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Original Article

Assessment of some Hematological and Biochemical Changes Among Workers Exposed to Liquified Natural Gas in Egypt

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Abstract

Background: Natural gas (NG) is one of the world's three primary energy sources. However, workers in the liquefied natural gas (LNG) industry are routinely exposed to various occupational hazards, including mechanical, physical, chemical, and psychosocial factors, as well as risks of fires and explosions.

Objective(s): to assess some hematological and biochemical changes among workers exposed to Liquified Natural Gas in Egypt.

Methods: A comparative cross-sectional study was performed on a sample of 181 workers in LNG industry, assigned into two groups: An exposed group including workers exposed to the different industrial processes, and a non-exposed group including workers engaged in administrative and clerical activities at the same LNG Company. Data were collected by a structured interviewing questionnaire and by performing a general clinical examination and some laboratory investigations, as complete blood count, fasting blood glucose, Liver enzymes, renal functions tests, tumor markers tests as Carcinoembryonic Antigen (CEA), Cancer Antigen 125 (CA-125), Carbohydrate Antigen 19-9 (CA 19-9), Cancer Antigen 15-3 (CA 15-3) and Prostate-Specific Antigen (PSA). Indoor air contaminants as Methanol, Methyl Ethyl Ketone, Ethylene Glycol, Hexane, Benzene, total Volatile Organic Compound, Nitrogen Dioxide, Hydrogen Sulfide and Carbon Dioxide were measured inside the LNG plant and the resultant measurement were presented as midrange and compared to the equivalent Threshold Limit Value according to Egyptian law.

Results: The study revealed that there were statistically insignificant differences between exposed and non-exposed workers regarding all measured parameters except for some blood indices (HB, RBCs, Hematocrit and lymphocytic count) and some of the evaluated tumor markers, as Carcinoembryonic Antigen (CEA) and Cancer Antigen 125 (CA-125).. Regarding air levels of measured air contaminants all over the plant, they were lower than threshold limit values, except for Benzene which was coming from nearby industries.

Conclusion: LNG exposure is safe, and no health effects are expected if its contaminants are kept below their threshold limit values. Benzene was the offending factor coming from nearby industries.

Keywords: Liquified natural gas, hematological changes, biochemical Changes, exposed worker.

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INTRODUCTION

Natural Gas (NG) is one of the three most important energy sources (coal, oil, and natural gas), and it continues to be favored as an ecologically attractive fuel as it has the lowest impact on environmental pollution. [(1)] Natural gas occurs naturally throughout the world and is transported to residences, industries, and other countries by pipelines. It is liquefied to supply it to consumers almost anywhere in the world, which makes storage and shipping economically feasible. [(2, 3)]

Workers engaged in various liquefied natural gas (LNG) industrial processes may experience occupational diseases, as well as hematological and biochemical changes. Clinically, eyes, skin, lungs, liver, and kidneys may be affected. [(4)] Hematological, some adverse changes in certain parameters were observed, including a significant change in circulating erythrocytes, hemoglobin, platelets, total white blood cells, and absolute numbers of lymphocytes and neutrophils, which were reported among exposed workers. [(5)] Biochemically, changes in concentrations of liver transaminases, blood urea, serum creatinine, and uric acid are common and are used as indicators of liver and renal damage. [(3)] Furthermore, recent studies showed that people living within 500 feet of oil and liquefying natural gas facilities have a cancer risk eight times higher than the upper limit. [(4)] So, the screening tests, which detect either excess absorption (in the case of chemicals) or early adverse effects (from chemical, physical, or biological agents), could be useful tools in diagnosing clinical conditions, predicting clinical outcomes and risks, and providing objective measurements of ongoing diseases or injuries.

Egypt is working to become a regional center for trading energy in the Eastern Mediterranean region during the upcoming period and it was announced that Egypt is ready to cooperate with other countries in the region to receive gas, liquefy it, and re-export to Europe. Many studies contributed to the risks of fires and explosions in the LNG industry.[6, 7] However, there are no enough studies on early hematological and biochemical findings among LNG operators. Accordingly, this study was conducted to evaluate the impact of exposures on workers' health and to assess some of the hematological and biochemical changes, considering that exposed workers are engaged in the different processes of LNG industry according to work needs.

METHODS

Study Setting and Design

A Comparative cross-sectional study was conducted at a Liquefied Natural Gas (ELNG) company in Egypt, while laboratory assessments were conducted at the laboratories of the Occupational Health and Air Pollution Department, High Institute of Public Health, Alexandria University.

Assuming that the effect size of laboratory tests for workers exposed to Liquefied Natural Gas (LNG) was 0.5, which is considered a medium effect size according to Cohen's guidelines. [8] Using alpha error = 0.05 and a power of 80%; the minimum required sample size is 128 (64 for each group, exposed and non-exposed). The power was calculated using G. Power software.

All available workers in the ELNG company were enrolled in the study. The total number of workers was 181 They were assigned into two groups: An exposed group of 106 workers including those exposed to the different industrial processes, and non-exposed group of 75 workers including those engaged in administrative and clerical activities at the same LNG Company.

Data collection methods and tools:

Pre-designed structured interview questionnaire: A predesigned questionnaire was used to collect data from exposed and non-exposed workers. Content validity was assessed by three occupational health specialists who reviewed the items for relevance and clarity. The questionnaire was pilot-tested among 15 workers not included in the final analysis, and minor modifications were made to improve comprehensibility. Internal consistency reliability was confirmed with a Cronbach's alpha of 0.82 for the overall instrument. The final version was administered in paper format during working hours.

The questionnaire consisted of three parts. The first part gathered information about socio-demographic information, including age, residence and education. The second part gathered information about occupational data including current job description regarding, daily working hours, working days, working years and previous job description (if found) regarding nature of exposure and working years. The third part gathered information about relevant medical history. Lastly, the fourth part gathered information about smoking data including years of smoking and pack year index.

Medical examination: General examination was carried out for everyone; weight and height were measured and body mass index (BMI) was calculated according to Johnson et al. [9] Arterial blood pressure was measured according to Muntner et al, [10]

Laboratory investigations: [11] Blood samples were collected by aseptic techniques. Hands were washed at the start of each session before collecting blood, and a new pair of disposable gloves was used for each participant. Venous blood samples were collected from each participant using the WHO-recommended procedure for blood collection (World Health Organization, 2010).[12] Eight ml of blood were collected from each participant in vacationer tubes. The sample was divided into 3 portions, i.e. 2 ml for complete blood picture in CBC tubes containing EDTA, 2ml in sodium fluoride (NaF) tubes (a glycolysis inhibitor to preserve blood glucose level) and 4 ml in serum test tubes for detection of the other biochemical parameters as complete blood count, fasting blood glucose, liver enzymes, renal functions tests, and tumor markers, like: Carcinoembryonic Antigen (CEA) for colorectal cancer (also pancreatic, gastric, breast, and lung cancers); Cancer Antigen 125 (CA-125) for epithelial ovarian cancer (also endometrial and peritoneal malignancies); Carbohydrate Antigen 19-9 (CA 19-9) for pancreatic adenocarcinoma (also cholangiocarcinoma, gastric and colorectal cancers); Cancer Antigen 15-3 (CA 15-3) for breast cancer (especially advanced or metastatic disease) and Prostate-Specific Antigen (PSA) for prostate cancer.

Unopened kits were stored at 2-8 °C. Sera were obtained by separation at centrifugation 4000 rpm for 15 minutes. Complete blood counts (CBCs) were determined using an automated hematology analyzer based on the Coulter principle (impedance method) and flow cytometry with light scatter technology, according to the manufacturer's standard protocol. [13] Determination of creatinine was performed on Olympus au 400 using Jaffe colorimetric method based on reaction of creatinine with sodium picrate.

[14] Determination of urea was performed on Olympus au 400 using urease –GLDH: enzymatic UV test. [15] Determination of AST and ALT was performed on Olympus au 400 using optimize UV test according to IFCC determined from the rate of decrease of NADH. [16] Determination of glucose was performed on Olympus au 400 using enzymatic UV test (Hexokinase method). [17]

Determination of cholesterol was performed on Olympus au 400 using enzymatic colorimetric test for cholesterol with lipid clearing factor (CHOD-PAP-Method). Determination of triglycerides was performed on Olympus au 400 using enzymatic colorimetric test for triglyceride with lipid clearing factor (GPO-PAP-Method). [18] Determination of tumor markers (CEA, PSA, Ca15.3, Ca19.9 and Ca125) was performed on Cobas-E (Roche) using electrochemiluminescence immunoassay (ECLIA) sandwich principle. [19]

Indoor air contaminants measurement: Airborne concentrations of Methanol, Methyl Ethyl Ketone, Ethylene Glycol, Hexane, Benzene, and Carbon Dioxide (CO_2) were measured using a MIRAN SapphIRe 205B-XL1A3N-ST-AA infrared analyzer (Thermo Environmental Instruments Inc., Franklin MA, USA), based on non-dispersive infrared spectrophotometry. [20] The analyzer was zero-calibrated with clean air and span-calibrated using certified gas mixtures traceable to ISO 6141. [21] Total volatile organic compounds (TVOCs) were determined using a Casella VOC Pro photoionization detector (PID) (Casella CEL Ltd., Bedford, UK) equipped with a 10.6 eV UV lamp and calibrated with isobutylene gas standards. [22]
Nitrogen dioxide samples were analyzed by ion chromatography (IC) according to OSHA Method ID-182. [23]
Hydrogen sulfide concentrations were determined following the NIOSH Manual of Analytical Methods, Method 6013 (NIOSH, 1994). [24]

Statistical analysis: Data was fed and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp) [25] Qualitative data were described using numbers and percentages. The Shapiro–Wilk (S-W) test was used to verify the normality of distribution. Quantitative data were described using range (minimum and maximum), and median. The significance of the results obtained was judged at the 5% level and 95% confidence interval.

Ethical considerations: Approval of the Ethics Committee of the High Institute of Public Health was obtained before conducting the study. The study was conducted in compliance with the International Guidelines for Research Ethics. [26] Informed written consents were taken from all study participants after an explanation of the purpose and benefits of the research. Anonymity and confidentiality were assured and maintained, and there was no conflict of interest.

RESULTS

The normality of data distribution was assessed using the Shapiro–Wilk test. As the data did not follow a normal distribution ($p < 0.05$), non-parametric tests were applied. Accordingly, comparisons between two groups were performed using the Mann–Whitney U test.
Table (1) shows the distribution of age and smoking parameters among exposed and non-exposed groups. There was no significant difference in age distribution between exposed and non-exposed groups ($\chi^2 = 4.435$, $p = 0.218$). Smoking history was reported by 40.6% of the exposed and 49.3% of the non-exposed participants, with no significant difference ($\chi^2 = 1.369$, $p = 0.242$). The smoking index ranged 20–900 in the exposed and 40–600 in the non-exposed group, with median values of 282 and 209, respectively; with no statistical significance ($U = 3306.5$, $p = 0.054$).

Table (1): Distribution of age and smoking parameters among exposed and non-exposed groups to liquified natural gas

Age and smoking parameters	Exposed group (n = 106)		Non-exposed group (n = 75)		Test of Sig.	p
	No.	%	No.	%		
Age (years)						
20 – 30	19	17.9	15	20.0	χ^2= 4.435	0.218
30 – 40	34	32.1	30	42.7		
40 – 50	44	41.5	31	34.7		
50+	9	8.5	2	2.7		
Smoking history						
No	63	59.4	38	50.7	χ^2= 1.369	0.242
Yes	43	40.6	37	49.3		
Smoking index (SI) (Cigarettes per day × Years of smoking)						
Range	20.0 – 900.0		40.0 – 600.0		U = 3306.5	0.054
median	282		209			

χ^2 : Chi square test U: Mann Whitney test p: p value for comparing between the studied groups
*: Statistically significant at p< 0.05

Table (2) shows comparison between the 2 studied groups according to occupational history. . The duration of current work did not differ significantly between groups (χ^2 = 0.903, *p* = 0.825). Regarding previous occupations, 45.3% of the exposed group reported prior work involving the same hazards compared to none of the non-exposed group (χ^2 = 46.219, *p* < 0.001).

Table (2): Comparison of occupational history findings between exposed and non-exposed groups to liquified natural gas

Occupational history	Exposed group (n = 106)		Non-exposed group (n = 75)		Test of Sig.	p
	No.	%	No.	%		
Duration of current work (years)						
≤5 –	29	27.4	23	30.7	χ^2= 0.903	0.825
10 –	38	35.8	24	32.0		
15 –	26	24.5	21	28.0		
20+	13	12.3	7	9.3		
Previous work						
Exposed to same hazards	**48**	**45.3**	**0**	**0.0**	χ^2= 46.219*	<0.001*
Non-exposed to hazards	58	54.7	75	100.0		

χ^2 : Chi square test p: p value for comparing between the studied groups *: Statistically significant at p < 0.05

Table (3). No statistically significant differences between exposed and non-exposed groups regarding medical history were observed except for Hypertension (10.4% vs. 2.7%, *p* = 0.048)

Table (3): Comparison of medical history between exposed and non-exposed groups to liquified natural gas

Medical history	Exposed group (n=106) No. (%)	Non-exposed group (n=75) No. (%)	Test of sig χ^2 / FE	p-value
Allergic sinusitis	29 (27.4)	15 (20.0)	χ^2 = 1.293	0.256
Gastro-esophageal Reflux Disease (GERD)	13 (12.3)	8 (10.7)	χ^2 = 0.109	0.741
Hypertension	11 (10.4)	2 (2.7)	χ^2 = 3.917	0.048*
irritable Bowel syndrome (IBS)	10 (9.4)	12 (16.0)	FE	0.248
Ischemic Heart Disease (IHD)	9 (8.5)	2 (2.7)	FE	0.126
Diabetes	7 (6.6)	4 (5.3)	FE	1.000
Chronic headache	6 (5.7)	3 (4.0)	FE	0.738
Back pain	5 (4.7)	1 (1.3)	FE	0.403
Peripheral neuropathy	3 (2.8)	0 (0.0)	FE	0.268
Renal problems	2 (1.9)	0 (0.0)	FE	0.512
Dry eye	1 (0.9)	3 (4.0)	FE	0.308
Prostatitis	1 (0.9)	2 (2.7)	FE	0.571
Thyroid problems	1 (0.9)	0 (0.0)	FE	1.000
Hodgkin's lymphoma	1 (0.9)	0 (0.0)	FE	1.000
Anemia	0 (0.0)	1 (1.3)	FE	0.414
Rheumatoid	0 (0.0)	1 (1.3)	FE	0.414

χ^2 : Chi square test FE: Fisher Exact p: p value for comparing between the studied groups *: Statistically significant at p < 0.05

Table (4) represents comparison between the two groups studied regarding general examination. There was no statistically significant difference between the exposed and non-exposed groups. The median systolic blood pressure was 120 mmHg in both groups, with ranges of 100–150 mmHg in the exposed group and 90–150 mmHg in the non-exposed group (U = 3958, p = 0.958). Similarly, the median diastolic blood pressure was 80 mmHg in both groups, ranging from 70–110 mmHg among exposed workers and 60–100 mmHg among non-exposed workers (U = 3555, p = 0.145). The same was observed concerning body mass index (BMI).

Table (4): Comparison of general examination findings between exposed and non-exposed groups to liquified natural gas

General examination	Exposed group (n = 106)	Non-exposed group (n = 75)	U	p
Systolic blood pressure (mm Hg)				
Range	100.0 – 150.0	90.0 – 150.0	3958	0.958
Median	120.0	120.0		
Diastolic blood pressure (mm. .Hg)				
Range	70.0 – 110.0	60.0 – 100.0	3555	0.145
Median	80.0	80.0		
BMI (kg/m^2)				
Range	18.6 – 49.3	21.1 – 49.0	3968	0.984
Median	28.6	28.0		

U: Mann Whitney test p: p value for comparing between the studied groups

Table (5): Comparison of complete blood count, biochemical parameters and tumor markers between exposed and non-exposed groups to liquified natural gas

	Exposed group (n =106) Median (range)	Non-exposed group (n = 75) Median (range)	U	p
Complete blood count				
Hemoglobin (g/dl)	14.7 (12.3 – 19.0)	14.10 (10.4 – 16.50)	2929.0	0.003*
Hemoglobin %	92.0 (68.0 – 119.0)	88.0 (61.0 – 103.0)	2902.50	0.002*
R.B.Cs (10^6 cells/mm^3)	5.5 (4.5 – 7.)	5.46 (3.9 – 6.5)	3130.0	0.015*
Hematocrit %	46.1 (14.2 – 58.8)	44.40 (32.6 – 53.1)	2947.0	0.003*
M.C.V mm3	83.7 (24.4 – 91.6)	82.0 (56. – 96.7)	3736	0.491
M.C.H pg	26.5 (18.4 – 42.2)	26.60 (17.4 – 30.)	3936	0.911
M.C.H.C (g/dl)	31.9 (29.5 – 100.)	31.90 (28.9 – 36.5)	3895.5	0.819
RDW %	14.3 (12.5 – 17.6)	14.40 (12.5 – 37.9)	3462	0.139
Platelets (10^{3}cells/mm^3)	241.0 (90.0 – 439.0)	259.0 (101.0 – 410.0)	3339.5	0.067
WBCs (10^3 cells/mm^3)	6.85 (2.1 – 14.4)	6.90 (2.6– 12.1)	3462	0.139
Basophils (10^3 cells/mm^3)	0.0 (0.0 – 6.0)	0.0 (0.0 – 1.0)	3562.5	0.123
Eosinophil's (10^3 cells/mm^3)	2.0 (0.0 – 16.0)	2.0 (0.0 – 18.0)	3846.5	0.705
Neutrophils (10^3 cells/mm^3) Total	53.0 (29.0 – 74.0)	56.0 (23.0 – 70.0)	3276.5	0.121
Lymphocytes (10^3 cells/mm^3)	38.50 (18.0 – 62.0)	36.0 (24.0 – 70.0)	3134.0	0.048*
Monocytes (10^3 cells/mm^3)	6.0 (1.0 – 13.0)	5.0 (2.0 – 12.0)	3387	0.259
Biochemical parameters				
Fasting blood glucose(mg/dl)	76.0 (50.0 – 160.0)	77.5 (47.0 – 156.0)	3840.5	0.587
Urea (mg/dl)	31.0 (14.0 – 56.0)	32.0 (14.0 – 51.0)	3602	0.282
Creatinine (mg/dl)	0.97 (0.5 – 1.51)	0.93 (0.6 – 1.3)	3794	0.602
ALT (U/L)	20.50 (9.0 – 90.0)	19.0 (8.0 – 48.0)	3497.5	0.169
AST (U/L)	22.0 (10.0 – 124.0)	20.0 (11.0 – 96.0)	3271.0	0.042*
Cholesterol (mg/dl)	194.5 (114.0 – 312.0)	199.0 (78.0 – 328.0)	3670.5	0.38
Triglycerides (mg/dl)	130.5 (51.0 – 492.0)	112.0 (38.0 – 379.0)	3601.5	0.282
Tumor markers				
CEA (ng/ml)	1.74 (0.5 – 5.6)	1.46 (0.3 – 12.9)	3077.0	0.010*
PSA (ng/ml)	0.77 (0.2 – 10.5)	0.91 (0.2 – 11.7)	3564	0.237
Ca 19-9 (U/ml)	9.06 (0.6 – 60.6)	7.28 (0.6 – 50.5)	3810.5	0.636
Ca 125 (U/ml)	8.12 (2.1 – 28.1)	9.76 (2.9 – 25.6)	3247.5	0.036*
Ca 15-3 (U/ml)	16.35 (5.1 – 217.0)	16.90 (6.9 – 245.0)	3519	0.189

RBCs: Red blood cells, MCV: Mean Corpuscle Volume, MCH: Mean Corpuscle Hemoglobin, MCHC: Mean Corpuscle Hemoglobin Concentration, RDW: Red Cell Distribution Width, WBCs: White Blood Cells, ALT: Alanine Aminotransferase, AST: Aspartate Aminotransferase, CEA: Carcinoembryonic Antigen, CA-125: Cancer Antigen, CA 19-9: Carbohydrate Antigen 19-9, CA 15-3: Cancer Antigen 15-3. PSA: Prostate-Specific Antigen
U: Mann Whitney test
p: p value for comparing between the studied groups
*: Statistically significant at $p < 0.05$

Table (5) illustrates a comparison between the exposed and non-exposed workers according to CBC. A statistically significant increase regarding Hemoglobin (p =0.003), Hemoglobin% (p = 0.002), Red Blood Cells count (RBCs) (p = .015) and Hematocrit level (p = 0.003) was observed among exposed workers. On the other hand, the study revealed that there was no statistically significant difference between the two groups regarding mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC) and red cell distribution width (RDW) (p = 0.491, 0.911, 0.819, 0.139) respectively.

No statistically significant differences were observed between the two groups regarding platelets' count (p = 0.067) and white blood cell count (WBCs) whether total count (p = 0.139) or differential except for lymphocytes that were significantly higher among exposed group compared to non-exposed group (p = 0.048).

Regarding biochemical markers, serum creatinine, ALT, AST and triglycerides were observed to be higher, while fasting blood sugar, blood urea and serum cholesterol were found to be lower among exposed workers compared to non-exposed workers. However, the differences were insignificant for all parameters except for AST (p = 0.042).

The table also shows the values of tumor markers among exposed and non-exposed groups. CEA and Ca 19.9 values were higher among exposed workers, but the differences were insignificant, except for CEA (p = 0.010). On the other hand, values of PSA, Ca125 and Ca 15.3 were higher among non-exposed subjects compared to exposed workers and the difference was significant only for Ca 125 (p = 0.036).

Table (6) represents mid ranges of air levels of the measured air contaminants all over the plant. The results indicate that the measured levels were lower than the threshold limit values, except for benzene which was higher than its threshold limit value. It should be taken into consideration that benzene is not one of the contaminants of LNG industry and it's an air contaminant coming from a close industry.

Table (6): Measurement of air levels of different air contaminants in the ELNG plant

Measured Gases	Organic Gases						Inorganic Gases		
	Methanol	Methyl Ethyl Ketone	Ethylene glycol	Hexane	Benzene	TVOCS	NO2	H2S	CO2
Mid-range all over the plant (mg/m^3)	3	15	0.1	34	22*	13	1.5	3.95	64
TLV-TWA (mg/m^3)	**260**	**590**	**100**	**176**	**1.6**	**200**	**5.6**	**14**	**9000**

*TLV-TWA Threshold Limit Value- Time Weighted Average According to the Egyptian Environmental Law No 4-1994 Amended by Law No. 9-2009 and its Executive Law No. 1095-2011 and the decision of Minister of Manpower and Immigration No. 12-200

DISCUSSION

In the ELNG industry, workers are engaged in different processes according to work needs and without specifications, consequently, they are exposed to different adverse environmental factors and stressors. So, no specific exposure could be blamed for being the causative agent for specific effects. Many studies were conducted to assess the risks of fires and explosions in the LNG industry, [6, 27] but unfortunately without focus on the health status of the exposed workers and on changes in their hematological and biochemical parameters.

In the present study, the age distribution was similar in both groups, with most participants aged 30–50 years and no significant difference (p = 0.218). Smoking habits, including prevalence and duration, were also comparable between groups (p = 0.242 and p = 0.928, respectively), suggesting similar lifestyle exposure patterns. The smoking index was higher among the exposed group (median = 282 vs. 209), showing a borderline difference (p = 0.054), which may indicate a tendency toward greater cumulative smoking exposure among exposed workers.

The duration of current employment was similar across both groups, suggesting comparable work experience and minimizing the potential confounding effect of job duration (p = 0.825*).* Nearly half of the exposed workers (45.3%) also reported previous employment involving similar hazards, while none of the non-exposed participants had such history (p < 0.001). This finding highlights the likelihood of cumulative exposure among the exposed group, which may enhance their overall risk of adverse health outcomes related to chronic occupational exposure.

Most medical conditions did not differ significantly between groups; however, hypertension was significantly higher among exposed workers (10.4% vs. 2.7%, p = 0.048). This may reflect the combined effects of occupational and environmental exposures, particularly the elevated ambient benzene levels coming from outside ELNG plant from nearby industry. Chronic benzene exposure has been linked to oxidative stress, endothelial dysfunction, and autonomic imbalance, all of which can contribute to increased blood pressure. Similar findings were reported among gas industry workers exposed to hydrocarbons and environmental pollutants (Ismail et al., 2023). [28]

Results of general medical examination showed statistically insignificant differences between the two examined groups regarding systolic and diastolic blood pressures and body mass index (BMI). Similar studies in petrochemical and oil & gas workforces have reported higher risks of hypertension and obesity primarily in populations with prolonged, high-intensity exposures or additional risk modifiers such as age and shift work. [28, 29]This likely reflects the effectiveness of engineering controls and health surveillance programs in LNG facilities, as well as the influence of shared lifestyle and demographic factors that overshadow potential exposure effects.

Hematological analysis disclosed significantly higher hemoglobin levels, red blood cell counts, and hematocrit values in exposed compared to non-exposed workers ($p = 0.002$, 0.015, and 0.003, respectively), whereas MCV, MCH, MCHC, and RDW did not differ significantly. These results are consistent with studies documenting hematologic changes due to natural gas, [30] though they contrast with findings by Adienbo and Nwafor, [5] who reported reduced hematological indices among oil and gas workers. These observed hematological changes may be attributed to the inhalation of hazardous air pollutants, particularly benzene, a well-established hematotoxin, which might come from outside ELNG plants from nearby industry. Chronic exposure to benzene has been associated with bone marrow suppression and altered red cell parameters. [31]

White blood cell counts were modestly elevated in the exposed group, with only lymphocytes showing a statistically significant increase. This pattern may suggest an adaptive immunological response or early subclinical inflammation triggered by chronic low-level exposure to environmental contaminants. Verheyen et al. (2021) demonstrated increased lymphocyte counts in populations exposed to ambient air pollution. [32] In contrast, several studies of petrochemical and benzene-exposed workers have documented lymphocyte suppression and overall leukopenia, reflecting hematotoxic effects of more intense or prolonged exposures. [33, 34] These discrepancies highlight that the hematological response may vary according to the type of exposure, intensity, and individual susceptibility. Importantly, there is a paucity of recent peer-reviewed studies specifically examining hematological or biochemical biomarkers among workers in the liquefied natural gas (LNG) industry. Most existing LNG research has focused on safety engineering, emissions, and accident risk assessments rather than on worker health monitoring. Therefore, the current study contributes novel evidence by documenting lymphocytic changes among LNG workers, underscoring the need for further longitudinal and multi-site investigations to clarify the long-term health implications of such findings.

Regarding biochemical parameters, exposed individuals exhibited non-significant reductions in fasting glucose and cholesterol, with a mild increase in triglyceride levels. Although these changes did not reach statistical significance, they may reflect subtle metabolic alterations associated with chronic occupational exposures. Similar observations have been reported in other fuel-related industries: Guo et al. (2024) found that male gas station workers exposed to methyl tetra-butyl ether (MTBE) exhibited elevated fasting glucose and increased triglyceride–glucose index values, suggesting early metabolic disruption.[35] Likewise, Wang et al. (2025) reported that long-term improvements in ambient air quality were associated with significant reductions in fasting glucose, cholesterol, and triglycerides, underscoring the sensitivity of these biomarkers to environmental exposures. [36] In contrast to these populations, LNG workers in the present study showed only mild, non-significant trends, which may indicate either lower exposure intensity or the influence of different exposure components. This suggests that while LNG operations may induce subtle metabolic responses, the magnitude of effect appears smaller compared to other fuel-handling occupations.

Liver enzyme analysis revealed elevated mean AST and ALT levels among exposed workers, with AST showing statistical significance. This might suggest the presence of mild hepatocellular stress that may be related to chronic low-level occupational exposures. Similar enzyme suggestions have been reported in recent petrochemical and fuel-handling studies. Zhang et al. (2022) observed significant increases in liver enzymes among workers chronically exposed to benzene, toluene, and xylene, indicating that even moderate solvent exposure can disrupt hepatic function.[37] Qiao et al. (2023) likewise demonstrated hepatic and splenic injury associated with benzene exposure in experimental models, reinforcing the sensitivity of AST and ALT to hydrocarbon-related toxicity. [33] Abou-ElWafa et al. (2015) documented elevated liver enzymes among petrol station attendants, consistent with occupational exposure to volatile hydrocarbons. [38, 39] While LNG itself is considered relatively safe when air contaminants remain below threshold limits, the significant rise in AST observed in this study may reflect either low-level chronic exposure to associated hydrocarbons or confounding from possible external benzene emissions or other hydrocarbons from nearby plants.

Renal function, assessed via blood urea and serum creatinine, indicated higher mean values in the exposed group, though differences were not significant, consistent with Abou-ElWafa et al. [38]

Importantly, tumor marker analysis revealed significant increases in CEA and CA125 among exposed workers. These results echo recent findings from the Colorado School of Public Health, suggesting elevated cancer risk near oil and gas facilities. While the exposure to benzene from adjacent industrial sources may contribute, other environmental and occupational carcinogens must also be considered . [40]Importantly, tumor marker analysis revealed significant increases in CEA and CA125 among exposed workers. Although these markers are non-specific and can be influenced by smoking, inflammation, and benign conditions, their elevation may nonetheless reflect early biological effects of chronic hydrocarbon exposure. Similar observations have been reported in fuel-handling and petrochemical settings. For example, a 2025 study of Egyptian gasoline station workers documented higher serum CEA levels in exposed individuals, linking these changes to chronic benzene and volatile organic compound exposure. [41] Likewise, workers in petrochemical plants and coking industries have shown elevations in CEA and other tumor markers, suggesting possible early neoplastic or inflammatory activity associated with long-term chemical exposure. [42] Taken together, the increases in CEA and CA125 observed in LNG workers in the present study are consistent with reports from other fuel-related industries, and while not diagnostic of cancer, they highlight the potential for low-level exposures to influence tumor-associated biomarkers.

All measured air contaminants were within acceptable limits, except for benzene, which exceeded the threshold limit value (TLV) due to emissions from adjacent industrial sources rather than internal LNG operations. This external benzene contamination may partly account for the mild hematological and biochemical alterations observed among exposed workers. Recent studies have demonstrated that even low-level ambient benzene exposure can induce oxidative stress, inflammatory responses, and subtle hematopoietic changes. [43]

In Egypt, LNG facilities operate under national occupational safety laws and environmental regulations that require employers to provide safe working conditions, monitor exposures, and conduct regular medical examinations of workers. These rules, combined with company-level HSE standards such as leak detection, ventilation systems, and protective equipment, help to minimize exposure to harmful chemicals. Unlike refineries or gasoline stations, LNG plants mainly deal with methane and have much lower levels of benzene and other toxic hydrocarbons. This strict regulatory environment likely explains why our study showed only weak and non-significant correlations between exposure and blood or tumor markers.

Significance of the study: This study provides one of the first systematic assessments of hematological and biochemical alterations among workers in the liquefied natural gas (LNG) industry in Egypt. While most LNG research to date has focused on safety engineering, emissions control, and accident risk, very few investigations have examined early biological effects in exposed workers. By documenting significant differences in red blood cell indices (hemoglobin, RBC count, hematocrit), lymphocyte counts, liver enzyme activity (AST), and tumor markers (CEA, CA125), the present study contributes novel evidence on potential health effects in this occupational group. Moreover, the lack of strong correlations between hematological or biochemical parameters and personal variables (age, smoking, duration of employment) underscores that occupational exposures, rather than demographic or lifestyle factors, are the most likely contributors. This supports the value of routine medical surveillance—including complete blood counts, liver function tests, and tumor markers—as an early warning system for LNG workers.

Limitations of the study: This study had several limitations that should be considered when interpreting the findings. First, the cross-sectional design restricts the ability to establish temporal or causal relationships between exposure to liquefied natural gas and the observed hematological and biochemical changes. Second, all workers in the company were selected without randomization. Third, the study was conducted at a single site with a relatively modest and demographically homogeneous sample, which may limit the generalizability of the results to other LNG facilities or more diverse worker populations. Fourth, only selected hematological, biochemical, and tumor markers were assessed, while other sensitive biomarkers such as oxidative stress, genetic damage, or immunological parameters were not included. Fifth, environmental air levels measurement should be done in different places, different times of the day and different days. Finally, all laboratory investigations were performed at a single point in time, which does not capture biological variability, and the potential influence of residual confounders, such as unmeasured environmental or lifestyle factors, cannot be fully excluded.

CONCLUSION AND RECOMMENDATIONS

In our study, some hematological and biochemical changes were detected among exposed workers, mainly in their CBC parameters, liver enzyme AST, and tumor markers (CEA and Ca125). Concerning air contaminants, the measured values were below TLV levels except for benzene. It is difficult to attribute these adverse health effects to a certain exposure since exposed workers are engaged in the different processes of the LNG industry according to work needs. Moreover, it is difficult to attribute these effects to any of the constituents of LNG as all the measured values in air were below their TLV. Exposure to factors outside the work environment as exposure to benzene from a nearby industry might be blamed as an offensive pollutant.

It is recommended that all workers in the LNG industry should undergo pre-placement medical examination, including a proper medical history, a thorough medical physical examination carried out by occupational health physician and blood tests, including complete blood count, kidney, liver, and some tumor markers assessment. Also, periodic medical examinations must be conducted for early detection of health impairment. Application of personal protective equipment (PPE) for exposed workers is a must to reduce exposure to occupational contaminants.

CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

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REFERENCES

1. Šebalj D, Mesaric J, Dujak D. Predicting Natural Gas Consumption - A Literature Review. In Central European Conference on Information and Intelligent Systems 2017 (pp. 293-300). Faculty of Organization and Informatics Varazdin. CECIIS Template 2017.
2. Aktemur C. An overview of natural gas as an energy source for various purposes. International Journal of Engineering Technologies IJET. 2017;3:91-104.
3. Sirdah MM, Al Laham NA, El Madhoun RA. Possible health effects of liquefied petroleum gas on workers at filling and distribution stations of Gaza governorates. East Mediterr Health J. 2013 Mar 1;19(3):289-94.
4. McKenzie LM, Witter RZ, Newman LS, Adgate JL. Human health risk assessment of air emissions from development of unconventional natural gas resources. The Science of the total environment. 2012;424:79-87.
5. Adienbo O, Nwafor A. Effect of Prolong Exposure to Gas Flaring on some Haematological Parameters of Humans in the Niger Delta Region of Nigeria. Journal of Applied Sciences and Environmental Management. 2010;14.
6. Kang K, Wang X. A Stepwise Risk Assessment for Explosion Events considering Probability Distribution of Explosion Load Parameters. Complexity. 2018;2018:1-14.
7. Li J, Huang Z. Fire and Explosion Risk Analysis and Evaluation for LNG Ships. Procedia Engineering. 2012;45:70-6.
8. Cohen J. Statistical power analysis for the behavioral sciences. 2 ed. Hillsdale, NJ: Lawrence Erlbaum Associates; 1988.
9. Johnson W, Norris T, Bann D, Cameron N, Wells JK, Cole TJ, et al. Differences in the relationship of weight to height, and thus the meaning of BMI, according to age, sex, and birth year cohort. Annals of human biology. 2020;47(2):199-207.
10. Muntner P, Shimbo D, Carey RM, Charleston JB, Gaillard T, Misra S, et al. Measurement of Blood Pressure in Humans: A Scientific Statement From the American Heart Association. 2019;73(5):e35-e66.
11. Burtis CA, Ashwood ER, Bruns DE. Tietz textbook of clinical chemistry and molecular diagnostics-e-book: Elsevier Health Sciences; 2012.
12. World Health Organization. WHO guidelines on drawing blood: best practices in phlebotomy. Geneva: WHO Press; 2010.
13. Tantanate C, et al. Performance evaluation of automated hematology analyzers in routine clinical practice. International Journal of Laboratory Hematology. 2020;42(2):e38-e41.
14. Boss K, et al. Comparative accuracy of Jaffe and enzymatic creatinine assays: insights from clinical evaluations. Clinical Kidney Journal. 2023;16(11):2147-55.
15. Tzovaras V, et al. Performance of enzymatic urea assays on clinical chemistry analyzers: a multicenter evaluation. Clinical Biochemistry. 2021;92:25-30.
16. Talli I, et al. Methodological innovations in AST/ALT measurement: pyridoxal-5′-phosphate effects. Clinical Chimica Acta. 2025;12:105-12.
17. Ceriotti F, et al. Reference measurement procedures for glucose: the hexokinase enzymatic method. Clinical Chemistry and Laboratory Medicine. 2020;58(7):1121-7.
18. Guder WG, et al. Enzymatic methods for cholesterol and triglycerides: update and analytical performance. Clinical Chimica Acta. 2019;495:117-25.
19. Roche D. Elecsys CEA/PSA/CA 15-3/CA 19-9/CA 125 II immunoassays: Method sheet. Mannheim, Germany: Roche Diagnostics; 2020.
20. Thermo Environmental Instruments Inc. MIRAN SapphIRe Series 205B Portable Ambient Air Analyzer: Operation and Service Manual. Franklin (MA): Thermo Environmental Instruments Inc.; 2003 [cited 2025 Oct 30]. Available from: https://www.thermofisher.com.
21. International Organization for Standardization (ISO). ISO 6141:2015 — Gas analysis: Contents of certificates for calibration gas mixtures. 4th ed. Geneva (CH): ISO; 2015 [cited 2025 Oct 30]. Available from: https://www.iso.org/standard/62190.html.
22. Casella CEL Ltd. VOC Pro PID gas monitor: operator’s manual. Bedford (UK): Casella CEL Ltd.; 2001 [cited 2025 Oct 30]. Available from: https://www.casellasolutions.com/uk/en.html.
23. Occupational Safety and Health Administration. Nitrogen dioxide in workplace atmospheres: Method no. ID-182. Salt Lake City (UT): U.S. Department of Labor, OSHA Technical Center; 1991 [cited 2025 Oct 30]. Available from: https://www.osha.gov/analyticalmethods/inorganic/id182.
24. National Institute for Occupational Safety and Health (NIOSH). Hydrogen sulfide: Method 6013, Issue 1. In: NIOSH Manual of Analytical Methods (NMAM), Fourth Edition. Cincinnati (OH): U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention; 1994 [cited 2025 Oct 30]. Available from: https://www.cdc.gov/niosh/nmam/pdfs/6013.pdf.
25. I. B. M. Corp. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp.; 2011.
26. Sciences CfIOoM, Organization WH. International ethical guidelines for health-related research involving humans. Geneva: CIOMS; 2016.
27. Jianhua L, Zhenghua H. Fire and Explosion Risk Analysis and Evaluation for LNG Ships. Procedia Engineering. 2012;45:70-6.
28. Ismail AU, Ibrahim SA, Gambo MD, Muhammad RF, Badamasi MM, Sulaiman I. Impact of differential occupational LPG exposure on cardiopulmonary indices, liver function, and oxidative stress in Northwestern city of Nigeria. Science of The Total Environment. 2023;862:160881.
29. Mannocci A, Pignalosa S, Nicosia V, Saulle R, Sernia S, La Torre G. Cardiovascular Diseases Risk Factors in oil and gas workers: a ten years observational retrospective cohort. Annali di igiene : medicina preventiva e di comunita. 2016;28(2):122-32.
30. Saadat M, Bahaoddini A. Hematological changes due to chronic exposure to natural gas leakage in polluted areas of Masjid-i-Sulaiman (Khozestan province, Iran). Ecotoxicology and

environmental safety. 2004;58(2):273-6.
31. Okoro AM, Ani EJ, Ibu JO, Akpogomeh BA. Effect of petroleum products inhalation on some haematological indices of fuel attendants in Calabar metropolis, Nigeria. Nigerian journal of physiological sciences : official publication of the Physiological Society of Nigeria. 2006;21(1-2):71-5.
32. Verheyen VJ, Remy S, Bijnens EM, Colles A, Govarts E, Martin LR, et al. Long-term residential exposure to air pollution is associated with hair cortisol concentration and differential leucocyte count in Flemish adolescent boys. Environmental Research. 2021;201:111595.
33. Qiao Y, Hu H, Zhao Y, Jin M, Yang D, Yin J, et al. Benzene induces spleen injury through the B cell receptor signaling pathway. Ecotoxicology and environmental safety. 2023;257:114924.
34. Zhang Z, Liu X, Guo C, Zhang X, Zhang Y, Deng N, et al. Hematological Effects and Benchmark Doses of Long-Term Co-Exposure to Benzene, Toluene, and Xylenes in a Follow-Up Study on Petrochemical Workers. 2022;10(9):502.
35. Mannocci A, Pignalosa S, Saulle R, Sernia S, De Sanctis S, Consentino M, et al. Prevalence of major cardiovascular risk factors among oil and gas and energy company workers. Annali dell'Istituto superiore di sanita. 2015;51(2):148-53.
36. Wang M, Sang C, He Y, Chen H, Jia J, Zheng S. Exploring the effects of altered concentrations of PM2.5 and its constituents on the incidence risk of type 2 diabetes mellitus and glucose and lipid indicators: a cohort study in Northwestern China. BMC Public Health. 2025;25(1):3030.
37. Zhang Z, Liu X, Guo C, Zhang X, Zhang Y, Deng N, et al. Hematological Effects and Benchmark Doses of Long-Term Co-Exposure to Benzene, Toluene, and Xylenes in a Follow-Up Study on Petrochemical Workers. Toxics. 2022;10(9).
38. Abou-ElWafa HS, Albadry AA, El-Gilany AH, Bazeed FB. Some Biochemical and Hematological Parameters among Petrol Station Attendants: A Comparative Study. BioMed research international. 2015;2015:418724.
39. Hu Y, Chen B, Qian J, Jin L, Jin T, Lu D. Occupational coke oven emissions exposure and risk of abnormal liver function: modifications of body mass index and hepatitis virus infection. Occupational and environmental medicine. 2010;67(3):159-65.
40. Ajugwo A. Negative effects of gas flaring: The Nigerian experience. Journal of Environment Pollution and Human Health. 2013;1:6-8.
41. Allam AA, Taha ST, Aziz SP, Shokry MA, Ahmed NMJTEJoH. Monitoring risks for hematological changes in gas station workers. 2025.
42. Lakshmi A, Lakshmanan A, Kumar G, A S. Effect of intensity of cigarette smoking on haematological and lipid parameters. Journal of clinical and diagnostic research : JCDR. 2014;8(7):Bc11-3.
43. Wang J, Ma Y, Tang L, Li D, Xie J, Sun Y, et al. Long-Term Exposure to Low Concentrations of Ambient Benzene and Mortality in a National English Cohort. American journal of respiratory and critical care medicine. 2024;209(8):987-94.