela de Medicina, Monterrey, Nuevo León, México. [2]Universidad Autónoma de Nuevo León, Facultad de Odontología, Monterrey, Nuevo León, México.

E-mail: elisabethd.meester@udem.edu

Fibromyalgia is characterized by chronic widespread pain, fatigue, and cognitive difficulties. Affecting 2-5% of adults, its cause is unknown, and there is currently no definitive diagnostic test. Therefore, we aimed to develop and optimize a laboratory test based on the hypothesis of an autoimmune etiology targeting the body´s fascia or nervous system. Since the antigen is unknown and human tissue scarcely available, screening was done against murine organs in two formats: crude protein extracts for ELISA and histological sections for immunohistochemistry (IHC). Hereto, adult mice (strain 129, either sex) were anesthesized, perfused, and sacrificed to disect brain, spinal cord with or without the vertebrate column, skeletal muscle, intestine, and kidney. Technical optimization was done with commercial detection antibodies against endothelium (anti-CD34) and axons (anti-TUBB-III) and horse-radish peroxidase-conjugated secondary antibodies. For IHC, optimization variables were: fixation, decalcification, epitope retrieval, slide adherence, and antibody concentration. For ELISA, organs were homogenized in T-PER solution. The protein concentration in crude extracts were quantified with Bradford. ELISA optimization involved dilution series. TUBB-III immunoreactivity was easily obtained, but IHC optimization proved challenging for the CD34 antibody due to the competing interests of adherence and antigen retrieval requirements and specific immunoreactive staining was only achieved on floating sections Thus, IHC optimization is an on-going process. As for the in-house developed ELISA protocol, reproducible results were easily obtained using adherent 96-well plates, albumin blocking, and 50 µg protein per well. In conclusion, the in-house developed ELISA is optimized and ready to be used for screening with diluted patient sera to verify whether they contain autoantibodies. Ethical approval: CEI-EM 11-2023-01; Funds: UDEM, UIN20061 and UANL, Proacti 2023.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Perinatal Sucralose Intake Impairs Antitumor Immunity and Promotes Colitis-Associated Tumorigenesis

Mejía -Muñoz, Aranza Daniela [1]; Arroyo -Olarte, Rubén Darío [1,2];
Rocha -Martínez, Karla Ximena [1,2]; Salinas -Lara, Citlaltépetl [3];
Sánchez -Garibay, Carlos [3]; León -Cabrera, Sonia Andrea [1,2].

[1]Facultad de Estudios Superiores-Iztacala. Universidad Nacional Autónoma de México. Unidad de Biomedicina. Edo. De México, Tlalnepantla, México. [2]Facultad de Estudios Superiores-Iztacala. Universidad Nacional Autónoma de México. Carrera de Médico Cirujano. Edo. De México, Tlalnepantla, México. [3]Instituto Nacional de Neurología y Neurocirugía "Manuel Velasco Suárez". Departamento de Neuropatología. Ciudad de México, México.

E-mail: aranzamar6@gmail.com

Colorectal cancer (CRC) is one of the most prevalent and lethal malignancies worldwide. Notably, its incidence is rising among younger populations in developed countries, a trend potentially linked to increased consumption of dietary additives, including non-caloric artificial sweeteners (NCAS). Sucralose, a commonly used NCAS, is frequently consumed during pregnancy. Emerging evidence suggests that maternal sucralose intake may predispose offspring to metabolic dysfunction and chronic low-grade inflammation. However, the long-term consequences of perinatal sucralose exposure on intestinal homeostasis and CRC susceptibility remain poorly understood. This study aimed to characterize the impact of gestational and lactational sucralose exposure on intestinal immune responses and CRC progression in murine offspring. Pregnant C57BL/6 mice were administered sucralose (8 mg/mL) or pure water throughout gestation and lactation. Offspring from sucralose-fed (F1-Suc) and control (F1-Ctl) dams were maintained on a standard chow diet post-weaning. F1-Suc mice exhibited intestinal dysbiosis, reduced mucin-producing goblet cells, and epithelial disruption compared to F1-Ctl. Upon induction of colitis-associated CRC via azoxymethane (AOM) and dextran sulfate sodium (DSS), F1-Suc mice showed exacerbated disease, with greater weight loss, elevated disease activity index, shortened colon length, increased tumor burden, and heightened inflammatory and carcinogenic markers. Immunohistochemical analysis revealed upregulation of pSTAT3, β-catenin, and epithelial-mesenchymal transition (EMT) markers in F1-Suc mice. Immunophenotyping of colonic lymphocytes showed a significant reduction in CD4^{+}CD8^{+}IFNγ^{+} effector T cells and an increase in CD4^{+}CD25^{+}CTLA-4^{+} regulatory T cells, suggesting a compromised antitumor immune response. Collectively, these findings indicate that maternal sucralose consumption during the perinatal period disrupts gut immune homeostasis and enhances susceptibility to CRC in offspring. These results underscore the need for further investigation into the long-term health impacts of NCAS exposure during critical developmental windows.

Funding Information: UNAM-PAPIIT IN201724.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

ATG16L1 is a negative regulator of the IL-6-Muc4 axis during enteric inflammation

Mendiola, Daniela [1]; González, Marisol I [1]; Eksteen, Bertus [2];
Vannan, Danielle [3]; Quintero-Hernández, Jorge [1];
Macedo Hernández, Mariano I [1]; Mateos, Diego [1]; García Huerta,
Eduardo [1]; Pantoja-Gonzáles, Raúl [1]; Reyes, José L [1].

[1]Laboratorio de Inmunología Experimental y Regulación de la Inflamación Hepato-Intestinal, UBIMED, FES Iztacala, UNAM, Tlalnepantla de Baz, Estado de México, México. [2]Aspen Woods Clinic, Calgary, AB, Canada. [3]Boston Scientific Corporation, Urology Division, 200 Boston Scientific Way, Marlborough, MA, USA.

E-mail: dani15.mendiola@gmail.com

ATG16L1 is a central protein involved in autophagy, a cellular process essential for survival and repair under homeostatic conditions. However, mutations in this protein are associated with inflammatory bowel diseases (IBD) such as Crohn's disease and ulcerative colitis (UC). However, the specific molecular markers altered in the intestinal inflammatory microenvironment by ATG16L1 deficiency are not characterized. Herein, we employed the DSS-induced colitis mouse model to explore the role of ATG16L1 during chemically-induced colitis and the underlying mechanisms. We observed that hypomorphic ATG16L1 (ATG16L1HM) mice are highly susceptible to colitis induction as compared to wild type (WT) littermates. ATG16L1HM mice presented enhanced bodyweight loss and colon damage, especially in the caecum. Interestingly, the levels of interleukin (IL)-6 were increased in blood samples and lymphoid organs from ATG16L1HM mice. Evidence suggests that IL-6, an inflammatory cytokine, can modulate mucin 4 expression via the STAT3 signaling pathway. Thus, we also sought to determine whether ATG16L1 could be regulating the expression of mucin 4, through IL-6. To address this, total mucins were assessed by alcian blue staining, muc 2 and muc 4 gene expression in caecum as well as in proximal and distal colonic tissue was quantified by RT-PCR. Our data suggest that the ATG16L1 protein participates in the regulation of mucin 4 via the IL6-STAT3 pathway, playing an important role in the progression of colitis.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Immunization with plasmids that codify SARS-CoV-2 fusion proteins induced an immune response in a preclinical model

Mendoza-Ramírez, Noe Juvenal [1]; García -Cordero, Julio [1];
Cedillo-Barrón, Leticia [1].

[1]Departamento de Biomedicina Molecular, CINVESTAV.

E-mail: n.juvenalmendoza@gmail.com

Acid nucleic vaccines have been explored against different pathogens for many years. For COVID-19, many RNA vaccines have been developed, but only a few DNA vaccines have been proposed. Preclinical studies have shown that the combination of spike and nucleocapsid protein of SARS-CoV-2 induced a robust immune response. In this work, we evaluated the immune response induced by DNA vaccination with plasmids that encode for fusion proteins of SARS-CoV-2. We determined the most immunogenic regions of Spike (S) and Nucleocapsid (N) protein of the SARS-CoV-2 Delta and Omicron B.5 strain. Then, the sequences were cloned in pcDNA3.1 and named pcDNA3.1/D-S1, pcDNA3.1/D-S1N, and pcDNA3.1/O-SN. The expression of the constructs was observed in vitro. Then, BALB /c mice were immunized with 25µg of the above plasmids. Control mice were immunized with the same dose of the plasmids pcDNA3.1/S1, pcDNA3.1/N, and the parental pcDNA3.1. Three doses at 20-day intervals were administered. After immunization, bleedings were performed, serum samples were obtained, and the humoral response was evaluated. We found specific IgM, IgG, IgG1, and IgG2 antibodies against S1 and N protein. Neutralization activity was determined, and the highest levels were in mice immunized with pcDNA3.1/D-S1N. Furthermore, splenocytes were obtained from immunized mice, and the cellular T-cell response was analyzed by flow cytometry. The production of IFN-γ was detected in CD4+ and CD8+ cells, and the highest levels were observed on pcDNA3.1/O-SN immunized mice. Proliferation of CD3+ cells was observed. Finally, SARS-CoV-2-infected cells were evaluated by immunofluorescence with sera from immunized mice. A serum cross-reaction was observed with different VOCs. Additionally, we observed that sera from immunized mice were capable of recognizing SARS-CoV-2 in infected cells. Our results indicate that DNA vaccination with plasmids may be an excellent alternative to induce specific immune responses, with cross-reaction with VOCs, and the recognition of SARS-CoV-2.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Inhibition of WNT/βcatenin in melanoma mouse model. Strategy to reverse immunotherapy resistance

Mendoza -Roldán, Fátima [1 3]; Gajón, Julián A. [2 3];
Fuentes Panana, Ezequiel M. [4]; Bonifaz, Laura C. [3 5].

[1]Facultad de Química, Universidad Nacional Autónoma de México, Ciudad de México, México. [2]Posgrado en Ciencias Bioquímicas, Universidad Nacional Autónoma de México, Ciudad de México, México. [3]Unidad de Investigación Médica en Inmunoquímica, Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México. [4]Unidad de Investigación en Virología y Cáncer, Hospital Infantil de México Federico Gómez, Ciudad de México, México. [5]Coordinación de Investigación en Salud, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México.

E-mail: mroldan.fatima@gmail.com

Melanoma is the deadliest skin cancer with highly mortality worldwide, being the exposure to UV-radiation the main risk factor that transform melanocytes. Currently, Immune Checkpoint Blockade Immunotherapy (ICB) is an efficient therapeutic strategy that has improve patient survival; however, 40% of patients are non-responsive to ICB. It´s been described that the lack of response is associated mainly with exhaustion phenotype on tumor infiltrating lymphocytes as well as tumoral intrinsic characteristics. Direct evidence from our research group showed that ICB resistant cell lines overexpress stemness associated markers such as OCT4, ALDH1A1 and NANOG. Moreover, these cells overexpress WNT3a, a canonical ligand of WNT/ βcatenin signaling pathway which regulates the acquisition of stemness, indicating that this pathway is responsible for ICB resistance. Therefore, we proposed a strategy based on WNT/βcatenin inhibition to revert resistance to ICB. An in vitro inhibition of resistant cell lines treated with XAV-939, demonstrated the decrease of stemness phenotype. Also, the in vivo inhibition in mice, improved immunotherapy responsiveness, suggesting the reversion of ICB resistance. These findings allow us to propose a possible combinatory therapy, by inhibiting the WNT/ βcatenin pathway and ICB, to improve ICB response in melanoma.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Impact of MIF Inhibition by CPSI-1306 in Colitis-Associated Colorectal Cancer

Mendoza-Sánchez, Dánae Berenice [1]; Rodríguez-Hernández, Mario Alberto Tonathiu [1]; Juárez-Avelar, Imelda [1]; Rodríguez-Sosa, Miriam [1].

[1]Unidad de Biomedicina (UBIMED), Facultad de Estudios Superiores Iztacala (FES-Iztacala), Universidad Nacional Autónoma de México (UNAM). Tlalnepantla, Estado de México 54090, México.

E-mail: mendoza.sa.danae@gmail.com

Colorectal cancer (CRC) is the third most commonly diagnosed malignancy and the second leading cause of cancer-related mortality worldwide. Chronic colitis is a recognized risk factor for CRC development. Macrophage migration inhibitory factor (MIF) is a key proinflammatory cytokine implicated in colorectal tumorigenesis. During the early stages, MIF contributes to a chronic inflammatory microenvironment that promotes tumor initiation; in advanced stages, it facilitates angiogenesis, apoptosis resistance, invasion, and metastasis. In this study, we investigated the effects of MIF inhibition using the small-molecule inhibitor CPSI-1306, both in vitro, in human CRC cell lines with varying degrees of malignancy, and in vivo, in a murine model of colitis-associated colorectal cancer (CAC) induced by azoxymethane and dextran sodium sulfate (AOM/DSS). In vitro, low doses of CPSI-1306 reduced the viability of transformed CRC cell lines without affecting total MIF production, suggesting potential functional interference with MIF activity not detectable through conventional quantification. In vivo, treatment with CPSI-1306 from day 40 to day 68 post-CAC induction improved survival, mitigated body weight loss, rectal bleeding, and histological inflammation, and preserved colon length. No significant differences were observed in tumor burden or in the proportions of infiltrating CD4$^+$ and CD8$^+$ T cells, NK cells, macrophages, or neutrophils. Collectively, these findings suggest that MIF inhibition via CPSI-1306 decreases tumor cell viability in vitro and ameliorates clinical manifestations of CAC in vivo, although without significantly altering tumor burden or immune cell infiltration. Further studies are warranted to elucidate the precise mechanisms by which CPSI-1306 modulates CAC progression and its potential as a therapeutic agent.

This work was supported by PAPIIT project number IV200425.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Study of mast cells during COVID-19 and its fibrotic lung sequelae

Meneses-Preza, Yatsiri Guadalupe [1]; Martínez-Martínez, Ricardo Emmanuel [1]; Becerril-Villanueva, Enrique [2]; Muñoz-Cruz, Samira [3]; Hernández-Pando, Rogelio [4]; Maravillas-Montero, José Luis [5]; Wong-Baeza, Isabel [1]; Pérez-Tapia, Sonia Mayra [1 6]; Chávez-Blanco, Alma [7]; Chacón-Salinas, Rommel [1].

[1]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, ENCB-IPN. México City, México. [2]Laboratorio de Psicoinmunología, Instituto Nacional de Psiquiatría Ramón de la Fuente. México City 14370, México. [3]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social. México City, México. [4]Sección de Patología Experimental, Departamento de Patología, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán. México City 14080, México. [5]Departamento de Medicina Molecular y Bioprocesos, Instituto de Biotecnología, Universidad Autónoma De México, Cuernavaca, Morelos, México. [6]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, ENCB-IPN. México city, México. [7]División de Ciencia Básica, Instituto Nacional de Cancerología (INCan, SS), México city, México.

E-mail: yatsiri.meneses@gmail.com

Severe cases of COVID-19 often result in long-term pulmonary complications, including fibrosis. While excessive inflammation has been recognized as a key driver of tissue damage, cellular mechanisms linking acute infection to chronic remodeling remain insufficiently defined. Mast cells (MC) have recently emerged as contributors to inflammatory and fibrotic processes in various diseases. In this study, we investigated whether MC activation plays a role in COVID-19 severity and post-infection fibrotic sequelae. We analyzed serum samples from COVID-19 patients quantifying levels of MC-derived mediators: tryptase, chymase, carboxypeptidase A3 (CPA3), and PGD2. These measurements were used to describe a serum activation profile of MC, which was found to be significantly altered in infected individuals, especially in those with severe disease, and correlated with laboratory markers of inflammation and coagulopathy. To further explore tissue-level involvement, we examined lung samples from deceased COVID-19 patients. Histological analyses revealed increased numbers of MC expressing CPA3, particularly in pneumonic, fibrotic and thrombotic areas. The extent of CPA3+ MC infiltration correlated positively with collagen deposition, suggesting a link between MC activity and fibrotic remodeling. To investigate the potential role of MC proteases in this process, we used LUVA cells—a human MC line—and induced their degranulation to obtain MC-preformed mediators. These were applied to A549 human alveolar epithelial cells to evaluate their effects. MC-derived mediators promoted cell proliferation without compromising viability, suggesting that MC mediators may influence epithelial behavior. While this response could support regeneration in early stages, persistent signaling in an inflammatory environment may favor aberrant tissue remodeling and fibrosis. Together, our findings indicate that MC activation is a characteristic feature of severe COVID-19 and is closely associated with pulmonary fibrosis. This study supports a model in which MCs participate in both inflammatory amplification and dysfunctional repair, highlighting their potential as therapeutic targets in post-COVID lung disease.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Vacunas**

AI-biotechnology convergence and the emergence of a biodigital regime in vaccines: a sectoral innovation proposal

Meza-de la Rosa, José Luis [1]; Cimoli, Mario [1]; Stezano-Pérez,
Federico Andrés [1]; Flores-Encarnación, Marcos [2]; Corona-
Alcántar, Juan Manuel [1].

[1]Posgrado Integrado en Economía, Gestión y Políticas de Innovación. Universidad Autónoma Metropolitana. Ciudad de México, México. [2]Laboratorio de Microbiología Molecular y Celular, Facultad de Medicina, Benemérita Universidad, Autónoma de Puebla. Puebla, México.

E-mail: joseluis.mezad11@gmail.com

TRABAJO
NO
PRESENTADO

This study aims to analyze how the technological convergence between artificial intelligence (AI) and modern biotechnology is transforming technological trajectories and organizational models in the development of new vaccines, shaping a new biodigital innovation regime. An exploratory methodology was employed, grounded in evolutionary economics and the sectoral systems of innovation approach. A bibliometric analysis (2010–2024) was conducted using VOSviewer and Dimensions.ai to identify scientific publications and patents integrating AI (machine learning, predictive modeling) and biotechnology (mRNA, structural antigen design). This was complemented by the study of three convergent platforms—DeepVax, Gritstone Bio, and Moderna—to characterize their technological architectures. A significant acceleration in hybrid scientific-technological production has been observed since 2020, particularly in areas such as rational antigen design, epitope prediction, and immunogenicity simulation. Biodigital platforms have enabled shorter preclinical development timelines, optimized vaccine candidate design, and expanded immune coverage using AI tools trained on genomic and structural data. The case studies reveal a paradigm shift in organizational models: from sequential experimental processes to iterative cycles driven by computational simulation and automated learning. A new biodigital regime is emerging that redefines technological trajectories in vaccine development, characterized by integrated platforms, data science, and alliances between biotech firms, pharmaceutical companies, and AI enterprises. This transformation poses challenges for regulation, national capability management, and technological sovereignty in health. The study proposes advancing an industrial policy aimed at strengthening biodigital platforms applied to immunology as a strategic pathway to accelerate responses to future pandemic threats. Promoting biodigital platforms in vaccine development is not merely a technological choice, but a strategic decision. Anticipating this new innovation regime will enable countries to lead—rather than follow—the next revolution in precision immunology and global health.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Effects of diet on the Leptin-Anti-LEP antibody axis in Wistar rats

Meza -Peña, Dennis Alberto [1]; Reyes Castillo, Zyanya [2]; Padilla-Galindo, María Del Rocío [1]; Enciso-Ramírez, Mayra Alejandra [2]; Vázquez-Solorzano, Rafael [1]; Ochoa-Acosta, Alicia [3]; Terán Cabanillas, Eli [3].

[1]Instituto de Investigaciones en Comportamiento Alimentario y Nutrición (IICAN), Centro Universitario del Sur, Universidad de Guadalajara; Cd. Guzmán, Jalisco, México. [2]Laboratorio de Biomedicina y Biotecnología para la Salud, Centro Universitario del Sur, Universidad de Guadalajara; Cd. Guzmán, Jalisco, México. [3]Facultad de Ciencias de la Nutrición y Gastronomía, Universidad Autónoma de Sinaloa; Culiacán, Sinaloa, México.

E-mail: dennis.meza2607@alumnos.udg.mx

Anti-leptin antibodies (anti-LEP), whose serum levels and affinity have been associated with obesity and type 2 diabetes, may originate through molecular mimicry with intestinal microbiota proteins. However, the role of diet in modulating this immune response remains unclear. This study aimed to evaluate the effects of a high-fat diet and a high-fiber diet on the leptin/anti-LEP antibody axis in Wistar rats. Thirty-five adult rats were assigned to one of three dietary groups: standard (SD), high-fiber (HFiD), and high-fat (HFD). Food and water intake were recorded during four weeks (ad libitum). Blood samples were collected at baseline and at the end of the intervention to measure leptin levels and anti-LEP IgA antibodies (free, total, and immune complex fractions) using ELISA. The most relevant findings were as follows: male rats in the DAF group exhibited significantly higher levels of free IgA antibodies compared to the DAG group ($p = 0.04$), and similarly, female rats in the DAF group had higher levels of these antibodies than those in DAG ($p = 0.02$). No significant differences were found in total antibody fractions based on sex or diet. IgA immune complex levels were significantly lower in the DAF group compared to DAG ($p = 0.03$). Within-group analysis revealed a reduction in total antibody levels at baseline compared to final measurements in DE ($p = 0.03$) and DAG ($p = 0.009$), and lower levels of immune complexes at baseline than at the end of the study in DE ($p = 0.03$) and DAG ($p = 0.001$). These findings suggest that a high-fiber diet may reduce the proportion of anti-leptin immune complexes, potentially due to: a) alterations in gut microbiota, b) changes in leptin levels, or c) fiber-derived short-chain fatty acids affecting B cell maturation and antibody affinity and specificity.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Regulatory networks in neonatal T cells: vaginal, term and preterm cesarean delivery

Mijares-Guevara, Sebastian [1]; Kempis-Calanis, Linda Aimara [1];
Rodríguez-Jorge, Otoniel [1].

[1]Laboratorio de Inmunología Celular y de Sistemas, Centro de Investigación en Dinámica Celular, Instituto de Investigación en Ciencias Básicas y Aplicadas, Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México.

E-mail: sebastian.mijares@uaem.edu.mx

The neonatal period is one of the most vulnerable periods in human life, with 2.3 million deaths annually, representing 48% of all deaths in children under five. The neonatal immune system has been described as immature; however, it is now understood to be distinct from the adult immune system. Notably, neonatal T cells have been described as tolerant and exhibit a bias toward an innate-like response. This study aims to understand the regulatory networks underlying neonatal CD4+ and CD8+ T cells during labor at term, as well as during preterm delivery. Using a systems immunology approach, we integrate transcriptome data and computational tools to generate regulatory networks and logical models. A differential gene expression analysis (DGEA) was performed, revealing 354 differentially expressed genes in T cells (CD4+ and CD8+) between neonates delivered by cesarean section and those delivered vaginally. Also, a gene set enrichment analysis (GSEA) was performed, showing enriched pathways involved in the regulation of the immune response (cytokine production, effector function, leukocyte activation) in neonates delivered vaginally. DGEA also shows 359 overexpressed genes in CD8+ T cells when analyzing preterm and term neonates delivered by cesarean section, and 25 overexpressed genes were identified in CD4+ T cells under the same condition. Interestingly, the GSEA shows similar results in CD8+ and CD4+, indicating a proliferative phenotype in preterm neonates. Currently, we are working on the generation of PPI networks and logical models describing the immune response imposed by the type of delivery and gestational age. The above results show differences in the transcriptome of CD4+ and CD8+ T cells, influenced by the type of birth, indicating the presence of regulators in the phenotypes of the immune response.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Immunomodulatory and Antitumor Properties of the GK-1 Peptide: Possible Involvement of TLR4 Signaling.

Moctezuma Rocha, Diego; Hernández-Aceves, Juan; Rodríguez Tovar, Héctor; Sciutto-Conde, Edda; Fragoso González, Gladis del Carmen.

[1]Instituto de Investigaciones Biomédicas, Departamento de Inmunología, Coyoacán, Ciudad de México, México.

E-mail: diegodmr96@gmail.com

GK-1 is an 18-amino acid peptide originally identified in the recombinant KETc7 protein of Taenia crassiceps. During vaccine development against cysticercosis, GK-1 was found to induce protective immunity even without adjuvants, suggesting intrinsic immunostimulatory properties. Subsequent studies showed that GK-1 upregulates genes associated with TLR signaling in peritoneal macrophages from BALB/c mice. In bone marrow-derived dendritic cells (BMDCs), GK-1 enhanced the expression of CD80 and MHC-II and promoted the production of the inmunoinflammatory cytokines IFN-γ, TNF-α, and CCL2. These effects were linked to MyD88-dependent signaling and NF-κB nuclear translocation, indicating a probable TLR involvement. In silico molecular docking predicted that GK-1 interacts with the LPS-binding site of TLR4. Based on this, we investigated the role of TLR4 in mediating the immunomodulatory effects of GK-1, including its ability to activate antigen-presenting cells (APCs). Considering prior evidence of antitumor and antimetastatic effects of GK-1 in a 4T1 murine breast cancer model, we further assessed the contribution of TLR4 to GK-1 effects on tumor biology, focusing on oxidative stress and mitochondrial dysfunction, and on its antimetastatic properties. GK-1 activated dendritic cells from BALB/c and C57BL/6 mice by inducing IL-6 and CCL2 but had no effect on cells from TLR4-deficient C3H/HeJ mice, confirming the involvement of TLR4 in its immunostimulatory activity. GK-1 also significantly reduced mitochondrial membrane potential in TLR4-expressing 4T1 tumor cells, but not in TLR4-negative HEK-293 cells. Furthermore, a wound healing assay (mimicking cell invasiveness) showed that GK-1 impaired the migration of 4T1 cells, with no effect on HEK-293 cells. Overall, these findings suggest that the immunomodulatory, antitumor, and anti-metastatic effects of GK-1 may involve TLR4 signaling, indicating a possible role for this receptor in mediating GK-1's biological activity and supporting its potential as an immunotherapeutic candidate.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

NETS from neutrophils of patients with spondyloarthritis and their interaction with osteoblasts

Mojica-Villa, Lassha Ludmila [1]; Luna -Herrera, Julieta [2];
Romero-López, José Pablo [3]; Domínguez López, María Lilia [1];
García-Romo, Gina Stella [4].

[1]ENCB, IPN, Laboratorio de Inmunoquimica I, CDMX, México.. [2]ENCB, IPN, Laboratorio de Inmunoquimica II, CDMX, México . [3]UNAM, FESI, Laboratorio A4, EDOMEX, México.. [4]UNAM, FESI, Unidad de morfología y función, EDOMEX, México.

E-mail: lasshamojica98@gmail.com

Spondyloarthritis (SpA) comprises a group of heterogeneous diseases that share genetic, pathophysiological, and clinical features. Joint and synovial membrane inflammation leads to osteoproliferation and progressive immobility in patients. Neutrophils play a key role in the pathogenesis of various systemic diseases by capturing and eliminating microorganisms through neutrophil extracellular traps (NETs), which are web-like structures composed of DNA fibers, histones, and cytotoxic proteins. These cells are abundant in the synovial fluid and peripheral blood of patients with SpA.

General Objective: To evaluate spontaneous NETosis in neutrophils from patients with spondyloarthritis and determine the osteogenic potential of this mechanism. Hypothesis: Patients with spondyloarthritis show increased spontaneous NETosis, and the interaction between NETs and osteoblasts promotes osteoblast activity and bone matrix production. Peripheral blood samples were collected from patients and healthy controls. Neutrophils were isolated using a Polymorphoprep density gradient. A total of 500,000 cells were cultured in phenol red-free RPMI medium at 37°C and evaluated at 30, 60, and 120 minutes using fluorescence microscopy with DAPI and an anti-myeloperoxidase (MPO) antibody. Additionally, healthy neutrophils were stimulated with plasma from SpA patients. Co-cultures were performed between NETs and HFOB1.19 osteoblasts to assess proliferation and bone matrix formation using Alamar Blue and Alizarin Red staining.

Results showed an increase in spontaneous NETosis in SpA patients, and this response was further enhanced when neutrophils were stimulated with patient plasma, suggesting the presence of circulating proinflammatory factors. Furthermore, NET exposure promoted both osteoblast proliferation and bone mineralization. These findings suggest that neutrophil activity and NETosis may play an important role in the osteoproliferation observed in AD patients and could help elucidate the mechanisms underlying pathological bone formation, offering potential targets for future therapeutic strategies.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Alleviation of indomethacin-induced enteric damage in Wistar rat by kombucha supplementation

Molina-Afanador, Damaris E [1]; Varela-Mendoza, Atziri A [1];
Martínez -Flores, Ma. Magdalena [1]; Paz-Jiménez, Melanie G [1];
Córdova-Dávalos, Laura E [1]; Barrios-García, Tonatiuh [2];
Salinas-Guardado, Valeria [1]; Jiménez-Vargas, Mariela [1];
Salinas-Miralles, Eva [1]; Cervantes-García, Daniel [1 3].

[1]Department of Microbiology, Basic Science Center, Autonomous University of Aguascalientes, Aguascalientes, Mexico. [2]Department of Morphology, Basic Science Center, Autonomous University of Aguascalientes, Aguascalientes, Mexico. [3]Secretariat of Science, Humanities, Technologies and Innovation, Mexico City, Mexico.

E-mail: molina.damaris.b@gmail.com

Kombucha is a green tea fermented beverage rich in antioxidants and probiotics with relevant benefits on human health. Supplementation with antioxidants and probiotics has been explored as a strategy to reduce inflammation. Enteric damage due uncontrolled and unsupervised consumption of non-steroidal anti-inflammatory drugs (NSAID) represents a significant cause of morbidity and mortality among NSAID users. This study aimed to evaluate the preventive effects of kombucha against indomethacin-induced enteric damage. Enteropathy was induced for seven days with daily oral administration of indomethacin at a dose of 6 mg/kg. Kombucha was orally supplemented for three days prior to and until the seventh day of the induction of enteric damage. Hematological parameters, ulcer index and histological changes in the small intestine were determined. Gene markers for inflammation and intestinal barrier function were evaluated. Proportion of Firmicutes, Bacteroidetes, Actinobacteria and Proteobacteria were quantified by qPCR. Notably, kombucha supplementation reduced ulcer index and inflammation in small intestines, which prevented the drop in levels of hemoglobin, hematocrit and erythrocytes. In rats with indomethacin-induced enteric damage histopathological analyses showed impaired integrity of villi with inflammatory infiltrates, and this damage was prevented with kombucha supplementation. Interestingly, rats that received kombucha upregulated the expression of tight junction genes Muc2, Cldn1 and Ocln. The upregulation of the oxidative and inflammatory markers Nos2, Mpo and Tnf in damaged intestinal tissue was reduced by kombucha intake. Dysbiosis was markedly avoided by kombucha in indomethacin-induced enteric damage rats, since its supplementation attenuated the proliferation of Proteobacteria. Besides, kombucha treatment increased the Firmicutes abundance in cecal content. This study demonstrated that kombucha consumption may delay NSAID-induced enteric damage through enhancement of intestinal barrier integrity and prevention of dysbiosis.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Determination of antimicrobial peptides in marsh turtle microbiota for the analysis of its immunological resistance

Molina-Guillén, Gilberto [1]; Ramírez-Franco, Ulises [1];
Montero-Ruíz, Oreth [1]; Gallardo-Hernández, Carlos Arturo [2].

[1]Facultad de Bioanálisis, Universidad Veracruzana, Xalapa, Veracruz, México. [2]Unidad de Servicios Analíticos en Salud Bioanálisis, Facultad de Bioanálisis, Universidad Veracruzana, Xalapa, Veracruz, México.

E-mail: gilbertomolina1q2w3e4r@gmail.com

The Trachemys venusta (marsh turtle), have a high life expectancy, living from 25 to 40 years, so the immune system of these, especially their phagocytic cells, have proven to be very effective against pathogens such as bacteria and viruses that are present in the natural habitat in which they develop. Recent studies have indicated that antimicrobial peptides (AMPs) are highly conserved components of the innate immune system found among all classes of life and can be present in the environment and fluids of different species. These molecules exert an immunomodulator effect that could enhance the immune response. In this regard, we identify the microbiota present in the environment of Trachemys venusta as well as samples of saliva and stool. The results demonstrate a broad spectrum of gram-negative bacteria in the water where the marsh turtle lives. Similarly, we found the presence of AMPs through electrophoresis in samples of serum and saliva from marsh turtles. This evidence shows that AMPs from Trachemys venusta could participate in their immune response against bacteria present in their habitat and highlights the possibility of isolating them and proposing them as a treatment strategy for human infections.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

HPV-binding function of CD73 in cervical cancer cells

Monroy-García, Alberto [1,2]; Muñoz-Godínez, Ricardo [2];
García-Rocha, Rosario [2]; Weiss-Steider, Benny [2];
Hernández-Montes, Jorge [2]; Don-López, Christian Azucena [2];
Díaz-Ramos, Juan Antonio [2]; Molina-Castillo, Gabriela [2];
Mora-García, María de Lourdes [2].

[1]Laboratorio de Inmunología y Cáncer, Unidad de Investigación Médica en Enfermedades Oncológicas, Hospital de Oncología, CMN SXXI, Instituto Mexicano del Seguro Social, Ciudad de México 06720, México. [2]Laboratorio de Inmunobiología, Unidad de Investigación en Diferenciación Celular y Cáncer, Unidad Multidisciplinaria de Investigación Experimental Zaragoza (UMIEZ), Facultad de Estudios Superiores Zaragoza, Universidad Nacional Autónoma de México, Ciudad de México 09230, México.

E-mail: albertomon@yahoo.com

Persistent infection with high-risk human papillomavirus (HR-HPV) is the main factor associated in the development of cervical cancer (CC). Despite the high immunogenicity of HPV, CD73 expression on tumor cells plays a significant role in producing immunosuppressive adenosine (Ado), which inhibits the anti-tumor immune response. In previous studies, we observed that HPV-positive (HPV+) cervical cancer cells express higher levels of CD73 on their cell membrane than HPV-negative ones. Taking into account the additional role of CD73 as a binding protein, in this study we analyzed the capability of HPV+ and HPV- CC cell lines to bind HPV virus-like particles (VLPs). For this purpose, C33A (HPV-, CD73-); CaSki (HPV-16+, CD73+); SiHa (HPV-16+, CD73+); HeLa (HPV-18, CD73+); MS751(HPV-18, CD73+), and human keratinocyte cell line HaCaT (HPV-, CD73+), used as a positive control, were incubated for 24hrs in the presence or absence of the nonavalent Gardasil vaccine labeled with the fluorochrome Alexa fluor 488-A (VLPs-9v-AF-488A). Flow cytometry analysis revealed that the capacity of CC cells to bind VLPs-9v-AF-488A was strongly related to CD73 expression levels. The mean fluorescence intensity (MFI) values for CD73/VLPs-9v-AF-488A in HPV+ CC cells were: CaSki =128290/7238; Siha= 21467/20641; HeLa=24850/8660 and MS751=31615/20062. In contrast, in the HPV- C33A cell line, the MFI was 759/574; while in the control HaCat the MFI was113821/26490. Interestingly, reducing CD73 expression in CaSki and HeLa cells using siRNA for CD73, decreased their capability to bind VLPs-9v-AF-488A to MFI = 26164/1672 and MFI = 5015/4662, respectively. This results provide evidence that, in addition to enzymatic function of CD73, its presence on the membrane of epithelial cells could favor HPV binding and infection. Supported by the grant from SECIHTI to proyect CF-2023-I-141.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Activation markers in NKG2C⁺NKp46⁺ Memory-Like NK Cells Subset against Influenza A Nucleoprotein

Montes-Zapata, Edgar I. [1]; De la Cruz-Almazán, Larissa M. [1];
Arce-Mendoza, Alma Y. [1]; Rosas-Taraco, Adrián G. [1].

[1]Universidad Autónoma de Nuevo León, Servicio y Departamento de Inmunología, Facultad de Medicina, Monterrey, Nuevo León, México.

E-mail: edivanmon@gmail.com

Natural Killer (NK) cells are innate immune cytotoxic lymphocytes that have recently been recognized for their roles in adaptive-like memory responses, particularly during viral infections. The expression of NKG2C, an activating receptor associated with memory-like NK phenotypes, in combination with NKp46, a natural cytotoxicity receptor, has been linked to enhanced antiviral activity that can be induced by certain proteins. This study aimed to identify and characterize the double-positive NKG2C$^+$NKp46$^+$ NK cell population in response to stimulation with influenza A H1N1 nucleoprotein (NP). NK cells were isolated from peripheral blood mononuclear cells (PBMCs) of 10 healthy donors by negative magnetic selection and expanded using CTS NK Xpander medium supplemented with either IL-2 or a combination of IL-2 and IL-15. After a 14-day expansion, cells were stimulated with IAV H1N1 NP and cultured for an additional 6 days using complete RPMI medium, followed of phenotypic analysis by flow cytometry. NK cells were gated as CD3$^-$CD56$^+$, and double-positive NKG2C$^+$NKp46$^+$ populations were quantified and analyzed for expression of LAMP-1, granzyme B, and IFN-γ. We identified a distinct NKG2C$^+$NKp46$^+$ subset following NP stimulation that was more frequent in cultures expanded with IL-2/IL-15 ($P= 0.0001$) compared to IL-2 alone ($P= 0.0097$). This IL-2/IL-15 expanded subset exhibited elevated expression of LAMP-1 ($P= 0.0069$) granzyme B ($P= 0.0012$) and IFN-γ ($P <0.0001$), indicating effector functions consistent with a memory-like phenotype directed against NP. In conclusion these findings suggest that the NKG2C$^+$NKp46$^+$ NK cell subset preferentially expands under IL-2/IL-15 stimulation and displays enhanced functional activity upon NP exposure. This functionally distinct memory-like NK subset may hold importance for vaccine design and immunotherapeutic strategies targeting conserved viral proteins such as NP.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Metabolic gene dysregulation in NK cells from patients with severe COVID-19

Montes -Zapata, Edgar Iván [1]; Osuna-Espinoza, Kenia Yaretzy [2];
Mejía-Torres, Manuel Guadalupe [3]; Camacho-Ortiz, Adrián [4];
Pérez-Alba, Eduardo [5]; Salinas-Carmona, Mario César [6];
Rosas-Taraco, Adrián Geovanni [7].

[1]Departamento de Inmunología, Facultad de Medicina, Universidad Autónoma de Nuevo León. Monterrey, N.L. México. [2]Departamento de Inmunología, Facultad de Medicina, Universidad Autónoma de Nuevo León. Monterrey, N.L. México. [3]Departamento de Inmunología, Facultad de Medicina, Universidad Autónoma de Nuevo León. Monterrey, N.L. México. [4]Departamento de Infectología. Hospital Universitario "Dr. José Eleuterio González". Monterrey, N.L. México. [5]Departamento de Infectología. Hospital Universitario "Dr. José Eleuterio González". Monterrey, N.L. México. [6]Departamento de Inmunología, Facultad de Medicina, Universidad Autónoma de Nuevo León. Monterrey, N.L. México. [7]Departamento de Inmunología, Facultad de Medicina, Universidad Autónoma de Nuevo León. Monterrey, N.L. México.

E-mail: edivanmon@gmail.com

Natural killer (NK) cells are innate lymphocytes with cytotoxic activity against tumors and viruses. The pandemic of the coronavirus disease 2019 (COVID-19) has increased the investigation of their role in disease severity. However, their functional status and modulators remain controversial. Recent studies highlighted the role of metabolism in immune function, but metabolic changes in NK cells during SARS-CoV-2 infection remain unexplored. This study compares metabolic (SIRT1, AMPKA, HIF1A, and GLUT1) gene expression, and flow cytometry-based assessment of activation markers in NK cells from severe COVID-19 patients (n=15) and the control group (n=10), and their association with clinical outcomes. The expression of the activation marker IFNg and granzyme B was significantly higher in NK cells from severe COVID-19 patients compared with control group ($P<0.05$), and its expression was related to their clinical outcome. Also, the inhibitory receptor KIR2DL1 was significantly higher in severe COVID-19 patients compared with the control group ($P< 0.05$). The expression of HIF1A and GLUT1 was reduce in severe COVID-19 patients compared with the control group ($P<0.05$). SIRT1 expression correlated with granzyme B expression in NK cells from severe COVID-19 patients ($P< 0.05$, $R= -0.499$). SIRT1 expression was higher in patients requiring intubation ($P< 0.05$). SIRT1, HIF1A, and GLUT1 were upregulated in deceased patients ($P< 0.05$). In conclusion, we demonstrate that NK cells from patients with severe COVID-19 exhibit increased activation markers and dysregulated metabolic gene expression associated with clinical outcomes.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Development of a recombinant viral vector as non-replicative rotavirus VP6 vaccine platform

Montiel-Martínez, Ana Gabriela [1 7]; Martínez-Rodríguez, Claudia Susana [2]; Aguillón-Segura, Jazmín Patricia [2]; Barrios-Payán, Jorge Alberto [3]; Liñán-Torres, Arturo [4]; Pastor-Flores, Ana Ruth [4]; Palomares-Aguilera, Laura Alicia [4]; Moreno-Fierros, Leticia [5]; Gutiérrez-Xicoténcatl, María de Lourdes [6]; Torres-Vega, Miguel Ángel [2]; Esquivel-Guadarrama, Fernando Roger [7].

[1]Posgrado en Ciencias Biológicas, Universidad Nacional Autónoma de México, CDMX, México. [2]Departamento de Gastroenterología, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, CDMX, México. [3]Departamento de Patología, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, CDMX, México. [4]Departamento de Medicina Molecular y Bioprocesos, Instituto de Biotecnología, Universidad Nacional Autónoma de México, Morelos, México. [5]Laboratorio de inmunología de mucosas, Facultad de Estudios Superiores Iztacala, Universidad Nacional Autónoma de México, México City, México. [6]Centro de Investigaciones Sobre Enfermedades Infecciosas, INSPSSA, Cuernavaca, Morelos, México. [7]Laboratorio de Inmunología Viral, Facultad de Medicina, Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México.

E-mail: agaby.m.m@gmail.com

The potential for zoonosis due to the emergence of novel rotavirus variants, along with the limited efficacy of current vaccines in low-income countries, underscores the need for next-generation rotavirus vaccines. In this study, we developed a recombinant viral vector based on adeno-associated virus serotype 8 (AAV8) expressing VP6, a highly conserved antigen from the intermediate capsid of rotavirus, with the aim of evaluating its structural stability and functional expression as a non-replicative vaccine candidate. A recombinant baculovirus (Bac-VP6) was constructed and co-infected with Bac-Rep/Cap-2/8 into Sf9 insect cells to produce adeno-associated virus particles (AAV-VP6). These viral particles were purified using an iodixanol gradient and characterized by transmission electron microscopy (TEM). VP6 expression was evaluated in vitro by Western blot in HEK293T cells transduced with the vector. In addition, the vector was administered to BALB/c mice via intragastric or intramuscular routes, followed by an intranasal booster, and antibodies specific to VP6 and to the AAV capsid were measured. Purified viral particles exhibited the typical AAV morphology under TEM. VP6 expression was confirmed in vitro in HEK293T cells. In mice, a significant serum antibody response against the AAV capsid was observed following intramuscular immunization, suggesting in vivo functionality of the vector. The AAV8-VP6 vector is structurally and functionally stable. Its ability to express antigen in both human cells and mice supports its potential as a safe and versatile vaccine platform. This study lays the groundwork for the development of recombinant AAV-based vaccines aimed at inducing mucosal immunity against rotavirus through conserved antigens such as VP6. AG Montiel Martínez is a doctoral student in the Doctoral Program in Biological Sciences at the UNIVERSIDAD NACIONAL AUTÓNOMA DE MÉXICO (UNAM) and received a fellowship (631032) from SECIHTI.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Association of CVD phenotypes with clinical activity and metabolic endotoxemia in SLE

Mora-García, Paulina Esmeralda [1 2]; Campos-López, Bertha [1 2]; Parra-Rojas, Isela [1 3]; Pesqueda-Cendejas, Karen [1 2]; Luquin-De Anda, Sonia [2]; Montoya-Buelna, Margarita [1 4]; Cerpa-Cruz, Sergio [5]; De la Cruz-Mosso, Ulises [1 2].

[1]Red de Inmunonutrición Y Genómica Nutricional en Las Enfermedades Autoinmunes, Centro Universitario de Ciencias de La Salud, Universidad de Guadalajara, 44340 Guadalajara, Jalisco, México. [2]Instituto de Neurociencias Traslacionales, Departamento de Neurociencias, Centro Universitario de Ciencias de La Salud, Universidad de Guadalajara, 44340 Guadalajara, Jalisco, México. [3]Laboratorio de Investigación en Obesidad Y Diabetes, Facultad de Ciencias Químico-Biológicas, Universidad Autónoma de Guerrero, 39087 Chilpancingo de los Bravo, Guerrero, México. [4]Laboratorio de Inmunología, Departamento de Fisiología, Centro Universitario de Ciencias de La Salud, Universidad de Guadalajara, 44340 Guadalajara, Jalisco, México. [5]Departamento de Reumatología, O.P.D. Hospital Civil de Guadalajara Fray Antonio Alcalde, 44280 Guadalajara, Jalisco, México.

E-mail: moragarciapaulinaesmeralda@gmail.com

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by damage to multiple organs. Its main cause of death is cardiovascular disease, because various traditional and non-traditional CVD risk factors, such as serum and intake vitamin D deficiency, high levels of CRP, hyperuricemia, hypoalbuminemia, and dyslipidemia contribute to its severity and mortality. The aim of this study was to characterize and associate CVD risk phenotypes with clinical disease activity and the presence of metabolic endotoxemia in SLE patients. A cross-sectional study was conducted in 120 SLE women classified according to the SLE ACR-1997 criteria and 189 women as control subjects (CS). LPS levels were assessed using a sandwich ELISA, and CVD risk phenotypes were classified as low CVD risk if they presented <3 factors and high risk if they presented ≥3 CVD risk factors. When evaluating the CVD risk phenotypes in both study groups, we observed that patients had a frequency of the high CVD risk phenotype (45% vs. 26%; p=0.001) compared to CS. SLE patients with a high CVD risk phenotype had a higher percentage of renal activity (50% vs. 22%; p=0.004) and clinical disease activity (53% vs. 31%; p=0.02). Regarding the prescribed medications, 48% of patients with a high CVD risk phenotype were administered with prednisone, compared to 29% of patients with a low CVD. Notably, when evaluating serum LPS levels in SLE presented higher levels (86.18 vs. 65.66 pg/mL, p=0.001) compared to CS. According to the CVD risk phenotype, we observed differences in CVD risk group (87.72 vs. 58.22; p = 0.002). We observed that 43% of SLE patients presented metabolic endotoxemia compared to only 23% of SC. In conclusion, patients with SLE presented a higher frequency of high CVD risk phenotype, characterized by higher clinical activity, dyslipidemia, hypovitaminosis D, hypoalbuminemia, hyperuricemia, and endotoxemia compared to CS.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Differential IL-17R family expression in B lymphocytes: potential targets for rheumatoid arthritis immunotherapy

Morales-Núñez, José Javier [1]; Sifuentes -Franco, Sonia [1];
Hernández -Bello, Jorge [2]; Muñoz-Valle, José Francisco [2];
Coronado-González, Christian Fernando [3].

[1]Centro Universitario de los Valles - Universidad de Guadalajara, Departamento de Ciencias de la Salud, Ameca, Jalisco, México. [2]Centro Universitario de Ciencias de la Salud - Universidad de Guadalajara, Departamento de Biología Molecular y Genómica, Guadalajara, Jalisco, México. . [3]Centro Universitario de Ciencias de la Salud - Universidad de Guadalajara, Lic. Médico Cirujano y Partero, Guadalajara, Jalisco, México.

E-mail: javier.morales@academicos.udg.mx

B lymphocytes are central players in rheumatoid arthritis (RA) pathogenesis, not only through autoantibody production but also by interacting with cytokines, such as IL-17. The IL-17 cytokine family exerts its biological effects via five receptors (IL-17RA to IL-17RE), which vary in structure and function, and are involved in key aspects of B cell biology. Among these functions include survival, class-switch recombination, plasma cell differentiation and affinity maturation, however, the expression profile of IL-17 receptors on B cells in RA is poorly characterized. This study aimed to characterize the surface expression of the IL-17 receptor family on B lymphocytes and explore associations with clinical variables in RA patients, compared to control subjects. Peripheral blood mononuclear cells were isolated from 34 RA patients and 9 healthy controls. Flow cytometry was used to assess the expression of CD19 and IL-17 receptors (IL-17RA, IL-17RB, IL-17RC, IL-17RD, IL-17RE). Both the percentage of receptor-positive cells and mean fluorescence intensity (MFI) were analyzed. Clinical activity was evaluated using the Disease Activity Score-28 (DAS28), and statistical comparisons were performed using Mann-Whitney U and Kruskal-Wallis tests. RA patients exhibited a trend toward increased CD19+ B cell frequency ($p=0.0521$) but significantly reduced total CD19+ MFI ($p=0.0073$). IL-17RC+ B cells were significantly reduced ($p<0.0001$), while IL-17RD+ B cells were increased ($p=0.0058$). Although the frequency of IL-17RA+ B cells did not differ significantly ($p=0.2136$), their absolute number was reduced ($p=0.0013$). Stratification by DAS28 showed higher IL-17RD expression in patients with active disease ($p=0.0344$). These findings highlight distinct patterns of IL-17 receptor expression in RA B cells, supporting their potential as therapeutic targets in the disease.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Analysis of the effect of cannabidiol on the immune response in horses

Morelos-Cruz, Elba Rebeca [1]; Martínez-Cortés, Ismael [2];
Martínez-Casares, Rubria Marlen [3]; Chaparro-Godínez, Beatriz [4];
Campillo-Navarro, Marcia [5].

[1]Universidad Autónoma Metropolitana Unidad Xochimilco, Maestría en Ciencias Farmacéuticas, México City, México. [2]Universidad Autónoma Metropolitana, Unidad Xochimilco, Departamento de Producción Agrícola y Animal, Laboratorio de Farmacología y Toxicología molecular, México City, México. [3]Universidad Autónoma Metropolitana, Unidad Xochimilco, Departamento de Sistemas Biológicos, Laboratorio de Biocatálisis, México City, México. [4]Universidad Autónoma Metropolitana, Unidad Xochimilco, Departamento de Sistemas Biológicos, Laboratorio de Dolor, México City, México. [5]Instituto Nacional de Cardiología, Oficina de Apoyo Sistemático para la Investigación Superior, México City, México.

E-mail: elbarebemore@gmail.com

With the need to find new therapeutic alternatives for various pathological cases, different methods of extraction from natural products have been developed. Among them, compounds derived from Cannabis, known as cannabinoids, have gained relevance in various fields. In particular, cannabidiol (CBD) has been shown to possess anxiolytic, anti-inflammatory, anticonvulsant and neuroprotective properties. The health effects of Cannabis consumption remain poorly understood, especially in the case of products administered intranasally. Therefore, the present work aims to evaluate the immunomodulatory activity of CBD administered by this route, considering its affinity for the CB2 receptor -predominant in cells of the immune system- and the direct contact that can be established with the nasal mucosa. For this purpose, two experimental groups with eight horses each will be used: group A (control, with administration of placebo) and group B (administration of 1.2 mg/kg of CBD). Whole blood samples will be collected to establish the pharmacokinetics of CBD after intranasal administration. In addition, with the DNA microarray technique, the immunomodulatory activity will be evaluated from the expression of genes related to the inflammatory process.

Acknowledgments:

•To SECIHTI for the postgraduate grant awarded, CVU 2000738.

•Laboratorio de Farmacología y Toxicología molecular, DPAA., UAM-X.

•Laboratorio de Biocatálisis, DSB., UAM-X.

•Laboratorio de Dolor, DSB., UAM-X.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Immune and Inflammatory Profiling in Elite Taekwondo Athletes During the Preparation and Recovery Phases of the France 2024 Olympic Games

Moreno-Eutimio, Mario Adán [1]; Pérez-Flores, Dulce María [2];
]Bedolla-Santos, Jesús [2]; Pérez-Pacheco, Ana Laura [2];
Patiño-Castillo, Aldo Jason [2]; Aguilar-Martínez, Agustín Eduardo [2];
Castillo-Hernández, René [2]; Herrera-Abarca, Jorge Eduardo [3];
Pastelin-Palacios, Rodolfo [1].

[1]Facultad de Química, Universidad Nacional Autónoma de México (UNAM), México City, México. [2]Dirección de Medicina y Ciencias Aplicadas, Comisión Nacional de Cultura Física y Deporte (CONADE), México City, México. [3]Clínica de Enfermedades Crónicas y de Procedimientos Especiales, S.C.

E-mail: marioadan@quimica.unam.mx

High-performance sports demand exceptional physiological and psychological resilience, often accompanied by significant immunological stress. In this pilot study, we evaluated immune and inflammatory markers in elite Mexican taekwondo athletes during the preparation and recovery phases associated with the France 2024 Olympic Games. Peripheral blood and serum samples were collected at two key time points: during the pre-competition phase and during a follow-up recovery phase several weeks after the athletes' return. Multiparametric flow cytometry and ELISA-based analyses were used to assess immune cell phenotypes, immune checkpoint expression, cytokine profiles, and selected molecular markers of inflammation, exhaustion, and recovery. This study represents a first step toward understanding immunological fluctuations linked to Olympic-level training and competition in taekwondo athletes. The data generated will contribute to developing evidence-based tools for immune monitoring, performance optimization, and overtraining prevention in elite sports.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Use of Imipramine in a murine model of Brucella abortus 2308

Moreno-Lafont, Martha C [1]; Maldonado-García, José Luis [1 2];
Damián-Morales, Gabriela [1]; Maldonado-Tapia, Jesús Octavio [1];
López-Santiago, Rubén [1]; Pérez-Sánchez, Gilberto [2];
Becerril-Villanueva, Enrique [2]; Álvarez-Herrera, Samantha [2];
Pavón-Romero, Lenin [2].

[1]Laboratorio de Inmunología Celular, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, México. [2]Laboratorio de Psicoinmunología, Instituto Nacional de Psiquiatría Ramón de la Fuente, México.

E-mail: mlafont@ipn.mx

Brucellosis infection causes non-specific symptoms such as fever, chills, sweating, headaches, myalgia, arthralgia, anorexia, fatigue, and mood disorders. In mouse models, it has been associated with increased levels of IL-6, TNF-α, and IFN-γ, a decrease in serotonin and dopamine levels in hippocampus, loss of muscle strength and equilibrium, and increased anxiety and hopelessness. Imipramine (ImiP), a tricyclic antidepressant, is used to alleviate neuropathic pain. This study evaluated the effects of ImiP on Balb/c mice infected with Brucella abortus 2308 (Ba) at 14- and 28-days post-infection. Serum levels of IFN-γ, IL-6, TNF-α, IL-12, MCP-1. and IL-10 were assessed by FACS. Additionally, the number of CFU in the spleen was measured. Finally, Serotonin levels in the hippocampus were analyzed via HPLC. Behavioral tests were conducted to assess strength, equilibrium, and mood by forced swimming test (FST) and tail suspension test (TST). Results showed that mice infected with Brucella abortus 2308 and treated with ImiP for six days (Im6Ba14) had significantly different outcomes compared to infected mice (Ba14) at day 14 post-infection. Improvement was observed in FST ($p < 0.01$), TST ($p < 0.0001$), and open-field test ($p < 0.0001$). Additionally, there was an increase in serotonin levels in the hippocampus ($p < 0.001$). Furthermore, there was an improvement in equilibrium ($p < 0.0001$) and muscle strength ($p < 0.01$). Lastly, there was a decrease in IL-6 levels ($p < 0.05$) and CFU count in the spleen ($p < 0.0001$). These findings suggest the potential for ImiP to be used as an adjuvant treatment for the symptoms of brucellosis. In addition, the results suggest that imipramine has immunomodulatory effects that should be investigated in future studies.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Metabolic Cross-Talk: Platelets as Dynamic Regulators of Neutrophil Immunometabolism

Mosso-Pani, Manuel Alejandro [1]; Barreda-, Dante [2];
Salazar-, Ma. Isabel [3].

[1]ODIN Bioscience, Miami, Florida, UU.EE.. [2]Aging and Metabolism Research Program, The Oklahoma Medical Research Foundation, Oklahoma City, OK, United States.. [3]Laboratorio Nacional de Vacunología y Virus Tropicales (LNVyVT), Escuela Nacional de Ciencias Biológicas Instituto Politécnico Nacional, Ciudad de México, México.

E-mail: qbp.moss@hotmail.com

Platelets are increasingly recognized as immune modulators far beyond their traditional roles in hemostasis and thrombosis. Among their most dynamic immunological functions is their ability to influence neutrophil behavior at multiple levels. Here, we highlighted cutting-edge insights into how platelets and platelet-derived signals—particularly metabolites—reshape neutrophil immunometabolism, impacting both innate immunity and inflammation.

We explored how platelet-neutrophil aggregates, extracellular vesicles, and soluble mediators orchestrate key neutrophil functions including recruitment, activation, endothelial adhesion, and the delicate balance between resolution and amplification of inflammation. A central focus was on how platelets drive metabolic reprogramming in neutrophils, shifting glycolytic and mitochondrial activity in a context-dependent manner. Moreover, we discussed how these platelet-neutrophil interactions reverberate through the immune system by modulating the activity of T cells, B cells, and the broader immune microenvironment.

By dissecting this underappreciated yet powerful axis of immune regulation, we aim to uncover novel therapeutic strategies to target neutrophil-driven pathology in inflammatory and immune-mediated diseases.

TRABAJO
NO
PRESENTADO

Área del artículo: **Inmunología veterinaria y evolutiva**

Effect of polyherbal supplementation on intestinal microbiota and immune response in horses.

Muñoz Cano-Villalpando, Raquel María [1]; Martínez-Cortés, Ismael [2]; De La Torre-Hernández, María Eugenia [3]; Martínez-García, José Antonio [4]; Campillo-Navarro, Marcia [5]; Solís-Jaquez, Roberto [6]; Carrillo-Barajas, Alberto [7]; Luna-García, Sergio [8].

[1]Universidad Autónoma Metropolitana Unidad Xochimilco, Departamento de Producción Agrícola y Animal, CDMX, México. [2]Universidad Autónoma Metropolitana Unidad Xochimilco, Departamento de Producción Agrícola y Animal, CDMX, México. [3]Investigadora por México SECIHTI-UAM Xochimilco, Sistemas biológicos, Departamento de Producción Agrícola y Animal, CDMX, México. [4]Universidad Autónoma Metropolitana Unidad Xochimilco, Departamento de Producción Agrícola y Animal, CDMX, México. [5]Oficina de Apoyo Sistemático para la Investigación Superior, Instituto Nacional de Cardiología Ignacio Chávez, CDMX, México. [6]Universidad Nacional Autónoma de México, Facultad de Medicina Veterinaria y Zootecnia, Departamento de Medicina, Cirugía y Zootecnia para Équidos, CDMX, México. [7]Pelotón de Veterinaria, Remonta y Forrajes del Grupo Montado y de Honores de la Comandancia del Ejército Mexicano, CDMX, México. [8]Grupo Montado y de Honores de la Comandancia del Ejército Mexicano, CDMX, México.

E-mail: 2242800882@alumnos.xoc.uam.mx

Herbal supplements have gained importance due to their pharmacological properties on the body, mainly due to their content of secondary metabolites with antioxidant, anti-inflammatory or immunomodulatory effects. The intestinal microbiota plays an important role in equine nutrition and health. Furthermore, supplementation with polyherbal products such as BioCholine® is known to influence intestinal microbiota homeostasis and gene expression in various metabolic processes, significantly promoting health and performance in animal production. The objective of this study is to evaluate the effect of BioCholine® and Organomin® supplementation on fecal microbiota and immune response gene expression in horses. For this purpose, 20 Santa Gertrudis breed horses weighing 500 ± 50 kg will be used, distributed into three groups: group A (control, base diet without supplements), group B (base diet plus 4 g/day of BioCholine®), and group C (base diet plus 5 g/day of Organomin®). Fecal samples will be collected to analyze microbial diversity (alpha and beta), including Lactobacillus, Faecalibacterium and Clostridiales, by 16S rRNA gene sequencing, and the expression of immune response genes related to production of IL-10, TNF-α, IL-6 and IL-1β will be determined using DNA microarray techniques.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

PD-L1 expression in cervical cancer tissue is strongly associated with the expression of CD73/TGF-β the percentage of CD8+/PD-1+ T cells and disease progression

Muñoz -Godínez, Ricardo [1]; Monroy -García, Alberto [1 2];
Hernández-Cueto, Ángeles María [3]; García-Rocha, Rosario [1];
Weiss Steider, Benny [1]; Hernández-Montes, Jorge [1]; Don-López, Christian Azucena [1]; Mora-García, María de Lourdes [1].

[1]Laboratorio de Inmunobiología, Unidad de Investigación en Diferenciación Celular y Cáncer, Unidad Multidisciplinaria de Investigación Experimental Zaragoza (UMIEZ), Facultad de Estudios Superiores Zaragoza, Universidad Nacional Autónoma de México, Ciudad de México 09230, México. [2]Laboratorio de Inmunología y Cáncer, Unidad de Investigación Médica en Enfermedades Oncológicas, Hospital de Oncología, CMN SXXI, Instituto Mexicano del Seguro Social, Ciudad de México 06720, México. [3]Banco de Tejidos, División de Laboratorios Especializados, CCILE, Instituto Mexicano del Seguro Social, Ciudad de México 02990, México.

E-mail: biolric1406@gmail.com

To investigate the associations between the expression of programmed cell death ligand-1 (PD-L1) with that od CD73 and TGF-β and the percentage of CD8+ T lymphocytes expressing programmed cell death protein 1 (PD-1) in cervical tissue samples from normal donors (NDs), patients with cervical intraepithelial neoplasia (CIN-I, CIN-II, CIN-III) and patients with cervical cancer (CC). This was a retrospective observational study of patients and control individuals. Patients with histopathological diagnoses of CIN1 (n = 27), CIN2 (n = 25), CIN3 (n = 23) and CC (n = 23), all of whom were free of treatment, underwent biopsies. ND tissue samples (n = 30) were also analyzed as controls. Tissue microarrays (TMAs) were generated in triplicate for each sample. TMAs were stained with anti-PD-L1 (E1L3N), anti-CD73 (NBP1-85740), anti-TGF-β1 (DY240), anti-PD-1 (EH33) and anti-CD8 (D8A8Y) monoclonal antibodies. Digital images were taken, and the area percentage of positive staining of each marker was calculated with the V9 algorithm via Aperio ImageScope software. We found that the expression of PD-L1, CD73 and TGF-β1, as well as the percentage of CD8+/PD-1+ T lymphocytes, increased in cervical tissues and were correlated with disease progression. The expression of PD-L1 was positively correlated with the expression of CD73 ($r = 0.8274$, $p < 0.001$) and with that of TGF-β 1 ($r = 0.8535$, $p < 0.001$). Similarly, the percentage of CD8+ cells was positively correlated with the expression of the marker PD-1 ($r = 0.7199$, $p < 0.01$). The expression of PD-L1 and PD-1 in CC tissues is strongly correlated with the expression of CD73 and TGF-β1, so the joint analysis of these biomarkers can be very useful for evaluating the prognosis of this disease and for establishing strategies that improve the efficacy of immunotherapy protocols in CC patients treated with ICIs. Supported by DGAPA-PAPIIT Nos. IN211822/IN216325 and IMSS/R-2023-3602-011, grants.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Relationship of anti-dsDNA autoantibodies and TLR9 haplotypes with risk in SLE patients

Muñoz-Valdivia, Josué Jacobo [1,2]; Campos-López, Bertha [1,2]; Meza-Meza, Mónica R. [1,2]; Ruiz-Ballesteros, Adolfo I. [1]; Pesqueda-Cendejas, Karen [1,2]; Luquin, Sonia [1,2]; Parra-Rojas, Isela [1,3]; Mora-García, Paulina E. [1,2]; De la Cruz-Mosso, Ulises [1,2].

[1]Red de Inmunonutrición y Genómica Nutricional en las Enfermedades Autoinmunes; Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud; Universidad de Guadalajara; Guadalajara, Jalisco 44340, México. [2]Instituto de Neurociencias Traslacionales, Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara 44340, Jalisco, México. [3]Laboratorio de Investigación en Obesidad y Diabetes; Facultad de Ciencias Químico-Biológicas; Universidad Autónoma de Guerrero; Chilpancingo de los Bravo 39087, Guerrero, México.

E-mail: josue.j.m.valdivia@gmail.com

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by anti-double stranded DNA (anti-dsDNA) autoantibodies production, which contribute to inflammation and organ damage. These antibodies form immune complexes recognized by intracellular receptors such as Toll-like receptor 9 (TLR9), triggering proinflammatory cytokine release. Single nucleotide variants (SNVs) in the TLR9 gene, such as rs352139 and rs352140, have been proposed as modulators of disease susceptibility in different populations. However, their role in the Mexican-mestizo population remains underexplored. This study aimed to determine the association of anti-dsDNA positivity and haplotypes conformed by rs352139 and rs352139 SNVs in TLR9 for the risk to SLE and its clinical variables in Mexican-Mestizo population. Was conducted in 253 SLE patients and 248 control subjects (CS), all females. To assess anti-dsDNA autoantibodies, indirect immunofluorescence was used with the haemoflagellate Crithidia luciliae method, and allelic discrimination for TLR9 SNVs was performed with real time PCR. Among SLE patients, 40.6% were positive for anti-dsDNA. Anti-dsDNA-positive patients had higher LDL-C levels (79.17 vs. 70.45 mg/dL; p=0.01) and lower albumin (3.84 vs. 3.94 g/dL; p=0.04). Also, hemoglobin (12.8 vs. 13.65 g/dL; p=<0.01) hematocrit (39.4% vs. 40.96%; p=0.04), lymphocytes (1.26 vs. 2.05 $\times 10^3/\mu L$; p=<0.001) monocytes (0.046 vs. 0.08 $\times 10^3/\mu L$; p=0.03), and basophils (0.05 vs. 0.08 $\times 10^3/\mu L$; p=<0.01) were significantly reduced. Both TLR9 SNVs were in Hardy-Weinberg equilibrium and were in high linkage disequilibrium (D’=0.8). Haplotype analysis showed that CT haplotype was a genetic risk factor for SLE (SLE: 45% vs. CS: 37%; OR=1.35; 95%CI: 1.05-1.73; p=0.02), while the CC haplotype was associated with no risk (SLE: 3% vs. CS: 8%; OR=0.39; 95%CI: 0.021-0.70; p=0.001). In conclusion, positivity to anti-dsDNA was related to biochemical and hematological alterations; notably, the CT haplotype of TLR9 was associated with genetic risk to SLE in Mexican-mestizo population.

ISSN: 2007-3380

Revista Bio Ciencias

https://revistabiociencias.uan.edu.mx

Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Association of C-reactive protein levels and CRP haplotypes with rheumatoid arthritis risk

Muro-Dávalos, Paulina F. [1 2]; López-Campos, Bertha [1 2];
Pesqueda-Cendejas, Karen [1 2]; Mora-García, Paulina E. [1 2];
Meza-Meza, Mónica R. [1 2]; Rivera-Escoto, Melissa [1]; Ruiz-Ballesteros, Adolfo I. [1]; De la Cruz-Mosso, Ulises [1 2].

[1]Red de Inmunonutrición y Genómica Nutricional en las Enfermedades Autoinmunes; Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud; Universidad de Guadalajara; Guadalajara, Jalisco 44340, México. [2]Instituto de Neurociencias Traslacionales, Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara 44340, Jalisco, México.

E-mail: paulina.muro9768@alumnos.udg.mx

Rheumatoid arthritis (RA) is an autoimmune disease associated with systemic inflammation and elevated cardiometabolic risk. In Mexican-Mestizo women, its prevalence and clinical variability pose a major health challenge. High C-reactive protein (CRP) serum levels could be associated with disease activity and may drive cardiovascular complications. However, genetic factors such as single nucleotide variants (SNVs) influencing CRP expression in RA remain insufficiently explored. Therefore, this study aimed to determinate the association of the −717 A>G, −409 G>A, +1444 C>T, and +1846 C>T CRP SNVs and CRP serum levels as potential biomarkers of risk in RA. this cross-sectional study enrolled 432 unrelated women: 216 with RA according to the 2010 ACR-EULAR RA criteria and 216 control subjects (CS). The clinical disease activity was assessed by the DAS28-CRP score; high-sensitivity CRP serum levels were quantified by turbidimetry. The CRP SNVs genotyping was carried out by allelic discrimination. Serum CRP levels were significantly higher in RA patients than controls (4.98 vs. 1.19 mg/dL; p<0.001), with 9.2-fold increased risk to AR (OR=9.2; CI=5.1-16.6; p<0.001). All CRP variants were in Hardy-Weinberg equilibrium. The CC +1444 C>T CRP genotype was more frequent among RA patients compared to controls (47% vs. 35%; p=0.035) and was associated with 1.7-fold genetic risk to RA (OR=1.7; CI=1.1–2.6; p<0.01). Moreover, CRP SNVs showed strong linkage disequilibrium (D’>0.8) and four haplotypes were identified; notably, AGCT CRP haplotype was associated with RA genetic risk (OR=1.34; CI=1.01-1.8; p=0.04). In conclusion, these findings indicate that in the RA Mexican-Mestizo population, high CRP serum levels, the CC genotype of the +1444 CRP SNV and the AGCT CRP haplotype were associated with RA risk.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Repositioned Drugs Treatment for Suppressing Dengue Infection and Cytokine Production in Macrophages

Niño -Herrera, Sujey Abigail [1]; González-Christen, Judith [1];
Montiel-Hernández, José Luis [1].

[1]Universidad Autónoma del Estado de Morelos, Facultad de Farmacia, Cuernavaca Morelos, México.

E-mail: sujey.nino@uaem.edu.mx

Dengue disease can exhibit a wide range of clinical manifestations, with some patients developing life-threating complications. Although the reasons why certain patients progress to severe dengue remain unclear, the dysregulated immune response is associated with disease pathogenesis due to high inflammatory cytokine production. Currently, approved treatment for this illness is not available. It is known that neuraminidases (Neu) participate in cellular transduction by TLR to produce inflammatory cytokines, and oseltamivir (OSV) has shown activity to inhibit this enzyme. On the other hand, some studies have reported that IVM inhibits Dengue replication in different cell lines; however, the only clinical study did not show a clinical improvement with this treatment. In this study, the combination of ivermectin (IVM) and OSV was evaluated in Dengue infected macrophages to analyze their effect on inflammatory cytokine production and Dengue replication. The inhibitory effect of OSV over Neu was determined following a fluorescence assay using a neuraminidase substrate and the LPS stimulation of macrophages. To determine IVM activity over Dengue replication on macrophages, an RT-PCR assay was done evaluating different concentrations of IVM alone or in combination with OSV. Also, the combined effect of IVM and OSV was evaluated by RT-PCR on IL-6 and TNF expression during dengue infection. We found that OSV can inhibit neuraminidase activity at 1 μM, and the combination of IVM and OSV can reduce Dengue expression by 40 % at 0.05 μM of IVM with 1 μM of OSV. This study suggests that a combined therapy to reduce dengue replication and modulate immune response could be a promising treatment strategy for this illness.

SANH is a fellowship from SECIHYT.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Immunomodulatory Effects of Purified and Encrypted Axolotl Peptides

Ocampo-Hernández, Melissa Citlali [1]; Ruelas-Galindo, Isabel [1];
Fleitas-Martínez, Osmel [2]; Jacinto-Castillo, Diana [3];
Rosenstein-Azoulay, Yvonne Jane [1]; Gurrola-Briones, Georgina [1];
Rebollar-Caudillo, Eria [3]; Bustamante-Santillán, Víctor Humberto [2];
Auvynet, Constance [1].

[1]Instituto de Biotecnología, Departamento de Medicina Molecular y Bioprocesos, Cuernavaca, Morelos, México. [2]Instituto de Biotecnología, Departamento de Microbiología Molecular, Cuernavaca, Morelos, México. [3]Centro de Ciencias Genómicas, Departamento de Microbiología Genómica, Cuernavaca, Morelos, México.

E-mail: melissa.ocampo@ibt.unam.mx

Among the new therapeutic strategies developed to combat antibiotic-resistant infections are host defense peptides (HDPs). These components of the eukaryotic innate immune system are characterized by their short length (12 to 50 amino acids), cationic and amphipathic nature, and high content of hydrophobic residues (approximately 50%), with most adopting an alpha-helical structure. Because most of the HDPs induce microbial death by interacting with membranes through electrostatic and hydrophobic interactions, leading to membrane disruption, they have been shown to generate less bacterial resistance compared to conventional antibiotics. In addition to their antimicrobial activity, HDPs often exhibit other biological functions, such as antitumor effects and modulation of the immune response. These include promoting chemotaxis, phagocytosis, apoptosis, cell proliferation, and the production of cytokines, chemokines, and reactive oxygen species. This combination of immunomodulatory and direct antimicrobial effects makes them promising candidates for infection treatment. HDPs can be produced either through classical ribosomal synthesis or via proteolytic processing. The latter has attracted growing interest due to the discovery of novel antimicrobial peptides known as cryptic host defense peptides. With the aid of computational tools, algorithms can be applied to scan the proteome for these cryptic peptides, based on HDP characteristics such as sequence length, net charge, average hydrophobicity, and other physicochemical properties, all integrated into a fitness function. Although amphibians represent the largest group of organisms with identified and characterized HDPs showing antimicrobial and immunomodulatory activities, very little is currently known about the host defense peptides of the Mexican axolotl (Ambystoma mexicanum). To identify new therapeutic candidates, peptides were either isolated from skin exudate by purification or identified from the whole proteome. Those showing antimicrobial activity were further characterized for immunomodulatory properties—such as chemotaxis, leukocyte viability, and proliferation—using classical assays including migration and MTT assays.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

BCG as an inducer of trained immunity in bone marrow-derived mast cells

Ogonaga-Borja, Ingrid Michelle; Ruiz-Sánchez, Bibiana Patricia [1];
Meneses-Preza, Yatsiri Guadalupe [1]; Chacón-Salinas, Rommel [1].

[1]Instituto Politécnico Nacional, Escuela Nacional de Ciencias Biológicas. Departamento de Inmunología. Ciudad de México. México.

E-mail: ogonagaingrid@gmail.com

Since 2011, the term trained immunity was coined to refer to the ability of innate myeloid cells to acquire immunological memory. Trained immunity explains how these cells can emit robust or diminished responses after restimulation with certain agents. This process has been evidenced mainly after the application of bacille Calmette and Guerin (BCG) vaccine, which is traditionally applied against systemic tuberculosis. However, research has shown that BCG stimulation of monocytes, macrophages, neutrophils, dendritic cells, natural killer cells and hematopoietic precursors generates differentiated responses when these cell groups face a second challenge. Interestingly, studying immunity trained from hematopoietic precursors would lead to the analysis of possible persistent phenotypic and functional changes in the different myeloid lineages and thus, their participation in various processes of the immune response. For example, the study of mast cells (MC) would be key to a more comprehensive understanding of their role in allergic and inflammatory processes. In this sense, in the following proposal, we intend to evaluate whether BCG is able to induce immune training in bone marrow-derived mast cells (BMMC). For this purpose, we will work with C57BL/6 mice in which the BCG inoculum will be applied intravenously (i.v.). Four weeks later, it will be determined whether BCG i.v. is able to reach the bone marrow of the murine model. Subsequently, bone marrow cells from tibia and femur will be obtained and derived to mast cells to verify if there are phenotypic differences between BMMCs exposed or not to BCG. Finally, in primary BMMC cultures, different secondary stimuli will be applied and the function of BMMCs will be analyzed through techniques that allow determining their degranulation processes and cytokine production.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Immune expression of CD38 as an important biomarker of diagnosis, prognosis, and monitoring of MASLD

Ojeda -Vázquez, Emanuel [1]; Palma-Jacinto, José Antonio [2]; Gallardo-Hernández, Carlos Arturo [3]; González-Herrera, Sandra Luz [4]; Pérez-Lara, Jocelyn Carolina [5].

[1]Facultad de Bioanálisis, Universidad Veracruzana, Xalapa, Veracruz, México. [2]Unidad de Servicios Analíticos en Salud Bioanálisis, Facultad de Bioanálisis, Universidad Veracruzana, Xalapa, Veracruz, México. [3]Unidad de Servicios Analíticos en Salud Bioanálisis, Facultad de Bioanálisis, Universidad Veracruzana, Xalapa, Veracruz, México. [4]Facultad de Bioanálisis, Universidad Veracruzana, Xalapa, Veracruz, México. [5]Centro de Investigación y Estudios Avanzados del Instituto Politécnico Nacional, Departamento de Biomedicina Molecular, Ciudad de México, Ciudad de México, México.

E-mail: emanuelov45@gmail.com

Metabolic dysfunction-associated steatotic liver disease (MASLD) is currently one of causes of liver-related morbidity, with an estimated global incidence of 30% in the adult population and in Mexico, its prevalence may reach 47%. The presence of type 2 diabetes mellitus, obesity, insulin resistance, and other cardiometabolic risk factors is closely linked to the development of MASLD. This affectation included a clinical spectrum ranging from simple steatosis to metabolic dysfunction-associated steatohepatitis (MASH), advanced fibrosis, cirrhosis, and hepatocellular carcinoma. One of the emerging molecular mechanisms that has gained relevance in the progression of this disease is the role of the NADase CD38. This protein is an ectoenzyme that regulate the NAD$^+$ metabolism. Similarly, CD38 is highly expressed in immune cells like a receptor and the crosslinking triggers multiple cellular mechanisms, including signaling pathways that induce cell activation. In this way, CD38 appears to be involved in the activation of hepatic stellate cells contributing to liver fibrogenesis and inflammatory state. The objective of this study was to evaluate the expression by flow cytometry of the CD38 protein from peripheral blood immune cells from patients diagnosed with MASLD and determine the correlation between CD38 expression and the different stages of disease. Interestingly, we observed a higher expression of CD38 on immune cells and a direct relationship between CD38 expression with respect to the severity of MASLD. These results demonstrate that CD38 play an important role in physiopathology of MASLD and could be as a non-invasive biomarker for diagnosis, prognosis, and monitoring of the disease in the general population.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

TLR4 expression in metabolically activated macrophages from patients with dyslipidemia.

Olalde-Rodríguez, Farid; Ramírez -Segovia, Silvia; Mendoza-Pérez, Alejandra; Monsivais-Urenda, Adriana Elizabeth.

[1]Centro de Investigación en Ciencias de la Salud y Biomedicina (CICSAB), UASLP. [2]Centro de Investigación en Ciencias de la Salud y Biomedicina (CICSAB), UASLP. [3]Centro de Investigación en Ciencias de la Salud y Biomedicina (CICSAB), UASLP. [4]Centro de Investigación en Ciencias de la Salud y Biomedicina (CICSAB), UASLP.

E-mail: faridolald@gmail.com

Metabolically activated macrophages (MMe) are immune cells that acquire a distinct proinflammatory phenotype upon in vitro stimulation with glucose, insulin, and palmitate. These cells are frequently overrepresented in the adipose tissue of individuals with dyslipidemia, a condition associated with chronic low-grade inflammation that may contribute to the development of trained immunity, a phenomenon in which Toll-like receptors (TLRs) play a central role. Specifically, palmitate and oxidized low-density lipoprotein (oxLDL) have been shown to potentiate the activation of various TLR4-mediated signaling pathways, and TLR4 itself has been implicated in the induction of trained immunity. Although TLR4 expression in MMe has been previously reported, its quantitative expression profile and association with lipid parameters in these cells remain poorly characterized. The aim of this study was to evaluate the expression of TLR4 in MMe derived from circulating monocytes isolated from individuals with and without dyslipidemia. Peripheral blood samples were collected from dyslipidemic patients and normolipidemic controls. Peripheral blood mononuclear cells (PBMCs) were isolated, differentiated into macrophages, and subsequently polarized to the MMe phenotype through metabolic stimulation. TLR4 expression levels were then assessed by flow cytometry. The data revealed no significant positive correlation between TLR4 expression and plasma levels of LDL cholesterol or triglycerides. Interestingly, a positive correlation was observed between elevated TLR4 expression and high-density lipoprotein (HDL) cholesterol levels. These findings suggest that increased HDL levels may be associated with upregulation of TLR4 expression in MMe. Nevertheless, further studies with a larger sample size are warranted to validate these preliminary observations.

Área del artículo: **Biología de linfocitos T**

Inhibins are required for T-Cell migration and homing to peripheral lymphoid organs

Olguín-Alor, Roxana [1 2]; Ortega-Francisco, Sandra [1]; Bolaños-Castro, Lizbeth [1]; De la Fuente-Granada, Marisol [1]; Soldevila-Melgarejo, Gloria [1 2].

[1]Department of Immunology, Biomedical Research Institute, UNAM. [2]National Laboratory of Flow Cytometry, Biomedical Research Institute, UNAM.

E-mail: rolguinalor@iibiomedicas.unam.mx

Inhibins and their co-receptor, the TGF-β type III receptor (TβRIII), have been implicated in T cell development within the thymus and in the induction of peripheral tolerance via modulation of dendritic cell (DC) maturation and regulatory T cell generation. Inhibins also modulate $CD4^+$ T cell differentiation by restraining Th17 polarization. Our group has previously demonstrated that inhibins modulate DC migration, since $Inh\alpha^{-/-}$ DCs display reduced in vitro chemotaxis to CCL19 and CCL21 and impaired homing to inflamed tissues, however, their role in mature T cell migration remains unknown. Here, we show that Inhibin A is produced by activated naive $CD4^+$ T cells, as early as 3 hours after stimulation, regardless of activation strength. Notably, $CD4^+$ T cells from $Inh\alpha^{-/-}$ mice exhibited impaired chemotactic responses to CCR7 ligands CCL19 and CCL21 in vitro, and reduced homing to peripheral lymph nodes (PLNs) in vivo, as shown by competitive adoptive transfer assays. This defect was specific to $CD4^+$ T cells, as $CD8^+$ T and B cell migration remained unaffected. Analysis of lymphoid organs in $Inh\alpha^{-/-}$ mice revealed an accumulation of naïve $CD4^+$ T cells in PLNs, but not in mesenteric lymph nodes (MLNs) or spleen (SP). Interestingly, CCR7 expression was increased in $CD4^+$ T cells within PLNs, but decreased in MLNs and SP, suggesting altered tissue-specific migration dynamics. Altogether, our findings uncover a previously unrecognized autocrine role for Inhibins in regulating $CD4^+$ T cell trafficking to secondary lymphoid tissues. Further investigation is needed to elucidate the molecular mechanisms underlying Inhibin-mediated modulation of T cell migration.

Funding: This work was supported by PAPIIT-DGAPA UNAM projects IN213319 and IN220124. Acknowledgments: We thank Mauricio Ortiz (National Laboratory of Flow Cytometry), Omar Collazo (National Laboratory of Genomic Resources) at the Biomedical Research Institute, UNAM, and Oscar Hernández for technical assistance in mice genotyping.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Expanding the Landscape of Ca^{2+} signaling in NK Cells: Evidence of Unexplored Ion Channels with Clinical Relevance

Olivas-Aguirre, Miguel Ángel [1]; Cruz-Aguilar, Laura Hadit [1];
Ramirez-Oseguera, Laura Cecilia [1]; Pottosin, Igor [1];
Dobrovinskaya-, Oxana [1].

[1] Laboratory of Cancer Pathophysiology, University Center for Biomedical Research, University of Colima, Colima 28040, Mexico. [2]Laboratory of Immunobiology and Ionic Transport Regulation, University Center for Biomedical Research, University of Colima, Colima 28045, Mexico. [3]Secretaria de Ciencia, Humanidades, Tecnología e Innovación (Secihti), Programa de Investigadores e Investigadoras por México, México City 03940, Mexico.

E-mail: miguel.a.olivas@gmail.com

NK cells are key effectors of the innate immune response with potent antitumor abilities. Primary mechanism of cancer cell killing by NK is via the release of cytotoxic granules in a Ca^{2+}-dependent fashion. Data on patients with defective mutant CRAC channels evidenced that reduced store-operated Ca^{2+} entry (SOCE) strongly suppressed the degranulation and target cell killing. However, NK cells express a broader repertoire of ion channels, with potential roles in Ca2+ signaling. These include calcium-permeable ones and auxiliary channels, such as potassium ones, whose activity can shape the Ca2+ signal. As a starting point, we investigated the role of the unique Ca^{2+}-activated K+ channel KCa3.1 in modulation of SOCE and cytotoxic activity of NK cells. Using the selective KCa3.1 blocker NS6180, we found that only a small (about 20%) fraction of resting NK cells exhibited significant KCa3.1 currents, which is significantly increased upon the activation with IL-2 and IL-15. Pharmacological block of KCa3.1 reduced SOCE and intracellular calcium elevation, triggered by cytokines or contact with target (Jurkat T-ALL) leukemic cells. Interestingly, while early degranulation was reduced, a prolonged coculture with Jurkat cells revealed an enhanced global NK-mediated cytotoxicity and increased apoptotic/necrotic cell death. In this case, the abolishment of KCa3.1 activity, which potentiated SOCE, optimized the time course of cytolytic granules release and supported a serial killing. Our ongoing research aims to characterize the expression, function and subcellular localization of additional Ca^{2+} entry channels, which can participate in Ca2+ signaling in alternative/additive to SOCE receptor-mediated modes, and Ca^{2+} regulators, such as mitochondria, to better understand how ion channel diversity contributes to the regulation of NK cell-mediated immunity against cancer. Understanding this ion channel landscape may open new avenues for modulating NK cell activity and improve NK-based immunotherapeutic strategies.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Detección de linfocitos T CD8 WT-1 e IE-1 específicos en pacientes con Cáncer de mama previo a la mastectomía.

Olvera-Gómez, Irlanda [1 6]; Pérez-Álvarez, María Paulina [1];
Pérez-Pérez, Carlos Manuel [2]; Narváez-Villanueva, Juan Manuel [2];
Pérez-Lara, Jocelyn [3]; Charis-Trujillo, Paul Ernesto [4]; Sosa-Durán,
Erik [4]; Ortiz-Navarrete, Vianney [3].

[1]Hospital Juárez de México, División de Investigación, Ciudad de México, México. [2]Hospital Juárez de México, Cirugía Oncológica, Ciudad de México, México. [3]CINVESTAV, Biomedicina Molecular, Ciudad de México, México. [4]Hospital Juárez de México, Oncología, Ciudad de México, México. [5]Universidad Anáhuac, Facultad de Ciencias de la Salud, Ciudad de México, México.

E-mail: irlandaolverag@gmail.com

In Mexico, breast cancer affects not only women in their 60s, but also younger women. The diagnosis is frequently in the highest stages: III and IV. Eventually, neoadjuvant chemotherapy (NC) is administered for six months. This will eliminate or shrink the tumor (and then make a surgery). NC will induce tumor cell death and the release of tumor antigens. These would be processed by antigen-presenting cells in an inflammatory environment and activate new clones of T cells. We are interested in knowing the frequency of WT-1 and IE-1-specific T cells in these patients and analyzing the correlation with the clinical response to neoadjuvant chemotherapy. To address this, we stimulate peripheral blood mononuclear cells (PBMC) from patients with a mixture of WT-1 (tumor antigen) or IE-1 (CMV protein) peptides, and by flow cytometry, we analyzed IFN- production of IFN-γ by T cells.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

"Computational insights into the mechanisms of haptenization of HSA-SMX"

Ordoñez-Rodriguez, Alma Mireya [1]; Castrejón-Flores, José Luis [1];
Gómez-Chávez, Fernando [2]; Orozco-Valencia, Ángel Ulises [3];
Gómez -Castro, Carlos Zepactonal [4].

[1]Instituto Politécnico Nacional, Unidad Profesional Interdisciplinaria de Biotecnología, Laboratorio de Inmunofarmacología, Ciudad de México, México. [2]Instituto Politécnico Nacional, Escuela Nacional de Medicina y Homeopatía (ENMyH), Laboratorio de Enfermedades Osteoarticulares e Inmunológicas, Ciudad de México, México. [3]Universidad de Sonora, Departamento de Investigación en Polímeros y Materiales, Hermosillo, Sonora, México. [4]Universidad Autónoma del Estado de Hidalgo, Pachuca, Hidalgo, México.

E-mail: alma.ordonez.rod@gmail.com

Drug-induced hypersensitivity reactions represent a significant clinical challenge and are often attributed to the formation of hapten complexes, where reactive drug metabolites covalently bind to endogenous proteins and trigger immune responses. Sulfamethoxazole (SMX), a widely used sulfonamide antibiotic, is metabolized to its nitroso derivative (SMXno), which is capable of haptenizing human serum albumin (HSA) through lysine residues, leading to documented hypersensitivity reactions in patients. While experimental techniques such as mass spectrometry have identified specific lysine residues as targets for SMXno adduct formation, detailed mechanistic and structural insights remain limited, partly due to challenges in capturing transient reaction intermediates and the dynamic nature of protein-drug interactions. To address these challenges, this work employs a comprehensive computational strategy integrating molecular docking, conceptual density functional theory (CDFT) calculations, intrinsic reaction coordinate (IRC) analyses, and hybrid quantum-mechanics/molecular-mechanics (QM/MM) molecular dynamics (MD) simulations. Our results pinpoint Lys199 and Lys195—assisted by Glu292 within HSA's binding site I as the most likely sites for haptenization, corroborating prior mass spectrometry data. Reaction pathway analyses reveal that SMXno undergoes nucleophilic attack by the lysine ε-amino group, leading to arylazoalkane adduct formation. Thermodynamic and kinetic evaluations demonstrate a strong preference for the trans adduct configuration over the cis form. Beyond mechanistic insights, this study establishes a systematic computational workflow for predicting hapten formation between drug metabolites and proteins. The methodology is generalizable to other drug-protein systems, enabling early identification of immunogenic risks during drug development. The present findings not only resolve issues regarding site specificity and stereochemical outcomes in SMXno-HSA haptenization but also provide a theoretical framework for understanding drug-induced hypersensitivity. Ultimately, this work supports the rational design of safer therapeutics and contributes to the broader goal of precision medicine by reducing adverse drug reactions through predictive computational modeling.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Nickel-Cobalt Nanocrystals and Their Anti-Inflammatory Potential

Orozco -Alvarez, Jazmin [1]; Varela-Rodríguez, Luis [1];
Sánchez-Ramírez, Blanca E [1].

[1]Universidad Auntonoma de Chihuahua, Facultad de Ciencias Quimicas, Chihuahua, Chihuahua, Mexico .

E-mail: p330046@uach.mx

TRABAJO NO PRESENTADO

Inflammation is a key response of the immune system to infections or tissue damage, aimed at containing harm and restoring homeostasis. However, chronic or dysregulated activation can lead to inflammatory and autoimmune diseases. In this process, inflammasomes play a central role. They detect danger signals of microbial (PAMP) or cellular (DAMP) origin and, once activated, promote the maturation of pro-inflammatory cytokines such as IL-1β and IL-18 through the activation of caspase-1, in addition to inducing pyroptosis, an inflammatory form of cell death. The best-known inflammasome is NLRP3, notable for its ability to respond to a wide variety of stimuli, from bacterial toxins to cellular dysfunctions, linking it to diseases such as type II diabetes, Alzheimer's, inflammatory bowel disease, and gout. Given its involvement in multiple disorders, precise regulation of its activation is critical.

Recent advances have identified an innovative pathway for inflammatory modulation using nanomaterials. Although many nanoparticles have traditionally been associated with inflammasome activation, it has been discovered that Ni-Co alloy nanocrystals (NCs) can exert an inhibitory effect. They act by interfering with inflammasome assembly through the suppression of the long non-coding RNA Neat1, thereby reducing the formation of the ASC inflammasome complex. This inhibitory effect was confirmed in murine models of colitis and peritonitis, where Ni-Co NCs significantly reduced inflammation. These findings suggest that certain nanomaterials not only can prevent inflammasome activation but also offer a promising therapeutic pathway for chronic inflammatory diseases, combining immune specificity with targeted delivery technologies.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

Trim33 regulates Th9 Lymphocyte Differentiation and In Vivo Function.

Orozco-Córdoba, Javier; Contreras-Castillo, Eugenio [1];
Muñoz-Paleta, Ofelia [1]; Zambrano-Romero, Daniel [1];
Ríos Ramírez, Diana [1]; Ramos-Balderas, José Luis [1];
Licona-Limón, Paula [1].

[1]Instituto de Fisiología Celular, Universidad Nacional Autónoma de México, México City, México.

E-mail: orozcocordobajavier@gmail.com

The TGF-β signaling pathway has been extensively studied in CD4+ T cells, particularly in TH17, Treg, and, more recently, in TH9 cells, characterized by high IL-9 production and its dependency on TGF-β and IL-4 for their generation. The canonical TGF-β pathway is regulated by Smad2/3 and Smad4; however, non-canonical TGF-β pathways in TH9 remain unknown. In this work, we focused on the alternative TGF-β pathway regulated by Trim33, an E3 Ubiquitin ligase and double epigenetic reader, which has remained underexplored in immune populations. By using a conditional knockout mouse model for Trim33 (CD4-Cre) in the IL-9, IL-4, and FOXP3 reporter background, we demonstrated that Trim33 plays a crucial role in cell commitment to the TH9 lineage by inhibiting IL-9 expression and TH9 differentiation in vitro. In vivo, during helminth infection with Nippostrongylus brasiliensis, Trim33 deficiency increases the generation of TH9 and TH9-like Treg cells, a poorly described population characterized by the concomitant expression of IL-9 and FOXP3, which together promote an enhanced antiparasitic response in Trim33-deficient mice, driven by increased numbers of ILC and mast cells. Furthermore, in a B16-OVA melanoma model, we found an enhanced antitumor response in cKO mice, marked by increased TH9 cells within the tumor and draining lymph nodes. Likewise, the generation of TH9-like Tregs and possible transitional populations towards this new population were characterized through bioinformatic assays. Mechanistically, Trim33 did not regulate the TGF-β or IL-1β pathways but instead modulated IL-4 signaling, as Trim33-deficient cells exhibited heightened IL-4 sensitivity and higher expression of STAT6 and IRF4 during differentiation. Collectively, our data indicate that Trim33 is a crucial molecule in the differentiation and function of TH9 lymphocytes, highlighting its potential as a therapeutic target in diseases where TH9 cells play a central role.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Decoding CMTM6 in Cervical Cancer: Subcellular Distribution and PD-L1 Binding Insights

Orozco-Jacobo, Ana Delia [1]; Delgadillo-Gutiérrez, Alan [1];
Carranza-Aranda, Ahtziri Socorro [2]; Viera-Segura, Oliver [3];
Rojas-Díaz, José Manuel [1]; Galván-Ledezma, Fernando [1];
Tellez-Bañuelos, Martha Cecilia [4]; Bueno-Topete, Miriam Ruth [1];
Haramati, Jesse [4]; Del Toro-Arreola, Susana [1 5].

[1]Instituto en Investigación en Enfermedades Crónico-Degenerativas, Departamento de Biología Molecular y Genómica, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara, Jalisco, México. [2]Departamento de Disciplinas Filosóficas Metodológicas e Instrumentales, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara, Jalisco, México. [3]Instituto de Investigación en Ciencias Biomédicas, Departamento de Biología Molecular y Genómica, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara, Jalisco, México. [4]Laboratorio de Inmunología Traslacional, Departamento de Biología Celular y Molecular, Centro Universitario de Ciencias Biológicas y Agropecuarias, Universidad de Guadalajara, Zapopan, Jalisco, México. [5]Instituto de Inmunología, Departamento de Fisiología, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara.

E-mail: ana.orozco4051@alumnos.udg.mx

Cervical cancer (CC) remains the second leading cause of cancer-related death among Mexican women. Chronic pro-inflammatory stimuli upregulate PD-L1 expression on cancer cells, inhibiting the effector functions of T lymphocytes and NK cells. The surface stability of PD-L1 is regulated by CMTM6, a transmembrane protein that prevents its lysosomal degradation, thereby extending its functional lifespan. Recent studies have shown that HSC70, a constitutively expressed intracellular chaperone that promotes PD-L1 degradation, competes with CMTM6 for PD-L1 binding. Additionally, CMTM6 has been detected in extracellular vesicles and soluble form, raising the question: does CMTM6 have a broader distribution that may reflect additional, yet uncharacterized, immunomodulatory functions beyond membrane stabilization? To answer this, we explored the cellular distribution of CMTM6 in cervical cancer cell lines and predicted its interaction site with PD-L1. We determined CMTM6 location in SiHa and HeLa cell lines by immunofluorescence. To elucidate the CMTM6-PD-L1 interaction site, an in silico approach was employed. AlphaFold3 models were refined using GalaxyWEB server and validated by Molprobity and SAVESv6.1. Molecular docking was performed using the HDOCK program. HSC70 was used as a positive control of interaction. Our main results revealed that CMTM6 is localized not only in the cytoplasm and plasma membrane but also in the nucleus, suggesting a potential nuclear function. Docking results revealed a conserved PD-L1 binding site at amino acid position 228. Our analysis also confirmed that HSC70 interacts with PD-L1 within the 171-265 amino acid region, consistent with previous reports and supporting competitive binding with CMTM6. Molecular dynamics simulations are in progress to validate these interactions and assess their conformational stability over time. In conclusion, the nuclear distribution of CMTM6 supports the notion of novel non-canonical roles. Docking models suggest non-previously described interaction regions. Further studies are required for a better understanding of the significance of CMTM6 in CC.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Modulation of Erythrocyte Oxidative Stress by Paenibacillus sp. Extracts at Different pH

Orozco-Ruiz, Eleazar [1]; Ramírez-Villalobos, Jesica María [2];
Quiñones-Flores, Celia María [1]; Castillo-González, Alva Rocío [1];
García, Andrés [3]; Gómez-Flores, Ricardo [2]; Núñez-Parra, Samantha Patricia [1]; Vásquez-de la Cruz, Jesús Geovani;
Romo-Sáenz, César Iván [1].

[1]Universidad Autónoma de Chihuahua, Facultad de Medicina y Ciencias Biomédicas, Chihuahua, Chihuahua, México. [2]Universidad Autónoma de Nuevo León, Facultad de Ciencias Biológicas, Departamento de Microbiología e Inmunología, San Nicolás de los Garza, Nuevo León, México. [3]Universidad Autónoma del Estado de Morelos, Centro de Investigación en Biotecnología, Cuernavaca, Morelos, México.

E-mail: a361342@uach.mx

Probiotics with antioxidant capacity are promising for immunomodulation and cellular protection against oxidative stress. In this study, the effect of methanolic extracts from a Paenibacillus sp. isolate, known for its antitumor activity, was evaluated on the neutralization of experimentally induced erythrocyte oxidative stress under different pH conditions (6, 7, and 8). Hemolysis assays induced by cold, heat, and hypotonicity were conducted, along with anti-hemolytic assays against H_2O_2 to assess cellular protection. ABTS, FRAP, and DPPH assays were used to measure the neutralization of reactive oxygen species (ROS). Results showed that the extracts did not cause significant hemolysis and exhibited protective effects against oxidative hemolysis, especially at pH 6. Antioxidant capacity measured by ABTS was highest at pH 6.

These findings suggest that methanolic extracts of Paenibacillus sp. favorably modulate erythrocyte oxidative stress in acidic conditions, likely by regulating membrane damage caused by lipid peroxidation. This supports their potential as immunoprotective and probiotic agents in diseases associated with chronic inflammation, autoimmune disorders, and cancer, where oxidative stress plays a key role in pathophysiology.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Area del artículo: **Inmunidad tumoral**

Inhibition of autophagic flux as strategy to reduce the Raji cells proliferation

Ortega-Díaz, Gloria Michel [1]; García Pérez, Blanca Estela [1];
Baltierra-Uribe, Shantal Lizbeth [1].

[1]Instituto Politécnico Nacional, Departamento de Microbiología, Laboratorio de Microbiología General, ENCB, CDMX, México.

E-mail: gloriaortegadiaz@gmail.com

Burkitt lymphoma (BL) is a highly aggressive B-cell malignancy characterized by an extremely high proliferation rate. In recent years, autophagy has gained attention for its role in tumor cell survival by promoting intracellular recycling, particularly in stressful microenvironments. This study aimed to evaluate the effects of autophagic flux modulation on Raji cells, a model derived from BL. In this study chloroquine and rapamycin were used as inhibitor and inductor of autophagic flux respectively. Metabolic activity was measured using Alamar Blue, cell proliferation was assessed via Ki-67 (APC), cell death by Annexin V/PI staining, and the expression of autophagy-related proteins (Beclin-1, LC3-I/II, and p62) was analyzed by Western blot. The inhibition of autophagic flux by chloroquine caused a reduction of metabolic activity and proliferation along with a progressive increase in cell death with a concentration-dependent effect. This effect correlated with the accumulation of proteins associated with the initiation of the autophagy process (Beclin-1) and with the blockage of autophagic flux (p62, LC3-II), suggesting a collapse of functional autophagy. In contrast the induction of autophagy with rapamycin did not significantly alter cell viability and proliferation of B cells. In conclusion, these findings suggest that disrupting autophagic flux rather than stimulating autophagy may offer a promising immunobiological approach to reduce the proliferation of neoplastic B cells in Burkitt lymphoma, potentially contributing to the development of novel therapeutic strategies.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Vacunas**

COH04S1 vaccine induces cross-reactive and protective immunity against mpox virus infection.

Ortega Francisco, Sandra [1]; Chiuppesi, Flavia [2];
Gutiérrez, Miguel-Ángel [2]; Li, Jing [2]; Mack-Onyeike, Jada [2];
La Rosa, Corinna [2]; Zhou, Qiao [2]; Wussow, Félix [2]; Taplitz,
Randy [3 4]; Diamond, Don [2].

[1]UNAM, Instituto de Biotecnología, Cuernavaca, Morelos, México. [2]City of Hope Medical Center, Department of Hematology and HCT, Hematologic Malignancies Research Institute, Duarte, California, USA. [3]City of Hope National Medical Center, Division of Infectious Diseases, Duarte, California, USA. [4]City of Hope National Medical Center, Deparment of Medicine, Duarte, California, USA.

E-mail: amyrisortega@gmail.com

Despite the official end of the mpox global health emergency caused by mpox virus (MPXV) clade IIb.1, mpox cases continue to be reported due to low vaccination rates and diminishing immunity. COH04S1 is a multiantigen COVID-19 vaccine candidate developed on a fully synthetic platform based on the highly attenuated modified vaccinia Ankara (MVA) vector, which also forms the basis for JYNNEOS, the only FDA-approved smallpox and mpox vaccine. Given the ongoing threat of MPXV resurgence and the need for additional vaccine options, this study investigated the ability of COH04S1 and its synthetic MVA (sMVA) backbone to induce MPXV-specific immunity. Here we analyzed orthopoxvirus-specific and MPXV cross-reactive immune responses in samples from a Phase 1 clinical trial of COH04S1 and from non-human primates (NHPs) vaccinated with COH04S1 or its sMVA backbone. In healthy adults, MPXV cross-reactive immune responses following COH04S1 vaccination were compared to those seen in individuals vaccinated with JYNNEOS. To assess protective efficacy, we evaluated whether COH04S1 and sMVA vaccination protected mpox-susceptible CAST/EiJ mice from MPXV infection. The study found that COH04S1 vaccination induces strong orthopoxvirus-specific humoral and cellular immune responses. These include antibodies that cross-react with MPXV-specific virion proteins and MPXV cross-neutralizing antibodies in nearly half of the vaccinated subjects. Similar MPXV cross-reactive antibody responses were observed in NHPs vaccinated with either COH04S1 or sMVA. Moreover, COH04S1-elicited MPXV cross-reactive humoral responses in humans were comparable to those induced by JYNNEOS. In animal models, both COH04S1 and sMVA provided protection against lung infection after exposure to MPXV clade IIb.1. Overall, the data demonstrate that synthetic MVA-based vaccines can generate cross-reactive and protective immunity against MPXV, supporting the potential of COH04S1 and sMVA as alternative or complementary vaccine options to combat mpox virus infections.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Functional characterization of anti-Spike monoclonal antibody for Fc effector activity

Ortega-Francisco, Sandra; Rustrián-Fernández, Estephania [1];
Liñan-Torres, Arturo [1]; Gutiérrez, Michelle [1]; Ramírez, Octavio T [1];
Palomares, Laura A [1].

[1]Instituto de Biotecnología UNAM, Departamento de Medicina Molecular y Bioprocesos, Cuernavaca, Morelos, México.

E-mail: sandra.ortega@ibt.unam.mx

The use of anti-SARS-CoV-2 antibodies as prophylactic and therapeutic agents proved effective in the early stages of the COVID-19 pandemic. However, the emergence of viral variants has compromised the efficacy of these treatments, primarily due to reduced neutralization resulting from mutations in key epitopes. This has underscored the importance of identifying antibodies with additional antiviral mechanisms, particularly those capable of mediating Fc effector functions, which are influenced by antibody glycosylation profiles. Furthermore, the development of low-cost, scalable, and regulatory-compliant assays is essential to support the clinical evaluation and approval of such antibodies. This study aims to optimize antibody-dependent cellular cytotoxicity (ADCC) assays and develop complementary antibody-dependent phagocytosis (ADP) assays using flow cytometry. These assays are designed to support functional screening of candidate therapeutic antibodies in compliance with regulatory requirements, enabling accessible implementation in Mexico. For ADCC optimization, HEK293 cells expressing the SARS-CoV-2 Spike protein were used as target cells. These were stained with CellTrace Violet (CTV) and incubated with varying concentrations of a monoclonal anti-Spike antibody. NK cells isolated by magnetic negative selection from healthy donor PBMCs served as effector cells. NK activation and target cell apoptosis were evaluated by multiparametric flow cytometry using viability dyes and surface markers such as CD56, CD16, and CD107a. For the ADP assay, fluorescent beads coated with the receptor-binding domain (RBD) of Spike were incubated with the candidate antibody, then exposed to the THP1 monocytic cell line. Phagocytosis was quantified by flow cytometry. These optimized ADCC and ADP assays provide robust, reproducible, and scalable platforms for evaluating the functional activity of monoclonal antibodies. Their implementation could enhance therapeutic antibody development and support regulatory evaluation processes.

ISSN: 2007-3380

Revista Bio Ciencias

https://revistabiociencias.uan.edu.mx

Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Evaluation of VSP 5G8 Expression in Giardia lamblia Under Different Experimental Conditions

Ortega-Tirado, David [1]; Bracamontes-Picos, Leslie [1]; Rascón-Durán, Lucila [1]; Garzón-Lizárraga, Thania [1]; López-Romero, Gloria [1]; Velázquez -Contreras, Carlos [1].

[1]Universidad de Sonora, Departamento de Ciencias Químico-Biológicas, Hermosillo, Sonora, México.

E-mail: david.ortega@unison.mx

Giardia lamblia genome encodes variable surface antigens known as variant-specific surface proteins (VSPs). VSPs influence the immune response to the parasite, and affect the course of Giardia infection. While it is already known that VSPs are involved in antigenic variation, little is understood about their specific functions or the mechanisms that control their expression. In our research group, we have identified and characterized the VSP 5G8 as an antigen highly immunogenic; however, it remains unclear how Giardia regulates its expression (in vivo or in vitro). In the present study, we evaluated the expression of the 5G8 protein under different experimental conditions (during Giardia infection and several in vitro culture conditions). Gerbils (Meriones unguiculatus) were infected with 2x10^6 G.lamblia trophozoites (strain variant GS-5G8(+). Two weeks later, gerbils were sacrificed, and trophozoites were recovered from the small intestine, and analyzed via flow cytometry. Additionally, a co-culture of G. lamblia strain variant GS-5G8(+) with human colon adenocarcinoma cells (Caco-2 cell line) was established. Trophozoites were recovered after 12 h culture and analyzed by flow cytometry. Our results showed that trophozoites recovered from infected animals at 14-days post-infection decreased the 5G8 expression from the trophozoite surface (35% to 5%). A similar reduced 5G8 protein expression level was observed during the in vitro Giardia-Caco-2 cells co-culture (the number of 5G8-positive trophozoites decreased from 75% to 42% after 12 h-co-culture). These results highlight the presence of additional mechanisms regulating VSP expression. Also, a model of study of 5G8 expression (in vivo or in vitro) can contribute to our understanding of how VSPs influence the host–parasite relationship and the progression of Giardia infection.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Evaluation of Renin-Angiotensin System in individuals with normal weight, obesity, and T2DM

Ortiz-Jiménez, Anahí [1]; García-Hernández, Mariana Haydee [1];
Portales-Pérez, Diana Patricia [2 3].

[1]Unidad de Investigación Biomédica, Delegación Zacatecas, Instituto Mexicano del Seguro Social, IMSS. [2]Facultad de Ciencias Químicas, Universidad Autónoma de San Luís Potosí, UASLP, San Luis Potosí, México. [3]Centro de Investigación en Ciencias de la Salud y Biomedicina, Universidad Autónoma de San Luis Potosí, San Luis Potosí, México.

E-mail: anahiortizjimenez08@gmail.com

Introduction. Obesity is a chronic disease characterized by excessive lipid accumulation in adipose tissue, which leads to a persistent state of systemic inflammation. This inflammatory environment promotes metabolic alterations that increase the risk of developing conditions such as type 2 diabetes mellitus (T2DM), a multifactorial metabolic disease characterized by insulin resistance, progressive β-cell dysfunction, and chronic hyperglycemia. Similar to obesity, T2DM presents an inflammatory state that is associated with the renin-angiotensin system (RAS), whose immunoregulatory role in pro-inflammatory processes has been recently studied. Objective. To determine the expression of RAS components in leukocytes from patients with obesity and T2DM, and to evaluate their correlation with clinical and metabolic parameters. Methods. A comparative experimental study was conducted including control subjects, patients with obesity, and patients with T2DM. Peripheral venous blood samples were obtained to isolate PBMCs. Gene expression of ACE1, AGT, AT1R, and AT2R was determined by RT-qPCR, while protein expression was evaluated by FACS. Results. No significant differences were observed in the relative gene expression of ACE1, AGT, AT1R, and AT2R among the groups, whereas MasR expression was significantly lower in individuals with obesity. At the protein level, a significant increase in ACE2 and in the ACE2/ACE1 ratio was observed. Additionally, a trend toward increased ACE1 and ACE2 expression in lymphocytes and monocytes from diabetic patients was detected. Conclusion. There is an alteration in the expression of RAS components in patients with obesity and T2DM, which may contribute to inflammation and insulin resistance.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Induction of Mast Cell Senescence by murine melanoma B16-F10 cells-conditioned media

Ortiz-Santos, Arely Desiree [1]; Ibarra-Sánchez, Alfredo [1];
González-Espinosa, Claudia [1].

[1]Centro de Investigación y de Estudios Avanzados (CINVESTAV) Unidad Sede Sur, Departamento de Farmacobiología, Ciudad de México, México.

E-mail: ortizsantosarely@gmail.com

This project investigates the interactions between mast cells (MCs) and malignant cells in the tumor microenvironment (TME), with the aim to identify those mediators that could lead to MCs senescence. MCs are key components of the innate immune system, primarily implicated in allergic and inflammatory responses. However, they also participate in malignant tumor growth through the production of inflammatory mediators in the TME. Preliminary data suggests that senescent MCs are part of TME but the functional impact of this state on the release of pro-inflammatory cytokines in this microenvironment remains unknown. Cellular senescence is a mostly irreversible state of cell cycle arrest that involves various alterations, including the senescence-associated secretory phenotype, or SASP. This phenotype has been extensively studied due to its paracrine effects in other cells, promoting further senescence. SASP includes a variety of mediators such as interleukins, chemokines, inflammatory factors, growth factors, and more. To analyze the effects of conditioned media (CM) of murine B16-F10 melanoma in the secretory profile of MCs and the possible induction of senescence of those cells, bone marrow-derived cells (BMMCs) were exposed to distinct dilutions of supernatants of confluent cultures of melanoma cells. Then, secretion of selected mediators of SASP (such as IL-6 and VEGF), together with the presence of senescence markers (such as p16, p21 and SA-b galactosidase) was evaluated. Results show that CM of B16-F10 melanoma cells cause a time-dependent secretion of the pro-inflammatory cytokine IL-6 in BMMCs, reaching maximal values 5 days after exposure. Our data provide evidence suggesting important changes on MCs phenotype by melanoma-produced mediators.

Supported by Grant Secihti-CF-3915.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Immunization against SARS-CoV-2 in immunostimulated rats with imiquimod

Pacheco-García, Ursino [1]; Serafín-López, Jeanet [2].

[1]Department of Cardio-Renal Pathophysiology, Instituto Nacional de Cardiología "Ignacio Chávez", Calle Juan Badiano No. 1, Tlalpan,14080, Ciudad de México. . [2]Department of Immunology, Escuela Nacional de Ciencias Biológicas (IPN), Carpio Esq. Plan de Ayala, Cuauhtémoc, Ciudad de México.

E-mail: upacheco@yahoo.com.mx

Abstract: SARS-CoV-2 infection causes COVID-19 disease in individuals with inefficient or dysregulated immune system, especially those with comorbidities such as obesity, diabetes, hypertension, lung damage, and advanced age. Those who develop severe COVID-19, experience post-COVID complications (Long COVID). To better control SARS-CoV-2 infection it is necessary to stimulate the immune system to acquire adequate levels of interferons and an efficient Th1 cellular immunity profile. Imiquimod (IMQ) is an immune response modifier that favors the control of some viral infections.

Objetive: To analyze whether IMQ induces an enhanced immune response in immunization against the SARS-CoV-2 virus.

Methods: Eight-month-old rats are stimulated with IMQ once or for 15 days and immunized with SARS-CoV-2 S1 Ag on days 0 and 20. Serum samples are taken on days 0, 1, 10, 21, and 28 after immunization. Interleukins concentrations were measured at different times and anti S1 antibodies on days 0, 21 and 28.

Conclusions: IMQ administered for 15 days induces an enhanced antibody immune response against SARS-CoV-2 S1 Ag in age-month-old rats and promotes the development of a Th1 cytokine profile.

Keywords: SARS-CoV-2, Long COVID, Vaccine, Imiquimod, Immunostimulation.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Virulence Factors of Staphylococcus aureus Isolates from Atopic Dermatitis Patients Correlate with Disease Severity in a Mexican Cohort

Pastelin-Palacios, Rodolfo [1]; Zumaya-Pérez, Luis Carlos [1];
Velázquez-Sámano, Guillermo [2]; Velasco-Medina, Andrea Aida [2];
Venegas-Flores, Claudia Leticia [1]; García-Bautista, Gabriel Alejandro [1];
Garza-Velasco, Raúl [1]; Rangel-Castañeda, Itzia Azucena [1];
Moreno-Eutimio, Mario Adán [1].

[1]Facultad de Química, UNAM, Mexico City, México. [2]Allergy and Immunology Service, General Hospital of México “Dr. Eduardo Liceaga”, México City, México.

E-mail: rodolfop@unam.mx

Staphylococcus aureus colonization is a hallmark of atopic dermatitis (AD) and contributes to disease exacerbation through the production of various virulence factors. However, the specific characteristics of S. aureus strains isolated from AD patients in Latin America remain poorly described. In this study, we analyzed S. aureus isolates obtained from lesional skin of Mexican patients with AD and compared them to isolates from healthy individuals. Bacterial lysates and culture supernatants were evaluated for the presence and expression of virulence factors, including superantigens, using qPCR and ELISA. Strains from lesional skin exhibited a higher prevalence and expression of superantigens compared to control isolates. Moreover, supernatants from lesional isolates induced increased production of thymic stromal lymphopoietin (TSLP) in a human keratinocyte cell line, suggesting a functional link between bacterial virulence and epithelial immune activation. These results highlight the contribution of S. aureus virulence traits to disease severity and support the importance of bacterial profiling in atopic dermatitis.

Funding: This work was supported by the Programa de Apoyo a Proyectos de Investigación e Innovación Tecnológica (PAPIIT), UNAM, through grants PAPIIT 2023 No. IA208423 and PAPIIT 2024 No. IN221024.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Genomic Characterization of the T-Cell Receptor loci in Ambystoma mexicanum

Pacheco-Olvera, Diana Laura [1 3]; Saint Remy-Hernández, Stephanie [2 3]; Godoy-Lozano, Elizabeth Ernestina [4]; Téllez-Sosa, Juan Mauricio [4]; Valdovinos-Torres, Humberto [4]; López-Macías, Constantino [3]; Martínez-Barnetche, Jesús [4].

[1]Posgrado en Ciencias Quimicobiológicas, Escuela Nacional de Ciencias Biológicas. Instituto Politécnico Nacional. Departamento de Bioquímica. México City. México. [2]Posgrado en Ciencias Quimicobiológicas, Escuela Nacional de Ciencias Biológicas. Instituto Politécnico Nacional. Departamento de Parasitología. México City. México. [3]Unidad de Investigación Médica en Inmunoquímica. UMAE Hospital de Especialidades. Centro Médico Nacional Siglo XXI. Instituto Mexicano del Seguro Social. México City. México. [4]Centro de Investigación Sobre Enfermedades Infecciosas. Instituto Nacional de Salud Pública. Cuernavaca, Morelos. México.

E-mail: diana.laura.pacheco@gmail.com

Amphibians serve as valuable models for comparative immunological research. Prior studies have demonstrated that Ambystoma mexicanum shares the general immunoglobulin loci architecture with Xenopus tropicalis, while lacking certain antibody gene features. In axolotl, T lymphocytes expressing T-cell receptor (TR) α, β, and δ chains have been identified, with restricted structural diversity observed in the δ chain.

Here, we aimed to characterize and annotate the TR loci within the A. mexicanum genome and to conduct comparative analyses with X. tropicalis and other tetrapods. To this end, we mapped, annotated, and analyzed TR loci in the A. mexicanum genome (UKY_AMEXF1_1), leveraging previously reported axolotl TR sequences and orthologous sequences from X. tropicalis, Homo sapiens, and Mus musculus. Additionally, gene models were refined using spleen-derived RNA-seq data from A. mexicanum.

Our results show that the TRA/TRD locus in A. mexicanum retains a largely conserved structural organization relative to other tetrapods. The TRA locus displays an increased number of V segments, with a predominance of functional TRAV genes (functionality ratio: 0.75). Notably, no gene encoding the pre-TCRα chain was identified, suggesting its absence in this locus. The TRD locus comprises two conventional TRDV genes; however, one appears to be a pseudogene. Furthermore, TRDD genes were absent. No genomic or transcriptomic evidence of TRG locus components (TRGV, TRGJ, TRGC) was detected. In contrast, the TRB locus exhibits structural similarity to those of other tetrapods, comprising five TRBD-TRBJ-TRBC translocons. Interestingly, one TRBC gene contains two exons encoding two Cβ domains. Functional TRBV genes predominate (functionality ratio: 0.75), mirroring the TRAV gene profile. Collectively, these findings offer new insights into T-cell receptor evolution in amphibians. The absence of TRDD segments implies direct TRDV-TRDJ rearrangements, limiting TRδ diversity, whereas the lack of a TRG locus supports the absence of bona fide γδ T cells in A. mexicanum.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

Early and transient CD40 expression in T cells

Parga-Ortega, Jesús Miguel [1]; Villalpando-Sánchez, Diana [2];
Medina-Contreras, Óscar [2]; Patiño-López, Genaro [1].

[1]Laboratorio de Investigación en Inmunología y Proteómica, Hospital Infantil de México, Federico Gómez. Ciudad de México, México. [2]Laboratorio de Inmunometabolismo, Unidad de Investigación Epidemiológica en Endocrinología y Nutrición, Hospital Infantil de México, Federico Gómez. Ciudad de México, México.

E-mail: gpatino@himfg.edu.mx

In a recent study we identified several potential new T cell activation molecules, among them, one very interesting candidate was CD40, CD40 is the canonical molecule for B cell-T cell help, that is antibody production (including Class Switch Recombination), germinal center formation and memory B cell maturation and is mainly expressed in antigen resenting cells (APC), although has been reported in epithelial cells, endothelial cells and some tumor cells. When CD40 interact with CD40-L in activated T cells activate a myriad of signaling pathways that induce IL-2 production and T helper differentiation, T cell memory and some effector functions, however, the expression of CD40 in T cells has been a controversial topic, earlier studies demonstrated that CD40 in CD8 T cells is essential for memory formation and other groups demonstrated high expression of CD40 in CD4 T cells from patients with autoimmune type I diabetes. Preliminary results in our lab indicate CD40 expression in murine and human T cells, also upon CD3-CD28 stimulation CD40 mRNA expression is transiently augmented at 6h with a decay phase after 18-24h, this kinetics are similar to other well-known activation molecules like CD69, current studies are underway to evaluate the functional consequences of CD40 overexpression and down-regulation (knock-down) in Jurkat cells to determine what are the main functions in T cells that are regulated by CD40 expression.

Work supported by Grants #320456 by CONAHCYT and grant HIM/2022/015 to GPL.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Impact of Arylamine N-Acetyltransferase Silencing on $CD4^+$ T Cell Polarization and Immunometabolism

Paz-Rodríguez, Víctor-Alejandro [1]; Castillo-Esquivel, Arian-Dalai [1];
Sánchez-Lucio, Lizeth-Sinai [1]; Turiján-Espinoza, Eneida [2];
Martínez-Leija, Miguel-Ernesto [1 2]; Cortez-García, Juan-Diego [2];
Garcia-Hernández, Mariana-Haydee [3]; Salazar-Gonzales, Raúl-Alejandro [4]; Portales-Pérez, Diana-Patricia [1 2].

[1]Center for Research in Health Sciences and Biomedicine, Autonomous University of San Luis Potosí, San Luis Potosí, México. [2]Faculty of Chemical Sciences, Autonomous University of San Luis Potosí, San Luis Potosí, México. [3]Biomedical Research Unit of Zacatecas, Zacatecas, México. [4]Preclinical Services and Toxicology, Discovery Life Sciences, MA, USA.
E-mail: victor.pazrdz98@gmail.com

The phenotype and function of T cells depend on cellular metabolism. Effector T cells rely on glycolysis, while regulatory T cells (Tregs) depend on mitochondrial metabolism. This process converts pyruvate into acetyl-CoA via pyruvate dehydrogenase (PDH). Cytosolic enzymes called arylamine N-acetyltransferases (NAT1 and NAT2) are involved in the metabolism of xenobiotics, such as drugs and carcinogens. Similarly, NATs play a role in regulating PDH. Deletion of NATs induces mitochondrial dysfunction and reduced PDH expression. However, the role of NATs in T cell metabolism and differentiation into pro-inflammatory (Th17) or anti-inflammatory (Treg) phenotypes remains unexplored. We cultured and transfected peripheral blood mononuclear cells (PBMCs) from healthy donors with small interfering RNAs (siRNAs) targeting NAT1 and NAT2. We then assessed the effects of the siRNAs on Th17 and Treg polarization conditions by flow cytometry. NAT1 and NAT2 expression was significantly higher in basal Th17 and Treg cells than in CD4+ and CD8+ T cells ($p < 0.0001$). Under polarized conditions, NAT2 expression increased in both Th17 and Treg cells compared to unstimulated controls ($p < 0.008$). Bioinformatic analysis suggested that the transcription factor PXR contributes to NAT upregulation following TCR activation. Inhibition assays of NAT1 and NAT2 with siRNAs revealed a significant decrease in Treg cells compared to controls with scrambled siRNAs ($p = 0.0012$). Conversely, NAT2 inhibition increased the proportion of Th17 cells compared to scrambled controls ($p = 0.00023$). These findings suggest that NAT1 and NAT2 are upregulated during T cell activation and highlight an unrecognized role of NAT1 and NAT2 in T cell differentiation. The increased expression of NAT1 and NAT2 in polarized Th17 and Treg cells, along with the opposing effects of their inhibition, suggests that these enzymes may contribute to the metabolic regulation of inflammatory responses.

ISSN: 2007-3380

Revista Bio Ciencias

https://revistabiociencias.uan.edu.mx

Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

“Comparative analysis of immunogenic proteins of Mycobacterium tuberculosis recognized by patients in active and latent phases”

Pelcastre-Gómez, Andrea Yamile [1 2]; Vázquez-Leyva, Said [1];
Olvera-Olguin, Vania [1 2]; Hernández-Pando, Rogelio [3];
Chávez-Galán, Leslie [4]; Chacón-Salinas, Rommel [2];
Castañeda-Casimiro, Jessica [2]; Pérez-Tapia, Sonia Mayra [1 2];
Pedraza-Escalona, Ma. Martha [5].

[1]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), CDMX, México. [2]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, CDMX, México. [3]Instituto Nacional de Ciencias Médicas y Nutrición, CDMX, México. [4]Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas (INER), CDMX, México. [5]SECIHTI-UDIBI. Escuela Nacional de Ciencias Biológicas, IPN, CDMX, México.

E-mail: yamile.pelcg@gmail.com

Tuberculosis (TB), caused by Mycobacterium tuberculosis (MTB), remains a significant global public health challenge. A major obstacle to effective TB control is the identification of reliable immunogenic antigens that can serve as therapeutic, diagnostic, or vaccine targets, owing to the bacterium’s structural complexity and its ability to evade the host immune response. In this study, a combined experimental and bioinformatic approach was employed to identify and characterize immunogenic proteins from the MTB H37Rv strain, recognized by antibodies present in sera from patients with active TB (ATB) and latent TB infection (LTBI). Human serum samples were analyzed by Western blot and immunoprecipitation against MTB protein extracts, revealing a greater number of recognized proteins in sera from ATB patients, indicative of a more robust immune response during active disease. Selected proteins were subsequently identified via MS/MS and functionally annotated using various bioinformatic tools. Analyses included subcellular localization, predicted biological functions, and the identification of linear and conformational B-cell epitopes, supported by three-dimensional structural modeling using AlphaFold. The results showed a predominance of proteins involved in central metabolism, lipid biosynthesis of the cell envelope, and immune evasion mechanisms—processes critical to MTB pathogenesis and persistence. Among the identified proteins, Rv3616c, Rv2007c, and Rv2742c emerged as promising therapeutic targets due to their high immunogenicity, structural accessibility, and potential roles in MTB virulence and survival.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Function of pVHL in the Apoptosis of Pancreatic β Cells

Pérez-Eguía, Neivy Lizeth [1]; Cancino-Díaz, Juan Carlos [2];
Hernández-Valenzuela, Marco Antonio [1 2]; Cancino-Díaz, Mario
Eugenio [1].

[1]Instituto Politécnico Nacional, Escuela Nacional de Ciencias Biológicas, Departamento de Inmunología, Ciudad de México. [2]Instituto Politécnico Nacional, Escuela Nacional de Ciencias Biológicas, Departamento de Microbiología, Ciudad de México.

E-mail: shilizpe@hotmail.com

Type 2 diabetes mellitus has been associated with the progressive dysfunction and apoptosis of pancreatic β cells, which compromises their ability to maintain glucose homeostasis. Several studies have shown that the von Hippel-Lindau protein (pVHL) plays a key role in regulating glucose metabolism. In transgenic mouse models, deletion of the VHL gene in β cells leads to hyperglycemia, suggesting a direct role in preserving their function. In this context, we investigated whether pVHL can protect β cells from apoptosis. The aim of this study was to evaluate the effect of pVHL on β-cell apoptosis by introducing the protein using either an adenoviral vector (AdVHL) or a recombinant soluble version fused to a cell-penetrating peptide (rTAT-VHL). The AdVHL and control AdGFP vectors were generated and validated in a psoriasis model, as well as in RIN-5F cells (a rat insulinoma-derived β-cell line) via immunofluorescence. In parallel, a Gblock was designed for the expression of rTAT-VHL in E. coli Rosetta, and the recombinant protein was purified by size exclusion chromatography (SEC). Internalization of rTAT-VHL into RIN-5F cells was confirmed by confocal microscopy. Apoptosis was evaluated using annexin V/PI staining and flow cytometry in cells treated with cisplatin. The results show that the expression of recombinant pVHL via the adenoviral vector reduces both basal and cisplatin-induced apoptosis after 24 hours of treatment. These findings suggest that pVHL may act as a protective agent for pancreatic β cells, helping to preserve their function under stress conditions, such as those present in diabetes.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Identification of Non-Replicating Persistent Salmonella in B cells during *in vitro* Infections

Pérez-Lara, Jocelyn Carolina [1]; Cruz-Cruz, Alonso D [3];
Velázquez-Valdez [1 2], Diana Zuleika [1,2]; Ortiz Navarrete, Vianney [1].

[1]Departamento de Biomedicina Molecular. CINVESTAV, Ciudad de México, CDMX, México. .
[2]Doctorado en Ciencias de la Producción y Salud animal en la Universidad Nacional Autónoma de México, Facultad de Medicina Veterinaria y Zootecnia, Ciudad de México, CDMX, México. .
[3]Programa de Maestría del Departamento de Biología Celular, CINVESTAV and Departamento de Microbiología Universidad Estatal de Ohio.

E-mail: jocelyn.perez@cinvestav.mx

Salmonella enterica serovar Typhimurium (S. Tm) can colonize different intracellular niches, either actively dividing or remaining dormant to persist. Bacterial persisters are phenotypic variants that temporarily enter a non-replicative state. This allows them to evade host cell defenses and antibiotics, leading to chronic infections. We previously reported that during chronic periods, Salmonella remains within B cells in the bone marrow and spleen. However, the dynamics of Salmonella replication and the formation of antibiotic tolerance in infected B cells have not been studied. Here we show that B cells are a favorable reservoir for bacterial persistence. In vitro experiments identified non-replicating, persistent Salmonella subsets in splenic B cells. These non-replicative Salmonella are tolerant to antibiotics (cefotaxime and ciprofloxacin), while replicative bacteria remain susceptible. Although acid intravacuolar pH and SPI-2 regulators (SsrA/SsrB) are not necessary for Salmonella persistence in B cells, the SehA/B toxin-antitoxin system facilitates the formation of the persistent phenotype in Salmonella. Overall, we show that B cells are a reservoir for non-replicating, antibiotic-tolerant Salmonella.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Role of CCR3 in neutrophil activation during interaction with influenza virus *in vitro*

Benard -Zamorano, Mariana; Cabellos- Avelar , Tecilli; García
Aguiar, Jesús Israel; Pérez López, Araceli.

[1]UNAM, Facultad de Estudios Superiores Iztacala, Unidad de Investigación en Biomedicina (UBIMED) , Tlalnepantla, Estado de México, México. [2]Universidad Autónoma de Occidente, Unidad de Investigación en Biotecnología Biomédica, Culiacán, Sinaloa, México.

E-mail: aracelipl83@gmail.com

Neutrophils are the most abundant leukocytes in the blood, and their activation is a defense mechanism against infections through processes such as the formation of neutrophil extracellular traps (NETs). However, uncontrolled activation of neutrophils can result in tissue damage to the host. One example is infections caused by influenza A virus (IAV), particularly severe cases, in which recruitment and activation of neutrophils in lung tissue is associated with fatal outcomes. Our group has demonstrated that neutrophils acquire the expression of chemokine receptor 3 (CCR3) when recruited to the lungs during IAV infection. However, it is unknown whether stimulation of CCR3 contributes to neutrophil activation during IAV infection. Therefore, the aim of this study was to evaluate the effect of CCR3 activation on NET production in neutrophils from humans and mice in response to IAV *in vitro*. Our results indicate that IAV interaction with neutrophils induces NET production in both mouse neutrophils (increase of ~35%) and human neutrophils (increase of ~25%) compared to uninfected cells. When neutrophils were stimulated with CCL28 (a ligand of CCR3) in presence of IAV, we observed a slight increase in NET formation in mouse neutrophils and no clear effect in human neutrophils. However, upon addition of the compound SB-328437 (a highly specific antagonist of CCR3), there was a significant reduction in NET production in both human neutrophils (reduction of ~25%) and murine neutrophils (reduction of ~15%) during IAV infection. Our data indicate that, in vitro, the CCR3 receptor participates in the induction of NETs in both human and mouse neutrophils during in presence of IAV.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Development and optimization of cancer vaccines for breast cancer treatment

Pérez-Méndez, Erasmo Leonel [1]; Almeida-Luna, Paloma [1];
Martínez-Badillo, Saraí Verónica [1]; Jiménez, Samanta [2];
González-Sánchez, Ricardo [2]; Idoyaga-, Juliana [3];
Fournier, Pierrick GJ [2].

[1]CICESE, Posgrado en Ciencias de la Vida, Ensenada, BC, México. [2]CICESE, Departamento de Innovación Biomédica, Ensenada, BC, México. [3]UCSD, Department of Pharmacology, La Jolla, CA, USA.

E-mail: eperez.mendez@cicese.edu.mx

Breast cancer (BC) is the primary cause of cancer death in women worldwide, and new treatments need to be tested. Using a syngeneic BC model with the 4T1 cell line, we assessed the efficacy of different methods of cancer vaccines to elicit an immune response against cancer cells. 4T1 cells were inoculated in mouse mammary glands to cause tumors and to prepare tumor cells as an antigen source. These tumor cells were fixed with paraformaldehyde and mixed with Freund's adjuvant to treat 4T1 tumor-bearing mice. However, two immunizations did not decrease tumor volume and weight compared to control mice. Thus, we turned to dendritic cell (DC) vaccines. Mouse bone marrow cells (BM) were differentiated into DCs using GM-CSF and IL-4. These GM-DCs were pulsed with tumor lysates and activated with LPS and PAMPs from a prophylactic vaccine (Vanguard-Plus). Although there were increased plasma levels of anti-4T1 antibodies in mice treated with GM-DCs (+69%), there was no increase in tumor-infiltrating lymphocytes and only a 16% decrease in tumor volume that could not be reproduced. DCs inoculated were all cDC2, which may not be optimal against cancer. To improve DC preparation, we cultured BM cells with FLT3L and Delta-like 1 (DLL1) to obtain a mix of cDC1 and cDC2 (~1:5 ratio; DLL1-DCs). DCs were cultured with different TLR agonists: LPS, Poly(I:C), Resiquimod, and CpG. The combined four agonists led to the best increase of activation markers CD80, CD86, MHC-II, and CCR7 measured by flow cytometry and of Il12b mRNA measured by qRT-PCR, in GM-DCs and DLL1-DCs. However, upon activation, Tnfa and Ifng levels were higher in DLL1-DCs than in GM-DCs. These results suggest that DLL1-DCs could be more effective than GM-DCs to cause a type I response against cancer cells, which will be tested against 4T1 cells in mice.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Preclinical Evaluation of a Multi-Epitope HIV-1 Vaccine Candidate Engineered on a B19 Parvovirus VLP Platform

Pérez-Saucedo, David Gilberto [1]; Castro-Perea, Nancy Vanessa [2];
Ruíz-Cruz, Antonio [1]; Bustos-Jaimes, Ismael [3];
Viveros-Rogel, Monica [4]; Huerta-Hernández, Leonor [5].

[1]Unidad de Investigación en Biomedicina, Facultad de Estudios Superiores Iztacala, UNAM. [2]Instituto Tecnológico de Tijuana, Centro de Graduados e Investigación en Química. Tecnológico Nacional de México. [3]Departamento de Bioquímica, Facultad de Medicina, UNAM. [4]Departamento de Enfermedades Infecciosas, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán. [5]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, UNAM.

E-mail: xlv2008@gmail.com

The high mutational rate and antigenic diversity of HIV-1, leading to immune escape and viral reservoirs, present major challenges for vaccine development. Modern strategies aim to focus immune responses on conserved epitopes to induce broadly neutralizing antibodies (bnAbs). To overcome the low immunogenicity of isolated epitopes, we developed a carrier system using Virus-Like Particles (VLPs) from the B19 human parvovirus.We engineered chimeric VLPs ("VLP-MHIV-A") to display multiple HIV-1 epitopes on their surface, which were selected based on their recognition by patient-derived bnAbs. Correct VLP morphology and assembly were confirmed by TEM and DLS, respectively. Antigenicity was verified by the binding of VLP-MHIV-A to sera from HIV+ patients. Immunogenicity was evaluated in C57bl/6 mice via intramuscular (I.M.) and subcutaneous (S.C.) routes. Both routes induced substantial specific antibody responses in serum and vaginal lavages, with antibodies detected against the CD4 binding site, V3 loop, and gp41 epitopes. Functional assessment in an in vitro reporter assay showed that both immunization routes produced neutralizing activity. The I.M. route, however, was demonstrably more effective, achieving a significant 50% inhibition of a tier 1 clade B virus. These results validate our chimeric VLP platform as an effective antigen for inducing neutralizing antibodies against HIV-1.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Role of autophagy in PD-L1 expression in a lung cancer cell model

Pérez-Torres, Christian [1]; García-Pérez, Blanca Estela [1];
Baltierra-Uribe, Shantal Lizbeth [1]; Castrejón-Jiménez, Nayeli
Shantal [2].

[1]Instituto Politécnico Nacional - Escuela Nacional de Ciencias Biológicas, Departamento de Microbiología, Ciudad de México, México. [2]Instituto de Ciencias Agropecuarias - Universidad Autónoma del Estado de Hidalgo, Área Académica de Medicina Veterinaria y Zootecnia, Tulancingo-Hidalgo, México.

E-mail: chris_copernicium@hotmail.com

Programmed death-ligand 1 (PD-L1) is a glycoprotein expressed in a wide range of cell types, including tumor cells. Its interaction with the PD-1 receptor on T lymphocytes leads to the inhibition of T cell-mediated immune responses. The expression of PD-L1 is regulated by various mechanisms, including cytokines, transcription factors, and microRNAs. However, recent studies have suggested that autophagy, an intracellular degradation pathway, may also serve as a regulatory mechanism of PD-L1 expression. Despite these findings, the exact molecular details underlying this regulation remain largely unknown. In the present study, we investigated whether PD-L1 expression in A549 and HeLa cells could be modulated through autophagy. To this end, both cell lines were treated with autophagy inducers and inhibitors, followed by evaluation of PD-L1 expression using western blotting and immunofluorescence analysis. Our results demonstrated that autophagy inhibition in A549 cells resulted in increased PD-L1 expression, whereas autophagy induction led to its downregulation. In contrast, HeLa cells exhibited decreased PD-L1 expression under both autophagy-inducing and -inhibiting conditions. These findings suggest that autophagy modulates PD-L1 expression in a cell type-dependent manner, highlighting the complexity of its regulation in different cancer contexts.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Vacunas**

"Evaluation of the immune response induced by vaccine formulations against Salmonella Typhi."

Pérez-Vergara, Ana Delia [1]; Lozano -Cisneros, Diego [1];
García-Valeriano, María Guadalupe [1]; Rivera -Hernández, Tania [1];
López-Macias, Constantino [1].

[1]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades "Bernardo Sepúlveda Gutiérrez", Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México.

E-mail: anadeliabqd@gmail.com

Salmonella enterica serovar Typhi is a clinically significant pathogen worldwide and the causative agent of typhoid fever, which is acquired through the consumption of contaminated food and water. The disease has a global distribution, with higher prevalence in low- and middle-income countries. Although improvements in quality of life have led to a reduction in typhoid fever morbidity and mortality, a new challenge has emerged with the appearance of multidrug-resistant strains to first-line antibiotics. Therefore, this study focuses on the evaluation of a vaccine formulation aimed at preventing this disease, based on molecules known as porins, which have been shown to elicit strong immunogenic responses. Additionally, we explore the use of an adjuvant to assess whether it enhances the immune response in animal models. To compare the immune response elicited by a Salmonella Typhi porin-based vaccine formulation with and without the addition of the MF59 adjuvant. Porins combined with the adjuvant induced higher IgG antibody titers compared to the control group that received antigen without the adjuvant. However, when evaluating protection against the disease, a lower bacterial load was observed in the liver and spleen of the groups administered only with porins. Therefore, in terms of protection against infection, the addition of the MF59 adjuvant does not confer a benefit to the protective capacity of S. Typhi porins. MF59 is an adjuvant that has been used for decades in various human vaccines due to its ability to enhance immunogenic responses; however, it is not suitable for use in a porin-based vaccine formulation.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

CRP variants and serum levels as cardiovascular and disease activity biomarkers in SLE

Pesqueda Cendejas, Karen [1 2]; Parra Rojas, Isela [1 3];
Campos López, Bertha [1 2]; Mora García, Paulina Esmeralda [1 2];
Meza-Meza, Mónica del Rosario [1 2]; Rivera Escoto, Melissa [1];
Ruiz-Ballesteros, Adolfo Isaac [1]; De la Cruz Mosso, Ulises [1 2].

[1]Red de Inmunonutrición y Genómica Nutricional en las Enfermedades Autoinmunes; Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud; Universidad de Guadalajara; Guadalajara, Jalisco 44340, México. [2]Instituto de Neurociencias Traslacionales, Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara 44340, Jalisco, México. [3]Laboratorio de Investigación en Obesidad y Diabetes; Facultad de Ciencias Químico-Biológicas; Universidad Autónoma de Guerrero; Chilpancingo de los Bravo 39087, Guerrero, México.

E-mail: karen.pesqueda20@gmail.com

Systemic lupus erythematosus (SLE) is an autoimmune disease where genetic factors have been related to SLE susceptibility and disease severity. Genetic variants in the CRP gene could influence the C-reactive protein (CRP) serum levels and contribute to the cardiovascular disease (CVD) risk and the clinical disease activity in SLE. This study aimed to determinate the association of the −717 A>G, −409 G>A, +1444 C>T, and +1846 C>T CRP single nucleotide variants (SNVs) and CRP serum levels as potential biomarkers of CVD and clinical disease activity risk in SLE. this cross-sectional study enrolled 369 unrelated women: 183 with SLE according to the 1997 SLE-ACR criteria and 186 control subjects (CS). The clinical disease activity was assessed by the Mex-SLEDAI score; CRP serum levels were quantified by turbidimetry. The CRP SNV genotyping was carried out by allelic discrimination. SLE patients had higher CRP levels than CS (SLE: 5 mg/L vs. CS = 1.1 mg/L; $p < 0.001$). In SLE patients, CRP levels ≥ 3 mg/L were associated with high clinical disease activity (OR = 2.5: IC: 1.03–6.2; $p = 0.04$; and β coefficient = 5.8; IC: 2.5–9.4; R2 = 0.15; $p = 0.001$). With an area under curve (AUC) of 0.67 it was determined that CRP levels have a moderate ability to discriminate between patients with and without clinical activity. Regarding -717 CRP SNV, AA genotype was associated with high CVD risk in SLE patients (OR = 3; CI: 1.2–7.6; $p < 0.01$). In conclusion, the −717 A>G CRP SNV was a risk factor for high CRP levels and high CVD risk. Moreover, CRP levels are a modest biomarker of clinical disease activity in Mexican-mestizo SLE patients.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Mitochondrial Dysfunction Drives Immune Dysregulation and Oxidative Stress in Spondyloarthritis

Prieto-Carraco, Rodrigo [1]; González -Chávez, Susana Aideé [1]; Chaparro-Barrera, Eduardo [1]; Loya-Rivera, Mario [1]; Cuevas-López, Belén [2]; García-Arroyo, Fernando E. [2]; Aparicio-Trejo, Omar Emiliano [2]; Pacheco-Tena, César [1].

[1]Universidad Autónoma de Chihuahua, Facultad de Medicina y Ciencias Biomédicas, Laboratorio de Patogenia y Biomedicina Molecular (PABIOM), Chihuahua, Chihuahua, México. [2]Instituto Nacional de Cardiología "Ignacio Chávez", Departamento de Fisiopatología Cardio-Renal, Ciudad de México, México.

E-mail: rprieto@uach.mx

Mitochondria are central regulators of cellular metabolism and immune responses. In chronic inflammatory diseases such as spondyloarthritis (SpA), mitochondrial dysfunction contributes to pathogenesis by promoting oxidative stress, metabolic imbalance, and activation of innate immune pathways through damage-associated molecular patterns (DAMPs). This study investigates mitochondrial alterations and their immunometabolic consequences during SpA progression in a murine model. Spontaneous arthritis (SpAD) was evaluated in male DBA/1 mice at 6 weeks (acute inflammation/osteogenesis) and 11 weeks (chronic ossification). BALB/c mice were used as healthy controls. Mitochondria were isolated from hind limb tissues to assess proteins involved in mitochondrial dynamics (fission, fusion), biogenesis, and mitophagy by western blot. Mitochondrial complex activity, ATP production, antioxidant enzyme activity (SOD, catalase, Gpx), and oxidative damage markers were analyzed. Transcriptomic changes were characterized by microarray and validated by RT-qPCR and western blotting. In the active phase (6 weeks), SpAD mice showed decreased fission and increased fusion, biogenesis, and mitophagy. This was associated with impaired complex II activity, reduced ATP synthesis, and elevated oxidative stress, as evidenced by increased SOD and reduced catalase/Gpx activity. In the chronic phase (11 weeks), mitochondrial dysfunction persisted with increased fission and mitophagy, decreased fusion, and sustained bioenergetic deficits. Antioxidant enzyme imbalance and redox stress markers remained elevated. These findings highlight mitochondrial quality control as a key regulator of immune activation in SpA. Dysregulated mitochondrial dynamics and mitophagy disrupt bioenergetic homeostasis and sustain oxidative stress, amplifying inflammation throughout disease progression. Targeting mitochondrial pathways may offer novel therapeutic opportunities to restore immune balance in spondyloarthritis.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos B**

Targeting B cell activation in vitro using antigen-decorated liposomes carrying Bcl-6 mRNA

Quinto-Manzanares, Rocio; Galván-Hernández, Arturo;
Téllez-Sosa, Juan Mauricio; Martínez-Barnetche, Jesús;
González-Sánchez, Hilda Minerva.

[1]Instituto de Investigación en Ciencias Básicas y Aplicadas, Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México. [2]Instituto de Ciencias Físicas, Universidad Nacional Autónoma de México, Cuernavaca, Morelos, México. [3]Centro de Investigación Sobre Enfermedades Infecciosas, Instituto Nacional de Salud Pública, Cuernavaca, Morelos, México. [4]Investigadores por México, Secihti, Centro de Investigación Sobre Enfermedades Infecciosas, Instituto Nacional de Salud Pública, Cuernavaca, Morelos.

E-mail: quinto.roci12@hotmail.com

The specific activation of B cells initiates the formation of germinal centers (GC), where B cells proliferate and undergo affinity maturation. Multivalent presentation of antigens on nanoparticles such as liposomes enhances BCR activation by promoting efficient crosslinking, mimicking natural antigen display. This approach also enables the co-delivery of functional molecules such as mRNA. A key factor in GC formation is the expression of Bcl-6, a transcriptional repressor essential for B cell entry into follicles and for initiating processes like somatic hypermutation and class-switch recombination. In this context, liposomes offer a versatile platform to simultaneously deliver antigenic and molecular signals critical for B cell activation. The project aims to develop a model of specific activation of human B cells using liposomes conjugated with antigen and modified Bcl-6 mRNA. DNA templates for Bcl-6 and a GFP control were designed for in vitro transcription, incorporating human codon optimization, UTRs, a Kozak sequence, promoter-binding region, and a poly(A) tail. Modified mRNAs were synthesized with modified nucleotides and a Cap analog, encapsulated into cationic liposomes. RNA encapsulation was evaluated by electrophoresis in agarose gels, and expression validated in mammalian cells by flow cytometry. Liposomes will be decorated with the SARS-CoV-2 RBD protein, and, as a control, with protein A (SpA) from Staphylococcus aureus, a superantigen. Size and surface charge will be assessed by tunable resistive pulse sensing (TRPS) and transmission electron microscopy (TEM). The enrichment of human antigen-specific B cells will be analyzed by flow cytometry and specific antibody secretion measured in culture supernatants. It is expected that antigen-decorated liposomes carrying Bcl-6 mRNA will enable the targeted activation, expansion, and differentiation of B cells into antibody-secreting cells.

Funding: Secretaría de Investigación, Ciencia, Tecnología e Innovación project No. CF-2023-I-2387, Fellowships CVU No.1079908.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Immunopathogenesis and Novel Therapeutic Strategies in Rheumatoid Arthritis.

Quiñones-Acuña, Diana Lizeth [1]; Alvarado-Ramos, Jessica Yuritzi [1]; Alvarado-Flores, Ernesto [1].

[1]Autonomous University of Zacatecas, Fresnillo Campus.

E-mail: lizethacu.14@gmail.com

Rheumatoid arthritis (RA) is a chronic autoimmune disorder marked by persistent synovial inflammation causing joint destruction and systemic morbidity, affecting around 0.2–1 % of the global population. Its pathogenesis involves dysregulated immune responses: genetic predispositions including HLA-DRB1, epigenetic modifications (e.g., DNA methylation, histone changes), and altered metabolic pathways in immune and stromal cells. The synovial microenvironment harbors pathogenic cell populations—such as Th1/Th17-skewed T cells, GZMK□ CD8□ T cells, Tfh-like and aging B cells, inflammatory fibroblast-like synoviocytes, and macrophage subsets—that sustain inflammation and osteoclastogenesis. Metabolically, CD4□ T cells shift from glycolysis to the pentose phosphate pathway, fibroblast-like synoviocytes show elevated anaerobic glycolysis, mitochondrial dysfunction, imbalances in polyunsaturated fatty acids (PUFAs), and altered amino acid metabolism contributing to persistent inflammation. Conventional disease-modifying antirheumatic drugs (DMARDs), biologics, and targeted small molecules provide clinical benefit, yet remission rates remain suboptimal. Emerging therapies focus on epigenetic regulators and metabolic modulators in preclinical and clinical trials. Precision medicine strategies based on synovial biopsy, RNA sequencing, mesenchymal stem cells, and CAR T cell approaches show promise. These advances—rooted in a deeper understanding of immunopathogenesis, immunometabolism, and epigenetics—are accelerating the translation of novel therapies in RA.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Optic Neuritis as Initial Manifestation of Multiple Sclerosis: A Case Report

Ramírez -Álvarez, Jamaliel Sachel [1]; Romo-Vera, Brenda
Guadalupe; Juárez-De Santiago, Zuemi [1].

[1]Universidad Autónoma de Zacatecas, Unidad Académica de medicina humana, Fresnillo,
Zacatecas, México.

E-mail: jamasa503@gmail.com

Multiple sclerosis (MS) is a chronic inflammatory, immune-mediated, demyelinating disease of the central nervous system characterized by perivascular lymphocytic infiltration, multifocal demyelination, reactive gliosis, and axonal damage. It primarily affects young adults, especially women, and may present with acute episodes (relapses) or gradual progression. We report the clinical, pathophysiological, and therapeutic features of optic neuritis as the initial manifestation of MS through a case study. A 45-year-old female patient with relapsing-remitting MS, under interferon beta treatment, presented with neurological exacerbation marked by right peripheral facial palsy, left optic neuritis with partial visual loss, persistent paresthesias in the left lower limb, progressive hemibody weakness, and marked fatigue. Neurological examination revealed right peripheral facial paralysis (cranial nerve VII), left optic neuritis evidenced by temporal optic nerve pallor, distal hypoesthesia on the left hemibody, mild muscle weakness (4/5) on the right hemibody, generalized hyperreflexia, paresthesias in lower limbs, and ataxic gait. Pathophysiology involves autoreactive CD4+ and CD8+ T lymphocytes, B cells, and macrophages crossing the blood-brain barrier, targeting myelin basic protein (MBP) and myelin oligodendrocyte glycoprotein (MOG), leading to autoimmune demyelination and axonal injury. B cells produce specific antibodies aggravating demyelination and inflammation, contributing to neurotoxicity, oxidative stress, and mitochondrial dysfunction. Remyelination attempts by oligodendrocyte precursor cells are limited in advanced disease stages, resulting in structural and functional sequelae. Optic neuritis as an acute MS relapse is a clinically significant and frequent manifestation that serves as a useful diagnostic and monitoring marker, reflecting the central immunopathology of MS.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Generation of an Anti-CD19 scFv Panel Using the ALTHEA Platinum III Phage Display Library

Ramírez-Florencio, Mireya [1 2]; Téllez-Severo, Filemón [1];
Gómez-Castellano, Keyla María [1 2]; Mendoza-Salazar, Ivette [1 2 3];
Vázquez -Leyva, Said [1 2]; Almagro, Juan Carlos [1 2 4];
Pérez-Tapia, Sonia Mayra [1 2 3].

[1]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, México City, México. [2]Laboratorio Nacional Para Servicios Especializados de Investigación, Desarrollo e Innovación (I+D+i) Para Farmoquímicos y Biotecnológicos, LANSEIDI-FarBiotec-CONACyT, México City, México. [3]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, México City, México. [4]Global Bio, Cambridge Massachusetts, United States.

E-mail: mireram26@gmail.com

Chimeric antigen receptor (CAR)-T cell therapy has emerged as a powerful strategy in cancer treatment, involving the genetic modification of autologous T cells to express chimeric receptors capable of recognizing and eliminating tumor cells. CD19, a surface protein highly expressed on malignant B cells, is a prominent target in CAR-T therapies. The scFv commonly used in approved anti-CD19 CAR-T therapies is FMC63, of murine origin. However, repeated administration can lead to increased immunogenicity and reduced efficacy. To address this limitation, this study aimed to discover and characterize novel human-derived scFvs targeting the CD19 antigen using phage display technology. As a first step, the FMC63 scFv was cloned, expressed in E. coli TG1, purified, and characterized by SDS-PAGE and UV absorbance at 280 nm. Its binding to recombinant biotinylated CD19 and CD19-expressing NALM-6 cells was confirmed via flow cytometry, validating the assay conditions. For novel scFv discovery, the ALTHEA Platinum III phage display library was used in three rounds of semi-automated solution-phase panning with decreasing concentrations of biotinylated human CD19 (50 nM, 10 nM, and 1 nM, respectively). An extended monoclonal screening of 270 clones was conducted, from which 19 positive clones were selected for sequencing. Among these, eight clones with unique scFv sequences were expressed and evaluated using periplasmic extracts. All eight clones showed binding to recombinant CD19, but only one clone (E7) exhibited low-level binding to NALM-6 cells. However, upon further evaluation, E7 failed to show consistent binding to CD19 on NALM-6 cells. These results highlight the successful identification of novel scFvs against CD19, with confirmed reactivity to the recombinant antigen. Further affinity maturation or engineering may be needed to improve binding to native CD19 on cell surfaces for therapeutic application.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Dynamics of immunoglobulin production in milk: IgA and IgG across lactation stages in C57BL/6 mice

Ramírez-Pérez, Irvin [1]; Santos-Argumedo, Leopoldo [1 2].

[1]Departamento de Biomedicina Molecular; Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, Cd. México. [2]Centro de Investigación sobre el Envejecimiento; Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, Cd. México Sede Sur.

E-mail: irvin.ramirez@cinvestav.mx

Given the importance of breastfeeding for proper offspring development and survival, an increasing number of studies are focusing on analyzing the components of breast milk and their significance for neonates. This interest has expanded to examine the relationship between maternal factors (such as nutrition, health status, and vaccination status) and the dynamic composition of milk, as well as its effects on offspring. The antibody composition of milk changes during lactation stages and in response to various factors. Its relationship with the development of the neonatal immune system represents a key area of scientific interest. Animal models allow direct study of the relationship between milk antibody composition and neonatal immune development. Murine models, particularly mice, are commonly used for this purpose. Characterizing antibody composition across different lactation stages in these models is essential. While existing literature reports the presence of IgA, IgG, and IgM in mouse milk, the variability in methods (milk extraction techniques, antibody detection protocols), mouse strains, lactation time points, and other factors precludes a comprehensive characterization and necessitates standardization. In this work, we evaluated the presence of IgA and IgG isotypes in colostrum, transitional milk, and mature milk from C57BL/6 mice using sandwich ELISA and western blot. Through the ELISA, we identified the presence of IgA in colostrum. On the other hand, Western blot analysis revealed the presence of IgA in colostrum, transitional milk, and mature milk. It should be noted that the presence of IgA in colostrum was lower than in the other lactation stages. Interestingly, using the same methodology in the same lactation stages, we were unable to identify any IgG isotypes, as have been reported by other authors. The presence of IgG in murine milk may be derived from a traumatic (then serum contamination) extraction procedure.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos B**

Characterization of B cells subsets in a murine model of accelerated aging

Ramírez Quintero, Rubí [1]; De Ita, Marlon [2]; Cisneros, Bulmaro [2];
Santos Argumedo, Leopoldo [1 3].

[1]Departamento de Biomedicina Molecular, Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, Ciudad de México, CDMX, México. [2]Departamento de Genética y Biología Molecular, Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, Ciudad de México, CDMX, México. [3]Centro de investigación sobre el envejecimiento, Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, Ciudad de México, CDMX, México.

E-mail: rubi.ramirez@cinvestav.mx

During aging, there is a gradual decline in both the proportion and function of B lymphocytes, leading to a weakened immune response. This decline is often accompanied by chronic low-grade inflammation, driven by the accumulation of senescent cells and continuous stimulation by antigens, such as self-DNA molecules. It has been proposed that defects in the nuclear envelope proteins may contribute to inflammation during aging. Progerin, a mutated isoform of nuclear Lamin A that accumulates with age, has been studied as a potential biomarker of aging and for its involvement in processes such as proliferation and autophagy. However, its role in inflammation and the immune system has not yet been fully explored. This study aimed to characterize different subsets of B lymphocytes, including follicular and marginal zone B cells in the spleen, and B1 and B2 lymphocytes in the peritoneal cavity, using a murine model of accelerated aging that expresses progerin, known as Lmna G609G. We have found a decreased percentage of total B cells in both tissues. In addition, we observed an increased proportion of marginal zone B cells in the spleen, as well as B1 cells in the peritoneal cavity, and a significant reduction of B2 cells. These findings suggest that progerin expression may alter B cell homeostasis during aging and contribute to an innate-like immune response by these cells, thereby promoting the chronic inflammation observed in the aging process.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Role of CD43 in erythroid differentiation

Ramírez-Vilchis, Gabriel [1 2]; Carpio-Torres, Samantha [2 3];
Flores-Alcantar, Ángel Francisco [2]; Melchy-Pérez, Erika Isabel [2];
Rojo-León, Verónica [2]; Rosenstein-Azoulay, Yvonne [2].

[1]UNAM, Centro de Ciencias Genómicas, Licenciatura en Ciencias Genómicas, Cuernavaca, Morelos, México. [2]UNAM, Instituto de Biotecnología, Depto. de Medicina Molecular y Bioprocesos, Cuernavaca, Morelos, México. [3]UAEM, Escuela de Estudios Superiores del Jicarero, Jojutla, Morelos, México.

E-mail: gabriel.ramirez.vilchis@gmail.com

Erythropoiesis occurs primarily in the bone marrow (BM) and involves the differentiation of erythroblasts through four stages: proerythroblasts, basophilic erythroblasts, polychromatic erythroblasts, and orthochromatic erythroblasts. Within BM, erythroblastic islands form around a central macrophage, where CD43 sialomucin on erythroblasts interacts with CD169 on macrophages, facilitating cell adhesion and supporting erythropoiesis. Notably, CD43 is expressed very early during erythropoiesis, suggesting its potential role in regulating this process. In this study, we evaluated the role of CD43 in the organization of erythroblastic islands and in erythroid differentiation. We used GIEMSA staining and microscopy to identify organizational differences in erythroblastic islands between WT and CD43-/- mice. Through flow cytometry and differential expression of CD44, CD71, and Ter-119 we quantified the different stages of erythroid differentiation. Additionally, we assessed hematological parameters in peripheral blood to further elucidate the role of CD43 in erythropoiesis. Our findings reveal that CD43 deficiency leads to the accumulation of orthoerythroblasts in both the bone marrow and spleen, along with disorganized erythroblastic islands. Despite this, erythroid cells can still complete differentiation into red blood cells, suggesting that extramedullary erythropoiesis may compensate for impaired bone marrow function. Notably, CD43-/- mice exhibit a 3-fold increase in orthochromatic erythroblasts and reticulocytes in the spleen, while peripheral red blood cell counts are reduced by approximately 1.6-fold, supporting the notion that extramedullary erythropoiesis is upregulated to mitigate the deficiency. We also found in hematology analyses that, even though CD43 absence does not affect either erythocytes or hematocrit counts, it does diminish neutrophils and lymphocytes in peripheral blood. Collectively, this data underscores a novel role for CD43 in hematopoiesis, particularly in erythropoiesis.

Supported by DGAPA/UNAM grant IN222523 and Postdoctoral fellow by SECIHTI 6002103.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Identification of receptors for protoxin Cry1Ac in macrophage plasma membrane proteins

Raya -Malvaez, Bryan José Antonio [1]; Gómez-Méndez, Eduardo [1];
Ilhuicatzi-Alvarado, Damaris [1]; Moreno-Fierros, Leticia [1].

[1]Facultad de Estudios Superiores Iztacala, UBIMED Laboratorio 9, Estado de México, Tlanepantla de Baz, México.

E-mail: bryanraya2011@gmail.com

Cry1Ac protoxin has been widely studied for the ability to induce immune activation, particularly in macrophages; in RAW264.7 cells, it has been demonstrated that pCry1Ac induce the phosphorylation of MAP kinases ERK1/2, p38, and JNK, implicating a receptor-dependent signaling surfaces. Through flow cytometry and confocal microscopy, a specific and saturable binding of pCry1Ac to the cell membrane was observed, followed by its internalization via the clathrin pathway. To identify Cry1Ac binding proteins, immunoprecipitation methods using anti-protoxin antibodies and/or chemical conjugation of the protoxin with Sepharose 4B beads were initially employed, followed by MALDI- TOFF analysis, identifying proteins such as HEAT SHOCK P70, vimentin, α-enolase, and actin. The direct interaction of HSP70 with pCry1Ac was confirmed, and the blockade reduced ERK1 signaling, functionally implicating it in macrophage activation. Additionally, an HSP70-dependent migration of peritoneal macrophages was observed in vivo. Those findings demonstrated cell surface HSP70 as a key component in the Cry1Ac-induced macrophage signaling; however, these approaches also identified multiple additional proteins, difficulting the selection of potential receptors for further evaluation, possibly due to co-precipitation of proteins that are part of complexes associated with the actual receptor, generating indirect interactions. Given this limitation, present work implemented a more specific methodology: after purifying endotoxin-free Cry1Ac and verifying its integrity (130 kDa, 6.13 μg/μL), the protein was biotinylated and coupled to streptavidin magnetic microbeads, and separated by magnetic sorting columns, this way was optimized the specificity through oriented binding expecting to preserve the functionality of the active Cry1Ac binding sites to its receptor. This strategy avoided centrifugation steps and reduced nonspecific interactions caused by mechanical drag, allowing more precise interactions with membrane proteins obtained from RAW264.7. Therefore, better candidates for MAPK-mediated effects beyond HSP70 will be presented.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Distribution and Functional Capacity of Monocytes in Compensated and Decompensated Cirrhosis Patients

Rea-Ortega, Grecia [1 2]; Juárez-Palacios, Jesús [1 2];
Gálvez-Basilio, Edna [2]; García-De la Rosa, Teresa [1 2];
Prieto-Chávez, Jessica [3]; Camargo-Juárez, María [1 2];
Rodríguez-Negrete, Elda [4]; López-Macías, Constantino [1];
Domínguez -López, María [2]; Arriaga -Pizano, Andrea [1].

[1]Medical Research Unit in Immunochemistry, Dr. Bernardo Sepúlveda Gutiérrez Specialty Hospital, Siglo XXI National Medical Center, IMSS. México. [2]Department of Immunology, National School of Biological Sciences, National Polytechnic Institute (IPN), México. [3]Flow Cytometry Laboratory, Instrument Center, Health Research Coordination, National Medical Center Siglo XXI, IMSS (IMSS) México. [4]Gastroenterology Service, Pancreas Clinic, Dr. Bernardo Sepúlveda Gutiérrez Specialty Hospital, Siglo XXI National Medical Center, Mexican Social Security Institute (IMSS), México.

E-mail: greciaf.rea@gmail.com

Cirrhosis is a chronic liver disease characterized by the formation of fibrotic tissue in the liver, which can lead to loss of liver function and death. Cirrhosis is a leading cause of morbidity and mortality worldwide, and its prevalence is increasing due to rising obesity, diabetes, and alcohol consumption. Compensated cirrhosis is defined as the presence of fibrotic tissue in the liver without clinical symptoms, while decompensated cirrhosis is characterized by the presence of clinical symptoms.IL-6 is a proinflammatory cytokine produced in response to inflammation and has been linked to cirrhosis because it is considered a chronic inflammatory disease. The aim of this study was to evaluate the distribution of monocytes and their subpopulations, as well as their IL-6 production, the frequency and absolute numbers of monocytes, the IL-6 production of LPS-stimulated monocytes. Peripheral blood samples were collected by venipuncture of the radial or cubital vein from 14 patients with cirrhosis attending the gastroenterology unit of the CMSXXI IMSS. Patients were divided into two cirrhosis groups (compensatedvs.decompensated). Monocytes and their subpopulations were identified and characterized using intracellular cytokine determination to evaluate IL-6 production and immunophenotyping. To date, eight patients with compensated cirrhosis and six with decompensated cirrhosis have been recruited, under two conditions:(SE)and(LPS). It was observed that the MFI of IL-6 in intermediate and non-classical monocytes presented a significant difference with LPSp= 0.0208, while in variables such as proportion of monocytes, absolute number of monocytes, proportion of monocytes for IL-6+ both in monocytes, and in the classical monocyte subpopulation, there was no statistically significant difference. In patients with compensated cirrhosis, all three monocyte subsets exhibit a robust and homogeneous pro-inflammatory response to LPS stimulation.This suggests their functional capacity in response stimuli preserved in this disease stage. Conversely, patients with decompensated cirrhosis display marked heterogeneity and subtype-specific monocyte immune dysfunction.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Bacterial Extract-Mediated Activation and Immune Training of Mast Cells

Rechy-Ahumada, Miguel [1]; Chacón-Salinas, Rommel [1];
Rodríguez-Cortés, Octavio [2]; Serafín-López, Jeanet [1]; Baltierra-Uribe, Shantal Lizbeth [1]; Flores-Mejía, Raúl [2].

[1]Escuela Nacional de Ciencias Biológicas del Instituto Politécnico Nacional, Laboratorio de Inmunología Molecular II, Ciudad de México, México. [2]Escuela Superior de Medicina del Instituto Politécnico Nacional, Laboratorio de Inflamación y Obesidad, Ciudad de México, México.

E-mail: miguel.rechy.93@gmail.com

Bacterial Extract-Mediated Activation and Immune Training of Mast Cells. Respiratory infections represent the leading cause of morbidity worldwide, and their complications can be potentially fatal. Although most are of viral origin, antibiotics are frequently prescribed, promoting adverse effects and contributing to the rise of antimicrobial resistance. Bacterial extracts are used prophylactically to prevent respiratory infections; in addition to reducing their incidence, they have also been shown to alleviate symptoms in patients with asthma and allergic diseases. Their mechanism of action involves both the adaptive and innate immune systems, and it has been proposed that trained immunity contributes to the observed heterologous protection. A key cell involved in pathogen elimination and allergic responses is the mast cell; however, no studies have evaluated its activation or potential for trained immunity in response to bacterial extracts. The aim of this study was to determine mast cell activation in response to two commercially available bacterial extracts (Broncho-Vaxom and IPI ASAC), and to assess the ability of these cells to modify their response using a trained immunity model. Our results show that neither extract induces mast cell degranulation, suggesting that these stimuli do not activate the classical pathway associated with allergic reactions. In contrast, both extracts promoted the release of IL-6 and IL-13, while TNF-α production was modestly induced only by IPI ASAC. These findings indicate that bacterial extracts support both early and late mast cell activation. Currently, the involvement of TLR2 and TLR4 in this activation is being investigated, along with the implementation of a training model to analyze responses to homologous and heterologous stimuli. Understanding the role of mast cells in bacterial immunotherapy will provide deeper insights into the mechanisms underlying the efficacy of these treatments and may open new avenues for preventing respiratory infections and managing allergic diseases.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Immune infiltrate and genome structure of TNBC sample by scRNA-seq and Hi-C

Rendón-Bautista, Luis [1 3 4]; Stephenson-Gussinye, Aura [1 4];
Blanco-Olais, Eduardo [1 4]; Ruiz-Medina, Blanca [1]; Poot-Hernández,
César [1 3]; Soto, Enrique [2]; Chávarri-Guerra, Yanin [2]; Furlan-Magaril,
Mayra [1]; Licona-Limón, Paula [1].

[1]Cellular Physiology Institute, UNAM, CDMX, México. [2]National Institute of Medical Sciences and Nutrition Salvador Zubirán, CDMX, México. [3]Cellular Physiology Institute, Bioinformatics Unit, UNAM, CDMX, México. [4]Master and Doctoral Program in Biochemical Sciences, UNAM, CDMX, México.

E-mail: lrendon@ifc.unam.mx

The concept of ”Hallmarks of cancer” has evolve in the last years, and has been a comprehensive way to understand the factors and mechanisms promoting carcinogenesis. Identification of cell populations within the tumor and determination of the genome structure in human samples, is important to understand the heterogeneity observed in the clinic and to provide mechanistic insights into the cell transformation. Such type of analysis are currently provided by technologies such as single cell RNA-seq and Hi-C. Particularly, analysis of the tumor immune microenvironment is relevant because of the different mechanisms to inhibit or promote tumor progression, as well as to determine a suitable therapeutic approach. The aim of this study was to characterize the cellular composition and the 3D genome structure of a TNBC sample. After QC, we obtained 5, 201 cells by scRNA-seq and 336 million valid paired reads by Hi-C. Using canonical markers, we identified two separate cellular components, eight clusters of-non hematopoietic cells (CD45-), and five clusters of hematopoietic cells (CD45+).Within the immune compartment, we identified myeloid, B and NKT/T cells. Particularly, one of the NKT/T cells expressed FOXP3, characteristic of regulatory T cells and increased expression of CTLA4, ICOS, LAIR2 and TIGIT, suggesting an exhausted phenotype in these cells and supporting the potential use of immunotherapeutic strategies. Regarding the Hi-C results, 87 structural variants were found in the tumor sample, many of them were validated by their expression at the single cell level, for example the gene ENO1, with a gene duplication leading to transcript overexpression compared to the control adjacent tissue. The characterization of tumor samples by these two powerful techniques will help to understand the mechanisms that promote cancer and the identification of potential biomarkers that guide the diagnosis and treatments toward personalized medicine.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

The NLRP3-Interleukin 15 Axis Transiently Regulates the Resistance in Oral Cavity Carcinogenesis

Reyes, José Luis [1]; Quintero-Hernández, Jorge [1];
González, Marisol Ibet [1]; Macedo-Hernández, Mariano [1];
Mateos-Arenas, Diego [1]; Ortiz-Sánchez, Betsaida [2];
Vannan, Danielle [3]; Eksteen, Bertus [4].

[1]Laboratorio de Inmunología e Inflamación Hepato-Intestinal, UBIMED, FES Iztacala, UNAM, Tlalnepantla de Baz, Estado de México, México. [2]Carrera de Cirujano Dentista, FES Iztacala, UNAM, Tlalnepantla de Baz, Estado de México, México. [3]Aspen Woods Clinic, Calgary, AB, Canada. [4]Boston Scientific Corporation, Urology Division, 200 Boston Scientific Way, Marlborough, MA, USA.

E-mail: jlreyes@iztacala.unam.mx

The role of the sensor NLRP3 during oral carcinogenesis has been addressed in cell lines mostly. We and others have reported that the lack of NLRP3 results in enhanced interleukin (IL)-15 production. Thus, we explored the role of NLRP3-IL-15 axis on chemically-induced oral carcinogenesis in the rodent model. To induce oral carcinogenesis, we employed the administration of 4-nitroquinolone-1-oxide (4NQO) in the drinking water of mice for 16 weeks (induction stage) and then mice were maintained drinking water without 4NQO for additional 8 weeks (development stage).We observed that wild-type (WT) mice presented tongue leukoplakia at 16 weeks of cancer induction, in contrast, NLRP3 deficient (NLRP3-/-) mice exhibited only mild leukoplakia, showing a significant protection. The observed resistance was accompanied by increased circulating IL-15 levels in NLRP3-/- mice during the induction stage. At 24 weeks, tumors in the tongue were evident in WT animals, interestingly, comparable tumors were observed in NLRP3-/- mice, suggesting that the resistance against oral carcinogenesis is not further sustained. In line with this, the lack of NLRP3 attenuated the lessions in oesophagus at 16 weeks, but similar tumors were observed at the end of the protocol (24 weeks). Thus, although NLRP3 drives migration and proliferation in oral cancer cell lines, the in vivo protective role seems not to be sustained during the distinct stages of oral cancer.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Mycobacterium tuberculosis alters cytoskeletal dynamics and Induces ROS and NOS in hepatocytes

Reyes-Martínez, Cyntia Denisse [1]; Garduño-Espinoza, Maried [1];
Baltierra-Uribe, Shantal Lizbeth [2]; Romero-Ramírez, Héctor [2]; García-Pérez, Blanca Estela [2]; Castrejón-Jiménez, Nayeli Shantal [1 2]; Hernández-González, Juan Carlos [1 2].

[1]Instituto de Ciencias Agropecuarias, Área Académica de Medicina Veterinaria y Zootecnia, Universidad Autónoma del Estado de Hidalgo, Av. Universidad No. 133. Col. San Miguel Huatengo, Santiago Tulantepec de Lugo Guerrero, Hidalgo, México, C.P. 43775.
[2]Departamento de Microbiología General, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Prolongación de Carpio y Plan de Ayala s/n, Ciudad de México 11340, México.

E-mail: re464224@uaeh.edu.mx

Mycobacterium tuberculosis remains one of the leading threats to global public health, especially with the rise of multidrug-resistant strains. In 2022, the World Health Organization (WHO) reported more than 10 million cases of tuberculosis, including those with extrapulmonary manifestations, such as liver tuberculosis. This form of tuberculosis, although rare, is clinically significant due to its potential impact on liver function and its difficult diagnosis, which is often confused with chronic liver diseases. Liver tuberculosis, as an extrapulmonary manifestation, is of particular interest because it can complicate disease management and affect patient prognosis. Therefore, it is important to understand the underlying mechanisms of liver tuberculosis in order to understand the immune response mechanisms involved in this type of infection. This work aimed to evaluate the infection of HepG2 cells by 1, the expression of reactive oxygen species (ROS) and nitrogen ions (NOS), and the cytoskeleton changes during hepatocyte infection. Cell monolayers were cultured and infected at different incubation times (2, 6, 24, 48, and 72 h). Colony-forming units (CFU) were quantified, and ROS and NOS levels were measured. The results showed that HepG2 hepatocytes are susceptible to M. tuberculosis infection. The mycobacterial bacillus persisted in the cells for up to 72 hours. However, no significant increase in ROS and NOS was observed in response to infection. Despite this, a cytoskeleton reorganization was evident, suggesting that M. tuberculosis could modulate the respiratory burst response to facilitate its persistence in this cell line.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Characterization of mast cells of the Lmna G609G progeric mouse

Reyna-Varela, Oscar Yoali [1]; Ibarra-Sánchez, Alfredo [1 2];
de Ita-Ley, Marlon [3]; Cisneros-Vega, Bulmaro [3];
González-Espinosa, Claudia [1 2].

[1]Centro de Investigación sobre el Envejecimiento, Cinvestav, Sede Sur, Ciudad de México, México.. [2]Departamento de Farmacobiología Cinvestav, Sede Sur, Ciudad de México, México.. [3]Departamento de Genética y Biología Molecular, Cinvestav, Zacatenco, Ciudad de México, México.

E-mail: oscar.reyna@cinvestav.mx

Mast cells (MC), known for their role in the inflammatory response and remodeling of tissue microenvironment, could participate in defective inflammatory reactions observed in Hutchinson-Gilford progeria syndrome (HGPS), characterized by structural deterioration and premature aging. The Lmna G609G murine model reproduces many of the clinical and cellular aspects of the human disease, allowing the underlying cellular mechanisms to be studied. While cardiovascular and muscular manifestations of progeria have been extensively investigated, their impact on immune cell function (particularly MC) remains poorly understood. This study aimed to characterize the morphological and functional alterations of MC in the Lmna G609G murine model, focusing on their capacity to respond to stimuli mediated by the high-affinity IgE (FcεRI) and Toll-like receptor 4 (TLR4). Bone marrow-derived mast cells (BMMCs) from WT and Lmna G609G mice were characterized by performing functional and morphological analyses, including FcεRI- and TLR4-induced secretion of inflammatory mediators such as Tumor Necrosis Factor (TNF), Interleukin-6 (IL-6), Vascular Endothelial Growth Factor (VEGF), and the chemokine CCL-2. Additionally, senescence markers such as senescence-associated β-galactosidase activity were detected. Ultrastructural analysis was performed by transmission and scanning electron microscopy. Preliminary findings revealed significant alterations in morphology and function of Lmna G609G-derived MC compared to controls. Changes in degranulation patterns and inflammatory mediator release were also observed, suggesting impaired innate immune responses in this laminopathy model. These findings provide novel insights into the immunological consequences of lamin A/C defects and may help explain the inflammatory phenomena associated with progeria.

Authors thank the National Laboratory of Surfaces and Structures (LaNSE) for granting access to their SEM and TEM facilities. Murine models essential for BMMC isolation were provided through project 0314-20. We acknowledge Dr. Cisneros and his research team for their valuable collaboration and guidance.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

The role of TGF-beta induced TIF1-gamma and Smad4 on regulatory T lymphocytes within the tumor microenvironment.

Ríos-Ramírez, Diana Berenice [1 2]; Marcial-Medina, Cleofas [1];
Ramos-Balderas, José Luis [1]; Ortiz-Navarrete, Francisco Vianney [2];
Licona-Limón, Paula [1].

[1]IFC-UNAM, Biología del desarrollo, Ciudad Universitaria, CdMx, México. [2]Cinvestav, Biomedicina molecular, Zacatenco, CdMx, México.

E-mail: diana.rios@cinvestav.mx

The role of TGF-beta induced TIF1-gamma and Smad4 on regulatory T lymphocytes within the tumor microenvironment. TGF-β induces the proliferation and differentiation of regulatory T lymphocytes within the tumor microenvironment, resulting in the inhibition of effector lymphocytes. TGF-β signaling is mediated by a protein complex called Smad2/3/4 that translocates to the nucleus and induces gene expression. The transcription factor TIF1-γ can interact with the Smad2/3 complex changing the outcome of gene induction and resulting in a different transcription profile and phenotype. Here, we aim to characterize the role of TIF1-γ and Smad4 in TGF-β-induced tumor-specific regulatory T cells. We generated a transgenic mouse strain with antigen-specific regulatory T lymphocytes. In a model of melanoma, we found that tumor-infiltrating regulatory T lymphocytes express less TIF1-γ than SMAD4. We then isolated the regulatory T lymphocytes and deleted SMAD4 and TIF1-γ with CRISPR/Cas9. Functionality assays of edited lymphocytes showed that Smad4-deficient cells maintain their stability and suppressive capacity compared to TIF1-γ-deficient cells. The SMAD4- or TIF1-γ-deficient regulatory T lymphocytes were adoptively transferred into mice with melanoma tumors with a positive impact on tumor growth. These results position SMAD4 and TIF1-γ as therapeutic targets against cancer.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Optimized IR-fluorophore platform for autoantibody/biomarker detection in TDM1 and LADA

Rivas-Saucedo, Estefanía [1]; Medina-Sosa, Kenia [1];
Lara-Ramírez, Edgar [3]; Rivas-Santiago, Bruno [2]; De Jesús-González, Luis Adrián [2]; Castañeda-Delgado, Julio Enrique [4].

[1]Universidad Autónoma de Zacatecas, Unidad Académica de Ciencias Químicas, Zacatecas, México. [2]Unidad de Investigación Biomédica de Zacatecas, Instituto Mexicano del Seguro Social, Zacatecas, México. [3]Instituto Politécnico Nacional. Centro de Biotecnología Genómica, Laboratorio de Biotecnología Farmacéutica. Tamaulipas, México. [4]Investigadores por México, Secihti, Secretaría de ciencias, humanidades, tecnología e innovación, México.

E-mail: estefaniarivas34@gmail.com

Type 1 diabetes mellitus (T1DM) and latent autoimmune diabetes of the adult (LADA) are diseases characterized by the presence of autoantibodies against proteins secreted by pancreatic beta cells, such as GAD65. In Mexico, the differential diagnosis between T1DM, type 2 diabetes mellitus (T2DM), and LADA, as well as the epidemiology of the latter, is limited by the high cost and low availability of autoantibody detection tests. Clinical practice guidelines prioritize the quantification of plasma glucose, C-peptide, and glycosylated hemoglobin, leaving autoantibodies out of the diagnosis. There are still gaps in our knowledge of the prevalence of LADA and regarding the late detection of T1DM in young adults. Therefore, it is necessary to develop accessible techniques that allow the detection of these biomarkers and improve the distinction of these types of diabetes. To develop a human biomarker detection platform using infrared fluorochrome-labeled antibodies, allowing the identification of both infectious and autoimmune diseases. Model proteins such as human IgG (Kedrigamma), Staphylococcus aureus protein A, and the SARS-CoV-2 spike protein obtained from the supernatant of infected cells were used. These proteins were printed on a nitrocellulose membrane using the Bio-Dot ZX1010 system. Detection was performed with primary antibodies and sera from patients with SARS-CoV-2, rheumatoid arthritis (RA), and healthy subjects, along with the secondary antibody IRDye Goat anti-Human 800CW. Visualization was performed on the Odyssey CLx system, and band quantification was performed using Image Studio v6.0.0.28 software. A detection prototype was standardized, optimizing the concentration of proteins and antibodies. We were able to detect the proteins, successfully distinguishing positive and negative samples for SARS-CoV-2. Based on this, we generated a prototype for detecting antibodies against GAD65, PTPRN2, and insulin. The developed system enables the semiquantitative detection of biomarkers in humans using infrared fluorochrome-labeled antibodies. It is low-cost, highly sensitive, and has potential for diagnostic applications.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Evaluation of Microorganism Derived Adjuvants in induction of Trained Immunity in BMDCs

Rivera Delgado, Jorge Luis [1 2]; Tepale Segura, Araceli [2];
Bonifaz Alfonso, Laura C . [3]; López-Macias, Constantino [2].

[1]Facultad de Química, Universidad Nacional Autónoma de México, México City, México. [2]Unidad de Investigación Médica en Inmunoquímica, Unidad Médica de Alta Especialidad (UMAE) Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México. [3]Coordinación de Investigación en Salud, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México.

E-mail: riveraj859@gmail.com

One of the main differences between the innate and adaptive immune response, is the capability of the last one to generate memory. However, recently researches have established that innate immune cells can also develop a memory-like response known as trained immunity, denoted by enhancing a more effective response after a previous stimulation with PRRs ligands such as β-glucan and BCG. This response is due to epigenetic and metabolic mechanisms, resulting in an increased production of proinflammatory cytokines (IL-1β, IL-6 and TNFα) when cells are challenged with microbial stimuli, either identical or different from the initial trigger. These changes can be observed in completely differentiated cells, such as, monocytes and macrophages and their precursors in bone marrow (BM). This way, trained immunity can be classified in peripheral and central respectively.

Dendritic cells (DCs) are considered the link between innate and adaptive responses due to their role in antigen presentation. It has been demonstrated that DCs can be trained using microbial stimuli, such as C. neoformans, and adjuvants, such as CTB (Cholera B Subunit). In this study, we evaluate the ability of Salmonella typhi porins (OmpC and OmpF) to induce trained immunity in vivo. C57BL/6 mice were immunized intradermally with porins, and BM cells were harvested for DCs differentiation and characterization of their precursors (pre-DCs) for analysis using spectral flow cytometry to determine proinflammatory cytokine and costimulatory molecule production. The results showed an increased percentage of pre-DCs and increased expression of TNFα in BMDCs, alongside increased expression of CD86 and CD40, which improves their function as antigen-presenting cells. This demonstrates that porin administration promotes central and peripheral trained immunity.

These findings could provide support in the development of new and improved vaccination schemes, since porins could be used as adjuvants in the establishment of an effective long-lasting immune response against infectious diseases.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Humoral immune proteins in the mucus of two evolutionarily basal marine animals.

Rivera-Ortega, Jacqueline [1 2]; Sánchez-Salgado, José Luis [3];
Thomé, Patricia E. [2].

[1]Posgrado en Ciencias del Mar y Limnología, Universidad Nacional Autónoma de México, Ciudad de México, México. [2]Laboratorio de Microbiología Molecular, Unidad Académica de Sistemas Arrecifales Puerto Morelos, Instituto de Ciencias del Mar y Limnología, Universidad Nacional Autónoma de México, Puerto Morelos, Quintana Roo, México. [3]Laboratorio Mixto Internacional Gly-CRRET UPEC-UNAM, Departamento de Bioquímica, Facultad de Medicina, Universidad Nacional Autónoma de México, Ciudad de México, México.

[4]E-mail: jacq.rior@gmail.com

Cnidarians are evolutionarily basal animals with barely explored immune mechanisms. The Phylum Cnidaria comprises hard and soft corals, anemones, jellyfish, and hydrozoans. Mucus is an external layer that presents immune properties and protects the organism from the external environment. It first appeared in the history of the planet Earth in the organisms of the Phylum Cnidaria. Thus, the study could help understand the mucosal immunity in other organisms. Here, we determined the phenoloxidase reaction and lectin-like activity in basal conditions. Hard coral Montastraea cavernosa colonies were sampled at Puerto Morelos Reef National Park, Mexico. Anemone Exaiptasia diaphana was collected from the tank facilities at Reef Systems Academic Unit, Puerto Morelos, UNAM, and acclimatised to laboratory conditions. M. cavernosa and E. diaphana showed an enzymatic reaction with all substrates (tyrosine, levodopa, dopamine and hydroquinone). Hence, mucus from both cnidarians has tyrosinase, catechol oxidase, and laccase-type phenoloxidases. On the other hand, M. cavernosa showed strong agglutination activity with A+, B+ and O+ erythrocytes. Conversely, E. diaphana showed low agglutination activity only with A+ erythrocytes. In conclusion, the mucus of M. cavernosa and E. diaphana contains proteins with immunological activity, such as phenoloxidases and lectins, that help ensure the organisms' preservation and survival.

We acknowledge Dr MA Pereyra's help standardising the lectin protocol, Dr E Zenteno's support for the project, R Del Río Arriaga, G Ordoñez-Ruiz and C Pelcastre de la Cruz's diving support, and blood donor volunteers. This project is part of JRO's PhD thesis (SECIHTI, CVU: 747651) and funded by DGAPA-PAPIIT Project IN201323.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Effect of amino acids on adult and neonatal CD4+ T cell responses

Robles-Reyes, Jonathan Ricardo [1]; Rodríguez-Jorge, Otoniel [1];
Carreón-Cortés, Ingrid Yaritzit [1].

[1]Centro de Investigación en Dinámica Celular (UAEM), Laboratorio de inmunología celular y de sistemas, Cuernavaca, Morelos, México.

E-mail: jonathan.robles@uaem.edu.mx

Neonates have a high mortality and morbidity rate. One of the leading causes of neonatal death is their high susceptibility to infections. CD4+ T cells are one of the main infection-fighting arms of the immune system. However, the immune response of neonatal CD4+ T cells is characterized by a tolerogenic bias, highlighting the need to study the neonatal immune response and seek strategies to enhance the response. Naïve CD4+ T cells have a mainly catabolic program based on the oxidation of available nutrients and OXPHOS for ATP synthesis. Naïve T cells are activated when they recognize their antigen through the TCR along with costimulatory signals, such as those from CD28. T cell activation induces metabolic reprogramming, changing to a glycolytic and anabolic metabolism, a metabolism necessary for the synthesis of the biomolecules necessary to fulfill its functions as an effector T lymphocyte. In this new program, some metabolites present in the tissue microenvironment or produced by metabolic reprogramming, like lactate, have immunoregulatory activity, which can affect T cell activation at different levels. Previously, we reported that neonatal T cells' metabolism is characterized by high glycolytic activity and proliferation in basal conditions, which could induce high lactate levels. In this work, we employ logical modeling to describe the influence of metabolism on CD4+ T cell activation and explore the influence of neonatal CD4+ T cell metabolic state on its activation. Through computational simulations and in vitro experiments, we explored the use of Arg or Ser supplementation as a possible strategy to enhance neonatal T cell immune responses. Our results point to an inhibitory role of Arg and Ser on the activation of CD4+ T cells, as they repress the production of most Th cytokines, without affecting the cells' viability. We are currently exploring more scenarios of CD4+ T cell immune stimulation.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Analysis of the Autophagy-Ferroptosis Crosstalk in a Cancer Cell Model

Rocha-González, Mariana [1]; Baltierra-Uribe, Shantal Lizbeth [1];
García-Pérez, Blanca Estela [1].

[1]Department of Microbiology, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional. Prolongación de Carpio y Plan de Ayala, s/n. 11340. México City. México.

E-mail: rocmary26@outlook.com

Maintaining a precise balance between cell survival and cell death is essential for preserving homeostasis in multicellular organisms. Disruption of this balance can lead to various pathological conditions, including cancer, where impaired cell death contributes to uncontrolled proliferation. Ferroptosis is a regulated form of cell death, characterized by iron dependency and the accumulation of lipid peroxides, which damage cellular membranes. Conversely, autophagy is a highly conserved catabolic process that plays a context-dependent dual role in cell fate, either promoting survival or contributing to cell death. Recent evidence suggests that autophagy may modulate susceptibility to ferroptosis. Given that lung cancer remains a leading cause of cancer-related mortality worldwide and that therapeutic resistance to conventional chemotherapies is a persistent challenge, investigating alternative strategies is imperative. This study explores the potential crosstalk between autophagy and ferroptosis in lung cancer cells (A549) treated with ursolic acid, a natural compound previously associated with autophagy induction. Experimental results indicate that ursolic acid significantly reduces A549 cell viability after 24 hours of treatment, suggesting activation of cell death pathways. Notably, pre-treatment with ferrostatin-1, a specific ferroptosis inhibitor, partially restores cell viability, implicating ferroptosis as a potential mechanism of action. Additionally, the use of BODIPY 581/591 C11 staining revealed increased lipid peroxidation in cells exposed to ursolic acid, further supporting ferroptotic involvement. Ongoing experiments aim to delineate the mechanistic interplay between autophagy and ferroptosis in this model. A better understanding of this interaction could contribute to the development of novel therapeutic approaches targeting drug-resistant lung cancers.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

Nitazoxanide shows an immunomodulatory effect in IFN-γ production in Vγ9Vδ2 T cells

Rodríguez-Cortés, Octavio [1]; Campos-Juárez, Ángel Daniel [1] [4]; Aquino-Naranjo, Oscar [1]; Cortés-Serrano, Jonathan Brian [1]; Gómez-Castro, Carlos Zepactonal [2]; Romero-Rodríguez, Damaris Priscila [3]; García-Núñez, Andrés Ademar [4]; Rodríguez-Cadena, Mónica Adriana [4]; Torres-Ávila, Itzel Pamela [4]; Castrejón-Flores, José Luis [4].

[1]Instituto Politécnico Nacional, Escuela Superior de Medicina, Sección de Estudios de Posgrado e Investigación. Ciudad de México, México. [2]CONACYT-Universidad Autónoma del Estado de Hidalgo, Mineral de la Reforma, Hidalgo, México. [3]Instituto Nacional de Enfermedades Respiratorias Laboratorio Nacional Conahcyt de Investigación y Diagnóstico por Inmunocitofluorometría, Ciudad de México, México. [4]Instituto Politécnico Nacional, Unidad Profesional Interdisciplinaria de Biotecnología, Ciudad de México, México.

E-mail: orodriguezc@ipn.mx

γδ T cells are a subset of non-conventional T lymphocytes characterized by their restricted T-cell receptor (TCR) repertoire and capacity to recognize non-peptidic antigens. The predominant γδ T-cell population in human peripheral blood expresses the Vγ9Vδ2 TCR and is typically activated by phosphoantigens (PAgs) through a non-canonical mechanism involving the BTN3A1 protein, particularly its intracellular B30.2 domain. While various synthetic PAgs have been proposed as γδ T-cell activators, no FDA-approved drug has been shown to directly induce their activation. In this study, we performed high-throughput virtual screening using the DrugBank database of FDA-approved compounds. Molecular docking was carried out targeting the B30.2 domain of BTN3A1 (PDB ID: 5ZXK) in complex with HMBPP, a potent natural PAg. Three different scoring methods—CMD, ΔG, and LRCD—were applied to select the best candidate ligands. From these, five drugs (aztreonam, azathioprine, dantrolene, tinidazole, and nitazoxanide [NTZ]) were selected for in vitro evaluation. Cytotoxicity assays using 7-AAD staining in PBMCs from healthy donors showed no reduction in cell viability at 1, 10, or 100 µM. Functional activation was assessed by intracellular IFN-γ staining after 4 h stimulation. Only NTZ (60 µM) significantly increased IFN-γ–producing γδ T cells. This effect was confirmed in PBMC cultures and in co-cultures of monocytes with purified γδ T cells. After 36 h, NTZ also increased HLA-DR expression. Interestingly, co-stimulation with HMBPP and NTZ reduced IFN-γ production compared to HMBPP alone, suggesting competition at the B30.2 binding site. Molecular docking supported this hypothesis, revealing putative interactions between NTZ and the B30.2 domain. These findings reveal nitazoxanide as a γδ T-cell modulator and highlight its potential as a scaffold for novel immunotherapeutic strategies.

Ethics committee approved ESM-CEI-02/16-02-23.

We would like to thank the Central Blood Bank of the Siglo XXI National Medical Center, as well as funding SIP20240536.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Anti-inflammatory activity of Rhus trilobata subbractions in lipopolysaccharide-stimulated J774A.1 macrophages.

Rodríguez-Guerrero, David [1]; Sánchez-Ramírez, Blanca Estela [1];
Mendoza-Chacón, Johan [1]; Infante -Ramírez, María del Rocío [1];
Siqueiros-Cendón, Tania Samanta [1]; González-Horta, María del Carmen [1].

[1]Facultad de Ciencias Químicas. Universidad Autónoma de Chihuahua. Chihuahua, Chihuahua, México.

E-mail: a345673@uach.mx

Inflammation is the body's response to focal aggression that can be caused by microorganisms, external agents or autoimmune diseases. Its persistence can contribute to the development of chronic diseases such as cancer, diabetes and cardiovascular diseases, which cause millions of deaths annually. Macrophages are involved because they are responsible for the recognition of pathogens and the control of inflammation. Nonsteroidal anti-inflammatory drugs are compounds, some isolated from plants, that are the conventional treatment for these diseases. Fraction 6 (F6) of the aqueous extract (EA) of Rhus trilobata (Rt) has been reported to possess compounds with anti-inflammatory activity. The objective of this study was to identify a subfraction of F6 capable of decreasing the expression of COX-2, TNF-α and IL-1β mRNAs in J774A.1 BMs stimulated with lipopolysaccharide (LPS) in vitro. Subfractions (S1F6 to S4F6) were obtained using methanol-water ratios, and their cytotoxicity was assessed with resazurin at 15 μg/mL at 24 h. Gene expression was analyzed by end-point RT-PCR. Unstimulated, LPS-stimulated, and dexamethasone-treated (10 μM) cultures were used as controls. Subfraction S3F6 showed the highest yield (54.22%) and did not present significant cytotoxicity. All subfractions decreased the expression of the analyzed genes; however, S3F6 achieved a significant reduction of all three, suggesting a higher concentration of active or synergistic compounds.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

MIF Deficiency Alters Gut Microbiota and Contributes to Colitis-Associated Colorectal Cancer Development

Rodríguez-Hernández, Mario Alberto Tonathiu; Juárez-Avelar, Imelda; Rodríguez-Sosa, Miriam.

[1]Facultad de Estudios Superiores-Iztacala (FES-I), Universidad Nacional Autónoma de México (UNAM), Unidad de Investigación en Biomedicina, Laboratorio de Inmunidad Innata, Tlalnepantla, Edo. México.

E-mail: tonathiurh@gmail.com

Colitis-associated colorectal cancer (CAC) is a serious complication of inflammatory bowel diseases (IBD), where dysbiosis and immune activation play critical roles. The cytokine macrophage migration inhibitory factor (MIF) is a key regulator of innate immunity and Toll-like receptor (TLR) expression. In a murine CAC model induced by AOM/DSS, we previously observed that MIF^-/- mice develop increased tumor burden, potentially due to defective immune responses. In this study, we investigated the impact of MIF deficiency on gut microbial composition and its contribution to CAC development using 16S rRNA sequencing and fecal microbiota transplantation (FMT). Naïve MIF-/- mice exhibited significant microbiota alterations compared to wild-type (WT), including an increased abundance of Firmicutes, Prevotellaceae, and Helicobacteraceae, and a reduction in Bacteroidaceae. FMT from MIF-/- into antibiotic-treated WT mice exacerbated weight loss after DSS exposure, shortened colon length, and increased tumor volume, compared to WT recipients of WT microbiota. Taxonomic analysis revealed that these microbial changes persisted during both acute inflammation (day 10) and tumor progression (day 68), highlighting the enrichment of pro-inflammatory bacteria such as Prevotella, Desulfovibrio, and Lactobacillus in recipients of MIF-/- microbiota. Our results indicate that MIF deficiency drives a pro-tumorigenic microbiota that exacerbates CAC development. This suggests that MIF plays a critical role in modulating the inflammation–microbiota–tumor axis in the colon. Understanding this interplay could offer novel insights into immune-microbiota crosstalk and its impact on colorectal carcinogenesis.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Area del artículo: **Inmunometabolismo**

Metabolic regulation of neonatal T cell activation and its therapeutic implications.

Rodríguez-Jorge, Otoniel [1]; Castañeda-Gómez, Francisco [1];
Carreón-Cortés, Ingrid Yaritzit [1]; Robles Reyes, Jonathan Ricardo [1];
Kempis-Calanis, Linda Aimara [1].

[1]Universidad Autónoma del Estado de Morelos. Centro de Investigación en Dinámica Celular.
Laboratorio de Inmunología Celular y de Sistemas. Cuernavaca, Morelos, México.

E-mail: orj@uaem.mx

T cells are central players in the adaptive immune response, requiring metabolic reprogramming upon activation. During this transition, both CD4^{+} and CD8^{+} T cells shift from a quiescent state to a highly glycolytic metabolism, commonly referred to as the Warburg effect, to meet the increased demands for energy and biosynthetic precursors. However, neonatal CD4^{+} T cells exhibit impaired activation and diminished effector function. These cells paradoxically overexpress genes associated with glycolysis while underexpressing key activation-related genes, suggesting a dysregulated metabolic-activation axis. This study aimed to dissect the role of major metabolic pathways, including glycolysis, oxidative phosphorylation, and β-oxidation, in the activation of neonatal CD4^{+} T cells. To this end, we constructed computational models integrating T cell activation networks with metabolic pathways. These models were informed by experimental data, including RNA-seq-based transcriptomic profiles and functional nutrient uptake assays using fluorescent analogs. Using in silico simulations, we investigated how the modulation of specific metabolic pathways affects activation outcomes in neonatal T cells. Experimentally, CD4^{+} T cells were stimulated in vitro with arginine, flagellin, or cytokines, yielding varied activation responses and offering insights into potential strategies for metabolic and signaling stimulation. Our findings suggest that targeted manipulation of signaling and metabolic circuits can overcome neonatal T cell hypo-responsiveness. Understanding and enhancing the metabolic-immune interface in neonatal T cells holds significant potential for improving early-life immunity, particularly in the context of infections and vaccine efficacy.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

Alterations in Immune Cell Populations in the Blood of Transgenic Mice with Alzheimer's-Like Pathology and Cerebral Amyloid Angiopathy (Tg-SwDI).

Rodriguez-Lopez, Gilberto Adrian [1] ; Gevorgyan, Goar [1].

[1]Instituto de Investigaciones Biomédicas, Departamento de Inmunología, UNAM, Ciudad de México, CDMX, México.

E-mail: lta556@gmail.com

Alzheimer's disease (AD) is a chronic and progressive neurodegenerative disorder, and the leading cause of dementia worldwide. Its strong association with cerebral amyloid angiopathy (CAA) has highlighted the importance of investigating the mechanisms driving neuroinflammatory progression when both conditions coexist. AD and CAA are characterized by the accumulation of beta-amyloid (Aβ) aggregates in the brain parenchyma and vasculature, respectively. Multiple studies have reported that dysfunction in the communication between the central nervous system (CNS) and the peripheral immune system, as well as infiltration of immune cells from the blood into the CNS, are associated with changes in the composition and function of circulating immune cells, in patients and in murine models of AD. In this study, we conducted a longitudinal analysis of immune cell populations in the peripheral blood of male and female Tg-SwDI transgenic mice, a model that develops Aβ deposits in both the brain parenchyma and cerebral vasculature. We observed a reduced proportion of T lymphocytes and an increased proportion of neutrophils in both sexes compared to age-matched wild-type (WT) controls. Additionally, an inversion of the CD4/CD8 ratio was detected in Tg-SwDI mice relative to WT counterparts. Our findings suggest that the neuropathology observed in the Tg-SwDI model is associated with early alterations in peripheral immune cell populations, which may reflect systemic inflammatory mechanisms contributing to the progression of Alzheimer's-like pathology

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Sex differences of immunological response in the induction of colorectal tumors

Rodríguez-Santiago, Yair; Garay-Canales, Claudia Angélica; Del Rio-Araiza, Víctor Hugo; Ruiz-Antonio, Diana Lizeth; Ríos-Avila, María del Sol; Zavala-López, César Antonio; Morales-Montor, Jorge.

[1]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, UNAM, CDMX, México. [2]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, UNAM, CDMX, México. [3]Departamento de Parasitología, Facultad de Veterinaria y Zootecnia, UNAM, CDMX, México. [4]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, UNAM, CDMX, México. [5]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, UNAM, CDMX, México Departamento de Inmunología, Instituto de Investigaciones Biomédicas, UNAM, CDMX, México. [6]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, UNAM, CDMX, México. [7]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, UNAM, CDMX, México.

E-mail: ryai791@gmail.com

Colorectal cancer (CRC) shows higher incidence and mortality in men than in women, both in humans and in experimental models. This difference has been linked to the influence of sex hormones. While females display more robust immune responses, males tend to develop more immunosuppressive tumor microenvironments. The aim of this study was to compare systemic and tumor immune responses between male and female mice induced with colorectal tumors. Balb/c mice treated with AOM/DSS were used to induce colorectal tumors. After 70 days, tumor, mesenteric lymph node, and spleen samples were collected. Immune populations were evaluated by flow cytometry, including T lymphocytes ($CD3^+$, $CD4^+$, $CD8^+$, γδ, FOXP3+), B cells ($CD19^+$), plasma cells ($CD138^+$), macrophages, NK cells, and mast cells. In the tumor, males exhibited significantly higher infiltration of γδ T and B cells, along with a reduction in total T lymphocytes and plasma cells. In females, $CD3^+$, $CD4^+$, $CD8^+$, and γδ T lymphocytes were markedly reduced, while plasma cells were preserved. In the mesenteric lymph nodes, macrophages increased in males and NK cells decreased, while both sexes showed reductions in B lymphocytes and plasma cells. In the spleen, an increase in macrophages was detected in females and in mast cells in both sexes. These findings reflect an immune profile with clear sex-related differences both systemically and in the tumor. This highlights the relevance of sex as a biological variable in colorectal cancer and underscores the importance of considering sex as an essential variable in the search for treatments.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Differential Effects of Agave Fructans on Inflammation, Pruritus and Gut Microbiota in Rats

Rodriguez-Villalpando, Kenya Nallely [1]; Fuentes-de Anda, Evelyn Danae [1]; Cervantes-García, Daniel [1 2]; Jiménez -Vargas, Mariela [1]; Verduzco-Grajeda, Lidia Elizabeth [3]; Córdova-Dávalos, Laura E [1]; Enríquez-Medrano, Francisco Javier [4]; Fabela-Sánchez, Omar [2 4]; Salinas-Miralles, Eva [1].

[1]Department of Microbiology, Basic Science Center, Autonomous University of Aguascalientes, México. [2]Secretariat of Science, Humanities, Technologies and Innovation, México City, México. [3]Technological University of Northern Aguascalientes, Rincón de Romos, México. [4]Center for Research in Applied Chemistry, Saltillo, México.

E-mail: kenyardz0302@gmail.com

Agave genus plants produce fructans as polysaccharide reservoirs, which consist of repeated fructose units linked by $\beta(2\rightarrow1)$ and $\beta(2\rightarrow6)$ bonds, with an internal glucose, and branched structure. Agave fructans are not digested by human enzymes but fermented by intestinal bacteria, and then they have prebiotic properties promoting beneficial health effects. The prebiotic potential depends on their degree of polymerization (DP), which varies according to geographic location and age of the source plant. There is growing interest in describing new health applications of agave fructans, driven by their economic and agronomic relevance in Mexico. This study aimed to analyze the effect of oral administration of fructans from Agave tequilana Weber var. azul with different DP on rat with acute inflammation and pruritus, and on gut microbiota. Commercial agave fructans with DP of 11 (AF1) or 17 (AF2) were used. Rats were orally treated with fructans (0.1 g/kg weight, dissolved in water) for 11 days, fecal samples were collected, and relative abundance of main bacterial phyla and genera was analyzed. On day 11, animals were intradermally injected with C48/80 to induce skin inflammatory reactions or with 5-HT to stimulate pruritus. Fructans intake significantly reduced scratching and grooming behaviors in animals, and the effect was markedly diminished with the lowest DP fructans. None of fructan treatments altered the diameter of the inflamed area, or the extent of extravasation induced by C48/80 in rats. Regarding intestinal bacterial phyla, both fructans significantly reduced the relative abundance of Proteobacteria, while AF2 increased Firmicutes. Genera-level analysis showed that both AF1 and AF2 promoted the proliferation of Lactobacillus, with AF2 inducing a 5.4-fold greater increase than AF1. However, only AF1 was able to increase Bifidobacterium levels in the gut. Therefore, agave fructans exert therapeutic effects on experimental acute pruritus by modulating gut microbiota in a DP-dependent manner.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Coagulation Times as Predictors of Mortality in Sepsis

Rojas-Guillen, Megan Zahori [1]; López-Álvarez, Xuxu Guadalupe [2];
Plata-Ramos, Ivonne Mónica [3]; Castillo-Cruz, Juan [4].

[1]Instituto Politécnico Nacional, Escuela Superior de Enfermería y Obstetricia, Ciudad de México, CDMX, México. [2]Instituto Politécnico Nacional, Escuela Superior de Enfermería y Obstetricia, Ciudad de México, CDMX, México. [3]IMSS, Hospital General Regional No. 72°, Vicente Santos Guajardo, Estado de México, México. [4]Instituto Politécnico Nacional, Escuela Superior de Medicina, Ciudad de México, CDMX, México.

E-mail: mrojasg2002@alumno.ipn.mx

Sepsis is characterized by a dysregulated immune response involving hemodynamic, metabolic, and hematological disturbances. Coagulation times—thrombin time (TT), prothrombin time (PT), and partial thromboplastin time (aPTT)—are closely linked to innate immunity through immunothrombosis. This process may contribute to organ dysfunction and increased mortality.

Objective:

To evaluate the prognostic value of coagulation times and their relationship with immune function and mortality in septic patients, using a multivariate model incorporating base deficit (BD) and fibrinogen.

Materials and Methods:

A retrospective, observational, analytical cohort study was conducted using clinical data from 164 patients (20–75 years old, both sexes) admitted to the emergency department with a diagnosis of sepsis. Patients were selected through systematic random sampling. Serum biomarkers—BD, TT, PT, aPTT, and fibrinogen—were measured every 24 hours for up to 72 hours from admission. The primary outcome was in-hospital mortality. A multivariate logistic regression model was applied.

Results:

Thrombin and prothrombin times significantly increased during the first 46 hours ($p < 0.05$), while fibrinogen levels significantly decreased at 72 hours. The multivariate analysis showed: Base Deficit: $\beta = 0.067$, OR = 0.93, 95% CI: 0.887–0.986, $p = 0.013$, indicating that each unit increase in BD reduces mortality risk by 7%. Fibrinogen: $\beta = 0.997$, OR = 0.002, 95% CI: 0.997–1.000, suggesting that lower fibrinogen levels are associated with increased mortality risk. The model's ROC curve showed moderate discriminatory ability (AUC = 0.648, $p = 0.002$, 95% CI: 0.561–0.734).

Conclusions: Early changes in coagulation times and fibrinogen levels are associated with immune dysfunction and mortality in sepsis. BD appears protective, while decreased fibrinogen increases risk. These biomarkers may support early risk stratification and improve clinical prognosis in septic patients.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Temporal transcriptomic metaanalysis of peripheral blood in early Ischemic Stroke

Rojas-Gutiérrez, Santiago Emilio [1]; Martínez-Canales, Rodolfo [1];
Tienda-López, Fernando [2]; Salinas-Carmona, Mario Cesar [1];
Góngora Rivera, Juan Fernando [2]; Macías-Segura, Noé [1].

[1]Servicio y Departamento de Inmunología, Hospital Universitario “Dr. Jose Eleuterio Gonzalez”, Universidad Autónoma de Nuevo Leon, Monterrey, Nuevo Leon, Mexico. [2]Departamento de Neurología, Hospital Universitario “Dr. Jose Eleuterio Gonzalez”, Universidad Autónoma de Nuevo Leon, Monterrey, Nuevo Leon, Mexico.

E-mail: santiago.rojasgu@uanl.edu.mx

Ischemic stroke triggers a systemic immune response that evolves dynamically over time. To characterize transcriptomic kinetics in peripheral blood during the first 48 hours post-stroke, we performed an integrative meta-analysis of gene expression datasets. Microarray datasets GSE58294 and GSE16561 were analyzed for the <24h window, GSE37587 for the 24–48h period, and GSE22255 as a chronic-phase reference. Additionally, we incorporated next-generation sequencing (NGS) data of exosomal miRNAs (GSE202709, GSE186844, GSE255087) and mRNA (GSE202518). Differentially expressed genes (DEGs) were identified using limma (for microarray) and DESeq2 (for NGS), with adjusted $p < 0.05$ and log□ fold change > 1 or < 1. Immune cell composition was inferred using CIBERSORT, and comparisons were made across <24h vs 24–48h, <24h vs chronic phase, and stroke vs healthy controls. GSVA and GSEA were applied for pathway enrichment, and miRNA–mRNA interactions were explored using miRWalk. Comparisons between compositions across studies were made using SPSS v26 and graphs were made using Graph Pad Prism v8, considering statistically significant $p < 0.05$. In total, 290 samples were analyzed. We observed clear temporal shifts in immune cell proportions, including early increases in monocytes and neutrophils. The acute phase showed strong upregulation of inflammatory and metabolic pathways, while later timepoints suggested partial resolution. Among notable findings, we identified a consistent downregulation of CCR7, a key marker of lymphocyte trafficking, during the acute phase. Exosomal miRNAs such as miR-210 and miR-1244-2 were differentially expressed, with predicted targets involved in hypoxia response, apoptosis, and leukocyte signaling.These results offer an integrated view of early immune-transcriptomic dynamics in ischemic stroke and highlight molecular signatures that could guide time-sensitive therapeutic interventions.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Identification of transcriptomic biomarkers in early preeclampsia in systemic lupus erythematosus

Rojas-Gutiérrez, Santiago Emilio [1]; Martínez-Canales, Rodolfo [1];
Pérez-Barbosa, Lorena [2]; Skinner-Taylor, Cassandra Michel [2];
Montoya-Rosales, Alejandra [3]; Salinas-Carmona, Mario Cesar [1];
Macías-Segura, Noé [1].

[1]Servicio y Departamento de Inmunología, Hospital Universitario "Dr. Jose Eleuterio González", Universidad Autónoma de Nuevo León. [2]Departamento de Reumatología, Hospital Universitario "Dr. José Eleuterio González", Universidad Autónoma de Nuevo León. Monterrey, Nuevo León, México. [3]Laboratorio de Inmunomoduladores, Centro de Investigación y Desarrollo en Ciencias de la Salud, Universidad Autónoma de Nuevo León, Monterrey, Nuevo León, México.

E-mail: santiago.rojasgu@uanl.edu.mx

Preeclampsia (PE) in patients with systemic lupus erythematosus (SLE) is a serious obstetric complication with high morbidity. This study aimed to identify differentially expressed genes that could serve as early biomarkers of PE in this population through a transcriptomic meta-analysis. A systematic search was conducted in the GEO database using the terms "preeclampsia" + "Homo sapiens" + "profiling by microarray." Transcriptomic datasets from placenta and peripheral blood samples related to PE (n=6) and SLE (n=2) were included. Genes were filtered based on statistical significance (FDR < 0.05) and expression change magnitude (|log2FC| > 2). Identified genes were analyzed using functional enrichment, co-expression networks, and hub gene detection through five centrality algorithms. A total of 115 commonly dysregulated genes were identified. Key hub genes included THBS1, COL4A3, THBD, PTGS2, and SERPINE1. Some genes such as HLA-DQA1, ZFY, UTY, and COL17A1 were found to overlap between PE and SLE. Independent validation was performed using external datasets (GSE98224, GSE99967, GSE48424), including ROC curve analysis and digital cytometry. Significant correlations were observed between immunological profiles and the presence of PE.

Our findings suggest the existence of a differential transcriptomic signature in blood and placenta that could be used to develop early diagnostic tools for identifying PE risk in SLE patients.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Biomarkers of fetal loss and infertility in rheumatic diseases: An integrative analysis

Rojas-Gutiérrez, Santiago Emilio [1]; Martínez-Canales, Rodolfo [1];
Pérez-Barbosa, Lorena [2]; Skinner-Taylor, Cassandra Michel [2];
Montoya-Rosales, Alejandra [3]; Salinas-Carmona, Mario Cesar [1];
Macías-Segura, Noé [1].

[1]Servicio y Departamento de Inmunología, Hospital Universitario “Dr. José Eleuterio González”, Universidad Autónoma de Nuevo León. Monterrey, Nuevo León, México. [2]Departamento de reumatología, Hospital Universitario “Dr. José Eleuterio González”, Universidad Autónoma de Nuevo León. Monterrey, Nuevo León, México. [3]Laboratorio de Inmunomoduladores, Centro de Investigación y Desarrollo en Ciencias de la Salud, Universidad Autónoma de Nuevo León, Monterrey, Nuevo León, México.

E-mail: santiago.rojasgu@uanl.edu.mx

Fetal loss and infertility are prevalent but poorly understood complications in women with rheumatic diseases. This study aimed to identify transcriptomic biomarkers and immunological pathways involved in spontaneous abortion and infertility associated with antiphospholipid syndrome (APS), systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and Sjögren’s syndrome.

We performed an integrative transcriptomic analysis using datasets from endometrial tissue and immune cells. Data were retrieved from GEO: GSE102215 (neutrophils, APS), GSE26787 (endometrium, spontaneous abortion), and GSE165004 (endometrium, recurrent pregnancy loss [RPL] and unexplained infertility [UIF]). Differentially expressed genes ($FDR < 0.05$, $|\log2FC| > 1$) were identified and analyzed for functional enrichment, immune cell infiltration, and hub gene detection using five centrality algorithms.

In APS and spontaneous abortion datasets, 30 key genes were identified. Notable hub genes included PECAM1, SELL, CD1D, SELPLG, GRB2, STAT2, and WAS. These genes showed strong correlation with immune cell infiltration in the endometrium. Validation in GSE165004 confirmed significant expression and immunological associations.

In SLE, 72 differentially expressed genes were identified. Hub genes included STAT5B, CD38, PRKCB, INPP5D, and IKZF1. In RA, six genes were found with relevance to immune signaling (LAMA4, BCL2L10, FBLN1, etc.). In Sjögren’s syndrome, 24 genes were differentially expressed, highlighting CD38, CXCL13, and PECAM1 as key immunological markers.

This study supports the existence of shared and disease-specific transcriptomic signatures linked to impaired fertility and pregnancy loss in rheumatic diseases, paving the way for early detection and targeted interventions.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Role of Neutrophil Extracellular Traps (NETs) in Inflammation and Thrombosis: New Horizons in Clinical Immunology

Rojas-Salazar, Jorge Gustavo; Rojas-Salazar, Yareli Lizbeth;
Gómez-Montañez, Emiliano.

[1]Universidad Autónoma de Ciudad Juárez, Ciudad Juárez, Chihuahua, México. [2]Instituto Nacional de Medicina Genómica, Ciudad de México, México. [3]Instituto Nacional de Medicina Genómica, Ciudad de México, México.

E-mail: rojas19111@gmail.com

Neutrophil extracellular traps (NETs) are web-like structures composed of DNA and antimicrobial proteins released by neutrophils to trap and neutralize pathogens; and while NETs play a key role in host defense, their excessive or dysregulated formation has been implicated in chronic inflammation and thrombosis, thus, they contribute to tissue damage and clot formation in diseases such as systemic lupus erythematosus (SLE) and deep vein thrombosis (DVT).

This study aims to explore the dual role of NETs in host defense and disease pathogenesis, particularly their involvement in inflammation and thrombosis, and to examine current and future therapeutic strategies aimed at regulating NET formation and degradation.

A systematic literature review was conducted using recent publications from scientific databases, focusing on the biological mechanisms of NET formation, their pathological consequences, and therapeutic interventions targeting NET-associated pathways, with a total of 25 articles to analyze.

NETs interact with vascular endothelium and platelets, promoting inflammation and thrombosis; in which dysregulated NET formation has been observed in autoimmune and thrombotic disorders. Key regulatory elements such as peptidylarginine deiminase 4 (PAD4) and DNases have been identified as potential therapeutic targets; and experimental treatments aiming to modulate NETs are under investigation to prevent tissue damage without impairing innate immunity.

To conclude, NETs represent a critical link between innate immunity, inflammation, and thrombosis; and understanding their regulation opens the door to novel therapies for autoimmune and thrombotic diseases. Future research on NET-targeted strategies may lead to more personalized and safer immunological treatments.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Cytokine Storms and Immune Dysregulation in Severe Viral Infections

Rojas-Salazar, Jorge Gustavo; Rojas-Salazar, Yareli Lizbeth;
Gómez-Montañez, Emiliano.

[1]Universidad Autónoma de Ciudad Juárez, Ciudad Juárez, Chihuahua, México. [2]Instituto Nacional de Medicina Genómica, Ciudad de México, México. [3]Instituto Nacional de Medicina Genómica, Ciudad de México, México.

E-mail: rojas19111@gmail.com

Cytokine storms are excessive and uncontrolled releases of pro-inflammatory cytokines that can occur in severe viral infections such as COVID-19, influenza, and Ebola; and this hyperinflammatory response contributes to tissue damage, organ failure, and high mortality.

This study aims to describe the mechanisms leading to cytokine storms and explore therapeutic strategies aimed at modulating the immune response in severe viral infections.

A review of recent studies and clinical data on cytokine profiles, immune signaling pathways, and immunomodulatory treatments in patients with severe viral diseases was performed, with 34 articles.

Key cytokines involved in cytokine storms include IL-6, TNF-α, and IL-1β. Treatments such as corticosteroids, IL-6 receptor blockers (e.g., tocilizumab), and JAK inhibitors have been employed to dampen the inflammatory response with varying degrees of success.

To conclude, the Cctokine storms represent a maladaptive immune response to viral infection; and targeted immunomodulation offers a strategy to reduce mortality, but must be carefully balanced to avoid impairing viral clearance.

Área del artículo: **Inmunotecnología e inmunoterapia**

Anti-PD-L1 Functionalized Nanoparticles to Enhance Immunotherapy in Resistant Murine Melanoma Models

Rojas-Salazar, Jorge Gustavo; Rojas-Salazar, Yareli Lizbeth;
Gómez-Montañez, Emiliano.

[1]Universidad Autónoma de Ciudad Juárez, Ciudad Juárez, Chihuahua, México. [2]Instituto Nacional de Medicina Genómica, Ciudad de México, México.

E-mail: rojas19111@gmail.com

Tumor resistance to immunotherapy remains one of the main challenges in clinical oncology; particularly, the overexpression of PD-L1 in the tumor microenvironment contributes to immune evasion; and immunotechnology enables the design of targeted delivery systems that can enhance immunotherapeutic efficacy.

The aim of this study was to develop and evaluate biocompatible nanoparticles functionalized with anti-PD-L1 monoclonal antibodies to restore the anti-tumor immune response in a murine model of resistant melanoma.

Biodegradable polymeric nanoparticles were synthesized by nanoprecipitation and covalently conjugated with anti-PD-L1 antibodies using EDC/NHS chemistry. A B16-F10 melanoma model was established in C57BL/6 mice, which were divided into four groups: control, plain nanoparticles, free antibody, and anti-PD-L1 functionalized nanoparticles. Tumor burden, CD8+ T-cell infiltration, and PD-L1 expression were evaluated using flow cytometry and RT-PCR.

Results showed that mice treated with functionalized nanoparticles exhibited a significant reduction in tumor volume ($p<0.01$) compared to the control and free antibody groups. A marked increase in CD8+ cytotoxic T-cell infiltration and a decrease in PD-L1 expression in tumor tissues were observed, indicating a sustained restoration of immune activity.

In conclusion, anti-PD-L1 functionalized nanoparticles represent a promising strategy to overcome immune resistance in melanoma. This approach combines the specificity of immunotherapy with the efficiency of nanotechnology-based delivery systems, paving the way for more effective treatments in refractory tumors.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Role of the Gut Microbiota in Modulating the Immune Response and Its Clinical Implications

Rojas-Salazar, Yareli Lizbeth; Gómez-Montañez, Emiliano;
Rojas-Salazar, Jorge Gustavo.

[1]Instituto Nacional de Medicina Genómica, Ciudad de México, México. [2]Instituto Nacional de Medicina Genómica, Ciudad de México, México. [3]Universidad Autónoma de Ciudad Juárez, Ciudad Juárez, Chihuahua, México.

E-mail: yarojas177662@gmail.com

The gut microbiota plays a crucial role in regulating the immune system by maintaining a balance between immune tolerance and activation, in which commensal bacteria influence the maturation of antigen-presenting cells and the differentiation of regulatory T cells, which are essential for preventing excessive inflammatory responses. Disruptions in the gut microbiota, known as dysbiosis, have been linked to autoimmune and inflammatory diseases such as inflammatory bowel disease and rheumatoid arthritis.

This study aims to analyze the role of gut microbiota in immune modulation and its clinical implications, and to review emerging therapies targeting microbiota modulation, including probiotics and fecal microbiota transplantation.

We conducted a literature review using scientific databases to select recent and relevant studies focused on gut microbiota–immune system interactions and clinical applications of microbiota modulation.

The gut microbiota regulates immune responses through antigen-presenting cell activation and regulatory T cell differentiation, in which microbial metabolites, such as short-chain fatty acids, modulate key immune receptors. Dysbiosis is associated with various inflammatory diseases and therapies like probiotics and fecal microbiota transplantation show promise in restoring microbial balance and improving immune function.

The gut microbiota is essential for immune regulation, and its modulation offers promising therapeutic opportunities for autoimmune and inflammatory diseases, providing more natural and less invasive treatment options.

TRABAJO
NO
PRESENTADO

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Checkpoint Inhibitors and Immune Regulation in Cancer Therapy

Rojas-Salazar, Yareli Lizbeth; Gómez-Montañez, Emiliano;
Rojas-Salazar, Jorge Gustavo.

[1]Instituto Nacional de Medicina Genómica, Ciudad de México, México. [2]Instituto Nacional de Medicina Genómica, Ciudad de México, México. [3]Universidad Autónoma de Ciudad Juárez, Ciudad Juárez, Chihuahua, México.

E-mail: yarojas177662@gmail.com

Immune checkpoints such as PD-1/PD-L1 and CTLA-4 are inhibitory pathways that regulate T cell responses and prevent autoimmunity; and cancer cells exploit these pathways to evade immune surveillance. Checkpoint inhibitors block these signals, enabling T cells to attack tumor cells.

This study aims to examine the mechanism of action of checkpoint inhibitors and their role in reactivating antitumor immunity, as well as to discuss the challenges associated with immune-related adverse events.

A review of clinical trials and immunological studies was conducted to evaluate the efficacy and safety of checkpoint inhibitors, including anti-PD-1, anti-PD-L1, and anti-CTLA-4 therapies, in which we obtained 25 articles to analyze.

Checkpoint blockade has revolutionized cancer treatment, showing significant benefits in melanoma, lung, and renal cancers; however, up to 20% of patients develop immune-related adverse events such as colitis, hepatitis, or thyroiditis due to nonspecific T cell activation.

Checkpoint inhibitors represent a major advancement in oncology, harnessing the immune system against tumors; and future efforts should aim at identifying biomarkers to predict response and toxicity, and at combining therapies to improve efficacy while minimizing adverse effects.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

CD43 regulates T-cell function and survival in a context-dependent manner

Rojo-León, Verónica [1]; Chipres-Naranjo, Luis Eduardo [1];
Cabañas -, Guadalupe [1 2]; Flores-Alcantar, Ángel Francisco [1];
Melchy-Pérez, Erika Isabel [1]; Rosenstein-Azoulay, Yvonne Jane [1].

[1]Instituto de Biotecnología, UNAM, Cuernavaca, Morelos, México. [2]Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México.

E-mail: veronica.rojo@ibt.unam.mx

CD43, a transmembrane glycoprotein also known as sialomucin or sialophorin, is broadly expressed on hematopoietic cells. By interacting with its different ligands, CD43 regulates adhesion/repulsion, cellular migration, and cell activation. We aimed to investigate whether CD43 regulates T cell proliferation. In vitro, we evaluated the proliferation and viability of CD4$^+$ and CD8$^+$ T cells from WT and CD43KO male C57BL/6 mice in response to PMA/ionomycin or CD3/CD28. Notably, CD8$^+$ CD43KO T cells showed a transient but significant decrease in numbers at 48 h, but recovered robustly by 72 h, maintaining proliferation and surpassing the CD8 WT cell population. In contrast, CD4$^+$ CD43KO T cells exhibited sustained lower viability and proliferation, alongside an increased Bax/BclXL ratio, suggesting a pro-apoptotic process. Consistent with this, CD43KO T-cells exhibited lower expression of the glucose transporter GLUT-1 compared to their WT counterparts. In vivo, we adoptively transferred CFSE-labeled lymph node cells from CD43WT and CD43KO OT-II mice into OVA-immunized C57BL/6 recipients. At 72 h post-transfer, we found that CD43KO CD4$^+$ T cells arrived in fewer numbers (approximately 50%) into the lymph node and proliferated seven-fold less than their WT counterparts. This indicates that CD43 is necessary for both migration into lymphoid tissues, metabolic remodeling, and efficient clonal expansion upon antigen encounter.

Our findings reveal that CD43 has a context-dependent and subset-specific role in T cell responses, with CD4$^+$ T cells being more susceptible to activation-induced cell death, failing to expand properly in its absence. In contrast, CD8$^+$ T cells can compensate for the loss of CD43. Thus, CD43 plays an essential role in promoting the survival, activation, and effective participation in immune response.

Funded by DGAPA/UNAM IN222523, Postdoctoral fellow by SECIHTI, 6002103.

ISSN: 2007-3380

Revista Bio Ciencias

https://revistabiociencias.uan.edu.mx

Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Viability of human leukemia cells with csGRP78 in an in vivo model

Román-Anguiano, Nadia Giovanna [1 2]; Gutiérrez-Muñoz, Paola Andrea [1 2]; Ángeles-Floriano, Tania [2 3]; Martínez-Rodríguez, Nancy Lucero [4]; Valle-Ríos, Ricardo [1 2].

Immunology and Proteomics Research Laboratory, Children's Hospital of México "Federico Gómez" (HIMFG), México City, México. [2]University Research Unit UNAM-HIMFG, Research Division, School of Medicine, National Autonomous University of México, México City, México. [3]Clinical Laboratory Department, Children's Hospital of México "Federico Gómez" (HIMFG), México City, México. [4] Epidemiology, Endocrinology & Nutrition Research Unit, Children's Hospital of México "Federico Gómez" (HIMFG), México City, México.

E-mail: naddokuro@gmail.com

Leukemia is the most common type of cancer in children. Leukemia cells have been observed to display the glucose-regulated protein GRP78 on their cell surface (csGRP78), and this presence has been linked to resistance to cancer therapies

GRP78 is a resident chaperone of the endoplasmic reticulum (ER). It is involved in protein folding and it is associated to three transmembrane proteins: i) IRE1 (inositol-1-requiring endoribonuclease); ii) PERK (eukaryotic initiation factor similar to double-stranded RNA-activated protein kinase); and iii) ATF6 (activator transcription factor 6). Under cellular stress, proteins accumulate in the ER lumen, GRP78 separates from these proteins, and moves to the cell surface.

Since csGRP78+ leukemia cells may have elevated survival skills, we measured the viability of these cells, in xenotransplanted athymic mice treated with vincristine. Transplanted cells were analyzed after 4 weeks. Bone marrow cells from vincristine treated mice revealed a modest increase in csGRP78+ CD19+ cells (from 39% to 41%) and a more pronounced increase in CD10+ cells (from 1.7% to 29%). The spleen showed an increase in csGRP78+ CD19+ cells from 0.5% to 5% and in CD10+ cells from 1.5% to 27%. Furthermore, the percentage of dead cells rose from 0% to 7% in bone marrow cells and stayed at 14% in the spleen.

Treatment with vincristine increased the percentage of csGRP78+ cells, suggesting that these cells may be under ER stress related to cancer survival.

Acknowledgments: This work received support from: Secretaria de Salud Funding: FPIS2024-HIM-6904 Hospital Infantil de México Federico Gómez Grant HIM/2021/038 SSA 1781 UNAM proyect 153/2017.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Myo1g participates in the expression of NK1.1 molecule and the cytotoxicity of NK cells.

Romero-Ramírez, Héctor [1 2]; López -Esparza, Vania Aminta Zuleica [1]; Flores-Castelan, Mariana [1 2]; Cortez-Hernández, Diana [2]; -, Santos; Argumedo-Leopoldo [1 2].

[1]Cinvestav, Departamento de Biomedicina Molecular, Ciudad de México, México. [2]Centro de Investigación sobre el envejecimiento. Ciudad de México, México.

E-mail: hromero@cinvestav.mx

Myo1g, a short-tail class I myosin, has been extensively studied for its role in cellular adhesion, migration, cytokine secretion, and receptor recycling on T and B lymphocytes. However, its involvement in other immune cell populations, particularly NK cells, remains a compelling question in immunology. Our research began using Western Blot to confirm the presence of Myo1g proteins in protein lysates from splenic NK cells from wild-type (WT) mice. We then found that mice deficient in Myo1g (Myo1g-/-) had a decrease in the frequency and absolute numbers of NK cells (defined by CD3- CD19- NK1.1+ lymphocytes) in 8-week-old bone marrow, blood, and spleen compartments compared to WT. Additionally, we analyzed the NK1.1 molecule on CD3- CD19- cells of Myo1g-/- mice and observed its reduced expression, but other molecules expressed by NK cells, such as CD49b and NKp46, were found in the same proportion. Afterward, we evaluated NK cells expressing Granzyme A+ in the CD3- population, finding reducing degranulation in cells from Myo 1g -/- compared to WT mice. Finally, we corroborated the previous observations using purified NK cells, demonstrating that cells from Myo1g deficient mice have reduced cytotoxicity than WT mice. Myo1g participates in the expression of NK1.1 molecule and cytotoxicity of NK cells. These results open new avenues for research, suggesting that Myo1g could be a potential target for modulating NK cell function and immune responses.

Founded by Secihti CF-2023-I-741

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Serological Profiling in Rickettsiosis Using a Multi-Species Recombinant Antigen

Romero-Terrazas, Diana [1]; Araujo-Henriquez, Norma [2];
Baquera-Arteaga, Maribel [3]; Zurawski, Sandra [3];
Zurawski, Gerard [3]; Espino-Solís, Gerardo Pavel [1].

[1]Universidad Autónoma de Chihuahua; Facultad de Medicina y Ciencias Biomédicas; Laboratorio de Investigación Traslacional; Chihuahua, Chih. México. [2]Hospital General Regional 01 de Chihuahua; Chihuahua, Chih. México. [3]Hospital Infantil de Especialidades de Chihuahua; Chihuahua, Chih. México. [4]Biotechnology Center at Baylor Institute for Immunology Research. Dallas, Texas, USA.

E-mail: a351811@uach.mx

Tick-borne rickettsial infections pose a diagnostic challenge due to their nonspecific clinical presentation and the lack of sensitive, standardized assays for early detection. In Chihuahua, Mexico, Rickettsia rickettsii is the predominant cause of severe rickettsiosis, with a case fatality rate of 55 % reported in 2024. This study aimed to develop and implement an ELISA-based assay for the detection of IgM, IgG, and IgA antibodies against a multi-species recombinant rickettsial antigen in peripheral blood samples from confirmed cases. Twenty-four serum samples, collected between 2023 and 2025, were analyzed and stratified by clinical severity. Patients with severe disease exhibited significantly elevated IgM titers, suggesting that early systemic humoral responses may contribute to immunopathology. In contrast, deceased patients showed absent or markedly reduced IgM levels, consistent with immune suppression in late-stage disease. IgG levels did not differ significantly across clinical categories, indicating limited utility in acute-phase assessment. These findings support the implementation of IgM- and IgA-based ELISAs as sensitive biomarkers for early detection of rickettsial infections. The use of a recombinant multi-species antigen enhances cross-reactivity and may facilitate assay standardization in endemic settings. Translationally, this serological platform offers potential for point-of-care diagnostics and integrated surveillance strategies, thereby improving early intervention, risk stratification, and clinical outcomes, particularly in resource-limited regions.

Acknowledgments: We gratefully acknowledge the participation of the patients and their families, whose trust made this study possible. We also thank the medical residents for their dedicated clinical support.

This work was funded by the “Más UACH, Más Ciencia” grant from the Universidad Autónoma de Chihuahua.

ISSN: 2007-3380
Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Cytokine alterations in the pediatric population chronically exposed to high concentrations of Particulate Matter

Romo-García, María Fernanda [1]; Mendoza-Cano, Oliver [3]; Murillo-Zamora, Efrén [4]; Rivas-Santiago, Bruno [2]; García Hernández, Mariana Haydee [2]; González-Curiel, Irma E. [1].

[1]Laboratorio de Inmunotoxicología, Unidad Académica de Ciencias Químicas, Universidad Autónoma de Zacatecas, Campus UAZ siglo XXI, Carretera Zacatecas-Guadalajara km 6, Col. Ejido La Escondida, Zacatecas 98160, México. [2]Unidad de Investigación Biomédica Zacatecas, Instituto Mexicano del Seguro Social, Alameda Trinidad García de La Cadena 438_2436A436, Zacatecas Centro, 98000 Zacatecas, Zacatecas, México. [3]Facultad de Ingeniería Civil, Universidad de Colima, Carretera Colima-Coquimatlán km 9, Col. Jardines del Llano, Coquimatlán 28400, México. [4]Unidad de Investigación en Epidemiología Clínica, Instituto Mexicano del Seguro Social, Av. Lapislázuli 250, Col. El Haya, Villa de Álvarez 28984, México.

E-mail: mromog@cinvestav.mx

Particulate matter (PM) pollution is a global concern. The sugarcane “zafra” process can release up to 60% of PM emissions (Lara *et al*., 2005); therefore, it is associated with adverse pulmonary effects (Arbex *et al*., 2007). The pediatric population is particularly vulnerable to PM as its exposure has been linked to short-term increases in asthma incidence (Halonen *et al*., 2008). To explore cytokine alterations in the pediatric population exposed to PM released by sugarcane harvesting process.

Serum samples were collected from exposed (<300 meters from PM emission) and non-exposed (>35 km from PM emission) population. A total of 202 individuals aged 5 to 12 years were included. Cytokine Th1/Th2/Th17 quantification was performed using a bead array (BD CBA Kit) on a FACS Canto II flow cytometer. PM concentration was measured using a Nanoenvi device over a four-month period. Statistical differences were analyzed using the Kruskal Wallis test with Dunn’s post hoc test.

PM concentration was 30 μg/m³, three times above the permissible limits outlined in NOM-021-SSA1-2021. In the exposed population, cytokine concentrations of IL-2, IL-10, IFN-γ, and IL-6 were increased by 1.6 times ($p < 0.001$) compared to the non-exposed population. Additionally, within the exposed population, higher IL-2 and IFN-γ concentrations observed in individuals located less than 900 meters closer to the emission point ($p < 0.05$). No significant differences were found in allergic incidence (13.4% vs. 7.6%, $p = 0.12$).

The pediatric population exposed to high levels of PM exhibited a significant increase in IL-2, IL-10, IFN-γ, and IL-6 cytokines, which are probably induced by inflammatory responses. This suggests that the cytokine response could be triggered as part of a pulmonary repair mechanism in reaction to inhaled PM and its deposition in lung tissue, indicating an immunologic response to a xenobiotic.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Synthetic Peptides of GP45 and GP90 as Biomarkers for Early EIAV Detection

Romo -Sáenz, César Iván [1]; Orozco-Ruiz, Eleazar [1];
Arzate-Quintana, Carlos [1]; Castro-Valenzuela, Beatriz Elena [3];
Tamez-Guerra, Patricia [2]; Gómez-Flores, Ricardo [2].

[1]Facultad de Medicina y Ciencias Biomédicas Universidad Autónoma de Chihuahua, Chihuahua, Chihuahua, México. [2]Facultad de Ciencias Biológicas, Universidad Autónoma de Nuevo León, San Nicolas de los Garza, Nuevos León, México. [3]Facultad de Zootécnica, Universidad Autónoma de Chihuahua, Chihuahua, Chihuahua, México.

E-mail: cromo@uach.mx

Early detection of Equine Infectious Anemia Virus (EIAV) is critical for effective control of this viral disease affecting horses, which is characterized by high antigenic variability and frequent asymptomatic carriers that complicate diagnosis. Due to the limited sensitivity of conventional methods, this study evaluated the ability of synthetic peptides derived from the glycoproteins GP45 and GP90, as well as the P26 protein, to detect specific antibodies using an indirect ELISA assay. Serum samples from 48 horses, including 6 symptomatic and 42 asymptomatic animals confirmed by agar gel immunodiffusion (AGID), were analyzed. Results showed that GP90 had the highest serological sensitivity (25.8%), followed by GP45 (16.1%) and P26 (14.5%). Bioinformatic analysis revealed that GP90 exhibited high amino acid variability (49.39%), while GP45 showed lower variability (35.12%), which affects immune recognition. Although GP90 demonstrated greater detection capacity, its high variability limits its use as a sole marker. Therefore, the combination of synthetic peptides from GP45 and GP90 is proposed to improve sensitivity and specificity of serological diagnosis, especially in asymptomatic animals. These findings highlight the potential of synthetic peptides as tools for early detection of EIAV, which could strengthen surveillance and control programs in equine populations.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Low-density neutrophils increase during obesity and display less phagocytosis

Rosales, Carlos [1]; Blanco-Camarillo, Carlos [1]; Uribe-Querol, Eileen [2]; Ferreira-Hermosillo, Aldo[3].

[1]Instituto de Investigaciones Bimédicas, Universidad Nacional Autónoma de México, México City, México. [2]Facultad de Odontología, Universidad Nacional Autónoma de México, México City, México. [3]Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México.

E-mail: carosal@iibiomedicas.unam.mx

Neutrophils, important leukocytes of innate immunity, have traditionally been considered a homogeneous cell population. Nevertheless, recent evidence has shown that neutrophils exist in several subpopulations. One such subpopulation is the “low-density neutrophils” (LDN). LDN are present in healthy conditions, but they increase significantly in diseases such as cancer, systemic lupus erythematosus, rheumatoid arthritis, psoriasis, and tuberculosis. In other diseases, such as obesity, there is no information about LDN. Obesity is a disease associated with several health complications, including an increased susceptibility to infections. However, the underlying mechanisms for this increased susceptibility remain poorly understood. Because, neutrophils play a critical role in the immune response to infections, we evaluated whether LDN also increase in obesity and whether they have a dysfunctional antimicrobial function. LDN from obese individuals were compared to those from non-obese individuals. The numbers of LDN increased dramatically in obese individuals. Purified LDN from obese individuals were functional as indicated by their higher capacity to produce reactive oxygen species (ROS) than LDN from normal-weight individuals. However, their antibody-mediated phagocytic activity was reduced. Mechanistically, expression of the phagocytic antibody Fc receptor Fc gamma RIIa was diminished on LDN from obese individuals. LDN from obese individual were also able to form neutrophil extracellular traps (NET). However, NET formation was reduced in response to bacteria. These findings suggest that obesity induces an increase in LDN, which although seem to be activated, they display inhibited antimicrobial mechanisms. This could impair pathogen clearance and contribute to infection susceptibility in obese individuals.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Expression and regulation of MPP1 in human monocyte-derived macrophages (MDM)

Rosas-García, Jorge [1]; Rivera-Almazo, Luis Eduardo [1 2]; Santos-Mendoza, María Teresa [1].

[1]Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas, Laboratorio de Transcriptómica e Inmunología Molecular, Ciudad de México, CDMX, México. [2]Universidad Autónoma Metropolitana, División de Ciencias Biológicas y de la Salud, Ciudad de México, CDMX, México.

E-mail: drjrg.jorge@gmail.com

MPP1 (membrane palmitoylated protein 1) belongs to the subfamily of membrane palmitoylated proteins (MPPs), a group enclosed in the large family of scaffold and multidomain proteins known as membrane-associated guanylate kinases (MAGUKs). MPP1 plays a key role in the stability of the erythrocyte membrane and is an important regulator of neutrophil polarity during chemotaxis. Recently, we found MPP1 transcriptional regulation in macrophages (MOS) and dendritic cells (DC) upon distinct PRR stimulation. Additionally, we have previously identified MPP1 as a target of the Envelope protein of SARS-CoV-2 (2E) in monocytes. Here, we analyzed the expression of MPP1 in human monocyte-derived macrophages (MDM) using western blot (WB) and its regulation upon stimulation with bacterial (lipopolysacaride [LPS]) and viral (polyinosinic-polycytidylic acid [poly I:C]) pathogen-associated molecular patterns (PAMPs) over time. Our results showed an increase in MPP1 expression at 6 and 12 h upon LPS stimulation, while the increase upon Poly I:C stimuli was greater until 24 hours. These results suggest that MPP1 plays important functions in MOS that should be addressed.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Silencing of IL-16 Expression in Human Monocyte-Derived Macrophages

Rosas-García, Jorge [1]; García-Mijares-Nevárez, Ricardo
Ramsés [1 2]; Santos-Mendoza, María Teresa [1].

[1]Instituto Nacional de Enfermedades Respiratorias "Ismael Cosio Villegas", Laboratorio de Transcriptómica e Inmunología Molecular, CDMX, México. [2]Universidad Autónoma Metropolitana Unidad Cuajimalpa, División de Ciencias Naturales e Ingeniería, CDMX, México.

E-mail: drjrg.jorge@gmail.com

Most of tissues express mRNA for IL-16, and presumably translate and secrete IL-16 under certain conditions. Upon cell activation IL-16 is cleaved, forming functional proteins, one regulating cell cycle and the other acting as chemoattractant for cells carrying CD4 or CD9. Recently, it was revealed that Mtb hijacks host macrophage-derived IL-16 to enhance its intracellular growth, suggesting an immunosuppressive role of IL-16 during Mtb infection. Our research group previously reported different bands observed in macrophages by Western blot of IL-16 that correspond to the protein at different levels of post-translational processing. Band 1 of approximately 80 kDa corresponding to Pro-IL-16 was observed, while band 2 of approximately 50 kDa corresponding to the N-terminal end of the protein once processed by caspase 3, while the portion corresponding to the C-terminal end of Pro-IL-16 was not observed. Furthermore, we previously observed a significant increase in the expression of IL-16 band 1 after stimulation at 6h with LPS of bands 1 and 2, while only band 2 was maintained until 24 h. In this study, macrophages were treated with SmartPool IL-16 siRNA and LPS, and we verified the results by means of western blot, which showed a trend towards decreased IL-16 expression at 6h of treatment of 6% regarding its control (SiRNA Scramble) for band 1, while for band 2, this was 35.8%. On the other hand, in the 24h treatment with the IL-16 SmartPool IL-16 SiRNA and LPS, a decrease in expression of 37% for band 1 and 75% for band 2 was observed. In conclusion, the expression of IL-16 was reduced in macrophages with SmartPool IL-16 siRNA stimulated with LPS at 24h , as an important tool for subsequent functional studies.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Diet quality and rs1800795 IL6 interact with cardiovascular risk in rheumatoid arthritis

Rosas-García, Karla Sofía; Campos-López, Bertha;
Mora-García, Paulina Esmeralda; Pesqueda-Cendejas, Karen;
Rivera-Escoto, Melissa; De la Cruz-Mosso, Ulises.

[1]Red de Inmunonutrición y Genómica Nutricional en las Enfermedades Autoinmunes; Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud; Universidad de Guadalajara; Guadalajara, Jalisco 44340, México. [2]Instituto de Neurociencias Traslacionales, Departamento de Neurociencias, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara 44340, Jalisco, México.

E-mail: sofia.rosas@alumnos.udg.mx

Rheumatoid arthritis (RA) is a chronic inflammatory disease that affects the joints, and cardiovascular system. It has been reported that dietary and genetic factors such single nucleotide variants (SNVs) could interact influencing disease outcomes. One such variant, -174 G/C (rs1800795) in the interleukin six (IL6) gene, could modulate serum IL-6 serum levels and contribute to increased cardiovascular risk. This study aimed to determine the interaction between diet quality and the -174 G/C (rs1800795) genetic variant in IL6 with cardiovascular risk in RA patients. 178 RA patients and 206 control subjects (CS), all females were included. Genotyping was performed using TaqMan allele discrimination assays, and diet quality was assessed using the alternative healthy eating index (AHEI-2010) The total AHEI-2010 scores were classified as low and moderate diet quality. Nutrient intake analysis showed that RA patients and CS had inadequate intake of vitamin E, vitamin D, iodine, biotin, and folate. Analysis of gene–diet interactions showed higher C-reactive protein (CRP) levels in the GG + low-quality diet group (3.1 mg/dL) compared to the GG + moderate-quality group (1.8 mg/dL; $p = 0.01$). Cardiovascular risk scores assessed by LAP and CRP/albumin ratio were also highest in the GG + low-quality diet group. Carriers of the GG genotype + low diet quality had significant risks of high BMI (OR = 3.9), high waist-to-height ratio (OR = 2.8), high CRP/albumin ratio (OR = 2.5), and rheumatoid factor (OR = 13.0; $p = 0.03$). In contrast, GC/CC carriers + moderate diet quality showed low risk. In conclusion; in both study groups, carriers of the GG genotype of -174 G/C IL6 with a low diet quality had higher cardiovascular risk than carriers of GC and CC genotypes with a moderate diet quality.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

MIF inhibitor (ISO-1) modulate ocular surface Inflammation in a Dry Eye model

Rosas-González, Karla Andrea [1]; Aguilar-Garduño, Arturo [1];
Zacarías -García, Mónica [1]; Gutiérrez -Ospina, Gabriel [2];
Rodríguez -Sosa, Miriam [3]; Juárez -Avelar, Imelda [3]; López -Pineda,
Katerine D [1]; Vázquez -Mendoza, Alicia [1].

[1]Laboratorio de Inmunología Ocular, Carrera de Optometría. Facultad de Estudios Superiores Iztacala, UNAM. Tlalnepantla de Baz, Estado de México, México.. [2]Laboratorio de Biología de Sistemas, Departamento de Biología Celular y Fisiología, Instituto de Investigaciones Biomédica, UNAM.. [3]Unidad de Investigación en Biomedicina, Lab 5, Inmunidad FES-I, UNAM. Tlalnepantla de Baz, Estado de México, México.

E-mail: lyandrea321@gmail.com

Dry eye disease (DED) is a multifactorial disease characterized by loss of tear film stability, inflammation and damage on the ocular surface. There is a close relationship between the worsening of the disease and the release of chemokines, metalloproteinases and inflammatory mediators such as: TNF-α, IFN-γ, IL-1β, IL-6, IL-17 and the macrophage migration inhibitory factor (MIF). In particular, it has been reported a significant increase of MIF in tears and serum from patients with DED. In this context, we recently demonstrated that genetic deficiency of MIF attenuates the signs of the disease. Hence, we aimed to evaluate the potential therapeutic effect of pharmacological inhibition of MIF, dry eye was induced in male mice of the Balb/c strain through the administration of 0.5mg of scopolamine intraperitoneally three times a day for 28 days. From day 14 post-scopolamine, the mice of the vehicle group (dimethyl sulfoxide (DMSO)) and the pharmacological inhibitor of MIF (ISO-1) received ophthalmic route 5 µl of DMSO (10%) or 1 mg/ml of ISO-1 twice daily for 14 days. The evaluation of tear film stability through the Schirmer and ferning test revealed that ISO-1 administration reduces loss of tear volume and mucin arborization pattern. These observations were associated with a low expression of ifn-γ, il-1β and tnf-α in cornea. Together, these results suggest that ISO-1could be used as a strategy to modulate inflammation on the ocular surface associated to dry eye.

This Project is currently funded by CONAHCYT: CBF 2023-2024-405

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Role of the CD43 sialomucin in macrophage differentiation in contrasting inflammatory backgrounds

Alemán-Navarro, Estefanía [1 2]; Chipres-Naranjo, Luis Eduardo [1 2];
Rubio-Blancas, Emiliano [3]; Rosenstein-Azoulay, Yvonne [1].

[1]Instituto de Biotecnología, UNAM Campus Morelos, Departamento de Medicina Molecular y Bioprocesos, Cuernavaca, Morelos, México. [2]Posgrado en Ciencias Bioquímicas, UNAM. [3]Centro de Investigación en Dinámica Celular, Universidad Autónoma del Estado de Morelos, Cuernavaca, Morelos, México.

E-mail: rubioemiliano12@gmail.com

Macrophages are key components of the immune system that are derived from monocytes through a process of differentiation. This process results in morphological and functional changes that enable macrophages to carry out antimicrobial and homeostatic functions. As the prevailing inflammatory conditions significantly impact this differentiation process, cellular receptors that sense changes in the extracellular microenvironment play a significant role. CD43 is a long and rigid transmembrane sialomucin abundantly expressed on the surface of leukocytes. Due to its long and rigid shape, CD43 is presumably one of the first molecules to sense environmental cues and modulate the resulting cellular response. While CD43 has been shown to regulate cellular adhesion, migration, activation, proliferation, survival, and cytokine/chemokine secretion, its contribution to macrophage differentiation remains unexplored. In this work, we investigated the impact of CD43 on macrophage differentiation in bone marrow-derived macrophages (BMDMs) from BALB/c and C57BL/6 cells, two strains with an anti- or pro-inflammatory biased immune response, respectively, as well as in the human monocytic cell line THP-1. Our results show that, regardless of the strain, BMDMs from WT and CD43KO expressed similar levels of the macrophage marker F4/80. Likewise, THP-1 macrophages expressing either normal (CD43hi) or reduced (CD43lo) levels of CD43 showed comparable levels of the macrophage markers CD11b and CD14. However, CD43-deficient macrophages exhibited altered expression of TLR2 and TLR4 in a strain-dependent manner. Specifically, CD43KO C57BL/6 macrophages showed increased TLR2 and TLR4 expression, whereas CD43KO BALB/c macrophages displayed increased TLR4 but decreased TLR2 expression. These results show that macrophage differentiation is not affected by the absence of CD43 or the host's inflammatory background. However, CD43 might influence the expression of PRRs, such as TLR2 and TLR4, depending on the host's immune response type.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Helios limits CD8 T cell antitumor capacity

Rubio-Robles, Rosa M. [1]; Suárez-Rojas, Gerardo [1,2];
Albarrán-Godínez, Adrián [1,2]; De León-Rodríguez, Saraí G. [3,4];
Aguilar-Flores, Cristina [4]; Fonseca-Montaño, Marco [1];
González-Yáñez, Marysol [5]; Bonifaz, Laura C. [3,6];
Madera-Salcedo, Iris K. [1]; Crispín-Acuña, José C. [1,7];
Rosetti-Sciutto, Florencia [1].

[1]Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Departamento de Inmunología y Reumatología, Ciudad de México, México. [2]Programa de doctorado en Ciencias Biomédicas, UNAM, Ciudad de México, México. [3]UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Unidad de Investigación Médica en Inmunoquímica, Ciudad de México, México. [4]Programa de doctorado en Ciencias Biológicas, UNAM, Ciudad de México, México. [5]Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Departamento de Investigación Experimental y Bioterio, Ciudad de México, México. [6]Coordinación de Investigación en Salud, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México. [7]Escuela de Medicina y Ciencias de la Salud, Tecnológico de Monterrey, Monterrey, México.

E-mail: rossieru97@gmail.com

Helios (Ikzf2) is a transcription factor associated with suppressive function in thymic-derived CD4 regulatory T cells. Previously, we documented Helios expression in CD8 cells exposed to antigen presented as self or associated with malignant cells. However, the role of Helios in CD8 T cells function is poorly understood. Here, we explored whether Helios expression was induced in tumor infiltrating CD8 T cells and assessed the effects of its absence. We found that Helios expression in CD8 T cells was promoted by the tumor microenvironment in patients with colorectal cancer and in mice with different cancer models. Notably, cells from tumor-draining lymph nodes remained Helios negative. Helios induction was driven by antigen activation, as it was absent in OT-I cells from tumors devoid of ovalbumin. Helios expression correlated with the progression of the exhaustion program. Helios deficiency in T cells (Cd4-Cre) and in CD8 T cells (E8III-Cre) enhanced their anti-tumor capacity, likely by expanding the pool of TCF1+ progenitor cells. This resulted in decreased tumor growth in models of melanoma and colon adenocarcinoma in mice and was associated with a reduction in terminally exhausted CD8 T cells and an increased frequency of TCF-1+ cells. We generated Pdcd1 and Ikzf2 double KO mice (dKO). These mice exhibited superior tumor control compared to those with single deficiencies, with a significant fraction showing complete tumor rejection. Tumor-infiltrating CD8 T cells from dKO mice produced higher levels of IFN-g and granzyme B. A small molecule screen identified a drug capable to inhibit Helios expression, and its administration improved the anti-tumor effects of PD-1 deficiency. Together, these findings suggest that Helios is induced in CD8 T cells by the tumor microenvironment and that its expression limits their anti-tumor capacity. Therefore, Helios represents a potential novel target for immunotherapy, that could work in combination with PD-1/PDL-1 blockade.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Tolerogenic innate immune cells compensate for regulatory T cells in Kawasaki disease

Ruedas-Montero, Rubén E. [1]; Song, Jaeyoon [1]; Tremoulet, Adriana H. [1]; Burns, Jane C. [1]; Franco, Alessandra [1].

[1]University of California San Diego, School of Medicine, Department of Pediatrics, Division of Allergy, Immunology, and Rheumatology, La Jolla, CA, USA.

E-mail: ruedasr@uabc.edu.mx

Kawasaki disease (KD) is an acute pediatric vasculitis of the coronary arteries. Natural regulatory T cells (nTreg) that recognize the heavy constant region of IgG (Fc) are poorly expanded in the acute phase. IVIG restores the expansion of Fc-specific nTreg in most patients, although the nTreg repertoire does not last. This raises the question of why individuals with a history of KD do not develop other inflammatory conditions. We enumerated and characterized innate immune cells in healthy subjects years after KD onset and IVIG treatment. We defined their capacities to regulate inflammation via the secretion of suppressive lymphokines as interleukin-10 (IL-10), and IL-1receptor antagonist (IL-1Ra).

Study population: Eight KD patients studied in the acute phase after IVIG (7 males, 1 female aged 6 months-7 years), and 18 healthy subjects with a history of KD (12 males and 6 females aged 3 to 56 years). Immunephenotyping: peripheral blood mononuclear cells (PBMC) were isolated and cultured 24 hours with and without purified Fc fragments to define innate cell lineages, and their maturation/activation stage. Fc-specific nTreg were studied after 4 days in culture. Lymphokines were measured by ELISA and intracellular staining.

Fc-specific nTreg in acute KD transiently expand after IVIG. Mature tolerogenic myeloid dendritic cells (TmDC, CD4+ ILT-4+) were abundant. Monocytes expand and mature when stimulated with Fc fragments. CD14+ cDC1 myeloid dendritic cells also respond to Fc stimulation secreting the suppressive lymphokines IL-10 and IL-1Ra. TmDC in vitro spontaneously secrete IL-1Ra, and IL-10 upon Fc stimulation.

Lack of Fc-specific nTreg in KD subjects is compensated for by innate myeloid cells with a tolerogenic phenotype.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Cytokines and alpha-interferon response of A549 cells against influenza A virus

Ruelas-Ruiz, Luis C [1]; Martínez-Castilla, Azalia M [1];
Medina-De la Garza, Carlos E [1]; Macias-Segura, Noe [1];
Rosas-Taraco, Adrián G [1].

[1]Universidad Autónoma de Nuevo León, Facultad de Medicina, Departamento y Servicio de Inmunología, Monterrey, Nuevo León, México.

E-mail: lcruelas25@gmail.com

Respiratory epithelial cells serve as the first line of defense against airborne pathogens, playing a key role not only as a physical barrier but also as active contributors in initiating immune responses. Upon infection with respiratory viruses such as Influenza A virus (IAV), these cells secrete a broad range of cytokines and chemokines that coordinate local and systemic immunity, notably type I and type III interferons. These interferons play essential roles in restricting viral replication and shaping the immune environment at mucosal surfaces, with type III IFNs particularly important for their antiviral role in the lung epithelium. However, excessive or dysregulated cytokine production may contribute to immunopathology, highlighting the importance of understanding epithelial cytokine dynamics during infection. The aim of this work was to evaluate the cytokine secretion profile of human respiratory epithelial cells infected with IAV. A549 human alveolar epithelial cells were cultured and infected with IAV at a multiplicity of infection (MOI) of 1 while another group was MOCK-treated. After 24 hours, supernatants were harvested and analyzed using the LEGENDplex™ Human Anti-Virus Response Panel, which quantifies 13 antiviral cytokines, including IL-1β, IL-6, TNF-α, IP-10, IL-8, IL-12p70, GM-CSF, IFN-α2, and IFN-λ1/2/3. Results showed significant increases in the secretion of IL-6, IP-10, IL-8, and IFN-α2 in the infected group compared to controls (Wilcoxon test, $p < 0.05$). Furthermore, viral titers were inversely correlated with IL-6 and IP-10 concentrations, with correlation coefficients of -0.63 and -0.64, respectively, suggesting a potential feedback mechanism or a role in viral clearance. In conclusion, our findings suggest that IAV infection induces a robust cytokine response in respiratory epithelial cells, highlighting their active role in shaping early antiviral immunity.

ISSN: 2007-3380

Revista Bio Ciencias

https://revistabiociencias.uan.edu.mx

Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Influenza vaccination modulates monocyte subsets and cytokine expresión upon viral challenge

Ruelas-Ruiz, Luis C [1]; Rendon-Pérez, Luis A [2]; Martínez-Castilla, Azalia M [1]; Montes-Zapata, Edgar I [1]; Ramirez-Garcia, Arianna [1]; Rosas-Taraco, Adrián G [1].

[1]Universidad Autónoma de Nuevo León, University Hospital “Dr. Jose Eleuterio Gonzalez”, Service of Immunology, Monterrey, Nuevo León, México. [2]Universidad Autónoma de Nuevo León, University Hospital “Dr. Jose Eleuterio Gonzalez”, Department of Internal Medicine, Monterrey, Nuevo León, México.

E-mail: lcruelas25@gmail.com

Monocytes are crucial components of the innate immune system, contributing to host defense through phagocytosis, efferocytosis, and tissue replenishment, notably by differentiation into macrophages in tissues such as the lung. Emerging evidence suggests that monocytes may also develop an enhanced response upon secondary stimulation known as trained immunity which could be modulated by prior vaccination. This study aimed to investigate the phenotypic and functional responses of monocyte subsets from influenza-vaccinated individuals following ex vivo infection with influenza virus. Peripheral blood samples were collected from healthy vaccinated subjects, and monocytes were isolated via density gradient centrifugation and negative magnetic separation. Cells were infected with influenza A virus (MOI=1) or MOCK-treated and cultured for 24 hours. Flow cytometry was employed to assess the expression of CD14, CD16, IFNγ, IL-4 and IL-10, as well as cell viability. Preliminary results revealed a significant reduction in the frequency of classical monocytes (CD14+CD16-) in IAV-infected cultures compared to MOCK-treated controls. These decreases correlated with increased cell death in the infected group, suggesting a potential vulnerability of classical monocytes to viral-induced cytotoxicity. Interestingly, surviving classical monocytes showed elevated IFN-γ expresión post-infection. In intermediate monocytes (CD14+CD16+), both IFNγ and IL-10 expression were increased following IAV-exposure, indicating a complex activation profile. These changes may reflect early phenotypic shifts toward macrophage-like states during in vitro culture. In conclusión, our findings suggest that prior influenza vaccination may prime monocytes for altered responses upon viral encounter, characterized by different subset dynamics and cytokine profiles supporting the relevance of innate immune memory in vaccine-induced protection through innate immune cells such as monocytes.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Human parvovirus B19 VLP's Induce SARS-CoV-2 neutralizing antibodies in mice.

Ruíz-Cruz, Alail Antonio [1]; Gutiérrez-Morales, Lourdes Selene [1]; Avalos-Carreón, Getsemani [1]; Jiménez-Chávez, Ángel de Jesús [1]; Ilhuicatzi-Alvárado, Damaris [1]; Ávila-Ríos, Santiago [2]; Bustos-Jaimes, Ismael [3]; Moreno -Fierros, Leticia [1].

[1]National Autonomous University of Mexico, Mucosal Immunology Laboratory, Tlalnepantla, Estado De México, México. [2]National Institute of Respiratory Diseases, Center for Research in Infectious Diseases, México City, México City, México. [3]National Autonomous University of Mexico, Faculty of Medicine: Department of Biochemistry, México City, México City, México.

E-mail: biol.antonio.ruiz@gmail.com

The COVID-19 pandemic, caused by SARS-CoV-2, severely impacted global health. While the public health emergency has ended, the persistent threat of viral resurgence due to factors like increased transmissibility, waning immunity, or mutations underscores the need for advanced vaccine strategies. The SARS-CoV-2 Spike (S) protein is crucial for viral infection, mediating entry into human cells, particularly in the respiratory tract. Existing vaccines largely target this protein, proving effective against severe disease. However, challenges remain in achieving broad immunity and preventing immune escape. Current approaches often focus on the full Spike protein, potentially leading to immunodominance that limits efficacy against infection. This highlights the importance of exploring new vaccine designs for broader, more effective protection. Research has identified immunodominant "hot spots" within the Spike protein, conserved across variants and crucial for infection. These regions including the Receptor Binding Domain (RBD), Fusion Protein (FP), Heptad Repeat 1 (HR1), Heptad Repeat 2 (HR2), and the TMPRSS2/cathepsin cleavage site in the S2 domain are frequent targets of neutralizing antibodies. Leveraging these epitopes in vaccine design promises more potent, targeted responses, often requiring adjuvants.

In this study, we developed meSpikeVP2, a novel multiepitopic vaccine prototype. It incorporates key immunodominant linear epitopes from Spike "hot spots" known to induce neutralizing antibodies. To enhance the immune response and address subunit immunogenicity challenges, this multi-epitopic construct was inserted into the surface loop (301-313) of the human parvovirus B19 VP2 protein (B19V). B19V VP2 is an ideal carrier; it spontaneously forms Virus-Like Particles (VLPs) even when modified, effectively presents antigens, and acts as a natural adjuvant by mimicking a native viral capsid. Our meSpikeVP2 prototype successfully formed stable VLPs. Intramuscular administration to C57BL/6 mice induced antibodies that effectively recognized the SARS-CoV-2 Spike protein. The multi-epitopic region was also recognized by IgG antibodies from convalescent patients infected with Wuhan, Delta, and Omicron variants. Critically, antibodies from meSpikeVP2-immunized mice neutralized SARS-CoV-2 in vitro, preventing cytopathic effects in VERO cells. These findings demonstrate that the meSpikeVP2 vaccine prototype can confer protection against SARS-CoV-2 in a murine model, highlighting its potential as a broad spectrum vaccine candidate.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Pathway Polarization in Human Leprosy Biopsies: From Immune Containment to Exhaustion

Ruiz-de la Cruz, María Luisa [1]; Rojas-Gutiérrez, Santiago Emilio [1]; Martínez-Canales, Rodolfo [1]; Barraza-Ramos, Brenda Alejandra [2]; Montoya-Rosales, Alejandra [3]; Salinas-Carmona, Mario César [1]; Macías-Segura, Noé [1].

[1]Universidad Autónoma de Nuevo León, Hospital Universitario "Dr. José Eleuterio González" Servicio y Departamento de Inmunología, Monterrey, Nuevo León México. [2]Universidad Autónoma de Nuevo León, Hospital Universitario "Dr. José Eleuterio González" Departamento de Medicina Interna, Monterrey, Nuevo León México. [3]Universidad Autónoma de Nuevo León, Centro de Investigación y Desarrollo en Ciencias de la Salud, Monterrey, Nuevo León México.

E-mail: mluisardlc@gmail.com

Leprosy, caused by Mycobacterium leprae, remains a considerable global health challenge due to its potential to induce irreversible neuropathy. Research utilizing human skin biopsies has elucidated its immunological spectrum: tuberculoid (TT) lesions display robust Th1/IFN-γ responses that facilitate bacterial containment, whereas lepromatous (LL) forms are characterized by a Th2 bias, expansion of regulatory T-cells, and checkpoint-mediated tolerance, notably involving PD-1 and LAG3. Despite recent advancements, the mechanisms contributing to the pathological immunosuppression seen in leprosy remain insufficiently characterized. Conducting pathway-level analyses of biopsies from patients offers unparalleled insights into in situ immune responses, potentially unveiling actionable targets for immunotherapy aimed at reversing tolerance and preventing disability. We conducted a quantification of the enrichment of 12 immune pathways across healthy controls (HC), TT, and LL skin biopsies using GSVA and compared with ANOVA and Kruskal-Wallis tests. This analysis aimed to identify polarization-specific signatures and explore novel therapeutic pathways. Both leprosy subtypes demonstrated significant hyperactivation in interferon responses and PD-1/TCR signaling in comparison to healthy controls. Furthermore, LL exhibited pronounced upregulation of the IL-6/JAK/STAT3 pathway, complement pathways, and reactive oxygen species (ROS) pathways compared to TT. Additionally, TNFα/NFκB signaling was markedly elevated in TT compared to HC and showed a trend toward elevation in comparison to LL. Biopsies from LL patients exhibit a triad of immunosuppressive features: IL-6/STAT3 pathways known to promote Th17 and Treg cell expansion, complement system activation which recruits inhibitory myeloid cells, and ROS overload that induces mitochondrial dysfunction—all of which can synergize with PD-1 to effectively inhibit immune responses. In contrast, the TNFα/NFκB signaling signature in TT supports granuloma-mediated containment of the infection. Targeting these identified pathways may facilitate repurposing therapeutic agents such as JAK inhibitors, complement blockers, or antioxidants in conjunction with immune checkpoint inhibitors targeting PD-1 in lymphocytes.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

PD-1/PD-L1 blockade exacerbates inflammatory tissue damage in experimental Nocardia brasiliensis mycetoma

Ruiz-de la Cruz, María Luisa [1]; Vázquez-Marmolejo, Anna Velia [1];
Mejía-Torres, Manuel Guadalupe [1]; Castro-Corona, María de los
Ángeles [1]; Salinas-Carmona, Mario César [1].

[1]Department of Immunology, School of Medicine and Dr. Jose Eleuterio Gonzalez University
Hospital, Universidad Autonoma de Nuevo León, Monterrey, Mexico.

E-mail: mluisardlc@gmail.com

Nocardia brasiliensis mycetoma, a neglected tropical disease, progresses to chronicity through unclear mechanisms, potentially involving T-cell exhaustion marked by PD-1/PD-L1 upregulation. This study evaluated the impact of PD-L1 blockade with a humanized monoclonal antibody, atezolizumab, in BALB/c mice with experimental mycetoma. Mice were inoculated with 1×10^7 CFU N. brasiliensis in the left footpad, followed by weekly intraperitoneal anti-PD-L1 (200 μg/dose) administration from week 10 to 14 post-infection. Bacterial load, immune cell populations (flow cytometry; CD4, CD8, Ly6G), expression of PD-1/PD-L1 (flow cytometry; MFI fold-change vs. FMO controls), and histopathology by H&E in a blinded evaluation was compared across three groups: healthy controls (HC), infected controls (IC), and treated groups (αPD-L1). Anti-PD-L1 treatment reduced survival with no difference in bacterial clearance compared to untreated IC ($p<0.05$). Both IC and αPD-L1 groups showed sustained neutrophilic (Ly6G+) infiltration in infected tissue, while CD4+/CD8+ T cell ratios remained unchanged, with a predominance of T helper cells. PD-1 (lymphocytes) and PD-L1 (neutrophils) expression did not differ significantly between groups. Histopathology revealed exacerbated tissue damage in αPD-L1 mice: severe edema, necrosis, and panniculitis ($p<0.01$ vs. IC), despite comparable bacterial burdens. These findings demonstrate that PD-L1 inhibition amplifies neutrophil-driven inflammation, worsening tissue pathology without enhancing bacterial control. The absence of modulation after the PD-1/PD-L1 blockade suggests off-target immune dysregulation rather than restored T-cell function as the driver of harm. This contrasts with checkpoint inhibitor benefits in cancer, highlighting context-dependent risks in chronic infections. Clinically, these results caution against PD-1/PD-L1 blockade in granulomatous diseases dominated by neutrophilic inflammation, where responses may override regulatory pathways critical for limiting immunopathology. By linking PD-L1 signaling to inflammation restraint in mycetoma, this study underscores the dual role of immune checkpoints in infection control and tissue preservation, advocating for pathogen-specific assessments before immune checkpoint inhibitors therapeutic modulation.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Immune dysregulation and bacterial structural adaptation in chronic Nocardia brasiliensis murine mycetoma

Ruiz-de la Cruz, María Luisa [1]; Vázquez-Marmolejo, Anna Velia [1]; Mejía-Torres, Manuel Guadalupe [1]; Castro-Corona, María de los Ángeles [1]; García-Baez, Jesús Ángel [1]; Salinas-Carmona, Mario César [1].

[1]Department of Immunology, School of Medicine and Dr. Jose Eleuterio Gonzalez University Hospital, Universidad Autonoma de Nuevo León, Monterrey, Mexico.

E-mail: mluisardlc@gmail.com

Actinomycetoma is a chronic granulomatous infection of skin and subcutaneous tissues, characterized by tumor-like swellings, draining sinus tracts, and pathogen-laden grains. Despite clinical efforts for early diagnosis, patients ask for medical treatment after years, allowing progression to irreversible tissue damage. This study analyzes cellular immune changes and bacterial grain dynamics during chronic Nocardia brasiliensis mycetoma in BALB/c mice over one year. Mice were inoculated with 1×10^7 CFU N. brasiliensis in the left footpad, with lesion volume, bacterial load, lymphoid/myeloid cell populations (flow cytometry: CD3, CD4, CD8, CD45, Ly6G, F4/80), serum cytokines and histopathology by morphometry, assessed at 70-, 100-, and 365-days post-infection (dpi). Lesion volume stabilized by 100 dpi, while splenomegaly was present during chronicity. Infected tissue showed massive neutrophilic infiltration (90–95% of total leukocytes), progressive macrophage decline, and sustained T-cell recruitment dominated by CD4+ T cell subsets (71–78% of total T cells). Systemic cytokine revealed transient IL-6 elevation and reduced MCP-1. Histopathology confirmed progressive necrosis, microabscesses, and bacterial grain accumulation, with N. brasiliensis grains adopting a circular morphology over time, suggesting structural adaptation immersed in the inflammatory infiltrate. Statistical analyses highlighted intergroup differences in immune and bacterial parameters. Chronic mycetoma is marked by dysregulated immunity: IL-6 elevation in serum, persistent neutrophilic inflammation and CD4+ T-cell skewing, with tissue destruction despite bacterial load stabilization. This remarkable finding is characteristic of an acute inflammation pattern in a chronic infection. The morphological shift in bacterial grains implies a pathogen evasion strategy, potentially shielding bacterium from host defenses. These significant findings underscore the dual role of maladaptive immune responses and bacterial adaptation in sustaining chronic infection, offering insights into therapeutic targets. By integrating longitudinal immune profiling with bacterial dynamics, this model bridges mechanistic gaps in mycetoma chronicity, emphasizing the need for early intervention to counteract immune-driven pathology and pathogen resilience.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

A Long-Term Culture Model for Investigating Senescence-Associated Dysregulation in Macrophages

Ruiz, Andy; Lucero-Gil, María Guadalupe; Torres, Martha;
Juárez, Esmeralda.

[1]Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas, Laboratorio de Alta Contención Biológica (LACBio), Tlalpan, Ciudad de México, México.. [2]Facultad de Ciencias, Universidad Nacional Autónoma de México, Ciudad de México, México. . [3]Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas, Laboratorio de Inmunobiología de la Tuberculosis (LIT), Tlalpan, Ciudad de México, México.

E-mail: doreydna@gmail.com

Aging is associated with low-grade chronic inflammation, known as inflammaging, which is partly driven by senescent cells. In this study, we developed a long-term culture model of human macrophages to investigate how aging dysregulates innate immune responses. Peripheral blood mononuclear cells (PBMCs) were isolated from healthy donors, and CD14+ monocytes were enriched by magnetic separation. These cells were differentiated into macrophages and maintained in culture for 7, 14, and 21 days. Cellular senescence was assessed through β-galactosidase, γH2AX, and p16INK4a staining. Flow cytometry and cytokine secretion analysis revealed a progressive acquisition of senescence markers, such as a significant increase in SA-β-gal activity ($p < 0.05$) and the accumulation of DNA damage markers like H2AXpS139 ($p < 0.05$) after 21 days. Phenotypically, senescent macrophages exhibited alterations in surface marker expression, such as CD14, CD64, and TLR2, transitioning from an M2 anti-inflammatory profile (present at 60% on day 7) to an M1 pro-inflammatory profile. Upon stimulation with LPS, on day 7, macrophages produced a robust cytokine profile (TNF-α, GM-CSF, IL-12p70, among others). In contrast, there was a significant reduction of IL-2, IL-12, IFN-g, IL-4, IL5, and GM-CSF ($p < 0.01$) and increased production of IL-10 and TNF- α on day 21 compared to day 7.

In addition, macrophages from days 14 and 21 (senescent macrophages) exhibited an increased secretion of senescence-associated secretory phenotype (SASP) factors such as IL-8 and MCP-1 ($p < 0.05$). These results suggest that macrophage senescence contributes to immune dysfunction observed during aging. This long-term culture model provides a valuable tool for studying the cellular mechanisms underlying immunosenescence and may serve as a platform for therapeutic interventions to restore innate immune function in older individuals.

Keywords: Senescence, macrophages, immune dysregulation, inflammation, M1 polarization, flow cytometry

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Recombinant Scorpion Toxin Targeting Ion Channels as a Strategy Against Breast Cancer

Saad-Manzanera, María Isabel, [1]; Mata-de los Ríos, Natalia, [1];
Gastelum-Arellanez, Argel, [1]; Clement, Herlinda, [2]; Alvarado-González, Karely Carolina, [1]; Corzo, Gerardo, [2]; Espino-Solís, Gerardo Pavel, [3].

[1]Facultad de Medicina y Ciencias Biomédicas, Autonomous University of Chihuahua, Circuito Universitario s/n, Campus II, Chihuahua 31125, México. [2]Instituto de Biotecnología—UNAM, Universidad Nacional Autónoma de México, Av. Universidad 2001, Col. Chamilpa, Cuernavaca 62210, México. [3]Laboratorio Nacional de Citometría de Flujo, Facultad de Medicina y Ciencias Biomédicas, Autonomous University of Chihuahua, Circuito Universitario s/n, Campus II, Chihuahua 31125, México.

E-mail: mariasaad9@gmail.com

Breast cancer continues to represent the most formidable oncological challenge among women globally, largely due to therapeutic resistance and the deleterious effects of conventional treatments. Increasingly, ion channels have emerged as pivotal regulators of cancer biology, orchestrating key processes such as proliferation, apoptosis, migration, and metastasis. Voltage-gated potassium (Kv) and sodium (Nav) channels are now recognized as integral components of oncogenic signaling networks. Capitalizing on nature's molecular elegance, this study investigates the therapeutic promise of recombinant venom peptides derived from Chihuahuanus coahuilae, a scorpion species native to northern Mexico. These bioactive peptides, known for their exquisite selectivity in modulating ion channel activity, were isolated through chromatographic and mass spectrometric techniques, and evaluated in breast cancer cell lines. Functional assays reveal that the peptides disrupt cancer cell homeostasis by modulating ion flux, inducing apoptosis, and suppressing invasive phenotypes. Preliminary data position these venom-derived molecules as potent and selective anticancer agents. Further elucidation of their molecular targets may illuminate a new paradigm in targeted breast cancer therapy—one defined by precision, elegance, and biotechnological finesse.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

The ITIM domain of CD5 modulates Th1 versus Th17 T cell differentiation by regulating cytokine dependent STAT signaling.

Saint Martin-Castellanos, Abril; Acevedo-Domínguez, Naray;
Alvarado-Luis, Brenda; Hernández-Campos, Oscar; Soldevila-Melgarejo, Gloria.

[1]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, UNAM, CU, Cd México. México. [2]Laboratorio Nacional de Citometría de Flujo, Instituto de Investigaciones Biomédicas, UNAM, CU, Cd México. México.

E-mail: abril.saintmartin@iibiomedicas.unam.mx

The immunoregulatory molecule CD5 is expressed on thymocytes and mature T cells. It contains an immunoreceptor tyrosine-based inhibitory motif (ITIM) within its cytoplasmic domain, which plays a crucial role in modulating T cell receptor (TCR) signaling thresholds. Given the established influence of TCR signaling strength on helper T cell differentiation, this study investigated the functional significance of tyrosines 429 and 441 within the CD5 ITIM domain in regulating Th1 versus Th17 effector T cell differentiation. Utilizing genetically modified murine models and in vitro polarization assays, we demonstrated that the abrogation of tyrosine residues 429 and 441 within the CD5 ITIM motif enhances Th1 effector differentiation in a 50% while simultaneously reducing Th17 lineage development by 50%. Flow cytometry analysis results indicate changes in signaling cascades downstream of TCR activation, particularly involving members of the STAT transcription factors family. Our results indicate that inhibition of the ITIM domain of CD5 reduces STAT3 phosphorylation by 45% at 72 hours after the induction of in vitro Th17 differentiation, highlighting the importance of the CD5 ITIM domain in T cell fate specification, which may have implications for the modulation of immune responses in autoimmunity and cancer immunotherapy.

Work supported by Conacyt Grant #253274

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

An update of the description of immunoglobulin loci in Ambystoma mexicanum

Saint Remy-Hernández, Stephanie; Pacheco-Olvera, Diana; Godoy-Lozano, Ernestina Elizabeth; Miguel -Ruíz, Juan Antonio; Valdovinos-Torres, Humberto; Téllez-Sosa, Juan Mauricio; Rivas-Hernández, Nancy; López-Macías, Constantino III Roberto; Martínez-Barnetche, Jesús.

[1]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional. Prolongación de Carpio y Calle Plan de Ayala s/n, Santo Tomás, Miguel Hidalgo, 11340 Ciudad de México, CDMX. [2]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional. Prolongación de Carpio y Calle Plan de Ayala s/n, Santo Tomás, Miguel Hidalgo, 11340 Ciudad de México, CDMX. [3]Instituto Nacional de Salud Pública. Centro de Investigación sobre enfermedades infecciosas. Avenida Universidad 655, Santa María Ahucatitlán 62 100 Cuernavaca, Morelos. [4]Centro Nacional de Investigación y desarrollo tecnológico. Interior Internado Plamira s/n, col. Palmira, C.P 62490, Cuernavaca, Morelos. [5]Instituto Nacional de Salud Pública. Centro de Investigación sobre enfermedades infecciosas. Avenida Universidad 655, Santa María Ahucatitlán 62 100 Cuernavaca, Morelos. [6]Instituto Nacional de Salud Pública. Centro de Investigación sobre enfermedades infecciosas. Avenida Universidad 655, Santa María Ahucatitlán 62 100 Cuernavaca, Morelos. [7]Instituto Nacional de Salud Pública. Centro de Investigación sobre enfermedades infecciosas. Avenida Universidad 655, Santa María Ahucatitlán 62 100 Cuernavaca, Morelos. [8]Hospital de Especialidades Centro Médico Nacional Siglo XXI. Av. Cuauhtémoc 330 Col. Doctores, 06020 Ciudad de México, CDMX. Tel: 55 5627 6900 ext. 21476 E-mail: constantino@sminmunologia.mx [9]Instituto Nacional de Salud Pública. Centro de Investigación sobre enfermedades infecciosas. Avenida Universidad 655, Santa María Ahucatitlán 62 100 Cuernavaca, Morelos E-mail: jmbarnet@insp.mx ; stephstremy2691@gmail.com

Ambystoma mexicanum genome has been sequenced since 2018,. It is ten times larger than the human genome, and one of the largest animal genomes sequenced so far (32 Gb) and most of the scaffolds have been linked to 14 chromosomes. However, its assembly has been a challenge given as a resulting in a new public assembly UKY_AMEXF1_1, where 26937 (99.1%) unmapped scaffolds are mapped to chromosomes. We published a genomic characterization of immunoglobulin loci in A. mexicanum. We present an update to this description based on the most recent genome version. The IGH locus was mapped at 350-364 Mbp in chr 13q; syntenic relationships are preserved across different genome assembly versions, and we confirm the misassembly of the last version, given that all the IGHV genes are in the same orientation as the rest of the locus. The locus is composed of 87 IGHV genes, 73% were functional, 15% have unusually long V-introns (>150 bp). No differences in the new annotation were observed on IGHD, IGHJ, and IGHC. The lambda chain, we identified significant differences compared to the previous annotation. A total of 13 IGLC-IGLJ clusters comprising 60 IGLJ genes. The sigma chain (Chr 1p) retains the previously described structure. We found a single IGVK pseudogene in chr6 in the homologous region where locus kappa is located in Xenopus. No evidence of kappa transcription in the spleen transcriptome was found, confirming that the kappa locus was lost. This study confirms previous observations but provides an accurate assembly and annotation of the Ig locus in A. mexicanum with the UKY_AMEXF1_1 version, where the genes are in the same orientation as biologically expected. The lambda chain annotation has a significant change in the number of the IGLC-IGLJ genes. This version could help the study of immune response evolution in the axolotl.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Characterization of tumor-infiltrating regulatory T cells

Salcedo-Magaña, Patricia [1 3]; Albarrán-Godínez, Adrián [2 3];
Suarez-Rojas, Gerardo [2 3]; Rubio, Rosa M. [3]; Madera-Salcedo,
Iris K. [3]; Rosetti, Florencia [3]; Crispín, José C. [3 4].

[1]Universidad Nacional Autónoma de México (UNAM), Posgrado en Ciencias Bioquímicas, Coyoacán, Ciudad de México, México. [2]Universidad Nacional Autónoma de México (UNAM), Posgrado en Ciencias Biomédicas, Coyoacán, Ciudad de México, México. [3]Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán (INCMNSZ), Departamento de Reumatología e Inmunología, Laboratorio de Inmunopatología, Tlalpan, Ciudad de México, México. [4]Tecnológico de Monterrey, Posgrado de Investigación, Tlalpan, Ciudad de México, México.

E-mail: paty16sm@gmail.com

The presence of FOXP3 regulatory T cells (Treg) in the tumor microenvironment has been associated with poor clinical outcomes. However, the nature of the infiltrating Tregs, particularly whether they represent thymic-derived Tregs or CD4 T cells that undergo a Treg differentiation process locally, remains an open question. The aim of this work was to determine the origin of tumor-infiltrating FOXP3 CD4 T cells (Tregs). B16 murine melanoma cells that express ovalbumin (B16-OVA) were inoculated into CD45.1 wild-type B6 mice. OVA-specific (OT-II) CD4 CD45.2 FOXP3GFP/RFP cells (naïve or activated) were adoptively transferred. CD4 T cell migration into the tumor and tumor draining lymph nodes was analyzed. FOXP3 expression in transferred OVA-specific cells was quantified. Migration of adoptively transferred naïve OT-II cells into the tumor was negligible, regardless of the number of transferred cells. Virtually, most of the FOXP3 CD4 T cells found in the tumor represented endogenous Helios+ thymic-derived Tregs. OT-II cells recovered from tumor draining lymph nodes were mostly FOXP3 negative. Activated OT-II cells exhibited a higher tendency to infiltrate B16-OVA tumors but did not acquire FOXP3 expression. Conversely to the observed when transferring naïve or activated OT-II cells to tumor-bearing wild-type B6 mice, migration of these cells into the tumor was significant when transferring them into B16-OVA tumor-bearing RAG-1/2 knockout mice. Our results indicate that (1) naïve cell migration into tumors is negligible; (2) migration of activated CD4 T cells into tumors depends on signals acquired during priming, presumably in draining lymph nodes. It will be important to identify the specific requirements that allow CD4 T cells to infiltrate tumors.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Generation of Stable CD19+ Cells to Identify Anti-CD19 scFv for CAR-T”

Salgado-López, Adrián [1]; Rodríguez-Flores, Sofía N. [1]; Gómez-Castellano, Keyla M. [1]; Almagro, Juan C.[1 2]; Pérez-Tapia, Sonia M. [1].

[1]Instituto Politécnico Nacional, UDIBI, ENCB, México City, México. [2] GlobalBio, Inc., Cambridge, MA , USA.

E-mail: adriisalgadool@gmail.com

Cancer is a global public health concern. In 2022, the World Health Organization (WHO) reported approximately 20 million new cases and nearly 10 million deaths related to the disease. Among pediatric cancers, acute lymphoblastic leukemia (ALL) is the most commonly diagnosed worldwide. Therapeutic approaches include chemotherapy, radiotherapy, and immunotherapy. The latter comprises strategies such as monoclonal antibodies, chemokines, and adoptive cell therapies, including chimeric antigen receptor (CAR) therapies using T cells (CAR-T) or natural killer cells (CAR-NK). One of the essential components of CARs is the antigen recognition domain, typically derived from single-chain variable fragments (scFvs). Currently, approved CD19-targeting CAR-T therapies rely on murine-derived scFvs such as FMC63. Although effective, these may elicit immune responses in patients. In contrast, human-derived scFvs may reduce immunogenicity and potentially enhance therapeutic safety and efficacy. Therefore, developing strategies for the discovery of fully human scFvs against CD19 is a key step toward advancing safer and more effective CAR-T therapies. In this context, semi-synthetic libraries of human origin constitute a widely used and effective approach for identifying scFvs against clinically relevant targets such as CD19, a surface antigen highly expressed in B-cell malignancies, including ALL. To enable the identification of human anti-CD19 scFvs via phage display, we established a stable CD19-expressing cell line. These cells serve as a platform for performing cell-based biopanning and for selecting specific binders from the phage library. This approach may facilitate the development of more effective and less immunogenic CAR-T cell therapies for the treatment of ALL and other B-cell malignancies.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Antipruritic effect of Agave tequilana fructans in experimental models of allergic itch

Salinas-Guardado, Valeria [1]; Jiménez-Vargas, Mariela [1];
Cervantes-García, Daniel [1 2]; Córdova-Dávalos, Laura E [1];
Salinas-Miralles, Eva [1].

[1]Autonomous University of Aguascalientes, Department of Microbiology, Aguascalientes, México. [2]Secretariat of Science, Humanities, Technologies and Innovation, México City, México.

E-mail: valeriasg.0204@gmail.com

Pruritus is a condition characterized by itching that significantly reduces patients quality of life. It occurs in various allergic pathologies, including urticaria and dermatitis. Stimuli from the skin microbiome, allergens, or molecules released by skin-resident cells activate itch receptors on cutaneous nerve fibers, transmitting signals to the spinal cord and brain, thereby triggering the itching sensation. Continuous research efforts are required to identify effective treatments for pruritus in allergies. Recent studies suggest that prebiotics, such as Agave tequilana fructans (AF), which are widely produced in Mexico, may offer a potential therapeutic alternative due to their immunomodulatory properties. This study aimed to evaluate the antipruritic effects of orally administered AF (0.1, 1, 5, and 10 g/kg) in acute (AP) and chronic (CP) pruritus in male C57BL/6J mice. AP was induced via intradermal injection of C48/80 (1.2 µg/µL) into the nape of the mice, while dry skin CP was triggered by topical application of acetone:ether:water. Scratching events were quantified using LinceWin10 over a 20-minute period for AP and 30- and 60-minutes for CP. Scratching behavior was classified into short and long durations in the latter. Oral administration of AF at 0.1, 1, and 10 g/kg for 11 days downregulated scratching events in the CP model when evaluated over a 30-minute period. While doses of 0.1 and 10 g/kg of AF significantly reduced the number of scratching events at 60 minutes. When scratching behavior was classified by duration, all AF doses significantly decreased the frequency of long-lasting scratching events. Although low-dose AF slightly attenuated itching in mice with AP induced by C48/80, the effect was not statistically significant. These results suggest that treatment with AF may alleviate CP in allergic condition characterized by dry skin, such as atopic dermatitis, mainly by reducing itching intensity.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Anti-inflammatory and immunomodulatory effects of Agave tequilana fructans in experimental atopic dermatitis

Salinas-Miralles, Eva [1]; Ríos-Carlos, Marcela [1]; Bermúdez-Humarán, Luis G [2]; Cervantes-García, Daniel [1 3]; Jiménez-Vargas, Mariela [1]; Córdova-Dávalos, Laura Elena [1]; Verduzco-Grajeda, Lidia Elizabeth [4]; Enríquez-Medrano, Francisco Javier [5]; Fabela-Sánchez, Omar [3 5].

[1]Department of Microbiology, Basic Science Center, Autonomous University of Aguascalientes, Mexico. [2]INRAE, Université Paris-Saclay, AgroParisTech, Micalis Institute, Jouy-en-Josas, France. [3]Secretariat of Science, Humanities, Technologies and Innovation, Mexico City, Mexico. [4]Technological University of Northern Aguascalientes, Rincón de Romos, Mexico. [5]Center for Research in Applied Chemistry, Saltillo, Mexico.

E-mail: maria.salinas@edu.uaa.mx

Atopic dermatitis (AD) is a skin allergic disease characterized by pruritus, inflammatory lesions, and cutaneous and intestinal dysbiosis. Agave fructans are indigestible by human enzymes. They are metabolized by intestinal microbiota and then producing substances with anti-inflammatory activity, such as short chain fatty acids (SCFAs). We examined the effect of rat supplementation with Agave tequilana fructans on the allergic response of AD. AD-like lesions were induced in the ear of animals and fructans (0.1, 1, 5g/kg) were orally administered for 13 days. Inflammation, pruritus, gene expression of transcriptional factors of immune response, and staphylococcal colonization were evaluated. Cytokine expression, relative abundance of main bacterial phyla and genera, and levels of SCFAs were analyzed at intestinal milieu. Treatment with 0.1g/kg fructans significantly decreased the ear thickness induced 1 and 6h after allergen application, without change at 24h. Likewise, the ear edema generated 1h after allergen challenge was reduced and the NF-κB inflammatory pathway was inhibited. Fructans at 0.1g/kg reduced serum IgE levels to control values. Dermal mast cell and eosinophil number, and epidermal thickening were significantly decreased with all fructan doses, although the effect was improved at 0.1g/kg. Scratching events remain unchanged in animals. The amount of S. aureus in the lesions was decreased after treatment with fructans, and S. epidermidis levels were restored to control values. Fructan treatment reduced the up-regulated expressions of Gata3, Rorc, Il4 and Il17a at lesional skin while prompted a regulatory profile at mesenteric lymph-nodes with high expression level of Foxp3, Il10, Tgfb1. Fructans at 0.1 and 1g/kg increased fecal abundance of Bifidobacterium and cecal levels of acetic acid; while doses of 1 and 5g/kg up-regulated Firmicutes, Lactobacillus and propionic acid. All doses decreased Proteobacteria abundance. Thus, agave fructans have therapeutic effects on AD through skin and gut microbiota modulation, without a dose-response effect.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Removing IgG from excreted/secreted helminth products enhances their antitumor effects

Sánchez-Barrera, Cuauhtémoc del Ángel [1]; Mendoza-Rodríguez,
Mónica Graciela [1]; Carrillo-Pérez, Jazmín Areli [1]; Fernández-
Muñoz, Karen Vanessa [1 2]; Terrazas-Valdés, Luis Ignacio [1 3].

[1]Facultad de Estudios superiores Iztacala, Unidad de Investigación en Biomedicina, Tlalnepantla, Estado de México, México. [2]Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional, Departamento de Biomedicina molecular, Ciudad de México, México. [3]Facultad de Estudios superiores Iztacala, Laboratorio Nacional en Salud, Tlalnepantla, Estado de México, México.

E-mail: angel.casb@hotmail.com

Colorectal cancer (CRC) is the third most common cancer worldwide and the second leading cause of cancer-related deaths, with rising incidence among individuals under 50, especially in high-income countries. The inflammatory response triggered by pathogens, environmental and dietary factors, and IBD can promote the formation of colorectal tumors. The hygiene hypothesis suggests a negative correlation between chronic inflammatory diseases, including inflammation-related cancer, and helminth infections. Helminths such as Taenia crassiceps exhibit antitumor effects. In the AOM/DSS-induced CRC model, prophylactic treatment with Taenia crassiceps excreted/secreted products (TcES) inhibited tumor formation. Notably, when TcES is combined with the chemotherapy drug 5-FU this effect is enhanced. The antitumor effects of TcES include immunoregulation and direct alterations to cancer cells. However, a limited understanding of its composition poses challenges for analyzing its properties. In this study, TcES was fractionated to investigate its interaction with CRC cells. Immunofluorescence assay demonstrated that TcES binds to the human CRC cell line HCT116 and inhibits its proliferation. Further analysis revealed that TcES consists of a mixture of parasite and host proteins, including mouse immunoglobulin G (IgG). Using affinity chromatography, TcES was separated into IgG-free TcES. SDS-PAGE indicated that IgG masked low-abundance proteins in TcES, suggesting that the presence of IgG could reduce the effect of TcES. To assess the activity of the fractions, dose-response curves were performed in HCT116 cells, demonstrating that the IgG-free TcES fraction promotes morphological changes associated with cellular stress, as well as a reduction in cell number compared to the control. Notably, these effects were observed at lower doses than those required for the total TcES. In summary, these results suggest that host proteins may interfere with the antitumor effects of TcES. Notably, IgG and removal enhance these effects. This process will also aid in the characterization of TcES proteins.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Expression of anti-CD19 chimeric antigen receptor (CAR) by neutrophil-like cells

Sánchez-Centeno, Luis Leonardo; López-Arredondo, Alejandra [1];
Cruz-Cárdenas, José Antonio [1]; Brunck, Marion Emilie Genevieve [1 2 3].

[1]Escuela de Ingeniería y Ciencias, Tecnológico de Monterrey, Monterrey, Nuevo León, México. [2]Escuela de Medicina y Ciencias de la Salud, Tecnológico de Monterrey, Monterrey, Nuevo León, México. [3]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, CDMX, México.

E-mail: a00840936@tec.mx

About 90% of patients receiving lymphodepletion chemotherapy exhibit profound neutropenia1 and 25% of patients experience life-threatening infections following CAR-T therapy2,3. With the advent of CAR cell therapies, it may be a possibility to replenish circulating neutrophil concentrations with cells that may also fight of the malignancy. Here we present the production of an anti CD19 CAR-expressing neutrophil cell line as proof-of-concept for the development of an adjunct treatment in neutropenic patients with B-cell malignancies. We used a third-generation plasmid system to produce lentiviral particles in HEK-293T cells. Lentiviral particles were then used to transduce HEK-293T cells with 5.7% transformed cells at 24h post-infection, 26% at 48 hrs and 28.8% at 72h, as validated by GFP expression measured with flow cytometry. We are currently transducing HL-60 cells which can be differentiated towards neutrophil-like cells following stimulation with 1.3% DMSO for 5 days. Transduced and differentiated neutrophil-like cells will be investigated for cytotoxic responses following co-incubation with B lymphocytes, and antimicrobial response following stimulation with pooled sera-opsonised E. coli.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Vacunas**

VLPs are a promising platform for multiepitope vaccines targeting SARS-CoV-2

Sánchez -Fragoso, Sandra Isabel [1]; Pérez-Saucedo, David Gilberto [1]; Moreno -Fierros, Leticia [1].

[1]Universidad Nacional Autónoma de México, Facultad de Estudios Superiores Iztacala Unidad de Biomedicina, Municipio Tlanepantla, Estado de México, México.

E-mail: 317341045@iztacala.unam.mx

Since late 2019, the COVID-19 pandemic, caused by SARS-CoV-2, has affected over 765 million people and resulted in approximately seven million deaths. Coronaviruses can be transmitted from animals to humans, as occurred with SARS-CoV and MERS-CoV. The Spike protein of SARS- CoV-2, particularly its Receptor-Binding Domain (RBD), plays a crucial role in infection, making it a key target for vaccine development. Several vaccine platforms, including mRNA and adenovirus-based vaccines, have been developed targeting the full-length Spike protein. A novel approach involves selecting conserved RBD epitopes capable of generating neutralizing antibodies, offering an alternative for vaccine design. However, the low immunogenicity of multi- epitope vaccines remains a challenge that can be addressed through delivery systems such as Virus-Like Particles (VLPs). VLP-based vaccines show great potential due to their high immunogenicity and adaptability. An innovative modification strategy is the SPYTAG/SPYCATCHER system, which allows specific and stable attachment of RBD epitopes to VLPs, enhancing their ability to induce an immune response. In this study, a construct named M.E RBD TAG was designed with SARS-CoV-2 RBD epitopes, using the VP2 protein from human parvovirus B19 as the carrier system.

The objective of this work was to design, express, produce, assemble, and evaluate the immunogenic potential of this vaccine prototype in a murine model. C57/BL6 mice were immunized via subcutaneous, intramuscular and intranasal routes. Pulmonary washes and blood samples were collected, and analysis revealed that the construct was immunogenic, achieving high antibody titers. The results indicate that VP2-based VLPs represent a promising system for the development of multiepitope vaccines.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Butyric acid produced by Staphylococcus epidermidis attenuates the expression of IL-36γ and improves murine psoriasis

Sánchez -Torres, María Marcela [1]; Pérez-Hernández, Elizabeth [3];
Gómez-Chávez, Fernando [3]; Cancino -Díaz, Mario Eugenio [2];
Cancino -Díaz , Juan Carlos [1].

[1]Departamento de Microbiología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional. [2]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional. [3]Seccion de Estudios de Posgrado e Investigación, Escuela Superior de Medicina y Homeopatía, Instituto Politécnico Nacional.

E-mail: mariamarcelasat58@gmail.com

Psoriasis is a chronic inflammatory disease characterized by altered differentiation and proliferation of keratinocytes. The initial trigger for the inflammatory process appears to be the aberrant expression of the IL-36 family, especially the gamma isoform; therefore, controlling the expression of this cytokine may help improve psoriatic lesions. On the other hand, Staphylococcus epidermidis, the main resident of the skin, can produce short-chain fatty acids—such as butyric acid—that modulates the immune response of various immune cells. The objective of this study is to determine whether products derived from glycerol fermentation by S. epidermidis can negatively regulate the expression of IL-36 and improve the phenotype of imiquimod-induce murine psoriasis. Several commensal and clinical strains of S. epidermidis were evaluated for their capacity to ferment 2% glycerol, identifying strain 59 as the most efficient. Strain 59, when grown in glycerol, produces butyric acid. Growth supernatant from strain 59, strain 59 alone and strain 59 in combination with either glycerol or butyric acid were applied to imiquimod-induced psoriatic skin. The results show that psoriatic skin of mice are treated with S. epidermidis in combination with 2% glycerol, or treated with butyric acid alone, exhibited improvements in characteristic signs of psoriasis, such as delayed acanthosis, parakeratosis, and cellular infiltration. In addition, a reduction in mRNA and IL-36 gamma protein expression was observed. These results strongly suggest that the butyric acid produced by S. epidermidis through glycerol fermentation has an immunomodulatory effect on psoriatic skin and that this molecule negatively regulates IL-36 gamma expression, resulting in an improvement of the psoriatic phenotype.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Alergias y autoinmunidad**

Glycomacropeptide modifies staphylococcal abundance and adhesion in experimental atopic dermatitis

Sánchez-Villalobos, Carlos Alberto [1]; Gallegos-Alcalá, Pamela [1];
Muñoz-De la Cruz, Fabiola C [1]; Mercado-Aguilera, Germán [1];
Cervantes-García, Daniel [1 2]; Jiménez-Vargas, Mariela [1];
Córdova-Dávalos, Laura E [1]; Salinas-Miralles, Eva [1].

[1]Department of Microbiology, Basic Science Center, Autonomous University of Aguascalientes, México. [2]Secretariat of Science, Humanities, Technologies and Innovation, México City, México.

E-mail: charlos.sav@gmail.com

Atopic dermatitis (AD) is an allergic skin disease characterized by eczematous lesions, dry skin, and cutaneous dysbiosis. Lesional skin presents reduced microbial diversity and the overgrowth of Staphylococcus aureus which exacerbates inflammation. Commensal bacteria, such as Staphylococcus epidermidis, may exert protective effects by inhibiting S. aureus colonization and modulating immune responses. Glycomacropeptide (GMP) is a 64-amino-acid bioactive peptide released from milk κ-casein through the action of digestive enzymes or chymosin. Oral administration of GMP has shown immunomodulatory activities in experimental AD. This study aimed to evaluate whether oral administration of GMP modifies the abundance of S. aureus in relation to S. epidermidis in AD-like lesions in rats and whether it alters bacterial adhesion to cultured keratinocytes. AD-like lesions were induced in Wistar rats and GMP (500 mg/kg/day) was orally administered prophylactically (AD-GP, 39 days) or therapeutically (AD-GT, 14 days). Animals with AD and orally administered with water were considered as disease control (AD-WP and AD-WT). Genomic DNA was extracted from skin samples and S. aureus and S. epidermidis were quantified by qPCR analysis of femA gene. In vitro assays of bacterial adhesion (MOI 10) to HaCaT cells were performed and the total number of adherent and internalized bacteria was evaluated by UFC and femA qPCR. In animals without AD, S. aureus was 2.6-times less abundant than S. epidermidis. In AD-lesions, the number of copies of S. aureus was 4.85-fold (AD-WP) and 3.96-fold (AD-WT) higher than those of S. epidermidis. GMP administration clearly showed a tendency to decrease the ratio of S. aureus/S. epidermidis in the AD-GP and AD-GT groups. In vitro, GMP treatment significantly increased S. epidermidis and decreased S. aureus adhesion to HaCaT cells, the latter in a concentration-dependent manner. Results suggest that GMP has potential to revert staphylococcal dysbiosis at AD lesional skin.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Development of an innovative carbon nanotubes-based nanoconjugate for ovarian cancer immunotherapy

Sánchez-Ramírez, Blanca Estela [2]; Guzmán-Mendoza, José Jesús [1]; Velarde-Calderón, Alejandro [1]; Jiménez, Alondra [1]; García-Campusano, Carolina [3]; Orrantia-Borunda, Erasmo [4]; Talamás-Rohana, Patricia [1].

[1]Centro de Investigación y de Estudios Avanzados, Departamento de Infectómica y Patogénesis Molecular, CDMX, México. [2]Universidad Autónoma de Chihuahua, Facultad de Ciencias Químicas, Chihuahua, México. [3]Universidad Nacional Autónoma de México (UNAM), Facultad de Estudios Superiores Iztacala, Laboratorio de Ingeniería Tisular y Medicina Traslacional, CDMX, México. [4]Centro de Investigación en Materiales Avanzados. Unidad Chihuahua.

E-mail: bsanche@uach.mx

Carbon nanotubes (CNTs) play a crucial role in advanced nano-vaccine development due to their bioconjugation properties and cellular uptake capabilities. In this study, we introduce an immunotherapeutic approach for ovarian cancer (OvCa) using CNTs conjugated with peptides from the overexpressed fucosyltransferase 4 (FUT4) protein. Predictive modeling identified high-affinity FUT4 epitopes that bind to MHC-I and -II; after peptide synthesis (PEP37), CNTs were bioconjugated to create f-CNTs. Physicochemical analysis confirmed conjugation, and the non-cytotoxic effect was demonstrated in vitro. To evaluate the potential of f-CNTs as a nano-vaccine, we used the immunocompetent C57BL/6J mouse model inoculated with transformed ID8 epithelial ovarian cells. FUT4 overexpression was shown in ID8 cells, notably induced by ascites stimulation, and heightened in cells from C57BL/6 mice compared to those in culture media, confirming the influence of the tumor microenvironment (TME) on FUT4 expression. Subsequent immunization of mice with f-CNTs, f-CNTs plus adjuvant, or PEP37 plus adjuvant, followed by ID8 cell inoculation, revealed significant immunoprophylactic effects from f-CNTs. These included reduced tumor development, decreased ascites accumulation, and improved overall survival, with f-CNTs exhibiting the most pronounced impact, followed by f-CNTs plus adjuvant and PEP37 plus adjuvant. *In vivo* experiments demonstrated changes in immune cell profiles within the OvCa TME, characterized by increased leukocytes, T lymphocytes, and cytotoxic T cells, alongside a shift from M2 to M1 macrophage polarization and a decrease in regulatory T cells (Tregs). Robust spleen cell proliferation was observed in mice immunized with f-CNTs, f-CNTs plus adjuvant, and PEP37 plus adjuvant, indicating the presence of T cells specific to the PEP37 antigen. Our findings highlight the ability of f-CNTs to modulate immune responses within the OvCa TME, mediated by cytotoxic T lymphocytes, M1 macrophages, and Tregs. This positions f-CNTs as a promising carrier system for exploration in OvCa immunotherapy.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Characterization of exhausted T cells in systemic lupus erythematosus

Santana-Sánchez, Paola [1 2]; Gajón, Julián A. [3]; Ramírez-Pérez,
Astrid Asminda [4]; Alberti-Minutti, Paolo [5]; Bonifaz, Laura C [3 8];
Amezcua-Guerra, Luis Manuel [6 7]; Chávez-Rueda, Adriana Karina [2].

[1]Universidad Autónoma Metropolitana, Doctorado en Ciencias Biológicas y de la Salud, Ciudad de México, México. [2]Instituto Mexicano del Seguro Social, Unidad de Investigación Médica en Inmunología, UMAE Hospital de Pediatría, CMN SXXI, Ciudad de México, México. [3]Instituto Mexicano del Seguro Social, Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, CMN SXXI, Ciudad de México, México. [4]Instituto Mexicano del Seguro Social, Servicio de Reumatología, UMAE Hospital de Especialidades, CMN SXXI, Ciudad de México, México. [5]Instituto Mexicano del Seguro Social, Servicio de Medicina Interna, UMAE Hospital de Especialidades, CMN SXXI, Ciudad de México, México. [6]Universidad Autónoma Metropolitana, Ciencias Biológicas y de la Salud, Ciudad de México, México. [7]Instituto Nacional de Cardiología Ignacio Chávez, Departamento de Inmunología, Ciudad de México, México. [8]Coordinación de investigación en salud, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México.

E-mail: pao-ss@live.com.mx

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease. Although prognosis has improved in recent decades, mortality in SLE patients remains higher than in the general population. T-cell activation contributes to the hyperreactive and chronic immune response observed in SLE. Dysregulation of cellular mechanisms may lead to an exhausted T-cell phenotype; however, analyzing this process in relation to disease activity may provide valuable insights into disease progression.

Peripheral blood samples were collected from patients with active and inactive SLE, as well as from age- and sex-matched healthy individuals. Cells were stained with markers associated with T-cell exhaustion and analyzed using a Cytek Aurora spectral flow cytometer (Cytek Biosciences, USA). Data were processed with FlowJo 10 software (Tree Star, USA).

We investigated exhausted CD8 T cells in relation to immune activity in SLE. Our analysis revealed differential expression of exhaustion markers and suggested a correlation between exhaustion profiles and disease activity status. These findings indicate that exhausted T cells may contribute to disease progression and inflammatory pathways. Moreover, we observed sustained expression of cytotoxic molecules in the exhausted CD8 T-cell subset.

Chronic inflammation in SLE promotes persistent exposure to autoantigens and continuous activation of autoreactive T-cell clones. Characterizing exhausted T cells in this context may be key for monitoring immune dysregulation and could support clinical follow-up and therapeutic strategies.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Cervical cancer mesenchymal stromal cells release PD-L1^high extracellular vesicles via adenosinergic signaling

Sandoval-Juárez, David [1 2]; García-Rocha, Rosario [1]; Molina-Castillo, Gabriela [3]; Mora -García, María de Lourdes [3]; Weiss-Steider, Benny [3]; Don-López, Christian Azucena [3]; Hernández-Montes, Jorge [3]; Díaz-Ramos, Juan Antonio [3]; Monroy-García, Alberto [1 3].

[1]Laboratorio de Inmunología y Cáncer, Unidad de Investigación Médica en Enfermedades Oncológicas, Hospital de Oncología, CMN SXXI, Instituto Mexicano del Seguro Social, Ciudad de México 06720, México. [2]Programa de Posgrado (Maestría) en Ciencias Biológicas, Universidad Nacional Autónoma de México, Facultad de Medicina, Ciudad de México 04510, México. [3]Laboratorio de Inmunobiología, Unidad de Investigación en Diferenciación Celular y Cáncer-UMIEZ, FES-Zaragoza, Universidad Nacional Autónoma de México, Ciudad de México 09230, México.

E-mail: biodasaju@comunidad.unam.mx

Cervical cancer (CeCa) is potentially immunogenic due to its association with human papillomavirus (HPV) infection. However, high PD-L1 expression in the tumor microenvironment (TME) suppresses the antitumor immune response. In previous studies, we observed that adenosine (ADO) signaling on A2A and A2B receptors in mesenchymal stromal cells derived from CeCa tumors (CeCa-MSCs) induces PD-L1 expression via the production of TGF-β1. In this study, we evaluated the effect of ADO on PD-L1 expression of extracellular vesicles (EVs) released by CeCa-MSCs. For this purpose, CeCa-MSCs were cultured in the presence of 1 mM ADO, with or without 10 μM of ZM241385 or MRS1754, specific antagonists of the A2A and A2B receptors, respectively, or SB505124, a selective TGF-β receptor-1 inhibitor. CeCa-MSCs treated with 40 ng/mL of TGF-β1 or left untreated were used as positive (PCtl) and negative (NCtl) controls, respectively. After 72 hours, supernatants were collected and EVs were isolated by sequential centrifugation. EVs were characterized by electron microscopy, and PD-L1 expression was analyzed by flow cytometry. EVs derived from CeCa-MSCs treated with ADO or TGF-β1 showed significantly increased PD-L1 expression (MFI=47,596 and MFI=39,758, respectively) compared to EVs from the NCtl group (MFI= 24,434). Notably, PD-L1 expression was markedly reduced in EVs upon treatment with ZM241385, MRS1754, or SB505124 (MFI=27,907, MFI=19,499, and MFI=14,880, respectively). These findings suggest that ADO and TGF-β in the CeCa TME may promote neoplastic progression by secreting PD-L1-enriched EVs. Therefore, inhibition of PD-L1+ EVs could represent a potencial therapeutic strategy in CeCa.

Supported by IMSS/R-2023-3602-011 and DGAPAPAPIIT No. IN216325, grants; and SECIHTI-scholarship to CVU No:2048151.

ISSN: 2007-3380

Revista Bio Ciencias

https://revistabiociencias.uan.edu.mx

Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Characterization of exhausted T cells in systemic lupus erythematosus

Santana-Sánchez, Paola [1 2]; Gajón, Julián A. [3]; Ramírez-Pérez, Astrid Asminda [4]; Alberti-Minutti, Paolo [5]; Bonifaz, Laura C [3 8]; Amezcua-Guerra, Luis Manuel [6 7]; Chávez-Rueda, Adriana Karina [2].

[1]Universidad Autónoma Metropolitana, Doctorado en Ciencias Biológicas y de la Salud, Ciudad de México, México. [2]Instituto Mexicano del Seguro Social, Unidad de Investigación Médica en Inmunología, UMAE Hospital de Pediatría, CMN SXXI, Ciudad de México, México. [3]Instituto Mexicano del Seguro Social, Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, CMN SXXI, Ciudad de México, México. [4]Instituto Mexicano del Seguro Social, Servicio de Reumatología, UMAE Hospital de Especialidades, CMN SXXI, Ciudad de México, México. [5]Instituto Mexicano del Seguro Social, Servicio de Medicina Interna, UMAE Hospital de Especialidades, CMN SXXI, Ciudad de México, México. [6]Universidad Autónoma Metropolitana, Ciencias Biológicas y de la Salud, Ciudad de México, México. [7]Instituto Nacional de Cardiología Ignacio Chávez, Departamento de Inmunología, Ciudad de México, México. [8]Coordinación de investigación en salud, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Ciudad de México, México.

E-mail: pao-ss@live.com.mx

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease. Although prognosis has improved in recent decades, mortality in SLE patients remains higher than in the general population. T-cell activation contributes to the hyperreactive and chronic immune response observed in SLE. Dysregulation of cellular mechanisms may lead to an exhausted T-cell phenotype; however, analyzing this process in relation to disease activity may provide valuable insights into disease progression.

Peripheral blood samples were collected from patients with active and inactive SLE, as well as from age- and sex-matched healthy individuals. Cells were stained with markers associated with T-cell exhaustion and analyzed using a Cytek Aurora spectral flow cytometer (Cytek Biosciences, USA). Data were processed with FlowJo 10 software (Tree Star, USA).

We investigated exhausted CD8 T cells in relation to immune activity in SLE. Our analysis revealed differential expression of exhaustion markers and suggested a correlation between exhaustion profiles and disease activity status. These findings indicate that exhausted T cells may contribute to disease progression and inflammatory pathways. Moreover, we observed sustained expression of cytotoxic molecules in the exhausted CD8 T-cell subset.

Chronic inflammation in SLE promotes persistent exposure to autoantigens and continuous activation of autoreactive T-cell clones. Characterizing exhausted T cells in this context may be key for monitoring immune dysregulation and could support clinical follow-up and therapeutic strategies.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

Glial dysfunction links chronic Toxoplasma gondii infection to spatial memory deficits

Sepúlveda -Cuéllar, Rodrigo Daniel [1 2]; García-Ruiz, Ricardo [3]; Soria-Medina, Diego Alberto [2]; García-Michaus, Valeria [2]; Luna-Pastén, Héctor [4]; Cañedo-Solares, Irma [4]; Gómez-Chávez, Fernando [5]; Molina -López, Liliana [2]; Correa-Beltrán, María Dolores [2].

[1]UNAM, Programa de Doctorado en Ciencias Biomédicas, CDMX, México. [2]Universidad Anáhuac, Facultad de Ciencias de la Salud. México, México. [3]Facultad de Medicina, Laboratorio de Endocannabinoides, CDMX, México. [4]Instituto Nacional de Pediatría, Laboratorio de Inmunología Experimental, CDMX, México. [5]Escuela Nacional de Medicina y Homeopatía, Laboratorio de Enfermedades Osteoarticulares e Inmunológicas, CDMX, México.

E-mail: danielsepulveda71@gmail.com

Chronic Toxoplasma gondii infection, affecting approximately 30% of humans, is associated with neuropsychiatric disorders; however, little is understood about how the neuroimmune response alters memory processes during T. gondii infection. Therefore, in this study, we examined the effects of chronic infection on hippocampal-dependent spatial navigation and its relationship with neuroimmune responses, using a murine model. Ten- to twelve-week-old male BALB/c mice (non-exposed, n = 6; exposed, n = 9) were inoculated i.p. with 1x10^4 tachyzoites of the non-virulent ME49 strain. Eight weeks post-infection, spatial memory was evaluated using the Barnes maze test, while GFAP (astrocytes), IBA-1 (microglia), and BDNF (brain-derived neurotrophic factor) were examined in the brains using confocal microscopy. Infection status was confirmed by detecting anti-T. gondii IgG antibodies in ELISA. Some exposed mice did not seroconvert, resulting in three experimental groups: non-exposed (n = 6), seronegative (n = 4), and seropositive (n = 5). The seropositive group exhibited spatial memory deficits, including prolonged latency to locate the escape hole and reduced search precision, along with neuroinflammatory changes such as hippocampal astrocytosis, increased activated microglia (with ameboid, hypertrophied, and bushy morphologies), and decreased BDNF. In contrast, the seronegative group displayed only mild memory impairment and a weaker neuroinflammatory response than seropositive mice. Strikingly, higher total IgG levels correlated with improved maze performance. Our findings suggest that chronic T. gondii infection induces morphological alterations in glial cells, which are linked to cognitive deficits and a sustained neuroinflammatory response.

Área del artículo: **Inmunología de enfermedades infecciosas**

Internalization of Candida glabrata from biofilm established on biotic surface by macrophages

Serna -Pérez, Amanda Belén [1]; García -Pérez, Blanca Estela [1].

[1]Escuela Nacional de Ciencias Biológicas, Microbiología, Ciudad de México, Ciudad de México, México.

E-mail: sernaamanda2@gmail.com

Candida glabrata (Nakaseomyces glabrata) is an opportunist yeast with a high mortality from invasive candidiasis. Recently it was included in the list of the seven high-priority human fungal pathogens by the WHO. Their virulence factors include resistance to antifungal agents, the ability to form biofilms on both biotic and abiotic surfaces, and the capacity to survive intracellularly and resist oxidative stress. The aim of this work was to evaluate the intracellular behavior of C. glabrata derived from biofilm in human macrophages and to determine the production of reactive oxygen and nitrogen species by human macrophages in response to these yeasts. For all assays, yeasts from biotic biofilm (biofilm established on epithelial cells, BB), yeasts from abiotic biofilm (BA) and planktonic yeasts (P) were used. The result showed a different pattern in the intracellular behavior of yeasts. The intracellular fungal load of the yeasts from BB was higher in contrast to the planktonic forms P and yeast from BA. Extracellular BB yeasts adhered to macrophages were observed forming yeast groups during the kinetics up to 72 h in medium with amphotericin, in contrast to the P and BA yeasts, which were observed in fewer numbers in the medium up to 48 hours. Furthermore, an increase in ROS production was observed at 4 hours post-infection with BB and BA, with no increase in those infected by P forms. Additionally, the three conditions exhibited NO production over time. Together, these results suggest that yeasts from BB showed a higher virulence that the yeasts from BA and P forms.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Toxicological profile of the immunomodulatory, antitumor, and antimetastatic peptide GK-1

Sifontes-Rodríguez, Sergio [1,2]; Cervantes Torres, Jacquelynne [3];
Hernández Aceves, Juan Alberto [1]; Moctezuma Rocha, Diego [1];
Sciutto, Edda [1]; Fragoso, Gladis [1].

[1]Universidad Nacional Autónoma de México, Instituto de Investigaciones Biomédicas, Departamento de Inmunología, UNAM, Ciudad de México, México. [2]Secretaría de Ciencias Humanidades, Tecnología e Innovación (SECIHTI), México. [3]Universidad Nacional Autónoma de México, Facultad de Medicina Veterinaria y Zootecnia, Unidad de Investigación, Ciudad de México, México.

E-mail: sifontes@iibiomedicas.unam.mx

GK-1 is an 18-amino acid peptide with demonstrated immunomodulatory, antitumor, and antimetastatic properties. This study provides a comprehensive preclinical toxicological evaluation of GK-1. In vitro cytotoxicity was assessed in tumor and non-tumor cell lines as well as primary cell cultures. In vivo toxicological assessments included single-dose toxicity in mice and repeated-dose toxicity in BALB/c mice and Wistar rats. Genotoxicity was evaluated using the Ames test (Salmonella typhimurium strains TA1535, TA100, TA102, TA98, TA1537), the in vivo micronucleus assay in Wistar rats (oral doses of 0.5–12.5 mg/kg), and the in vitro mammalian chromosomal aberration test. Dermal sensitization was examined in guinea pigs, and pyrogenicity was tested in New Zealand rabbits. GK-1 showed no cytotoxic effects in vitro at concentrations up to 500 µM in any cell type tested. No local or systemic toxicity was observed following single-dose administration in mice (up to 1000 mg/kg) or repeated-dose administration in mice (300 mg/kg) and rats (12.5 mg/kg). In the Ames test, GK-1 did not induce increases in revertant colonies at any tested concentration (0.125–2 mg/plate). Similarly, no significant changes in micronucleus frequency were observed in rat erythrocytes, nor were chromosomal aberrations induced in human cells treated with GK-1 (0.1–100 µM). Furthermore, GK-1 did not produce erythema or swelling in the dermal sensitization test and did not induce a pyrogenic response in rabbits at 0.5 mg/kg. In conclusion, GK-1 is a promising therapeutic candidate, currently under development as a novel antitumor agent for triple-negative breast cancer and exhibits an excellent preclinical safety profile across multiple toxicological endpoints.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Senescence-associated changes in electrophysiological properties of mast cells

Solares-Rioja, Eva Marcela [1]; González -Espinosa, Claudia [1];
Galván-Espinosa, Emilio Javier [1].

[1]Centro de Investigación y Estudios Avanzados del IPN. Departamento de Fatmacobiología.
Ciudad de México.

E-mail: eva.solares@cinvestav.mx

Mast cells (MC) are long-lived, tissue resident innate immune cells able to secrete distinct inflammatory mediators. They are recruited to solid tumors where they are subjected to extreme conditions (like cyclic hypoxia) found in tumor microenvironment (TME). Despite the knowledge on their participation in distinct immune reactions, changes induced by replicative and stress-induced senescence in that cell type are not fully described. Here, we analyzed the effect of senescence on electrophysiological properties of MC. To study replicative senescence, bone marrow-derived mast cells (BMMC) were generated from young C57BL6/J (WT) mice and maintained in culture for distinct times to generate mature, non-senescent (5wk-old) or senescent (13 wk-old) MC. Also, MC precursors were isolated from aged (60 wk-old) WT mice and mature BMMC were generated after 5 wk in culture to mimic "in vivo senescence". To analyze stress-induced senescence, 5 weeks-old cultures were subjected to cyclic hypoxia. We found important alterations in resting membrane potential in senescent MCs, with no important changes in other parameters. The more depolarized potential observed in senescent MC could make MC more prone to secrete inflammatory mediators. In addition, we assessed calcium influx by depolarizing the cells with KCl. In the in vivo senescence group, we observed a decreased latency to the peak calcium response compared to the control group. Furthermore, the addition of nifedipine, a selective L-type calcium channel blocker, reduced calcium entry and resulted in a prolonged latency to the maximal response in non-senescent than in senescent cells. These findings suggest that L-type calcium channels are actively involved in the altered calcium dynamics associated with senescent cells.

Supported by Conahcyt Grants CF-2019-51488 and Secihti CF-3915.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Prevalence and diagnostic approaches to autonomic dysfunction in Guillain-Barre syndrome

Solís-Gómez, Raymundo [1 2]; Hernández-Campos, María Elena [1]; Vargas-Cañas, Edwin Steven [2]; López-Hernández, Juan Carlos [2].

[1]Sección de Estudios de Posgrado e Investigación, Escuela Superior de Medicina del Instituto Politécnico Nacional. Plan de San Luis y Díaz Mirón s/n, Casco de Sto. Tomás, Ciudad de México, México. [2]Clínica de Enfermedades Neuromusculares, Instituto Nacional de Neurología y Neurocirugía “Manuel Velasco Suárez”. Ciudad de México, México.

E-mail: ray.sogo99@gmail.com

Autonomic dysfunction in patients with Guillain-Barre syndrome occurs because of inflammatory lesions caused by infiltration of lymphocytes and macrophages in the tissue surrounding endoneural blood vessels in the peripheral nervous system. This systematic review aims to determine the main affected systems and diagnostic methods reported in the literature. We conducted a search in the databases PubMed, Scopus, Web of Science, Cochrane, and Embase using keywords such as ‘Guillain-Barre syndrome’ and ‘dysautonomia’ or ‘autonomic dysfunction’ to identify original articles reporting patients with dysautonomia and Guillain-Barre syndrome. The search covered all records available from the inception of the databases until March 12, 2025. Demographic and clinical data were collected from each study. Twenty-eight studies, including 9 cross-sectional studies, 8 cohort studies, 7 case-control studies, and 2 clinical trials with a combined sample of 744 patients with GBS and dysautonomia and 10,348 controls, were included. The most affected systems were the cardiovascular system, with manifestations reported in 84.8% of patients; the gastrointestinal system, affected in 42%; the genitourinary system, with manifestations in 27%; and the sudomotor system, affected in 10%. Vasomotor and visual manifestations were reported less frequently. Regarding diagnostic methods, four studies measured catecholamine and cortisol concentrations in plasma and urine; one study performed a sudomotor test; two studies used 24-hour heart rate power spectrum analysis; three studies applied the Valsalva index; three conducted the Quantitative Sudomotor Axon Reflex Test (QSART); one reported skin biopsy; and one used the Sympathetic Skin Response (SSwR) test. This review highlights that autonomic dysfunction is a common and complex complication in Guillain-Barre syndrome, primarily impacting cardiovascular, gastrointestinal, and genitourinary systems. The high prevalence of cardiovascular involvement necessitates close monitoring to prevent severe complications. Diagnostic methods vary, and further research is needed to standardize assessments and develop targeted treatments to improve patient outcomes.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Effect of long-term exposure to particulate matter on pulmonary immune response against P. aeruginosa in a murine model

Solís-Torres, Nancy Gabriela [1]; Escudero-Lourdes, Claudia [2]; González-Curiel, Irma [3]; Rivas-Santiago, Bruno [4]; Osornio-Vargas, Álvaro [5]; Quintana-Belmares, Raúl [6]; Reyes-Escobedo, Fuensanta [7]; Rivas-Santiago, César [8].

[1]Universidad Autónoma de San Luis Potosí, Posgrado en Ciencias Farmacobiológicas, San Luis Potosí, San Luis Potosí, México. [2]Universidad Autónoma de San Luis Potosí, Laboratorio de Inmunotoxicología, San Luis Potosí, San Luis Potosí, México. [3]Universidad Autónoma de Zacatecas, Laboratorio de Inmunotoxicología y Terapéutica experimental, Zacatecas, Zacatecas, México. [4]Unidad de Investigación Biomédica del IMSS, Zacatecas, Zacatecas, México. [5]Universidad de Alberta, Departamento de Pediatría, Alberta, Edmonton, Canadá. [6]Instituto Nacional de Cancerología, Ciudad de México, México. [7]Universidad Autónoma de Zacatecas, Unidad Académica de Ciencias Químicas, Zacatecas, Zacatecas, México. [8]Investigadores por México Secretaría de Ciencia, Humanidades, Tecnología e Innovación-Universidad Autónoma de Zacatecas, Zacatecas, Zacatecas, México.
E-mail: nnancy_618@hotmail.com

Particulate Matter (PM) exposure has become a significant global public health concern. Exposure to PM has been linked to adverse health outcomes, principally in the respiratory system. Besides, several studies have shown that PM exposure impairs innate immune response mechanisms, increasing the susceptibility to infectious diseases. Among these, infections caused by Pseudomonas aeruginosa—an opportunistic pathogen ranked as the fourth leading cause of infection-related mortality globally—have gained significant relevance for study. The impact of PM exposure on opportunistic bacterial infections has not been thoroughly investigated. This study aims to examine the effect of long-term PM exposure on P. aeruginosa infection in a murine model. To determine whether long-term exposure to PM2.5 enhances pulmonary infection with Pseudomonas aeruginosa, exacerbates lung tissue damage, and induces changes in specific immune response parameters within the lung tissue of exposed mice compared to unexposed control animals. Eight-week-old BALB/c mice were grouped in: control, infection, PM, and PM + infection. Then, mice were exposed intratracheally to PM (64 µg) for 28 days. After this time, mice were infected with P. aeruginosa (1.7×10^8 CFU/mouse). Subsequently, at day 3 and 7 post-infection, mice were euthanized and blood, lungs, and other organs were collected and stored until further analysis. Long-term exposure to urban PM2.5 resulted in a significantly higher bacterial load in the lungs of PM-exposed infected mice on days 3 and 7, compared to mice infected with P. aeruginosa alone. Also, the exposure to PM2.5 induced an imbalanced inflammatory response. Long-term exposure to PM2.5 of BALB/c mice favored P. aeruginosa infection, increasing the bacterial load in the lungs of pre-exposed mice compared to unexposed controls. In addition, the pre-exposure to PM2.5 induced a pro-inflammatory environment, contributing to tissue damage and inefficient control of bacterial load.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Immunopotentiator "RTLV14-24" modulates intratumoral T cells phenotype of cancer mouce model

Solorio-Cárdenas, Manuel [1 2]; López-Macías, Constantino [1]; Lozano-Cisneros , Diego [1 3]; Fragoso Gonzáles, Gladis Del Carmen [4]; Cortés Morales Víctor Adrián [1 5].

[1]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México. [2]Facultad de Química, Universidad Nacional Autónoma de México, México City, México. [3]Posgrado en Ciencias Biológicas, Facultad de Medicina, Universidad Nacional Autónoma de México, México City, México. [4]Departamento de Inmunología, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, México City, México. [5]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City, México.

Corresponding: v.adrian.cortes@gmail.com. E-mail: manuel203b@gmail.com

Currently, breast cancer is the most prevalent and deadly cancer among female population, both globally and nationally. An increased lymphocytic infiltrate within the tumor is associated with a better prognosis, and correlates with increased survival and improved response to treatment. Consequently, therapies aimed at reactivating and enhancing the immune system have gained relevance, given the crucial role of cellular immunity in cancer control and eradication. The immunopotentiator RTLV-1424, derived from Salmonella Typhi, has been shown to enhance T lymphocyte responses, promoting the generation of T cell IFN-γ producing phenotype in response to bacterial infections. Our objective was to evaluate the ability of the immunopotentiator RTLV-1424 to modulate the phenotype of intratumoral T lymphocytes with antitumor potential in a murine breast cancer model induced by 4T1 tumor cell line. RTLV-1424 was administered intratumorally, with and without a booster dose, once the tumor was established. Tumor tissue was dissociated 28 days post-treatment, and flow cytometry was employed to assess IFN- γ expression and proliferation capacity over T cells. We observed a reduction in tumor weight after treatment with RTLV-1424. Our treatment increased the frecuency of CD45+ tumor infiltrating leukocytes, as well as CD3+ T lymphocytes and their CD4+ and CD8+ subpopulations. Interestingly, RTLV-1424 also increased the frequency of IFN-γ producing T cells in both T cell subpopulation with and without booster dose; however, it did not alter their proliferation capacity with a phytohemagglutinin stimulation in a in vitro system assessed by flow cytometry. Our preliminary results suggest that administration of RTLV-1424 induces the generation of tumor infiltrating IFN- γ producing T lymphocytes.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Neuroinmunobiología**

Microglia's dual role in neurogenesis and behavior in adult rodents: literature review

Soria -Medina, Diego Alberto [1 2]; Sepúlveda-Cuéllar, Rodrigo Daniel [3]; Cañedo-Solares, Irma [4]; Gómez-Chávez, Fernando [5]; Correa, Dolores [2].

[1]Laboratorio de Neurobiología Molecular y Celular de la Glía; Departamento de Bioquímica, Facultad de Medicina, UNAM, Ciudad de México, México. [2]Centro de Investigación en Ciencias de la Salud, Fac. de Ciencias de la Salud, Universidad Anáhuac, Huixquilucan, México. [3]Programa de Doctorado en Ciencias Biomédicas, UNAM, Ciudad de México, México. [4]Laboratorio de Inmunología Experimental, Subdirección de Medicina Experimental, INP. S.S., Ciudad de México, México. [5]Laboratorio de Enfermedades Osteoarticulares e Inmunológicas, Sección de Estudios de Posgrado e Investigación, ENMyH - IPN, Ciudad de México, México.

E-mail: diegosm2d@gmail.com

Microglia are resident macrophages in the central nervous system (CNS), existing in a quiescent state characterized by a ramified morphology, or acquire an "amoeboid state" once activated by various insults. Quiescent microglia promote neurogenesis in two ways: phagocytosing apoptotic newborn neural progenitor-stem cells (NPSC) that have not completed differentiation, and producing low levels of cytokines, like TNF-α, that stimulate NPSC proliferation, differentiation, or migration through TNFR2 directly. When activated by various insults, microglia increase their phagocytic activity, destroying the surrounding tissue and producing high levels of pro-inflammatory cytokines. In this sense, high levels of TNF-α act through TNFR1, which returns NPSCs to multipotentiality. Regarding the regulators of microglia state, the fractalkine (CX3CL) receptor, CX3CR1, is of utmost importance, since deletion of its gene or receptor blocking with antibodies reduces NPSC proliferation and differentiation. It has to be stressed that NSPCs do not express CX3CR1, emphasizing the role of the microglia. Neurogenesis in the dentate gyrus of the hippocampus and the subventricular zone-olfactory bulb axis induces changes in spatial learning, memory, and social behaviors, and most studies suggest that they are all affected by the lack of TNF-α or its receptors in knockout mouse models. Despite the need for more models on the neurogenesis in the dentate gyrus and subventricular zone-olfactory bulb and the behaviors dependent on it, microglia seem to have a dual role depending on their activation state, at least TNF-α explains this paradoxical phenomenon. The effect of other cytokines differentially produced by microglia in different states is less clear.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Alterations of endothelial progenitor cells in Mexican women with cardiovascular risk factors

Soriano -Cruz, Marina [1 2 3]; Vázquez-González, Wendy Guadalupe [1 2]; Madrid-Miller, Alejandra [4]; Puente-Barragán, Adriana [5]; Núñez-Enríquez, Juan Carlos [1]; Chávez-Rueda, Adriana Karina [1]; Chávez -Sánchez, Luis [1 2].

[1]Unidad de Investigación Médica en Inmunología, Hospital de Pediatría, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City 06720, México. [2]Unidad de Investigación Médica en Enfermedades Metabólicas, Hospital de Cardiología, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, México City 06720, México. [3]Facultad de Medicina y Cirugía, Universidad Autónoma Benito Juárez de Oaxaca, Oaxaca 68020, México. [4]Consultorio de cardiología clínica, Torre de Especialidades Médicas Tepic, México City 06720, México. Vocal del capítulo de Cardiopatías en la mujer de la Asociación Nacional de Cardiólogos de México A.C. [5]Sección Médica del Servicio de Cardiología, Centro Médico Nacional 20 de noviembre, ISSSTE, México City 03104, México. Coordinadora del Capítulo de Cardiopatías en la Mujer de la Asociación Nacional de Cardiólogos de México A.C.

E-mail: marina674@live.com.mx

Cardiovascular risk factors in women may affect the phenotype and function of endothelial progenitor cells (EPCs). Here, we analyzed the phenotype and function of EPCs of adult Mexican women with low risk and borderline-intermediate for developing cardiovascular diseases. We analyzed cardiovascular risk factors and using flow cytometry, we explored EPCs number, phenotypes, molecules associated with cardiovascular disease as well as EPCs migration. We determined that women with cardiovascular risk had a higher presence of cardiovascular risk factors than women without risk, as well as a lower expression of characteristic markers of EPCs. Also, women with risk showed a reduction in EPC colony-forming units and migration, as well as an increase in molecules inducing cardiovascular diseases in relation to women without cardiovascular risk. These findings offer new insights into the effect of cardiovascular risk factors on EPCs as targets in the homeostasis of the vasculature in women.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Mapping HIV epitopes restricted by Amerindian HLA-B*35 subtypes using IFNγ ELISpot and synthetic HAP-containing peptides

Sosa Flores, Jesús Cora [1]; Valenzuela Ponce, Humberto [1];
Soto Nava, Maribel [1]; Cervantes Valenzuela, Mariela [1];
Félix Soto, Lucero [1]; Del Arenal Sánchez, Silvia [1];
Flores Andrade, Xavier [1]; Ávila Ríos, Santiago [1].

[1]CIENI Centre for Research in Infectious Diseases, Instituto Nacional de Enfermedades Respiratorias, Tlalpan, Mexico City, Mexico.

E-mail: humbertovzla@gmail.com

HLA loci are considered major drivers of HIV evolution. Some HLAs select mutations within the viral population that favor HIV and allow it to evade cytotoxic T lymphocyte responses. These escape mutations follow predictable molecular patterns, imposing an HLA subtype-specific "footprint" (i.e., HLA-associated polymorphisms, HAPs) on HIV. Earlier, we identified in Mexican HIV+ cohorts, Amerindian B*35 subtypes (i.e., B*35:12/14/17, common in Mexican mestizos), which impose a population-level footprint on Gag/Pol. We aimed to test B*35 HIV epitopes using IFNg ELISpot assays with Gag/Pol peptides as stimuli encompassing described B*35-related HAPs. Overlapping 9mer peptides (OLPs) were synthesized for each Gag/Pol region containing B*35 HAPs (i.e., 63 OLPs for 7 HAPs). OLPs were tested individually using IFNg ELISpot using PBMCs from recently HIV-diagnosed B*35+ donors (PLWH). Positive spot-forming units (SFU) were identified using the non-parametric DFR method. We included 26 B*35+ PLWH. Seven of twelve B*35:12+ PLWH had positive ELISpot responses, encompassing adjacent LEEMNLPGR and EEMNLPGRW OLPs (HAP-Pol-37, Kruskal-Wallis $p=0.022$, median SFU=12 and 16, respectively). KALTEVVPL and ALTEVVPLT (HAP-Pol-393, $p<0.001$, SFU=18.43 and 16.03, respectively), and TEEAELELA and EEAELELAE (HAP-Pol-403AE, $p<0.001$, SFU=13.82 and 16.03, respectively). For B*35:14+ assays, 1/4 of PLWH had positive responses; SPAIFQSSM had a significantly higher SFU ($p=0.01$, SFU=273); also restricted by common B*35:01. For B*35:17+, 3 OLPs had positive responses (FLGKIWPSH, GKIWPSHKG, HKGRPGNFL), however, median SFU values did not reach statistical significance. Our ongoing analysis across B*35 subtypes continues to reveal consistent and reproducible recognition of OLPs encompassing HAPs, supporting the utility of our epitope mapping strategy. Continued ELISpot assays will further validate these HIV-specific epitopes restricted by B*35 alleles. This approach not only proves methodologically sound but also holds significant promise for informing future immunological strategies, particularly in the rational design of geographically adapted HIV vaccines targeting underrepresented Amerindian populations.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Phenotype and immunogenicity of plasma extracellular vesicles in latent and active tuberculosis

Sta.Maria-Peregrino, Abraham Eliud [1]; Castañeda-Casimiro, Jessica [1 2]; Vallejo-Castillo, Luis [3 4]; Gutiérrez-Domínguez, Eduardo [1]; Vallejo-Castillo, Luis [3 4]; Soria-Fragoso, Mishelle [1]; Chacón-Salinas, Rommel [1]; Serafín-López, Jeanet [1]; Hernández-Solis, Alejandro [5 6]; Wong-Baeza, Isabel [1].

[1]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Laboratorio de Inmunología Molecular II, Departamento de Inmunología, Ciudad de México, México. [2]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Departamento de Microbiología, Ciudad de México, México. [3]Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, Unidad de Desarrollo e Investigación en Bioprocesos (UDIBI), Ciudad de México, México. [4]Laboratorio Nacional para Servicios Especializados de Investigación, Desarrollo e Innovación (I+D+i) para Farmoquímicos y Biotecnológicos, LANSEIDI-FarBiotec-CONAHCyT, Ciudad de México, México. [5]Universidad Nacional Autónoma de México, Facultad de Medicina, Ciudad de México, México. [6]Hospital General de México "Dr. Eduardo Liceaga", Servicio de Neumología, Secretaría de Salud, Ciudad de México, México.

E-mail: eliudsp.eb@gmail.com

Tuberculosis (TB) presents two major clinical phases—latent (LTB) and active (ATB)—which are shaped by the host's metabolic and immunologic status. These differences define a molecular landscape that can be explored to better understand TB pathophysiology. In this context, extracellular vesicles (EVs) carry molecular fingerprints that may serve as potential biomarkers or mediate disease progression. With the objective of describing EV dynamics during LTB and ATB and explore their ability to carry mycobacterial molecules recognized by TLRs, we isolated plasma-derived EVs from LTB and recently diagnosed ATB individuals. Characterization included protein quantification, electron microscopy, nanoparticle tracking analysis, and nanoscale flow cytometry to differentiate exosomes (CD81+/CD63+) and microvesicles (annexin V+/CD35+). With the aim of describing the potential participation of plasma EVs in immune cell activation, we stimulated TLR2/6-expressing HEK cells with purified EVs and measured IL-8 production.

Our findings show that EVs from both clinical TB phases have an increased protein content. While total EV concentrations did not differ significantly between LTB and ATB donors against healthy controls; LTB individuals exhibited reduced exosome percentages, suggesting selective modulation of this EV subpopulation. Moreover, EVs from both LTB and ATB phases failed to induce IL-8 production in HEK cells, indicating that early TB-associated EVs do not carry mycobacterial molecules recognized by TLR2/6.These results support the notion that EVs are involved in TB development. Notably, LTB and ATB can alter exosome production, suggesting that full EV profiling may reveal the contribution of distinct host cell types. The presence of mycobacterial molecules in EVs should be further investigated in advanced ATB stages.

Funding. SIP-IPN and CONAHCYT/SECIHTI (CF-2019/217572). Scholarship received. BEIFI 202110601, CONACYT/SECIHTI.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

Helios favors CD8 T cell effector differentiation by modulating IL-2 signaling

Suárez-Rojas, Gerardo [1 2]; Rubio-Robles, Rosa M. [1];
Albarran-Godínez, Adrian [1 2]; Aguilar-Fuentes, Rosa E. [1 3];
Magallón, Eduardo [1]; Madera-Salcedo, Iris K. [1];
Rosetti, Florencia [1]; Crispín, José C. [1 3].

[1]Departamento de Inmunología y Reumatología, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Ciudad de México, México. [2]Doctorado en Ciencias Biomédicas, Universidad Nacional Autónoma de México, Ciudad de México, México. [3]Escuela de Medicina y Ciencias de la Salud, Tecnológico de Monterrey, Ciudad de México, México.

E-mail: gerardo.suarez.699@gmail.com

Helios (Ikzf2) belongs to a family of transcription factors that regulate lymphoid differentiation and function. In thymic-derived FoxP3 regulatory T cells, Helios promotes suppressive function. Previously, we observed that Helios is induced in CD8 T cells under tolerance-induced contexts. The aim of this work was to analyze the signals that drive Helios expression and function in CD8 T cells. We established an in vitro assay to identify factors that modulate Helios expression. Productive CD8 activation, achieved by high affinity ligands, inhibited Helios expression. In contrast, activation with low-affinity ligands led to robust Helios induction. Pharmacological dissection of signaling pathways indicated that activation of the IL-2-STAT5 pathway inhibited Helios induction. We analyzed Helios expression kinetics in CD8 T cells during an acute infection, where clonal expansion relies on IL-2 abundance. We observed a transitory Helios expression that peaked at 72 hours post infection. Ikzf2-deficient CD8 T cells exhibited an impaired proliferation, expansion and effector responses when exposed to their cognate antigen in the context of infection (i.e. Listeria and LCMV-Arm). scRNA sequencing of CD8 T cells revealed an expansion of cells with memory/progenitor features in Helios-deficient samples when compared to wild-type cells, suggesting that Ikzf2-deficient CD8 T cells are less differentiated during an acute infection. Furthermore, transcriptional analysis showed that phosphatases implicated in the IL-2 signaling were enriched in Helios deficient cells. As IL-2 signaling is a key pathway in T cell acquisition of effector functions, Helios could act as a repressor of CD8 T cell effector functions through the modulation of IL-2 signaling. In conclusion, Helios might represent a key regulator of CD8 T cell effector differentiation. This regulatory mechanism could be an interesting pathway to target, in order to improve or restrain CD8-madiated immune responses in the context of autoimmunity or cancer.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Comparative Analysis of Exhaustion Markers in Blood and Tumor in Renal Cell Carcinoma

Suárez -Vázquez, Tomás [1]; González-Garza, Raquel [1];
Gutiérrez-González, Adrián [2]; Garza-Guajardo, Raquel [3];
Salinas-Carmona, Mario César [1]; Mejía-Torres, Manuel [1].

[1]Hospital Universitario “José Eleuterio González”, Universidad Autónoma de Nuevo León, Departamento de Inmunología, Monterrey, Nuevo León, México. [2]Hospital Universitario “José Eleuterio González”, Universidad Autónoma de Nuevo León,Servicio de Urología, Monterrey, Nuevo León, México. [3]Hospital Universitario “José Eleuterio González”, Universidad Autónoma de Nuevo León, Servicio de Patología, Monterrey, Nuevo León, México.

E-mail: suarez.ta@hotmail.com

Renal cell carcinoma (RCC) is a neoplastic disease associated with poor prognosis. Treatments for advanced disease often require the use of immunotherapy, but currently, no method is available for predicting treatment success or failure. The gold standard in assessing the expression of tissue antigens is immunohistochemistry but flow cytometry offers complementary advantages for evaluating the “exhaustion markers” as a guide for the use of immune checkpoint inhibitors. In this study, we describe with a quantitative approach the expression of five biomarkers associated with immune dysfunction in a group of 20 patients diagnosed with localized clear cell RCC. We confirm that ccRCC tissue is highly positive to CD3, with low expression of PD-L1 and CTLA-4. Relative to healthy controls, the RCC patients showed abnormal proportions of both blood and infiltrating leukocytes. In addition, patients had increased expression of PD-1 and TIM-3 in CD3, but decreased PD-L1 expression in CD14 monocytes, with levels decreasing further within the tumor infiltrating monocytes. We found that decreased PD-1 and increased infiltration with CD3 identified the patients with poorest prognosis according to nuclear grade. Collectively, our results suggest the valuable and complementarity use of conventional flow cytometry and immunohistochemistry for risk assessment and possibly for predicting therapeutic response to immunotherapy in patients with localized ccRCC.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Immune Exhaustion Marker Dynamics Following Nephrectomy in Patients with Renal Cell Carcinoma

Suárez-Vázquez, Tomás [1]; González-Garza, Raquel [1];
Gutiérrez -González, Adrián [2]; Garza-Guajardo, Raquel [3];
Salinas-Carmona, Mario César [1]; Mejía-Torres, Manuel [1].

[1]Hospital Universitario “José Eleuterio González”, Universidad Autónoma de Nuevo León, Departamento de Inmunología, Monterrey, Nuevo León, México. [2]Hospital Universitario “José Eleuterio González”, Universidad Autónoma de Nuevo León, Servicio de Urología, Monterrey, Nuevo León, México. [3]Hospital Universitario “José Eleuterio González”, Universidad Autónoma de Nuevo León, Servicio de Anatomía Patológica, Monterrey, Nuevo León, México.

E-mail: suarez.ta@hotmail.com

Clear cell renal cell carcinoma (ccRCC) is a neoplastic disease often associated with poor prognosis. While localized tumors are typically managed with nephrectomy, advanced disease frequently requires immunotherapy, for which predictive biomarkers remain limited. This study aimed to assess immune exhaustion markers in peripheral blood from patients with localized ccRCC before and after radical nephrectomy. We evaluated the expression of PD-1 and CTLA-4, TIM-3 and LAG-3, using flow cytometry in 20 patients. Postoperative analysis revealed an increase in granulocyte proportions, whereas monocytes and total lymphocytes decreased. Among lymphocyte subsets, only NKT cells showed a significant reduction post-nephrectomy. Notably, at the 12-month follow-up, we observed a marked and sustained decrease in the expression of PD-1 on $CD8^+$, $CD4^+$, and NKT cells, as well as a significant reduction of CTLA-4 on $CD4^+$ T cells. Additionally, we observed a reduction in TIM-3 cells among CD8+ and NKT lymphocytes, as well as a significant decline in LAG-3 cells on CD4+ lymphocytes. These findings suggest a partial restoration of peripheral immune competence following tumor removal, although persistent exhaustion features may reflect ongoing immune dysregulation. Our results highlight the potential of longitudinal immune monitoring to understand immune recovery and potential responsiveness to immunotherapy in ccRCC patients.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Mast cell deficiency affects the development of actinomycetoma caused by Nocardia brasiliensis

Suárez -Vázquez, Tomás Alejandro [1]; Salinas -Carmona, Mario Cesar [1]; López-López, Nallely [1]; Campillo-Hernández, Jesús Armando [1].

[1]Department of Immunology, School of Medicine and Dr. Jose Eleuterio Gonzalez University Hospital, Universidad Autónoma de Nuevo León, Monterrey, Mexico.

E-mail: suarez.ta@hotmail.com

Chronic infection's establishment depends on failures in their containment during early stages, primarily due to deficiencies in the innate immune response or immune evasion mechanisms. Nocardia brasiliensis, a facultative intracellular pathogen and the main cause of actinomycetoma in Mexico, evades immunity by targeting critical immune cells like mast cells. These cells, localized at environmental interfaces like skin and mucosal barriers, regulate tissue homeostasis and initiate host defense. Activated mast cells, release mediators (e.g., histamine, tryptase, TNF-α) that help contain pathogens, recruit immune cells, and enhance antimicrobial responses. This study aims to investigate the role of mast cells in the progression of actinomycetoma using a W-sh mouse model, which is deficient in these cells due to a mutation in the c-kit gene. We demonstrate that in the absence of mast cells, the bacteria load was significantly higher, with an impaired bacterial clearance. This finding appears to contrast with the granular morphology of Nocardia microcolonies in H&E-stained tissue sections, which showed fewer granules and smaller area in the W-sh, suggesting a failed antibacterial mechanism. Regarding innate immune cell populations, granulocytes accounted for 80–90% of cells in both W-sh and WT models during the chronic stage, with no apparent effect in mast cell deficient mice. However, in the adaptive immune response, the W-sh model exhibited a mixed CD3+ T-cell profile (30% CD4+, 70% CD8+), contrasting sharply with the WT model, which displayed a purely helper T-cell response (100% CD4+). No significant difference in IgG levels was observed between WT and W-sh. Our findings demonstrate that mast cells play a key role restricting bacterial replication and modulating granule formation through coordinated regulation of inflammatory cell recruitment and promotion of actinomycetoma development. This multifaceted mechanism enhances N. brasiliensis containment and clearance, establishing mast cells as promising therapeutic targets for chronic infection management.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Mast Cell Activation by Nocardia brasiliensis Drives Immunopathology in Actinomycetoma

Suárez -Vázquez, Tomás Alejandro [1]; Salinas -Carmona, Mario César [1]; López-López, Nallely [1]; Mejía -Torres, Manuel Guadalupe [1]; Garza-Guajardo, Raquel [2]; Chacón -Salinas, Rommel [3]; Meneses -Preza, Yatsiri Guadalupe [3]; Ruíz -Sánchez, Bibiana Patricia [3].

[1]Department of Immunology, School of Medicine and Dr. Jose Eleuterio Gonzalez University Hospital, Universidad Autónoma de Nuevo León, Monterrey, Mexico . [2]Anatomic Pathology and Cytopathology Service, Dr. Jose Eleuterio Gonzalez University Hospital, Universidad Autónoma de Nuevo León, Monterrey, Mexico. [3]Department of Immunology, National School of Biological Sciences, Instituto Politécnico Nacional (ENCB-IPN), Ciudad de México, Mexico.

E-mail: suarez.ta@hotmail.com

Mast cells act as immune sentinels and regulate inflammation in peripheral tissues. Chronic infections disrupt homeostasis through persistent mediator release, creating a pathological cycle of inflammation, tissue damage, and immunosuppression that hinders pathogen clearance. This study investigated mast cell-mediated immune responses to Nocardia brasiliensis infection using complementary in vitro and in vivo approaches, including mast cell deficient W-sh (c-kit mutant) mice. To assess Nocardia effect on bone marrow-derived mast cells (BMMCs), we determined a MOI that preserved cell viability (MOI 1). Subsequent β-hexosaminidase assays in the cocultures revealed peak degranulation at MOI of 3. We then assessed the inflammatory response measuring IL-6, TNF-α, and TGF-β cytokine levels using ELISA after Nocardia stimulation. We found significant increases in IL-6-TNF-α across MOIs of 0.5–3, whereas TGF-β showed a notable increase only at MOI of 3 compared to unstimulated controls. We confirmed that Nocardia activated BMMCs in vitro, inducing degranulation and cytokine release without compromising viability. To further investigate the in vivo relevance, we established an actinomycetoma model using Nocardia brasiliensis in W-sh mice to evaluate the role of mast cell deficiency in disease progression. Mast cell deficiency increased bacterial load and impaired clearance, despite smaller, less granular Nocardia microcolonies in W-sh mice (H&E staining), suggesting defective antibacterial mechanisms. Another key finding was that lesion volumes were larger in W-sh mice than in WT controls, prompting further histopathological analysis of chronic phase tissue sections. W-sh mice exhibited heightened plasma cell infiltration, fibroblast proliferation, perineural inflammation, neuritis, osteomyelitis, and tissue atrophy in infected areas, along with reduced lymphoid hyperplasia by day 72 post-infection. Our findings established that N. brasiliensis activates mast cells in vivo and in vitro, triggering proinflammatory cytokine release that affects leukocyte recruitment and drives the histopathology underlying actinomycetoma development. Mast cell deficiency disrupts antibacterial mechanisms and exacerbates chronic immunopathology.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

The store-operated calcium entry inhibitor 2-aminoethyl diphenylborinate suppresses NOX-dependent netosis

Sumoza-Toledo, Adriana [1 2]; Herrera-Hernández, Edgar Javier [2].

[1]Universidad Veracruzana. Instituto de investigaciones Médico-Biológicas. Veracruz, Ver. México. [2]Universidad Veracruzana. Facultad de Bioanálisis. Veracruz, Ver. México.

E-mail: asumoza@uv.mx

Neutrophil extracellular traps (NETs) play a significant role in the pathogenesis of various human diseases, including chronic inflammation, autoimmunity, severe viral infections, cardiovascular disease, thrombosis, and cancer metastasis. Consequently, pharmacological strategies aimed at modulating NETosis are currently under active investigation. An increase in intracellular calcium levels is critical for NET formation, as it contributes to the production of reactive oxygen species (ROS) and the citrullination of histone H3, two key events in NETosis. This study investigate the effect of 2-aminoethoxydiphenyl borate (2-APB), a putative inhibitor of store-operated calcium (Ca2+) entry (SOCE), on the formation of NETs induced by phorbol 12-myristate 13-acetate (PMA). Our findings indicate that NET formation, along with the colocalization of myeloperoxidase (MPO) and citrullinated-H3, is dependent on extracellular calcium influx. Moreover, NETosis was significantly inhibited by 2-APB, raising the possibility that it may be modulated through the inhibition of calcium channels.

Funding for carrying out this research was provided by Instituto de Investigaciones Médico Biológicas, Universidad Veracruzana.

TRABAJO NO PRESENTADO

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Analysis of DNMT2 function during dengue virus infection in *Aedes aegypti* mosquitoes

Tapia-Rodríguez, Ana [1]; Claudio-Piedras, Fabiola [1];
Recio-Tótoro, Benito [1 2]; Pacheco-Guillén, Sabino [2];
Lanz-Mendoza, Humberto [1].

[1]Centro de Investigación Sobre Enfermedades Infecciosas, Instituto Nacional de Salud Pública, Cuernavaca, Morelos, México. [2]Instituto de Biotecnología, Universidad Nacional Autónoma de México, Cuernavaca, Morelos, México.

E-mail: ana.tapia.rod9@gmail.com

Dengue virus (DENV) is an arbovirus transmitted by infected mosquitoes, mainly Aedes aegypti. It is a public health problem in tropical regions of developing countries. There is no specific treatment against this infection, so the development of transmission-blocking strategies based on the understanding of virus-vector molecular interactions is crucial. The small interfering RNA pathway is the principal antiviral defense system in A. aegypti. This pathway includes dicer 2 (dcr2), a dsRNA-binding protein called r2d2, and argonaute 2 (ago2). Hsp70, a heat shock protein, is linked with the host RNA silencing machinery by coordinating with the argonaute proteins. Recent work from our research group has shown that dengue virus-infected mosquitoes overexpress dnmt2, a cytosine-C5-methyltransferase that is present as a single copy in A. aegypti. Coexpression analysis shows that dnmt2 and r2d2 transcripts are the most highly correlated in infected mosquitoes. Furthermore, the overexpression of dnmt2, dcr2, r2d2, ago2 and hsp70, coupled with a reduction in viral infection prevalence from 83% to 30% after a 5mC-inhibition treatment, suggests that dnmt2 plays a pivotal role during infection. Although the use of methylation inhibitors such as azacytidine is a well-established approach for analyzing methylation function in nucleic acids, a more specific approach that limits the indirect effects is required. This research project aims to analyze the function of DNMT2 in CRISPR-Cas9 genetically edited mosquitoes infected with DENV. The establishment of a Δdnmt2-53 A. aegypti line was successful, and it's currently being screened and selected for homozygosity. Up to now, four generations with no signs of deleterious effects on development have been produced. Once homozygosity is reached; infected mosquitoes will be analyzed for: (1) transcriptional response of dnmt2, dcr2, r2d2, ago2, hsp70; and (2) DENV prevalence and intensity of infecction.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Vacunas**

Hybrid Immunity Enhances Effector Function and Stem-like Memory T Cells Against SARS-CoV-2

Torres-Flores, Alejandro [1 2]; Gajón-Martínez, Julián [1];
Rivera-Hernández, Tania [1 3]; Lozano-Cisneros, Diego [1 4];
Berlanga-Taylor, Antonio [5]; Cérbulo-Vázquez, Arturo [6]; Ferat-Osorio, Eduardo [7]; Arriaga-Pizano, Lourdes [1]; Bonifaz-, Laura [1 7];
Wong-Baeza, Isabel [8]; López-Macías, Constantino [1].

[1]Unidad de Investigación Médica en Inmunoquímica, UMAE Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social (IMSS), Ciudad de México (CDMX), México. [2]Posgrado en Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, CDMX, México. [3]Investigadores por México, Consejo Nacional de Humanidades, Ciencias y Tecnologías (CONAHCYT), CDMX, México. [4]Posgrado en Ciencias Biológicas, Facultad de Ciencias, Universidad Nacional Autónoma de México, CDMX, México. [5]Unidad de Medicina de Datos, Centro Médico Nacional Siglo XXI, IMSS, CDMX, México. [6]Hospital General de México "Dr. Eduardo Liceaga", Servicio de Medicina Genómica, CDMX, México. [7]Coordinación de Investigación en Salud, IMSS, División de Investigación Clínica, CDMX, México. [8]Laboratorio de Inmunología Molecular, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, CDMX, México.

E-mail: t.alejandro78@yahoo.com.mx

Hybrid immunity, resulting from natural SARS-CoV-2 infection combined with vaccination, has been associated with more robust and persistent immune responses. T-cell responses have emerged as key correlates of protection, especially when antibody titers wane or viral variants escape neutralization. In this context, we aimed to characterize the dynamics of SARS-CoV-2–specific T-cell responses in hybrid immunity and evaluate their role in shaping durable cellular memory. We conducted a longitudinal study in 36 adult volunteers categorized into three cohorts: vaccinated-only (Vax), vaccinated after infection (Vax-Inf), and infected prior to vaccination (Inf-Vax). Peripheral blood samples were collected at baseline and at 3, 6, and 12 months following heterologous booster immunization. Antigen-specific activation of $CD4^+$ ($CD69^+CD134^+$) and $CD8^+$ ($CD69^+CD137^+$) T cells was assessed by multiparametric flow cytometry. Hybrid immunity markedly enhanced both the magnitude and persistence of T-cell responses. Effector memory subsets displayed distinct enrichment profiles, with hybrid groups showing improved functional signatures and cytokine production. Notably, booster vaccination in the setting of hybrid immunity promoted the induction of stem cell-like memory T cells (TSCM; $CD45RA^+CCR7^+CD95^+CD27^+$) in both $CD4^+$ and $CD8^+$ populations. These TSCM cells, known for their self-renewal and multipotency, are central to the maintenance of long-lived immune memory. While individual frequencies varied, TSCM expansion was consistently observed in hybrid cohorts. These findings indicate that hybrid immunity and booster vaccination synergize to diversify memory T-cell landscapes and promote durable cellular immunity. The induction of TSCM may represent a promising biomarker of long-lasting protection, relevant to SARS-CoV-2 booster design and potentially to future vaccine platforms against emerging infectious diseases.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Peripheral Immune Biomarkers for Alzheimer’s Diagnosis: Transcriptomic and Digital Cytometry Analysis

Torres- García, Aarón [1]; Rojas-Gutiérrez, Santiago Emilio [1]; Martínez-Canales, Rodolfo [1]; Montoya-Rosales, Alejandra del rocío [1]; Salinas-Carmona, Mario César [1]; Macías-Segura, Noé [1].

[1]Facultad de Medicina “Hospital Universitario Dr. José Eleuterio González” UANL, Servicio y Departamento de Inmunología, Monterrey, Nuevo León, México.

E-mail: aaron.torresg@uanl.edu.mx

Alzheimer’s disease (AD) is a neurodegenerative disorder characterized by β-amyloid plaques, neuroinflammation and immune dysregulation. Blood-based immune biomarkers offer a promising non-invasive alternative to current diagnostic tools, which are often costly or inaccessible.

To identify immune-related blood biomarkers to support the development of accessible and non-invasive diagnostic tools for AD.

We extracted transcriptomic datasets from 2013 to 2023 from NCBI GEO, including 238 brain and 192 peripheral blood samples from AD patients and healthy controls (HC). Brain datasets included GSE48350, GSE122063, GSE138261, and GSE193391, while blood data were GSE97760 and GSE140829. Differentially expressed genes (DEGs) were identified using GEO2R, studied via STRING. Immune cell composition was calculated from samples using CIBERSORTx. ROC curve analyses were conducted to evaluate the diagnostic performance of peripheral DEGs. Statistical analyses included t-tests and Pearson correlation, considering statistical significance as $p < 0.05$.

Peripheral blood from AD patients showed systemic immune activation. HSPA1A ($p = 0.0338$) and HSPB6 ($p = 0.0011$), linked to stress response, and UBC ($p = 0.0115$), related to proteostasis, were upregulated. Downregulated genes included HNRNPA2B1 ($p = 0.0008$), HNRNPA0 ($p = 0.0493$), and RBM3 ($p = 0.0526$), implicating RNA processing and reduced neuroprotection. Immune deconvolution revealed increased proportions of activated CD4+ memory T cells and monocytes in AD blood compared to controls. In brain tissue, regulatory T cells and mast cells showed significant variation.

ROC analysis identified HNRNPA2B1 (AUC = 0.5919), HSPB6 (AUC = 0.5887), and UBC (AUC = 0.5689) as top-performing biomarkers. Network analysis revealed enrichment in immune and stress response pathways.

Using a bioinformatic approach we found a potential peripheral transcriptomic signature with immune-related gene expression changes with potential biomarkers. Findings support further development of blood-based biomarkers as non-invasive tools for early detection and monitoring of AD.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Area del artículo: **Inmunometabolismo**

Dietary Fiber: Impact on Immunity Mediated by the Gut Microbiome

Torres -Salgado, Madeleine [1].

[1]Unipuebla, Puebla, México .

E-mail: maddiets75@gmail.com

Dietary fiber is a crucial component that significantly influences the modulation of the immune system through its interaction with the gut microbiome, a key factor in maintaining overall health. The primary objective of this study was to examine the effect of dietary fiber on immunity mediated by the microbiome by conducting a comprehensive review of current and relevant scientific literature. To achieve this, a careful selection of articles and reviews addressing the relationship between fiber types, microbial composition, and immune response was undertaken, applying stringent criteria for quality and relevance. The findings demonstrate that adequate fiber intake promotes the proliferation of beneficial bacteria such as bifidobacteria and lactobacilli, which ferment fiber to produce short-chain fatty acids (SCFAs). These metabolites play an essential role in regulating inflammation, strengthening the intestinal barrier, and modulating systemic immune responses. Moreover, diets rich in fiber are associated with reductions in inflammatory markers and improvements in immune function across various clinical contexts. The discussion underscores the importance of dietary fiber as a modulator of both the microbiome and immunity, highlighting individual variability in response and the consequent need for personalized nutritional strategies. In conclusion, encouraging sufficient fiber consumption represents an accessible and effective approach to enhancing immunity and preventing disease, thereby providing a robust foundation for future research and clinical applications in nutrition and public health.

Área del artículo: **Inmunidad tumoral**

HPV E6/E7 regulate Grainyhead-like 2 and 3 expression in a CaCU model

Torres-Reyes, Luis Alberto [1]; González-Aldaco, Karina [1];
Jave-Suárez, Luis Felipe [2]; Aguilar-Lemarroy, Adriana [2].

[1]Centro Universitario de los Valles, Universidad de Guadalajara, Carretera Guadalajara-Ameca Km. 45.5, Ameca 46600, Jalisco, México. [2]Centro de investigación Biomédica de Occidente, División de Inmunología, Instituto Mexicano del Seguro Social, Guadalajara 44340, Jalisco, México.

E-mail: luis.torres9386@academicos.udg.mx

Transcription factors GRHL2 and GRHL3 were characterized in humans during the past decade. Both proteins regulate the expression of key genes involved in various cellular processes, ranging from embryogenesis to the establishment and maintenance of epithelial homeostasis in adult organisms. However, their overexpression or inhibition in different cellular models has been associated with carcinogenic events. In this context, our research group previously reported a markedly reduced expression of both transcription factors in a cervical cancer (CaCU) cell line model, suggesting a possible role as tumor suppressor genes disrupted in this type of cancer. Therefore, we aimed to inhibit or overexpress the HPV oncogenes E6 and E7 to determine their potential regulatory effect on GRHL2 and GRHL3 in our CaCU model. To achieve this, specific shRNAs or lentiviral particles using the Tight-puro system were used to downregulate or induce E6 and E7 expression in HeLa (HPV18□) and HaCaT (non-tumorigenic keratinocytes) cell lines, respectively. As a result, downregulation of E6/E7 in HeLa cells led to increased expression of GRHL2 and GRHL3. Conversely, exogenous expression of E6/E7 in HaCaT cells resulted in a slight decrease in GRHL2 expression and a marked increase in GRHL3. These findings suggest a possible direct or indirect role of viral oncogenes E6 and E7 in modulating GRHL2 and GRHL3 on cervical carcinogenesis. This research was funded by Fomento a la Investigación 2024, UDG-CUValles to Torres-Reyes LA.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Combination therapy with Doxorubicin and Cry1Ac in a murine model of TNBC

Trejo-De la Mora, Sandra Sulibeth [1]; Flores-Correa, Edgar Emiliano [1]; Ilhuicatzi-Alvarado, Damaris [1]; Moreno-Fierros, Leticia [1].

[1]Unidad de Biomedicina (UBIMED) Laboratorio de Inmunidad en Mucosas Facultad de Estudios Superiores Iztacala Avenida de los barrios Número 1, Los Reyes Iztacala Tlalnepantla, Estado de México, C.P. 54090 . E-mail: suli256trejo@gmail.com

Breast cancer (BC) is the most common and deadliest cancer among women, with 670,000 deaths reported in 2022. In Mexico, its incidence is 27.64 cases per 100,000 inhabitants. BC is classified into four molecular subtypes: luminal A, luminal B, HER2+, and triple-negative breast cancer (TNBC), the latter lacking ER, PR, and HER2 expression. TNBC accounts for ~10% of cases and is the most aggressive subtype, with high metastatic capacity, poor prognosis, early recurrence, limited therapeutic options, and higher mortality.

Active immunotherapy is a promising strategy for TNBC, yet its efficacy is limited by low tumor immunogenicity and an immunosuppressive microenvironment. Although TLR agonists have been tested as adjuvants, their use is restricted because many tumors express TLRs, and their activation can promote tumor growth. Safer, more effective adjuvants are needed. Cry1Ac protoxins from Bacillus thuringiensis have shown adjuvant potential through systemic and mucosal routes. In prophylactic models using tumor cell lysates, Cry1Ac induced antitumor immunity, reduced metastases, and decreased MDSC levels in murine TNBC. However, its therapeutic potential as an adjuvant remains to be explored.

Inducing immunogenic cell death, such as that caused by Doxorubicin, can enhance antigen release. In this study, we evaluated the antitumor effect of combining Cry1Ac protoxin with Doxorubicin in a 4T1 murine TNBC model. Tumor, spleen, and inguinal lymph nodes were analyzed by flow cytometry to assess MDSCs and proinflammatory cytokines.

Combined therapy reduced tumor size, pulmonary metastases, and MDSC populations. Additionally, intratumoral proinflammatory cytokine levels increased, supporting the immunostimulatory and antitumor potential of this approach.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Effect of microRNA overexpression on macrophage polarization markers

Turiján-Espinoza, Eneida [1,2]; Martínez-Leija, Miguel Ernesto [2,3]; Martínez-Tejada, Lorena [3]; Castillo-Esquivel, Arian [3]; Sánchez-Lucio, Lizeth Sinai; Jiménez-Maldonado, Brenda Vanessa [3]; González-Hernández, Osiel [3]; Zavala-Reyes, Daniel [3]; Portales-Pérez, Diana Patricia [2,3].

[1]Universidad de Guadalajara, Centro Universitario de Ciencias de la Salud (CUCS), Guadalajara, Jalisco. México. [2]Universidad Autónoma de San Luis Potosí, Laboratorio de Inmunología y Biología Celular y Molecular, Facultad de Ciencias Químicas, San Luis Potosí, S.L.P. México. [3]Universidad Autónoma de San Luis Potosí, Centro de Investigación en Ciencias de la Salud y Biomedicina, San Luis Potosí, S.L.P. México.

E-mail: eneida.turijan.espinoza@gmail.com

Effect of microRNA overexpression on macrophage polarization markers. Macrophages with an M1 phenotype are characterized by promoting antitumor immune responses, whereas M2 macrophages contribute to cancer progression by facilitating angiogenesis, invasion, and immune evasion. MicroRNAs (miRNAs) are known to regulate the differentiation and function of monocytes and macrophages; specifically, miR-125a is associated with the induction of the M1 phenotype, while miR-146b is linked to M2 polarization. Therefore, elucidating the role of miRNAs offers a promising avenue for developing novel therapeutic strategies in contexts such as breast cancer. This study aimed to evaluate the effects of miR-125b and miR-146a overexpression on M1 and M2 macrophage polarization using $CD14^+$ human peripheral blood monocytes and the THP-1 monocytic cell line. Differentiation protocols for M1 and M2 macrophages were standardized using specific polarization stimuli. Transfection conditions were optimized using miRNA mimics for miR-125b and miR-146a under each polarization condition. Phenotypic characterization was performed through the analysis of molecular markers associated with M1 (CD86, TNF-α, STAT1, and NOS2) and M2 (CD206, STAT6, CCL22, and ARG1) phenotypes by flow cytometry and real-time PCR. In vitro assays showed that both primary monocytes and THP-1 cells developed characteristic amoeboid (M1) and fibroblast-like (M2) morphologies upon polarization. Gene expression analysis in THP-1 cells revealed upregulation of STAT1 and TNF-α in M1 conditions, and increased STAT6 and CCL2 expression in M2 conditions. Flow cytometry confirmed an increase in $CD86^+$ (M1) and $CD206^+$ (M2) cells relative to non-polarized controls. Notably, overexpression of miR-146b led to a reduction in CD206 protein levels, suggesting a potential inhibitory effect on M2 macrophage development.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Diazinon-induced neuroimmunotoxicity mediated through mAChR in Oreochromis niloticus challenged with Aeromonas hydrophila

Ulloa-Ramírez, Brandon Bladimir [1]; Toledo-Ibarra, Gladys Alejandra [2]; Covantes-Rosales, Carlos Eduardo [2]; Girón-Pérez, Manuel Ivan [2]; Barcelos-García, Rocío Guadalupe [2]; Martínez-Pérez, Vasti Elena [1]; Mercado-Salgado, Ulises [2]; Girón-Pérez, Daniel Alberto [2].

[1]Unidad Académica Ciencias Químico Biológicas y Farmacéuticas. Universidad Autónoma de Nayarit. Tepic, Nayarit, México. [2] Laboratorio Nacional para la Investigación en Inocuidad Alimentaria (LANIIA) Unidad Nayarit. Universidad Autónoma de Nayarit. Tepic, Nayarit, México. * E-mail: gladys.toledo@uan.edu.mx,

E-mail: 16035371l@uan.edu.mx

The nervous and immune systems are closely interconnected, playing a critical role in regulating organismal homeostasis and facilitating effective responses to pathogens. However, environmental pollutants can disrupt neuro-immune communication. Diazinon, an organophosphate pesticide, is a neuroimmunotoxic compound as it inhibits the acetylcholinesterase enzyme, leading to increased acetylcholine concentration. This overstimulation of muscarinic (mAChR) and nicotinic (nAChR) cholinergic receptors disrupts both the neuronal and leukocytic cholinergic systems. Additionally, this pesticide has immunotoxic effects, altering both humoral and cellular immunity parameters. However, the mechanism of toxicity remains unclear. Consequently, this work aims to evaluate the expression of muscarinic receptors (mAChR M2–M5) in spleen leukocytes and the brain of Nile tilapia fish (Oreochromis niloticus) exposed *in vivo* to diazinon (0.97 mg/L, 24 hours) and subsequently challenged with Aeromonas hydrophila (1x109 CFUs). To achieve this, adult male O. niloticus will be exposed in vivo to 0.97 mg/L (1/8 LC50) of diazinon for 24 hours and then challenged with A. hydrophila for 72 hours. Following the treatment period, the brain and spleen will be collected, RNA will be isolated using the TRIzol method, cDNA synthesis will be conducted, and gene expression of mAChRs (M2–M5) will be assessed using real-time PCR. The data obtained will help elucidate the role of mAChRs in the neuroimmunotoxicity mechanism of organophosphate pesticides, particularly regarding the induction of immunosuppression and increased susceptibility to infections in exposed organisms. This information will contribute to mitigating the negative impact of organophosphate pesticides on environmental health.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Cytokine Signatures in Immune Thrombocytopenia Across Clinical Stages

Unzueta-Villalobos, Katya Unzueta [1 3]; Alvarado-González,
Carmen Carolina [2 3]; Davizon-Castillo, Pavel [4]; Chávez-Trillo,
Carlos [5]; Espino-Solís, Gerardo Pavel [3].

[1]Instituto Tecnológico y de Estudios Superiores de Monterrey Campus Guadalajara, Facultad de Medicina y Ciencias de la Salud, Zapopan, Jalisco, México. [2]Universidad Autónoma de Chihuahua, Facultad de Ciencias Químicas, Chihuahua, Chihuahua, México. [3]Universidad Autónoma de Chihuahua, Laboratorio de Investigación Traslacional, Facultad de Medicina y Ciencias Biomédicas, Chihuahua, Chihuahua, México. [4]Bloodworks Northwest Research Institute, Seattle, Washington, EUA. [5]Hospital de Pensiones Civiles del Estado de Chihuahua, Hematología, Chihuahua, Chihuahua, México.

E-mail: katya.unzueta@gmail.com

Immune Thrombocytopenia (ITP) is an autoimmune disorder marked by decreased platelet counts and heterogeneous clinical outcomes ranging from newly diagnosed to chronic, relapsing, or refractory phases. T cell dysregulation and impaired regulatory T cell (Treg) functions are central to its pathophysiology. Cytokines—critical mediators of immune cell signaling—are key contributors to the inflammatory milieu driving disease progression. Elevated pro-inflammatory cytokines (e.g., IL-6, TNF-α) and dysregulated anti- inflammatory responses, including altered IL-10 production, may reflect or influence disease activity. This study aims to profile cytokine expression in 20 participants divided into four groups: healthy controls (n=5), newly diagnosed ITP (n=5), patients in remission (n=5), and those experiencing relapse (n=5). Peripheral blood samples will be collected for complete blood counts and plasma cytokine analysis. Cytokine concentrations (IL-6, TNF-α, IFN-γ, IL-17A, IL-10, IL-2 and IL-4) will be quantified via Cytometric Bead Array (CBA) and analyzed using flow cytometry. We hypothesize that newly diagnosed and relapsing patients will exhibit elevated levels of pro-inflammatory cytokines, while remission-phase patients will show normalized or elevated anti-inflammatory cytokine levels. An inverse correlation between pro-inflammatory cytokines and platelet counts—and a direct correlation with IL-10—is anticipated. By delineating cytokine dynamics across disease states, this study seeks to identify potential immunological biomarkers of disease activity and therapeutic response. These insights could contribute to more precise, personalized strategies for disease monitoring and treatment in ITP.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

In-silico transcriptomic study of $P2X_7$ and PA_{2A} in T lymphocytes in metabolic syndrome

Urbina-Rodríguez, Rubén Omar [1,2]; Paz-Rodríguez, Víctor Alejandro [1,2]; Muñoz-López, Alejandra [3]; Cortes-García, Juan Diego [1]; Turiján-Espinoza, Eneida [1]; Parra-Rojas, Isela [4]; Vargas-Morales, Juan Manuel [1]; Portales-Pérez, Diana Patricia [1,2].

[1]Facultad de Ciencias Químicas de la Universidad Autónoma de San Luis Potosí. San Luis Potosí, San Luis Potosí, México. [2]Sección de Medicina Molecular y Traslacional, Centro de Investigación en Ciencias de la Salud y Biomedicina (CICSaB). San Luis Potosí, San Luis Potosí, México. [3]Universidad del Valle de México, Campus San Luis. San Luis Potosí, San Luis Potosí, México. [4]Laboratorio de Investigación en Obesidad y Diabetes, Facultad de Ciencias Químico Biológicas, Universidad Autónoma de Guerrero, Chilpancingo, Guerrero, México.

E-mail: omar.urbina.rdz@gmail.com

Metabolic syndrome (MetS) is a multifactorial disorder involving central obesity, dyslipidemia, hypertension, and hyperglycemia. MetS is associated with chronic, low-grade inflammation, or metainflammation. This inflammation is driven by dysfunctional adipocytes that release proinflammatory cytokines, as well as extracellular adenosine triphosphate (ATP) and adenosine. T lymphocytes may contribute to this inflammatory environment through purinergic receptors, particularly P2X7 (encoded by P2RX7) and A2A (encoded by ADORA2A). These receptors function as immunometabolic sensors. P2X7 promotes polarization toward proinflammatory T cell phenotypes (Th1/Th17), while A2A supports anti-inflammatory responses and regulatory T cell development. This study aimed to identify genes differentially expressed in MetS and evaluate their expression in immunometabolic tissues. Additionally, we explored the functional relationship of these genes with P2RX7 and ADORA2A in T lymphocytes. We analyzed eight transcriptomic datasets (GSEs) related to obesity, dyslipidemia, being overweight, and type 2 diabetes. Using DESeq2 in RStudio, we identified differentially expressed genes and visualized them with volcano plots. Using UMAP and heatmaps, we assessed gene expression and cellular clustering in white and brown adipose tissue, as well as in peripheral blood mononuclear cells. We constructed functional interaction networks with Cytoscape. A total of 139 differentially expressed genes were identified across the eight GSEs (67 were overexpressed, and 72 were underexpressed). Thirty-six of these genes were expressed in all three cell types in cluster 13. Of these, eight showed functional interactions with P2RX7 and ADORA2A: NLRP3, TNF, CD163, CLEC7A, FYN, TIMP1, HIF1A, and PANX1. Elevated extracellular ATP levels in metabolic syndrome (MetS) may trigger P2X7 hyperactivation, promoting proinflammatory gene expression. Concurrently, reduced ADORA2A signaling could impair immunomodulatory responses. These findings suggest a potential mechanism linking purinergic signaling to inflammation in MetS; however, they require experimental validation.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Effect of Probiotics on Secretory Immunoglobulin A in Saliva of Healthy Adults

Urenda-Castañeda, Cynthia Gabriela [1]; Urenda Castañeda, Denisse [2].

[1]Universidad Autónoma de Ciudad Juárez, Departamento de Ciencias de la Salud, Ciudad Juárez, Chihuahua, México. [2]The University of Texas at El Paso, Department of Mathematical Science, El Paso, Texas, United States of América.

E-mail: al190348@alumnos.uacj.mx

Secretory immunoglobulin A (sIgA) in saliva is a key biomarker of mucosal immunity. Emerging evidence suggests oral probiotics may modulate sIgA levels, but results remain inconsistent. To evaluate the effect of oral probiotic supplementation on salivary sIgA concentration and secretion rate in healthy adults using a systematic review and meta-analysis of randomized controlled trials (RCTs). This review was conducted following PRISMA 2020 and Cochrane Handbook guidelines. The protocol was registered in PROSPERO (CRD420251053952). A comprehensive search was performed in PubMed, Cochrane Library, ScienceDirect, and Google Scholar from 2010 to April 2025. Inclusion criteria followed PICOS: healthy adults (P), defined oral probiotics (I), placebo (C), outcomes measured as sIgA in µg/mL or µg/min (O), and double-blind RCTs (S). Risk of bias was assessed using RoB 2. Quantitative data were synthesized via fixed-effects meta-analysis; qualitative synthesis was performed when meta-analysis was not feasible. The meta-analysis of RCTs revealed a significant increase in salivary IgA favoring probiotics. Notably, three studies showed statistically significant effects (95% CI), while others reported non-significant or opposing trends. Three studies measuring secretion rates (µg/min) reported consistent increases (+101.5 µg/min, $p < 0.05$). Subgroup analysis revealed that probiotic interventions increased salivary IgA levels, with high-dose regimens ($\geq 10^{10}$ CFU/day; 3/3 studies, $p < 0.05$) and Lactobacillus strains (3 studies, $p < 0.05$). Short-term (≤4 weeks) and long-term (>4 weeks) interventions both showed positive effects, though high-dose protocols demonstrated robust responses. Non-Lactobacillus strains (Streptococcus, Pediococcus) also elevated IgA, except for Bifidobacterium ($p = 0.04$). Oral probiotic supplementation, particularly with high-dose Lactobacillus strains, likely increases salivary sIgA in healthy adults. While evidence quality was moderate (GRADE), findings support probiotic use for enhancing mucosal immunity.

Funding: Self-funded.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Helicobacter pylori induces proteomic changes in human PBMCs and their exosomes

Urieta-Benítez, Luis Ángel [1 2]; Encarnación-Guevara, Sergio [3]; Garibay-Cerdenares, Olga Lilia [4]; Hernández-Ortiz, Magdalena [3]; Martínez-Batallar, Gabriel [3]; Román-Fernández, Ilce Valeria [7]; Ramírez-Ruano, Mónica [5]; Avalos-Gómez, Christian [6]; Fernández-Tilapa, Gloria [1 2].

[1]Autonomous University of Guerrero, Faculty of Chemical Biological Sciences, Infectious Diseases and Cancer Research Laboratory, Chilpancingo, Guerrero, México. [2]Autonomous University of Guerrero, Faculty of Chemical Biological Sciences, Biomolecules Research Laboratory, Chilpancingo, Guerrero, México. [3]National Autonomous University of México, Center for Genomic Sciences, Cuernavaca, Morelos, México. [4]Autonomous University of Guerrero, Faculty of Chemical Biological Sciences, Molecular Biomedicine Laboratory, Chilpancingo, Guerrero, México. [5]Autonomous University of Guerrero, Faculty of Chemical Biological Sciences, Functional Proteomics and Genomics Laboratory, Chilpancingo, Guerrero, México. [6]National Polytechnic Institute, Center for Research and Advanced Studies, Cell Biology, México City, México. [7]University of Guadalajara, University Center of Health Sciences, Biomedical Sciences Research Institute, Guadalajara, Jalisco, México.

E-mail: luanuribee@gmail.com

In the human gastric mucosa, Helicobacter pylori induces chronic inflammation sustained by proinflammatory cytokines secreted by mononuclear cells that infiltrate the infected site. To analyze changes in the cellular and exosomal proteomes of human peripheral blood mononuclear cells (PBMCs) in response to infection with H. pylori s1m1/cagA+, PBMCs from four healthy donors—H. pylori-negative and without dyspeptic symptoms—were infected for 24 hours with the ATCC 26695 strain. Infection was confirmed by western blot detection of CagA. Exosomes were isolated from the supernatants of Control-PBMCs and H. pylori-PBMCs via ultracentrifugation, and characterized by electron microscopy and by detection of CD9, CD63, and CD81. Total proteins from Control-PBMCs, H. pylori-PBMCs, and their exosomes were analyzed by LC-MS/MS mass spectrometry. Both human and H. pylori proteins were identified in the proteomes of H. pylori-PBMCs and their exosomes. More than 95% of the proteins identified in Control-PBMCs, H. pylori-PBMCs, and their exosomes were shared among the four donors. While 4,319 proteins were commonly detected in Control-PBMCs, this number decreased to 3,649 in H. pylori-PBMCs, of which 132 were exclusively found in the infected cells. In exosomes from Control-PBMCs, 2,066 proteins were common to all four donors, decreasing to 1,727 in exosomes from H. pylori-infected PBMCs. Among these, 131 proteins were detected only in exosomes from H. pylori-PBMCs. These results indicate that PBMCs exposed to H. pylori reduce the number of proteins they express, synthesize specific proteins, and load their exosomes with both host-derived and bacterial proteins. Special thanks to Rojas-Morales María de Lourdes, from the National Laboratory for Experimental Services (LaNSE), for her valuable support in acquiring the transmission electron microscopy images.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Therapeutic vaccine protocol against breast cancer

Urrutia-Medina, Estefany Daniela [1]; Sánchez-Ramírez, Blanca Estela [1].

[1]Facultad de Ciencias Químicas. Universidad Autónoma de Chihuahua. Chihuahua, Chihuahua, México.

E-mail: a330002@uach.mx

The development of a therapeutic vaccine against breast cancer aims to activate the patient's immune system to recognize and destroy tumor cells. A common approach uses MCF-7 breast cancer cells, which contain specific antigens on their surface, as targets for the immune system. The process begins with the isolation of dendritic cells from the patient. These immune cells are responsible for capturing and presenting antigens to other immune cells. The dendritic cells are incubated with MCF-7 tumor cells to absorb the proteins and antigens from their surface. Once loaded with these tumor antigens, the dendritic cells are stimulated to activate their ability to induce an effective immune response. An innovative approach in this protocol is the use of messenger RNA (mRNA). mRNA is used to encode the specific antigens of the tumor cells, allowing the patient's immune system to produce these antigens and present them to T cells. This method has the advantage of being highly personalized, as it adapts to the molecular characteristics of each patient's tumor. In addition, in some cases, specific oncogenes such as HER2, commonly overexpressed in certain types of breast cancer, are included. These oncogenes serve as targets for T cells, directing the immune response more precisely to the tumor cells. Once activated, the loaded and stimulated dendritic cells are reintroduced into the patient, where they induce an immune response directed against the tumor cells. This approach has the potential to offer personalized and more effective treatments for breast cancer, improving precision and reducing side effects.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Loss of arpin expression in acute myeloid leukemia cells triggers bone marrow spheroid colonization

Valencia-Escamilla, Esther Alejandra [1]; Hernández Almaraz, Karina Belén [1]; Sánchez Argáez, Ana Beatriz [1]; Correa Lara, Maximiliano Víctor Manuel [1]; Vargas Robles , Hilda [1]; Núñez -Enríquez, Juan Carlos [2]; Schnoor, Michael [1].

[1]Centro de Investigación y Estudios Avanzados del Instituto Politécnico Nacional (CINVESTAV-IPN), Biomedicina Molecular, CDMX, México. [2]Hospital de Pediatría, CMN SXXI, IMSS. División de Investigación en Salud, CDMX, México.

E-mail: esther.valencia@cinvestav.mx

Acute myeloid leukemia (AML) is a myeloproliferative neoplasm with an overproduction of neoplastic blasts of myeloid precursors. These blasts exhibit a high capacity for extramedullary infiltration, which requires the formation of lamellipodia and the rearrangement of the actin cytoskeleton, which is regulated by the Arp2/3 complex. Arpin is an endogenous inhibitor of the Arp2/3 complex responsible for regulating the persistence and speed of migration. It has been reported that arpin expression is decreased in breast cancer, gastric adenocarcinoma, and PDAC, but its role in leukemia is unknown. In this study, the aim is to evaluate the relationship between arpin expression, actin remodeling and the migratory capacity of THP-1 cells, an AML cell line, and primary tumor cells from patients with AML. THP1 cells were used to generate an arpin knock-down with a 95% decrease in protein expression. Arpin-depleted THP-1 cells were used to perform colonization assay in spheroids formed by the mesenchymal stromal cell line HS-5. we found that the decrease of arpin was significantly correlated with an increase in the colonization capacity. To verify that arpin exerts this effect via the Arp2/3 complex, the knock-down cells and control cells were treated with CK-666 and then allowed to colonize HS5-derived spheroids. Interestingly, CK666 treatment in arpin-depleted cells decreased colonization, suggesting that arpin regulates BM colonization by AML cells via the Arp2/3 complex. Finally, bone marrow mononuclear cells of patients with AML were analyzed by flow cytometry, and a decrease in arpin expression was observed compared to healthy donors. In conclusion, arpin is decreased in primary AML cells and loss of arpin in the AML cell line THP-1drives the BM colonization capacity. We are currently investigating if the observed effects depend on arpin-regulated actin dynamics and chemokine receptor expression in AML cells.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

The role of FMNL-2 during inflammation in cremasteric postcapillary venules

Valenzuela-Padilla, Arturo Armando [1]; Vargas-Robles, Hilda [1];
Schnoor, Michael [1].

[1]Department of Molecular Biomedicine, CINVESTAV-IPN, México City, México.

E-mail: arturo.valenzuela@cinvestav.mx

Inflammation is a complex biological response of the body´s immune system against stimuli such as pathogens, damaged cells, or irritants. This response is mediated by tissue-resident cells, generating a proinflammatory microenvironment, which activates endothelial cells leading to the recruitment of circulatory neutrophils to the damaged tissue. Neutrophil extravasation is an adhesive and migratory event that depends on actin remodeling, a cellular response regulated by different actin-binding proteins (ABP). We have previously analyzed the role of ABP such as cortactin and the Arp2/3 complex during inflammation in the mouse cremaster, but functions of the G-actin elongation factor formin like-2 (FMNL2) during cremasteric inflammation remain unexplored. FMNL2 is a ubiquitous and highly conserved ABP in mice and humans that is involved in filopodia and lamellipodia formation, vesicular trafficking, and cell-cell junction formation. It has also been shown to regulate cell adhesion and migration. Therefore, we hypothesize that FMNL2 depletion reduces neutrophil extravasation in vivo. FMNL2-dficient mice are viable. We found that neutrophils and cremasteric postcapillary endothelial cells express FMNL2. Intravital microscopy studies showed that in FMNL2-deleted mice neutrophils roll faster, adhere and transmigrate less compared to wild-type littermate mice suggesting that FMNL2 plays an important role during the innate immune response. We are currently studying the molecular mechanism underlying this neutrophil extravasation defect including actin polymerization.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

Characterization of Exhausted T Cell Subpopulations in Type 1 Diabetes Mellitus

Vaquero-García, Ricardo Michelle [1 2]; Chávez-Rueda, Adriana Karina [1]; Legorreta-Haquet, María Victoria [1]; Santana-Sánchez, Paola [1].

[1]Unidad de Investigación Médica en Inmunología, UMAE Hospital de Pediatría, Centro Médico Nacional Siglo XXI, IMSS. Ciudad de México. [2]Posgrado en Ciencias Biológicas, UNAM. Ciudad de México.

E-mail: ricardo-michelle@hotmail.com

Type 1 Diabetes Mellitus (T1D) is an autoimmune disease characterized by the destruction of pancreatic beta cells, which produce insulin. This process is primarily mediated by cytotoxic CD8+ T cells, with the cooperation of activated CD4+ T cells. The resulting dysregulated immune response leads to progressive loss of insulin secretion and dependence on exogenous insulin. During the early stages of the disease, exhausted T cell populations (Tex) have been identified. These cells are characterized by the expression of inhibitory receptors (PD-1, TOX, CTLA-4, TIGIT, TIM-3) and reduced effector functions. Tex cells arise in the context of prolonged antigenic stimulation and can be classified into progenitor (pTex), intermediate (iTex), and terminal (termTex) subpopulations, with progressive loss of function and proliferative potential.

The aim of this study was to characterize Tex subpopulations in patients with T1D. Peripheral blood mononuclear cells (PBMCs) were isolated from patients with T1D and healthy controls. Cells were phenotyped by multiparametric flow cytometry using antibody panels targeting Tex subpopulations. Analysis of inhibitory marker expression was performed using unsupervised methods, applying UMAP for dimensionality reduction and FlowSOM for subpopulation clustering.

Results showed a similar overall expression of inhibitory markers between the two groups. However, total CD8+ Tex frequencies were significantly lower in T1D patients. Subpopulation analysis revealed comparable frequencies of iTex between groups, but a marked reduction of pTex in T1D patients. No termTex cells were detected in peripheral blood in either group. These findings suggest that the reduction of pTex and iTex in peripheral blood may reflect the recruitment of more differentiated Tex cells into pancreatic tissue in response to antigenic stimulation.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Both the TNF-α SNP and the IL-12 SNP are important for congenital transmission and disease caused by Toxoplasma gondii in humans

Vargas-Villavicencio, José Antonio [1]; Rico-Torres, Claudia Patricia [1]; Correa-, Dolores [2].

[1]Instituto Nacional de Pediatría, Secretaría de Salud.. [2]Centro de Investigación en Ciencias de la Salud. Facultad de Ciencias de la Salud, Universidad Anáhuac México.

E-mail: javvcs@yahoo.com.mx

Toxoplasma gondii infection of females during pregnancy may cause congenital toxoplasmosis in several species, including humans. Both the trimester of gestation and the virulence of the parasite are quite well established as risk factors for vertical transmission and offspring damage degree. Although maternal and fetal inflammatory/regulatory profiles are suggested to influence congenital infection or clinical outcome, studies on this topic remain limited. To further support knowledge acquisition about this, we studied SNPs in regulatory regions of cytokine genes in 30 mother/newborn pairs, five mothers and two babies with known status regarding vertical transmission and clinical outcome of positives. The binomials came from three different sources: prenatal and postnatal screening and clinical cases arrived at third-level hospitals for medical care. Polymorphisms of IL-2, IL-10, IL-12, IL-17, TNF-a, and TGF-b genes were obtained by sequencing, and the genotypes and allelic frequencies were related to transmission and offspring clinical outcome. In children, the polymorphic allele “G” of the TNF-a -308 (associated with low levels of the cytokine), as well as the low and high level-related homozygous GG or TT genotypes of the IL-12reg (rs 3212227) UTR gene region, were related to congenital infection. Both cytokines have been shown expressed by the cyto- and syncytiotrophoblast; thus, the fetus might be able to regulate the infection at the placental level. In mothers of infected children, the T allele of the IL-12reg gene region was associated with more severe disease in their children, suggesting that a strong maternal response is necessary to reduce the spread of the parasite through the umbilical cord blood.

Área del artículo: **Inmunología veterinaria y evolutiva**

Influence of Pathogen Virulence on the Proteomics of Immune Memory in Tenebrio molitor

Vázquez-Fuerte, Fernanda [1]; Bucio-Caravantes, Anette [1];
González-Hernández, Angélica [2]; Torres-Guzmán, Juan Carlos [2];
Contreras-Garduño, Jorge [1].

[1]Escuela Nacional de Estudios Superiores, Unidad Morelia, UNAM, Edificio de Investigación Í, Morelia, Michoacán, México. [2]Universidad de Guanajuato, Departamento de Biología de la División de Ciencias Naturales y Exactas, Guanajuato, Guanajuato, México.

E-mail: vaf.fernanda@gmail.com

An immune memory-like response has been demonstrated in invertebrates and its plasticity is shaped by pathogen virulence, but it remains unclear which molecular mechanisms are involved in this response. We tested whether the virulence of Metarhizium brunneum of two strains, Cherry (moderate virulence, expressing red fluorescent protein) and CAT (high virulence, overexpressing catalase), affects the immune memory of Tenebrio molitor. Larvae were primed with sublethal doses and re-challenged with lethal doses of homologous or heterologous strains ten days later. Survival, total protein kinetics were analyzed. Our results showed that tarvae challenged with homologous Cherry strain exhibited significantly higher survival than those with heterologous Cherry-CAT exposure, indicating strain-specific immune protection. In contrast, the CAT strain did not show evidence of innate memory. Interestingly, total protein levels reached their peak 24 hours after the second homologous challenge, regardless of the strain. Moreover, the response kinetics exhibited a biphasic pattern from the time of priming through to post-challenge. Preliminary gene expression results indicate differential expression of Tenecin 3, particularly after Cherry homologous challenges. To deeper understand the mechanisms, we are conducting a proteomic analyze to identify proteins linked to immune memory. We expect to find different protein profiles depending on the fungal strain used. These data will help us pinpoint specific pathways and molecular players involved in regulating memory, potentially through epigenetic, metabolic, or endocycle mechanisms. For now, our findings suggest that immune memory in T. molitor is influenced by pathogen virulence, with potential involvement of changes in proteins. Altogether, this work aims to shed light on how pathogen virulence affects immune memory in insects and how this could be linked to changes at the protein level.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

IgG-ghrelin and leptin-reactive antibodies differ in individuals according to types of diet

Vázquez -Solórzano, Rafael [1]; García- Bustillos, Adriana [1]; Lares -Michel, Mariana [2]; Reyes Castillo, Zyanya [1].

[1]Laboratorio de Biomedicina y Biotecnología para la Salud, Centro Universitario del Sur, Universidad de Guadalajara, México. [2]Instituto de Investigaciones en Comportamiento Alimentario y Nutrición (IICAN), Centro Universitario del Sur, Universidad de Guadalajara, México.

E-mail: rafael.vazsol@gmail.com

Low-affinity reactive-antibodies to hormones such as ghrelin and leptin have been identified in healthy subjects and altered in individuals with metabolic diseases. Diet impacts gut microbiota composition, and molecular mimicry of microbial proteins with these hormones may modulate this antibody system. We analyzed the circulating levels of IgG antibodies reactive to ghrelin and leptin in adults who follow an omnivorous or plant-based diet and its relationship with anthropometric, biochemical parameters and relative abundance of four intestinal microorganisms with mimetic proteins to leptin. This cross-sectional study included 89 adults (18-50 years) categorized in two groups: omnivorous diet (n=56) and plant-based diet (including vegetarians, lacto-ovo-vegetarians and vegans, n=33). Body composition and biochemical parameters (glucose, triglycerides, total cholesterol) were evaluated. An in-house ELISA was performed to quantify IgG´s ghrelin and leptin antibodies in their different fractions (free, total and immune-complexes percentage). qPCR was performed to quantify the relative abundance of Candida albicans, Lactococcus Lactis, Akkermansia muciphila and Faecalibacterium prausnitzii. The free fraction of IgG ghrelin reactive antibodies levels was found lower in omnivores than plant-based individuals ($p=0.0128$), total and the immune complexes fraction was higher in omnivorous compared to individuals following a plant-based diet ($p=0.0165$, $p=0.0398$). No differences were detected in IgG anti-leptin antibodies according to diet, sex or BMI. Relative abundance of Candida albicans was higher in plant-based diet individuals compared to omnivores ($p=<0.0001$). In omnivore group, a negative correlation between the free fraction of IgG and body fat was detected ($r=-0.2668$, $p=0.0469$). In plant-based group a positive correlation between IgG immune-complexes fraction and body fat was found ($r=0.3614$, $p=0.0421$). These findings suggest that different dietary patterns might modulate antibody production possibly by the interaction between gut microbiota and different nutrients intake, evidencing novel interactions between diet, the immune system and metabolism.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Evaluation of CD147, MMPs, TIMPs in Antibody-Mediated Renal Rejection: CD147-Induced MMP Production

Vázquez-Toledo, Miguel Ángel [1]; Sánchez-Muñoz, Fausto [2];
Springall-del Villar, Rashidi [3]; Chacón-Salinas, Rommel [1].

[1]Instituto Politécnico Nacional, Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, CDMX, México. [2]Instituto Nacional de Cardiología Ignacio Chávez, Departamento de Fisiología, CDMX, México. [3]Instituto Nacional de Cardiología Ignacio Chávez, Departamento de Inmunología, CDMX, México.

E-mail: miguelpradyu1401@gmail.com

Acute antibody-mediated rejection (ABMR) is one of the main causes of renal allograft dysfunction. Current diagnostic methods, mainly renal biopsy, are limited by their invasive nature, cost, and low sensitivity. Therefore, the identification of noninvasive, specific, and reliable biomarkers is a clinical necessity. Recent evidence suggest that CD147 (EMMPRIN), metalloproteinases (MMPs), and their inhibitors (TIMPs) play key roles in graft injury through their involvement in inflammation, fibrosis, and glomerular dysfunction; however, their utility as biomarkers has not been explored. In addition, macrophage infiltration is associated with poor ABMR outcomes, but the specific role of CD147 in facilitating infiltration through MMP production remains unclear. To evaluate the levels of CD147, MMPs, and TIMPs in transplant recipients and their association with histopathological findings, as well as CD147-mediated MMP production CD147 in macrophages. Twenty-seven kidney transplant recipients with and without ABMR were evaluated, and plasma levels of CD147, MMPs, and TIMPs were subsequently quantified by ELISA. In addition, THP-1 macrophage-like cells were stimulated with patient serum to evaluate MMP production in vitro. Patients with ABMR showed a significant increase in MMP-1, MMP-2, and MMP-3. However, TIMP-3 levels increased in patients without rejection, showing in both cases correlations with the severity of histopathological findings and a good diagnostic capacity to discriminate patients with rejection. In addition, serum from patients with high concentrations of CD147 increased MMP production in THP-1 cells. MMPs and TIMPs are emerging as promising biomarkers. In addition, high levels of CD147 were associated with increased MMP production in macrophages.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

In vitro modulation of inflammatory biomarkers by metabolites from probiotic microorganisms

Vega-Balderrama, Diana [1]; Ochoa-González, Fátima [1];
Castañeda-Delgado, Julio Enrique [2].

[1]Universidad Autónoma de Zacatecas, Unidad Académica de Ciencias Químicas, Zacatecas, Zacatecas, México. [2]Instituto Mexicano del Seguro Social (IMSS), Unidad de Investigación Biomédica (UIBMZ), Zacatecas, Zacatecas, México.

E-mail: 42101816@uaz.edu.mx

Chronic inflammation has become a key factor in the development of various pathologies, including metabolic and autoimmune disorders. In recent years, the study of probiotic microorganisms has emerged as a promising strategy for modulating the immune response and reducing inflammatory biomarkers. Multiple studies have reported that various species of Lactobacillus, Bifidobacterium, and certain yeasts can influence the regulation of the balance between pro- and anti-inflammatory cytokines in animal models. However, some areas remain underexplored, such as the implementation of in vitro studies, the identification of the most effective strains, and the role of bioactive compounds in contributing to immunomodulation.

This study proposes the isolation and culture of mononuclear (PBMC) and polymorphonuclear (PMN) cells from human peripheral blood, and the evaluation of the anti-inflammatory effects of bioactive compounds produced by Lactobacillus probiotics. These effects will be assessed by stimulating the cells with an aqueous-phase extract from the respective microbial cultures, followed by a comparative analysis of inflammatory biomarker levels (IL-6, IL-10, TNF-α) using polymerase chain reaction (PCR).

Although the experiments are still ongoing, existing literature supports the notion that bacterial metabolites derived from probiotics possess anti-inflammatory activity and, therefore, may exert immunomodulatory effects on PBMC and PMN cell cultures. Thus, this study presents an in vitro strategy to explore the immune response to bacterial metabolites. The results (currently in progress) will contribute to a better understanding of the relationship between the microbiota and the immune system, which is highly relevant in the context of chronic inflammatory diseases, infections, and the development of probiotic-based therapies. As such, this proposal opens the door to future research in the field of immunomodulation.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Innate Immune Elements of Prostate Epithelial Cells Modulated by Trichomonas vaginalis

Vega-Muñoz, Frida [1]; Ramírez-Ledesma, María Guadalupe [2];
Alva-Murillo, Patricia Nayeli [1].

[1]Laboratorio de Inmunomicrobiología Molecular, Departamento de Biología, División de Ciencias Naturales y Exactas, Campus Guanajuato, Universidad de Guanajuato, Noria Alta S/N; CP 36050, Guanajuato, Guanajuato, México. [2]Departamento de Neurobiología Celular y Molecular, Instituto de Neurobiología, Universidad Nacional Autónoma de México, Boulevard Juriquilla #3001, CP 76230, Juriquilla Querétaro, Querétaro, México.

E-mail: f.vegamunoz@ugto.mx

Trichomoniasis is the most common non-viral sexually transmitted infection (STI) worldwide, caused by the parasite Trichomonas vaginalis. This disease affects both women and men; however, most cases are asymptomatic, which is related to disease transmission and adverse health outcomes. The pathogenesis of this STI has been more studied in women than in men. Nevertheless, only a few reports address host-pathogen interactions in men. It is known that these interactions depend on the parasite strain, the multiplicity of infection (MOI), stimulation time, and the host cell type. In this context, prostate epithelial cells may be among the first to detect the parasite, suggesting a key role in the innate immune response of the male genitourinary tract. Therefore, the aim of this study was to evaluate innate immune elements -cytokines, chemokines, and nitric oxide- of prostate epithelial cells (RWPE-1) in response to T. vaginalis (CDC-085 strain). First, the cytotoxic effect of T. vaginalis (MOI 1:0.4 and 1:4) on RWPE-1 cells was evaluated using the MTT reduction assay. The results showed that MOI of 1:4 (24 h) reduced the metabolic activity of epithelial cells up to 40%. Stimulation with T. vaginalis (MOI 1:0.4) induced nitric oxide (NO) production in RWPE-1 cells, reaching the highest concentration (125.26 µM) at 24 h post-interaction (hpi). Additionally, the relative gene expression of cytokines and chemokines (IL-1β, TNF-α, IL-8, IL-10, and TGF-β) was evaluated in epithelial cells stimulated by T. vaginalis at 0.5, 3, 6, 24, and 48 hpi. At 48 hpi, the mRNA levels of proinflammatory cytokines TNF-α and IL-1β increased 1312- and 1416-fold, respectively. Likewise, the mRNA level of the chemokine IL-8 increased 60- and 2156-fold at 6 and 48 hpi, respectively. These results suggest that T. vaginalis CDC-085 strain modulates the inflammatory response in prostate epithelial cells, providing new insights into the pathogenesis of trichomoniasis in men.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de enfermedades infecciosas**

Trichomonas vaginalis modulates innate immune elements of vaginal epithelial cells

Vega-Muñoz, Frida [1]; Rodríguez -Hernández, María Elena [1];
Rodríguez-Solís, Mayra Cecilia [1]; Reyes-Cortés, Ruth [2];
Palomera-Sánchez, Zoraya [3]; Alva-Murillo, Patricia Nayeli [1].

[1]Laboratorio de Inmunomicrobiología Molecular, Departamento de Biología, División de Ciencias Naturales y Exactas, Campus Guanajuato, Universidad de Guanajuato, Noria Alta S/N; CP 36050, Guanajuato, Guanajuato, México.. [2]Laboratorio de Biología del Parasitismo, Departamento de Biología, División de Ciencias Naturales y Exactas, Campus Guanajuato, Universidad de Guanajuato, Noria Alta S/N; CP 36050, Guanajuato, Guanajuato, México.. [3]Facultad de Medicina Veterinaria y Zootecnia, Universidad Michoacana de San Nicolás de Hidalgo, Avenida Acueducto esq. Tzintzuntzan S/N, Col. Matamoros, CP 58240, Morelia, Michoacán, México.

E-mail: f.vegamunoz@ugto.mx

Trichomonas vaginalis the parasite responsible for trichomoniasis, the most common non-viral curable sexually transmitted infection (STI) worldwide, affecting both women and men. This parasite colonizes the mucosal surfaces of the urogenital tract, and ~50% of infected women remain asymptomatic, which facilitates the establishment of an inflammatory environment in the genitourinary system. In women, this STI is associated with health complications such as an increased risk of acquiring human immunodeficiency virus and human papillomavirus, and cervical cancer. In pregnant women, it may lead to preterm birth. Although trichomoniasis is the most common curable STI worldwide, with 26.4 million new cases annually, it remains an understudied – particularly in terms of the vaginal epithelial immune response. In this sense, the aim of this study was to evaluate the effect of T. vaginalis (GT-13 and CDC-085 strains) on innate immune components of vaginal epithelial cells (VK-2), including cytokines, chemokines, and nitric oxide. The results showed that both strains affected the metabolic activity of VK-2 cells in a multiplicity of infection (MOI)-dependent manner. MOIs of 1:0.5 and 1:1 were selected for further analysis. GT-13 strain (MOI 1:0.5) downregulated mRNA levels of IL-10 and TNF-α in VK-2 cells at 12 h post-interaction (hpi); however, IL-8, IL-1β, and TNF-α mRNA levels increased at 24 hpi. The CDC-085 strain (MOI 1:0.5) induced the relative gene expression of IL-10, IL-8, and TNF-α at both 12 and 24 hpi. Nevertheless, IL-1β gene expression increased (4.6-fold induction) at 12 hpi but reduced (0.3-fold induction) at 24 hpi. The relative expression of innate immunity genes at a MOI of 1:1, as well as nitric oxide production at both MOIs, remain to be determined. To date, the results suggest that T. vaginalis differentially modulates innate immune responses in vaginal epithelial cells in a strain-dependent way. This approach enhances our understanding of host-pathogen interactions and may provide insights into the immunopathogenesis of trichomoniasis.

Área del artículo: **Inmunidad innata**

Cryopreservation induce changes in phenotype and functionality of antigen-specific NK cells

Velarde-Rocha, Fernanda Michelle [1]; Montes -Zapata, Edgar Iván [1]; García -Taboada, Diego [1]; Salinas -Carmona, Mario César [1]; Rosas -Taraco, Adrián Geovanni [1].

[1]Universidad Autónoma de Nuevo León, Servicio y Departamento de Inmunología, Facultad de Medicina.

E-mail: fmichelle.vr@gmail.com

Cryopreservation is fundamental for biomedical research and recently supporting the development of cell therapies. However, the physical and chemical stress on frozen cells during cryopreservation affect the viability, phenotype, and functionality of immunological cells. CD4$^+$T lymphocytes are the most affected, whereas monocytes, NK cells, and B lymphocytes are more tolerant. The effects on NK cells depend on the cell origin, the type of manipulation prior to freezing and cryoprotective agents. The present study aimed to analyze the effect of cryopreservation on the phenotype and functionality of influenza A virus nucleoprotein-specific memory NK cells.

Peripheral blood NK cells from healthy donors were isolated by negative selection, expanded in IL-2/IL-15-supplemented medium, and stimulated with H1N1 influenza A virus nucleoprotein peptides to generate specific-memory NK cells. Cells were evaluated under two conditions: fresh and cryopreserved (3 months at −170 °C). Memory and activation markers (CD56, NKG2C, NKp46, LAMP-1, granzyme B, and IFN-γ) were analyzed by flow cytometry. The release of granzyme B, IFN-γ, and TNF-α in co-culture of NK/infected A549 was quantified by ELISA. Cytotoxic activity was assessed through cell death assays in A549 cells infected with H1N1 IAV.

Similar viability was observed in NK cells previous and after cryopreservation ($P<0.05$). NK cells showed similar NKG2C and IFN-γ previous and after cryopreservation ($P<0.05$), but high levels of NKp46 ($P=0.0015$) and LAMP-1 ($P= 0.0403$). Low intracellular and secreted granzyme B levels were found in cryopreserved NK cells ($P<0.05$). Cytotoxic activity of cryopreserved NK cells was reduced in 43%compared with those NK cells previous to cryopreservation ($P< 0.0001$).

In conclusion, these results showed that cryopreservation impacts the phenotype and functionality of influenza A virus nucleoprotein-specific memory NK cells.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Evaluation of interactions between CD40 and CD40L complex in missense (p.Ala123Glu, p.Ser222Phe, p.Gli257Arg) mutations in patients with Hyper-IgM syndrome.

Velásquez-Ortiz, María Guadalupe [1]; Jardón-Valadez, Eduardo [2]; Maravillas-Montero, José Luis[3].

[1]Posgrado en Ciencias Biológicas. UNAM, Ciudad de México. México. [2]Universidad Autónoma Metropolitana, Lerma. Departamento de Recursos de la Tierra. Estado de México, México. [3]Departamento de Medicina Molecular y Bioprocesos, Instituto de Biotecnología, UNAM, Cuernavaca, Morelos, México.

E-mail: guadalupe.vocarm@gmail.com

CD4⁺ T cells express CD40L on their membrane after activation; this molecule binds to its cognate receptor CD40, expressed on B cells. Upon complex formation, several immune responses are triggered, including somatic hypermutation and the terminal differentiation of B cells into memory B cells or plasma cells. Mutations in CD40L cause Hyper-IgM Syndrome type 1, a primary immunodeficiency characterized by elevated serum IgM levels and reduced levels of other immunoglobulin isotypes, leading to increased susceptibility to recurrent infections. In this study, we investigated the interactions within the CD40-CD40L complex that may be disrupted by CD40L mutations. The selected mutations were identified from previous reports by different research groups. A structural model of the extracellular region of the CD40-CD40L complex (comprising the homotrimeric CD40L interacting with the homotrimeric CD40) was built based on the crystal structure PDB:3QD6 and completed using AlphaFold. After model validation, mutations were introduced using psfgen, and molecular dynamics simulations were carried out in NAMD. Two 300-nanosecond trajectories were obtained and analyzed from 100 to 300 ns, once system stability was reached. We observed that the dynamics of the missense mutations differed from the wild-type complex. Specifically, RMSD and RMSF values were higher for p.Ser222Phe and p.Gly257Arg, while their radius of gyration was lower. In addition, contact analysis revealed that these mutations directly affect CD40L–CD40 interactions. Our findings suggest that missense mutations in CD40L impair CD40 binding, leading to defective B cell activation and signaling in Hyper-IgM Syndrome.

Funded by PAPIIT-DGAPA-UNAM, project IN215424.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

Influence of gestational age and birth type on neonatal CD4+ T cell activation

Ventura -Martínez, Carlos Jesús [1]; Kempis Calanis, Linda Aimara [1];
Santana Calderón, María Angélica; Rodríguez, Jorge Otoniel [1].

[1]Universidad Autónoma del Estado de Morelos, Instituto de investigación en Ciencias básicas y Aplicadas, Centro de Investigación en Dinámica Celular, Laboratorio de inmunología celular y de sistemas, Cuernavaca, Morelos, México.

E-mail: carlo.ventura@uaem.edu.mx

Neonates are a highly vulnerable population, responsible for over 40% of deaths in children under five, mainly due to infections. Their T cells exhibit a Th2-skewed response and distinct epigenetic features, including reduced chromatin accessibility at Th1 loci such as IFNG. Neonatal immune development is influenced by factors like maternal nutrition, smoking, and delivery mode. Cesarean-delivered neonates have a higher risk of developing asthma and chronic inflammation. Additionally, gestational age plays a key role, as preterm neonates (born at 26–32 weeks) show an even stronger Th2 bias and are at increased risk for conditions like necrotizing enterocolitis, bronchopulmonary dysplasia, and sepsis. In this study, we collected cord blood from healthy full-term and preterm neonates, born via vaginal delivery or cesarean section at Hospital General Parres, Cuernavaca, Morelos. Purified CD4+ T cells or cord blood mononuclear cells (CBMCs) were stimulated with either anti-CD3 + anti-CD28 or anti-CD3 + flagellin, based on evidence suggesting that TLR5 signaling provides co-stimulatory signals. We assessed proliferation, transcription factor activation (NFAT, NF-κB, AP-1), and cytokine production using flow cytometry. This included Th cytokines (e.g., IL-2, IL-4, IL-10, IFN-γ), innate cytokines (IL-1β, IL-7, IL-8), and cytotoxic molecules (granzyme, perforin). T cells from cesarean-delivered neonates exhibited enhanced proliferation, while cytokine and transcription factor responses varied depending on the stimulus. Full-term cesarean-born neonates produced fewer Th cytokines, whereas preterm neonates secreted higher levels of both Th and innate cytokines. Notably, flagellin enhanced T cell proliferation across all groups, highlighting its role as a potent TLR5-mediated co-stimulatory signal. Overall, gestational age and delivery mode significantly influence neonatal T cell responses. While vaginal delivery may provide beneficial activation cues, preterm neonates demonstrate heightened responses and a proinflammatory profile, suggesting the need for targeted immune-modulating strategies.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

CXCL10-Mediated Migration and Phenotypic Stability of Alloantigen-Specific Tregs

Vigil-Mora, Marco Antonio [1 2]; Cortés-Hernández, Arimelek [1 2]; Arteaga-Cruz, Saúl [1 2]; García-Cruz, Axel [1 2]; Álvarez-Salazar, Evelyn [1 2]; Olguín-Alor, Roxana [1 2]; Soldevila-Melgarejo, Gloria [1 2].

[1]Department of Immunology, Biomedical Research Institute, National Autonomous University of México, México City, México.. [2]National Laboratory of Flow Cytometry, Biomedical Research Institute, National Autonomous University of México, México City, México.

E-mail: vigil.marco@ciencias.unam.mx

Alloantigen-specific regulatory T cells (Tr-AS), ex vivo expanded for therapeutic purposes, express chemokine receptors such as CXCR3, which are essential for their migration to sites of inflammation. In the context of transplantation, this migratory ability may facilitate the establishment of immune tolerance toward the allograft. In vitro evaluation of Tr-AS chemotaxis —mediated by the interaction between chemokine receptors and their ligands— is crucial for predicting their in vivo migratory behavior in animal models and, ultimately, in transplant recipients.

In this study, we assessed the migratory capacity of Tr-AS cells using in vitro chemotaxis assays in response to varying concentrations of CXCL10, one of the three ligands of CXCR3. In addition, we analyzed the phenotype of migrating cells via flow cytometry. The results showed that resting Tr-AS cells migrated toward CXCL10 in a concentration-dependent manner. Notably, the migrating cells exhibited high expression of Foxp3, CD25, and CTLA-4, markers associated with regulatory function. Furthermore, other molecules —such as Helios, TIGIT, HLA-DR, and CD27— displayed increased expression depending on the concentration of CXCL10. Interestingly, after exposure to an in vitro inflammatory stimulus, Tr-AS cells retained both their migratory response to CXCL10 and the expression of immunosuppressive markers.

These findings demonstrate that ex vivo expanded Tr-AS cells not only migrate efficiently in response to CXCL10 but also preserve their migratory capacity and immunosuppressive phenotype in an inflammatory environment, reinforcing their therapeutic potential in the context of transplantation.

This work was supported by Conacyt-Fordecyt #302815 (Pronace-Salud), Mexico.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx

Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología Veterinaria y Evolutiva**

Seroprevalence of orthohantavirus in rodents from the Trans-Mexican Volcanic Belt.

Ana Laura Vigueras-Galván [1], Montserrat Elemi García-Hernández [1], Rosa Elena Sarmiento-Silva [1], Heliot Zarza Villanueva [2 *]

[1] Laboratorio de Virología. Departamento de Microbiología e Inmunología, Facultad de Medicina Veterinaria y Zootecnia, Universidad Nacional Autónoma de México. Ciudad de México, México. [2] Departamento de Ciencias Ambientales, División de Ciencias Biológicas y de la Salud, Universidad Autónoma Metropolitana Unidad Lerma. Lerma de Villada, Estado de México, México.

E-mail: ana.vigueras09@gmail.com

Orthohantaviruses are zoonotic viruses primarily transmitted to humans through contact with urine or feces from infected rodents, especially species of the Cricetidae family in the Americas. Landscape transformation, particularly due to agricultural activities, can promote increases in the abundance of key reservoirs such as *Peromyscus maniculatus*, thereby elevating the risk of transmission. Additionally, environmental factors such as precipitation, temperature, and altitudinal gradients influence the distribution of potential hosts. In Mexico, the high diversity of *Peromyscus* species suggests that other species may also participate in the virus's ecology. The Trans-Mexican Volcanic Belt, a region of high biodiversity, presents marked altitudinal variation and remains poorly studied regarding orthohantavirus ecology. This study aimed to detect the presence and seroprevalence of hantavirus in rodent communities along an altitudinal gradient in this region. Sampling was conducted in two localities: the Nevado de Toluca Flora and Fauna Protection Area (State of Mexico) and La Malinche National Park (Tlaxcala-Puebla), at three elevations: 3300, 3800, and 4200 meters above sea level. A total of 94 rodents from the family Cricetidae were captured, mainly from the genus *Peromyscus*. Serum samples were analyzed using an indirect ELISA to detect IgG antibodies against hantavirus (Sin Nombre virus). No samples tested seropositive. Although results were negative, all sampled species belong mainly to the genera *Peromyscus* and *Reithrodontomys*, which include confirmed zoonotic orthohantavirus reservoirs in North and Central America. Species such as *Peromyscus leucopus* and *P. maniculatus* are tolerant to habitat modifications and maintain coevolutionary associations with hantaviruses; therefore, *Peromyscus melanotis*, present in both volcanoes, could potentially act as a reservoir at these altitudes, although further research is needed to confirm this.

We thank SECIHTI for funding the Basic and Frontier Science project CBF2023-2024-3630 and the postdoctoral fellowship awarded to ALVG (2331314 CONV-2022).

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología Veterinaria y Evolutiva**

Hantavirus seroprevalence in rodents from the Yucatan peninsula, Mexico.

Ana Laura Vigueras-Galván 1, Laura Daniela Jaimes Serrano 1
, Montserrat Elemi García-Hernández 1 , Benjamín Roche 1 2 ,
Gerardo Suzan 3, Rosa Elena Sarmiento-Silva 1 2, Audrey Arnal
1 2 *

[1] Laboratorio de Virología. Departamento de Microbiología e Inmunología, Facultad de Medicina Veterinaria y Zootecnia, Universidad Nacional Autónoma de México. Ciudad de México, México.[2] MIVEGEC, Université de Montpellier, IRD, CNRS, Montpellier, France. [3] Departamento de Etología Fauna Silvestre y Animales de Laboratorio, Facultad de Medicina Veterinaria y Zootecnia, Universidad Nacional Autónoma de México. Ciudad de México, México.

E-mail: ana.vigueras09@gmail.com

Orthohantaviruses are zoonotic viruses transmitted by rodents that can cause epidemic outbreaks, mainly in rural areas. In Mexico, hantavirus circulation in rodents has been documented, although no human cases of Hantavirus Pulmonary Syndrome have been recorded. These viruses maintain a close evolutionary relationship with their hosts, and their persistence is influenced by viral, host, and environmental factors such as habitat transformation and host density. The Yucatán Peninsula has experienced high deforestation and habitat loss, which may affect rodent abundance and distribution. Previous studies have confirmed hantavirus circulation in rodents and serological evidence in humans from this region. This study evaluated hantavirus seroprevalence in rural communities of the Yucatán Peninsula and its relationship with the relative abundance of cricetid rodents, to identify potential epidemiological hotspots. We analyzed 220 serum samples from eigth rural localities in Campeche, Yucatán, and Quintana Roo. The overall seroprevalence was 12.56%. Among species, *Heteromys desmarestianus* showed the highest value (26.92%), while *Sigmodon toltecus* had the lowest (5.08%). By state, seroprevalence was highest in Quintana Roo (17%), followed by Campeche (15%) and Yucatán (5%). A generalized linear model revealed a significant negative relationship between cricetid rodent abundance and hantavirus seroprevalence. These findings suggest that hantavirus transmission in the Yucatán Peninsula may not be directly linked to host density, possibly reflecting a density-independent transmission pattern or the role of additional host species. The identification of high-seroprevalence localities underscores the need to strengthen epidemiological surveillance in rural areas undergoing landscape change, to prevent potential human outbreaks. This research was supported by SECIHTI through a postdoctoral fellowship awarded to ALVG (2331314 CONV-2022) and the PRONAII Virología 303002 project, with partial funding from the Institute de Recherche pour le Développement.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología veterinaria y evolutiva**

Impact of water pollution on the immune system and oxidative stress in the gills of Skiffia lermae

Villa-Villaseñor, Ivette Marai [1]; Contreras-Garduño, Jorge Alberto [2];
Herrera-Vargas, Ma. Antonia [4]; Hernández-Morales, Rubén [3];
Meléndez-Herrera, Esperanza.

[1]Estancia Posdoctoral por México-SECIHTI. Escuela Nacional de Estudios Superiores Unidad Morelia, Universidad Nacional Autónoma de México. Morelia, Michoacán, México. [2]Escuela Nacional de Estudios Superiores Unidad Morelia, Universidad Nacional Autónoma de México. Morelia, Michoacán, México. [3]Laboratorio de Biología Acuática, Facultad de Biología de la Universidad Michoacana de San Nicolás de Hidalgo. Morelia, Michoacán, México. [4]Laboratorio de Ecofisiología Animal, Instituto de Investigaciones sobre los Recursos Naturales, Universidad Michoacana de San Nicolás de Hidalgo. Morelia, Michoacán, México.
E-mail: 0620850k@umich.mx

The Goodeinae subfamily is one of the most representative groups in central Mexico. However, nearly all of its species are threatened because of multiple factors of human origin, including the contamination of water bodies. Poor habitat quality can impact the development and physiological condition of organisms, as well as their abundance. Skiffia lermae, a member of this subfamily, has seen a significant decline in its populations over the past decades. In the present study, we evaluated: (1) water quality parameters and the abundance of S. lermae over six months (June-November 2024) in La Mintzita Spring and Zacapu Lake, Michoacán, México. 2) Morphometric histological analysis, mucous cell density, phenoloxidase, catalase, and lipid peroxidation in gills of male fish from both ecosystems at the end of six months. Results revealed significant differences in water quality between the ecosystems. La Mintzita Spring exhibited higher concentrations of nitrates, electrical conductivity, and total dissolved solids, while Zacapu Lake displayed elevated oxygen levels, pH, and water temperature. Statistical analyses identify key water quality parameters (nitrate, pH, oxygen, and electrical conductivity) as predictors of S. lermae abundance. In Morphometric histological analysis, and lipid peroxidation, no significant differences were observed between organisms exposed to the water of La Mintzita Spring and those from Zacapu Lake. Exposure to water from La Mintzita Spring causes a significant increase in phenoloxidase and catalase activity. The results suggest that males from Zacapu Lake (the less polluted site) are in better physiological condition than the population of La Mintzita Spring. An ongoing study is testing potential differences in immune response and oxidative stress.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunidad innata**

Origin and Heterogeneity of Neutrophils in Oral Gingiva

Villagómez-Olea, José Guillermo; Sabalic, Maja; Marichi-Rodriguez, Francisco; Alvarez-Perez, Marco Antonio; Rosales, Carlos; Uribe-Querol, Eileen.

[1]Laboratorio de Biología del Desarrollo, División de Estudios de Posgrado e Investigación, Facultad de Odontología, Universidad Nacional Autónoma de México, México City, 04510, México. [2]Departamento de Ortodoncia, División de Estudios de Posgrado e Investigación, Facultad de Odontología, Universidad Nacional Autónoma de México, México City, 04360, México. [3]The ADA Forsyth Institute, Cambridge, MA, United States. . [4]Laboratorio de Bioingeniería de Tejidos, División de Estudios de Posgrado e Investigación, Facultad de Odontología, Universidad Nacional Autónoma de México, México City, 04360, México.

E-mail: villagomezolea@gmail.com

Neutrophils are key innate immune cells essential for maintaining periodontal health. Traditionally, neutrophils migrating to the gingival barrier have been though as short-lived, terminally differentiated cells preprogrammed for classical effector functions such as phagocytosis and pathogen clearance. However, during the pathogenesis of periodontal disease, their excessive recruitment contributes to tissue-destructive responses that sustain chronic inflammation.

Recent evidence challenges this simplistic view by revealing a spectrum of neutrophil states across different tissues, suggesting that these cells can acquire tailored functions in response to specific microenvironmental cues. Herein, through integrative reanalysis of multiple single-cell RNA sequencing (scRNA-seq) datasets, we demonstrate that gingival neutrophils represent a heterogeneous population encompassing distinct stages of maturation and differentiation. Notably, we uncover novel tissue-adapted functions linked to gingival homeostasis, including non-canonical roles such as epithelial repair. Our findings also indicates that the alveolar bone serves as the principal hematopoietic niche for neutrophil supply to the periodontium. By leveraging publicly available samples across health, disease, recovery, and bone marrow transplantation conditions, we identify previously unrecognized subpopulations shaping the neutrophil landscape in a context-dependent manner. Collectively, our study redefines the paradigm of oral neutrophils, revealing their dynamic heterogeneity and functional plasticity in periodontal health and disease.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunotecnología e inmunoterapia**

Development and Characterization of D119E, a Novel Bispecific Immune Checkpoint Inhibitor: Simultaneous Targeting of PD-1 and CTLA-4

Villanueva -López, María Alejandra [1 2]; Gómez-Castellano, Keyla María [1 2]; Pérez-Vega, Daniel [1 2]; Elizarraras-Rodríguez, Luis Javier [1 2]; Mendoza-Salazar, Ivette [1 2]; Damián-Morales, Gabriela [1 2]; Vázquez-Leyva, Said [1 2]; Pérez-Tapia, Sonia Mayra [1 2 3]; Almagro, Juan Carlos [1 2 4].

[1]Unidad de Desarrollo e Investigación en Bioterapéuticos (UDIBI), Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, CDMX, México. [2]Laboratorio Nacional para Servicios Especializados de Investigación, Desarrollo e Innovación (I+D+i) para Farmoquímicos y Biotecnológicos, LANSEIDI-FarBiotec-CONACyT CDMX, México. [3]Departamento de Inmunología, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional (ENCB-IPN), CDMX, México. [4]GlobalBio, Inc., Cambridge, MA, USA.

E-mail: alejavillanueva.lopez@gmail.com

Bispecific antibodies (BsAbs) have emerged as promising tools in cancer immunotherapy. Unlike conventional monoclonal antibodies that target a single antigen, bispecific antibodies are engineered to simultaneously bind two different epitopes, enabling them to bridge tumor and immune cells—such as T lymphocytes—to enhance antitumor responses. Their dual-targeting capacity allows for greater specificity, reduced off-target effects, and the potential for synergistic immune modulation. Here, we describe the development, expression and characterisation of D119E, a symmetric tetravalent BsAb, comprised of the anti-PD-1 domain of UDIZ-007, a human IgG1-LALA Ab developed at UDIBI, together with the anti-CTLA-4 domain derived from Quavonlimab (Cadonilimab).The BsAb was transiently expressed in Expi293F cells and purified using Protein A affinity chromatography. Physicochemical characterization by SE-UPLC and analysis of the mAb revealed a high purity of the monomeric form (97.3%) and expected structural integrity was demonstrated by SDS-PAGE under reducing and non-reducing conditions. The binding specificity was assessed by ELISA, and D119E exhibited similar or better EC 50 values compared with the reference antibodies. D119E bound PD-1 with an EC 50 of 0.034 nM, very close to Keytruda® (0.035 nM), and was more potent than control bispecific PD1 × CTLA-4 (Cadonilimab). In the case of CTLA-4, D119E had a EC 50 of 0.15 nM, which is similar to that of Cadonilimab (0.14 nM). The dual-biding ELISA data was obtained to determine the simultaneous binding of PD-1 and CTLA-4 (EC 50 = 70.3 nM), indicating that both arms were functioning correctly. Altogether, these results point to D119E as a compelling bispecific checkpoint inhibitor candidate with strong potential in cancer immunotherapy. Its binding performance and structural quality closely match and may even surpass those of Cadonilimab, supporting the evaluation of efficacy in cancer models.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Evaluation of the NOTCH pathway during DSS-induced ileal enteritis

Vizcarra-Soto, Mónica [1]; De la Torre-Báez, Juan Raúl [2]; Ortiz-Navarrete, Vianney Francisco [1]; Nava-Domínguez, Porfirio [2].

[1] Department of Molecular Biomedicine Of The Center Of Research And Advanced Studies, México City, México. [2] Department Of Physiology, Biophysics And Neuroscience Of The Center Of Research And Advanced Studies, México City, México México City, México.

E-mail: monica.vizcarra@cinvestav.mx

The intestinal epithelium acts as a physical barrier that allows nutrients absorption while prevents pathogens entry. Mantaining a functional barrier requires a balance of intestinal epithelial cell proliferation, differentiation, and death. Disruption of intestinal epithelial cell homeostasis induces the breakdown of the epithelial barrier and triggers inflammation. In this context, previous studies have shown that inflammatory events inhibit stem cell proliferation and disrupt the homeostasis of the secretory lineage, mainly Muc2 production, a process strongly regulated by the NOTCH signaling pathway. Furthermore, Muc2 ablation has been reported to induce intestinal inflammation and promote the development of colorectal cancer. Therefore, the regulation of differentiation by NOTCH signaling may be involved in intestinal epithelial barrier disruption during inflammation.

To evaluate the role of NOTCH signaling in intestinal homeostasis, we used a DSS-induced enteritis model in C57BL/6J mice. Our research focused on the ileum, as DSS treatment has previously been shown to induce mainly ileitis. On day 3 of DSS treatment, we observed increased cell death and mucus secretion in the surface enterocytes that line the villi. However, on day 6, cell death decreased, and cell proliferation increased, concomitantly the number of progenitor cells decreased, while the secretory lineage increased. Regarding Notch signaling, we observed an inhibition of NOTCH signaling on day 3, while on day 6, we noticed an increase in NOTCH activation. To identify the specific involvement of NOTCH signaling in all these processes, we treated organoids with a NOTCH activator or inhibitor and observed that the treatments differentially regulated cell death, proliferation, and mucus production and secretion.

These results suggest that in an inflammatory context, NOTCH signaling regulates the differentiation of secretory cells in the small intestine, supporting the protection and recovery of the intestinal epithelial barrier.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos B**

Mitochondrial dynamics of germinal center B cells and plasma cells in response to a lipid antigen

Wong-Baeza, Carlos [1]; Barrera-Aveleida, Giovanna Berenice [1];
Galarce-Sosa, Edgar Iván [1]; Ramos-Monteagudo, Rodrigo [1];
Runquist-Sánchez, Edgar José [1]; Trejo-Sánchez, Gabriel [1];
Cayetano-Centeno, Belén 1; Tescucano-Alonso, Rubén [2];
Reséndiz-Mora, Claudia Albany [1]; Baeza-Ramírez, Isabel [1].

[1]Laboratorio de Biomembranas, Departamento de Biquímica, Escuela Nacional de Ciencias Biológicas, Instituto Politécnico Nacional, México City, México. [2]Research Department of the Biological Pharmaceutical Chemistry Bachelor, University of Ixtlahuaca, Ixtlahuaca, México.

E-mail: charlywong@icloud.com

Cell membrane lipids can exhibit antigenic properties when they form non-bilayer phospholipid arrangements (NPAs) in bilayers, which certain drugs, such as chlorpromazine, can form and stabilize. In response to these lipid antigens, B cells can form germinal centers and generate plasma cells that produce anti-NPA IgG antibodies. These antibodies have been identified in patients with Systemic Lupus Erythematosus and contribute to autoimmunity. During these response mechanisms, changes occur in cellular metabolism where the mitochondria play a crucial role. These changes are associated with the fusion and fission of mitochondrial membranes, which directly affect the mitochondrial membrane potential (ΔΨm) and the production of mitochondrial reactive oxygen species (mtROS). In this study, we examined mitochondrial dynamics, ΔΨm, and mtROS production in germinal center B cells and IgG+ plasma cells within a mouse model of lupus induced by administering liposomes bearing stable NPAs. Anti-NPA IgG antibodies were detected in the serum of lupus mice starting from day 10 post-immunization, with a gradual increase towards day 35 post-immunization. Germinal center B cells and IgG+ plasma cells showed a significant increase in response to the NPAs. Germinal center B cells displayed fused mitochondria, with an increase in ΔΨm and a decrease in mtROS production. In contrast, IgG+ plasma cells displayed fissioned mitochondria, along with a decrease in ΔΨm and an increase in mtROS production. Our results suggest a significant association between ΔΨm and mtROS production, which influences the differentiation of germinal center B cells into plasma cells that produce anti-NPA IgG autoantibodies.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Characterization of the immunoglobulin heavy chain locus in native Mexican populations

Yáñez-Mendoza, Nefte Meni [1]; Martínez Barnetche, Jesus [2];
Godoy-Lozano, Elizabeth Ernestina[3].

[1]National Autonomous University of México, Institute of Biotechnology, Cuernavaca, Morelos, México. [2]National Institute of Public Health of México, Center for Infectious Diseases Research, Cuernavaca, Morelos, México. [3]National Institute of Public Health of México, Bioinformatics Department of Infectious Diseases, Cuernavaca, Morelos, México.

E-mail: kevinjacob11762@gmail.com

The adaptive immune system is characterized by two essential cell types: T cells and B cells. B cells express B cell receptors (BCRs) on their surface, which in soluble form are known as immunoglobulins (IGs). These molecules are central to adaptive immunity, mediating antigen recognition, pathogen neutralization, and the establishment of immunological memory. They are encoded by highly complex genomic loci, including the immunoglobulin heavy chain locus (IGH) located at the telomeric end of chromosome 14. IGH is the most diverse among these loci, containing variable (V), diversity (D), and joining (J) gene segments that contribute to antibody diversity. However, allelic diversity in IG loci remains poorly characterized due to their repetitive tandem architecture and structural complexity, which pose persistent challenges for short-read sequencing and reference-based assembly. Additionally, existing immunogenomic data are heavily skewed toward individuals of European ancestry, leaving global genetic diversity largely unexplored.

To address this gap, we analyzed 95 whole-genome sequences from 31 Native Mexican populations across Mexico using a tailored bioinformatic pipeline for short-read data, incorporating a custom IGH reference. Our analysis revealed a high density of small variants across the IGH locus, as well as novel alleles and potential functional adaptations that may confer selective advantages in these populations. These findings highlight the importance of including underrepresented groups in immunogenomic research and demonstrate how population-specific approaches can uncover hidden genetic variation, deepening our understanding of IG germline diversity and its relevance to health and disease.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Regulación inmune**

Effect of pharmacological inhibition of MIF in a diabetic retinopathy mouse model.

Zacarías-García, Mónica [1]; Rosas -González, Karla Andrea [2];
Aguilar -Garduño, Arturo [3]; Merlos-Juárez, Raúl [4]; Garfias -Becerra,
Yonathan Omar [5 6]; Vivanco-Rojas, Oscar [7 8]; Vázquez -Mendoza,
Alicia [9].

[1]Laboratorio de Inmunología Ocular, FES Iztacala, UNAM. Estado de México, México. [2]Laboratorio de Inmunología Ocular, FES Iztacala, UNAM. Estado de México, México. [3]Laboratorio de Inmunología Ocular, FES Iztacala, UNAM. Estado de México, México. [4]Laboratorio de Inmunología Ocular, FES Iztacala, UNAM. Estado de México, México. [5]Cell and Tissue Biology, Research Unit, Institute of Ophthalmology, Conde De Valenciana. Mexico City, Mexico. [6]Department of Biochemistry, Faculty of Medicine, UNAM. Mexico City, Mexico. [7]Department of Biochemistry, Faculty of Medicine, Universidad Nacional Autónoma de México. Mexico City, Mexico. [8] Cell and Tissue Biology Department, Research Unit, Institute of Ophthalmology Conde de Valenciana. Mexico City, Mexico. [9]Laboratorio de Inmunología Ocular, FES Iztacala, UNAM. Estado de México, México.

E-mail: qwermonic@gmail.com

Diabetic retinopathy (DR) is a complication of the retinal microvasculature associated with diabetes and one of the principal causes of preventable blindness in the world. The chronic hyperglycemic state underlying diabetes promote the infiltration of a wide variety of immunological cells and induces the production of inflammatory mediators such as: IL-1, IL-6, TNF-α and the macrophage migration inhibitory factor (MIF), which together perpetuate damage to the retina. In this context, given the immunological functions of the inflammatory cytokine MIF, the effects of its pharmacological inhibition are currently being explored. In particular, we evaluate the immunomodulatory activity of the MIF inhibitor (ISO-1) in a murine model of hyperglycemia induced with streptozotocin (STZ). For this purpose, 20 male mice of the Balb/c strain were used, divided into four groups: control, STZ, STZ + vehicle (dimethyl sulfoxide (DMSO)) and STZ+ISO-1. Hyperglycemia was induced by two doses of STZ (100 mg/kg) and treatment with ISO-1 (10 mg/kg) or vehicle (DMSO 10%) administration was performed intravenously at 4, 6, 8, and 10 weeks post-STZ. At 12 weeks, the expression and production of inflammatory mediators and angiogenic factors in the retina were evaluated by RT-PCR and ELISA. By fundoscopy, eye fundus images were taken. The results showed that ISO-1 did not reduce glucose levels. However, it did attenuate the retinal damage characteristic of DR, such as neovascularization, hemorrhages, and loss of pupil morphology. These findings were associated with a lower expression of il-β, il-18 and hif-1α in the retina. Together, these results suggest that the pharmacological inhibition of MIF could be a promising therapeutic in DR.

Acknowledgments: CONAHCyT for the postgraduate scholarship (2049061). This work was partially supported by PAPIIT – IN228225 and CONAHCyT–CBF-2023-2024-405.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Biología de linfocitos T**

TGF-β via TIF1γ modulates the activation and proliferation of CD8+ T cells.

Zambrano-Romero, Jesús Daniel [1]; Contreras-Castillo, Eugenio [1];
Ramos-Balderas, José Luis [1]; Flavell, Richard [2]; Licona-Limón, Paula [1].

[1]Cell biology and development department, Institute of Cellular Physiology, National Autonomous University of Mexico, Mexico City, Mexico.. [2]Department of Immunobiology, Yale University School of Medicine, New Haven, CT, USA.

E-mail: zambranojdr@gmail.com

Transforming growth factor-beta (TGF-β) is a key regulator of naïve CD8+ T cell quiescence and activation upon antigen stimulation. TIF1γ, an E3 ubiquitin ligase, controls a distinct arm of the TGF-β signaling pathway independent of Smad4. However, its role in CD8+ T cells remains unknown. This study aims to define the TGF-β-dependent functions of TIF1γ in CD8+ T cell activation. Using a Cd8-Cre floxed genetic system, we generated TIF1γ conditional knockout mice to investigate its role in CD8+ T cells. In vitro, TIF1γ KO CD8+ T cells exhibited impaired TGF-β-mediated regulation of proliferation and survival, correlating with increased c-MYC and BCL-2 expression after 24- and 48-hours post-activation. Despite TGF-β presence, TIF1γ KO cells failed to downregulate Irf4 and Nur77, suggesting altered activation dynamics. Western blot analysis revealed heightened p-ERK and p-p65 levels upon TGF-β stimulation in activated TIF1γ KO cells, indicating enhanced TCR signaling strength. Additionally, RNA sequencing analysis revealed TIF1γ KO cells upregulated proliferation and activation-associated genes in the presence of TGF-β compared to WT counterparts. In vivo, adoptively transferred TIF1γ KO CD8+ T cells in RAG2 KO mice underwent hyperproliferation and displayed an effector phenotype. By culturing naïve CD8+ T cells in suboptimal activation conditions and the presence of γ common chain cytokines, we demonstrated that TIF1γ modulates the TCR signal, but not IL-7 or IL-15 signaling. Furthermore, in a Listeria monocytogenes infection model, Cd8-Cre TIF1γflx/flx mice exhibited higher numbers of Tetramer+ CD8+ T cells in blood during the recall at day 6 post-infection, suggesting heightened activation and proliferation.These findings identify TIF1γ as a novel regulator of TGF-β-dependent CD8+ T cell activation, acting through modulation of TCR signaling in both homeostatic and proinflammatory conditions. This work expands our understanding of TGF-β's functions in immune regulation and suggests potential implications for targeting TIF1γ in immune therapies.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunidad tumoral**

Bone marrow immune microenvironment in childhood leukemia: building a prognostic test

Zamora-Herrera, Gabriela Rocío [1]; Rosas-Cruz, Arely [1];
Romo-Rodríguez, Rubí [1]; López-Blanco, Jebea Anaclara [1];
Itzmoyotl-Hernández, Ma. Teresa [2]; Solís-Poblano, Juan Carlos [2]
Pérez-Corro, Miguel Ángel [2]; Casique-Aguirre, Diana [1 3]; Ramírez-Ramírez, Dalia [1]; Pelayo, Rosana [1 4].

[1]Laboratory of Oncoimmunology and Cytomics of Childhood Cancer, Centro de Investigación Biomédica de Oriente. Instituto Mexicano del Seguro Social. Puebla, México. [2]High Specialty Medical Unit (UMAE) “Manuel Ávila Camacho”. Instituto Mexicano del Seguro Social. Puebla, México. [3]Ministry of Science, Humanities, Technology and Innovation (Secihti). México. [4]Education and Research Unit. Instituto Mexicano del Seguro Social. México City, México.

E-mail: zamorah.gabriela@gmail.com

Acute leukemia is the most common pediatric cancer, and despite global advances in therapy, children in low- and middle-income countries—including Mexico—remain disproportionately affected by poor outcomes, largely due to treatment failure and early relapse. Immunological factors within the bone marrow (BM) microenvironment are increasingly recognized as key determinants of therapeutic response and disease progression. We investigated the BM immune landscape in pediatric acute leukemia patients at diagnosis and follow-up. Primary Mesenchymal Stromal Cells (pMSC) derived from BM aspirates (n=81) were immune-profiled and correlated with clinical characteristics of patients. Also, BM aspirates and biopsies were analyzed employing mass cytometry (MC) and imaging mass cytometry (IMC) for high-dimensional profiling with over 70 markers. Results were obtained using FlowJo™, MCD Viewer, HistoCAT, CellProfiler, and visualized with R and Python. Elevated expressions of chemokine receptors CXCR3 and CXCR7 in leukemic BM compared to healthy controls were observed. Patients achieving measurable residual disease (MRD) negativity exhibited pro-inflammatory microenvironments enriched in CXCL8 and CXCL10, whereas those with persistent MRD showed immunoregulatory profiles marked by increased CD39, Galectin-9, and PD-1 expression. These immune signatures highlight the dichotomy between effective anti-leukemic responses and immune evasion mechanisms. Our findings emphasize the relevance of immune profiling in pediatric leukemia, identifying potential immunological biomarkers for risk stratification. This study contributes to our understanding of how immune modulation within BM impacts leukemia outcomes. This study was supported by SECIHTI through PRONAII Leucemia, project “Laboratorio de Citómica del Cáncer Infantil” (R-2020-785-177), and by IMSS through the project “Mesentipificación” (R-2023-785-047). We thank the clinicians from UMAE “San José” and other PRONAII hospitals, as well as the patients and their families, whose participation was essential to this research.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

HMGB1 is released during ACPA-positive serum-NET induction and correlates with disease stage and severity in Rheumatoid Arthritis

Zapata-Zúñiga, Martin [1 2 5]; Castañeda-Delgado, Julio Enrique [1]; Ochoa-González, Fátima de Lourdes [1]; Fernández-Ruiz, Julio César [1 4]; Bastian, Yadira [1]; López, Jesús Adrián [3]; Enciso-Moreno, José Antonio [1].

[1]* Unidad de Investigación Biomédica de Zacatecas, IMSS, Zacatecas, México. [2]Facultad de Medicina Humana y Ciencias de la Salud, Universidad Autónoma de Zacatecas, Zacatecas, México. [3]Laboratorio de microRNAs y Cáncer, Unidad Académica de Ciencias Biológicas, Universidad Autónoma de Zacatecas, Zacatecas México. [4]Universidad Autónoma de San Luis Potosí, San Luis Potosí, México. [5]Hospital Rural # 51, IMSS Bienestar, Villanueva, Zacatecas.

E-mail: martinzz1@yahoo.com

Rheumatoid Arthritis. (RA) is a chronic, systemic inflammatory disease that primarily affects peripheral synovial joints, leading to cartilage destruction, bone erosion, and joint deformity. Synovial inflammation is characterized by leukocyte infiltration and subsequent cell proliferation. Among the cells that infiltrate the synovium are neutrophils, which, in response to various cytokines present in synovial tissue, form neutrophil extracellular traps (NETs), a new form of cell death termed "NETosis." In NETosis, there is a marked increase in the production of reactive oxygen species. These indirectly lead to elevated cytoplasmic calcium concentrations, the activation of various enzymes and signaling pathways that lead to cytokine secretion, and ultimately to the extracellular release of the characteristic molecules of NETs, including DNA, histones, and proteins present in the neutrophil cytoplasmic granules. Among these molecules, a damage-associated molecule, such as HMGB1, may be present. This is a nuclear protein that binds to DNA and histones and, when expelled from the nuclear space, acts as an alarmin, increasing the inflammatory response. The mechanisms involved in the mobilization and intracellular dynamics of HMGB1 during the formation of NETs induced by various cytokines and molecules present in the serum of patients with RA, and whether these molecules are capable of inducing HMGB1 mobilization as an early event in NET induction, are unknown. The objective of this study was to evaluate the dynamics of HMGB1 during the formation of neutrophil extracellular traps induced by serum from patients with RA and to assess its relationship with various markers of disease activity characteristic of the inflammatory process in rheumatoid arthritis.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología clínica y traslacional**

Intestinal Protozoa Infection and systemic inflammation markers in rheumatoid arthritis

Zaragoza-García, Oscar [1]; Nogueda-Torres, Benjamín [2];
Gutiérrez -Pérez, Ilse Adriana [1]; Briceño-Cárdenas, Olivia [3];
Guzmán-Guzmán, Iris Paola [1].

[1]Laboratorio de Investigación Multidisciplinaria e Innovación Biomédica, Facultad de Ciencias Químico-Biológicas, Universidad Autónoma de Guerrero, Chilpancingo, Guerrero, México. [2]Departamento de Parasitología, Escuela Nacional de Ciencias Biológicas del Instituto Politécnico Nacional, Ciudad de México, México. [3]Centro de Investigación en Enfermedades Infecciosas, Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas, Ciudad de México, México.

E-mail: ozaga789@gmail.com

Protozoa, nematodes, and platyhelminths are of clinical interest due to their role in the modulation of immune responses. In patients with Rheumatoid Arthritis (RA), some protozoa like Blastocystis, Giardia, Cryptosporidium, and Entamoeba histolytica have been related to inflammation and increased intestinal permeability principally in the early stages of development of autoimmunity diseases. This study aims to assess the relationship between infection with intestinal parasites and systemic inflammation markers. A cross-sectional study was conducted in 50 RA patients. A fecal sample of each participant was analyzed for parasitic identification. The neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), eosinophil-to-lymphocyte ratio (ELR), and systemic inflammation response index (SIRI), were calculated from routine blood work and are used as indicators of systemic inflammation. 28% of the total sample tested were positive for the presence of intestinal protozoa. Blastocystis sp. and Endolimax nana were the most frequently identified protozoa. The NLR and ELR values were greater between patients with coinfection by protozoa, although not statistically significant (p=0.13, and p=0.18, respectively). On the other hand, SIRI and MLR values showed a tendency to diminish in the presence of single infection and coinfection for protozoa. More research is needed to understand the roles of protozoa because the infection by intestinal protozoa appears to play a dual act as both triggers or potential regulators, so should be clinically screened because the infection by intestinal parasites could be an associated factor to the clinical variability of rheumatoid arthritis.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunometabolismo**

Inflammatory Profile Assessment in Veracruz Population with Metabolic Dysfunction-Associated Steatotic Liver Disease

Zaragoza-Jiménez, Daniel [1]; Palma-Jacinto, José Antonio [2];
Montero-Ruiz, Oreth [1]; Gallardo-Hernández, Carlos Arturo [2];
González-Herrera, Sandra Luz [1]; Santiago-Roque, Isela [1].

[1]Facultad de Bioanálisis, Universidad Veracruzana, Xalapa, Veracruz, México. [2]Unidad de Servicios Analíticos en Salud Bioanalisis, Facultad de Bioanalisis, Universidad Veracruzana, Xalapa, Veracruz, México.

E-mail: dan.zj07@gmail.com

Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most common liver disease in the world, with an estimated incidence of 30% in the adult population. In Mexico, the incidence is about the 47% and is related with the increase in risk factors such as obesity, type 2 diabetes and dyslipidemia. The MASLD initially began with simple steatosis (MASL) to steatohepatitis (MASH) and can evolve into fibrosis, cirrhosis and hepatocellular carcinoma (HCC). This progression is mediated by inflammatory mechanisms involving proinflammatory cytokines such as IL-1β, IL-6 and TNF-α. The diagnosis of MASLD depends mainly on non-specific methods and, in some cases, invasives procedures such as liver biopsy, whose clinical application is limited by cost, availability and associated risks. In this context, there is a need to identify non-invasive biomarkers that allow for a more accessible, sensitive, and specific assessment of the clinical status of patients. In this perspective, we implement an observational, cross-sectional and comparative study for determinate the inflammatory profile of patients from Veracruz, México diagnosed with MASLD by ELISA quantitation serum levels of IL-1β, IL-6 and TNF-α, and their correlation with clinical stage of the disease. The results showed an increase in the serum concentration levels of IL-1β, IL-6 and TNF-α in the patients with MASLD. Moreover, a positive correlation were observed between serum levels of pro-inflammatory cytokines and the clinical stages of MASLD, supporting their usefulness as biomarkers for the non-invasive diagnosis, prognosis, and monitoring of the disease. These results may contribute to the development of more accessible and safer diagnostic strategies for the population, minimizing the use of invasive methods and improving the clinical care for patients with this disease.

Revista Bio Ciencias
ISSN: 2007-3380
https://revistabiociencias.uan.edu.mx
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de mucosas**

Chronic stress decreases IgA and its pIgR-mediated transport in mice lungs.

Zárate-Ayón, Erick José [1]; Arciniega-Martínez, Ivonne Maciel [2]; Yépez-Ortega, Mariazell [2]; Mendoza-Arroyo, Belén [1]; Martínez-Morales, Javier Alejandro [1]; Reséndiz-Albor, Aldo Arturo [1].

[1]Escuela Superior de Medicina del Instituto Politécnico Nacional, Laboratorio de Inmunidad de Mucosas, Sección de Estudios de Posgrado e Investigación, Ciudad de México, México. [2]Escuela Superior de Medicina del Instituto Politécnico Nacional, Laboratorio de Inmunonutrición, Sección de Estudios de Posgrado e Investigación, Ciudad de México, México.

E-mail: ezaratea1400@alumno.ipn.mx

Uncertainties remain regarding the impact of stress on the respiratory system, which is recognized to have the second greatest population of lymphoid tissue. This study evaluates the impact of long-term stress on lung IgA synthesis and secretion. There were two groups: 1) control and 2) chronic stress (four hours a day for eight days). Enzyme immunoassay was used to measure the levels of corticosterone in serum. By using ELISA, the levels of total IgA and s-IgA in the tracheobronchial fluid were determined. Using flow cytometry, the percenteges of IgA-producing plasma cells and intracellular CD4+ T cytokines (IL-4, IL-5, IL-10, and TGF-β) that stimulate IgA switching were ascertained. The lung cells expression of the J-chain gene and the glucocorticoid receptor gene (NR3C1). The NR3C1 and PIGR genes were identified in epithelial cells, so as the genes of interleukins (IFN, TNF, and TGF) involved in pIgR regulation. Ultimately, lung samples were recollected for histological analysis of pIgR expression. According to our findings, chronic stress raises corticosterone in serum, the glucocorticoid receptor NR3C1 in leukocytes and epithelial cells while lowering pIgR and TNF genes and decreases the pIgR expression bronchi and bronquioles as well as s-IgA, IgA-producing plasma cells and TGF-β; while the cytokines IL-4, IL-5 and IL-10 in CD4+ T cells increased. We can conclude that in the lung of BALB/c mice, prolonged stress reduces sIgA production and its pIgR-mediated transport in bronchi and main bronquioles. Exposure to constraint stress caused by pro-inflammatory stimuli can modify the respiratory humoral immune system in part. Furthermore, the findings suggest that there are other regulatory mechanisms involved in the transport and secretion of IgA through interaction with pIgR.

This work was supported by SIP 20250872 and COFFA-IPN.

Revista Bio Ciencias
https://revistabiociencias.uan.edu.mx
ISSN: 2007-3380
Memorias del XXVI Congreso
Nacional de Inmunología 2025

Área del artículo: **Inmunología de sistemas e inmunoinformática**

Bioinformatic identification of a CD8Tex lymphocyte signature in an aggressive breast cancer

Zavala-Reyes, Daniel [1]; Paz-Rodríguez, Víctor Alejandro [1]; Martínez-Leija, Ernesto [1]; González-Hernández, Osiel [1]; Turiján-Espinoza, Eneida [2]; González-Varela, Pedro Saúl [1]; Martínez-Tejeda, Lorena [1]; Jiménez-Maldonado, Brenda Vanesa [1]; Guel-Pañola, Arturo [3]; Portales-Pérez, Diana Patricia [1 2].

[1]Section of Molecular and Translational Medicine, Research Center in Health Sciences and Biomedicine (CICSaB), UASLP, SLP, México. [2]Laboratory of Immunology and Cell and Molecular Biology, Faculty of Chemical Sciences, UASLP, SLP, México. [3]Oncology Área, High Specialty Hospital, Ignacio Morones Prieto, SLP, México.

E-mail: danizr1494@gmail.com

Breast cancer is a significant global health problem and the leading cause of cancer-related deaths among Mexican women. It accounts for 28% of all cancers in this population. Molecular subtypes, such as triple-negative breast cancer (TNBC) and HER2-positive breast cancer, are considered more aggressive. These subtypes generally lead to a poorer prognosis and increased resistance to chemotherapy. Tumor-infiltrating CD8+ T lymphocytes are crucial for an effective anti-tumor response. These cells release perforins, granzymes, and cytokines, such as IFNγ, which induce cancer cell death. However, within the tumor microenvironment, CD8+ T lymphocytes can develop an "exhaustion phenotype" (Tex). This state is characterized by the expression of multiple inhibitory receptors and an altered transcriptional program. These receptors negatively regulate the functions of CD8+ T lymphocytes, reducing their ability to release granzymes and perforins, which ultimately diminishes their anti-tumor activity. The objective of this study is to identify prognostic biomarkers by bioinformatically analyzing the transcriptional profile of CD8Tex associated with breast cancer aggressiveness. Public repositories (TCGA and GEO) were searched for RNA-seq datasets from breast cancer patients, specifically from tumor tissue and peripheral venous blood. A differential expression analysis was performed using the DESeq2 algorithm in R Studio. Biological pathways and cellular processes were identified in the differentially expressed genes using the GSEA algorithm. Clustering and gene regulatory network analyses were conducted using Cytoscape. The following genes were identified as potential biomarkers: PD-1, CTLA4, PFN1, GZMB, NKG7, and GNLY. These genes are associated with aggressive breast cancer subtypes, such as triple-negative breast cancer (TNBC) and HER2-positive breast cancer (HER2+). The results of this study could generate new transcriptional profiles in CD8+ T lymphocytes associated with aggressiveness. These profiles could enable more accurate cancer classification and treatment, ultimately improving survival rates and quality of life for patients.