ORIENTAL JOURNAL OF CHEMISTRY
An International Open Access, Peer Reviewed Research Journal
www.orientjchem.org
ISSN: 0970-020 X
CODEN: OJCHEG
2025, Vol. 41, No.(3):
Pg. 771-782

Early DNA Damage in Buccal Cell Induced by Mixed Chemical Exposure Among Paint Manufacturing Workers

NURUL AFIQAH MD SAIRONI[1]*, NORMAH AWANG[1]*, FARAH WAHIDA IBRAHIM[1], ANUAR ITHNIN[2] and FARAHANA KAMALUDDIN[1]

[1]Centre of Toxicology and Health Risk Studies (CORE), University of Kebangsaan, Malaysia (UKM), 50300, Kuala Lumpur, Malaysia.
[2]Centre of Community and Health Studies (ReaCH), University of Kebangsaan Malaysia (UKM), 50300, Kuala Lumpur, Malaysia.
*Corresponding author E-mail: afiqah96.saironi@gmail.com, norm@ukm.edu.my

http://dx.doi.org/10.13005/ojc/410309

(Received: March 18, 2025; Accepted: May 01, 2025)

ABSTRACT

Paint manufacturing exposes workers to hazardous chemicals, increasing health risks. This study assessed DNA damage in buccal cells among workers. Forty workers were categorized into an exposed group (19 manufacturing workers) and a control group (21 office workers). A WHO standard questionnaire collected demographic and occupational data, including training on occupational hazards, smoking habits, and awareness of job-specific risks. Buccal cell samples were taken in the early morning (three per worker) to measure DNA damage using the comet assay, a method based on electrophoresis that evaluates DNA strand breaks by measuring tail length, %DNA in the tail, and tail moment. In the exposed group, 63% of workers had received training on occupational hazards, 30% were smokers, and 30% were aware of job-specific risks. The exposed group showed a marginally significant increase in tail moment length and %DNA in the tail ($P< 0.05$), indicating greater DNA damage compared to the control group. Prolonged chemical exposure in paint manufacturing is linked to increased DNA damage. Strengthening safety measures, improving hazard awareness, and implementing regular health monitoring are essential to reducing occupational risks.

Keywords: Occupational health risk, DNA damage, Paint manufacturing workers, Health monitoring in paint industry.

INTRODUCTION

Paints are widely used across various industries and have diversified over time, with each formulation tailored to a specific function. These materials serve to decorate, protect, and extend the durability of natural and synthetic surfaces by providing a barrier against environmental stressors. Application methods include brushing, rolling, dipping, flowing, conventional air spraying, disc spraying, and powder coating.[60] The formulations typically consist of complex mixtures of solvents and chemical compounds, each contributing to their overall performance. These including pigments, binders, extenders, and additives, with solvents acting as carriers for other components.[3]

Paint formulations typically consist of 10% thinner paints, 45% high-solvent paints (substance more volatile), and 45% low-solvent paints (substance less volatile).[31] Common solvents used in paint manufacturing include hydrocarbon aromatic solvents (e.g., toluene, xylene, ketones, alcohols)[55], solvent mixtures like white spirits, and chlorinated solvents such as methylene chloride.[15]

Paint production involved multiple stages, including weighing raw materials, mixing, milling, shearing, canning, and packaging.[3] Workers in paint manufacturing are frequently exposed to various chemicals including hazardous chemicals, volatile organic compound (VOC). Despite ongoing safety measures, VOC remain a major component of paints, leading to routine occupational exposure.[13] The term 'occupational hazard' refers to workers' exposure to potentially harmful substances. The World Health Organization (WHO) defines occupational hazards as risks, harm, or dangers faced by workers due to job-related exposures. Occupational diseases are illnesses that can be associated with exposure to chemical agents encountered in the workplace.[55] While chemicals are essential to the operations of the paint manufacturing industry, their use also raises serious health concerns. In particular, workers in paint manufacturing are routinely exposed to mixed chemical agents that may lead to early biological effects, including DNA damage and neurobehavioral impairments.

Globally, increasing attention has been directed toward the harmful effects of occupational chemical exposure on human health. Studies from various countries-including Canada, Korea, New Zealand, Spain, Iran, Pakistan, Thailand, Argentina, Mexico, and Colombia-have examined the occupational exposure of painters to VOC.[12] Workers in these industries encounter multiple hazards, which can pose potential health risks, especially for those directly handling chemicals. These VOCs contribute to a range of health issues, including respiratory problems, neurotoxic effects, and long-term DNA damage. VOCs, alongside other chemicals in the paint production environment, heighten the risk of occupational diseases. Understanding the collective impact of these substances is crucial for assessing their role in workers' health outcomes. However, the severity of the health effects caused by chemical exposure can vary depending on several factors, such as an individual's diet, smoking habits, physical activity, and pre-existing health conditions.

These factors can influence how the body metabolizes and responds to toxic substances. In addition to these individual factors, the production, storage, transportation, and use of chemicals-especially in industries like paint manufacturing-can pose significant risks to human health and safety. Different types of chemicals, particularly solvents, pigments, and additives in paints, can have harmful effects on workers and the surrounding environment. Numerous studies have linked solvents used in the paint industry to health concerns, including central and peripheral nervous system damage.

Paint production exposes workers to volatile organic compounds (VOCs) which contribute to nucleic acid damage, oxidative stress, genotoxicity, and inflammation.[37] However, the severity of these effects vary depending on factors such as diet, smoking habits, physical activity, pre-existing health conditions, environmental exposure, age, metal state, and stress levels.[21] Workers in paint manufacturing are exposed to complex chemical mixtures, such as organic hydrocarbons, including toluene, ketones, and alcohol esters.[33] While the International Agency for Research on Cancer (IARC) classifies these substances as non-carcinogenic,[30] exposure to certain chemical mixtures, including metals may elevate the risk of cancer.[68] Studies have linked exposure to complex chemical mixtures to neurological,[9] auditory,[61] hepatic,[36] and respiratory[56] issues. Occupational exposure to organic solvents[5,16,26,4] and certain metals[41,44,49] has also been associated with DNA damage and oxidative stress, though the precise mechanisms remain unclear.

Additionally, exposure to organic solvents has been implicated in hemotoxic conditions, such as anaemia and leukaemia.[36] Previous reports indicate that paint exposure can cause DNA damage. The International Industrial Accident Committee (IIAC) analysed epidemiological data and concluded that painters face twice the occupational cancer risk compared to the general population.[13,70] Increased chromosomal aberrations, sister chromatid exchange, and DNA

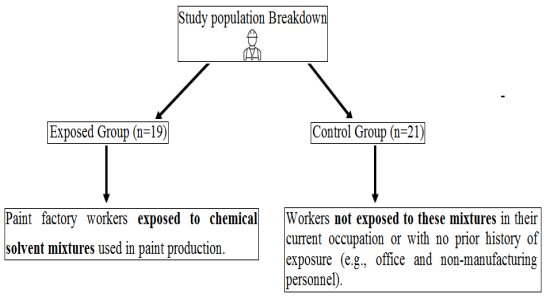
damage had been reported in the leukocytes of workers exposed to paints. Studies also detected elevated micronuclei frequencies in peripheral lymphocytes and oral mucosal cells of these workers.[53] Ten epidemiological studies identified an association between lung cancer and occupational exposure in Korean commercial painters. However, limited research has focused specifically on workers in paint manufacturing. Therefore, this study aims to assess the genotoxic effects of occupational chemical exposure by evaluating DNA damage in buccal cells of paint.

MATERIALS AND METHODS

Study design

This cross-sectional study was conducted at a small industrial paint manufacturing plant in Bangi, Selangor, Malaysia, from November 2021 to December 2022.

Population



Ethical consideration

This study was approved by the Research and Ethics Committee of the Faculty of Health Sciences, Universiti Kebangsaan Malaysia. The workers were informed of the study's aim, and that the data would only be used for scientific purposes; they also had the right to refuse to participate in this study. All the participants provided informed consent.

Sampling technique

A total of 40 workers with at least one year of uninterrupted employment and no major diseases related such as cancer, genetic disorders, or other health conditions that could confound DNA damage results, participated. The exposed group had[19] respondents, while the control group had[21]. Each respondent completed a structured questionnaire and provided three buccal samples for DNA damage assessment.

Data Collection Using Questionnaire

A predesigned four-part questionnaire collected:

1. Sociodemographic data (age, gender, education level)
2. Lifestyle factors (smoking status)
3. Medical history (genetic disorder, cancer, poisoning)
4. Occupational exposure (working sectors, duration of chemical exposure, personal protective equipment usage, safety awareness).

Buccal cell sampling

Buccal samples were collected on workdays (Monday-Friday) between 7 and 9 a.m to minimize smoking-related DNA damage. Participants rinsed their mouths twice before sampling and refrained from eating 15 min before collection. Buccal cells were obtained by gently scraping both sides of the inner cheeks with cotton swabs for 15-30 seconds. Swabs were immersed in 30 mL of cold Titenko-Holland buffer solution (4°C) containing 0.01 M Tris HCl, 0.1 M EDTA, and 0.02 M NaCl (pH 7). Samples were transported to the laboratory within 2–4 h and centrifuged (10 min at 2500 rpm). The cell pellet was washed with phosphate-buffered saline (PBS) and mixed with low-melting-point agarose (LMA) before slide preparation.

Comet assay

The standard alkaline comet assay procedure was adapted from a study by Rojas *et al.,*(2014). Fully frosted microscope slides were pre-coated with 0.7% normal-melting-point agarose (NMA). The cell pellet was layered with LMA, covered with coverslip and solidified on an ice-cold tray. After that, slides were immersed in lysis solution (2.5 M NaCl, 100 mM Na4 EDTA, 10 mM Tris-HCl (pH 10), 1% Triton X-100 and 10% DMSO) for 1 h at room temperature. DNA was unwound in electrophoresis buffer (1 mM Na2EDTA, 300 mM NaOH, pH<13) for 20 min and electrophoresed (25 V, 300 mA, 20 minute). All steps were conducted in dim light to prevent additional DNA damage. Later,

slides were taken out from the tank and neutralized (0.4 M Tris, pH 7.5) and stained with ethidium bromide (20 µL/mL). The stained slides were covered with a coverslip and stored in a humidified box at 4°C, and comet image were analysed using a fluorescence microscope (Olympus, BX51, 546 nm excitation, 590 nm barrier filter). DNA damage was quantified using TriTex CometScore Freeware v1.5. Tail DNA percentage (%DNAT) was calculated as: [%DNAT=100 × DNAT/(DNAT+DNAH)].

Statistical analysis

Quantitative and qualitative data are presented as percentages. An independent t-test was used to compare tail moment mean percentage between exposed and non-exposed groups. Pearson correlation assessed relationships between DNA damage parameters (tail moment, %DNA damage, tail length) and independent variables (age, education level, work duration, smoking history, exposure frequency). Linear and multivariate regression analyses determined associations between independent variables and mean tail moment percentage. All collected data were computerized and statistically analysed using SPSS version 26.0 for the calculations.

RESULTS

Table 1 shows the primary demographic features of the two groups tested (non-exposed/ control and exposed workers). The exposed and non-exposed workers were statistically comparable, with no significant differences in smoking habits (p=2.950) or age (p=0.334). Nearly 70% of the control group were males, compared to 90% in the exposed group, though age differences were not significant. On average, exposed workers spent 1.40±0.50 hours daily handling chemicals, with work hours varying based on workload. The longest recorded workday was 9 h for exposed workers and 10 h for non-exposed workers. Smoking was more prevalent among exposed workers (36.8%) than in the control group (23.8%). This indicates that more exposed workers are smokers compared to the non-exposed workers.

Table 1: Demographic characteristics of the study group

Characteristic	Exposed Workers N = 19 n (%)	Mean ± SP	Control Workers N = 21 n (%)	Mean ± SP
Age				
18 – 35 Years	13 (68.4)	1.33 ± 0.49	14 (66.7)	1.32 ± 0.48
36 – 55 Years	6 (31.6)		7 (33.3)	
Gender				
Male	18 (94.7)	_____	14 (66.7)	_____
Female	1 (5.3)		7 (33.3)	
Daily Exposure to Paint				
Production (Hours)	Range	8.22 ± 0.43		
	8h-9h	_____	_____	
Working Period				
5 Years ≤		1.33 ± 0.49		1.41 ± 0.50
13 (68.4)		12 (57.1)		
< 5 Years	6 (31.6)		9 (42.9)	
Smoking Status				
Yes	7 (36.8)		5 (23.8)	
No	12 (63.2)		16 (76.2)	

Occupational data were gathered from the second section of the questionnaire. While 52.6% of exposed workers had formal safety training, 52.6% were unaware of workplace hazards and potential health risks, compared to 71.4% of non-exposed workers who received training (Table 2). Although 89.5% of exposed workers reported using personal protective equipment (PPE), observations revealed improper mask usage, inconsistent glove use, and inadequate protection, such as wearing short sleeves while handling chemicals. During production, workers occasionally removed masks due to respiratory discomfort, exposing themselves to hazardous chemicals used in paint production, including toluene, xylene, methanol, and alcohol.

Table 2: Occupational information

Characteristic	Exposed Worker N = 19 n (%)	Mean ± SP	Control Worker N = 21 n (%)	Mean ± SP
Formal Training on Hazard and Safety Measures	Yes (45.5)	n:9	Yes (71.4)	n:14
	No (50.0)	n:10	No (28.6)	n:7
Aware of Hazards Related to Their Job	Yes (47.4)	n:9	_____	_____
	No (52.6)	n:10		
Use of Individual Protection Equipment	Use	17		
	Not Use	3		_____
Frequency Involves with Paint Production Process (Chemical)	Frequent (73.7)	n:14	_____	_____
	Occasionally (26.3)	n:5		

Table 3 summarizes the minimum values and standard deviations for comet assay parameters indicating DNA damage, including comet tail length, %DNA in the comet tail, and tail moment for both groups. In the exposed group, the comet tail length is 10.86±3.45, %DNA in the tail is 8.97±2.16, and tail moment is 2.77±2.35. In contrast, the control group shows comet tail length of 6.35±2.40, %DNA in the tail of 5.42±1.24, and tail moment of 0.87±0.67. An independent t-test revealed significant differences ($p<0.05$) in all three parameters between the groups. The exposed group's comet tail length is 1.71 times higher, %DNA in the tail is 1.65 times higher, and tail moment is 3.18 times higher compared to the control group, confirming greater DNA damage in exposed workers.

Table 3: DNA damage in buccal cells

	Type of Workers	N	Mean	Std. Deviation	p-value
Tail length (px)	Exposed	19	10.86	3.45	<0.001
	Non-Exposed	21	6.35	2.40	
Tail DNA (%)	Exposed	19	8.97	2.16	<0.001
	Non-Exposed	21	5.42	1.24	
Tail Moment	Exposed	19	2.77	2.35	<0.001
	Non-Exposed	21	0.87	0.67	

Pearson correlation analysis (Table 4) was used to assess the relationship between DNA damage parameters-comet tail length, %DNA in the comet tail, and tail moment-and enabling factors such as age, education level, duration of employment, years of smoking, and frequency of chemical exposure in paint manufacturing. Results indicate a moderate negative correlation between the duration of employment and %DNA in the comet tail in the exposed group ($p=0.047$, $r=-0.46$). However, duration of employment showed no significant correlation with comet tail length or tail moment. Similarly, no significant correlations were found between age, education level, smoking duration, or frequency of exposure and any DNA damage parameters in both exposed and control groups ($p > 0.05$).

Table 4: Correlation Between DNA Damage Parameters and Enabling Factors

Group	Factor	%DNA in tail		Tail Length (px)		Tail Moment	
		r	P	r	P	r	P
Non-Exposed (N=21)	Age	-0.29	0.203	-0.23	0.315	-0.10	0.669
	Education level	0.17	0.469	-0.07	0.773	-0.16	0.498
	Duration of employment (year)	-0.07	0.751	-0.09	0.706	0.19	0.409
	Smoking status	-0.06	0.814	0.37	0.096	0.03	0.887
	Frequency of exposure to chemicals during paint manufacturing process	0.27	0.241	0.40	0.071	0.26	0.249
Exposed (N=19)	Age	-0.23	0.335	-0.01	0.982	0.04	0.864
	Education level	0.04	0.866	-0.18	0.471	-0.20	0.404
	Duration of employment (year)	-0.46	0.047*	0.06	0.804	0.12	0.618
	Smoking status	-0.08	0.747	-0.30	0.216	-0.20	0.406
	Frequency of exposure to chemicals during paint manufacturing process	-0.23	0.343	0.23	0.336	0.26	0.276

The simple and multiple linear regression (SLR & MLR) analyses (Tables 5 to 7) examined the relationship between the DNA damage parameters (comet tail length, %DNA in the comet tail, and tail moment) and independent variables. SLR results indicate that the frequency of chemical exposure in paint manufacturing significantly correlates with all three DNA damage parameters ($p<0.05$). Specifically, increased exposure frequency is associated with a rise of 0.65 units in comet tail length, 0.63 units in %DNA in the comet tail, and 0.54 units in tail moment. However, MLR results show that no independent variables significantly affect comet tail length. For %DNA in the comet tail, smoking duration emerges as the most significant predictor (β=0.42), suggesting that prolonged smoking increases DNA damage. Tail moment readings remain unaffected by any independent variables in the MLR model

Table 5: The simple linear regression (SLR) and multiple linear regression (MLR) analyses for comet tail length parameters with the independent variables

	SLR					MLR				
Factor/Parameter				95.0% Confidence Interval for B					95.0% Confidence Interval for B	
Tail Length (px)	B	Beta	P	Lower Bound	Upper Bound	B	Beta	P	Lower Bound	Upper Bound
Age	-0.02	-0.05	0.756	-0.16	0.12	0.04	0.08	0.835	-0.31	0.38
Education level	-1.04	-0.26	0.102	-2.30	0.22	-0.32	-0.08	0.753	-2.43	1.79
Smoking status	0.03	0.08	0.643	-0.11	0.18	0.01	0.03	0.915	-0.21	0.24
Duration employment	-0.03	-0.03	0.870	-0.35	0.29	0.00	0.00	0.993	-0.52	0.52
Frequency of exposure to chemicals during paint manufacturing process	1.82	0.65	0.000*	1.13	2.51	2.20	0.79	0.101	-0.47	4.87

Table 6: The simple linear regression (SLR) and multiple linear regression (MLR) analyses for % of DNA in tail parameters with the independent variables

	SLR					MLR				
Factor/Parameter				95.0% Confidence Interval for B					95.0% Confidence Interval for B	
%DNA in Tail	B	Beta	P	Lower Bound	Upper Bound	B	Beta	P	Lower Bound	Upper Bound
Age	-0.04	-0.14	0.395	-0.13	0.05	-0.15	-0.52	0.093	-0.33	0.03
Education level	-0.36	-0.14	0.399	-1.23	0.50	0.10	0.04	0.849	-0.98	1.18
Smoking status	0.05	0.15	0.345	-0.05	0.14	0.13	0.42	0.033*	0.01	0.24
Duration employment	-0.15	-0.23	0.154	-0.36	0.06	0.06	0.09	0.634	-0.21	0.33
Frequency of exposure to chemicals during paint manufacturing process	1.17	0.63	0.000*	0.70	1.65	-0.40	-0.21	0.550	-1.77	0.97

Table 7: The simple linear regression (SLR) and multiple linear regression (MLR) analyses for tail moment parameters with independent variables

	SLR					MLR				
Factor/Parameter				95.0% Confidence Interval for B					95.0% Confidence Interval for B	
Tail Moment	B	Beta	P	Lower Bound	Upper Bound	B	Beta	P	Lower Bound	Upper Bound
Age	0.01	0.03	0.857	-0.07	0.08	-0.03	-0.12	0.815	-0.27	0.22
Education level	-0.56	-0.27	0.087	-1.22	0.09	-0.05	-0.03	0.943	-1.55	1.44
Smoking status	0.00	0.00	0.977	-0.08	0.07	-0.08	-0.34	0.317	-0.24	0.08
Duration employment	0.04	0.09	0.601	-0.12	0.21	0.13	0.25	0.482	-0.24	0.50
Frequency of exposure to chemicals during paint manufacturing process	0.79	0.54	0.000*	0.39	1.18	0.32	0.22	0.726	-1.57	2.22

DISCUSSION

The rapid development of the paint manufacturing industry has contributed significantly to economic growth in many countries, including Malaysia. However, inadequate workplace monitoring, lack of awareness regarding hazardous agents, and insufficient safety protocols pose serious occupational health risks to workers. Exposure to complex chemical mixtures in paint

manufacturing may lead to increased cytogenetic damage, highlighting the need for biomonitoring and workplace safety improvements. Hence, this study aimed to assess early DNA damage among paint manufacturing workers and contribute to understanding the occupational exposure risks in this industry, as numerous studies have reported significant DNA damage in workers exposed to complex chemical and paint mixtures.[17]

Our findings indicate a significant increase in DNA damage among exposed workers compared to the control group. Using the comet assay, we observed higher values for comet tail length, percentage of DNA in the comet tail, and tail moment parameters in exposed workers. Independent t-test analysis confirmed statistically significant differences between the two groups ($p<0.05$). These findings align with previous studies that reported increased DNA damage among individuals exposed to paint chemicals, reinforcing concerns about the genotoxic potential of occupational chemical exposure.

A major challenge in assessing health risks in the paint industry is the lack of transparency regarding the complete chemical composition of paints. However, substances such as toluene, xylene, ethylbenzene, butyl and ethyl acetate, acetone, methyl isobutyl ketone, and aluminum are commonly present and have been linked to DNA damage and oxidative stress.[8] While much of the existing literature focuses on painters, limited research has been conducted on workers involved in paint manufacturing. This study helps bridge that gap by evaluating the early biological effects of chemical exposure in this underrepresented population.

The comet assay has been widely recognized as a sensitive and reliable tool for detecting DNA damage, including strand breaks and alkali-labile sites. It is particularly useful in occupational health studies as it provides rapid and cost-effective assessments of genotoxic exposure.[10] The use of buccal mucosa (BM) cells in this study further strengthens the findings, as BM cells serve as a first-line defense against inhaled or ingested carcinogens.[70,75] Additionally, buccal cell sampling is non-invasive and does not cause undue stress to participants, making it an effective method for biomonitoring occupational exposure.[47]

In this study, DNA damage was found to correlate significantly with employment duration but not with smoking status. Workers with shorter employment durations (<5 years) exhibited DNA damage levels comparable to those with longer exposure periods, suggesting that even short-term exposure may be harmful. This finding is consistent with previous studies[18] that reported a positive correlation between occupational exposure duration and increased genetic damage. However, the negative correlation observed in this study suggests that newly employed workers may be particularly vulnerable to DNA damage, potentially due to differences in metabolic detoxification pathways, which warrants further investigation into genetic polymorphisms and exposure thresholds.[63]

Although some studies have reported no significant DNA damage among exposed workers, this variation may be attributed to differences in chemical concentrations, exposure duration, and individual genetic responses. Many chemicals used in modern paint formulations are classified as non-carcinogenic (Category 3), but cumulative exposure over time may still pose genotoxic risks.[50,73] This underscores the need for more stringent occupational safety measures, particularly in environments where workers handle multiple chemicals simultaneously.

The findings of this study have significant implications for occupational health and safety in the paint manufacturing industry. Employers and regulatory authorities, such as the Department of Occupational Safety and Health (DOSH), must enforce strict safety guidelines, including the mandatory use of personal protective equipment (PPE) and proper ventilation systems. Additionally, implementing biomonitoring programs to assess workers' chemical exposure levels can enhance workplace safety. Future research should focus on validating specific biomarkers for chemical exposure, exploring genetic susceptibility to genotoxic effects, and developing safer, environmentally friendly alternatives in paint production.

Journalism Ethical Considerations

Ethical issues (including plagiarism, informed consent, errors, falsification and/or fabrication of data, multiple publications and/or submissions, redundancy) have been fully addressed by the authors.

CONCLUSION

This cross-sectional study (November 2021–December 2022) examined DNA damage among paint factory workers in Bangi, Selangor, Malaysia. Significant differences in comet assay parameters (DNA tail length, %DNA in the tail, and tail moment) were observed between exposed and non-exposed workers ($p<0.001$), reinforcing existing evidence that occupational exposure in the paint industry poses a high genotoxic risk. Despite this, identifying specific genotoxic hazards from the various chemicals used in paint production remains challenging. Thus, stringent occupational safety regulations are essential, particularly in paint manufacturing. Regulatory bodies such as the Department of Occupational Safety and Health (DOSH) can use this study as a reference to implement targeted enforcement and preventive measures. Employers must ensure proper personal protective equipment (PPE) usage, improve ventilation, and establish exposure monitoring programs using suitable biomarkers. Future research should focus on safer, environmentally friendly chemicals and sustainable manufacturing processes to mitigate long-term health and environmental risks. Prioritizing workers' health not only enhances their well-being but also contributes to overall workplace productivity and industry sustainability.

ACKNOWLEDGEMENT

The authors would like to thank the Ministry of Higher Education and the National University of Malaysia (UKM) for their funding and support.

Funding

Financial support for this research was provided by the Universiti Kebangsaan Malaysia (UKM) through a grant from the Ministry of Higher Education Malaysia, Fundamental Research Grant Scheme (FRGS) with the grant number FRGS/1/2020/STG02/UKM/02/4.

Conflict of interest

The authors declare no conflict of interest relating to the material presented in this article. Its contents, including any opinions and/or conclusions expressed, are solely those of the authors.

REFERENCES

1. Aliyu, A.A.; Shehu, A.U. Occupational hazards and safety measures among stone quarry workers in Northern Nigeria., *Nigerian Medical Practice.*, **2006**, *50*, 42–7.
2. Awodele, O.; Popoola T.D.; Ogbudu, B.S.; Akinyede, A.; Coker, H.A.B.; Akintonwa, A. 2014. Occupational hazards and safety measures amongst the paint factory workers in Lagos, Nigeria., *Safety and Health at Work.*, **2014**, *5*, 106–111.
3. Abdullah Youssef, Abdal-Rhman. Paints Industry: Raw materials & unit operations & Equipment & Manufacturing & Quality tests., **2019.**
4. Balkhyour, M.A.; Chakroun, R.; & Faidi, F. Evaluation of environmental and biological monitoring methods for toluene exposure assessment in paint industry., *Saudi Journal of Biological Sciences.*, **2023**, *30*(2).
5. Bayil, S.; Cicek, H.; Cimenci, I.G.; Hazar, M. How volatile organic compounds affect free radical and antioxidant enzyme activity in textile workers., *Archives of Industrial Hygiene and Toxicology.*, **2008**, *59*, 283–7.
6. Bilban, M. Mutagenic. Testing of Workers Exposed to Toluene-Diisocyanates during Plastics Production Process., *American Journal of Industrial Medicine.*, **2004**, *45*, 468–474.
7. Bolognesi, C. Genotoxicity of pesticides: A review of human biomonitoring studies., *Mutation Research.*, **2003**, *543*, 251-72.
8. Chang, F.K.; Mao, I.F.; Chen, M.L.; Cheng, S.F. Urinary 8-hydroxydeoxyguanosine as a biomarker of oxidative DNA damage in workers exposed to ethylbenzene., *Annals of Occupational Hygiene.*, **2011**, *55*, 519–25.
9. Chen, R.; Dick, F.; Seaton, A. Health effects of solvent exposure among dockyard painters: Mortality and neuropsychological symptoms., *Occupational Environment Medicine.*, **1999**, *56*, 383–7.
10. Collins, A.R. The comet assay for DNA damage and repair: Principles, applications, and limitations., *Appl Biochem Biotechnol Part B Mol Biotechnol.*, **2004**, *26*, 249–261.
11. Cárdenas-Bustamante, O.; Varona-Uribe, M.; Patiño-Florez, R.I.; Groot-Restrepo, H.;

Sicard-Suárez, D.; Tórres-Carvajal, M.M.; Pardo-Pardo, D.; Bogotá. Paint-industry workers exposure to organic solvents and genotoxic effects., *Revista de Salud Publica*., **2007**, *9*, 275-288.

12. Castaño, B.P.; Ramírez, V. & Cancelado, J.A. Controlling Painters' Exposure to Volatile Organic Solvents in the Automotive Sector of Southern Colombia., *Safety and Health at Work*., **2019**, *10*(3), 355–361.
13. De Oliveira, H.M.; Dagostim, G.P.; Mota, A.D.S.; Tavares, P.; Da Rosa, L.A.Z.C.; De Andrade, V.M. Occupational risk assessment of paint industry workers., *Indian Journal of Occupational and Environmental Medicine.,* **2011**, *15*, 52–58.
14. De Rosa, E.; Brugnone, F.; Bartolucci, G.B.; Perbellini. L.; Bellomo, M.L.; Gori, G.P.; Sigon, M.; Chiesura, P.C. The validity of urinary metabolites as indicators of low exposures to toluene., *International Archives of Occupational and Environmental Health*., **1985**, *56*, 135-45.
15. Dick, F.D. Solvent neurotoxicity., *Journal of Occupational and Environmental Medicine*., **2006**, *63*, 221–226.
16. Duan, H.; Leng, S.; Pan, Z.; Dai, Y.; Niu, Y.; Huang, C.; Bin, P.; Wang, Y.; Liu. Q.; Zheng, Y. Biomarkers measured by cytokinesis-block micronucleus cytome assay for evaluating genetic damages induced by polycyclic aromatic hydrocarbons., *Mutation Res*., **2009**, *677*, 93–9.
17. Da Silva, J. DNA damage induced by occupational and environmental exposure to miscellaneous chemicals., *Mutat Res Rev Mutat Res*., **2016**, *770*(PtA), 170-182.
18. Dos Reis Filho, A.P.; Silveira, M.A.D.; Demarco, N.R.; D'Arce, L.P.G. Increased DNA damage, instability and cytokinesis defects in occupationally exposed car painters., *In vivo*., **2019**, *33*(6), 1807-1811.
19. El Mahdy. N, M.; Radwan, N.M. Assessment of different health hazards in painting industry., *The Egyptian Journal of Occupational Medicine*., **2009**, *33*, 211–232.
20. Esra AlDhaen. Awareness of occupational health hazards and occupational stress among dental care professionals: Evidence from the GCC region., **2022**.
21. Fidler, A.T.; Baker, E.L.; Letz, R.E. Estimation of long-term exposure to mixed solvents from questionnaire data: a tool for epidemiologic investigations., *British Journal of Industrial Medicine*., **1987**, *44*, 133.
22. Fracasso, M.E.; Doria, D.; Bartolucci, G.B.; Carrieri, M.; Lovreglio, P.; Ballini, A.; Soleo, L.; Tranfo, G.; Manno, M. Low air levels of benzene: Correlation between biomarkers of exposure and genotoxic effects., *Toxicology Letters*., **2010**, *192*, 22–28.
23. Gabelova, A.; Collins, A.; Langie, S.A.S. 2023. Minimum Information for Reporting on the Comet Assay (MIRCA): recommendations for describing comet assay procedures and results., *Nat Protoc.,* **2020**, *15*(12), 3817-3826.
24. Ghobakhloo, S.; Khoshakhlagh, A.H.; Morais, S.; Mazaheri Tehrani, A. Exposure to Volatile Organic Compounds in Paint Production Plants: Levels and Potential Human Health Risks., *Toxics*., **2023**, *11*(2), 111.
25. Hininger, I.; Chollat-Namy, A.; Sauvaigo, S.; Osman, M.; Faure, H.; Cadet, J.; Favier, A.; Roussel, A.M. Assessment of DNA damage by comet assay on frozen total blood: method and evaluation in smokers and non-smokers., *Mutat Res*., **2004**, *558*(1-2), 75-80.
26. Heuser, V. D.; Erdtmann, B.; Kvitko, K.; Da Silva, J. Evaluation of genetic damage in Brazilian footwear-workers: Biomarkers of exposure, effect and susceptibility., *Toxicology*., **2007**, *232*, 235-47.
27. Holland, N.; Bolognesi, C.; Kirsch-Volders, M.; Bonassi, S.; Zeiger, E.; Knasmueller, S.; Fenech, M. The micronucleus assay in human buccal cells as a tool for biomonitoring DNA damage: the HUMN project perspective on current status and knowledge gaps., *Mutation Research*., **2008**, *659*, 93-108.
28. Hegazy, S.I.; El-Raghi, H.A.; Mohammed, Asmaa, Rizk, S.A.; Badawy, N.A. Prevalence of Renal Impairment among Workers of a Paint Manufacturing Factory., **2016** Oct.
29. Hawari, N.S.S.L.; Latif, M.T.; Othman, M.; Hanif, N.M.; Hamid, H.H.A.; Mustaffa, N.I.H. & Mohtar, A.A.A. One-year monitoring of aromatic volatile organic compounds (VOCs) in urban areas of Malaysia. *IOP Conference Series: Earth and Environmental Science*., **2021**, 880(1).

30. IARC. Inorganic and organic lead compounds. Monograph on the evaluation of the carcinogenic risk of chemicals to humans: IARC Press, Geneva., **2007**.
31. Jafari, M.J.; Karimi, A.; Azari, M.R. The role of exhaust ventilation systems in reducing occupational exposure to organic solvents in a paint manufacturing factory., *Indian Journal Occupational Environmental Medicine*., **2008**, *12*, 82-87.
32. James, I.C.; Imaga, N.A.; Dokunmu, T.M.; Adedeji, I.O.; Emmanuel, O.O.; Orija, M.E. Effects of chronic exposure to paint fumes among artisans in Lagos State, Nigeria., *Toxicology Reports*., **2022**, *28*(9), 663-669.
33. Jiménez-López, A.M.; & Hincapié-Llanos, G.A. Identification of factors affecting the reduction of VOC emissions in the paint industry: Systematic literature review-SLR., *Progress in Organic Coatings*. **2022**, *170*.
34. Jiang N.; Naz S.; Ma Y.; Ullah Q.; Khan MZ.; Wang J.; Lu X.; Luosang D-Z.; Tabassum S.; Chatha AMM., An Overview of Comet Assay Application for Detecting DNA Damage in Aquatic Animals., *Agriculture*., **2023**., *13*(3), 623.
35. Kamal, A.; Malik, R.N. Hematological Evidence of Occupational Exposure to Chemicals and Other Factors among Auto-Repair Workers in Rawalpindi, Pakistan., *Osong Public Health and Research Perspectives*., **2012**, *3*(4), 229-38.
36. Kaukiainen. A.; Vehmas, T.; Rantala, K.; Nurminen, M.; Martikaine, R.; Taskinen, H. Results of common laboratory tests in solvent-exposed workers., *Int Arch Occup Environ Health*., **2004**, *77*, 1432–46.
37. Kianmehr, M.; Hajavi, J.; Gazeri, J. Assessment of DNA damage in blood lymphocytes of bakery workers by comet assay., *Toxicology and Industrial Health*., **2017**, *33*, 726–735.
38. Kwon, J.W.; Park, H,W.; Kim, W.J.; Kim, M.J.; Lee, S.J. Exposure to volatile organic compounds and airway inflammation., *Environment Health*., **2018**, *17*(1), 65.
39. Ladeira, C.; Gajski, G.; Meneses, M.; Geri, M.; Viegas, S. The genotoxicity of an organic solvent mixture: a human biomonitoring study and translation of a real-scenario exposure to *in vitro*., *Regulatory Toxicology and Pharmacology*., **2020**, 116.
40. Lee, K.H.; Ichiba, M.; Zhang, J.; Tomokuni, K.; Hong, Y.C.; Ha, M.; Ho, J.K.; Koh, S.B.; Choi, H.R.; Lee, K.H.; Park, C.G.; Cho, S.H.; Ari, H.; Paul, T.S.; Roel, V.; Richard, B.H.; Kang, D. Multiple biomarkers study in painters in a shipyard in Korea., *Mutation Research*., **2003**, *540*, 89-98.
41. Liu, H.H.; Lin, M.H.; Liu, P.C.; Chan, C.I.; Chen, H.L. Health risk assessment by measuring plasma malondialdehyde (MDA), urinary 8-hydroxydeoxyguanosine (8-OH-dG) and DNA strand breakage following metal exposure in foundry workers., *Journal of Hazard Material*., **2009**, *170*(2-3), 699–704.
42. Londoño-Velasco, E.; Martínez-Perafán, F.; Carvajal-Varona, S.; García-Vallejo, F.; Hoyos-Giraldo, L.S. Assessment of DNA damage in car spray painters exposed to organic solvents by the high-throughput comet assay., *Toxicology Mechanism and Methods*., **2016**, *26*(4), 238-42.
43. Marczynski, B.; Pesch, B.; Wilhelm, M.; Rossbach, B.; Preuss, R.; Jens-Uwe, H.; Rabstein, S.; Raulf-Heimsoth, M.; Seidel, A.; Hans-Peter, R.; Adams, A.; Scherenberg, M.; Erkes, A.; Engelhardt, B.; Straif, K.; Käfferlein, H.U.; Angerer, A.; Thomas B. Occupational exposure to polycyclic aromatic hydrocarbons and DNA damage by industry: a nationwide study in Germany., *Arch Toxicol*., **2009**, *83*(10), 947–57.
44. Mateuca, R.; Aka, P.V.; De Boeck, M.; Hauspie, R.; Kirsch-Volders, M. Influence of hOGG1, XRCC1 and XRCC3 genotypes on biomarkers of genotoxicity in workers exposed to cobalt or hard metals dusts., *Toxicology Letter*., **2005**, *156*(2), 277–88.
45. Migliore, L.; Colognato, R.; Naccarati, A.; Bergamaschi, E. Relationship between genotoxicity biomarkers in somatic and germ cells., *Findings from a biomonitoring study*. **2006**, *21*(2),149-52.
46. Mo, Z.; Lu, S.; Shao, M. Volatile organic compound (VOC) emissions and health risk assessment in paint and coatings industry in the Yangtze River Delta, China., *Environmental Pollution*., **2021**, *269*, 115740.
47. Moller, P. The alkaline comet assay: Towards validation in biomonitoring of DNA damaging exposures., *Basic Clinical Pharmacology Toxicology*., **2006**, *98*(4), 336–345.

48. Manda, N.; Mohamed-Katerere.; Jennifer. Chemicals: our environment, our health., *Africa Environ Outlook*., **2004**, *5*, 42-69.
49. Mohammad, I.K.; Mahdi, A.A.; Raviraja, A.; Najmul, I.; Iqbal, A.; Thuppil, V. Oxidative stress in painters exposed to low lead levels., *Archives of Industrial Hygiene and Toxicology*., **2008**, *59*(3),161–9.
50. Moro, A.M.; Brucker, N.; Charão, M.; Bulcão, R.; Freitas, F.; Baierle, M.; Nascimento, S.; Valentini, J.; Cassini, C.; Salvador, M. Evaluation of genotoxicity and oxidative damage in painters exposed to low levels of toluene., *Mutat. Res. Toxicol. Environ. Mutagen*., **2012**, *746*, 42–48.
51. Møller, P.; Azqueta, A.; Boutet-Robinet, E. Minimum Information for Reporting on the Comet Assay (MIRCA): recommendations for describing comet assay procedures and results., *Nat Protoc*., **2020**, *15*, 3817–3826.
52. Myong, J.P.; Cho, Y.; Choi, M.; Kim, H.R. Overview of occupational cancer in painters in Korea., *Annals of Occupational and Environmental Medicine*., **2018**, *30*, 10.
53. Olurin A., *Evaluation review of occupational effect of car paint component mixture fume exposure on lungs in osogbo*., **2021**, *7*(11), 50–60.
54. Packard, R.M. Industrial production; health and disease in sub-Saharan Africa., *Social Science Medicine*., **1989**, *28*, 75–96.
55. Pandey, P.; Kiran, U.V. Solvent based paint and its impact on environment and human beings., Environment and Society., **2020**, 198–207
56. Park, J.; Lee, C.G.; Ryu, S.Y. Factors related to the prevalence of respiratory symptoms in workers in a petrochemical complex., *Journal Occupational and Health*., **2006**, *48*, 216–22.
57. Pinto, D.; Ceballos, J.M.; García, G.; Guzmán, P.; Del Razo, L.M.; Vera, E.; Gomez, H.; Garcia, A.; Gonsebatt, M,E. Increased cytogenetic damage in outdoor painters., *Mutation Research*., **2000**, *467*(2), 105–11.
58. Pitarque, M.; Vaglenov, A.; Nosko, M.; Pavlova, S.; Petkova, V.; Hirvonen, A.; Creus, A.; Norppa, H.; Marcos, R. Sister Chromatid Exchanges and Micronuclei in Peripheral Lymphocytes of Shoe Factory Workers Exposed to Solvents., *Environmental Health Perspectives*., **2002**, *110*(4), 399-404.
59. Priha E. Burden of occupational diseases [internet]. Europian Agency for Safety and Health at Work. **2012** [cited 2022 Oct 26]. Available from: https://oshwiki.osha.europa.eu/en/themes/burden-occupational-diseases
60. Pandey, P.; Kiran, U.V. Exposure to paints and respiratory health ailments among painting workers., *Cogent Engineering*., **2023**, *10*(1).
61. Rabinowitz, P.M.; Galusha, D.; Slade, M.D.; Dixon-Ernst, C.; O'Neill, A.; Fiellin, M.; Cullen, M.R. Organic solvent exposure and hearing loss in a cohort of aluminum workers., *Occupational and Environmental Medicine*., **2008**, *65*(4), 230–5.
62. Rojas, E.; Valverde, M.; Sordo, M.; Ostrosky-Wegman, P. DNA damage in exfoliated buccal cells of smokers assessed by the single cell gel electrophoresis assay., *Mutatation Research*., **1996**, *370*, 115–120.
63. Roma-Torres, J.; Teixeira, J.P.; Silva, S.; Laffon, B.; Cunha, L.M.; Méndez, J.; Mayan, O. Evaluation of genotoxicity in a group of workers from a petroleum refinery aromatics plant. *Mutation Research-Genetic Toxicology and Environmental Mutagenesis*., **2006**, *604*, 19–27.
64. Ram.; Amritanshu.; Banerjee.; Birendranath.; Hosakote.; Vadiraja, S.; Rao.; Raghavendra, M.; Nagarathna; Raghuram. Comparison of lymphocyte apoptotic index and qualitative DNA damage in yoga practitioners and breast cancer patients: a pilot study., *International Journal of Yoga*., **2013**, *6*(1), 20-25.
65. Rahman, I. N.; Oginawati, K.; Ismawati, Y.; Buftheim, S.; Nugrahaningsih, D.A. Relationship between Lead Exposure and Genotoxic Effect in Paint Industry Workers., *Devotion Journal of Research and Community Service*., **2023**, *4*(7), 1507–1517.
66. Sánchez-Alarcón, J.; Milić, M.; Gómez-Arroyo, S.; Montiel-González, J.M.R.; Valencia-Quintana, R. Assessment of DNA Damage by Comet Assay in Buccal Epithelial Cells: Problems, Achievement, Perspectives [Internet]. *Environmental Health Risk-Hazardous Factors to Living Species. InTech*., **2016**. Available from: http://dx.doi.org/10.5772/62760

67. Sarto, F.; Tomanin, R.; Giacomelli, L.; Iannini, G.; Cupiraggi, A.R. The micronucleus assay in human exfoliated cells of the nose and mounth: Application to occupational exposures to chronic acid and ethylene oxide., *Mutation Research*., **1990**, *244*, 345-351.
68. Scélo, G.; Metayer, C.; Zhang, L.; Wiemels, J.L.; Aldrich, M.C.; Selvin, S. Household exposure to paint and petroleum solvents, chromosomal translocations, and the risk of childhood leukemia., *Environmental Health Perspectives*., **2009**, *117*(1), 133–9.
69. Silva, J.M.; Santos-Mello, R. Chromosomal aberrations in lymphocytes from car painters., *Mutation Research.,* **1996**, *368*(1), 21-5.
70. Sorahan, T.; Cross, H.; Sadhra, S.; Ayres, J. Occupational cancer risks in commercial painters [internet]. A review prepared for the Industrial Injuries Advisory Council (IIAC). **2010** April (Cited on 20 February **2023**). Available online: https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/330365/review-of-cancer-painters.pdf
71. Spivack, S.D.; Hurteau, G.J.; Jain, R.; Kumar, S.V.; Aldous, K.M.; Gierthy, J.F.; Kaminsky, L.S. Gene-environment interaction signatures by quantitative mRNA profiling in exfoliated buccal mucosal cells., *Cancer Res*., **2004**, *64*(18), 6805-13.
72. Squier, C.A.; Kremer, M.J. Biology of oral mucosa and esophagus., *J Natl Cancer Inst Monogr*., **2001**, *29*, 7-15.
73. Sisto, R.; Cavallo, D.; Ursini, C.L.; Fresegna, A.M.; Ciervo, A.; Maiello, R.; Paci, E.; Pigini, D.; Gherardi, M.; Gordiani, A.; L'Episcopo, N.; Tranfo, G.; Capone, P.; Carbonari, D.; Balzani, B. & Chiarella, P. Direct and Oxidative DNA Damage in a Group of Painters Exposed to VOCs: Dose Response Relationship., *Front. Public Health*., **2020**, *8*, 445.
74. Testa, A.; Festa, F.; Ranaldi, R.; Giachelia, M.; Tirindelli, D.; De Marco, A.; Owczarek, M,; Guidotti, M.; Cozzi, R. A multi-biomarker analysis of DNA damage in automobile painters., *Environmental and Molecular Mutagenesis*., **2005**, *46*, 182-188.
75. Vondracek, M.; Xi, Z.; Larsson, P.; Baker, V.; Mace, K.; Pfeifer, A.; Tjälve, H.; Donato, M.T.; Gomez-Lechon, M.J.; Grafström, R.C. Cytochrome P450 expression and related metabolism in human buccal mucosa., *Carcinogenesis*., **2001**, *22*(3), 481-488.
76. Whysner, J.; Reddy, M.V.; Ross, P.M.; Mohan, M.; Lax, E.A. Genotoxicity of benzene and its metabolites., *Mutation Research-Reviews in Mutation Research*., **2004**, *566*(2), 99–130.
77. Yamaguchi, N. H. Smoking, immunity, and DNA damage., *Translational Lung Cancer Research*., **2019**, *8*(S1), S3–S6.
78. Zhou, Y.; Wang, K. & Wang, B. Occupational benzene exposure and the risk of genetic damage: a systematic review and meta-analysis., *BMC Public Health*., **2020**, *20*, 1113.