Occupational Medicine & Health Affairs

Research Article Open Access

Effect of Gasoline Exposure on Lipid Profile of Smoker and Nonsmoker Workers at Gasoline Refueling Service Stations

Hussain Al Wandawi[1], Dhiyaa SaleemJassim , Mohammed Essay Mohammad , Ali - Mahdi Mobarak

Department of Pharmacy, AL - Yarmouck University, Iraq

Abstract

Background: Using lipid profile contents as cardiovascular biomarker risk factors, the cumulate effect of cigarette smoking and exposure to gasoline(petrol) was studied in a group of workers occupationally exposed at gasoline refueling stations. Gasoline components are toxic, and too much exposure can be deadly. Low and moderate doses may cause eyes, nose and throat irritation, headache, nausea, and dizziness. Higher doses may damage some vital organs such as, liver, kidneys, heart, neurological and hematopoietic systems and in some cases may lead to vision loss.

Objective: There is little or no documentation on the weather smokers occupationally exposed to gasoline vapors and spills are at gasoline refueling car stations are at higher risk of cardiovascular disease compared to nonsmoker coworkers.

Methodology: Several gasoline refueling car stations were chosen at random in Hila city at central part of Iraq. The number of workers at each station was 8-10, provided they were non obese and non- diabetes, taking no medicine for heart problems, work 8 hours a day, 7 days a week, one day off). Each worker was informed about: the aim of the study and privacy of their personal data. They signed a written consent confirming their acceptance to participate in this study. Their personal database included age, duration of smoking and years of service. Blood samples were obtained by venipuncture technique, collected in EDTA and delivered to the lab within an hour after collection. ALipidPlus device**,** serum total cholesterol, NON – HDL cholesterol, HDL cholesterol, and triglycerides (TGs). LDL cholesterol was estimated using Friedewald equation.

Results: The interpretation of the results presented in tables and conclusions made, are mainly based checking with reference levels documented in medical practices, because no similar studies can be traced in literature. The elevation in the levels of triglycerides, total cholesterol, non –HDL cholesterol among smoker workers compared to nonsmokers, and the high ratios for, TC (total cholesterol) / HDL – C (high density lipoprotein cholesterol) and TGs (triglycerides) / HDL – C (high density lipoprotein cholesterol) may also provide additional support to our conclusion that smokers occupationally exposed to gasoline are at higher risk of cardiovascular disease compared to their nonsmoker colleagues.

Conclusions: In spite of difference in age and duration of service, smoker workers at gasoline refueling stations are at higher risks of cardiovascular disease compared to their nonsmokers colleagues. This may be due to the passive synergistic effect of tobacco smoke and gasoline exposure, because both contain of some toxic components that affect the circulatory system,. The another biomarker test, namely, the ratio of HDL - C / LDL- C values was showed to be of no significant value in our case and differences can be seen among smokers and nonsmokers

Keywords: Smoke; Cardiovascular disese; Tabacoo; Gasoline; Fuel

Introduction

Since no studies similar to ours can be traced inliteratures, our review will be limited to the properties, routes of exposure, and health problems resulting from exposure to gasoline fumes and spill at gasoline refueling stations and similar facilities. Gasoline is known as petrol in (Britain), benzin in (Germany). Gasoline is one of the most consume products worldwide. In 2019, U.S alone consumed about 142.23 billion gallons (or about 3.39 billion barrels) of finished motor gasoline, which accounts to about 389.68 million gallons (or about 9.28 million barrels) per day [1]. Gasoline refueling stations are particularly hazardous work place where, fuel is stored and transferred between tanker trucks, underground storage tanks and vehicle tanks. Gasoline is a unique product with one specific commercial use: fuel for internal combustion engine [2]. Gasoline is toxic, volatile, and extremely flammable liquid, contains more than 150 or more chemical compounds, some are toxic including benzene, toluene, xylene, and sometimes lead [3-4]. Some of these potential toxic compounds can be found in atmosphere of gasoline refueling stations and surroundings [5-7]. The main routes for gasoline exposure is by breathing vapors. Exposure to gasoline or gasoline vapors in large amounts or over an extended period can cause serious health complications [8-10]. Gasoline may also present certain risks even for those living in close vicinity of a petrol refueling station, or petrol refinery [2,11]. However, occupational exposures are considered to be great health concern [12,13], car because of exposure vapors and sills, but also for exposure to car combustion products in the vicinity of the stations is additional risk. Some acute cases of gasoline exposure at sites of washing petrol tanks have also been reported [14]. Sniffing of gasoline is also an important route

***Corresponding author:** Hussain Al Wandawi, Department of Pharmacy, AL - Yarmouck University, Iraq, Tel: + 964 7704261368; E-mail: dr_windawi @yahoo.com

Received October 14, 2019; **Accepted** July 20, 2020; **Published** July 27, 2020

Citation: Wandawi HA, Jassim DS, Mohammad ME, Mobarak A (2020) Effect of Gasoline Exposure on Lipid Profile of Smoker and Nonsmoker Workers at Gasoline Refueling Service Stations. Occup Med Health Aff 8: 309.

for exposure, which may cause serious problems such as muscle weakness, dementia, nephritis etc. [15-18]**.** The majority of reported cases of gasoline intoxication involve oral ingestion or inhalation [19]. Absorption of gasoline and its components including benzene through the lungs and gastrointestinal tract (GIT) is quite efficient [20]. Misuse of gasoline as a solvent or cleaner can cause skin and eye irritation and central nervous system toxicity after extensive overexposure [21]. Thus, Severe exposure cases to gasoline or gasoline vapors can cause kidney cancer [22] cortical atrophy [23-24], reproductive toxicity [25-27] neurological and cognitive impairment [28-29]. The association between lung cancer and cumulative exposure to gasoline was found to be weakly positive. Gasoline poisoning may also cause vision loss, vomiting with or without blood ;bloody stools; dizziness, staggered gait, slurred speech and confusionand very high concentrations may result in rapid unconsciousness and death due to respiratory failure. Chronic eyes exposure to gasoline without protection may cause damage to the cornea, retina ciliary body, genotoxicity. Positive association was suggested between exposure the exposure to gasoline inhalation and the development of adverse reproductive endpoints including menstrual and reproductive hormone profile. Elevated levels of blood lipids are well-documented risk factors for cardiovascular disease (Nelson, 2013). Epidemiologic and clinical trials show that elevated triglycerides and low levels of high-density lipoprotein cholesterol (HDL-C) are independent risk factors for coronary heart disease. For predicting risks of cardiovascular disease, there is a good deal of agreements that determining non – HDL cholesterol may be more useful than calculating cholesterol ratio (Total cholesterol / high density lipoprotein cholesterol; TC / HDL-C), and either option appears to be a better risk factor assessment than total cholesterol level or even low density lipoprotein cholesterol(LDL – C) Thus, it was found that non – HDL – C provides a measure of cholesterol contained in all atherogenic particles, therefore, non – HDL – C was introduced as secondary target of therapy in persons with triglycerides ≥ 200 mg / dL. Even in the presence of tightly controlled LDL-C levels, evidence indicates that high TG and low HDL-C levels are independent cardiovascular risk factors. High-risk individuals, especially with cardiometabolic disease, who achieve LDL-C goals, remain at high risk of CV events. The effects of exposure to gasoline on alteration in lipid profile among smokers, and even non – smokers has received little attention. In the present study we investigated the effect of gasoline exposure among two groups of smoker and non – smoking workers at refueling gasoline station to investigate if there is an effect of cigarette smoke on their lipid which may give a predictor risk for cardiovascular disease.

Discussion

Examination of the published literature reveals no or only scanty information concerning the effects of gasoline exposure on lipid profile in general, and of smoker workers occupationally exposed to gasoline vapors and spills at gasoline refueling stations in particular. It is useful to remember that gasoline is one of the highly volatile liquid products of petroleum fractionation, and its evaporation generates vapors into the work place, surroundings and environment. Sniffing of and contact with unburned gasoline are also routes of exposure. Moreover, even persons whose clothing or skin is contaminated with liquid gasoline can cause secondary contamination by direct contact or through off-gassing vapors. Hence, population at greater risk of frequent exposure are, those occupationally exposed, as well as those residing in traffic-congested areas. Gasoline and its vapor are toxic and exposure to them can seriously damage a person's health. The association between gasoline exposure and cardiovascular disease has been documented. Since cardiovascular diseases remain the biggest cause of disability and premature death throughout the world. For more than a century, increasing evidence has replicated athergenic lipid factors in the development of atherosclerotic cardiovascular disease (ASCVD). A routine lipid profile measurement that been used in our study, is the most commonly used laboratory measure to evaluate a patients' atherosclerotic risk, and includes the measurement of total cholesterol (TC) and high - density lipoprotein cholesterol (HDL – C), low density lipoprotein cholesterol (LDL – C), and *triglycerides (TGs)*. Replacing TC and HDL – C with various lipid parameters does not improve the risk prediction of CVD, with, no meaningful improvement from addition of apolipoproteins or, direct measurement of calculated LDL-C. On the other hand, **s**moking which is an independent risk factor of CVD, but, its effect is compound through association with other risk factors in the plasma such as, high blood cholesterol levels (hypercholesterolemia), high low density lipoprotein cholesterol (LDL-C), high triglycerides (hyperglyceridemia), and low level of high density lipoprotein cholesterol(HDL – C). Our study investigated the possible passive synergistic effects on cardiovascular risks of exposure to gasoline on cardiovascular risk factors, because of dearth of information regarding this subject. The results of our study shown in Tables 1 & 2 reveal that in spite of variation of age, and duration of service, the average, TG levels in smokers was 221.91(ranked high), normal level is below (150 mg / dL). Moreover, none of smokers showed TG level below or equal to (150 mg/dL). In nonsmoker workers, however, the average (TG) levels was (118.70) which is ranked (optimum or near ideal), only 2 of such workers showed (TG) ≥ (150 mg / dL). The important of this finding stems from the fact that, a long standing association was reported to exist between triglyceride levels and cardiovascular disease, and there is increasing evidence suggest that (TG) level is important in assessing risk of atherosclerotic events. So the high (TG) levels among smoker workers under conditions reported in our study suggest that smokers are at higher risk of CVD compared to their nonsmoker coworkers. Such elevated level of TG may be due the passive synergistic effect of some toxic compounds in both cigarette smoke and gasoline. For example, cigarettes smoke is known to contain a high concentrations of benzene which is toxic and volatile and gasoline has been to contain (0.62%) by volume with maximum allowable level 1.3 % by volume and in one study, 1 to 4 % v/v may be prevalent, depending on the type of crude oil. Benzene is also reported to be associated with cardiovascular disease risk therefore, such elevation in TG may be due passive synergistic effect of gasoline exposure and smoking. The importance of measuring non - high density lipoprotein (non – the major HDL) holds that the major atherogenic lipoproteins are low density lipoproteins (LDL) and very low density lipoprotein (VLDP) together they constitute non – high density lipoproteins (non – HDL). The results of our study also reveal that the levels of total cholesterol (TC) and non – HDL cholesterol were relatively in smoker workers (165.27 and 110.80) respectively, compared to (146.80 and 95.70) in nonsmoker workers. Total cholesterol to HDL cholesterol ratio was calculated because such ratio has been reported to be helpful in predicting the risk of developing atherosclerosis, where high ratios indicate higher risks of heart attack and vice versa. In our study, the ratio of TC / HDL - C was relatively higher in smokers (3.4) compared to (2.82) in nonsmokers are considered good (below 4), and approaching the best values(2 or 3). In our study, non – HDL cholesterol was also considered, because it does not require fasting, and it is superior predictor of atherosclerotic cardiovascular events than measured LDL- C. We also found that, although the average non – HDL cholesterol levels in both groups were within an optimal level (less than 130 mg /dL), it was relatively higher in smokers group (110.82 mg / dL), compared to (95.38 mg / dL)

in nonsmokers (Tables 1 & 2). The non – HDL- C has been considered in this study because with the exception of lipid profile, none of the other lipid parameters found its way in clinical use ; non – HDL cholesterol was an exception, and proved to be superior to LDL cholesterol for prediction of cardiovascular events. The non – HDL cholesterol, was also bend more useful than calculating cholesterol ratio(TC / HDL cholesterol). An optimal level of non – HDL cholesterol is less than 130 mg / dL. Numbers higher than (4:1 ratio) mean a higher risk of heart disease ratios. The results presented in Tables 3 and 4 show that the average of non – HDL cholesterol is 118.82 and 95.10 for smokers and nonsmokers respectively. These values are well below the recommended value of (130 mg / dL), which means there is no or minimum indication risk of CVD). Significant difference in ratios of TG / HDL – C in smokers was (4.06) compared to (2.51). However, the vales for the ratios of HDL- C / LDL – C showed no difference; 0.80 for smokers and 0.76 for nonsmokers. Finally, pooling the results of this study, it can be suggested that smoker occupational expose to gasoline vapors and spills are at higher risk of cardiovascular disease compared to nonsmoker coworkers. Finally, the results of this study show that smoking strengthen the risks of CVD when coexist with gasoline exposure or vice versa.

Subject	Age	Years of service	TC	TGs	LDL-C	HDL- C	TC –Non-HDL-C
1	63	24	161	224	61	55	106
2	44	10	151	385	22	52	99
3	48	10	175	196	80	56	119
4	31	13	137	154	53	53	84
5	33	7	156	258	49	55	101
6	42	22	210	222	105	61	149
7	49	10	120	228	30	44	76
8	57	28	204	156	115	58	146
9	49	9	183	114	101	59	124
10	36	15	132	264	50	49	83
11	57	24	189	240	84	57	132
Average	46.27	15.67	165.27	221.91	68.18	54.46	110.82

Table 1: Background Data for Smoker Workers.

Reference range: TC : ≤ 200 mg/dL is normal; TGs : 10 – 150 mg/dL is normal; 150 to 199mg/dL is border high; 200 to 499 mg/dL is high, LDL – C : 70 – 130 mg/ dL is normal(100 mg / dL is optimal) , HDL – C , 40 to 65 mg / dl is normal range for adult men.

Subject	Age	Years of service	TC	TGs	LDL-C	HDL- C	TC-Non-HDL-C
1	69	8	144	140	66	50	94
2	33	10	130	147	53	48	82
3	39	8	182	84	108	57	125
4	42	10	133	105	61	51	82
5	31	5	144	63	78	53	91
6	61	36	141	66	74	54	87
7	44	6	204	252	96	58	146
8	39	15	132	108	59	51	81
9	37	14	144	162	59	53	91
10	39	10	114	60	56	46	68
Average:	43.4	12.2	146.8	118.7	71	52.1	95.7

Table 2: Background Data for Non-smoker Workers.

Reference range: TC : ≤ 200 mg/dL is normal; TGs : 10 – 150 mg/dL is normal; 150 to 199 mg/dL is border high; 200 to 499 mg/dL is high, LDL – C : 70 – 130 mg/ dL is normal(optimal ,100 mg /dL or lower), HDL – C 40 – 65 mg / dl is normal for adult men.

Subject No.	Total cholesterol mg / dL	LDL mg / dL	HDL mg / dL	TG mg / dL	TC/ HDL	TGs / HDL	HDL/LDL
1	161	61	55	225	2.93	4.09	0.9
2	151	22	52	385	2.9	7.4	2.36
3	175	80	56	196	3.13	3.5	0.7
4	137	53	53	155	2.59	2.93	1
5	156	49	55	258	2.84	4.69	1.12
6	210	105	61	222	3.44	3.64	0.58
7	120	30	44	228	2.73	5.18	1.47
8	204	115	58	156	3.52	2.69	0.5
9	183	101	59	114	3.1	1.93	0.58
11	189	84	57	240	3.32	4.21	0 .68
Average	165.3	68.2	54.5	222.1	2.77	4.15	0.9

Table 3: Smoker group

Reference range: TC : ≤ 200 mg/dL is normal; TGs : 10 – 150 mg/dL is normal; 150 to 199mg/dL is border high; 200 to 499 mg/dL is high, LDL – C : 70 – 130 mg/ dL is normal(100mg/ dL), HDL – C 40 – 65 mg / dL is normal for adult men.

Subject No.	Total cholesterol mg / dL	LDL mg / dL	HDL mg / dL	TG mg / dL	TC/ HDL	TGs/HDL	HDL/LDL
1	144	66	50	140	2.88	2.8	0.76
2	130	53	48	147	2.71	3.06	0.91
3	182	108	57	84	3.19	1.47	0.53
4	133	61	51	105	2.61	2.06	0.84
5	144	78	53	63	2.72	1.19	0.68
6	141	74	54	66	2.61	1.22	0.73
7	204	96	58	252	3.52	4.35	0.6
8	132	59	51	108	2.59	4.41	0.05
9	144	59	53	162	2.72	3.06	0.86
10	114	56	46	60	2.48	1.3	0.82
Average	145.09	67.27	51.36	128.64	2.8	2.54	0.75

Table 4: Non-smoker group

Reference range: TC : ≤ 200 mg/dL is normal; TGs : 10 – 150 mg/dL is normal; 150 to 199mg/dL is border high; 200 to 499 mg/dL is high, LDL – C : 70 – 130 mg/ dL is normal(100 mg/ dL is optimal) , HDL – C 35 – 65 mg / dl is normal for adult men.

Conflicts of Interest

The authors declare they have no conflicts of interest.

Funding Information

No funding of any was awarded for this study

Acknowledgement

The authors would like to acknowledge l volunteer workers who agreed to take part in this study.

References

1. https://www.eia.gov/tools/faqs/faq.php?id=727&t=6
2. Dolan JA (2008) Handbook of analytical separations 6: 873-922.
3. Page NP, Mehlam M (1989) Health effects of gasoline refueling vapors and measured exposures at service stations. ToxicolInd Health 5: 869-890. ATSDR (2004) Gasoline 8006- 61 – 69.
4. Hilpert M, Mora BA, Ni J (2015) Hydrocarbon release during fuel storage and transfer at gas stations : Environmental and health effect. Curr Environ Health Reports : 412-422.
5. Hazrati S, Rostami R, Fazlzadeh M (2016) Benzene, toluene, ethyl benzene and xylene concentrations in atmospheric ambient air of gasoline and CNG refueling stations. Air Quality, Atmosphere and Health 9: 403-409.
6. Hilpert M, Rule AM, Andria – Mora B (2018) Vent pipe emission from storage tanks at gas stations : Implication for setback distances. J Total Environ 650: 2239 -2250.
7. Caprino L, Tonga G (1998) Potential effects of gasoline constituents: A review of current literature (1990 –1997) on toxicological data. Environ Health Perspect 106: 115-125.
8. Ekpenyong CE, Asuquo AE (2016) Recent Advances in occupational and environmental hazards of workers exposed to gasoline compounds. Int J Occup Med Environ Health 29: 55-69.
9. Huizen J (2009) How does gasoline affect a person's health? Medical News Today.
10. Terres IMM, Minarro MD, Ferrdas EG, Caracena AB, Rico JB, et al. (2010) Assessing the impact of petrol stations on their immediate surroundings 91: 2754 - 2762.
11. Azari MR, Konjin ZN, Zayeri ZN (2012) Occupational Exposure of petroleum depot workers to BTEX compound. Int J Occup Environ Med 12: 39 -44.
12. TunsaringkamT, Soogarun S, Palasuwan A (2012) Occupational Exposure to Benzene and Changes in hematological Parameters and Urinary Trans, Trans-Muconic Acid. Int J Environ Med Occup 45 -49.
13. Takamiya MH, NiitsuK, Saigusa (2003) A case of acute gasoline intoxication at scene of washing a petrol tank. Legal Medicine 5: 165 -169.
14. Poklis A, Burket cd (1977) Gasoline sniffing: A review .Clin Toxicol 11: 35 -41.
15. Remington G, Hoffman BF (1984) Gas sniffing as a form substance abuse. Can J Psychiatry 29:31-35.
16. Fortenberry JD (1985) Gasoline sniffing Am J Med 79: 740 -744.
17. Cairney S (2002) The neurobehavioral consequences of petrol (gasoline) sniffing. Neurosci Biobehav Rev 26: 81-89.
18. Domej W , Mitterhammer H, Stauber R, Kaufmann P, Smolle KH, et al. (2007) Successful outcome after intravenous gasoline injection 3: 173 -177.
19. Reese E, Kimbrough RD (1993) Acute toxicity of gasoline and some additives 101: 115 -131.
20. TSDR - 1993: Toxic Substances and Disease Registry- Gasoline.
21. Uboh FE, Akpanabiatu MI, Eteng MU (2010) A risk factor for nephrotoxicity in rats. Int J Tox 7.
22. Rinsky RA, Hornung RW, Silver SR, CY Tseng, et al. (2002) Benzene exposure and hematopoietic mortality: A long-term epidemiologic risk assessment. Am J Ind Med 42: 474 -480.
23. Abubakar MB, Abdulah WZ, Sulaiman SA, Ang, BS (2015) The effects of exposure to petrol vapors on growth, hematological parameters and oxidative markers in Sprague Dawlely male rats. Malays J Med Sci 22: 23-25.
24. Webb E, Bushkin-Bedient S, Cheng A, Kassotis CD, Balise V, et al. (2014) Developmental and reproductive effects of chemicals associated with unconventional oil and natural gas operations. Rev EnvironHealth 29: 307-318.
25. Owaaboriaye FO, Deke GA, Ashidi JS, Aladesida AA, Olooto WE, et al. (2018) Effect of gasoline fumes on reproductive function in male albino rats. Environ Sci Pollution Res 25: 4309-4319.
26. White KL, Peachee VI, Armstrong SR, Armstrong SR, Twerdok LE (2014) Health assessment of gasoline and fuel oxygenate vapors : Immunotoxicity evaluation. Regul Toxicol Pharmacol 70: S43 – S47.
27. Maruff P (1998) Neurological and cognitive abnormalities associated with chronic petrol sniffing. Brain 121: 1903-1917.
28. Kinawy AA (2009) Impact of gasoline inhalation on some neurological characteristics of male rats. BMC Physiology 9: 21.
29. Karamis S, Boffeta P, Brennan P, Stewart PA, Zaridze D, et al. (2011) Renal cancer and occupational exposure to polycyclic aromatic hydrocarbons and plastics. J Occup Environ Medicine 53: 218-223.