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Original Article

Effects of Gasoline and Smoking on Lipid Profile and Liver Functions among Gasoline Exposure Workers in Iraq

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ABSTRACT

Introduction: The rapid and recent rise in the pandemic of cardiovascular disease implies that the environment plays a significant role. Numerous biological systems, such as the cardiovascular, blood-forming organs, liver, and kidneys, can be affected by gasoline and smoking. Because filling station employees, repair service workers, gasoline truck drivers, and refinery workers are all at a greater risk of being exposed to gasoline fumes. Even though gasoline and smoking have been investigated for so many years, few studies have looked into the effects of gasoline exposure combined with smoking on a variety of physiological mechanisms. As a result, we propose that combining gasoline exposure with smoking is a risk factor for cardiovascular diseases and impaired hepatic function.

Methods: The study included 95 male adult volunteers who worked with gasoline and were exposed to different fuel derivatives as study group and age and sex-matched seemingly healthy non-exposed people as the controls. Questionnaire interviews were used to collect socio-demographic data and a standard technique was used to collect the blood samples. The levels of cholesterol, HDL4, LDL-C, triglyceride, and VLDL were measured, as well as for liver enzymes ALP, AST, ALT, indirect bilirubin, direct bilirubin, and total bilirubin were measured.

Results: Our data suggest that smoking with gasoline exposure causes an increase in total and bad cholesterol levels, as well as a significant shift concerning the control group in lipid profile and liver enzymes. the exposed group had higher levels of ALP, and AST and significantly increased. In the nonsmoker exposed group D-bilirubin decreased in comparison to the control and exposed smoker group.

Conclusion: This research concluded that the liver enzymes (ALP, AST, ALT) were higher among workers who smoke and are exposed to gasoline than in control subjects, similarly, the bad cholesterol also increase. Therefore, people who smoke and are handled with gasoline are at a higher risk of having heart and hepatic diseases.

Keywords: Bilirubin, Cardiovascular, Cholesterol, Gasoline, Hepatoxicity.

Introduction

Waste management is identified as one of many severe environmental problems. Waste problems can surely disrupt the environment around the community, such as soil pollution. A polluted environment will also have an impact on public health. There are several causes for the increase in waste, specifically the increase in population, the level of community activity, the socio-economic level of the community, technological advances, and also the pattern of people's lives.[1]

Technological boosts also increase the amount of

waste. It can be seen from the use of personal devices and frequently updating them to the latest ones.[2]

The world population is rapidly increasing Gasoline, a highly flammable liquid mixture, is the primary contaminant in the environment and is mainly utilized for the internal combustion of vehicles.[1] The majority of them are made up of hydrocarbons (aromatic, saturated, and unsaturated) and non-hydrocarbons (N, S, O2, vanadium, and nickel).[2] Because the gasoline vapor concentration in the atmosphere (approximately 2000 ppm) is not healthy when breathed even for a brief time (seconds), filling station personnel, service station attendants, gasoline truck drivers, and refinery workers are all at heightened risk from exposure to gasoline fumes.[3] The most common method of exposure is inhalation, but cutaneous absorption is also possible. It is important to note that production employees, distribution, and gasoline usage are all in danger of acute or chronic toxicity.[4]

Smoking tobacco involves thousands of compounds that have detrimental and poisonous effects on the body. Alterations in lipid profile, increased insulin resistance, decreased nitric oxide (NO) availability, endothelial dysfunction, increased insulin resistance, platelet dysfunction, high blood viscosity, alternations in fibrinolysis, ongoing inflammatory responses with rising inflammatory markers, and more recently free radicals-mediated oxidative stress play a vital role in the mediation of atherothrombosis appear to play an important role in the mediation of atherothrombotic.[5] Smoking cigarettes raises plasma catecholamine levels, which causes lipolysis and the production of free fatty acids, which the liver absorbs. The lipid-driven inflammatory disease of the artery wall is known as atherosclerosis.[6] Low-Density Lipoprotein-Cholesterol (LDL-C) and Very Low-Density Lipoprotein-Cholesterol (VLDL-C) are toxic to cells, while HDL-C (High-Density Lipoprotein-Cholesterol) is a protective factor against coronary atherosclerosis.[5] Tobacco smoking has been linked to higher levels of total cholesterol, triglycerides, LDL–C, VLDL, and lower levels of HDL–C, according to a previous study.[7,8,9] Other research, however, has yielded contradictory outcomes.[10] Cigarette smoking appeared to accelerate atherosclerosis in part due to its effect on lipid profiles.[11,12] It's also been discovered that the amount of cigarettes smoked is closely tied to the chance of developing cardiovascular diseases LDL-C, IDL-C, and VLDL are all potentially atherogenic apoB-containing lipoprotein particles. Non-HDL-C is a progressive measure that includes all possibly atherogenic apoB-containing lipoprotein particles.[13]

The liver is the body's main gland, and it plays a variety of roles in the control of numerous physiological activities. As a result, fatal liver disease could be the major cause of mortality. Drug-induced liver damage would be one of those life-threatening disorders that necessitate extensive clinical and surveillance assessments.[14] Breathing small quantities of gasoline fumes can cause nasal and throat irritation, as also headaches, dizziness, nausea, vomiting, confusion, and breathing difficulties. Dermal contact with gasoline can cause rashes, redness, and swelling, along with many other symptoms. Although allergic reactions (hypersensitivity) have been observed, they are uncommon.[15] Long-term exposure to gasoline can cause hepatotoxicity, and the severity of benzene poisoning is dependent on the amount, route, and length of time of exposure, as well as the exposed person's age and pre-existing medical condition.[16]

The most typically requested tests for heart and liver investigations are lipid profile and liver function tests (LFTs).[17] As a result, the goal of this study was to determine the impact of gasoline exposure combined with smoke on the lipid profile parameters and liver function tests in gasoline station workers in Zakho, Duhok City, Kurdistan Region, Iraq.

Methods

The study included 95 male adult volunteers from Zakho city, who worked with gasoline and were exposed to different fuel derivatives and provided

informed consent to participate in the research. Age and sex-matched seemingly healthy non-exposed people from various locations in Zakho served as the controls. Participants with the following criteria were excluded from the study: people diagnosed with cardiovascular disorders, those with a family history of malignancies, subjects with chronic renal and respiratory disease, individuals on corticosteroid therapy, radiotherapy or chemotherapy, liver damage or disease, and those who were already taking medication that affected their cardiovascular and liver functions.

Questionnaire interviews were used to collect data over three months, concentrating on socio-demographic information, periods of exposure, time of working (hours/day), health conditions, and habits of smoking. A standard technique was used to collect the blood. To reduce errors, the blood sample container was tagged with the participant's name. A blood sample was collected from the peripheral vein on the arm of each of the volunteers by venipuncture using a sterile 10ml needle and syringe. 7mls of venous blood was taken and 4mls were transferred immediately into a sterile labeled plain vial while 3mls were transferred into well-label potassium EDTA anticoagulant vials. The blood in the plain vial was allowed to clot and retract. It was centrifuged at 18000g, serum extracted, for heart and liver enzyme assay.

This investigation was carried out after the research and ethical committee of Zakho University's Department of Biology Faculty of Science proved (5/238) its permission. To get authority to conduct biochemical analysis in the central laboratories, an official letter of request was issued to Bedare Hospital. After describing the study's goal, all research participants gave their written informed consent to participate willingly.

Data obtained were presented as median ± S.E.M. One-way ANOVA followed by Bonferroni post hoc test comparison was used to compare among control, non-smoking exposure, and smoking exposure groups. All statistical tests were two-tailed and a ($P \leq 0.05$) was considered statistically significant. All the calculations and statistical analyses were performed using GraphPad Prism 7 (GraphPad Software, San Diego, California, USA).

Results

The information about the participants' demographics was acquired from a record based on the service station employees. The participants' ages ranged from 23 to 53 years for nonsmokers (mean 33.43 years), 31.82 for smokers in the exposed group, and 20 to 53 years for the unexposed group (mean 40.03 years). Workers were employed for 3 to 35 years and were exposed to various fuel derivatives for at least 10 hours per day (Table 1).

Figures 1, 2, and Table 2 show the mean level of cholesterol (mg/dl), HDL-4 (mg/dl), LDL-C (mg/dl), Triglyceride (mg/dl), VLDL (mg/dl), and Liver enzymes of workers exposed to gasoline including smokers and non-smokers in comparison to the unexposed group. The mean results of the exposed non-smoker group provided the following values for cholesterol (161.9 ± 2.95), HDL-4 (39.03 ±1.23), LDL-C (120.2 ±1.16), Triglyceride (177.9 ±37.54), and VLDL (20.08 ±1.12). Whereas, in the case of the exposed smoker group, mean values of (165.7 ± 3.19), (39.38 ±1.26), (118.7 ±2.41), (121.2 ±6.55), and (23.38±1.39) were obtained for cholesterol (mg/dl), HDL-4 (mg/dl), LDL-C (mg/dl), Triglyceride (mg/dl), and (VLDL (mg/dl), respectively. For Control were cholesterol (126.9 ± 6.01), HDL-4 (44.98 ±0.64), LDL-C (102.2 ±2.28), Triglyceride (107.3 ±2.597), VLDL (22.11 ±1.288). Comparing the two sub-groups with the control indicated, the (cholesterol, HDL-4, and LDL-C) parameters were significantly different, whilethe parameters for (Triglyceride and VLDL) were insignificant.

Table 1. Some of the demographic Criteria for Participation

Demographic Criteria	control	Non-Smoker Exposure	Smoker Exposure
Age	40.03	33.43	31.82
Weight	75.33	79.64	75.61
Years/work	-	11.43	16
Exposure hours	-	10	10
packet	-	NO	1-4 packet

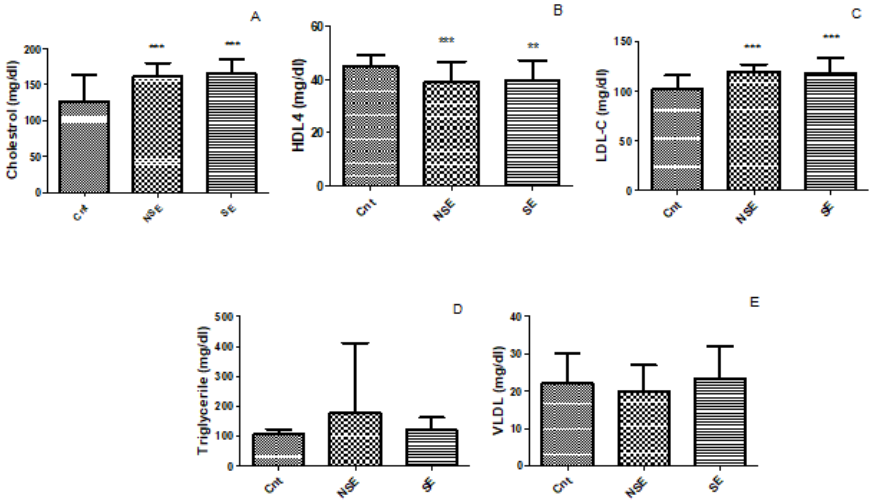

Figure 2. Comparison graph for levels of total plasma cholesterol (A), LDL-4 (B), HDL-C (C), triglycerides (D), and VLDL (E) in the Non-smoker (NSE) and smoker (SE) group compared with non-exposed controls (cnt); *: $p<0.05$.

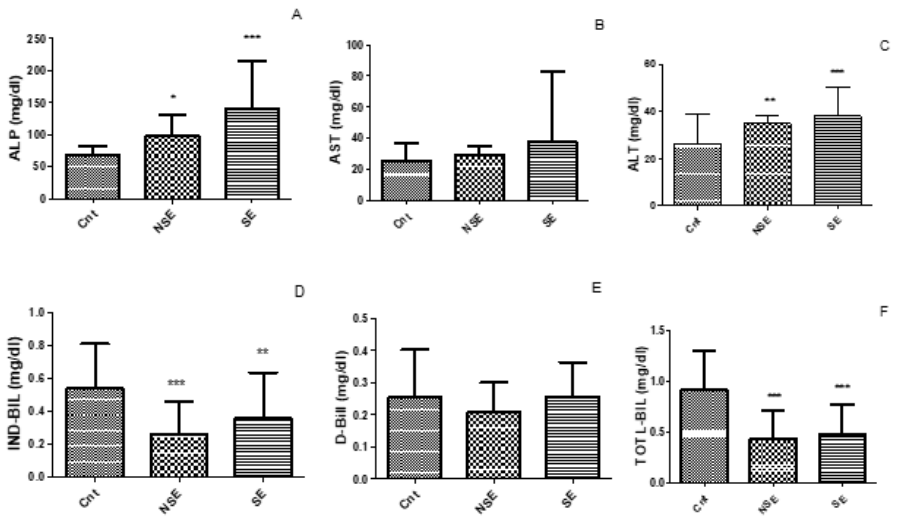

Figure 2. Comparison graph for levels of Liver enzyme: ALP (A), AST(B), ALT (C), IND-BIL(D), D-BIL (E), Total Bill (F) in the Non-smoker and smoker group compared with non-exposed controls; *: $p<0.05$

Table 2. The effect of gasoline inhalation on lipid profile parameters among smoker and nonsmoker gasoline exposure workers

Parameter	control	Non-Smoker Exposure	Smoker Exposure	P-value
Cholesterol (mg/l)	126.9 ± 6.01	161.9 ± 2.95 ***	165.7 ± 3.19 ***	< 0.0001
HDL4 (mg/l)	44.98 ±0.64	39.03 ±1.23 ***	39.38 ±1.26 **	< 0.0001
LDL-C (mg/l)	102.2 ±2.28	120.2 ±1.16 ***	118.7 ±2.41 ***	< 0.0001
Triglyceride (mg/l)	107.3 ±2.597	177.9 ±37.54	121.2 ±6.55	0.0603
VLDL (mg/l)	22.11 ±1.288	20.08 ±1.12	23.38±1.39	0.1829

Values are means±SEM (n=96), a P<0.05, significant change concerning the control group, ***changes between P<0.01 and P<0.001, SEM: Standard error of the mean, HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol, VLDL: Very-low-density lipoprotein cholesterol.

The blood ALP level for the exposed group of smokers was 140.7± 11.85 mg/l higher than the exposed group of non-smokers which was 98.14 ± 4.99 mg/l, both of them were significant when compared with the control. On the other hand, the AST concentration for the exposed group of smokers was 38.1 ± 1.95 mg/l which was insignificantly higher than the mean value for the non-smokers exposed group 34.93 ±0.49 mg/l, both of them were insignificant when compared with control. The mean levels of alanine transaminase (ALT) of all participants were evaluated. The result revealed a significant (P=0.05) increase in ALT in the smoker and non-smoker exposed groups (34.93±0.49 and 38.1±1.95 mg/l), respectively, when compared with the control (26.54 ± 1.98 mg/l). However, the D-BILL concentration for the exposed group of smokers was 0.2556 ± 0.017 mg/l which was insignificantly higher than the mean value for non-smokers in the exposed group (0.2082 ±0.02 mg/l). The result of the study revealed a significant decrease (P≤0.001) in the mean IND-BILL in the nonsmoker-exposed group. A significant decrease was noted between both control and non-smokers exposed groups for both total bilirubin and indirect bilirubin. Data from Table 3 indicated indirect bilirubin in the exposed group of smokers increased significantly in comparison to the control. On the other hand, the mean of total bilirubin decreased significantly in the non-smoker smoke-exposed group in comparison to the control (0.911± 0.062 control, 0.4369± 0.044 non-smokers, and 0.4805± 0.046 smokers). (Figure 2, Table 3).

Table 3. The effect of gasoline inhalation on liver function among smoker and nonsmoker gasoline exposure workers.

Parameter	Control	Non-Smoker Exposure	Smoker Exposure	P-value
ALP (mg/l)	66.92 ± 2.38	98.14±4.99 *	140.7± 11.85 ***	< 0.0001
AST (mg/l)	25.67 ± 1.81	29.45 ± 0.89	37.69 ± 7.24	0.1391
ALT (mg/l)	26.54 ± 1.98	34.93 ±0.49 **	38.1 ± 1.95 ***	< 0.0001
D-BILL (mg/l)	0.254 ± 0.02	0.208 ±0.015	0.256 ± 0.017	0.1444
IND-BILL (mg/l)	0.5385± 0.04	0.2628 ± 0.03 ***	0.3626 ± 0.04 **	< 0.0001
TOT-BILL (mg/l)	0.911± 0.062	0.4369± 0.044 ***	0.4805± 0.046 ***	< 0.0001

Values are means ± SEM, a P<0.05, significant change concerning the control group, *, ** and *** changes between P<0.01 and P<0.0001, SEM: Standard error of the mean, ALP: Alkaline phosphatase, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, D-BILL: direct bilirubin, IND-BILL indirect bilirubin, TOT-BILL: Total bilirubin.

Discussion

The risks of gasoline exposure are caused by chemicals in the gasoline mixture, such as xylenes toluene, ethylbenzene, benzene, and methyl tertiary butyl ether (MTBE). The CNS is the most common systemic impact of acute gasoline exposure, but respiratory tract irritation and hematological effects such as anemia and hypothermia can also occur at high concentrations. Chronic sniffing of gasoline has been linked to cardiac problems, as well as gasoline being a hepatotoxic substance.[17] Recently, it has been indicated by Rahimi et al (2020), that gasoline elevated proteinuria, liver enzymes, and fatty liver changes among exposed workers.[18] Through an oxidative process known as CYP450 2E1, gasoline hydrocarbons that are inhaled are digested in the liver, releasing free radicals and quinine metabolites such as hydroquinone, benzoquinone, phenol, and 1,2,4 benzenetriol. By these free radicals and hazardous metabolites, lipid peroxidation and damage to the hepatic plasma membrane are caused.[19] Assessment of cardiac parameters and liver enzymes may provide significant information regarding the effects of gasoline and smoke products on the heart cells and liver in Iraq because there are no legal guidelines or supervision for fuel components.[2]

Because of the predictive relationship between blood lipids and cardiovascular disorders, particularly coronary artery disease, measuring blood lipids in the clinical laboratory has become increasingly relevant. Total cholesterol, triglycerides, HDL cholesterol, and LDL cholesterol are frequently measured in lipid profiles during health screenings. The total cholesterol, LDL-C, increased statistically significantly as a result of this investigation. Smoking is linked to significantly higher total cholesterol and low-density lipoprotein levels in the blood (LDL). Triglycerides were found to be greater in the non-smoker exposure group. Smoking, on the other hand, lowers HDL cholesterol levels in the blood, which is a strong preventive factor against the development of atherosclerosis. These findings could be linked to exposure and inhalation of the hydrocarbon component of gasoline and cigarette smoke, both of which can produce oxidative stress due to reactive oxygen species (ROS). Tobacco smoke contains around 5000 hazardous chemical constituents, including polycyclic aromatic hydrocarbons (PAHs), free radicals, and oxidative gases.[20] As a result, in addition to inducing ROS intracellularly, the components of cigarette smoke suppress intracellular antioxidant systems, resulting in oxidative stress.[21] Pollutants in the environment, such as gasoline fumes, have been shown to increase oxidative stress in cells. When breathed, fumes from petroleum products can be degraded as xenobiotics through a sequence of reactions and biotransformations. During these reactions and biotransformations, ROS are created as undesirable by-products. Free radicals, which are produced by cigarette smoke, are thought to have harmful consequences, causing oxidative stress.[22]

Oxidative stress occurs when the cell's ROS and antioxidant systems are out of equilibrium. Under healthy settings, the cell produces ROS through oxygen metabolism, which is crucial for cellular signaling and is also harmless. Excessive formation of reactive oxygen species (ROS) causes lipid peroxidation, DNA strand breakage, and other impairments to the structure and functionality of cells when oxidative stress is present.[23] Extrinsic and intrinsic ROS can accumulate inside cells. Inhaled hazardous gases are the principal source of exogenous ROS (e.g., cigarette smoke, car exhaust fumes, and environmental pollutant). Peroxisomes, mitochondrial respiration, the NADPH oxidase system, and inflammatory cells, among other sources, create endogenous ROS.[24] The findings of this study, like those of many others, showed that cigarette smoke contributes to oxidative stress. In a case-control study involving 78 smoking and 82 nonsmoking men, Karademirci et al.(in press) found that the total antioxidant status (TAS), vitamin C, and vitamin E values were considerably higher in the nonsmoker group than in the smoker group. The smokers had increased total oxidant status and oxidative stress index values.[25] Other animal investigations have shown that cigarette smoke intake results in diminished and faulty antioxidant defense (decreased glutathione

peroxidase and superoxide dismutase activity, increased lipid peroxidation, and mitochondrial dysfunction) that causes a rise in H_2O_2 production.[26] As a result of this imbalance, oxidative stress caused by tobacco smoking causes greater heart damage.[27] The communication of NO with free radicals in smoke reduces NO's bioactivity, influencing its vasodilatory, antithrombotic, antioxidant, and anti-inflammatory effects, as well as its effects on endothelium permeability and myocardial function.[18,28]

In recent years, academics have been particularly interested in the physiological impacts of gasoline and smoking. In four primary studies, demographic data, smoking status, and other background information were gathered.[19] In five investigations, the frequency of cigarette smoking was recorded in both exposed and unexposed groups.[29] In terms of liver function measures, our findings revealed that non-smokers and smoke-exposed individuals had higher ALP, AST, and ALT levels than unexposed workers. Furthermore, the average levels of ALP and ALT were considerably higher in the smoker group, with no significant variations in AST levels between the exposed and unexposed groups. Smoking has been linked to an increase in liver enzymes and the development of chronic renal disease.[30] Furthermore, smoking has been linked to liver cancer and chronic renal disease, as well as an increase in liver enzymes.[31] Nicotine's poisonous components (CO, cyanide, potassium nitrate, cadmium, chloroform, vinyl chloride, and copper) are to be regarded.[32]

Some components of the smoke can inhibit the establishment of intracellular junctions, which can lead to tissue damage.[33] The rates of IND BILL and Total BILL were increased, according to the findings. Even though D-BILL was not statistically significant, it did indicate a little rise in the smoking exposure group, suggesting that environmental hazardous chemicals may hurt the liver. This data can be attributed to enzyme leakage, primarily as a result of enhanced cell membrane permeability. In addition, the liver is responsible for the metabolism of hazardous chemical compounds, which explains why the organ is susceptible to metabolic-induced hepatotoxicity.[34]

In addition, multiple investigations found that employees subjected to organic solvents had much higher liver enzymes than controls, supporting the current findings. Similar to our data, two of the investigations have reported an elevation in ALT and AST among the exposed than the unexposed group.[35] Furthermore, another study reported an increase in ALT and AST among the exposed group and similar concentrations in direct bilirubin and ALP in both exposed and unexposed persons.[36] While, on the other hand, Akinosun et al. record lower concentrations of ALT, AST, total protein, total albumin, and total bilirubin in both exposed and unexposed groups.[37] Workers in refueling stations had higher levels of ALT and AST enzymatic activity than those in the control group.[38] Contrary to popular belief, exposure to a variety of organic solvents does not affect the levels of liver enzymes such as ALT and AST.[39] Some investigations, however, have found that exposure to a mixture of organic solvents did not affect the levels of liver enzymes like ALT and AST.[40] Furthermore, the hallmark liver alterations (e.g., elevated ALP, ALS, and ALT) are depending on the method of administration (e.g., inhalation vs. cutaneous absorption), dose, and exposure period.[41] Another study conducted by Mohammed in Sulaimaniya City (Iraq) discovered changes in the hematological and biochemical profiles of gasoline station personnel, smokers, and non-smokers who were occupationally exposed to gasoline in connection to the observed lead levels.[42]

Conclusion

Based on the findings of this study, we can conclude that the harmful effects of gasoline, particularly when combined with smoking, are linked to negative health effects such as lipid profile parameters and liver, and that people who smoke are at a higher risk of having heart and hepatic diseases.

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