Respiratory Medicine Case Reports 27 (2019) 100836

Contents lists available at ScienceDirect

Respiratory Medicine Case Reports

journal homepage: www.elsevier.com/locate/rmcr

Case report

Biochemical study on occupational inhalation of benzene vapours in petrol station

H.A. Abdel Maksoud[a], Mohamed G. Elharrif[b,*], M.K. Mahfouz[a], M.A. Omnia[a], M.H. Abdullah[c], M.E. Eltabey[a]

[a] Biochemisry Department, Benha University, Egypt
[b] Basic Medical Sciences Department, Shaqra University, KSA, Saudi Arabia
[c] Biochemisry Department, 6th October University, Egypt

ARTICLE INFO

Keywords:
Benzene vapours
Petrol stations workers
Oxidative stress

ABSTRACT

inhalation of benzene vapours promote various and dangerous health problems. Fuel station workers are most susceptible to benzene inhalation toxicity. Samples were collected twice, at beginning of the study and 6 months later from 40 fuel station workers from different egyptian governorates and 10 control healthy volunteers. Fuel station workers were sub divided into four groups according to years working in the station. All of them are exposed to benzene vapours and exhausts during their duties, their work shifts were 8 hrs./day. Results indicated that; benzene vapours exposure induced significant increasing in serum Lead and Cadmium and Myeloperoxidase (MPO) enzyme activity levels. This goes with marked immunologic changes presented by decreases in immunoglobulins; IgA and IgG, along with increases in levels of IgM and IgE. Also, Malondialdehyde (MDA) levels were significantly increased. Meanwhile, reduction in some other biochemical parameters including; Copper, Zinc and Iron micronutrients, as well as; Superoxide Dismutase (SOD), Catalase (CAT) enzyme levels and Glutathione (GSH) content. Hence, the study inferred that prolonged benzene inhalation can lead to biochemical and immune disorders, probably through potentiating oxidative stress and inflammation pathways.

1. Introduction

Air pollution was increased with urbanization and rapid increasing number of automobiles in most of the towns and cities, Numerous epidemiological studies have documented decrements in pulmonary function and various other health problems associated with long term air pollution exposure [1].

Benzene long term exposures can give rise to effects that include haematotoxicity, genotoxicity, immunological and reproductive effects as well as various cancers [2].

Benzene is a volatile compound in urban air pollution induce DNA oxidation [3]. Moreover, iron and other transition metals leaching from particles or by their presence on particle surfaces play a role in the generation of ROS in biological systems [4]. when oxidative stress is relatively low, various transcription factors, such as the nuclear factor erythroid-2 (Nrf 2), induce a series of antioxidant and detoxification enzymes (e.g., CAT, SOD, GST that counteract ROS formation protecting from adverse biological outcomes [5].

The volatile nature of petrol makes it readily available in the atmosphere any time it is dispensed, especially at petrol filling stations and depots. People are exposed to gasoline fumes during fuelling and refueling at gas stations, but the gas station attendants are more at risk by virtue of their occupational exposure [6]. So, spotting on fuel station workers who are chronically exposed to gasoline seemed necessary, Workers were mostly suffering neurological symptoms including; headache, tiredness, irritability and disturbed sleeping, as recorded in the utilized questionnaires and remarked by Ref. [7].

2. Materials and methods

The study was carried out on 40 fuel station workers from different egyptian governorates and 10 control healthy volunteers after taking consent of them. Fuel station workers are exposed to benzene vapours and exhausts during their duties, their work shifts were 8 hrs./day and sub divided into four groups according to years working in the station. **subgroup 1**: Consists of 10 workers working in petrol station for 2 years. **subgroup 2:** Consists of 10 workers working in petrol station for 4 years. **subgroup 3:** Contains 10 workers working in petrol station for

* Corresponding author.
E-mail address: al_harrif@yahoo.com (M.G. Elharrif).

https://doi.org/10.1016/j.rmcr.2019.100836
Received 15 March 2019; Received in revised form 2 April 2019; Accepted 3 April 2019

6 years. **subgroup 4**: Compromise 10 workers working in petrol station more than 6 years. All attendants were subjected to medical and occupational history including age, marital status, respiratory and chronic diseases, history of operation, drug intake, duration of employment, location and use of protective equipment. Any individual with history of diabetes, renal or hepatic disease and atrophy or allergy was excluded.

Blood samples were collected twice, at beginning of study and 6 months later by withdrawing of ten cubic centimeter of venous blood through a vein puncture using sterile vacutainer tubes. Half of the blood samples taken in heparin tubes (green caped tubes). The other taken to plain tubes (red caped tubes) which allow to coagulate then centrifuge for 10 minutes at 4000 r.p.m for getting clear and non hemolyzed sera aspirated carefully by Pasteur automatic pipette and transferred into dry and sterile labeled vials.

The sera were subjected to estimation of the following parameters; Copper, Zinc, Iron, Cadmium, and Lead by using 5 pg atomic absorption spectra-photometry (Pye Unicum), according the methods, described by Wilse, [8] and Bauer [9]. as well as Myeloperoxidse enzyme activity (Myeloperoxidase ELISA kit, Kamiya Biomedical Company) and total protein immunoglobulines; IgM, IgG, IgA [10], IgE [11] and nitric oxide [12].

Heparin 0.5% tubes used for preparation of hemolysate by Digitonine after washing erythrocytes by physiological saline as described by Korrnburg and Korecker [13] This hemolysate was subjected for quantitative determination of erythrocyte catalase [14,15], total super oxide dismutase [16], reduced glutathione [17], glutathione peroxidase [18] and malondialdhyde [19].

Statistical analysis: Data analysis was expressed as mean ± S.E. and analyzed for statistical significance by one-way ANOVA followed by Tukey's post-hoc test for multiple comparisons, using SPSS program for Windows version 22.0 (SPSS Inc., Chicago, USA). Values were considered statistically significant at $P < 0.05$ correlations between the measured variables were assessed by linear regression analysis by the least squares method.

3. Results

Results obtained in Table 1 showed a significant decrease in serum Copper, Zinc and Iron levels ($P < 0.05$) in fuel station workers exposed to benzene vapours for 2 and 4 years and highly significant reductions in Cu, Zn after 6 and more than 6 years of exposure when compared to control. This significant decrease was still noticeable in the 2nd sample after 6 months of the first one. Also, a highly significant decrease was found in serum immunoglobulines IgG and IgA levels at ($P < 0.05$) in spite of the increase in of IgM and IgE and serum Lead and Cadmium levels in workers exposed to benzene vapours for 2 and 4 years and highly significant increase after 6 and more than 6 years of exposure when compared to control, these significant changes were persisted in the 2nd sample. Moreover, serum nitric oxide showed a significant decrease at ($P < 0.05$) in workers exposed to benzene vapours for 2 and 4 years and highly significant decrease after 6 and more than 6 years of exposure in comparison with control and also, NO levels were still decreased significantly in 2nd sample after 6 months. Serum MPO levels showed a significant increasing in workers exposed to benzene vapours for 2 and 4 years and highly significant increasing ($P < 0.05$) after 6 and more than 6 years of exposure when compared to control in the first and second samples.

The data recorded in Table 2 showed; a significant decrease in RBCs SOD, CAT, GPx and GSH levels ($P < 0.05$) in fuel station workers exposed to benzene vapours for 2 and 4 years and highly significant reduction in RBCs SOD, CAT, GPx and GSH levels after 6 and more than 6 years of exposure when compared to control. This significant decreasing still registered in the 2nd sample after 6 months from first sample. Despite of increasing of Lipid peroxidation, MDA levels in workers exposed to benzene vapours for 2 and 4 years and highly

Table 1
Biochemical effect of benzene vapour on some serum biochemical parameters in Fuel station workers after 2, 4, 6 and more than 6 years of exposure.

IgE *IU/ml*	IgA *mg/dl*	IgG *mg/dl*	IgM *mg/dl*	MPO *ng/ml*	NO *μmol/l*	Cd *μg/dl*	Pb *μg/dl*	Fe *μg/dl*	Zn *μg/dl*	Cu *μg/dl*	Parameters & Groups		
28.28 ± 0.74^{e}	95.66 ± 1.31^{a}	763.70 ± 3.71^{a}	95.00 ± 0.87^{e}	20.32 ± 1.51^{e}	58.68 ± 0.94^{a}	26.19 ± 1.19^{e}	27.62 ± 1.01^{e}	110.22 ± 5.62^{a}	81.29 ± 2.76^{a}	78.60 ± 1.72^{a}	1st sample	Control normal	
31.08 ± 0.79^{e}	92.46 ± 1.23^{a}	752.80 ± 3.57^{a}	96.21 ± 0.91^{e}	22.13 ± 1.44^{e}	56.20 ± 1.14^{a}	27.34 ± 1.03^{e}	28.99 ± 0.94^{e}	106.72 ± 4.35^{a}	87.47 ± 2.55^{a}	86.79 ± 1.76^{a}	2nd sample		
46.94 ± 1.10^{d}	83.03 ± 1.35^{b}	653.80 ± 3.14^{b}	118.34 ± 0.92^{d}	26.89 ± 1.06^{d}	53.06 ± 1.77^{b}	28.91 ± 2.26^{d}	33.43 ± 2.64^{d}	68.73 ± 1.13^{c}	69.34 ± 1.83^{b}	70.82 ± 2.17^{b}	1st sample	2 years working	Fuel station workers
52.23 ± 1.06^{d}	77.42 ± 1.32^{b}	620.30 ± 3.65^{b}	124.90 ± 1.22^{d}	33.61 ± 1.13^{d}	45.76 ± 1.83^{b}	31.12 ± 1.95^{d}	35.93 ± 2.41^{d}	62.83 ± 1.36^{d}	65.44 ± 2.51^{b}	68.81 ± 2.25^{b}	2nd sample		
66.57 ± 0.90^{c}	74.67 ± 0.87^{c}	600.80 ± 2.24^{c}	139.50 ± 1.24^{c}	39.55 ± 0.93^{c}	40.63 ± 1.96^{c}	36.62 ± 1.76^{c}	47.23 ± 2.98^{c}	56.07 ± 2.04^{e}	61.48 ± 2.67^{c}	63.77 ± 2.59^{c}	1st sample	4years working	
73.63 ± 1.00^{c}	62.87 ± 1.08^{c}	564.50 ± 2.56^{c}	158.18 ± 1.65^{c}	46.36 ± 1.15^{c}	33.86 ± 2.12^{c}	41.30 ± 2.65^{c}	53.21 ± 1.25^{c}	51.12 ± 2.76^{e}	57.08 ± 2.58^{c}	58.41 ± 1.96^{c}	2nd sample		
81.09 ± 1.04^{b}	62.51 ± 1.14^{d}	537.40 ± 2.11^{d}	168.54 ± 1.82^{b}	49.02 ± 1.49^{b}	30.55 ± 2.00^{d}	44.91 ± 2.17^{b}	57.02 ± 1.37^{b}	63.68 ± 1.51^{d}	49.88 ± 2.86^{d}	51.77 ± 1.94^{d}	1st sample	6 years working	
90.22 ± 1.04^{b}	55.06 ± 1.07^{d}	511.40 ± 2.31^{d}	177.84 ± 1.55^{b}	53.19 ± 0.88^{b}	26.65 ± 2.56^{d}	49.89 ± 2.25^{b}	61.94 ± 1.67^{b}	71.29 ± 2.02^{c}	42.07 ± 2.02^{d}	48.32 ± 1.43^{d}	2nd sample		
102.55 ± 1.93^{a}	45.67 ± 1.35^{e}	486.40 ± 3.04^{e}	187.30 ± 1.20^{a}	59.20 ± 1.03^{a}	23.14 ± 1.96^{e}	53.94 ± 3.63^{a}	66.51 ± 1.22^{a}	74.59 ± 1.96^{b}	39.27 ± 2.96^{e}	40.22 ± 1.09^{e}	1st sample	More than 6 years working	
123.67 ± 1.55^{a}	36.38 ± 1.06^{e}	463.90 ± 2.35^{e}	195.94 ± 1.42^{a}	65.45 ± 1.04^{a}	19.80 ± 1.98^{e}	59.12 ± 3.41^{a}	72.42 ± 1.41^{a}	78.98 ± 1.53^{b}	35.47 ± 2.85^{e}	37.07 ± 1.11^{e}	2nd sample		

Data shown are mean ± standard deviation of number of observations within each group. Mean values with different superscript letters in the same column are significantly different at ($P < 0.05$).Small letters are used for comparison between the means within the column.

Table 2
Biochemical effect of benzene vapours on RBCs' lipid peroxidation and antioxidant parameters in Fuel station workers after 2, 4, 6 and more than 6 years of exposure.

GPx mU/mL	CAT U/L	SOD U/g. Hb	GSH mg/dl	MDA nmol/g. Hb	Parameters & Groups		
121.92 ± 1.33[a]	135.25 ± 3.41[a]	96.55 ± 1.84[a]	72.90 ± 1.34[a]	46.42 ± 1.36[e]	1st sample	Control normal	
118.11 ± 1.22[a]	130.26 ± 3.30[a]	92.41 ± 1.05[a]	69.23 ± 1.45[a]	43.52 ± 1.28[e]	2nd sample		
105.93 ± 1.09[b]	122.85 ± 3.12[b]	80.09 ± 1.66[b]	49.94 ± 0.78[b]	53.32 ± 1.30[d]	1st sample	2 years working	Fuel station workers
100.04 ± 0.98[b]	115.92 ± 2.02[b]	71.12 ± 1.81[b]	41.81 ± 0.96[b]	58.12 ± 1.39[d]	2nd sample		
94.28 ± 1.77[c]	109.11 ± 3.44[c]	76.58 ± 1.83[c]	42.65 ± 1.04[c]	64.84 ± 1.28[c]	1st sample	4years working	
88.48 ± 0.97[c]	98.06 ± 1.95[c]	68.41 ± 1.64[c]	36.41 ± 0.92[c]	74.02 ± 1.24[c]	2nd sample		
83.28 ± 1.09[d]	97.52 ± 1.96[d]	65.28 ± 1.90[d]	34.92 ± 1.05[d]	75.32 ± 1.15[b]	1st sample	6 years working	
77.50 ± 1.10[d]	91.02 ± 1.84[d]	59.08 ± 1.92[d]	29.12 ± 0.83[d]	81.72 ± 1.39[b]	2nd sample		
76.78 ± 1.05[e]	88.92 ± 1.08[e]	60.02 ± 1.76[e]	28.91 ± 1.26[e]	80.82 ± 1.34[a]	1st sample	More than 6 years working	
70.48 ± 0.84[e]	83.02 ± 1.04[e]	53.15 ± 1.91[e]	24.52 ± 1.01[e]	88.30 ± 1.48[a]	2nd sample		

Data shown are mean ± standard deviation of number of observations within each group. Mean values with different superscript letters in the same column are significantly different at ($P < 0.05$).Small letters are used for comparison between the means within the column.

significant increase after 6 and more than 6 years of exposure when compared to control and also, MDA levels were still increased significantly in 2nd sample.

4. Discussion

Risk of benzene exposure is mostly related to four main components; benzene, toluene, ethylene and xylene that known as BTEX [20]. However, benzene was found to be the most hazardous component due to mutagenic and carcinogenic effects of its metabolites [22]. Spotting on fuel station workers who are chronically exposed to benzene vapours seemed necessary, as properly achieved in the present study.

Moderate and long term exposures to benzene which carry the risk of numerous health problems including inorganic elements in such hazards. Our results was in agreement with [23] who found that, all of the accumulated metals are proposed to contribute to oxidative stress by different mechanisms. The underlying mechanisms of their toxicity involve formation of the superoxide radicals, hydroxyl radicals and other reactive oxygen species (ROS). Increased formation of ROS overwhelms body antioxidant protection [24], which may be the underlying cause of the decreased antioxidant activity observed in the present study, leading to possible induction of numerous conditions detrimental to health.

Results obtained in this study showed significant decrease in copper and zinc metal levels in comparison to control group. This came in agreement with Mona et al. [25], who reported that; both trace elements copper and zinc were significantly lower compared to peoples that are not exposed to fuels. These results are consistence with [21] who reported that; long to moderate benzene exposed individuals under oxidative stress due to decreased levels of antioxidants, including copper and zinc, in the plasma and red blood cells. In this study, copper and zinc were reduced by 51% and 56%, respectively, than lower limit of the accepted reference intervals and amounted 63% and 64% decrease to control individuals. Zinc and Copper are essential antioxidants in the human body [26]. These trace elements are essential component of many enzymes and proteins involved in protection against oxidative stress damage. Zinc deficiency associated with reduced immunity [27], DNA damage and increases other metals induced oxidative toxicity [28]. Copper is essential micronutrients for normal growth and protection of many organs and electron transport [29]. Copper deficiency increases risk of cellular oxidative damage due to decreased activity of superoxide dismutase (SOD) [30]). The balance between antioxidant, such as copper and zinc, and rate of free radicals levels are essential factor to prevent organs and tissue from oxidative damages [31]. The deficiency of copper and zinc antioxidants observed in petroleum fuel stations workers indicates the increasing of oxidative stress in such populations showed in previous results of our study.

The most risky toxic metal those lead which can cause abnormal alterations in the functioning of many vital organs [32] and associated with increased risk of hematological malignancies. Unfortunately, the majority of the workers are neglecting or lacking the protective safety measures such as; facemasks, protective cloths, this carelessness makes them more susceptible to those toxic fumes. Lead exposure may increase the susceptibility of membranes by altering their integrity via causing deterioration of their components [33]. Cadmium metal was found to be elevated in the blood benzene exposed individuals. Increase in lipid peroxidation represented by increased MDA level, has been observed in experimental animals treated with cadmium [34]. While cadmium itself is unable to generate free radicals directly, indirect generation of various radicals has been reported. Such mechanism involves displacement of other redox-active metals from their binding sites thus increasing their free form and enhancing their capability of producing free radicals [35]. found that; Fuel station workers who have exposed to benzene showed highly significant reductions in total antioxidant capacity (TAC) activities and high significant increased plasma MDA levels [36]. Malondialdehyde (MDA) is one of the end-products of the peroxidation of membrane lipids caused by ROS formation, especially by the superoxide ion. This is in agreement with other studies which illustrated that Benzene exposure has been associated with increases in the overall formation of MDA [37].

MPO is one of the most promising biomarkers of oxidative stress for clinical cardiologists [38]. Elevated circulating MPO levels have been found to be associated with the presence of CAD. In our present study, high MPO levels were able to predict increased risk of developing CAD in benzene vapours exposed workers, this was in agreement with Meuwese et al., [39] who stated that; high MPO levels were able to predict increased risk of developing coronary artery diseases (CAD) in healthy individuals. Most of attention was directed toward gasoline related immunotoxicity through decreasing number of immunoglobulins (IgA, IgG) which are often measured to give information about immune system homeostasis [40]. Current study recorded remarkable decreases in IgA and IgG levels with significantly elevated levels of serum IgM and IgE in fuel stations individuals compared to the unexposed ones. It was found that people with primary immunodeficiency have decreased levels of serum IgG and IgA and normal or elevated levels of IgM [41]. Decreased levels of immunoglobulins in gasoline exposed workers were explained to either suppression of immunoglobuline producing cells or decreased cell mediated immunity [3].

5. Conclusion

By the end of this study we concluded that, benzene vapours inhalation lead to potential biochemical and immunological disorders appeared as elevation of serum Lead, Cadmium, (MPO) and (MDA) levels. Also, markedly decreases in IgA and IgG, along with increases in levels of IgM and IgE. Meanwhile, reduction in some other biochemical parameters including; Copper, Zinc and Iron, (SOD), (CAT) enzyme levels and glutathione (GSH) content. This probably through potentiating oxidative stress and inflammatory pathways.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.rmcr.2019.100836.

References

[1] N. Aprajita, N.K. Parwar, R.S. Sharma, A study on the lung function tests in petrol-pump workers, J. Clin. Diagn. Res. 5 (5) (2011) 1046–1050.
[2] J. Kirkeleit, T. Riise, B.T. Gjertsen, B.E. Moen, M.B.Ø. Bråtveit, Effects of benzene on human hematopoiesis, Open Hematol. J. 2 (2008) 87–102.
[3] N. Uzma, S.S. Kumar, M.A.H. Hazari, Exposure to benzene induces oxidative stress, alters the immune response and expression of p53 in gasoline filling workers, Am. J. Ind. Med. 53 (12) (2010) 1264–1270.
[4] A.J. Ghio, J.H. Richards, J.D. Carter, M.C. Madden, Accumulation of iron in the rat lung after tracheal instillation of diesel particles, Toxicol. Pathol. 28 (4) (2000) 619–627.
[5] J.D. Hayes, M. McMahon, NRF2 and KEAP1 mutations: permanent activation of an adaptive response in cancer, Trends Biochem. Sci. 34 (4) (2009) 176–188.
[6] M. Lewne, G. Nise, M.L. Lind, P. Gustavsson, Exposure to particles and nitrogen dioxide among taxi, bus lorry drivers, Int. Arch. Occup. Environ. Health 79 (2006) 220–226.
[7] H.S. Abou-ElWafa, A.A. Albadry, A. El-Gilany, F.B. Bazeed, Some biochemical and hematological parameters among petrol station attendants: a comparative study, BioMed Res. Int. (2015) 1–6.
[8] J.B. Wiles, The determination of metals in blood serum by atomic spectrophotometery, Spectrochem. Acta 16 (1960) 259–272.
[9] J.D. Bauer, Clinical Laboratory Methods, ninth ed., the C.V. Mosby company, 1982, p. 11830 waistline industrial Missouri: 63146 U.S.A p: 112.
[10] J.T. Whicher, C. warren, R.E. Chambers, Immunochemical assays for immunoglobulines, Ann. Clin. Biochem. J. 21 (1984) 78–91.
[11] M. Plebani, D. Bernardi, D. Basso, F. Borghesan, D. Faggian, Measurement of specific immunoglobulin E: intermethod comparison and standardization, Clin. Chem. J. 44 (1998) 1974–1979.
[12] H.A.C. Montgomery, J.F. Dymock, The determination of nitrate in water, Analyst 86 (1961) 414–416.
[13] A. Korunburg, D. Korecker, Methods in Enzymology, Acad. Press, New York, 1955, p. 323.
[14] H. Aebi, Catalase *in vitro*, Methods Enzymol. 105 (1984) 121–126.
[15] P. Fossati, L. Prencipe, G. Berti, Use of 3,5-dichloro-2-hydroxybenzenesulfonic acid/4-aminophenazone chromogenic system in direct enzymic assay of uric acid in serum and urine, Clin. Chem. 26 (2) (1980) 227–231.
[16] M. Nishikimi, N.A. Rao, K. Yagi, The occurrence of superoxide anion in the reaction of reduced phenazine methosulfate and molecular oxygen, Biochem. Biophys. Res. Commun. 46 (1972) 849–854.
[17] E. Beutler, O. Duron, B.M. Kelly, Improved method for the determination of blood glutathione, J. Lab. Clin. Med. 61 (1963) 882–888.
[18] D.E. Paglia, W.N. Valentine, Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase, J. Lab. Clin. Med. 70 (1967) 158–169.
[19] K. Satoh, Serum lipid peroxide in cerebrovascular disorders determined by a new colorimetric method, Clin. Chim. Acta 90 (1978) 37–43.
[20] M.B. Abubakar, W.Z. Abdullah, S.A. Sulaiman, B.S. Ang, The effects of exposure to petrol vapours on growth, haematological parameters and oxidative markers in sprague-dawley male rats, Malays J Med Sci. 22 (1) (2015) 23–31 Jan-Feb.
[21] K. Ma, S.E. Rs, E. Ea, R. Refaat, Disturbed Homeostasis of Some Inorganic Elements Associated with Chronic Exposure to Low Levels of Benzene and Possible Associated Health Hazards vol. 3, (2014), pp. 22–30.
[22] A.O. Odewabi, O.A. Ogundahunsi, M. Oyalowo, Effect of exposure to petroleum fumes on plasma antioxidant defense system in petrol attendants, Br. J. Pharmacol. Toxicol. 5 (2) (2014) 83–87.
[23] M.A. Kotb, H.S. Ramadan, R.S. Shams El-Din, E.A. El-Bassiouni, R. Refaat, Disturbed homeostasis of some inorganic elements associated with chronic exposure to low levels benzene and possible associated health hazards, Eur. Sci. J. 3 (2014) February, 1857–7881.
[24] G.I. Evan, K.H. Vousden, Proliferation, cell cycle and apoptosis in cancer, Nature 411 (2001) 342–348.
[25] M.A.W. Ahmed, Amn Osman M. Zein, N.M.A. Shrif, Trace elements disturbance and liver toxicity in Sudanese fuel stations workers, Sch. Acad. J. Biosci. 4 (6) (2016) 498–501.
[26] Q. Shazia, Z.H. Mohammad, T. Rahman, H.U. Shekhar, Correlation of Oxidative Stress with Serum Trace Element Levels and Antioxidant Enzyme Status in Beta Thalassemia Major Patients: a Review of the Literature, Anemia, 2012, p. 270923.
[27] A.S. Prasad, Zinc in human health: effect of zinc on immune cells, Mol. Med. 14 (5–6) (2008) 353–357.
[28] K. Jomova, M. Valko, Advances in metal-induced oxidative stress and human disease, Toxicology 283 (2011) 65–87.
[29] M. Valco, H. Morris, M.T.D. Cronin, Metals, toxicity and oxidative stress, Curr. Med. Chem. 12 (2005) 1161–1208.
[30] Y. Pan, G. Loo, Effect of copper deficiency on oxidative DNA damage in Jurkat Tlymphocytes, Free Radic. Biol. Med. 28 (2000) 824–830.
[31] R.M. Schapira, A.J. Ghio, R.M. Effros, J. Morrisey, U.A. Almagro, C.A. Dawson, A.D. Hacker, Hydroxyl radical production and lung injury in the rat following silica or titanium dioxide instillation in vivo, Am. J. Respir. Cell Mol. Biol. 12 (2) (1995) 220–226.
[32] A. Ali Khan, R. Sultan, G.Y. Zamani, S.U. Rahman, Biochemical and hematological analysis after exposure to hazardous materials during shoe making, J. Biol. Life Sci. 4 (2) (2013) 116–128.
[33] H. Gurer, N. Ercal, Can antioxidants be beneficial in the treatment of lead poisoning, Free Radic. Biol. Med. 29 (2000) 927–945.
[34] V. Eybl, D. Kotyzová, L. Lesetický, M. Bludovská, J. Koutenský, The influence of curcumin and manganese complex of curcumin on cadmium-induced oxidative damage and trace elements status in tissues of mice, J. Appl. Toxicol. 26 (3) (2006) 207–212.
[35] A. Galan, L. Garcia-Bermejo, A. Troyano, N.E. Vilaboa, C. Fernandez, E. Deblas, P. Aller, The role of intracellular oxidation in death induction (apoptosis and necrosis) in human promonocytic cells treated with stress inducers (cadmium, heat, X-rays), Eur. J. Cell Biol. 80 (2001) 312–320.
[36] R.M. Hegazy, H.F.M. kamel, Oxidant hepatic &/or haem.Injury on fuel station workers exposed to benzene vapors, Possible protection of antioxidants, Am. J. Med. Med. Sci. 4 (2) (2014) 35–46.
[37] T. Georgieva, A. Michailova, T. Panev, T. Popov, Possibilities to control the health risk of petrochemical workers, Int. Arch. Occup. Environ. Health 75 (2002) 21–26.
[38] R.K. Schindhelm, L.P. van der Zwan, T. Teerlink, P.G. Scheffer, Myeloperoxidase: a useful biomarker for cardiovascular disease risk stratification? Clin. Chem. 55 (2009) 1462–1470.
[39] M.C. Meuwese, E.S. Stroes, S.L. Hazen, J.N. van Miert, J.A. Kuivenhoven, R.G. Schaub, N.J. Wareham, R. Luben, J.J. Kastelein, K.T. Khaw, S.M. Boekholdt, Serum myeloperoxidase levels are associated with the future risk of coronary artery disease in apparently healthy individuals, J. Am. Coll. Cardiol. 50 (2007) 159–165.
[40] R.E. Marques, P.E. Marques, R. Guabiraba, M.M. Teixeira, Exploring the Homeostatic and sensory roles of the immune system, Front. Immunol. Mar. 7 (125) (2016) 1–7.
[41] O.M. Akinosun, O.G. Arinola, L.S. Salimonu, Immunoglobuline classes in liver function tests in Nigerian petrol attendants, Indian J. Occup. Environ. Med. 10 (2) (2006) 58–61.