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Biological Monitoring of Fuel Stations Workers Occupationally Exposed to Petroleum Products

Khaled F. El-Said*, Amr El-Noueam*

Abstract: The increased use of petroleum products in automobiles and industry has led to the deterioration in air quality and human health. Petrol (or gasoline) is a volatile and inflammable petroleum derived liquid mixture primarily used for internal combustion of engines. Occupational exposure to gasoline and air pollutant from vehicular sources are relatively unexplored. The main purpose of this study was conducting biological monitoring for petrol workers occupationally exposed to petroleum product and air pollutants. The study was conducted on fifty male workers employed in petrol filler stations and fifty subjects were recruited as a control group matched for age, sex and socio-economic status. Results revealed that presence of high significant association in the levels of liver enzymes and some haematological changes among exposed workers. In addition to the significant decline in the lung function. A significant increase in urinary phenol as a biomarker of benzene exposure was found. Sulphur dioxide nitrogen dioxide, carbon dioxide and carbon monoxide did not exceed the standards indoor air quality guidelines. However, particulate matter exceeded the limits as indoor air quality guidelines. This study demonstrated that it is possible to detect human health risks at an early stage using sensitive techniques. It is concluded that petrol pump workers should be carefully monitored and should take adequate protective measures to prevent long term effects.

INTRODUCTION

The increased use of petroleum products in automobiles and industry has led to the deterioration in air quality and human health. These products contain a number of toxins that are considered to be carcinogenic to humans.(1)

In filling stations, the volume of fuel dispensed as well as the ambient temperature contributes significantly to the increased emission of volatile hydrocarbons. Benzene (BZ) could be considered to be the most hazardous; xylene (XYL) and toluene (TOL) have toxicities (acute central nervous system effects) in line with other aromatics of lower/different concern than BZ.(2)

*Assistant Professor of Industrial Hygiene and Air pollution, Occupational Health and Air pollution Department, High Institute of Public Health. Alexandria University, Egypt.
** Professor of Physiology, Physiology Department, Faculty of Medicine, Alexandria University, Egypt.

Several studies to determine the concentrations of benzene, toluene, and xylene (BTX) in the ambient air of occupational sites have shown increased levels.[3]

Petrol filling workers are exposed to a mixture of hydrocarbons in fuel vapours during dispensing fuel and to the gases from vehicular exhaust. The rapidly multiplying number of automobiles in most towns and cities and the corresponding increase in air pollution is a cause of grave concern. Certain groups, by virtue of their occupation, face an increasing threat of its adverse health effects.[4] In addition to automobile exhaust fumes, these workers are also at a risk of inhaling fuel vapour. The combined effects of the two may result in accelerated decline of lung function. Fuel handlers as well as personnel working in close proximity to fuel exhaust such as vehicle mechanics.[4]

Petrol (or gasoline) is a volatile and inflammable petroleum derived liquid mixture primarily used for internal combustion of engines. It consists of hydrocarbons (aromatic, saturated and unsaturated) and non-hydrocarbons (N,S, O_2, vanadium and nickel).[5]

Components of car-exhaust as a result of internal combustion of petrol, which are harmful, are CO_2, NO_2, SO_2, benzene, formaldehyde and polycyclic hydrocarbons. These substances are known to produce harmful effects on the blood, bone marrow, respiratory system, lymph nodes, pregnancy and pregnancy outcomes. They also interact with the immune system to cause local and systemic responses ranging from overactive immune responses to immunosuppresion.[5]

Atmospheric concentration of gasoline vapor (approximately 2000 ppm) is not safe when inhaled even for a brief period of time (seconds). During fuelling of vehicles, the concentration of gasoline vapor in the air is between 20 and 200 ppm.[6]

Health effects of occupational exposure

to gasoline and air pollution from vehicular sources are relatively unexplored among petrol filling workers. Neglect of ventilation in the work place or failure to use personal protective equipment when using petrol containing solvents like benzene will increase the incidence of toxic effects of benzene in humans, which includes haematotoxicity, immunotoxicity, neurotoxicity and carcinogenicity. (7)

Air pollutants and chemicals with known adverse effects like Benzene, Lead, other heavy metals, and Carbon monoxide (CO) and their metabolites can cause adverse health effects by interacting with molecules crucial to the biochemical or physiological processes of the human body. All these have been found to lead to deleterious effect on respiratory, endocrine and haematopoietic systems. High ambient air concentrations of solvents and pollutants had well defined and marked systemic pulmonary inflammatory response with decreased forced vital capacity(FVC), forced expiratory volume in the first second (FEV1), Inspiratory and expiratory flow rates .(8)

The main purpose of this study was conducting biological monitoring for petrol workers occupationally exposed to petroleum product and air pollutants.

MATERIAL AND METHODS

Subjects

The present study included 50 petrol filling workers occupationally exposed to petroleum product and air pollutants for more than 5 years as workers group , and 50 subjects selected from administrative departments never occupationally exposed to petroleum product as control group. Verbal consent, approval of the Ethical committee was obtained. All participants signed individual consents prior to the study, occupational and medical questionnaire were completed with the workers through personal interview.

Collection and Assay: Blood samples were collected from all cases by sterile disposable syringes. Samples were left to clot and centrifuged, the separated serum was used for estimation of Serum aminotransferases (ALT and AST) by using Beckman's autoanalyser.[9]

Spot urine samples were collected from all subjects for analysis of urinary phenol as described by Van Roosmalen, *et al*.[10] The rest of EDTA blood were used to measure complete blood picture (CBC) according to Standard methods.[11]

Ventilatory Function Tests: Spirometric measurements were performed in sitting position using a portable spirometer, according to the criteria of the American Thoracic Society (1995).[12] All ventilatory function parameters in the form of forced expiratory volume in the first second (FEV1), forced vital capacity (FVC), peak expiratory flow rate (PEFR) and forced expiratory flow (FEF25-75) were expressed as percent of the predicted value for each person after adjustment for age, gender, race, height and weight.

Environmental monitoring of air pollutants such as carbon monoxide (CO), Carbon dioxide (CO_2),nitrogen dioxide (NO_2), Sulphur dioxide(SO_2) and particulate Matter (PM_{10}) were measured using direct reading instrument applying infrared analyzer principal.[13,14]

Statistical Analysis: The collected data and the laboratory results were computerized. Statistical analysis was done through SPSS version 17.0. The quantitative results were expressed as means ± standard deviation (SD), and Independent t-test, Pearson's correlation coefficient and ANOVA were used in the analysis of the results.

RESULTS AND DISCUSSION

Data of characterization of the subjects studied revealed that all parameters as age and smoking were not significant. The only significant association in the levels of BMI

Table 1: Characterization of the subjects

	Subjects (mean±S.D)		
Character	**Control**	**Exposed**	**P-Value**
Age(years)	34.7±5.2	35.0±6.3	NS
Duration of Exposure(years)	-	6.5±3.5	-
BMI Kg\m^2(Body Mass Index	21.0±2.1	25.3±3.5	P<0.001
Cigarettes Smoking			
Non-Smokers	27(54%)	36(72%)	NS
Smokers	23(46%)	14(28%)	NS

NS: Not significant

Table2: Hematological Parameters among exposed workers and control subjects

	Subjects (Mean±SD)		
Measured parameters	**control subjects (n=50)**	**Petrol exposed workers (n=50)**	**p**
Hb	13.51±0.771	13.25±0.939	NS
Ht %	44.7±3.59	44.19±4.35	NS
RBCs $\times 10^6$ (cell/mm^3)	4.79±0.329	4.29±0.317	P<0.01
WBCs $\times 10^6$ (cell/mm^3)	5.58±1.888	6.38±1.590	NS
PLT $\times 10^3$ (cell/mm^3)	299.15±87.81	252.39±58.65	P <0.01

NS: Not significant

In Table 2, Workers exposed to petro products and air pollutants showed low levels of Hb, Ht value, RBCs and PLT count; and high levels of WBCs relative to non-exposed workers. The differences between control and exposed workers a were insignificant except for RBCs, and PLT counts (P<0.05). The significant fall in blood parameter which is observed in the present study among exposed workers relative to controls indicates that exposure to petroleum product and air pollutants has an impact on erythropoiesis.

Moreover, the total WBC, differential counts and platelets were found to decrease in workers with longer periods of exposure. This may be explained by The studiy conducted by Qing Lan et al(2004).[(15)] which showed that solvents cause toxicity to progenitor cells of WBCs and platelets instead of the circulating cells.

Table 3: Liver function enzymes and urinary phenol in petrol filler workers and control subjects

parameter	Subjects (Mean±SD)		t-test	
	Control(n=50)	Exposed (n=50)	t	P
ALT(U\L)	23.5±5.6	45.3±17.9	7.13	P<0.001
AST(U\L)	21.6±7.15	35.7±12.7	8.27	P<0.001
Urinary Phenol (mg/g creatinine)	8.3±1.6	24.3±12.0	9.32	P<0.001

Table 3 showed that the level of urinary phenol as a biomarker of benzene exposure was highly significant in petrol workers in comparison to the control group indicating heavy benzene exposure among petrol stations workers.

Liver enzymes as ALT is an enzyme present in hepatocytes (liver cells), and it leaks into blood when liver cells are damaged. It rises dramatically in acute liver damage and during liver inflammation. Similarities in the levels of ALT in petroleum attendants and controls were found by Qing Lan et al., (2004)[15]. The other liver enzyme AST is associated with liver parenchymal cells. It is raised in acute liver damage and is also present in red cells and cardiac muscle. Since AST is not specific for the assessment of liver function, slightly raised AST in petrol attendants might be due to gradual lyses of red blood cells.[15] Results (table 3) of Liver function revealed that significant higher levels of ALT and AST among petrol filler workers, similar results were in others studies.[16,17]

Table 4: Concentrations of measured airborne pollutants in the petrol station.

	SO2 (µgm-3)	NO2 (µgm-3)	PM10 (µgm-3)	CO (µgm-3)	CO2(µgm-3)
Mean±SD	0.21±0.36	0.035±0.05	4.18±2.4	0.49±0.71	1188.15±2101
Range	3.0	00.19	8.21	2.79	7797.00
Egyptian Standard **(**µgm-3**)**	5	6	3	25	5000

All measured airborne pollutants (Table 4) did not exceed the Egyptian standards except for PM which exceeded the standards and these might be due to working in open areas.

Most of the petrol filling stations were situated near to the heavy traffic; workers were prone to exposure to CO. The ambient air concentration of CO was maximum during the peak working hours (6AM – 2PM), meaning that workers were exposed to high levels of CO along with other air pollutants and solvents.

Particulate matter of the size of 2.5 m and 10m (PM10, PM2.5) and NO2 have been found to be significantly associated with reduced FVC. Similar effects of automobile exhausts on the FVC and FEV1 were being reported in tunnel and bridge workers, traffic wardens and shopkeepers. Smoking has been reported to accelerate the decline in FEV1.[18,19]

Table 5 shows that FVC and FEV1 in petrol stations workers were decreased compared to the control subjects; but without significant difference. While PEFR, FEV1/FVC and FEF25-75 smokers were significantly decreased compared to the controls. Additionally, FEF25-75 of non-smoker workers was significantly decreased compared to that of the non-smoker controls.

Table 5: Comparison between petrol filler and controls subjects regarding ventilatory function.

	Subjects (Mean±SD)				
Paremeter	**Control (N=50)**		**Exposed (N=50)**		**AVOVA**
					P
	Smoker	Non Smoker	Smoker	Non Smoker	
FVC	77.2±19.1	84.0±16.22	69.2±13.1	76.1±1.27	NS
FEV1	81.2±19.3	92.4±12.9	86.9±15.6	87.1±17.1	NS
PEFR	83.9±25.1	91.6±22.2	69.3±18.2	74.1±20.5	P<o.o1
FEV1/FVC	118.2±7.2	122±3.9	109±16.2	114.1±9.3	P<o.o1
FEF 25-75	118.9±26.9	122.1±20.6	98.2±16.5	118.9±28.9	P<o.o1

Generally, most of the lung function parameters were decreased significantly in petrol pump workers as compared to controls. Although FEVl and FVC decreased in petrol pump workers, their ratios did not differ between the two groups. These findings indicate the restrictive nature of pulmonary involvement in the study subjects.[20]

Although multiple pollutants were detected in the indoor air of the workplaces in the present study, yet non of them exceeded the Egyptian standards, except for PM10 which might play casual factors in higher percentage of respiratory complaints in petrol filler makers. The FEF25-75 was found to be the most affected pulmonary function parameter.

These findings came in accordance with pervious studies[21,22] that linked occupational exposure in petrol stations workers with the development of diffuse lung disease. Taking in consideration smoking habits of the included subjects. Reductions in the ventilator capacity tests were similarly detected in petrol stations makers compared to their controls, but significant differences were only for PEFR, FEV1/FVC and FEF25-75.[21]

The findings of the present study indicate that small airways probably bear the brunt of the air pollution and fuel vapour related lung injury. This finding is in agreement with most studies on pollution inflicted changes in lung function. [22]

CONCLUSION

In conclusion, any significant decline in the lung function with time merits attention, as well as the biochemical and hematological changes in petrol workers that work in polluted environment. Preventive measures should be taken to avoid risk of exposure to toxic air pollutants and petroleum products.

REFERENCES

1. EPA.US Environmental Protection Agency. Integrated risk information system (IRIS) of benzene. National Center for Environmental Assessment. Washington, DC: Office of Research

and Development.2002

2. ACGIH.TLVs and BEIs. Based on the documentation of the threshold limit values for chemicals substances and physical agents and biological exposure indices. Cincinnati, OH: American Conference for Governmental and Industrial Hygienists.2009
3. Hoet P, Desmedt E, Ferrari M. Evaluation of urinary biomarkers of exposure to benzene: correlation with blood benzene and influence of confounding factors. Int Arch Occup Environ Health. 2009; 82: 985–95.
4. Singhal M, Khaliq F, Singhal S, Tandon P. Pulmonary functions in petrol pump workers: a preliminary study. Indian J Physiol Pharmacol. 2007; 51(3): 244–8.
5. Lewne M, Nise G, Lind L, Gustavsson P. Exposure to particles and nitrogen dioxide among taxi, bus lorry drivers. Int Arch Occup Environ Health. 2006;79:220-6.
6. Pranjic N, Mujagic H, Nurkic M, Karamehic J, Pavlovic S. Assessment of health effects in workers at gasoline station. Bosn J Basic Med Sci. 2002;2:35-45.
7. Aksoy M. Hematotoxicity and carcinogenicity of benzene..Environ. Health Perspect. 1989; *82:*193-7.
8. Kesavachandran C, Mathur N, Anand M, Dhawan A.Lung function abnormalities among petrol pump workers of lucknow, north India. Current Science. 2006; 90: 1177-78.
9. Olaniyi J, Arinola O, Esogurah B, Akibinu M. Liver and renal functions in Nigerian sickle cell disease patients. Science Focus. 2003;5:78-81.
10. VanRoosmalen B, Purdham J, Drummond I. An improved method for the determination of phenol in the urine of workers exposed to benzene or phenol. International Archives of Occupational and Environmental Health. 1981; 48(2): 159-63.
11. Dacie V, Lewis S. Practical haematology. 5th ed. Edinburgh: Churchill Livingstone, 1975.
12. American Thoracic Society. Standardization of spirometry 1994 Update. Am J Respir Crit Care Med. 1995;152:1107–360.
13. Poirot P, Subra I, Gérardin F, Baudin V, Grossmann S, Héry M. Determination of short-term exposure with a direct reading photoionization detector. Ann Occup Hyg. 2004, 48:75-84.
14. Katz M. Methods of air sampling and analysis. Edited by Morris Katz, Interdisciplinary Books & Periodicals, 1977; pp.578.
15. Qing L, Luoping Z, Guilar L, Rappaport S , Min S, Alter B , Yongi W, William K, Suranya W, Charles R, Weitong G. Hematotoxicity in workers exposed to low levels of benzene. Science. 2004,306, 1774-6.
16. Olaniyi J, Arinola O, Esogurah B, Akibinu M. Liver and renal functions in Nigerian sickle cell disease patients. Science Focus 2003;5:78-81.
17. Akinosun M, Arinola G, Salimonu S. Immunoglobulin Classes and liver Function tests in Nigerian petrol attendants. Indian Journal of Occupational and Environmental Medicine. 2006; 10 (2):58-61
18. Min J, Min K, Cho S, Paek D. Combined effects of cigarette smoking and sulphur-dioxide n lung functions in Koreans. J Toxicol Environ Health. 2008; 71(5): 301–3.
19. Xu X, Wang L. Synergistic effects of air pollution and personal smoking on adult pulmonary function. *Arch* Environ Health*.* 1998; 53(1): 44–53.
20. Innes J, Reid P. Respiratory disease. In:

Boon NA, Coiledge NR, Walker BR, Hunter JA, editors. Davidson's principles and practice of Medicine. 20th edition Churchill Livingston, Elsevier Publishers. 2006: 647–738.

21. Weisel C. Assessing Exposure to Air Toxics Relative to Asthma. Environmental Health Perspectives 2002; 110 (4): 527-37.

22. Mohan N, Patel T, Raiyani C.Pulmonary function status of shopkeepers of Ahmedabad exposed to automobile exhaust pollution. Indian J Physiol Pharmacol.1992;36(1):60-4.