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# Genotoxicity of Mexican woodworkers: micronucleus frequency and nuclear abnormalities in buccal mucosa cells

# Genotoxicidad en carpinteros mexicanos: frecuencia de micronúcleos y anomalías nucleares en células de la mucosa bucal

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## ABSTRACT

Occupational exposure to wood dust is associated with increased health risks, including genetic damage. This study evaluated cytotoxicity, genotoxicity, and genomic instability in oral mucosal cells of Mexican carpenters. 75 oral mucosa samples were collected from male wood industry workers and 73 from unexposed individuals. For each participant, 2,000 exfoliated cells were analyzed using fluorescence microscopy to determine the frequency of micronucleated cells (MNc) and other biomarkers of cellular damage. The mean age was 38.1 years, and the average body mass index (BMI) was 27.7. Wood dust exposure in the selected workshops exceeded the recommended occupational limit by more than 25 times. Among exposed individuals, a significantly higher frequency of DNA damage (MNc, $p < 0.003$), cell death ($p < 0.001$; karyorrhexis [KR] $p = 0.006$, pyknosis [PK] $p = 0.0001$, karyolysis [KL] $p = 0.0001$), and cytokinesis defects (binucleated cells [BN], $p < 0.001$) was observed compared to the control group. Exposure to wood dust and solvents, combined with tobacco use and alcohol consumption, further increased the frequency of certain biomarkers ($p < 0.01$). Workers in the wood industry face an elevated occupational risk due to chronic exposure to wood dust and solvents, as evidenced by increases in several biomarkers. These findings underscore the need to strengthen biosafety measures and implement ongoing biomonitoring programs in high-risk industrial environments.

**KEY WORDS:** Wood workers, wood dust, occupational exposure, biomonitoring, cytotoxicity, genotoxicity, genomic instability, oral mucosa cells, micronucleus assay, nuclear abnormalities.

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## RESUMEN

La exposición ocupacional al polvo de madera está asociada con mayor riesgo para la salud, incluido el daño genético. Este estudio evaluó la citotoxicidad, genotoxicidad e inestabilidad genómica en células de mucosa oral de carpinteros mexicanos. Se recolectaron 75 muestras de mucosa oral de varones de la industria de la madera y 73 de individuos no expuestos. En cada participante se analizaron 2000 células exfoliadas mediante microscopía de fluorescencia para determinar la frecuencia de células micronucleadas (MNc) y otros biomarcadores de daño celular. La media de la edad fue de 38.1 años y la del índice de masa corporal (BMI) de 27.7. La exposición al polvo de madera en los talleres seleccionados superó 25 veces el límite ocupacional recomendado. En los expuestos se observó mayor frecuencia significativa en el daño al DNA (MNc, $p < 0.003$), muerte celular ($p < 0.001$; cariorexis [KR] $p = 0.006$, picnosis [PK] $p = 0.0001$, cariólisis [KL] $p = 0.0001$) y daño a la citocinesis (células binucleadas [BNc] $p < 0.001$), respecto al grupo control. La exposición de los polvos de madera y solventes en combinación con el uso de tabaco y consumo de alcohol incrementó aún más la frecuencia de algunos biomarcadores ($p < 0.01$). Los trabajadores de la industria de la madera enfrentan riesgo laboral elevado debido a la exposición crónica al polvo de madera y disolventes, como lo evidencian los incrementos en los diferentes biomarcadores. Estos hallazgos subrayan la necesidad de fortalecer las medidas de bioseguridad e implementar programas continuos de biomonitoreo en entornos industriales de alto riesgo.


**PALABRAS CLAVE:** Wood workers, wood dust, occupational exposure, biomonitoring, cytotoxicity, genotoxicity, genomic instability, oral mucosa cells, micronucleus assay, nuclear abnormalities.

## Introduction

Woodworking is an ancient activity that has developed various methods and tools across cultures and regions. Several urban centers in Mexico, such as Campeche, Quintana Roo, Tabasco, Yucatán, Mexico City, Michoacán, and Jalisco, are recognized for their wood processing and consumption hubs, which often emerge as small businesses and artisanal industries related to carpentry. Unfortunately, woodworkers are exposed to occupational hazards, including both acute and chronic exposure to molds, bacteria, fungicides, pesticides, wood dust, solvents, paints, lacquers, and both natural and synthetic substances (Guzmán-Silva *et al.,* 2018). Continuous exposure to these elements poses a significant risk for the development of eczema, urticaria, rhinoconjunctivitis, and extrinsic allergic alveolitis (Ramoneda-Paniagua & van der Haar, 2016). In

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several cases, occupational cancer may also occur, including adenocarcinoma of the nasal cavity and paranasal sinuses (Meng *et al.,* 2020), as well as cancers of the lung, gastrointestinal tract, and hematopoietic and lymphatic systems (Hodgkin's disease) (Gómez-Yepes, 2010; Scarabelli *et al.,* 2021).

The International Agency for Research on Cancer (IARC, 2012; 1995) classified wood dust as a carcinogen to humans (NIOSH, 2019). Similarly, the American Conference of Governmental Industrial Hygienists (ACGIH) has classified certain types of wood as suspected human carcinogens (Rojas-García & Peñalver-Paolini, 2015). The Threshold Limit Value-Time-Weighted Average (TLV-TWA) established by the National Institute for Occupational Safety and Health (NIOSH) for wood dust exposure is 1 mg/m$^3$ (NIOSH, 2019). Despite these classifications, many workplaces lack adequate safety measures, leaving workers at risk of genotoxicity and cytotoxicity (Huff, 2001). Therefore, prioritizing occupational risk assessment associated with exposure to hazardous residues is critical.

Some methods used to assess mutagenic and genotoxic risk tend to be costly, complex, invasive, or inaccessible. However, an easy-to-perform and non-invasive alternative evaluates the frequency of micronuclei (MN) and nuclear anomalies (NA) in the oral mucosa. MN are extranuclear genetic material formed during mitosis, primarily due to chromosome breakage or spindle apparatus damage, often resulting in cell death. Recent findings indicate that the genetic material contained within MN can undergo multiple rearrangements, including gains or losses of material (chromothripsis/chromoanagenesis), potentially leading to genetic chaos and genomic instability (Kwon *et al.,* 2020). MN are mislocalized DNA fragments in the cytoplasm and are recognized by the cyclic GMP-AMP synthase (cGAS)-stimulator of interferon genes (STING) (cGAS-STING) pathway, which triggers the innate immune response and chronic inflammation. This process has been linked to autoimmune diseases, other chronic degenerative conditions, and premature aging (Guo *et al.,* 2021; Kirsch-Volders *et al.,* 2020). MN thus serve as a hallmark of severe DNA damage and may initiate multiple complex genomic rearrangements, acting as indicators for various cancers, particularly the most aggressive types (Krupina *et al.,* 2021).

The MN assay is a widely accepted and cost-effective method for examining the morphology of superficial epithelial cells (Bonassi *et al.,* 2011; Romo-Huerta *et al.,* 2021). It has proven advantageous in the early detection of health risks caused by exposure to carcinogens and other harmful substances in occupational settings (Aguiar-Torres *et al*., 2019; Ceppi *et al.,* 2023; Pastor-Sierra *et al.,* 2023; Ursini *et al.,* 2019). This study employs the MN assay to monitor nuclear anomalies in oral mucosa cells, assess cytotoxic, genotoxic, and genome instability effects, and identify potential health risks associated with occupational exposure to carcinogens, particularly wood dust and solvents, in woodworkers.

## Material and Methods

### Study design and participant selection

An observational study was conducted using intentional non-probabilistic sampling.

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Participants were selected from the Guadalajara Metropolitan Area and the Ocotlán municipality, both located in the Jalisco state, Mexico.

### Sampling sites

Samples were collected from 16 workshops specializing in the manufacture of furniture made from softwood veneers. These workshops presented comparable conditions in terms of infrastructure, type of machinery used, and number of employees. Typical carpentry processes were carried out in all workshops, including milling, carving, shaping, sanding, and lacquer application.

### Exposed group

This group consisted of wood industry workers, including carpenters, lacquerers, and assistants, employed in the previously selected workshops. All participants were directly and continuously exposed to wood dust and vapors derived from lacquers and solvents generated during their daily tasks. Only males over 18 years of age were included, with at least six months of continuous employment in their current position, no work interruptions during the week prior to sample collection, and a minimum work schedule of eight hours per day, at least five days per week.

### Control group

The control group was composed of apparently healthy males with no reported history of substance abuse or occupational exposure to wood dust, chemicals from lacquers or solvents, or other industrial contaminants. Individuals with chronic diseases were also excluded. Participants were selected from offices and corporate environments located in the same geographic areas as the workshops to control for potential environmental confounders.

### Sociodemographic data collection

Prior to any procedure, all participants signed an informed consent form. Subsequently, oral mucosa samples were collected, and vital signs were recorded. A structured questionnaire was administered to identify potential risk factors, including demographic, anthropometric, and clinical information. Collected variables included: age, sex, weight, height, general health status, consumption of coffee, tobacco, medications, drugs, and alcohol, date of last dental visit, personal medical history, occupational exposure, lifestyle, and presence of acute or chronic illnesses. Participants who withdrew their consent during the process or whose biological samples were unsuitable for analysis due to technical limitations were excluded from the study.

### Assessment of wood dust production

Wood dust production was evaluated over a 10-hour workday (from 9:00 a.m. to 6:00 p.m.) in two carpentry workshops where workers agreed to participate in the study. Dust quantities were

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expressed in mg/m$^3$, using a predefined one-square-meter surface. Collected dust samples were weighed to determine the final value. This procedure was repeated over two consecutive days, and the average dust concentration was calculated for each session.

### Oral mucosa sample collection and processing

Before sample collection, participants were instructed to rinse their oral cavity with potable water. A gentle scraping of the inner cheek epithelium was then performed using a microscope slide. The collected samples were smeared onto pre-coded slides and allowed to air dry at room temperature. Once dried, the slides were fixed in 80 % ethanol for 48 hours and stained with acridine orange (CAS No. 10127-02-3; Sigma, USA), following the protocol described by Torres-Bugarín *et al.* (2014).

All samples were analyzed by a single observer who was blinded to the participants' group assignment. For each individual, 2,000 cells were evaluated using an epifluorescence microscope (Axiostar Plus, Carl Zeiss®) equipped with an immersion objective (100×) (APLAN Carl Zeiss®). Biomarkers of genotoxicity, cytotoxicity, and genomic instability were quantified and categorized into three main groups:

- *DNA damage and genomic instability* (DD): micronucleated cells (MNc) and lobulated nuclei (LN)

- *Cell death* (CD): condensed chromtin (CC), lobulated nuclei (LN), karyorrhexis (KR), and karyolysis (KL)

- *Cytokinesis damage* (CytD): binucleated cells (BNc)

Biomarker identification and scoring were conducted according to the criteria proposed by Tolbert *et al.* (1992) and Torres-Bugarín *et al.* (2014) (see Figure 1).

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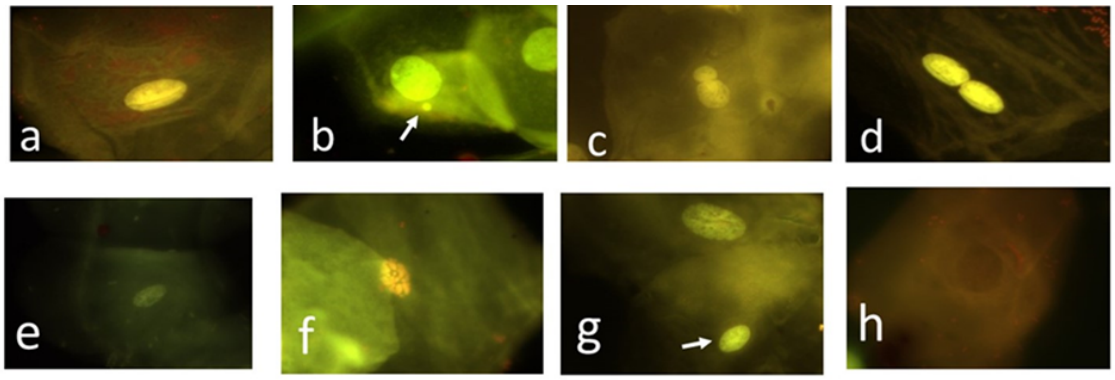


**Figure 1. Exfoliated buccal mucosa cells.**

a) Normal cells; b) Micronucleate cells (MNc), arrows show the micronucleus; c) Lobed nucleus (LN); d) Binucleated cell (BNc); e) Chromatin condensed cell (CC); f) Karyorrhexis (KR); g) Pyknosis (PK); and h) Karyolitic (KL) cell.

### Statistical analysis

Qualitative variables, such as educational level, daily income, occupational exposure time, and habits related to tobacco, alcohol, and diet, were described using absolute frequencies and percentages. Except for age, the distribution of quantitative variables did not meet the assumptions of normality according to the Shapiro-Wilk test ($p > 0.01$). Consequently, both Student's t-test and the Mann-Whitney U or Kruskal-Wallis tests were used to assess significant differences between groups for quantitative variables. To identify factors associated with increased frequency of MNc, odds ratios (OR) and 95 % confidence intervals were calculated. Fisher's exact test was used to determine statistical significance in comparisons involving categorical variables. Differences were considered statistically significant at $p < 0.05$. All statistical analyses were performed using GraphPad Prism, version 5.

## Results

### Biomarker evaluation: Comparison between control and exposed groups

### Wood dust exposure levels

Measurements taken in the selected carpentry workshops indicated that workers were exposed to a mean total dust concentration of 25.2 ± 12.14 mg/m$^3$, with values ranging from 7.8 to 35.1 mg/m$^3$, based on surface sample estimates. These levels far exceed the recommended occupational limit of 1 mg/m$^3$ (NIOSH, 2019).

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### Age and body mass index (BMI)

A total of 148 oral mucosa samples were analyzed, all from male participants, of which 75 belonged to the exposed group (carpentry workers) and 73 to the control group (no occupational wood exposure).

Statistical analyses showed no significant differences between groups in terms of age ($t = 0.26$) or BMI ($t = 0.92$), indicating that these variables were comparable between participants (table 1).

### Analysis of cytotoxicity and genotoxicity biomarkers

The analysis of the observed frequencies of cytotoxicity, genotoxicity, and genomic instability biomarkers grouped into three categories: DNA damage, cell death, and cytokinesis damage, yielded the following results when comparing the exposed and control groups (table 1):

In the cell death category, no significant global differences were observed between the two groups. Similarly, the LN frequency was comparable between the exposed and control groups. However, a significantly higher frequency of MNc was recorded in the exposed group ($p < 0.003$).

Regarding the overall frequency of cell death and cytokinesis damage, as well as the individual biomarkers comprising these categories, significantly higher frequencies were observed in the exposed group compared to controls. This trend was consistent across most biomarkers and exposure durations (see *p-values* in table 1).

Overall, the results indicate that while no significant differences were found in certain specific biomarkers, such as LN or in the global DNA damage frequency, the exposed group exhibited higher frequencies of genetic and cellular damage, particularly in MNc ($p < 0.003$), and in most biomarkers associated with cell death and cytokinesis damage. These differences were statistically significant compared to the control group. This pattern was observed across different exposure time ranges, suggesting a possible cumulative relationship between occupational exposure and cellular damage.

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## Table 1. Nuclear abnormalities and micronucleated cells in individuals exposed and non-exposed to wood dust and solvents

| Biomarkers | | No Exposed | ♦ *P* | *OR (IC 95%)* | Exposed | Years of exposure | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | **0.5 – 5** | 6 - 10 | 11 - 15 | > 16 |
| n (%) | | **73(100)** | NS | | 75(100) | 23(30.7) | 6(8.0) | 7(9.3) | 26(34.7) |
| Age | | 38.1 ±10.5 | t 0.26 | | 36.7 ± 13.1 | 31.3 ± 12.5 | 35.5 ± 9.8 | 36.0 ±12.3 | 43.4 ± .4 |
| BMI (Range) | | 27.4 ± 5.5 (20.1 - 39.2) | t 0.92 | | 26.9 ± 5.7 (19.8 – 41.1) | **11.5 ± 6.4**** | 16.5 ± 5.1 | 15.6 ±7.3 | 18.8 ± 9.1 |
| ***DNA damage* (DD)** | **MNc** | 1.2 ± 1.6 | **< 0.003** | 3.3 (1.5-1.7) 0.003 | 2.6 ± 2.6 | 2.3 ± 1.9 | 1.8 ±1.2 | 2.6 ± 2.1 | 2.9 ± 2.8 |
| | LN | 3.4 ± 3.2 | NS | 0.48 (0.3-0.9) 0.027 | 3.6 ± 3.3 | 3.2 ± 3.4 | 4.2 ± 4.3 | 3.9 ± 3.8 | 3.7 ± 3.3 |
| | DD | 4.6 ± 3.3 | NS | | 6.2 ± 4.6 | 5.5 ± 4.8 | 6.0 ± 5.0 | 6.4 ± 3.8 | 6.6 ± 4.3 |
| ***Cell death (CD)*** | **CC** | 2.8 ± 4.2 | **< 0.01** | 1.6 (0.8-3.0 0.24 | 4.2 ± 3.8 | 2.4 ± 2.5 | 3.8 ± 2.6 | 2.6 ± 1.7 | **5.3 ± 3.7*** |
| | KR | 0.8 ± 1.2 | **< 0.006** | 4.5 (1.5-1.4) 0.006 | 2.5 ± 2.9 | **0.9 ±1.1*** | 3.8 ± 2.6 | 2.9 ± 2.9 | 3.2 ± 3.3 |
| | PK | 0.6 ± 1.1 | **< 0.001** | 11 (3.6-32) 0.0001 | 3.8 ± 3.4 | 3.0 ± 2.3 | 1.7 ± 1.9* | 2.9 ± 2.5 | 4,2 ± 3.6 |
| | KL | 2.9 ± 2.6 | **< 0.001** | 4.9 (2.5-0.7) 0.0001 | 6.1 ± 4.9 | 5.1 ± 4.1 | 7.2 ± 4.4 | 7.3 ± 4.3 | 6.1 ± 5.9 |
| | CD | 7.1 ± 5.8 | **< 0.001** | | 16.6 ± 9.8 | 11.4 ± 6.4 | 16.5 ± 5.0 | 15.6 ± 7.3 | 18.8 ± 9.1 |
| ***Cyt damage*** | **BNc** | 2.6 ± 2.6 | **< 0.01** | 9.8 (4.7-20) <0.001 | 5.5 ± 3.2 | **2.4 ± 2.5*** | 6.0 ± 3.7 | 4.7 ± 4.2 | 5.3 ± 2.6 |

Mean ± SD; N- Sample size; BMI- Body mass index; NS- not significant; MNc- Micronucleated cells; LN- Lobed nucleus; DD- DNA damage; CC- Abnormally condensed chromatins; KR-Karyorrhexis**; PK**- Pyknosis; KL- Karyolysis; CD-Cell death; BNc- Binucleated cell. CytD-Cytokinesis damage; Kruskal-Wallis analysis [Statistical significance $p < 0.05$]. ).◄ "n" varies in each case because not all participants provided the information. ♦ U de Mann-Whitney, * Kruskal-Wallis (*$p < 0.05$; **$p < 0.01$).

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Impact of sociodemographic factors, lifestyle, and health status on cytotoxicity, genotoxicity, and genomic instability biomarkers in the exposed group (tables 1 and 2)

### Age and body mass index (BMI) in relation to occupational tenure

Exposed workers reported workdays ranging from 8 to 10 hours, six days per week. As shown in table 1, the average duration of employment in the wood industry was 10.9 ± 11.1 years, ranging from six months to 34 years.

Table 1 also presents the distribution of age and BMI across different ranges of occupational exposure time among woodworkers. No statistically significant differences were observed in these variables across exposure levels. However, one exception was noted in the subgroup with 0.5 to 5 years of exposure, which showed the lowest BMI among all subgroups (11.5 ± 6.4 *vs* 26.9 ± 5.7; $p < 0.01$), and even lower than the control group (27.4 ± 5.5). This may reflect an initial effect of working conditions on nutritional status, or it may be attributable to the fact that this subgroup is also the youngest, although no statistically significant differences in age were found.

### Cytotoxicity, genotoxicity, and genomic instability biomarkers according to occupational tenure

Biomarker analysis among exposed workers, stratified by duration of occupational exposure, did not show statistically significant differences in the overall frequencies of DNA damage, cell death, or cytokinesis damage categories.

However, significant differences were observed in specific individual biomarkers:

Workers with more than 16 years of exposure exhibited a significantly higher CC frequency (5.3 ± 3.7 *vs* 2.8 ± 4.2; *control*; $p < 0.05$).

The subgroup with 0.5 to 5 years of exposure showed the lowest KR frequency (0.9 ± 1.1 *vs* 2.5 ± 2.9 global).

The 6 to 10-year exposure subgroup recorded the lowest PK frequency.

These findings suggest that prolonged exposure to the carpentry work environment may induce subtle, biomarker-specific cellular alterations, particularly those related to cell death processes. While no global differences were observed in the primary biomarker categories, the variation in individual biomarkers supports the need for longitudinal studies to evaluate the progression of cellular damage over time.

*Age:* Younger workers (18-25 years) showed lower frequencies of CC ($p < 0.05$) and KR ($p < 0.05$) compared to older age groups, suggesting reduced cellular damage in early stages of occupational exposure.

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*Body mass index (BMI):* No statistically significant global differences in biomarker frequencies were found across BMI ranges. However, the subgroup with a BMI > 35 exhibited higher frequencies of overall DNA damage and, specifically in LN (7.5 ± 6.1 *vs* global 3.6 *vs* 3.3; $p < 0.01$), as well as higher global cell death (21.5 ± 13.3 *vs* global 16.6 ± 8.2). Notably, two of the four biomarkers within this category— (KR 6.8 ± 6.7 *vs* global 2.5 ± 2.9; $p < 0.01$) and KL 7.8 ± 5.7 *vs* global 4.8 ± 3.7; $p < 0.01$), also showed elevated frequencies. Overall, there was a general trend toward increased biomarker levels with rising BMI, with some differences reaching marginal ($p < 0.05$) or strong ($p < 0.01$) statistical significance. These patterns suggest that severe overweight may contribute to the accumulation of cellular damage.

*BMI/Waist-to-hip ratio (WHR):* Men with a WHR between 0.96 and 0.99 showed elevated trends across several biomarkers, including the highest frequency of MNc among subgroups (3.4 ± 1.7), indicating increased genotoxic damage. Higher averages were also observed for KL (7.0 ± 4.9), CC (4.2 ± 1.9), the general average and BNc (7.0 ± 4.9), suggesting enhanced cytotoxicity.

Conversely, the subgroup with a WHR > 1.0 displayed generally lower values, especially in MNc, which may seem contradictory. This could be due to the small sample size (n = 7) or uncontrolled differences in other factors such as age, diet, or exposure levels. The WHR < 0.95 subgroup exhibited moderate but consistent values across most biomarkers.

In male carpenters, an intermediate WHR (0.96-0.99) was associated with a higher frequency of several nuclear biomarkers, suggesting a potential link between body fat distribution and cellular damage. Although WHR values > 1.0 are traditionally associated with greater metabolic risk, this was not reflected in increased nuclear damage in the present study, possibly due to the small sample size. These findings indicate that WHR may act as a modifying factor in occupationally induced cellular damage. However, its interpretation should be approached with caution and considered alongside other factors such as BMI, age, diet, and toxic habits.

*Pathologies*: A total of 14 carpenters (18.7 %) reported having at least one chronic condition, including obesity, diabetes mellitus, hypertension, bipolar disorder, or rheumatoid arthritis. Although overall analysis revealed no statistically significant differences in biomarker frequencies between participants with and without chronic conditions, some individual cases showed exceptionally high values, suggesting increased biological vulnerability to occupational and environmental exposure.

Particularly noteworthy is the case of a lacquer worker with overweight and rheumatoid arthritis, who exhibited the highest frequency of genomic instability among all participants (16 micronucleated cells per 2000 cells analyzed, compared to the group average of 2.6 ± 2.6 in the exposed population). Similarly, the highest level of cell death was observed in another lacquer worker diagnosed with overweight and depression, reaching 50 cells per 2000 with signs of death, a figure considerably higher than the overall average in the exposed group (16.6 ± 8.2). The greatest number of binucleated cells (17/2000) was recorded in a carpenter with a 10-year history of diabetes mellitus, also significantly above the group average (3.9 ± 2.4).

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While these are isolated cases, the findings support the hypothesis that certain physiopathological conditions may heighten susceptibility to cellular damage caused by occupational and environmental exposures, possibly through mechanisms involving oxidative stress, chronic systemic inflammation, or immunometabolic dysfunction (Sienes-Bailo *et al*., 2020). Therefore, the concept of individual vulnerability is especially relevant in settings of prolonged occupational exposure, underscoring the importance of tailored surveillance strategies for workers with clinical histories that may elevate their risk of genotoxic or cytotoxic damage.

*Tobacco use:* Regarding smoking habits, the group of carpenters was divided into three subgroups: non-smokers (27/73; 33.3 %), passive smokers (22/75; 26.2 %), and smokers (24/73; 32.0%). Statistical analysis of biomarker frequencies revealed no significant differences between groups, except for the smokers, who exhibited a significantly higher frequency of cell death ($p < 0.01$). Passive smokers also showed elevated levels of KR and KL, both markers of cell death (table 2).

*Alcohol consumption: Seventy-five percent of the* woodworkers *consumed alcohol (mainly beer), which was associated with a higher frequency of MNc ($p < 0.05$) compared to non-drinkers, indicating DNA damage and genomic instability ($p < 0.01$). They also showed an increased frequency of cell death ($p < 0.01$).*

Alcohol users were classified into low, moderate, and high consumption subgroups. The results revealed no substantial differences among these levels of intake; however, the moderate consumption group showed the highest MNc frequency (4.1 ± 2.2 *vs* 1.8 ± 1.6 in non-drinkers; $p < 0.01$) and DNA damage (9.4 ± 5.1 *vs* 4.2 ± 2.6 in non-drinkers; $p < 0.01$).

*Drug use:* Analysis of the questionnaires revealed that 6 % of carpenters reported recreational drug use. Among this group, the frequency of CC was higher compared to non-users. No statistically significant differences were found for the remaining biomarkers.

*Consumption of fruits, vegetables, red meat, processed meats, vitamins, and education level:* No significant associations were observed between these variables and biomarker frequencies; therefore, they were not included in the tables. In terms of education, 38 % of the participants had completed only primary school, and 43 % had completed secondary education.

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## Table 2. Biological and lifestyle factors, health status, and frequency of nuclear abnormalities in the woodworkers.

| Risk and protective factors *n (%)* | | | ***DNA damage* (DD)** | | | ***Cell death* (CD)** | | | | | ***Cy-D*** |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | **MNc** | **LN** | **DD** | **CC** | **KR** | **PK** | **KL** | **CD** | **BN** |
| **Control** *73 (100)* | | | 1.2 ± 1.6 | 3.4 ± 3.2 | 4.6 ± 3.3 | 2.8 ± 4.2 | 0.8 ± 1.2 | 0.6 ± 1.1 | 2.9 ± 2.6 | 7.1 ± 5.8 | 2.8 ± 4.2 |
| Woodworkers **Total** | -- | *36.7* ± 13.1 *73 (100)* | 2.6 ±2.6 | 3.6 ±3.3 | 6.2 ±4.6 | 4.2 ±3.8 | 2.5 ±2.9 | 3.8 ±3.4 | 0.8 ± 1.2 | 16.6 ±8.2 | 3.9 ±2.4 |
| Age-years *75(100)* | 18- 25 | 19.9 ± 2.7 *14(18.5)* | 1.8 ±1.4 | 2.3 ±1.3 | 4.1 ±2.0 | **1.6 ±2.5*** | **0.8 ±1.1*** | 2.8 ±2.7 | 0.6 ± 1.1 | 11.2 ±7.0 | 4.9 ±3.1 |
| | 26-35 | 30.5 ± 3.4 *17 (22.7)* | 2.1 ±1.7 | 3.6 ±4.3 | 5.6 ±4.7 | 4.9 ±3.5 | 3.1 ±3.9 | 3.4 ± 3.2 | 2.9 ± 2.6 | 17.6 ± 10.6 | 6.2 ± 3.2 |
| | 36-44 | 40.1 ± 3.1 *17 (22.7)* | 3.3 ±2.4 | 4.5 ±4.1 | 7.8 ±5.8 | 5.5 ±4.0 | 3.8 ±2.5 | 4.7 ±4.4 | 7.1 ± 5.8 | **19.7 ±10.2*** | 5.4 ±3.0 |
| | > 45 | 49.4 ± 3.9 *23 (30.7)* | 2.7 ±2.8 | 3.5 ±2.5 | 6.1 ±3.8 | 3.9 ±3.1 | 1.9 ± 2.3 | 4.1 ±3.5 | 6.7 ±5.6 | 16.6 ±8.7 | 5.6 ±3.6 |
| **BMI** 27.4± 4.4 *56 (100)* | 18.5-24.9 | 23.1±1.4 *17 (30.4)* | 2.8 ± 2.3 | 3.5 ± 3.3 | 6.2 ± 4.9 | 4.9 ± 3.4 | 2.2 ± 1.9 | 3.4 ± 3.2 | 4.8 ± 3.7 | 15.2 ± 6.4 | 6.9 ± 4.0 |
| | 25.0-29.9 | 27.0± 1.5 *26 (46.4)* | 2.7 ± 1.5 | 3.8 ± 3.47 | 6.2 ± 4.9 | 4.5 ± 3.9 | 1.9 ± 2.1 | 4.3 ± 4.3 | 6.5 ± 5.4 | 17.2 ± 9.8 | 5.3 ± 2.7 |
| | 30.0-34.9 | 32.0± 1.9 *9 (16.1)* | 2.9 ± 2,5 | 3.2 ± 3.1 | 6.1 ± 3.8 | 4.8 ± 3.7 | 3.3 ± 2.8 | 4.7 ± 2.6 | 6.7 ± 5.1 | 19.4 ± 10.1 | 5.4 ± 2.6 |
| | >35.0 | 35.6 ± 0.7 *4 (7.1)* | 3.0 ± 1.4 | **7.5 ± 6.1**** | **10.5 ± 5.5*** | 3.8 ± 1.9 | **6.8 ± 6.7*** | 3.3 ± 1.5 | **7.8 ± 5.7*** | **21.5* ± 13.3** | 6.3 ± 4.9 |
| **WHR** 0.9 ± 0.1 | 40 (100) | < 0.95 28 (70) | 2.5 ± 2.3 | 4.3 ± 4.1 | 6.8 ± 5.4 | 3.4 ± 2.8 | 3.2 ± 3.5 | 3.4 ± 2.7 | 5.8 ± 3.2 | 15.8 ± 9.0 | 5.8 ± 3.2 |
| | | 0.96- 0.99 5 (12.5) | **3.4 ± 1.7*** | 4.6 ± 5.1 | **8.0 ± 4.3*** | 4.2 ± 1.9 | 1.4 ± 1.1 | 3.0 ± 2.7 | 7.0 ± 4.9 | 15.6 ± 7.3 | 7.0 ± 4.9 |
| | | >1 7(17.5) | 1.3 ± 1.1 | 3.3 ± 3.1 | 4.6 ± 2.8 | 3.7 ± 1.9 | 2.1 ± 2.9 | 4.1 ± 2.5 | 5.4 ± 1.8 | 15.3 ± 8.8 | 5.4 ± 1.8 |
| **Pathology** *75 (100)* | Without | *53 (76.7)* | 2.3 ± 2.1 | 3.1 ± 3.1 | 5.4 ± 3.8 | 4.1 ± 3.7 | **2.6 ± 2.9*** | 3.4 ± 3.5 | 5.9 ± 4.8 | 15.1 ± 9.8 | 5.1 ± 2.9 |
| | **With** | 14 (18.7) | 3.4 ± 2.6 | 5.2 ± 3.6 | **8.6 ± 5.6*** | 3.5 ± 2.9 | 1.4 ± 1.6 | 4.9 ± 3.2 | 7.5 ± 5.3 | 17.3 ± 6.3 | 6.4 ± 4.5 |
| **Tobacco** consumption 73 (100) | **No** | *27 (33.3)* | 2.1 ± 1.5 | 3.0 ± 3.0 | 5.1 ± 3.7 | 2.7 ± 3.2 | 2.0 ± 2.3 | 2.4 ± 2.3 | 6.1 ± 4.8 | 13.2 ± 8.6 | 4.6 ± 2.7 |
| | **Passive** | *22 (26.2)* | 5.7 ± 2.8 | 5.3 ± 3.6 | **2.3 ± 2.5*** | 4.1 ± 3.2 | **16.3 ± 6.8*** | 4.6 ± 3.0 | **16.0 ± 3.2**** | 16.0 ± 6.8 | 4.6 ± 3.0 |
| | **Smoker** | *24 (32.0)* | 2.4 ± 2.6 | 4.0 ± 3.7 | 6.3 ± 4.4 | 4.6 ± 3.4 | 2.6 ± 3.4 | 4.6 ± 4.3 | 7.5 ± 5.8 | **19.3 ±10.5**** | 6.1 ± 3.9 |

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**Table 2. Biological and lifestyle factors, health status, and frequency of nuclear abnormalities in the woodworkers.**

| Risk and protective factors *n (%)* | | | ***DNA damage* (DD)** | | | ***Cell death* (CD)** | | | | | ***Cy-D*** |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | **MNc** | **LN** | **DD** | **CC** | **KR** | **PK** | **KL** | **CD** | **BN** |
| **Control** *73 (100)* | | | 1.2 ± 1.6 | 3.4 ± 3.2 | 4.6 ± 3.3 | 2.8 ± 4.2 | 0.8 ± 1.2 | 0.6 ± 1.1 | 2.9 ± 2.6 | 7.1 ± 5.8 | 2.8 ± 4.2 |
| **Alcohol** 67(100) | **No** | *17 (25.0)* | 1.8 ± 1.6 | 2.5 ± 1.7 | 4.2 ± 2.6 | 1.9 ± 1.2 | 1.2 ± 1.5 | 2.4 ± 2.6 | 5.0 ± 4.0 | 10.5 ± 6.4 | 4.7 ± 2.4 |
| | **Yes** | *50 (75.0)* | **2.8 ± 1.6*** | 4.5 ± 4.4 | **7.3 ± 4.7**** | 4.2 ± 3.1 | **3.4 ± 3.9*** | 3.4 ± 2.6 | 6.4 ± 5.9 | **17.2 ± 10.2**** | 5.5 ± 3.1 |
| **[ml/week]◄** 50 (100) | ***Under*** *20 (36.8)* | *355 - 710 ml* | 2.3 ± 1.8 | 3.2 ± 2.7 | 5.5 ± 3.0 | 4.7 ± 3.8 | 2.7 ± 2.7 | 4.2 ± 3.0 | 5.3 ± 4.0 | 16.9 ± 8.0 | 5.9 ± 5.1 |
| | ***Medium*** *11 (22.8)* | 1065 – 1420 ml | **4.1 ± 2.2*** | 5.3 ± 3.8 | **9.4 ± 5.1*** | 2.8 ± 2.5 | 2.6 ± 1.9 | 3.8 ± 2.5 | 6.4 ± 2.8 | 15.6 ± 5.7 | 6.0 ± 3.4 |
| | ***High*** *19 (40.4)* | >1421 ml | 2.3 ± 2.6 | 3.7 ± 3.8 | 6.0 ± 4.8 | 4.8 ± 4.1 | 2.5 ± 3.5 | 3.9 ± 4.4 | 5.5 ± 3.0 | 16.7 ± 11.2 | 7.0 ± 5.7 |
| **Drugs** 75(100) | **No** | *70 (94)* | 2.4 ± 2.2 | 3.5 ± 3.1 | 5.9 ± 4.2 | 3.3 ± 3.4 | 2.2 ± 2.6 | 3.4 ± 3.3 | 5.4 ± 3.0 | 14.3 ± 8.9 | 5.8 ± 4.8 |
| | **Yes** | *5 (6)* | 1.8 ± 0.8 | 3.0 ± 2.5 | 4.8 ± 2.3 | **6.2 ± 2.2*** | 1.8 ± 1.5 | 3.8 ± 1.3 | 7.6 ± 5.7 | 19.4 ± 4.7 | 5.6 ± 4.0 |

(%)- percentage of individuals; DD-DNA damage; CD-Cell death; Cy-D- Cytokinesis damage; MNc- Micronucleated cells; LN- Lobed nucleus; BNc- Binucleated cell; CC- Abnormally condensed chromatins; KR- Karyorrhexis; PK- Pyknosis; KL- Karyolysis; IMC- BMI- Body mass index; WHR- waist/hip ratio. Kruskal-Wallis assay (*$p < 0.05$ **$p < 0.01$) ◄ "n" varies in each case because not all participants provided the information. Alcohol consumption per week, expressed in ml.

## Discussion

### Genotoxic and cytotoxic damage associated with occupational exposure in carpenters

The results of this study demonstrate a clear association between chronic occupational exposure to wood dust and solvents—common in carpentry workshops—and the presence of cytogenetic and cytotoxic damage in buccal mucosa cells. Through the assessment of nuclear biomarkers (DNA damage—MNc and NL; Cell death—CC, PN, KR, KL; and Cytokinesis failure—BNc), statistically significant differences were observed between exposed workers and controls.

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These findings suggest that, even under seemingly normal working conditions, cellular processes such as genomic instability, cell death, and cytokinesis impairment may occur.

These results are consistent with prior scientific literature that classifies wood dust as a Group 1 carcinogen, according to the IARC (1995: 2012). Studies conducted in Turkey, India, Brazil, Switzerland, and Australia (table 3) have reported a significant increase in nuclear alterations in exposed carpenters, particularly MNc, KL, KR, and BNc, findings similar to those observed in this Mexican sample. For instance, Celik and Kanik (2006) and Rekhadevi *et al.* (2009) reported notable elevations in MNc among workers exposed to wood dust, regardless of smoking habits, age, or education level. In this study, the frequency of MNc reached 2.5 ± 2.4 in the exposed group versus 1.3 ± 1.6 in controls, which represents a sustained biological risk pattern (Celik & Kanik, 2006; Rekhadevi *et al.,* 2009).

These findings align with previous reports indicating that wood processing, even without lacquering, can have adverse health effects (Acheson *et al.,* 1968; Alonso-Sardón *et al.,* 2015; Edwards *et al.,* 2021; Gómez-Yepes, 2010; Meng *et al.,* 2020; Ramoneda- Paniagua & van der Haar, 2016; Rojas-García & Peñalver-Paolini, 2015; Scarabelli *et al.,* 2021). In the Guadalajara Metropolitan Area, numerous small family-owned businesses, carpentry workshops, and furniture makers employ between 3 to 15 workers. While these enterprises provide essential employment and family support, their working conditions are often suboptimal, mainly due to confined spaces, poor ventilation, and limited or neglected safety measures. A notable example is the dust concentration found in one workshop where measurements were taken, which was 25 times higher than the recommended level (NIOSH, 2019). These conditions clearly pose a significant health risk to workers and highlight the urgent need for effective, low-cost, and continuous monitoring methods to detect potential cellular damage under such occupational settings.

Genotoxic damage from exposure to wood dust and its additives has previously been evaluated using the Ames test, comet assay, chromosomal aberrations, and the frequency of MN in lymphocytes and exfoliated buccal and nasal cells (Bruschweiler *et al.,* 2014; Çelik & Kanık, 2006; Coronas *et al.,* 2016; Elavarasi *et al.,* 2002; Rekhadevi *et al.,* 2009; Wultsch *et al.,* 2015). Notably, Elavarasi *et al.* (2002) and Rekhadevi *et al.* (2009) found a positive correlation between years of wood dust exposure and MNc frequency. Similar to the present study, both authors reported that genotoxic and cytotoxic damage was more significant in the exposed group than in the control group (Odds ratio [OR] ≥ 2), regardless of comorbidities, age, education, smoking, alcohol use, drug use, or dietary habits (tables 1, 2, and 3). Carpenter-lacquerers showed a higher frequency of MNc, cell death, and cytokinesis failure, with certain biomarkers being more evident in individuals with > 16 years of exposure. This may be linked to apoptosis, a process by which genetically damaged buccal epithelial cells are eliminated (Tolbert *et al.,* 1992). Therefore, this study supports the notion of elevated risk in individuals occupationally exposed to wood dust and solvents, aligning with the findings reported by Bruschweiler (2014) in Swiss woodworkers (table 3).

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**Table 3. Nuclear abnormalities in buccal mucosa cells in different research works.**

| Country | Groups | n | DNA damage (DD) | | Cell death (CD) | | | | Cyt-D | Cells/ Staining | Ref |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | MNc | NL | CC | KR | PK | KL | BN | | |
| México | *Control* | 73 | 1.3 ±1.6 | 3.4 ± 3.2 | 2.8 ±4.2 | 0.8 ±1.2 | 0.6 ±1.1 | 2.9 ±2.6 | 2.6 ±2.6 | 2000/ Acridine Orange | This work |
| | *Exposed* | 91 | 2.5 ±2.4 | 3.4 ± 3.1 | 3.6 ±3.6 | 2.4 ±2.7 | 3.5 ±3.3 | 5.9 ±4.7 | 5.6 ±3.2 | | |
| | *No Tabacco* | 28 | 2.4 ± 1.4 | 2.8 ±2.1 | 2.6 ±3.2 | 2.1 ±2.4 | 2.4 ±2.2 | 5.9 ±4.5 | 5.2 ±3.0 | | |
| | *Passive* | 22 | 2.2 ± 3.4 | 3.3 ± 3.4 | 2.9 ±2.4 | 2.1 ±2.1 | 3.2 ±2.6 | 4.7 ±3.5 | 5.0 ±3.1 | | |
| | *Tobacco* | 34 | 2.2 ± 3.3 | 3.9 ± 3.4 | 4.8 ±3.6 | 2.7 ±3.4 | 4.4 ±4.0 | 6.7 ±5.8 | 6.0 ±3.5 | | |
| Brazil | *Control* | 19 | 0.2 ± 0.1 | 0 | 2.3 ±0.5 | 14.6 ±2.6 | 1.8 ±0.5 | 4.2 ±0.9 | 2.0 ±0.2 | 1000/ Feulgen | (Coronas *et al*., 2016) |
| | Exposed | 38 | 0.1 ± 0.1 | 0.1 ±0.03 | 3.7±0.4 | 19.0 ±1.9 | 2.4 ±0.4 | 5.9 ±0.9 | 3.4 ±3.0 | | |
| Australia | *Control* | 65 | 0.6 ± 0.2 | 1.5 ±0.6 | ND | 5.3 ±1.5 | ND | 7.5 ±2.0 | 7.8 ±0.2 | 2000/ Schiff's | (Wultsch *et al*., 2015) |
| | *Exposed* | 51 | 0.7 ± 0.3 | 2.1 ±0.6 | ND | 8.5 ±1.5 | ND | 10.9 ±1.0 | 10.3 ±2.5 | | |
| Switzerland | *Control* | 19 | 1.6 ±0.8 | 0.6 ±0.1 | 53.0±3.1 | 92.0 ±5.0 | 22.0 ±2.0 | 123.0 ±7.5 | 7.0 ±0.5 | 2000/ Feulgen | (Bruschweiler *et al*., 2014) |
| | *Exposed* | 31 | 2.8±1.5 | 1.1 ±0.1 | 43.0±2.6 | 1860 ±9.8 | 36.0 ±3.0 | 259.0 ±14.5 | 4.2 ±0.2 | | |

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**Table 3. Nuclear abnormalities in buccal mucosa cells in different research works.**

| Country | Groups | n | DNA damage (DD) | | Cell death (CD) | | | | Cyt-D | Cells/ Staining | Ref |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | MNc | NL | CC | KR | PK | KL | BN | | |
| India | Control | 60 | 0.4 ± 0.1 | ND | ND | ND | ND | ND | ND | 1000/ DAPI | (Elavarasi *et al*., 2002; Rekhadevi *et al*., 2009) |
| | Exposed | | | | | | | | | | |
| | No Tobacco | 36 | 0.4 ± 0.1 | ND | ND | ND | ND | ND | ND | | |
| | Tobacco | 24 | 0.5 ± 0.2 | ND | ND | ND | ND | ND | ND | | |
| Turkey | Control | 10 | 0.9 ± 0.6 | 0.3 ±0.1 | N | 1.1 ±0.3 | ND | 1.07 ±0.3 | 0.7 ±0.3 | 1000/ Feulgen | (Celik &Kanik, 2006) |
| | Exposed | | | | | | | | | | |
| | NoTobacco | 10 | 5.5 ± 1.1 | 5.1 ±1.7 | ND | 8.7 ±2.0 | ND | 7.6 ±1.7 | 9.4 ±1.9 | | |
| | Tobacco | 10 | 7.8 ± 1.3 | 5.9 ±1.8 | ND | 9.9 ±2.1 | ND | 8.1 ±1.4 | 12.0 ±1.4 | | |

Mean ± SD; N- Sample size; DD-DNA damage; CD-Cell death; Cyt-D- Cytokinesis damage; MNc- Micronucleated cells; NL- Lobed nucleus; BNc- Binucleated cell; CC- condensed chromatins; KR- Karyorrhexis; PK- Pyknosis; KL- Karyolysis.

## Individual factors and susceptibility: A key dimension of risk

Although overall group comparisons revealed significant differences, the data also indicate that cellular damage is not uniformly distributed among workers. Individual factors such as age, BMI, body fat distribution (WHR), comorbidities, and lifestyle habits like tobacco and alcohol consumption modulated the extent of nuclear damage. An intermediate waist-to-hip ratio (0.96-0.99) was associated with higher frequencies of MNc, KL, and BNc, possibly due to a chronic inflammatory profile linked to visceral fat, a known source of oxidative stress and DNA damage (De Tursi-Ríspoli *et al.,* 2013). Additionally, workers with chronic illnesses such as rheumatoid arthritis or depression exhibited atypically high frequencies of MNc (up to 16/2000 cells) and cell death (up to 50/2000 cells), suggesting increased biological vulnerability potentially mediated by immune or DNA repair mechanisms (Torres-Bugarín *et al.,* 2024).

These data are relevant as they point to the need for stricter, individualized surveillance measures even within the same occupational category. Age also appears to influence outcomes:

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younger workers showed lower frequencies of KR and CD, supporting the hypothesis of cumulative exposure effects on cellular deterioration.

### Tobacco, alcohol, and synergistic effects

Although not all differences reached statistical significance, subgroup analyses revealed a trend toward increased cell death biomarkers (CC, KR, and KL) in individuals with active or passive tobacco exposure. These results are consistent with findings from India (Rekhadevi *et al.,* 2009) and Turkey (Celik & Kanık, 2006), where a progressive increase in these biomarkers was observed, although without statistical significance. These observations suggest that interactions between environmental contaminants and personal habits may exert synergistic effects, exacerbating cellular damage and potentially increasing long-term cancer risk.

### Poor working conditions and expanded environmental risk

Beyond the demonstrated biological risk, the working conditions observed in the participating carpentry workshops reveal a potentially hazardous health environment. Inadequate ventilation, lack of personal protective equipment, and workdays of up to 10 hours are common in small family businesses across the Guadalajara Metropolitan Area. In one workshop where particle concentration was measured, the dust load was found to be 25 times above recommended levels, emphasizing the severity of exposure.

This issue is not exclusive to Mexico. In Europe, approximately 3.5 million people work in wood-related activities, and their risk of respiratory and oncological diseases is estimated to be up to 900 times higher than the general population (Bruschweiler *et al.,* 2014; Llorente *et al.,* 2009). As Acheson *et al.* (1968) demonstrated, workers in this industry are at greater risk of nasal adenocarcinoma, and other studies have shown a significant increase in lung cancer risk with exposure levels above 5 mg/m$^3$ (Demers *et al.,* 1995; Wultsch *et al.,* 2015).

Moreover, the environmental risk extends to nearby communities: the dispersion of dust and toxic vapors into surrounding areas may affect vulnerable populations such as children, nearby residents, and unprotected workers, thereby posing a broader public health concern.

### Value of the buccal cytome assay as a monitoring tool

A key methodological contribution of this study is the validation of the buccal cytome assay as an accessible, non-invasive, and cost-effective tool for occupational surveillance. Its ability to detect early genetic and cellular damage in exposed populations is particularly valuable in resource-limited settings such as small artisan workshops. Furthermore, it allows for assessment of both cumulative exposure and the impact of individual variables such as comorbidities, habits, and age. The combined use of genotoxic (MNc, NB) and cytotoxic (KR, KL, CD, and BNc) biomarkers provides a more comprehensive view of cellular impact. Including these variables in occupational monitoring systems could enhance predictive capacity for early interventions and inform decision-making in occupational health.

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### Implications for health policy and preventive training

The data obtained from this Mexican population reflect a global phenomenon and highlight the urgent need to implement differentiated surveillance protocols, technical training, and biosafety measures. The evidence supports the implementation of mandatory practical courses on the safe handling of materials, the installation of proper ventilation systems, and regular biological monitoring.

Active surveillance should include biomarker evaluation and the identification of workers at greater risk due to advanced age, obesity, chronic illness, or combined exposure to tobacco and alcohol. Incorporating these variables would allow a shift from reactive strategies to proactive ones, focused on preventing cumulative genetic damage in vulnerable labor sectors.

Therefore, the results from this study focused on woodworkers in Jalisco, Mexico, are consistent with global trends. Regardless of workplace conditions, chronic exposure to wood dust and processing chemicals may significantly affect the genetic integrity of workers (tables 1, 2, and 3). Risk evaluation should not rely solely on genotoxic biomarkers (MNc and NB), but also consider the evolution of cytotoxic markers such as CC, KL, KR, and BNc for a more complete picture. These findings suggest that the buccal epithelium of lacquering carpenters could serve as a critical tissue for identifying health risks. Nonetheless, practical information is urgently needed to explain the importance of knowledge and implementation of basic safety measures. Continuous monitoring and community engagement are essential to improve health outcomes in these high-risk groups.

## Conclusions

The findings of this study demonstrate a significant increase in the frequency of biomarkers of cytotoxicity, genotoxicity, and genomic instability in wood industry workers chronically exposed to wood dust and solvent vapors. Notably, marked increases were observed in MNc, KR, KL, and BNc, indicating cumulative cellular damage associated with occupational exposure.

Additionally, individual factors that may modulate the response to environmental damage were identified, including age, body mass index, waist-to-hip ratio, presence of chronic comorbidities, and tobacco and alcohol consumption habits. These elements reinforce the need for an integrative surveillance approach that considers both external exposure and individual biological susceptibility.

In this regard, the use of the buccal cytome assay is consolidated as an effective, non-invasive, painless, simple, and low-cost tool for the early detection of cellular alterations in high-risk occupational settings.

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## Author contributions

Work conceptualization: TBO, CFM, and RIM; Methodology development: TBO, CFM, and RIM; Software management: TBO, AGME, MNL; Experimental validation: TBO, AGME; Data analysis: TBO, CFM, RIM; Data management: MNL, AGME; Writing and manuscript preparation: TBO, RIM, AGME; Sample collection: RIM, MSL, GJF, VA; sample analysis: CFM.

All authors have read and approved the published version of the manuscript.

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## Ethical declarations

Institutional Review Board Statement: This study was approved by the research committee of the Universidad Autónoma de Guadalajara and registered under number TTS-05-333-1326-17-089. The study complied with the Mexican Research Regulations and the Declaration of Helsinki.

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## Conflict of Interest

The authors declare no conflict of interest.

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