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Assessment of DNA Damage in Auto Repair Workers Exposed to Heavy Metals in Al-Diwaniyah Province, Iraq

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ABSTRACT

Objective: *In Al-Diwaniyah auto repair workshops, workers encounter daily contact with lubricants, greases, paints, and solvents containing lead, cadmium, chromium, and zinc. Such metals can trigger DNA breaks via oxidative mechanisms. DNA damage levels assessed in blood lymphocytes from auto workers using alkaline comet assay.* ***Method:*** *Blood samples from 33 male workshop workers (mechanics, welders, painters) and 12 office-based male controls were analyzed. For each participant, we scored ≥50 cells, measuring tail length (μm), tail DNA percentage, and tail moment. Mann–Whitney U test compared groups, Cohen's d estimated effect magnitude.* ***Results:*** *Workshop personnel displayed substantially higher tail length (230.16 ± 12.00 μm versus 70.52 ± 1.15 μm in controls), tail DNA (9.16 ± 0.70% versus 1.13 ± 0.05%), and tail moment (13.86 ± 1.12 versus 1.42 ± 0.11), all $p < 0.001$. Effect sizes exceeded 12 for every parameter. Parameters were highly intercorrelated reflecting a common genotoxic stress.* ***Novelty:*** *Correlates of significant DNA damage found with Al-Diwaniyah work exposures justify protective equipment, sustained biological monitoring and Optimal ventilation are very important. Workers in car repair environments are mostly exposed to.*

INTRODUCTION

Workers in automotive repair shops, which include mechanics, painters, and welders, are constantly exposed to materials that are high in heavy metals. Lead, cadmium, chromium, and zinc are introduced in the workplace in the form of engine oil, which contains metal-based friction modifiers, lubricants, paints, welding rods, and brake dust [1], [2], [3]. Inadequate air venting in these workshops, coupled with the use of working environments with poor protective gear, is not uncommon; metal can accumulate in these individuals over a long period of time through inhalation and dermal contact as well [3], [4].

Heavy metals, which include lead (Pb) and cadmium (Cd), have also been implicated in the generation of reactive oxygen species (ROS), depletion of glutathione, and inhibition of enzymes responsible for DNA repair mechanisms in the body [5], [6]. In the cell, hexavalent chromium compounds are reduced, leading to the formation of DNA adducts as well as ROS [7], [8], [9]. This leads to DNA strand breaks and oxidative damage, which are a result of heavy metal exposure [5], [8]. Severe chromosomal damage and cancer risk are also linked with heavy metal exposure in different industries [5], [10].

Only small volumes of blood are needed, and results are rapid, making this a viable option for the detection of genotoxic damage before the onset of disease symptoms

[11], [12]. This approach, coupled with large-scale reviews of literature, which include the hCOMET database, where hundreds of studies are compiled, have been used to validate the use of this approach for the detection of DNA damage in metal-exposed working populations [11], [13], [14].

Elevated DNA damage has been reported in published biomonitoring efforts among automobile workers in South Asia and Africa [8], [10], [15]. Parallel investigations in Middle Eastern contexts have remained limited. Metal pollution has been documented in Iraqi cities and industries [16], [17], yet comprehensive genotoxicity screening of high-exposure occupations has not been completed. In Al-Diwaniyah, a population center in southern Iraq, numerous repair facilities are operated where metal-laden oils, paints, and welding materials are handled for extended periods without sufficient safeguards.

DNA damage in peripheral lymphocytes from Al-Diwaniyah auto workers was therefore measured, with office employees used as a reference group, to clarify the genotoxic impact of metal exposures arising from lubricants, greases, paints, and solvents.

RESEARCH METHOD

Study Design and Participants

Between 2025 and 2026, 33 male auto repair workers (including mechanics, welders, and painters) from Al-Diwaniyah workshops, along with 12 age-matched male office workers lacking industrial chemical contact, were recruited. Exposed workers were required to have at least 5 years in the trade and to be 18–60 years old. Individuals with a history of cancer, prior chemotherapy or radiotherapy, recent infections, autoimmune conditions, or recent medical radiation were excluded. Job type, employment duration, protective equipment habits, and smoking status were recorded using a questionnaire [10], [13], [14]. Ethics approval was granted by the institutional review board, and written consent was obtained from all participants before sampling.

Table 1. Demographic characteristics of study participation

Characteristic	Exposed Group (Workers n=33)	Control Group (n=12)	p-value / Notes
Age (years), mean ± SD (range)	Age-matched (18-60)	Age-matched (18-60)	Matched, p>0.05
Smoking status	All non-smokers	All non-smokers	No confounding
Non-smokers, n (%)	33 (100%)	12 (100%)	-
Employment duration (years), mean ± SD (range)	Recorded (typical 5-25 years)	N/A	N/A

Blood Collection and Cell Isolation

Venous blood (3 mL) was drawn into EDTA tubes, and samples were processed within 24 hours to minimize artifactual damage [11], [12], [18]. Leukocytes were

separated by density gradient centrifugation in accordance with standard protocols [11], [18].

Alkaline Comet Assay Protocol

The alkaline comet assay at pH > 13 was performed following validated guidelines [11], [14], [18]. Cells were isolated and then suspended in low-melting agarose on slides. They were lysed in a high-salt detergent buffer at 4°C for an hour. After that, the cells were placed in an alkaline solution (pH > 13) for twenty minutes to allow the DNA to unwind.

Electrophoresis was run at 0.7 V/cm and 4°C for 20 minutes. After neutralization in Tris buffer (pH 7.5), slides were stained with fluorescent dye and examined under fluorescence microscopy with image analysis. At least 50 randomly chosen nucleoids per person were scored, and tail length (μm), tail DNA (%), and tail moment were recorded as recognized markers of strand breaks in occupational genotoxicity research [11], [14], [18].

Statistical Approach

Data were presented as mean ± standard deviation (SD). The Shapiro–Wilk test was used to assess normality. due to the exposed group exhibited non-normal distributions, the two-tailed Mann–Whitney U test was applied for group comparisons. Magnitude of the impact was quantified using Cohen's d. Pearson correlation coefficients were designed to study the relationships between comet variables, and the significance The minimum limit has been set at $p < 0.05$.

RESULTS AND DISCUSSION

Result

Comet Parameters in Workers Versus Controls

DNA damage across all measured endpoints was markedly higher among workshop employees (Table 1). Tail length was found to average 230.16 ± 12.00 μm in workers compared to 70.52 ± 1.15 μm in controls, representing a 3.3-fold difference. Tail DNA percentage increased 8.1-fold (9.16 ± 0.70% in workers versus 1.13 ± 0.05% in controls). Tail moment was elevated 9.8-fold (13.86 ± 1.12 in workers versus 1.42 ± 0.11 in controls), indicating substantial strand break accumulation.

Table 2. Comet assay outcomes in auto repair workers and office controls. P-values from Mann–Whitney U test; Cohen's d in parentheses.

Parameter	Controls (Mean ± SD)	Workers (Mean ± SD)	p-value (Cohen's d)
Tail length (μm)	70.52 ± 1.15	230.16 ± 12.00	< 0.001 (15.39)
Tail DNA (%)	1.13 ± 0.05	9.16 ± 0.70	< 0.001 (13.34)
Tail moment	1.42 ± 0.11	13.86 ± 1.12	< 0.001 (12.87)

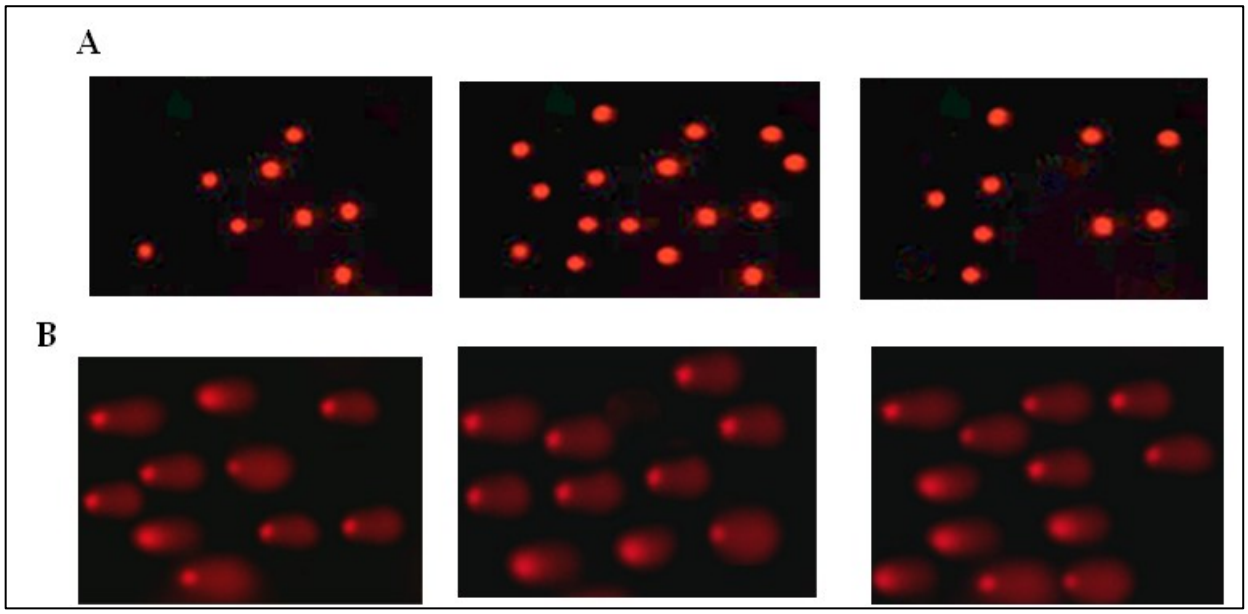

Figure 1. Images of the alkaline comet assay for peripheral blood lymphocytes. In control subjects (A), predominantly intact nucleoids with minimal comet tails are observed, indicating low levels of DNA strand breaks. In contrast, auto repair workers (B) have long, bright comet tails with more DNA in the tail, which shows that they have a lot of DNA damage.

Statistical Comparisons and Correlations

Highly significant differences for every parameter were confirmed by Mann–Whitney U tests ($p < 0.001$; Table 2). Effect sizes were found to range from 12.87 to 15.39, demonstrating large genotoxic effects associated with occupational metal contact. Among workers, comet measures were strongly intercorrelated (Pearson $r = 0.65–0.85$, all $p < 0.001$), indicating coherent damage patterns consistent with prior biomonitoring literature [11], [13], [14].

Discussion

Al-Diwaniyah auto workers showed 3- to 10-fold increases in DNA damage indices, with effect sizes exceeding 12. These findings point directly to chronic metal exposures from oils, greases, paints, and welding fumes prevalent in repair workshops.

Sources and Mechanisms of Metal Genotoxicity

Several mechanisms for the entry of heavy metals into the work environment have been identified, including the use of engine oils and lubricants, greases, paints, and corrosion inhibitors, which contain lead and zinc compounds that volatilize and become available for dermal absorption and inhalation, respectively [1], [3], [4]. Similarly, paints and corrosion inhibitors contain hexavalent chromium and lead, and aerosolized particles, which contain large amounts of heavy metals, are produced during the spray application of paints [3], [4]. Welding and soldering activities also produce cadmium and chromium, which are components of alloys and coatings, respectively, and particles of cadmium and chromium are produced during the abrading of brake and clutch liners, respectively.

Once inside the body, heavy metals induce oxidative stress through a variety of mechanisms, including the generation of reactive oxygen species (ROS), depletion of antioxidant defenses, and disruption of DNA repair mechanisms [5], [7]. For instance,

lead and cadmium induce oxidative stress through the generation of ROS, depletion of glutathione, and disruption of DNA repair mechanisms, including base excision repair, nucleotide excision repair, and DNA replication and transcription, which allow DNA breaks and oxidized bases to persist in the genome [5], [6], [19], [20], [21], [22], [23]. Similarly, the reduction of hexavalent chromium, which is contained in paints and corrosion inhibitors, to trivalent chromium also generates ROS and forms DNA adducts that interfere with DNA replication and transcription [7], [9], [24]. Zinc, although less damaging, also has the potential for inducing oxidative stress by being displaced from antioxidant enzymes, which displace other metals, thereby inducing oxidative stress.

Similarly, studies conducted in Pakistan, Nigeria, and Egypt, where workers in automobile factories were monitored for heavy metals, showed that workers in these factories had 2- to 8-fold increases in DNA damage, which was consistent with increased levels of lead, cadmium, and chromium in the blood and urine, respectively [8], [15], [19], [20]. Automotive emissions and polycyclic aromatic hydrocarbon exposure constitute additional genotoxic hazards in repair environments and other occupational settings [25], [26], [27], while oxidative-stress biomarkers can provide complementary evidence for interpreting occupational DNA damage [28].

Implications for Worker Health and Policy

Prompt action in Iraqi auto repair settings is therefore called for. Effective measures are considered to include the installation of local exhaust ventilation near welding and painting zones, replacement of metal-heavy materials where possible (such as with water-based paints and low-metal lubricants), and consistent use of respirators (P100-rated) together with dermal barriers like nitrile gloves and coveralls [3], [4], [21]. The comet assay's sensitivity, minimal sample requirement, and ability to reveal preclinical damage make it well-suited for routine monitoring in this occupational group [11], [13], [14]. A more complete exposure–effect profile can be obtained, and interventions can be better evaluated, by pairing comet data with blood or urine metal measurements and oxidative stress markers (8-OHdG, MDA, GSH) [11], [13], [21], [28].

CONCLUSION

Fundamental Finding: Severe DNA damage 3-10-fold elevations in comet assay parameters with effect sizes exceeding 12 was observed in auto repair workers in Al-Diwaniyah. **Implication:** These genotoxic outcomes, driven by oxidative stress from lead, cadmium, chromium, and zinc, are taken to require immediate workplace improvements (ventilation upgrades, material substitutions, protective equipment enforcement) together with regular comet-based biomonitoring. **Limitation:** Linked to ongoing metal exposures from lubricating oils, greases, paints, and welding activities. **Future Research:** Risk assessments can be sharpened, and tailored prevention strategies for this vulnerable worker population can be supported, by integrating biological metal measurements, oxidative stress markers, and DNA repair genotyping in future studies.

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