ASSESSMENT OF TOXICITY IMPACT AND BIOCHEMICAL CHANGES RESULTING FROM DIRECT AND INDIRECT EXPOSURE TO LIQUEFIED PETROLEUM GAS

Ibrahim H. Borghsh [(1)]; Magdy M. Mahmoud [(2)]; Inas Fawzy Gaballah [(3)]; Rasha E. Hassan [(2)]

1) Department of Environmental Basic Sciences, Faculty of Graduate Studies and Environmental Research, Ain Shams University 2) Department of Biochemistry, Faculty of Science, Ain Shams University 3) Department Occupational and Environmental Medicine, Faculty of Medicine, Cairo University

ABSTRACT

Mercaptans are volatile organic compounds (VOCs) used in industries such as natural gas odorization and chemical manufacturing. Prolonged or high-level exposure to these substances may pose serious health risks. This study investigated the chronic toxicity of ethyl mercaptan in male workers involved in the gas cylinder filling process. Eighty male workers aged 30–50 years, with at least 10 years of employment at liquid petroleum gas (LPG) filling factory, were examined. They were divided into two groups: 60 workers with direct exposure (further classified by employment duration—10, 20, or 30 years) and 20 with indirect exposure for 20 years. All participants underwent clinical and laboratory assessments both at the start and during their employment. The findings revealed significant negative health changes associated with prolonged exposure to ethyl mercaptan. Notably, there was a marked decline in total antioxidant capacity (TAC) and reduced glutathione (GSH), along with increased serum malondialdehyde (MDA), indicating heightened oxidative stress. Liver function was impaired, as shown by elevated levels of ALT, AST, and total bilirubin. Renal dysfunction was also indicated by increased serum urea and creatinine. Lipid profile disturbances were evident with higher total cholesterol and triglycerides. Furthermore, elevated levels of insulin- like growth factor binding protein-2 (IGHBP-2), homocysteine, cytokeratin fragment21-1 (CYFRA21-1), and high-sensitivity C-reactive protein (HsCRP) suggested systemic inflammation and potential early markers of carcinogenic or cardiovascular risk. In conclusion, the study emphasizes the toxicological impact of long-term occupational exposure to ethyl mercaptan and highlights the urgent need for improved workplace safety, regular monitoring, and preventive health measures to protect workers from associated health risks.

Key words: LPG; Ethyl mercaptan; oxidative stress, renal and hepatic dysfunction

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

INTRODUCTION

Liquid petroleum gas (LPG), also referred as simply propane or butane, is a flammable mixture of odorless hydrocarbon gases used as fuel in heating, cooking equipment and vehicles **.** Ethyl mercaptan, or ethanethiol (C_2H_5SH), is a volatile, sulfur-containing organic compound known for its strong, disagreeable odor, often described as similar to rotten cabbage or garlic. It is a colorless gas at room temperature and is part of the thiol group of compounds, characterized by the -SH functional group. Ethyl mercaptan is extremely odoriferous, detectable by the human nose at concentrations as low as 0.02 parts per million (ppm), which makes it particularly useful as a warning agent in various applications **(Drew *et al*., 2023; PubChem, 2025).**

Ethyl mercaptan is widely spread and critically used as an odorant in natural gas and liquefied petroleum gas (LPG). Since both methane and propane are odorless, ethyl mercaptan is added to allow people to detect gas leaks easily. This significantly improves safety by providing an early warning before concentrations reach explosive levels. It is typically added at 0.5–1.0 ppm concentrations in natural gas pipelines **(Occupational Safety and Health Administration, 2018)**. Ethyl mercaptan serves as a chemical intermediate in the production of other organosulfur compounds and pharmaceuticals. It is used in the synthesis of pesticides, antioxidants, and accelerators in rubber processing. It also plays a role in polymer manufacturing and refining processes, acting as a precursor or reagent for introducing sulfur atoms into larger molecules **(ATSDR, 2025).** In agriculture, ethyl mercaptan has been used as a component in fumigants and repellents. Its pungent smell acts as a deterrent to burrowing animals like moles and gophers. However, its use in pest control has declined due to its strong odor and the development of more effective agents **(WHO, ٢٠٢٥).** Also it can be used as warning agent in other fuels aside from natural gas, ethyl mercaptan is sometimes added to other odorless fuels, such as butane or ethanol used in industrial settings, to help detect leaks. This helps reduce the risk of accidental fires and explosions **(Occupational Safety and Health Administration, 2018)**.

Inhalation is the most common route of exposure. Short-term exposure to low concentrations (below 0.5 ppm) is generally safe and non-toxic. However, exposure to levels

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

above 100 ppm may cause significant health effects, including respiratory tract irritation, nausea, dizziness, headache, and in severe cases, pulmonary edema or asphyxiation due to displacement of oxygen **(EPA, 2025)** .OSHA has set the permissible exposure limit (PEL) at 0.5 ppm as an 8-hour time-weighted average **(Occupational Safety and Health Administration, 2018)**.

Direct contact with liquid ethyl mercaptan or high-concentration vapors may cause irritation to the skin and eyes. Symptoms include redness, itching, tearing, and burning sensations. Prolonged skin exposure may result in dermatitis, particularly with repeated occupational contact **(EPA, 2025).** Although ingestion is rare, ethyl mercaptan is toxic if swallowed. Ingestion may cause abdominal pain, vomiting, and central nervous system (CNS) depression. The compound is also rapidly absorbed through mucous membranes, and in large doses can potentially affect liver and kidney function, although such outcomes are more common in animal studies **(Brook *et al*., 2023).** However, according to the findings of **Jia *et al*., (2021) and Choi *et al*., (2021) w**orkers exposed to volatile organic compounds (VOCs) reported significant elevations in hepatic enzymes.

Exposure to methyl mercaptan has been shown to disrupt oxidative balance and antioxidant enzyme activity, **Fang et al. (2019**) reported significant increases in lipid peroxidation (MDA levels) and decreases in key antioxidant enzymes such as superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px), indicating elevated oxidative stress and potential cellular damage.

Exposure to moderate-to-high levels of ethyl mercaptan may result in central nervous system effects such as confusion, dizziness, fatigue, and, in extreme cases, loss of consciousness. These effects are more likely in enclosed or poorly ventilated environments where gas accumulation may occur undetected despite its odor **(Drew *et al*., 2023; ATSDR, 2025).** Prolonged exposure to low levels of ethyl mercaptan especially in occupational settings, can result in chronic respiratory issues such as bronchitis or asthma-like symptoms. There is limited evidence on its carcinogenicity, and no studies have conclusively shown it to be a cancer-causing agent in humans. However, long-term exposure to thiols in general has been associated with liver and kidney damage in animal studies **(Brook *et al*., 2023).**

Due to its volatility and odor, ethyl mercaptan can be detected easily even at low concentrations, which is beneficial for leak detection. However, this also means that occupational exposure is common in industries dealing with natural gas or sulfur chemistry.

Aim of the study: The current study aimed to analyze the biochemical alterations and toxicity aspects of ethyl mercaptan in liquefied petroleum gas among male workers filling gas cylinders.

SUBJECTS AND METHODS

Setting and Design:

One petroleum company that was considering the filling of gas cylinders with liquid petroleum gas equipment was chosen in the current study. The gas company's main headquarters is in Cairo, and there are other operational locations around the Upper Egypt province. The present observational study was conducted on 80 workers chosen from 379 of various employment categories from Sohag factory of Petroleum Company to assess the biochemical changes and evaluate the toxicity of liquid petroleum gas along their occupational period.

1- Subjects

Workers Selection:

Participants were selected from among the attendants accompanying company employees at the Sohag factory during their mandatory annual medical check-up.

Inclusion Criteria: The study included only male workers aged between 30 and 50 years who had been employed at a factory for filling gas cylinders with LPG in Sohag governorate for a minimum of 10 years and maximum 30 years.

Study Groups:

For the current investigation, participants were categorized into two main groups based on the location and duration of workplace exposure:

- **Group (I):** This group consisted of 60 high-risk workers who had direct exposure to ethyl mercaptan in liquid petroleum gas through filling gas cylinders. It was further subdivided into three subgroups based on the duration of exposure:

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

- **Group (I-a):** 20 workers with 30 years of direct contact to ethyl mercaptan in liquid petroleum gas (working in the filling area)
- **Group (I-b):** 20 workers with 20 years of exposure
- **Group (I-c):** 20 workers with 10 years of exposure
- **Group (II):** included 20 moderate-risk workers who were subjected indirectly (working in nearby offices within the same factory) to ethyl mercaptan along 20 years of employment.

The variables under study were compared for each group of participants before and during the occupational period, meaning that the primary clinical assessment for all of the eighty recruited employee served as control.

Participants were informed adequately about aims, methods and anticipated benefits of the study. Informed verbal consent of the subjects to participate in the study with a full right to withdraw was obtained with assurance of confidentiality and anonymity of the data.

2- Methodology

Data collection included subjects interviewing, blood sampling, work environment and air measurements methods of data collection are comprised:

2.1 Subjects interviewing.

All the subjects were interviewed with regard to general characteristics, history of work, smoking habitat and health symptoms (Face to face interview).

2.1.1. Questionnaire

The questionnaire included following parts

a) Personal history:

Including name, age, and smoking habits.

b) Occupational history:

In details, including duration of employment, location, description of the current job, body mass index (BMI) and previous jobs, if present, either in the same place or in other places and the use of protective equipment.

c) Medical History:

Including present, past history (earlier severe exposure to toxic agents, chronic liver or kidney diseases before attending their jobs)

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

2.2 Samples collection

2.2.1 Blood samples

Five milliliters of blood were collected from each worker through venipuncture at the just at annual medical check before administration of any medication by using sterile plastic syringes. Each blood sample was divided into 2 parts, one part was collected on heparin for assessing complete blood count (CBC) and the other part was obtained in EDTA coated tubes for the other biochemical investigations. Sera were separated after centrifugation of the non-heparinized blood at 1000 g for 10 minutes at 4 °divided into aliquot s and stored at -20 °C until use.

Biochemical investigations: Biochemical parameters included serum liver transaminases (ALT, AST), total bilirubin, urea, creatinine; total cholesterol (TC), triglycerides (TG) and high-sensitivity C - reactive protein (Hs-CRP) were analyzed by the automated Cobas 6000 system. Total antioxidant capacity (TAC) and malonaldhyde (MDA) were determined according to **Koracevic *et al*. (2001),** reduced glutathione (GSH) was assessed as described by using colormetric kit purchased from Biodiagnostic chemical company – Dokki, Giza, Egypt Homocysteine, was evaluated using ELISA kit provided by IBL, Hamburg, Germany. Insulin-like growth hormone-binding protein-2 (IGHBP-2) and (CYFRA21-1) were quantified using ELISA kit purchased Biodiagnostic chemical company respectively complete blood picture was analyzed using Sysmex (Automated Hematology Analyzer XT-2000i/XT-1800i).

Statistical analysis: Statistical analysis was performed using SPSS version 17.0 software (Chicago, USA). Results are expressed as mean ± standard deviation (SD). Data were tested for normality using the **Shapiro- Wilk** test and all variables were found to be normally distributed ($p > 0.05$). Paired t-test was used to compare each group corresponding to control.The results were considered statistically significant at $p < 0.05$. Percentage of change representing the percent of variation with respect to control values was also calculated. Pearson correlation was calculated to assess the relationship between studied parameters. Receiver operating characteristic (ROC) curve analysis was conducted for each group versus

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

control to evaluate the potentiality of the measured variables as diagnostic markers for ethyl mercaptan toxicity.

RESULTS

Table (1) shows how direct and indirect exposure to LPG containing ethyl mercaptan affects oxidative stress markers as well as hepatic and renal function biomarkers in exposed workers throughout varied occupational periods. Workers with direct exposure (GI), particularly those with longer occupational periods (GI-b and GI-c), had significantly lower antioxidant defense measures. All exposure groups had significantly reduced total antioxidant capacity and GSH levels than the control group (P<0.0001), with GI-c showing the lowest values. MDA levels were significantly higher in all exposure groups (P<0.0001), indicating increased oxidative stress. GI c had the highest level.

Serum ALT and AST levels were considerably higher in all exposed groups compared to control (P<0.0001). The rise was particularly evident in the GI-b and GI-c subgroups. Total bilirubin levels were also considerably raised in GI-b, GI-c, and GII (P<0.0001). Urea and creatinine levels were significantly higher in all groups relative to the control, with P values ranging from <0.05 to <0.0001. The most significant elevations were detected in the GI-c, indicating decreased renal function possibly due to chronic toxic exposure.

Table 1: Effect of direct and indirect exposure to ethyl mercaptan in LPG on serum oxidative stress, liver and kidney biomarkers of workers at different occupational periods

GROUPS / PARAMETERS	CONTROL (N=80)	GI DIRECT EXPOSURE (N=60)			GII INDIRECT EXPOSURE (N=20)
		GI-a(n=20)	GI-b (n=20)	GI-c (n=20)	
TAC (mM) mean± SD	2.71±0.62	2.69±0.31 (-0.74%)	1.87 ± 0.20[a] (-31%)	0.94 ±0.19 [a] (-65.3%)	1.67±0.17 [a] (-38.4%)
GSH (mM) mean± SD	15.48±1.8	8.31±0.91[a] (-46.32%)	4.13 ± 1.2 [a] (-73.32%)	0.80 ±0.15 [a] (-94.83%)	4.38±1.17 [a] (-71.71%)
MDA (nmol/ml) mean± SD	0.37 ± 0.13	0.79±0.17[a] (113.5%)	0.69 ± 0.39[a] (86.49%)	2.62 ±0.32[a] (608.1%)	1.61±0.21[a] (335.1%)
ALT (IU/L) Mean ± SD	13.12±3.61	29.2 ±8.3 [a] (122.6%)	44.0±12.63 [a] (235.36%)	43.00±5.43[a] (227.74%)	27.6 ±10.1[a] (110.36%)
AST (IU/L) Mean ± SD	11.07±3.41	24.25±7.2[a] (119.1%)	41.94±10.6[a] (278.86%)	40.90±5.23 [a] (269.47%)	21.25 ± 6.6 [a] (91.96%)
T. bilirubin (mg/dl) Mean ± SD	0.51 ± 0.16	0.44 ±0.23 (13.73%)	0.77 ± 0.23 [a] (50.1%)	0.85 ± 0.14 [a] (66.67%)	0.72 ± 0.16 [a] (41.18%)

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

Urea(mg/dl Mean ± SD	25.16±4.4	28.9±5.53[b] (14.86%)	32 .0± 7.39 [a] (27.19%)	36.9± 8.81 [a] (46.66%)	29.4± 7.13 [b] (16.85%)
Creatinine (mg/dl) Mean ± SD	0.60 ± 1.82	0.86±0.10[a] (43.33%)	0.86 ± 0.13[a] (43.33%)	0.85± 0.13 [a] (41.67%)	±0.15 [b] (13.33%)

[a] highly significant change at *p*<0.001 compared with control results prior to employment (paired-t-test)
[b] Significant change at *P*<0.05
(%): percent of change from control values

Data presented in Table (2) shows that chronic exposure to ethyl mercaptan in liquefied petroleum gas (LPG) causes significant metabolic and systemic changes in both directly and indirectly exposed personnel. Serum TC and TAG levels were considerably higher in all groups compared to control values (*P*< 0.0001). The GI-c category had the highest TC elevation, indicating a potential cumulative dyslipidemic effect from long-term direct exposure. Similarly, TAG levels were considerably higher in all exposed subgroups, with the highest concentration in GI-c.

Homocysteine, a measure of cardiovascular and oxidative stress, elevated significantly across all exposure groups (*P*< 0.0001), with the GI-c subgroup having the highest mean value. In addition, higher levels of Hs-CRP were detected in GI-b, GI-c, and GII versus their control values. The increase in GI-c was statistically significant (*P*<0.0001), indicating a systemic inflammatory response, especially among individuals with extended direct exposure. The tendency toward higher values over longer exposure periods suggests a dose-response connection, Table (2). Furthermore, IGHBP-2 levels were markedly elevated in all exposed subgroups (*P* <0.0001), with the highest mean level observed in GI-c. Obviously, only GI-c had a substantial rise in CYFRA21-1 (*P*<0.0001), while the other categories showed non-significant or minor declines, Table (2).

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

Table 2: Effect of direct and indirect exposure to mercaptans in LPG on serum TC, TG, HC, Hs-CRP, IGHBP-2 and CYFRA21-1

GROUPS / PARAMETERS	CONTROL (N=80)	GI DIRECT EXPOSURE (N=60)			GII INDIRECT EXPOSURE (N=20)
		G I-a (n=20)	G I-b (n=20)	GI-c (n=20)	
TC (mg/dl) Mean ± SD	80.8±14.43	139.8±42.1[a] (73.02%)	151.9±42.1[a] (88%)	233.6±35.9[a] (189.11%)	180.4±46.9[a] (123.27%)
TG (mg/dl) Mean ± SD	59.18±12.39	91.35±17.3[a] (54.36%)	95.10±30.6[a] (60.7%)	124±28.86[a] (109.5%)	110.9±53.6[a] (87.4%)
Homocysteine (mmol/L) Mean ± SD	2.17± 0.55	10.71± 2.2[a] (393.54%)	12.13±2.95[a] (459%)	13.65±1.38[a] (529%)	10.45± 2.3[a] (381.57%)
HsCRP (g/ml) Mean ± SD	1.00± 0.71	0.92 ±0.54 (-8%)	1.52 ± 0.89[b] (52%)	1.86± 0.83[a] (86%)	1.44 ± 0.80[a] (44%)
IGHBP-2 (ng/ml) Mean ± SD	0.43± 0.17	14.4 ±2.38[a] (3248.8%)	16.26±2.69[a] (3681.4%)	17.97±4.1[a] (4079%)	17.23± 3.9[a] (3907%)
CYFRA21-1 (ng/dl) Mean ± SD	0.59± 0.89	0.10 ±0.10 (-83.1%)	0.56±0.11 (-5.1%)	1.59± 0.49[a] (169.49%)	0.24 ± 0.22 (-59.32%)

[a] highly significant change at $p<0.001$ compared with control results prior to employment (paired-t-test)
[b] Significant change at $P<0.05$
(%): percent of change from control values

The analysis of complete blood count (CBC) parameters among workers exposed to mercaptans in liquefied petroleum gas (LPG) revealed significant hematological alterations, particularly in those with prolonged direct exposure, (Table 3). White blood cell counts were significantly elevated in workers with prolonged direct exposure (GI-b and GI-c) and in the indirectly exposed group (GII), with ($P<0.0001$). The highest WBC count was observed in GI-c, suggesting that WBC elevation may depend on cumulative exposure duration. Clearly, RBC counts remained statistically unchanged in most groups, with the exception of a little but significant decline in the GI-c group ($P = 0.01$). Moreover, a progressive decline in hemoglobin concentration was observed in exposed groups, with statistically significant reductions in GI-b, GI-c, and GII ($P<0.0001$). These values fall below the normal reference range for adult males, indicating the development of exposure-related anemia. The trend suggests that hemoglobin concentration is inversely related to the duration and intensity of

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

exposure. On the opposite side, platelet counts remained largely unaffected across groups, with no statistically significant differences except in the GI-a subgroup, which showed a notable decrease (P<0.0001), Table (3). This finding can be explained by the toxicokinetic and mechanistic properties of ethyl mercaptan.Unlike aromatic hydrocarbons like benzene, which decrease bone marrow and impair hematopoiesis, ethyl mercaptan operates largely as a respiratory irritant and central nervous system depressant at high concentrations **(ATSDR, 2001)**. Its metabolism is rapid hepatic oxidation to sulfoxides and sulfates, followed by urine excretion, with no bioaccumulation or generation of reactive metabolites that can harm hematopoietic stem cells **(Hsu and Maibach, 1999)**. Platelet count changes are typically caused by either impaired megakaryocyte function or increased peripheral destruction; neither mechanism is associated with ethyl mercaptan at concentrations found in LPG handling environments **(IPCS, 1992).** Furthermore, detectable thrombocytopenia requires sustained or severe marrow injury due to the short biological half-life of platelets (7–10 days), which is unlikely under current exposure settings. There have also been no notable changes in hematological parameters, such as platelet indices, in animals exposed to comparable sulfur-containing substances in sub-chronic inhalation experiments **(NIOSH, 2016).**

Concerning hematocrit percentage, GI-c workers possessed the lowest percentage (P< 0.01), which is consistent with the hemoglobin findings and supporting the presence of anemia. Interestingly, HCT was significantly elevated in the GII group (P<0.0001), potentially reflecting hemoconcentration or reactive polycythemia secondary to subclinical hypoxia or environmental stress.

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

Table 3: Effect of direct and indirect exposure to mercaptans in LPG on CBC among workers along different occupational periods.

GROUPS / PARAMETERS	CONTROL (N=80)	GI DIRECT EXPOSURE (N=60)			GII INDIRECT EXPOSURE (N=20)
		G I-a (n=20)	GI-b (n=20)	GI-c (n=20)	
WBCs (10^3/Cmm) Mean ± SD	5.45±0.77	5.15±0.75 (-5.5%)	7.11±0.87 [a] (30.46%)	7.68±1.08 [a] (40.91%)	6.49±1.31 [a] (19.1%)
RBCs (10^6/Cmm) Mean ± SD	4.74±0.51	5.09± 0.86 (7.38%)	4.62± 0.64 (-2.53%)	4.25±0.69[b] (-10.33%)	4.91±0.76 (3.57%)
Hb (g/dl) Mean ± SD	14.58±0.7	14.19±1.3 (2.67%)	12.27±1.5[a] (-15.84%)	10.33±1.9 [a] (-29.14%)	12.67±1.8 [a] (-13.1%)
Platelets (10^3/Cmm) Mean ± SD	370.3±56.4	286.2±65.9[a] (-22.65%)	367.40±63.8 (-0.78%)	388.9±57.5 (5%)	361.6±58.1 (-2.35%)
HCT (%) Mean ± SD	42.87±3.35	44.20±3.27 (3.1%)	44.55±3.32 (3.91%)	30.87±3.51[b] (-28%)	46.15±2.52[a] (7.65%)

[a] highly significant change at $p<0.001$ compared with control results prior to employment (paired-t-test)
[b] Significant change at $P<0.05$, (%): percent of change from control values

Pearson's correlation analysis was performed to evaluate the relationships between oxidative stress parameters (TAC, GSH, MDA) and serological biomarkers (IGFBP-2, homocysteine, CYFRA 21-1, and Hs-CRP) across all study participants, Table (4). The results revealed several statistically significant associations, reflecting the complex biochemical interplay between antioxidant status and systemic toxicity markers due to mercaptan exposure. TAC showed a slight but significant negative connection with GSH (p = 0.012), CYFRA 21-1 (r = -0.373, p < 0.001), and Hs-CRP (r = -0.210, p = 0.008). These data indicate that decreased antioxidant defense may be linked to increased inflammation and epithelium damage. GSH had substantial inverse associations with MDA (r = -0.801, p < 0.001), IGFBP-2 (r = -0.884, p < 0.001), and homocysteine (r = -0.879, p < 0.001) while it exhibited moderate to week relationship with CYFRA 21-1 (r = -0.492, p < 0.001), and Hs-CRP (r = -0.295, p < 0.001), respectively. These negative relationships suggest that low GSH levels may be a key predictor of oxidative stress progression, inflammatory response, and early tissue damage in exposed people.

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

In contrast, **MDA**, a key marker of lipid peroxidation, was positively and strongly correlated with **IGFBP-2** ($r = 0.856$, $p < 0.001$), **homocysteine** ($r = 0.868$, $p < 0.001$), and **CYFRA 21-1** ($r = 0.716$, $p < 0.001$), reinforcing the notion that oxidative damage is associated with increased cellular turnover and endothelial dysfunction. Also, it showed a moderate positive association with **Hs-CRP** ($r = 0.369$, $p < 0.001$), indicating a link between lipid peroxidation and systemic inflammation.

Further substantial positive correlation was noticed between IGFBP-2 and homocysteine ($r = 0.905$, $p < 0.001$), CYFRA 21-1 ($r = 0.548$, $p < 0.001$), and Hs-CRP ($r = 0.305$, $p < 0.001$). Similarly, homocysteine levels were substantially linked with IGFBP-2 and MDA, and marginally with CYFRA 21-1 ($r = 0.586$, $p < 0.001$) and Hs-CRP ($r = 0.369$, $p < 0.001$), highlighting its role in oxidative stress, vascular dysfunction, and systemic inflammation. Finally, CYFRA 21-1 and Hs-CRP showed a significant positive correlation ($r = 0.305$, $p < 0.001$), indicating increased epithelium damage and inflammation in affected workers.

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

Table (4): Correlations of antioxidants and serological markers among different studied groups

		TAC	GSH	MDA	IGFBP2	HOMOCYSTEINE	CYFRA 21	HSCRP
TAC	Pearson Correlation	1	-.197*	-.069	.014	.040	-.373**	-.210**
	Sig. (2-tailed)		.012	.385	.857	.613	.000	.008
	N	160	160	160	160	160	160	160
GSH	Pearson Correlation	-.197*	1	.801**	-.884**	-.879**	-.492**	-.295**
	Sig. (2-tailed)	.012		.000	.000	.000	.000	.000
	N	160	160	160	160	160	160	160
MDA	Pearson Correlation	-.069	.801**	1	.856**	.868**	.716**	.369**
	Sig. (2-tailed)	.385	.000		.000	.000	.000	.000
	N	160	160	160	160	160	160	160
IGFBP2	Pearson Correlation	.014	.884**	.856**	1	.905**	.548**	.305**
	Sig. (2-tailed)	.857	.000	.000		.000	.000	.000
	N	160	160	160	160	160	160	160
Homocysteine	Pearson Correlation	.040	.879**	.868**	.905**	1	.586**	.369**
	Sig. (2-tailed)	.613	.000	.000	.000		.000	.000
	N	160	160	160	160	160	160	160
CYFRA21	Pearson Correlation	.373**	.492**	.716**	.548**	.586**	1	.305**
	Sig. (2-tailed)	.000	.000	.000	.000	.000		.000
	N	160	160	160	160	160	160	160
HsCRP	Pearson Correlation	.210**	.295**	.369**	.305**	.369**	.305**	1
	Sig. (2-tailed)	.008	.000	.000	.000	.000	.000	
	N	160	160	160	160	160	160	160

** Significance correlation

To assess the diagnostic accuracy of oxidative stress, inflammatory, and epithelial injury biomarkers in discriminating mercaptan-intoxicated workers from unexposed controls, ROC curve analyses were conducted across the exposure groups: 10, 20, and 30 years of direct exposure, and 20 years of indirect exposure , **Figure (1)**. The results demonstrated that MDA, IGFBP2, and homocysteine consistently showed excellent diagnostic performance across all exposure periods and modes of contact. The biomarkers had an Area under the Curve (AUC) of 1.000 ($p < 0.001$) in all cases except for MDA at 10 years (AUC = 0.975), showing complete or near-perfect discrimination between exposed patients and controls.

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

CYFRA 21-1 demonstrated excellent discrimination after 20 and 30 years of direct exposure (AUC = 1.000, $p < 0.001$). The benefit of indirect exposure was moderate (AUC = 0.664, $p = 0.023$). While, Hs-CRP showed steadily increasing AUCs with longer exposure times. After 10 years of exposure (AUC = 0.506, p = 0.935), the effect became statistically significant at 20 years (AUC = 0.673, $p = 0.017$) and even greater at 30 years (AUC = 0.801, $p < 0.001$). In the indirect exposure group, it likewise demonstrated a moderate diagnostic value (AUC = 0.699, $p = 0.006$).

Obviously, GSH exhibited a strong inverse relationship with exposure, with its AUC decreasing gradually with exposure length. It had no discriminative ability at 10 years (AUC = 0.000), remained low at 20 and 30 years of direct exposure (AUCs = 0.025 and 0.000, respectively), and 20 years of indirect exposure (AUC = 0.025), all statistically significant (p < 0.001). This pattern indicates significant antioxidant depletion over time.

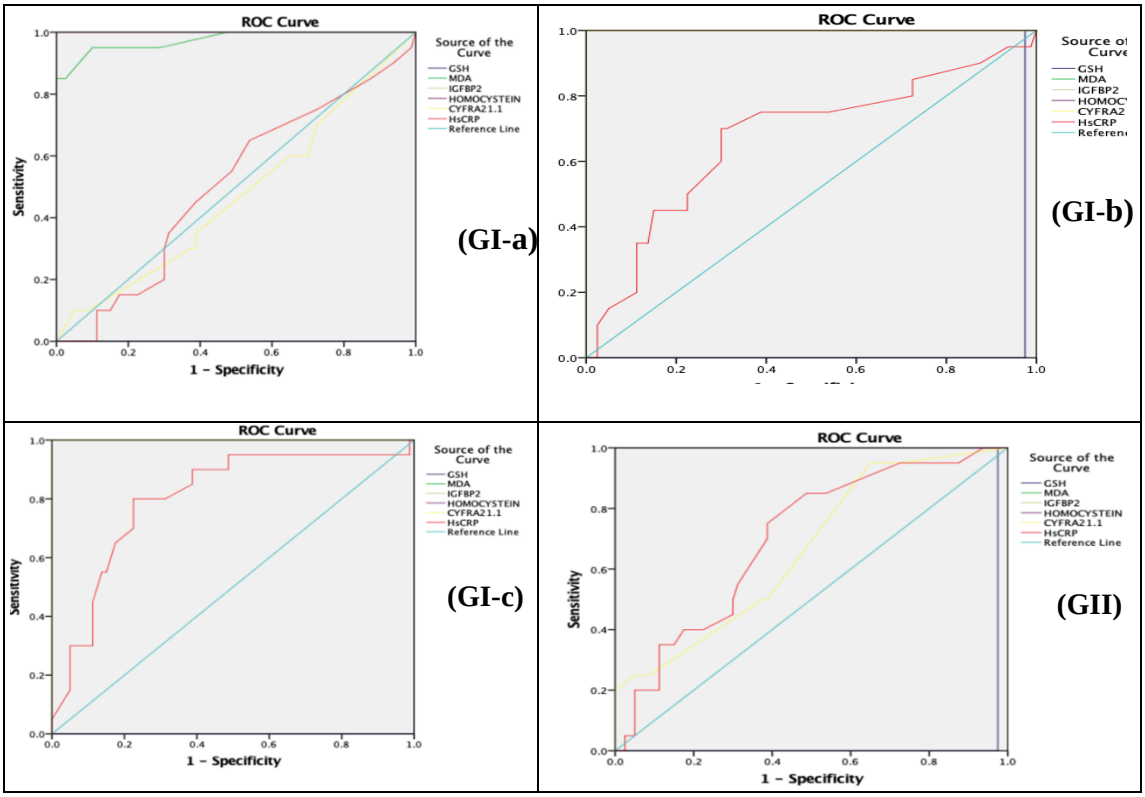

Figure (1): ROC curves across exposure groups: (GI a-c)10, 20, and 30 years of direct exposure, and (GII) 20 years of indirect exposure

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

Table 5: Area Under the Curve (AUC) and 95% Confidence Intervals (CI) for biomarkers across different ethyl mercaptan exposure groups.

BIOMARKER		10Y DIRECT EXPOSURE	20Y DIRECT EXPOSURE	30Y DIRECT EXPOSURE	20Y INDIRECT EXPOSURE
GSH	**AUC**	0.000 (0.000–0.000)	0.025 (0.000–0.059)	0.000 (0.000–0.000)	0.025 (0.000-0.059)
	95% CI				
MDA	**AUC**	0.975 (0.936–1.000)	1.000 (1.000–1.000)	1.000 (1.000–1.000)	1.000 (1.000-1.000)
	95% CI				
IGFBP2	**AUC**	1.000 (1.000–1.000)	1.000 (1.000–1.000)	1.000 (1.000–1.000)	1.000 (1.000–1.000)
	95%CI				
Homocyst.	**AUC**	1.000 (1.000–1.000)	1.000 (1.000–1.000)	1.000 (1.000–1.000)	1.000 (1.000–1.000)
	95% CI				
CYFRA 21-1	**AUC**	0.472 (0.329–0.615)	1.000 (1.000–1.000)	1.000 (1.000–1.000)	0.664 0.537–0.792)
	95% CI				
Hs-CRP	**AUC**	0.506 (0.368–0.644)	0.673 (0.529–0.816)	0.801 (0.691–0.912)	0.699 (0.579–0.818)
	95% CI				

DISCUSSION

The chronic toxicological impact of occupational exposure to ethyl mercaptan (EM), a volatile sulfur-containing compound used in LPG odorization, is comprehensively investigated in this study, which focuses on workers involved in cylinder filling over varying exposure periods. The findings reflect significant biochemical, hematological, oxidative, inflammatory, and serological alterations that correlate with exposure duration and mode (direct or indirect).

Our study revealed a significant depletion in TAC and GSH levels among workers, particularly those who had been directly involved in cylinder filling for extended periods of time (10-30 years). This decline was closely linked to increasing MDA levels, indicating oxidative lipid peroxidation and cellular membrane damage. Correlation study revealed a substantial unfavorable association between GSH, MDA, IGFBP2, and homocysteine, indicating that EM exposure increases the oxidative stress burden.. In addition, directly exposed groups showed elevated levels of liver enzymes (ALT, AST) and total bilirubin,

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

especially after 10 and 20 years of exposure, indicating hepatic stress or early hepatocellular injury. The metabolic transformations of mercaptans within the body result in reactive intermediates, which imply that oxidative stress is the joker mechanism behind mercaptan-induced cellular damage.

Comparable results were obtained by **(Akinmoladun *et al*., 2021 and Ismail *et al*., 2023).** On contrary, **Sambo *et al.* (2022)** detected no significant changes in liver enzymes among LPG vendors in Calabar, Nigeria.

The induction of oxidative stress by hydrocarbons in LPG is mostly due to their excellent lipophilicity and their capacity to cross and interact with membrane bilayers **(Dreiem *et al*., 2002)**. Mercaptans are organic compounds with a sulfur-hydrocarbon SH group attached to an organic molecule, which contributes to their lipophilicity. Mercaptans in LPG cause oxidative stress through a variety of processes, including the production of intracellular reactive oxygen species (ROS) and the dysregulation of antioxidant defenses. Through their metabolism within the body, mercaptans can undergo oxidation processes mediated by enzymes such as cytochrome P450 or peroxidases, creating superoxide anion (O-) and hydrogen peroxide (H_2O_2) **(Chan *et al*., 2022).** Mercaptan-induced oxidative stress can overcome intracellular antioxidant defenses. Furthermore, ROS can directly react with antioxidants, such as glutathione, and deplete their levels **(Włodek, 2022)**. ROS produced by hazardous mercaptan exposures cause lipid peroxidation, which can damage cell membranes. They interact with polyunsaturated fatty acids in cell membranes, producing lipid peroxides, causing oxidative damage and impair the integrity and function of cellular membranes leading to the leakage of hepatic enzymes **(Dere and Ari, 2009, Kurmi *et al*., 2013)**. According to **Tochukwu *et al.* (2020)**, LPG-induced liver damage is related to circulatory failure, hypoxia, and myocardial infarction; thus, exposure to excessive levels of LPG could lead to mortality where longevity of exposure had been considered.

Kidney indicators, such as serum urea and creatinine, were considerably higher in longer-exposure groups, indicating probable nephrotoxicity. These changes are most likely due to EM's systemic effects via inhalation and skin absorption, which cause cumulative tissue burden and organ failure. The present data are in-agreement with those published by

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

(Tochukwu *et al*., 2020) who related the current findings to high lipophilicity of petroleum compound and promotion of oxidative stress

Kurmi *et al*., (2013) referred the changes in urea and creatinine levels to the biotransformation of petroleum and particulate compounds into reactive intermediates that bind covalently to macromolecules and alter their function, resulting in cell damage. Furthermore, **Ekpenyong, and Asuquo, (2017)** mentioned that petroleum products have been shown to attach to the membrane lipid bilayer and proteins due to their high lipophilicity, causing damage to the membrane bilayer and proteins and inhibiting Na+/K+/ATPase action, resulting in ion homeostasis disturbance and cell injury.

A marked elevation in TC, TG, and homocysteine levels was documented in all exposed groups, with the highest values recorded in the 30-year direct exposure group. In accordance, **Tochukwu, *et al*., (2020)** noticed an elevation in serum levels of lipid profile parameters of male albino rats group exposed to LPG.

Ekpenyong and Asuqo (2017) attributed the obtained findings to LDL-cholesterol alteration by petroleum fumes and particulate debris, which
accelerates its oxidation.
As a result, increased oxidized LDL cholesterol is a risk factor for cardiovascular disease.

Homocysteine, a sulfur-containing amino acid, is linked to cardiovascular risk, **(Arreola *et al*., 2017).** Elevated levels of homocysteine can lead to endothelial dysfunction and increased risk of arterial diseases. Mercaptans, by influencing thiol/disulfide balance, may disrupt homocysteine metabolism. According to **(Arreola *et al*., 2017),** disturbances in thiol homeostasis have been associated with elevated homocysteine levels, suggesting a potential interaction between mercaptans and homocysteine metabolism. However, direct studies on this interaction remain limited.

High-sensitivity C-reactive protein (Hs-CRP) is a marker of systemic inflammation and has been associated with cardiovascular diseases (**Banait *et al*., 2022).** Although not consistently elevated across all groups, Hs-CRP levels were significantly higher in longer exposures, reflecting systemic inflammation. Moreover, Hs-CRP showed **exposure-dependent diagnostic improvements**, with AUCs increasing from 0.506 at 10 years to 0.801 at 30 years. This suggests

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

a gradual establishment of systemic inflammation, which may be subclinical in earlier exposure stages but becomes diagnostically significant with chronicity. Post exposure disturbances in thiol/disulfide balance have been linked to inflammatory conditions, suggesting that mercaptans might modulate Hs-CRP levels through redox-sensitive inflammatory pathways (**Singh *et al*., 2020).**

Insulin-like growth-factor-binding protein-2 (IGHBP-2) is involved in regulating the availability of insulin-like growth factors. IGHBP-2 protein has been implicated in metabolic regulation, tumorigenesis, and cellular stress responses (**Savvidis *et al*., 2024)**. Its elevation in exposed workers may indicate early cellular or oncogenic stress, potentially mediated by mercaptan-induced genotoxic or epigenetic changes.While specific studies on the direct effects of mercaptans on IGHBP-2 are limited, thiol compounds can influence protein structures and functions through redox reactions, suggesting that mercaptans may modulate IGHBP-2 activity, potentially affecting growth and metabolic processes **(Schmidt *et al*., 2019).**

Cytokeratin fragment 21-1 (CYFRA 21-1) is a fragment of cytokeratin 19 and serves as a tumor marker, in various epithelial cancers and in lung cancers, particularly in non-small cell lung cancer (NSCLC) **(Rowe *et al*., 2025).** Although there is no direct evidence linking mercaptans to CYFRA21-1 levels, thiol compounds can influence cellular processes, including those involved in cancer cell proliferation and apoptosis. Thus, mercaptans may indirectly affect CYFRA21-1 expression through modulation of cellular redox states and signaling pathways **(Dastgheib *et al*., 2020).**

In clinical settings, CBCs are often utilized to assess the impact of mercaptan exposure. For example, blood tests may reveal anemia or leukopenia in individuals with significant mercaptan exposure, aiding in the diagnosis and management of potential toxicity **(Chang *et al*., 2019).** In the present work, CBC analysis recorded a notable reduction in Hb and HCT levels among workers with 20–30 years of direct contact. While WBCs levels were raised in these groups, suggesting an inflammatory response, while platelet counts remained rather stable. These data point to mercaptan-induced hematological dysregulation, possibly secondary to oxidative stress or direct bone marrow suppression. In consistence, **Nawaz *et al*. (2020)** mentioned that exposure to high levels of mercaptans has been associated with oxidative damage to red blood cells, resulting in their rupture and subsequent anemia symptoms such as fatigue and weakness. This

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

oxidative stress may also affect white blood cell function and platelet aggregation, potentially altering CBC parameters **(Zhao *et al*., 2020).** Additionally, **Maguire *et al*., (2021**) reported that the disruption in thiol balance caused by mercaptans can impair neutrophil and lymphocyte activity, possibly influencing the neutrophil-lymphocyte ratio, a component assessed in CBCs. This balance disturbance may also contribute to immune dysregulation, leading to shifts in CBC values, particularly in the context of systemic inflammation (**Liu *et al*., 2020).**

Biomarkers such as IGFBP2 and CYFRA21-1 showed significant elevation in exposed groups. ROC curve analysis demonstrated exceptional diagnostic accuracy for IGFBP2, homocysteine, and MDA (AUC = 1.000) across most exposure groups, particularly in those with 20–30 years of direct or 20 years of indirect exposure. GSH levels showed poor diagnostic utility (AUC ≈ 0.000–0.025), possibly due to depletion in early toxic stages, while CYFRA21-1 and HsCRP had moderate predictive values depending on the group. These findings support the utility of these markers in occupational health surveillance for early detection of EM toxicity.

The correlation matrix further strengthens the mechanistic understanding by highlighting that IGFBP2 and homocysteine are positively correlated with MDA and negatively correlated with GSH. CYFRA21-1, a well-known epithelial cell injury marker, also presented strong correlations with oxidative and inflammatory indices, implying that it could serve as a sensitive biomarker of tissue damage produced from mercaptan exposure.

Finally, the **combined diagnostic profile** of oxidative, inflammatory, and epithelial markers provides a robust framework for detecting and monitoring **chronic ethyl mercaptan exposure**. The data support the use of **MDA, homocysteine, and IGFBP2 as early, high-sensitivity biomarkers**, with **GSH serving as a reliable depletion marker** and **Hs-CRP/CYFRA 21-1 offering insights into inflammatory and structural consequences** of long-term exposure to EM

CONCLUSION

Chronic occupational exposure to ethyl mercaptan during LPG cylinder filling cause severe oxidative stress, organ dysfunction, metabolic abnormalities, and hematological alterations. These harmful side effects worsen with time and mode of exposure. The diagnostic efficacy of biomarkers like IGFBP2, MDA, and homocysteine emphasizes their value in occupational

monitoring programs. Preventive strategies, exposure control, and regular bio monitoring are critical for protecting worker health in gas-filling environments.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). (2001). Toxicological Profile for Sulfur Dioxide and Related Compounds.

Agency for Toxic Substances and Disease Registry (ASTRD). (2025). Toxicological profile for ethyl mercaptan. Retrieved June 3, 2025.

Akinmoladun, A. C., Aladesanmi, O. O., Ojo, F. E., Bello, M., Taiwo, B. J., & Akindahunsi, A. A. (2021). Modifying influence of polyphenols on hematotoxicity, cardiotoxicity, and hepatotoxicity induced by liquefied petroleum gas in rats. *Toxicology Research, 10*(4) 751–760.

Arreola, R., Hernández-Fuentes, M. E., & Cruz-Fuentes, C. (2017). The effect of thiol compounds on homocysteine metabolism and cardiovascular risk: A review. Biological Chemistry, 398(5), 517–528.

Banait, T., Wanjari, A., Danade, V., Banait, S., & Jain, J. (2022). Role of high-sensitivity C-reactive protein (Hs-CRP) in non-communicable diseases: A review. *Cureus, 14*(10), e30225.

Brook, R. D., Rajagopalan, S., Pope, C. A., Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., & Kaufman, J. D. (2023). Air pollution and cardiovascular disease: A review of the literature. Current Environmental Health Reports, 10(4), 403–417.

Cevik, M., Erdem, H., & Yilmaz, O. (2019). The role of CBC in diagnosing mercaptan toxicity. Toxicology Reports, 6, 298–305.

Chan, A., Lee, J., Bhadra, S., Bortey-Sam, N., Hendry-Hofer, T. B., Bebarta, V. S., Mahon, S. B., Brenner, M., Logue, B., Pilz, R. B., & Boss, G. R. (2022). Development of sodium tetrathionate as a cyanide and methanethiol antidote. Clinical Toxicology, 60(3), 332–341.

Chang, Y. C., Huang, K. L., Lin, C. Y., Huang, H. C., & Huang, Y. L. (2019). Changes of oxidative stress, glutathione, and its dependent antioxidant enzyme activities in patients with hepatocellular carcinoma before and after tumor resection. Nutrients, 9(6), 623.

Chen, J., Wang, J., Lu, L., Zhang, Y., Xu, Y., Chen, J., & Han, X. (2019). Effects of methyl mercaptan on hematological and biochemical parameters and histological changes in rats. Regulatory Toxicology and Pharmacology, 107, 104423.

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178

Choi, Y., Kim, S., Lee, K., & Park, D. (2021). Exposure to volatile organic compounds and their effects on liver enzymes in workers at a shoe factory. Environmental Science and Pollution Research, 28(8), 10332–10340.

Dastgheib, L., Chen, X., & Wang, Y. (2020). Redox modulation in cancer therapy: The role of mercaptans and thiol groups in tumor progression. Biochemical Pharmacology, 177, 113985.

Dere E, Ari F. (2009). Effect of benzene on liver function in rats (*Rattus norvegicus).* Environ Monit Assess. (154):23–27.

Dreiem A., O. Myhre O. and Fonnum F. (2002). Relationship between lipophilicity of C6–10 hydrocarbon solvents and their ROS-inducing potency in rat cerebellar granule cells. Neurotoxicology, 23 (6): 701-709.

Drew, R. D., Rajagopalan, S., Pope, C. A., Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., & Kaufman, J. D. (2023). Air pollution and cardiovascular disease: A review of the literature. Current Environmental Health Reports, 10(4), 403–417.

Ekpenyong, C. E., & Asuquo, A. E. (2017). Recent advances in occupational and environmental health hazards of workers exposed to gasoline compounds. *International Journal of Occupational Medicine and Environmental Health, 30*(1), 1–26.

Fang, J., Xu, X., Jiang, L., & Qiao, J. (2019). Preliminary results of toxicity studies in rats following low-dose and short-term exposure to methyl mercaptan. Toxicology Reports, 6, 431–438.

Hsu, C. H., and Maibach, H. I. (1999). Toxicology of sulfur-containing gases. Toxicology and Industrial Health, 15(7), 480–493.

International Programme on Chemical Safety (IPCS) (1992). *Ethyl Mercaptan Health and Safety Guide*.

Ismail, A. U., Ibrahim, S. A., Gambo, M. D., Muhammad, R. F., Badamasi, M. M., & Sulaiman, I. (2023). Impact of differential occupational LPG exposure on cardiopulmonary indices, liver function, and oxidative stress in Northwestern city of Nigeria. *Science of the Total Environment, 862*, 160881.

Jia, Y., Wang, F., Liu, Y., & Chen, L. (2021). Occupational exposure to VOCs and their association with liver function biomarkers in petrochemical workers. International Journal of Environmental Research and Public Health, 18(4), 2067.

Koracevic, D. K., G., D., V., & Andrejevic, S. &. (2001). Total antioxidant capacity. J. Clin. Pathol. May, 356-361.

Kurmi OP, Dunster C, Ayres JG, Kelly FJ. (2013). Oxidative potential of smoke from burning wood and mixed biomass fuels. Free Radic Res. (47):829–835.

Liu, Y., Zhang, T., & Sun, J. (2020). Disruption of thiol/disulfide balance and its impact on the immune system in response to oxidative stress. Cellular Immunology, 354, 104163.

Maguire, J. F., Johnson, P. R., & Brown, S. L. (2021). Thiol/disulfide balance and its effect on immune cell function in response to mercaptan exposure. Immunology and Cell Biology, 99(5), 343–350.

National Institute for Occupational Safety and Health. NIOSH. (2016). *NIOSH Pocket Guide to Chemical Hazards: Ethyl Mercaptan (Ethanethiol)*.

Nawaz, S., Ahmed, F., Khan, M. A., & Qureshi, M. (2020). Mercaptan-induced oxidative damage to red blood cells and its effect on anemia. Environmental Toxicology, 35(3), 365–375.

Occupational Safety and Health Administration. (20١٨). Ethyl mercaptan (ethanethiol). U.S. Department of Labor. Retrieved June 3, 2025.

PubChem. (2025). Ethyl mercaptan. U.S. National Library of Medicine. Retrieved June 3, 2025.

Rowe, D. J., Khalil, T. A., Kammer, M. N., Godfrey, C. M., Zou, Y., Vnencak Jones, C. L., Xiao, D., Deppen, S., & Grogan, E. L. (2025). A deeper evaluation of cytokeratin fragment 21-1 as a lung cancer tumor marker and comparison of different assays. *Biosensors and Bioelectronics: X, 23*, 100593.

Sambo, A., Chinyere, A. N., Sunday, N. E., Henrietta, M. E., Denkok, Y., Obiora, R. E., Ukeme, E. A., Larrycarr, L. N., & Paul, C. U. (liu) (2022). Tumour necrotic factor alpha level and some biochemical parameters as a measure of health risks due to exposure to liquefied petroleum gas on vendors in Calabar, Nigeria. *International Journal of Tropical Disease & Health, 43*(20), 39–47

Savvidis, C., Kouroglou, E., Kallistrou, E., Ragia, D., Dionysopoulou, S., Gavriiloglou, G., Tsiama, V., Proikaki, S., Belis, K., & Ilias, I. (2024). IGFBP-2 in critical illness: A prognostic marker in the growth hormone/insulin-like growth factor axis. Pathophysiology, 31(4), 621–630.

Schmidt, M. J., Smith, A. L., Johnson, R. T., & Lee, H. K. (2019). Impact of redox modulation on IGF-I and IGFBP-2: A new perspective in endocrinology. Journal of Molecular Endocrinology, 62(1), R1–R10.

Singh, V., Patel, K., & Mehta, R. (2020). Thiol redox imbalance and inflammation: A critical pathway in cardiovascular disease. Antioxidants & Redox Signaling, 32(4), 271–283.

Tochukwu, U., Meludu, S. C., Dioka, C. E., Chikezie, O. D., Awalu, C. J., & Ibekailo, S. N. (2020). Comparative nephrotoxicity and hepatotoxicity effects of kerosene, gasoline, liquefied petroleum gas and biomass fuel exposure on male albino rats. *Asian Journal of Research in Biochemistry, 7*(4), 45–52.

U.S. Environmental Protection Agency. EPA (2025). Toxicological review of ethyl mercaptan. Retrieved June 3, 2025.

Włodek L. (2022). Beneficial and harmful effects of thiols. Pol J Pharmacol., 54 (3): 215-23.

World Health Organization (WHO). (2025). Ethyl mercaptan in the environment. Retrieved June 3, 2025.

Zhao, Y., Li, X., Wang, J., & Chen, L. (2020). Impact of mercaptans on platelet aggregation and white blood cell function. Journal of Clinical Hematology, 18(4), 243–251.

تقييم السمية والتغييرات الكيميائية الحيوية الناتجة عن التعرض لغاز البترولي المسال

إبراهيم حلمي ابراهيم احمد برغش[١]– **مجدي محمود محمد** [٢]– **ايناس فوزي جاب الله**[٣]– **رشا الشريف حسن** [٢]
١) قسم العلوم الاساسية البيئية، كلية الدراسات العليا والبحوث البيئية، جامعة عين شمس ٢) قسم الكيمياء الحيوية، كلية العلوم، جامعة عين شمس ٣) قسم الطب المهني والبيئي، كلية الطب، جامعة القاهرة

المستخلص

المركبتانات هي مركبات عضوية متطايرة تُستخدم في صناعات مثل تمييز الغاز الطبيعي وتصنيع المواد الكيميائية. قد يشكل التعرض المزمن أو المرتفع لهذه المواد مخاطر صحية خطيرة. تبين هذه الدراسة السمية المزمنة لمركب إيثيل مركبتان بين العمال الذكور المشاركين في عملية تعبئة أسطوانات الغاز. تم فحص ثمانين عاملاً ذكراً تتراوح أعمارهم بين ٣٠–٥٠ عاماً، وكان لديهم على الأقل ١٠ سنوات من العمل في مصنع ملئ الغاز البترولي السائل. تم تقسيمهم إلى مجموعتين: ٦٠ عاملاً مع تعرض مباشر (المصنّف بشكل فرعي حسب مدة العمل –١٠، ٢٠، أو ٣٠ سنة) و٢٠ عاملاً مع تعرض غير مباشر لمدة ٢٠ سنة. خضع جميع المشاركين لتقييمات سريرية ومخبرية في البداية وأثناء فترة عملهم. كشفت النتائج عن تغييرات صحية ملحوظة مرتبطة بالتعرض المزمن لمركب إيثيل مركبتان. ولاحظت الدراسة انخفاضًا ملحوظًا في القدرة الإجمالية لمضادات الأكسدة (TAC) وانخفاض الجلوتاثيون المختزل (GSH)، بالإضافة إلى زيادة في مركب المالومداهيد(MDA). يشير ذلك إلى زيادة الإجهاد التأكسدي. تأثرت وظائف الكبد، كما يتضح من ارتفاع مستويات ALT و AST وبليروبين الكلي. تمت الإشارة إلى خلل وظائف الكلى من خلال زيادة اليوريا والكرياتينين. كما لوحظ اضطراب صورة الدهون من خلال ارتفاع مستويات الكوليسترول الكلي والدهون الثلاثية. علاوة على ذلك دلت المستويات المرتفعة من IGHBP–٢ والهوموسيستين و٢١–١ (CYFRA٢١–١) وبروتين سي التفاعلي عالي الحساسية (HsCRP) على وجود التهاب وعوامل محتملة مبكرة لمخاطر سرطانية أو قلبية وعائية. وختاما، تؤكد الدراسة على الأثر السمي للتعرض المهني على المدى الطويل وتبرز ضرورة تحسين سلامة العمل والمراقبة المنتظمة لمستويات التعرض وتدابير الصحة الوقائية لحماية العمال من المخاطر.

الكلمات المفتاحية: الغاز النفطي المسال، الإيثيل مركابتان، الإجهاد التأكسدي، الخلل الكلوي، الخلل الكبدي

Vol. (54); No. (9); Spt. 2025
Print ISSN 1110-0826
Online ISSN 2636 - 3178