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Evaluation of the effects of occupational exposure to toxic gases on respiratory system biomarkers among oil company workers in Basra, Iraq

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Occupational exposure to toxic gases in oil industry settings poses significant risks to respiratory health, making the assessment of biomarkers essential for early detection of pulmonary and systemic effects among worker. The aim of this study is to evaluate the effects of occupational exposure to toxic gases on respiratory system biomarkers among oil company workers in Basra, Iraq. The study was a case-control investigation at Basra General Hospital involving 130 petroleum workers from three sites and 56 healthy controls, aged 28–59 years, employed >5 years, excluding smokers or those with chronic diseases. Conducted from December 2024 to July 2025, blood samples were analyzed for CC16, α-GST, KIM-1, SP-A, LDH, and CRP using ELISA, photometric, and turbidimetric assays to assess inflammatory and organ-specific biomarkers. The study showed no significant differences in age or BMI across groups or work sites. Occupational exposure to toxic gases was associated with significant increases in inflammatory markers (CRP, LDH), renal biomarkers (α-GST, KIM-1), and respiratory marker SP-A, while CC16 remained unchanged. Exposure duration and work location affected some biomarkers, particularly α-GST and SP-A. Risk assessment indicated that elevated LDH, α-GST, KIM-1, and SP-A significantly increased the likelihood of exposure, highlighting the impact of toxic gases on cellular, renal, and respiratory functions. Occupational exposure to toxic gases in oil workers significantly elevates inflammatory, renal, and respiratory biomarkers, likely due to oxidative stress and cellular damage induced by inhaled toxins. CC16 remains unaffected, suggesting selective organ-specific responses to exposure.

Keywords: occupational exposure; toxic gases; respiratory biomarkers; renal biomarkers; inflammatory markers; oil workers.

Introduction

Frequent exposures of workers to noxious gases at workplaces, especially in the petroleum industry, has been a significant health issue of concern in the industrial environment, as employees are regularly exposed to various volatile organic substances, hydrocarbons, and chemical wastes. Such exposures may cause serious negative outcomes on various organ systems, such as the respiratory, hepatic, and renal ones, and lead to both acute and chronic health disorders (Benson et al., 2021; Otitolaiye et al., 2022). Because of the recurrent and long-term exposure, petroleum workers are particularly susceptible to subclinical and clinical alterations that cannot be easily seen, hence the need to find ways of early diagnosis and following through on biological indicators of organ dysfunction (Otitolaiye et al., 2022).

One area that is highly vulnerable is the respiratory system when affected by the inhaled toxicants. Repeated exposure to hydrocarbons and particulate matters in oil production fields may trigger oxidative stress, inflammation, and epithelial damage in the lungs, which may result in chronic respiratory infections, low pulmonary functionality, and increased vulnerability to infections (Singh et al., 2021). C-reactive protein (CRP), surfactant protein A (SP-A) and Clara cell protein 16 (CC16) are also becoming important biomarkers of systemic inflammation and integrity of epithelial cells of the lungs (Mocelin et al., 2022). CRP is a nonspecific acute-phase protein that represents systemic inflammatory reaction to the exposure to environmental pollutants, SP-A and CC16 are released by the airway and alveolar epithelial cells, respectively, and are sensitive indicators of pulmonary damage and defenses (Straumfors et al., 2018; Anggraini et al., 2022).

Besides its respiratory effects, occupational exposure to the toxicants of petroleum can affect the renal and hepatic functions. Enzymatic biomarkers, such as α-glutathione-S-transferase (0-GST) and Kidney Injury Molecule-1 (KIM-1), are also becoming commonly used in occupational health studies to monitor the early tissue damage. 0-GST is expressed in the hepatocytes and is a marker of liver detoxification response and hepatocellular stress, with KIM-1 being an early biomarker of tubular injury highly sensitive to nephrotoxicity (Rahimi et al., 2020; Tonbra Egoro et al., 2024). The high concentration of these biomarkers in the exposed individuals is an indicator of the cumulative toxicity of the hydrocarbons and its metabolites, which is important in the realization of the need to combine renal and hepatic surveillance in occupational health surveillance programs (Polyong et al., 2024).

Basra, Iraq is a very sensitive area to petroleum production in the Middle East, with many oil mining and refining plants. Employees at these plants are regularly exposed to complicated blends of volatile solvents, such as hydrogen sulfide, benzene, toluene, and other hydrocarbons, in less-than-optimal conditions of ventilation, and with uneven compliance with protective provisions. Although the occupational hazards have been identified, lack of studies on the combined effect of the exposures on respiratory biomarkers and systemic health parameters is still evident in Iraq. The bulk of the past literature has centered on environmental surveillance or the pulmonary functional assessments, and little has been done in integrating biochemical and immunological measures that would give early indicators of subclinical organ damage (Mallon et al., 2019; Alghara et al., 2022).

The current study is intended to close this gap by assessing the effects of occupational exposure to toxic gases on respiratory and systemic biomarkers in oil company workers of Basra. The study seeks to present a detailed evaluation of the health risks with exposure by including various sites, i.e., Al-Masfa, Al-Barjisia, and Al-Tuba and stratifying the participants based on the period of exposure and age. The biomarkers of interest in the investigation are CRP, LDH, alpha-GST, KIM-1, SP-A, and CC16 that collectively indicate systemic inflammation, hepatic detoxification, renal tubular integrity and pulmonary epithelial responses. This research represents an integrative approach that enables early subclinical alterations to be identified that can lead to an overt disease. This approach can be useful in occupational health surveillance and prevention factors (Scammell et al.,

2019; Calabrese et al., 2024). Assessing the biochemical and physiological conditions of workers that are exposed to toxic gases can not only improve the health and safety of the workers but also govern regulation policy, monitoring of the workplace and the structure of intervention programs to minimize the occupational risk. Due to the economic impact of petroleum industry in Basra and the use of manual labor, such research offers vital evidence to guide the practice of occupational health, improve the level of protection and eventually decrease the work-related morbidity (Raja et al., 2019; Benson et al., 2021).

Finally, it is necessary to estimate respiratory biomarkers, as well as systemic biomarkers in petroleum workers who are under the influence of toxic gases, to identify the occupational risks at an initial stage. The research gap that is bridged in the present study concerns the relations between exposure, biochemical alterations, and possible health hazards in Iraq, which will help to develop evidence-based occupational health interventions and maintain the safety of workers in the high-risk industrial workplaces.

Material and methods

The study was approved by the Human Ethics Committee of Basra General Hospital, Everyone who took part in the study was informed about it and asked to sign a consent form. Each patient was also guaranteed that his information would be kept private.

The study was a case-control study at Basra General Hospital in volving a total of 130 petroleum workers and 56 healthy controls. The exposed group included the employees in the Al-Masfa Refinery (n = 52), the Al-Barjisia Field (n = 38), and the Al-Tuba Field (n = 40) age 28–59 years. All test subjects were eligible in terms of inclusion criteria that required the men to have been continuously employed over a period of more than five years, and none of the test subjects was rejected due to the exclusion criteria, which encompassed smoking, cancer or chronic ailments. The research was conducted between the months of December 2024 and July 2025. The blood of all the participants was sampled to determine the levels of certain biochemical and respiratory biomarkers. To measure concentrations of human Clara cell protein 16 (CC16), glutathione S-transferase α (3-GST), kidney injury molecule 1 (KIM-1) and surfactant protein A (SP-A), ELISA kits were used based on the following principles: the stop solution transforms the blue color of each protein to yellow, and optical density was measured at 450 nm. Also, the lactate dehydrogenase (LDH) activity was photometrically measured by the conversion of L-lactate into pyruvate with NADH formation depending with the activity of the enzyme, and C-reactive protein (CRP) was quantified by a particle-enhanced turbidimetric assay on the Cobas Integra system, where human CRP reacts with latex particles that have been coated with monoclonal anti-CRP antibodies and the precipitate is quantified at 552 nm. Such an approach allowed us to assess in detail both inflammatory and organ-specific biomarkers concerning occupational exposure to toxic gases.

Data were analyzed using SPSS version 26.0. Normality of continuous variables was checked with the Shapiro-Wilk test and presented as mean ± SD. Comparisons between exposed and control groups, and among site-specific groups (Al-Masfa, Al-Barjisia, Al-Tuba), were performed using independent t-tests or one-way ANOVA with Tukey's post-hoc test for multiple comparisons. Binary logistic regression was used to calculate odds ratios (OR) and 95% confidence intervals (CI) for the overall and site-specific exposed groups, including age, BMI, FBG, urea, creatinine, ALT, AST, CRP, LDH, α-GST, KIM-1, SP-A, and CC16 as predictors. Spearman correlation assessed linear relationships, and significance was set at $P < 0.05$.

Results

The study results presented in Table 1 showed that workers exposed to toxic gases in the oil sector (n = 130) did not differ from the control group in age (40.9 ± 7.7 vs. 39.3 ± 9.9 years, P = 0.273) or body mass index (BMI) (27.4 ± 3.1 vs. 26.5 ± 2.9 kg/m², P = 0.056). However, the exposed group showed a significant increase in CRP (mean 2.65 ± 3.52 compared to 1.26 ± 1.15, P = 0.007) for the control group and LDH (mean 214.6 ± 55.1 compared to 153.6 ± 38.1, P = 0.0001) for the control group. α-GST was also significantly elevated in the exposed group (248.6 ± 197.3) compared to the control group (147.6 ± 89.1, P = 0.001), as was KIM-1 (75.8 ± 17.7) compared to control (58.0 ± 18.4, P = 0.0001). Regarding respiratory markers, SP-A was elevated in the petroleum workers (171.0 ± 71.6) compared to control (133.3 ± 45.2, P = 0.001), while no difference was observed in CC16 between the two groups (44.1 ± 11.9) compared to (44.0 ± 12.4, P = 0.975). These results indicate a clear effect of occupational exposure on inflammatory, respiratory, and renal markers.

Table 1
Mean levels of demographic variables
and biomarkers among study groups (mean ± SD)

Variables	All exposed petroleum workers (n =130)	Non-exposed control (n =56)	P-value
Age, year	40.9 ± 7.6	39.3 ± 9.8	0.273
BMI, kg/m²	27.4 ± 3.1	26.5 ± 2.9	0.056
CRP, mg/L	2.65 ± 3.52	1.26 ± 1.15	0.007
LDH, U/L	214.6 ± 55.1	153.6 ± 38.1	0.0001
α-GST	248.6 ± 197.3	147.6 ± 89.1	0.001
KIM-1	75.8 ± 17.7	58.0 ± 18.4	0.0001
SP-A	171.0 ± 71.6	133.3 ± 45.2	0.001
CC16	44.1 ± 11.9	44.0 ± 12.4	0.975

The results for the group of workers at the Al Masfa (Basra) Refinery exposed to toxic gases (n = 52, Table 2) showed no significant difference in age (38.6 ± 7.6) compared to control (39.3 ± 9.8 years) (P = 0.376) or body mass index BMI (27.3 ± 3.1), compared to control (26.5 ± 2.9 kg/m², P = 0.171). However, a significant increase in CRP was observed among the petroleum workers, with a mean of 2.16 ± 1.99 compared to 1.26 ± 1.15 (P = 0.006) in the control, as well as a significant increase in LDH, with a mean of 226.3 ± 59.2 compared to 153.6 ± 38.1 (P = 0.0001) in control. α-GST also increased to 191.9 ± 154.1 in the exposed group compared to 147.6 ± 89.1 in the control group, although this increase was not statistically significant (P = 0.080). KIM-1 showed a significant increase with a mean of 78.5 ± 19.2 compared to 58.0 ± 18.4 (P = 0.0001) in control. Regarding respiratory markers, SP-A increased significantly in the petroleum workers with a mean of 163.9 ± 73.8 compared to 133.3 ± 45.2 (P = 0.013) in control, while no difference was recorded in CC16, which was 41.2 ± 10.0 compared to 44.0 ± 12.4 (P = 0.210) in control. These results indicate a significant impact of occupational exposure in the refinery on some inflammatory, renal, and respiratory indicators.

Table 2
Mean values of demographic and biochemical indicators
in both study groups (mean ± SD)

Variables	Masfa Al-Basra exposed petroleum workers (n = 52)	Non-exposed control (n =56)	P-value
Age, year	38.6 ± 7.6	39.3 ± 9.8	0.376
BMI, kg/m²	27.3 ± 3.1	26.5 ± 2.9	0.171
CRP, mg/L	2.16 ± 1.99	1.26 ± 1.15	0.006
LDH, U/L	226.3 ± 59.2	153.6 ± 38.1	0.0001
α-GST	191.9 ± 154.1	147.6 ± 89.1	0.080
KIM-1	78.5 ± 19.2	58.0 ± 18.4	0.0001
SP-A	163.9 ± 73.8	133.3 ± 45.2	0.013
CC16	41.2 ± 10.0	44.0 ± 12.4	0.210

The study results shown in Table 3 comparing the exposed workers in the Barjisia area (n = 38) and the control group showed no significant differences in age (42.4 ± 8.4 vs. 39.3 ± 9.8 years, P = 0.094) or body mass index (BMI, 27.4 ± 2.7 vs. 26.5 ± 2.9 kg/m², P = 0.145). However, the biomarkers showed a significant increase in the exposed petroleum workers, with CRP levels reaching 3.65 ± 5.26 compared to 1.26 ± 1.15 (P = 0.002) in the control group, and LDH levels rising to 208.7 ± 43.8 compared to 153.6 ± 38.1 (P = 0.0001) in the control group. α-GST also showed a significant increase, with a mean of 277.8 ± 182.0 in the petroleum workers compared to 147.6 ± 89.1 (P = 0.0001) in the control group, in addition to an increase in KIM-1 to 76.0 ± 16.6 compared to 58.0 ± 18.4 (P = 0.0001) in the control group. Regarding respiratory markers, SP-A increased significantly,

with a mean of 186.4 ± 73.5 compared to 133.3 ± 45.2 (P = <0.0001) in control, while CC16 showed no significant difference between the two groups, at 44.8 ± 12.5 versus 44.0 ± 12.4 (P = 0.764). These data indicate clear effects of occupational exposure in Barjisia on inflammatory, renal, and respiratory markers.

Table 3
Mean values of demographic
and respiratory–system biomarkers in both groups (mean ± SD)

Variables	Al-Barjisia exposed petroleum workers (n = 38)	Non-exposed control (n = 56)	P-value
Age, year	42.4 ± 8.4	39.3 ± 9.8	0.094
BMI, kg/m^2	27.4 ± 2.7	26.5 ± 2.9	0.145
CRP, mg/L	3.65 ± 5.26	1.26 ± 1.15	0.002
LDH, U/L	208.7 ± 43.8	153.6 ± 38.1	0.0001
α-GST	277.8 ± 182.0	147.6 ± 89.1	0.0001
KIM-1	76.0 ± 16.6	58.0 ± 18.4	0.0001
SP- A	186.4 ± 73.5	133.3 ± 45.2	<0.0001
CC16	44.8 ± 12.5	44.0 ± 12.4	0.764

The analysis of workers exposed in the Al-Tuba region (n = 40) shown in Table 4 indicated that there were no important differences between them in age (42.3 ± 6.2 vs. 39.3 ± 9.8 years, P = 0.064) or BMI (27.6 ± 3.6 vs. 26.5 ± 2.9 kg/m^2, P = 0.102). But the exposed workers showed a strong increase in CRP (2.33 ± 2.85 vs. 1.26 ± 1.15 mg/L, P = 0.018) and LDH (205.1 ± 57.5 vs. 153.6 ± 38.1 U/L, P < 0.0001) together with an increase in renal biomarkers. There was also a significant difference in the pulmonary marker SP-A (165.6 ± 66.3 vs. 133.3 ± 45.2 ng/mL, P = 0.007) but no significant difference in CC16 (47.1 ± 13.0 vs. 44.0 ± 12.4 ng/mL, P = 0.255). Such results suggest that the occupational exposure in this region has negative impacts on inflammatory, renal and respiratory biomarkers.

Table 4
Mean values of demographic and respiratory-related biomarkers
in both study groups (mean ± SD)

Variables	Al-Tuba exposed petroleum workers (n = 40)	Non-exposed control (n =56)	P-value
Age, year	42.3 ± 6.2	39.3 ± 9.8	0.064
BMI, kg/m^2	27.6 ± 3.5	26.5 ± 2.9	0.102
CRP, mg/L	2.33 ± 2.85	1.26 ± 1.15	0.018
LDH, U/L	205.1 ± 57.5	153.6 ± 38.1	0.0001
α-GST	294.5 ± 243.0	147.6 ± 89.1	0.0001
KIM-1	72.2 ± 16.6	58.0 ± 18.4	0.000
SP-A	165.6 ± 66.3	133.3 ± 45.2	0.007
CC16	47.1 ± 13.0	44.0 ± 12.4	0.255

The regression analysis of the exposed workers of various ages shown in Table 5 revealed no significant difference in BMI ranging between 27.1 ± 4.2 in the 20–30 age group to 27.9 ± 3.5 in the 51–60 age group (P = 0.52). On the same note, there was no significant difference in CRP levels across age groups (2.51 ± 2.45 to 3.36 ± 3.98 mg/L, P = 0.618) and LDH levels (209.6 ± 31.6 to 217.8 ± 46.4 U/L, P = 0.569), whereas α-GST had significant difference across the age groups (220.2 ± 148.0. However, SP-A levels showed significant differences among age groups (P = 0.003), and their mean values were 174.3 ± 97.0, 171.3 ± 77.3, 170.6 ± 60.8 and 169.7 ± 70.8 ng/mL indicating statistical variance among age groups. There were no significant changes in the levels of CC16 (39.9 ± 9.2 to 46.2 ± 11.9 ng/mL, P = 0.999). In general, it can be proposed that these results indicate that the majority of vital and biochemical markers did not change significantly in relation to age, except for SP-A, which revealed significant change with aging.

Table 5
Comparison of mean biomarker levels
according to age categories (mean ± SD)

Variables	20–30 years (n = 14)	31–40 years (n = 51)	41–50 years (n = 49)	51–60 years (n = 16)	P-value
BMI,kg/m^2	27.1 ± 4.2	27.9 ± 3.2	27.9 ± 4.2	27.9 ± 3.5	0.520
CRP, mg/L	2.51 ± 2.45	2.17 ± 2.28	2.90 ± 4.50	3.36 ± 3.98	0.618
LDH, U/L	209.5 ± 31.6	217.8 ± 46.4	213.3 ± 63.4	212.6 ± 66.3	0.569
α-GST	285.0 ± 302.1	220.2 ± 148.0	238.5 ± 160.9	325.2 ± 292.1	0.957
KIM-1	63.2 ± 22.1	74.3 ± 19.4	75.8 ± 15.0	86.9 ± 10.3	0.206
SP-A	174.3 ± 97.0	171.3 ± 77.3	170.6 ± 60.8	169.7 ± 70.8	0.003
CC16	39.9 ± 9.2	46.2 ± 11.9	42.4 ± 12.6	45.2 ± 11.0	0.999

It was established (Table 6) that there was no significant difference in age and body mass index between groups of different exposure periods (age 33.8 ± 5.6 to 51.8 ± 4.7 years, P = 0.437; BMI 26.9 ± 3.7 to 29.1 ± 3.4, P = 0.125). Conversely, the longer the exposure, the higher the IgM inflammatory markers rose: CRP from 1.64 ± 1.79 in the 1–10 years exposure category to 5.41 ± 6.30 mg/L in the 31–40 years exposure category and LDH from 181.4 ± 48.7 in the 1–10 years exposure category to 221.9 ± 70.4 U/L (P = 0.0001) in the 21–30 years exposure category. Renal marker KIM-1 also significantly increased from 64.1 ± 20.7 in the 1–10 years exposure category to 90.4 ± 9.2 (P = 0.001) in the 31-40 years exposure category while SP-A increased significantly but moderately (146.6 ± 63.2 and 184.0 ± 70.9 in the 1–10 years and 31–40 years exposure categories, respectively, P = 0.047). CC16 or α-GST did not show any significant changes. These results reveal that the cumulative occupational exposure is linked to increased inflammatory, renal and respiratory biomarkers, but CC16 and α-GST are not affected significantly.

Table 6
Comparison of mean biomarker levels across different exposure periods (mean ± SD)

Variables	1–10 years (n = 40)	11–20 years (n = 64)	21–30 years (n = 21)	31–40 years (n = 5)	P-value
Age, years	33.8 ± 5.6 a	41.8 ± 5.4 b	48.9 ± 4.7 c	51.8 ± 4.7 d	0.437
BMI, kg/m^2	26.9 ± 3.7 a	28.9 ± 2.7 a	27.9 ± 5.0 a	29.1 ± 3.4 b	0.125
CRP, mg/L	1.64 ± 1.79 a	2.20 ± 1.84 b	4.36 ± 6.69 c	5.41 ± 6.30 d	0.0001
LDH, U/L	181.4 ± 48.7 a	213.3 ± 60.9 b	221.9 ± 70.4 c	188.0 ± 15.7 a	0.001
α-GST	202.8 ± 171.4	223.2 ± 177.6	277.2 ± 211.7	265.7 ± 209.7	0.350
KIM-1	64.1 ± 20.7 a	75.8 ± 16.1 b	80.4 ± 14.7 c	90.4 ± 9.2 d	0.001
SP- A	146.6 ± 63.2 a	175.1 ± 69.7 b	170.5 ± 68.8 b	184.0 ± 70.9 c	0.047
CC16	43.7 ± 11.9	45.2 ± 11.8	43.2 ± 14.1	41.0 ± 9.8	0.770

The outcome (Table 7) showed that there were no significant variations in the age of the workers (Al-Tuba 42.3 ± 6.2, Al-Barjisia 42.4 ± 2.7, Al-Masfa Refinery 38.6 ± 7.6, P = 0.061), or BMI (27.6 ± 3.5, 27.4 ± 2.7, 28.1 ± 4.5, P = 0.091). There was also no significant difference between CRP levels at the sites (2.33 and 2.85 to 3.65 and 5.26, P = 0.110), but the levels of 2-alphagamma-S-glutamylglutamic acid transglutamase varied significantly (Al-Tuba: 294.5, Barjisia: to 277.8 and Basra: 191.9 to 154.1). There were no statistically significant differences in KIM-1, SP-A, and CC16 was close to significant (P = 0.056). The exposure time of workers varied with sites (Al-Tuba: 16.3 ± 8.3, Al-Barjisia: 17.4 ± 7.9, Basra: 12.0 ± 6.3 years, P = 0.002). These results indicate that the location of work can affect some of the biochemical indicators including the α-GST and the duration of exposure whereas other markers were not significantly altered.

The risk assessment results for workers exposed to toxic gases (Table 8) showed that age (OR = 1.010; 95% CI: 0.938–1.087; P = 0.799) and body mass index (OR = 0.971; 95% CI: 0.832–1.133; P = 0.707) did not significantly affect the likelihood of exposure. Similarly, CRP level was not a significant risk factor (OR = 1.245; 95% CI: 0.869–1.783; P = 0.232). Conversely, LDH levels showed a significant increase in risk (OR = 1.039; 95% CI: 1.020–1.059; P < 0.001), as did α-GST (OR = 1.009; 95% CI: 1.002–1.016; P = 0.012), KIM-1 (OR = 1.044; 95% CI: 1.006–1.082; P = 0.022), and SP-A (OR = 1.014; 95% CI: 1.004–1.024; P = 0.008). CC16, however, was not a

significant risk factor (OR = 0.965; 95% CI: 0.922–1.010; P = 0.128). These results indicate that exposure to toxic gases is associated with a significant increase in risk, with elevated levels of certain biomarkers related to cellular damage, respiratory function, and liver function.

Table 7
Comparison of mean biomarker levels across different work locations (mean ± SD)

Variables	Al-Tuba (n = 40)	Al-Barjisia (n = 38)	Al-Masfa (n = 52)	P-value
Age, year	42.3 ± 6.2 a	42.4 ± 8.4 a	38.6 ± 7.6 b	0.061
BMI, kg/m²	27.6 ± 3.5	27.4 ± 2.7	28.1 ± 4.5	0.091
CRP, mg/L	2.33 ± 2.85	3.65 ± 5.26	2.16 ± 1.99	0.110
LDH, U/L	205.1 ± 57.5	208.7 ± 43.8	226.3 ± 59.2	0.138
α-GST	294.5 ± 243.0 a	277.8 ± 182.0 a	191.9 ± 154.1 b	0.025
KIM-1	72.2 ± 16.6	76.0 ± 16.6	78.5 ± 19.2	0.250
SP-A	165.6 ± 66.3	186.4 ± 73.5	163.9 ± 73.8	0.288
CC16	47.1 ± 13.0	44.8 ± 12.5	41.2 ± 10.0	0.056
Duration	16.3 ± 8.3 a	17.4 ± 7.9 a	12.0 ± 6.3 b	0.002

Table 8
Odds ratios and 95% confidence intervals for biochemical and demographic variables

Variable	Odds ratio	95% CI for OR	P-value
Age, year	1.010	0.938–1.087	0.799
BMI, kg/m²	0.971	0.832–1.133	0.707
CRP, mg/L	1.245	0.869–1.783	0.232
LDH, U/L	1.039	1.020–1.059	<0.001
α-GST	1.009	1.002–1.016	0.012
KIM-1	1.044	1.006–1.082	0.022
SP-A	1.014	1.004–1.024	0.008
CC16	0.965	0.922–1.010	0.128

Discussion

The current research assessed the biochemical and inflammatory parameters of petroleum workers who were exposed to toxic gases compared with those who were not exposed to toxic gas. Exposure to occupational toxins resulted in extensive modification of numerous biomarkers in analysis of the whole exposed population (n = 130) indicating systemic and organ-specific effects of occupational exposure. The concentrations of C-reactive protein (CRP) in the exposed petroleum workers (2.65 ± 3.52 mg/L) were higher than those of controls (1.26 ± 1.15 mg/L), which suggests the presence of a chronic inflammatory process, which might have been induced by inhaled hydrocarbons and petroleum byproducts. This finding is in line with previous research that found high levels of CRP in fuel station workers and refinery employees who are exposed to benzene and volatile organic substances, which indicates long-term low-grade inflammation because of workplace exposure (Sajid et al., 2020). On the other hand, other studies failed to find significant CRP differences, potentially because of lower exposure intensity, or adequate protective mechanisms, or differences in immune status of the participants, and the need to recognize the effect of environmental and work conditions in shaping the systemic inflammation (Cordiano et al., 2022).

The cellular injury marker lactate dehydrogenase (LDH) was significantly elevated in exposed petroleum workers (214.6 ± 55.1 vs. 153.6 ± 38.1 U/L, P = 0.0001), with no difference between different subgroups by site (Al-Masfa: 226.3 ± 59.2; Barjisia: 208.7 ± 43.8). High LDH is indicative of general tissue destruction or oxidative stress caused by petroleum toxins. Other elevations have been documented in workers exposed to hydrocarbon fumes and gasoline vapors, where increased LDH is attributed to oxidative cell damage (Moneim et al., 2023). The variability among studies in which certain groups of people responded less to LDH elevation can indicate variability in duration of exposure, composition of the hydrocarbons, or adaptive responses in the cell that can counteract the effects of oxidative stress (Rizk et al., 2020).

The level of α-GST enzyme was significantly higher among the exposed individuals (248.6 ± 197.3 U/L) than the level of the controls (147.6 ± 89.1 U/L, P = 0.001). High α-GST is a sign of an increased enzyme activity to detoxify the reactive intermediates produced during metabolism of hydrocarbons, which is evidence of subclinical hepatic or renal stress. This observation is supported by earlier studies which have shown that enhanced α-GST activity in petroleum and chemical industry workers is an initial biomarker of occupational toxicity (Lal et al., 2018). In the Masfa subgroup, however, the difference was not so significant (191.9 ± 154.1 vs. 147.6 ± 89.1in the control, P = 0.080), which may be explained by the differences in exposure concentration or efficient use of personal protective equipment, in which case environmental and behavioral factors play an important role (Chatrin et al., 2020).

KIM-1 is a renal specific injury marker that was significantly increased in exposed workers (75.8 ± 17.7 vs. 58.0 ± 18.4 U/L, P < 0.0001) which indicated early tubular damage. This finding is in agreement with reports that suggested that KIM-1 is a dynamic biomarker of nephrotoxicity caused by chemical exposure, such as petroleum hydrocarbons and heavy metals (Kang et al., 2021; Moneim et al., 2023). The site-specific data also gives a steady elevation across Al-Masfa (78.5 ± 19.2), Al-Barjisia (76.0 ± 16.6), and Al-Tuba (72.2 ± 16.6), which is in favor of occupational exposure and not genetic predisposition. The comparative studies that could not find significant changes in KIM-1 could have used shorter exposure times or less cumulative toxin loads (Moetiara, 2020).

The level of Surfactant Protein-A (SP-A) was also found to be significantly elevated among the exposed workers (171.0 ± 71.6 vs. 133.3 ± 45.2, P = 0.001) as an indicator of pulmonary epithelial reaction to long term exposure to the toxic gases. The increase in SP-A is the evidence of the activation of alveolar type II cells, which is one of the protective mechanisms against oxidative stress and inflammation. This is in line with other occupational studies that have reported high SP-A in workers who are exposed to diesel exhaust and petroleum fumes (Arora et al., 2019; Kaur et al., 2024). Interestingly, other studies did not record any increase in SP-A, perhaps, as a result of variation in the composition of pollutants, exposure levels or methodological disparities in ELISA sensitivity.

Conversely, Clara cell protein 16 (CC16) did not exhibit any significant differences among the exposed and control groups (44.1 ± 11.9 vs. 44.0 ± 12.4, P = 0.975) indicating either maintenance of Clara cell activity or insufficient exposure to induce any significant depletion. Reduced epithelial injury has been suggested by previous research in conditions where chronic inhalation of high-concentration pollutants reduced CC16, and cholinergic eosinophils were involved (Duan et al., 2019; Nangola et al., 2023). The interchange can be explained by moderate levels of exposure, adaptation of cellular response, or the differences in personal protective mechanisms.

Patterns of biomarkers were also dependent on age and length of exposure. Age was not a significant factor for most of the markers but longer exposure (>20 years) was associated with increased CRP (4.36–5.41 mg/L), KIM-1 (80.39–90.42 U/L) and SP-A (170.5–184.0 U/L) indicating that cumulative effects of long-term exposure to hydrocarbons on systemic inflammation, renal, and pulmonary systems occurred. The findings reflect longitudinal occupational research that showed dose-response patterns in regard to the duration of exposure and subsequent increases in biomarkers (Moro et al., 2019). On the contrary, other studies found little correlation with the period of occupational exposure, probably because of variations in environmental control indices, or the difference in population heterogeneity (Rahimi Moghadam et al., 2020).

Local differences were found; α-GST was much more pronounced in Al-Tuba (294.5 ± 243.0) and Al-Barjisia (277.8 ± 182.0) than in Al-Masfa (191.9 ± 154.1, P = 0.025), and this could be attributed to local exposure levels, local ventilation or local operation activities. LDH was consistently high and KIM-1 was consistently high in all sites, illustrating that petroleum work is in general a risk factor of cellular and renal stress. The existence of such spatial variability has been observed in previous field research, where it is clear that localized occupational health evaluation is essential (Lima et al., 2024).

Conclusion

Overall, the paper indicates that individuals working in the petroleum industry who are exposed to poisonous gases have systemic inflammation, renal tubular stress, liver detoxification, and lung epithelial activation. These results are mostly in line with the existing occupational toxicology literature, and divergences in CC16 and site-specific differences are most probably attributed to the difference in the intensity and the duration of exposure, the protective measures, or the individual adaptation. All these findings lend credence to the fact that LDH, α-GST, KIM-1, and SP-A should be included in standard health surveillance of petroleum workers to allow subclinical kidney injury to be detected and preventive measures to be taken.

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