RESEARCH ARTICLE

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Impact of environmental pollutants in oil sector workers on some immunological parameters (cardiovascular, genetics, health): A critical analysis

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ABSTRACT This study investigated the hematological, inflammatory, and genetic responses in oil sector workers exposed to long-term and short-term environmental toxins. A total of 300 participants were included: 100 with long-term exposure, 100 with short-term exposure, and 100 controls. Blood samples were collected after overnight fasting for hematological assays, DNA extraction, malondialdehyde (MDA) assays, glycemic determination, and interleukin-6 (IL-6) analysis. The TP53 codon 72 (rs1042522) polymorphism was genotyped using RFLP-PCR. Results revealed a significant decrease in hemoglobin concentration and a significant increase in erythrocyte sedimentation rate (ESR) in both exposed groups compared to controls. IL-6 levels were significantly elevated in both exposure groups, with the Arg/Arg genotype of TP53 codon 72 being more prevalent in the long-term exposure group (52%) compared to controls (5%). Workers with the Arg/Arg genotype exhibited significantly higher IL-6 concentrations than those with Pro/Pro or Arg/Pro genotypes in both exposure groups. The findings suggest that the TP53 Arg/Arg genotype may increase susceptibility to environmental toxins, highlighting the role of genetic factors in individual responses to occupational exposures. This study underscores the importance of genetic screening and monitoring in workers exposed to environmental hazards.

KEYWORDS TP53 polymorphism, oil sector worker, MDA, pollution, cardiovascular, genetics, health

1. INTRODUCTION

Air pollution, which has a significant toxicological effect on human health and the atmosphere, has been a major issue in recent decades. Pollution sources range from small units such as tobacco and natural sources, like volcanic activity, to significant quantities of pollutants from vehicle engines and manufacturing activities [1].

Chronic exposure to environmental pollutants, particularly those encountered in industrial settings, has been increasingly recognized as a significant public health concern. Among these, oil pollution stands out due to its widespread occurrence and the diverse range of toxic substances it contains. Workers in the oil industry, including those involved in drilling, refining, and transportation, are often exposed to a complex mixture of hydrocarbons, heavy metals, and other contaminants, potentially leading to a variety of adverse health effects. Previous studies have documented the respiratory, dermatological, and systemic impacts of such exposure, yet the underlying mechanisms and the role of genetic susceptibility remain poorly understood [2].

One of the key areas of interest in understanding the health impacts of oil pollution is the role of genetic polymorphisms in modulating individual responses to toxic exposure. The TP53 gene, known for its critical function in regulating the cell cycle and apoptosis, has been implicated in various environmental and occupational health studies. Polymorphisms in TP53 may influence susceptibility to cancer and other chronic diseases in exposed populations. However, the extent to which these genetic variations contribute to the health risks associated with long-term oil pollution exposure has not been thoroughly investigated [3].

This study aims to fill this gap by exploring the association between chronic oil pollution exposure and a range of health outcomes, with a particular focus on the role of TP53 gene polymorphisms [4]. We hypothesize that workers with certain TP53 polymorphisms may exhibit higher susceptibility to the adverse effects of oil pollution, as reflected in both clinical symptoms and alterations in hematological and biochemical parameters. The study aimed to assess the prevalence of respiratory, dermatological, and systemic health effects among workers chronically exposed to oil pollution and to investigate the association between oil pollution exposure and changes in hematological and biochemical markers, as well as to determine the role of TP53 gene polymorphisms in modulating the health effects of chronic oil pollution exposure.

2. MATERIALS AND METHODS

The study involved a cohort of 300 workers from the occupational service of the oil sector, attending between January 1, 2022, and December 31, 2023. The participants were categorized into three distinct groups: a long-term exposure group consisting of 100 employees with direct and prolonged exposure to pollution, a short-term/indirect exposure group comprising 100 administrative employees with indirect or short-term exposure to pollution, and a control group of 100 employees with no exposure to pollution. Oil stations were randomly selected, with a preference for locations housing a higher number of workers potentially affected by pollution. The inclusion criteria required participants to be actively employed in the oil sector with documented exposure to air pollution, while the exclusion criteria ruled out individuals undergoing treatment with antibiotics or antiviral medications, and those with hyperthyroidism, hypothyroidism, severe renal failure, chronic hepatic diseases, malignant diseases, autoimmune diseases, lung cancer, or chronic obstructive pulmonary disease (COPD).

The study protocol received approval from the Scientific and Ethical Committee of the Western Health Area at the College of Medicine, Babylon University. Informed consent was obtained from all participants after a comprehensive explanation of the study's objectives. Participants also completed a specially designed questionnaire.

Blood samples were collected from all participants after overnight fasting. Two milliliters of blood were collected in an EDTA tube for hematological assays and DNA extraction, while three milliliters were collected in a gel tube for serum isolation for interleukin (IL) and malondialdehyde (MDA) assays. Glycemic indices were determined following standard protocols. For serum human IL-6 estimation, the samples were allowed to clot at room temperature for 10-20 minutes and were then centrifuged at 2000-3000 RPM for 20 minutes. The enzyme-linked immunosorbent assay (ELISA) kit, precoated with a human IL-6 antibody, was used according to the manufacturer's instructions.

Genomic DNA was extracted from blood samples using the G-spin Total DNA extraction kit (Intron) following the manufacturer's protocol. Polymerase Chain Reaction (PCR) was conducted using primers specific to TP53 codon 72 (rs1042522), designed with Primer-BLAST software and purchased from Bioneer, Korea (Table 1) [5]. Stock solutions of 100 pmol/µl were made and diluted to working solutions of 10 pmol/µl. PCR conditions such as annealing temperature, amplification cycles, and DNA and primer concentrations were optimized and presented in Table 2. RFLP-PCR analysis was performed by digesting the PCR products with the BstUI enzyme, and the resulting restriction fragments were separated by electrophoresis on a 2% agarose gel and visualized under UV light. Concordance was maintained by retesting more than 10% of the samples, with a 100% match rate.

Agarose gel electrophoresis was performed by preparing a gel with 1.5 g of agarose dissolved in 100 ml of 1x TBE buffer (pH 8). Ethidium bromide was added to the cast gel, and samples were loaded into wells. Electrophoresis was conducted, and the bands were visualized using a UV transilluminator and documented with a digital camera. Data analysis was conducted using SPSS version 26 (IBM, US). Scale variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. The Student's t-test was employed for comparisons between two groups, and one-way ANOVA was utilized for comparisons among three or more groups. Odds ratios (OR) with 95% confidence intervals (CI) were calculated to assess treatment response. Bivariate correlation tests were used to evaluate the association between SNP and diabetic variables, with correlation coefficients (R) indicating the strength of the relationship.

TABLE 1. Primer sequences of P53codon72 " rs1042522"

P53codon72" rs1042522"	F R	Forward primer "5 GCTCTTTTCACCCATCTACAG -3" Reverse primer "5 TGAAGTCTCATGGAAGCCAGC -3"	279 bp

TABLE 2. Mix reaction for genotyping of P53codon72 " rs1042522"in Polymerase chain

Component	Volume (µl)
Forward primer	Volume (µl)
Reverse primer	1.25
DNA template	1.25
Deionized water	5
Premix	12.5

3. RESULTS

The study evaluated clinical signs and symptoms in workers exposed to oil pollution. A range of physical symptoms was noted (Figure 1), including sneezing (28.6%), chest pain (25.5%), wheezing (21.1%), and coughing (19.9%). Some workers also exhibited delayed growth and a bump at the root of the nose (13.9%), potentially related to the accumulation of petroleum derivatives, which may lead to a decline in lung capacity and an increased risk of chronic illnesses. Dermatological symptoms were also prevalent among the exposed workers, with observations of dry skin, a distinctive bronze color, pigmentation on the extremities, severe itching, and visible scars (Figure 2). These findings align with elevated ferritin levels, consistent with previous research. Nail changes were common, with 80% of workers exposed long-term showing hard nails with transparent cracks, and 7% experiencing nail atrophy. Additionally, 40% of long-term exposed workers exhibited eye redness and congested capillaries, with a few showing swelling under the eyeball. Hair characteristics remained mostly unaffected, although 5% of the workers had white hair, possibly due to hyperpigmentation.

The population studied included 100 participants each in the long-term exposure, short-term exposure, and control groups, with age ranges from 29 to 59 years (Table 3). Long-term exposure was most prevalent in the 55-59 age group, suggesting an increased risk of complications such as iron and steel foundry overload in older workers. Blood parameter

tests revealed no significant differences in blood group distribution between the long-term, short-term, and control groups ($P > 0.05$), with O+ being the most common blood group among exposed workers, though not statistically significant.

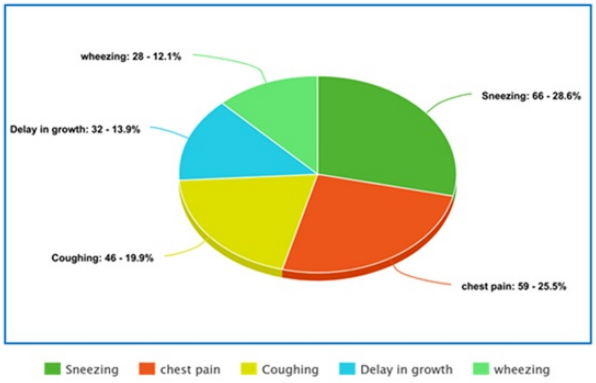

FIGURE 1. Side effects of occupational lung disease exposure to enviromental pollution in oil sectors

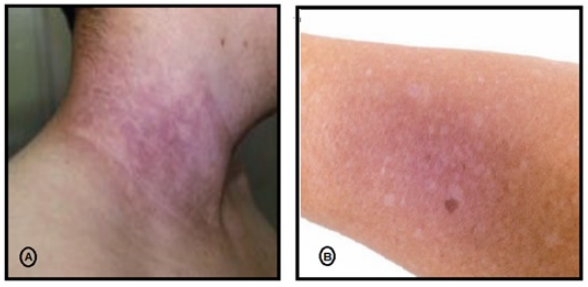

FIGURE 2. Showed Skin pigmentation is a common condition that can be triggered in workers oil sectors A/ represented hyperpigmentation in neck and B/ represented pigmentation of skin in arm

TABLE 3. Distribution of oil sector workers according to age

Age (Years)	Oil sector workers		Control
	Long exposure pollution	Short exposure pollution	
25-29	27	17	25
35-39	19	25	8
45-49	22	19	40
55-59	32	39	27
Total	100	100	100

There was a significant decrease in hemoglobin concentration in both exposed groups compared to the control group ($P = 0.0135$). The long-term exposure group had a mean hemoglobin concentration of 20.77 ± 2.4 g/dL, the short-term exposure group had 17.777 ± 3.5 g/dL, and the control group had 13.207 ± 3.4 g/dL. Erythrocyte sedimentation rate (ESR) showed a significant increase in both exposed groups compared to the control group ($P = 0.042$), with the long-term exposure group recording 50.7 ± 3.8 mm/hr, the short-term group 43.9 ± 4.6 mm/hr, and the control group 13.5 ± 4.3 mm/hr. Additionally, a significant increase in total white blood cell (WBC) count was observed among the exposed groups compared to the control group ($P = 0.0023$). The long-term exposure group had a WBC count of 8.72 ± 1.2 × 10^3/mL, the short-term exposure group 7.373 ± 1.6 × 10^3/mL, and the control group 5.49 ± 0.85 × 10^3/mL. Significant differences were also observed in other hematological parameters, including lymphocytes, neutrophils, MID, MCV, MCH, MCHC, and PLT between the exposed and control groups (Table 4).

TABLE 4. Blood counts and related hematological parameters measurements against long and short term exposure pollution with control health

N.	The test	Means± SD			P-value
		Control	Short exposure	Long exposure	
1	WBC (103 /mL)	5.59	7.373	8.72	0.00023
2	LYM (103/ mL)	32.687	47.453	53.029	0.0387
3	NEUT (103/ mL)	40.86	66.867	69.836	0.019
4	MID (103/ mL)	7.047	9.057	10.881	0.066
5	MCV (fL)	86.194	95.176	125.755	0.039
6	MCH (pg)	27.219	27.787	38.646	0.0301
7	MCHC (g/dL)	31.401	32.401	44.745	0.0054
8	PLT (103 /mL)	140.203	148.073	201.385	0.0128

Biochemical parameters showed that interleukin 6 (IL-6) levels were significantly elevated in both exposure groups compared to the control group ($P < 0.05$). The long-term exposure group had a mean IL-6 concentration of 203.5 ± 15.98 ng/mL, the short-term exposure group 88.06 ± 15.31 ng/mL, and the control group 49.52 ± 9.35 ng/mL (Figure 3). Malondialdehyde (MDA) concentrations were also significantly higher in the exposed groups ($P < 0.05$), with the long-term exposure group recording 366.4 ± 20.03 pg/mL, the short-term exposure group 189.55 ± 12.16 pg/mL, and the control group 58.52 ± 7.36 pg/mL (Figure 4).

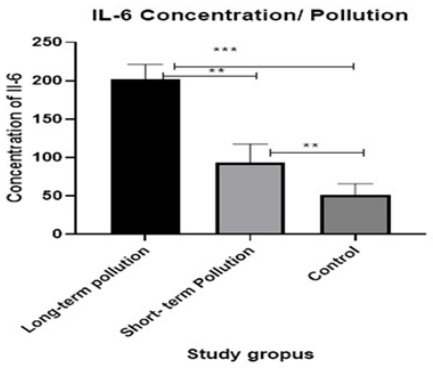

FIGURE 3. Concentration of IL-6 in long and short term exposure pollution and control groups

Analysis of the TP53 gene polymorphism at codon 72 (rs1042522) revealed three genotypes: Pro/Pro, Arg/Arg, and Arg/Pro. Significant differences in genotype distribution were found between the exposed and control groups. The Arg/Arg genotype was more prevalent in the long-term exposure group (52%) compared to controls (5%), with an odds ratio (OR) of 0.204 (95% CI 0.114–0.337, $P < 0.0001$). A similar pattern was observed in the short-term exposure group, where the Arg/Arg genotype was present in 36% of workers compared

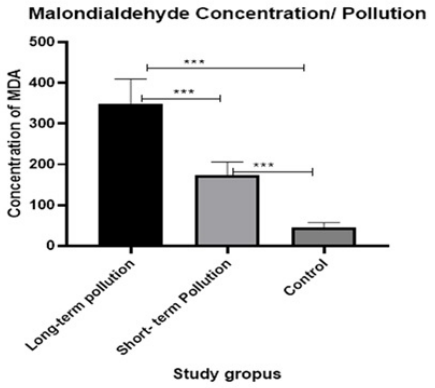

FIGURE 4. **Malondialdehyde (MDA) concentration in studies groups**

to 5% in the control group (OR = 4.21, 95% CI 2.71–6.43, P < 0.01) (Table 5 and 6).

Moreover, IL-6 concentrations were significantly higher in workers with the Arg/Arg genotype compared to those with the Pro/Pro or Arg/Pro genotypes in both long-term and short-term exposure groups (P < 0.05) (Table 7). On the other hand, no significant association was found between IL-6 levels and TP53 polymorphism in the control group. MDA concentrations also showed significant differences with TP53 polymorphism, with higher levels observed in the Arg/Arg genotype (Table 8).

4. DISCUSSION

The findings from this study highlight the severe health impacts of long-term exposure to oil pollution, emphasizing the multifaceted nature of these risks and the urgent need for improved protective measures within the industry. The high prevalence of respiratory symptoms, including sneezing, chest pain, wheezing, and persistent coughing among exposed workers, is particularly concerning. These symptoms suggest significant respiratory distress due to airborne exposure to volatile organic compounds and particulate matter associated with oil pollution. This is consistent with existing literature linking chronic exposure to petroleum-derived pollutants with adverse respiratory outcomes, such as an increased incidence of chronic obstructive pulmonary disease (COPD), asthma, and other chronic respiratory conditions [6], [7].

Globally, air pollution has been associated with various health effects, including lung diseases (such as asthma, respiratory infections, COPD, and cancer), cardiovascular, and neurological disorders [8], [9]. Particulate matter (PM), especially PM2.5, is frequently investigated due to its ability to penetrate alveoli and induce systemic effects [10]. Furthermore, air pollution exposures are related to immune-mediated diseases like asthma and allergy [6], [11], and autoimmune diseases such as systemic lupus erythematosus [12] and rheumatoid arthritis [8]. The biological mechanisms underlying these associations include oxidative stress, inflammatory responses, tissue damage, vascular alteration, and DNA damage [11], [13].

The dermatological symptoms observed in the exposed groups—such as dry skin, hyperpigmentation, and severe itching—support the hypothesis of systemic absorption of toxic substances from oil pollution. Skin contact with oil and its derivatives has been documented to cause dermatological issues, indicative of deeper systemic involvement. The systemic absorption of these toxicants likely contributes to the observed hematological and biochemical changes, suggesting widespread physiological stress and potential damage beyond the respiratory and dermal systems [14].

There were significant reductions in hemoglobin levels in both long-term and short-term exposure groups, indicating potential bone marrow suppression or direct hemolytic effects due to chronic exposure to oil pollutants. This decline, coupled with elevated erythrocyte sedimentation rate (ESR) and white blood cell (WBC) counts, points to a sustained inflammatory state, a recognized risk factor for chronic diseases, including cardiovascular and metabolic disorders [15]. The elevated WBC counts suggest an ongoing immune response to prolonged exposure to harmful substances, including volatile organic compounds and polycyclic aromatic hydrocarbons, contributing to increased ESR and systemic inflammation, which may predispose individuals to chronic health conditions beyond immediate symptoms [16], [17].

Biochemical analyses reveal significantly elevated levels of Interleukin 6 (IL-6) and malondialdehyde (MDA) in the exposed groups. IL-6, a key pro-inflammatory cytokine, is central to the immune response and is often elevated in chronic inflammatory conditions. The increased IL-6 levels among exposed workers suggest that oil pollution induces a broader inflammatory response with potential long-term health implications [18], [19]. Elevated MDA levels further indicate increased oxidative stress, reflecting the depletion of antioxidant defenses due to continuous exposure to toxic substances. Oxidative stress leads to cellular damage, lipid peroxidation, and DNA damage, which are implicated in chronic diseases, including cancer and cardiovascular disorders [20], [21].

Of particular interest is the significant association found between the TP53 gene polymorphism at codon 72 and exposure to oil pollution. The increased prevalence of the Arg/Arg genotype among workers in the long-term exposure group is especially noteworthy, as it suggests a potential genetic predisposition to the harmful effects of oil pollutants. The TP53 gene, known for its role as a tumor suppressor, plays a crucial role in regulating the cell cycle and preventing the proliferation of damaged cells. The Arg/Arg genotype has been associated with higher susceptibility to cancer and other inflammatory diseases, as it may alter the tumor suppressor function of the TP53 protein, reducing its ability to respond effectively to DNA damage [22]–[24].

The elevated IL-6 levels observed in workers with the Arg/Arg genotype further suggest that this genetic variant may exacerbate the inflammatory response to environmental toxins, potentially increasing the risk of developing chronic

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TABLE 5. Genotype and Allele Frequencies of the Polymorphism rs1042522 gene TP53 among occupational long exposure of pollution

TP53 (rs1042522)	Healthy (n = 100) No (%)	Pollution (n = 100) No (%)	Odds ratio (95% CI)	P-value
CC (Pro/Pro)	62 (62%)	25 (25%)	1	-
CG (Pro/Arg)	33 (33%)	23 (23%)	0.57 (0.28–1.137)	0.128
GG (Arg/Arg)	5 (5%)	52 (52%)	0.038 (0.009–0.098)	<0.0001*
CG (Pro/Arg) + GG (Arg/Arg)	38 (38%)	75 (75%)	0.204 (0.114–0.337)	<0.0001*
TP53 alleles				
C	157 (78.5%)	73 (36.5%)	-	
G	43 (21.5%)	127 (63.5%)	0.157 (0.101-0.245)	<0.0001*

TABLE 6. Genotype and Allele Frequencies of the Polymorphism rs1042522 gene TP53 among occupational short exposure of pollution

TP53 (rs1042522)	Healthy (n = 100) No (%)	Pollution (n = 100) No (%)	Odds ratio (95% CI)	P-value
CC (Pro/Pro)	62 (62%)	29 (29%)	1	-
CG (Pro/Arg)	33 (33%)	35 (35%)	2.26 (1.18–4.33)	0.128
GG (Arg/Arg)	5 (5%)	36 (36%)	15.3 (5.44–43.3)	<0.01*
CG (Pro/Arg) + GG (Arg/Arg)	38 (38%)	71 (71%)	3.99 (2.21-7.215)	<0.0001*
TP53 alleles				
C	157 (78.5%)	93 (46.5%)	-	
G	43 (21.5%)	107 (53.5%)	4.21 (2.71-6.43)	<0.0001*

TABLE 7. Relationship between three groups of il-6 concentration with Polymorphism rs1042522 gene TP53

Genotype	No. of genotype Frequency rs1042522	Concentration of IL-6 Pg/ml Mean ±SD
Long exposure of pollution Groups	CC (Pro/Pro)	84.2± 5.2
	CG (Pro/Arg)	123.6±8.3
	GG (Arg/Arg)	197.5± 19.5
P value		0.001
Short exposure of pollution Groups	CC (Pro/Pro)	27.6± 3.2
	CG (Pro/Arg)	62.9 ± 3.28
	GG (Arg/Arg)	98.6± 11.3
P value		0.035
Healthy Groups	CC (Pro/Pro)	48.6± 5.7
	CG (Pro/Arg)	41.39 ± 6.2
	GG (Arg/Arg)	39.8± 5.1
P value		0.48

TABLE 8. Relationship between three groups of MDA concentration with Polymorphism rs1042522 gene TP53

Genotype	No. of genotype Frequency rs1042522	Concentration of MDA Pg/ml Mean ±SD
Long exposure of pollution Groups	CC (Pro/Pro)	102.4± 16.5
	CG (Pro/Arg)	289.5±17.7
	GG (Arg/Arg)	374.6 ± 18.3
P value		0.001
Short exposure of pollution Groups	CC (Pro/Pro)	44.15± 11.5
	CG (Pro/Arg)	97.4 ± 15.7
	GG (Arg/Arg)	184.8± 10.5
P value		0.035
Healthy Groups	CC (Pro/Pro)	49.7± 8.9
	CG (Pro/Arg)	48.05 ± 15.5
	GG (Arg/Arg)	59.4± 10.4
P value		0.69

diseases, including cancer. These findings align with a growing body of evidence that suggests genetic polymorphisms can modulate an individual's response to environmental exposures, leading to differential health outcomes.

The implications of these findings are profound. First, they underscore the critical importance of genetic screening in occupational health settings, particularly for workers exposed to high levels of environmental pollutants. Identifying individuals with high-risk genotypes, such as the TP53 Arg/Arg variant, could enable targeted interventions aimed at mitigating the adverse health effects of long-term exposure. For instance, workers identified as being at higher genetic risk could be provided with enhanced protective measures, more frequent health screenings, and early interventions to reduce their exposure and monitor for the development of chronic diseases. Additionally, the observed hematological and biochemical changes suggest that routine monitoring of these parameters could serve as early indicators of health deterioration in exposed workers, allowing for timely medical intervention before more severe health outcomes develop [23].

5. CONCLUSION

In conclusion, this study provides compelling evidence of the significant health risks associated with long-term exposure to oil pollution, particularly in relation to respiratory health, systemic inflammation, and genetic susceptibility. The findings highlight the need for stringent occupational health regulations, including the implementation of protective measures, regular health screenings, and targeted interventions for workers identified as being at higher genetic risk. Future research should focus on the long-term health outcomes of workers with high-risk genotypes and investigate potential interventions to mitigate the effects of chronic exposure to oil pollutants. Moreover, there is a need for further studies to explore the molecular mechanisms underlying the observed associations, which could lead to the development of novel therapeutic strategies to protect and improve the health of workers in this industry. These efforts are crucial not only for protecting the health of current workers but also for informing policy and regulatory decisions that could prevent future generations from experiencing the same risks.

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