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The Biochemical Effects of Lead Concentration on Oxidative Stress Parameters in Workers

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Abstract

The goal of this study was to determine the blood lead concentration in various occupational workers in Sanandaj and its effects on oxidative stress parameters and other blood parameters. This descriptive-analytic study was carried out in Sanandaj, Iran. A total of 278 subjects were selected as the sample size for this study, of which 64 did not have exposure to theoccupational lead, and 214 had the occupational exposure tolead. The blood lead levels were measured by the atomic absorption spectrophotometer apparatus AA6800. Oxidative stress parameters and other blood parameters were measured by special devices. There was a significant positive relationship between the blood lead concentration and the oxidative stress parameters including lipid peroxidation, malondialdehyde, glutathione, antioxidant serum, and catalase. There was a significant negative relationship between the blood lead concentration and hematological parameters. Also, there was a significant positive relationship between the blood lead level and the lead concentration in the air.

Keywords: Blood lead levels, Oxidative stress, Blood parameters, Occupational exposure, Sanandaj

Introduction

Lead is one of the oldest metal toxins and is one of the most harmful pollutants in the environment that is found almost everywhere. Although lead is one of the most beneficial industrial elements but this metal is harmful to the function of the organs in the human body. Indeed, this metal cannot be decomposed in nature, so it will remain there as it enters the environment. Lead is used in more than 900 industries includes mining, melting, battery plants, color production, and production of polished oil (Karrari et al., 2012). Lead exposure can be occurred in many ways, including through food, drinking water pipes, smoking, and inhalation of polluted air in industrial areas or near roads (Vaglenov et al., 2001). Lead has been shown to have adverse health effects that include neurotoxicity, renal nephron toxicity, harmful effects on the cardiovascular system, immune system diseases such as allergy type 1, birth weight loss, early birth and stomach, lung and bladder cancers (Ekong et al., 2006). The International Agency for Research on Cancer (IARC) in 1986 classified lead as carcinogen in Group 2B, and in 2006, based on new animal experimentation data, this category was changed to group 2A, "possibly carcinogenic to humans" (Baan, 2010, on the Evaluation, 2006). There is 2% of the body's absorbed lead in the blood and its half-life is 35 days. Bones and teeth absorb 95% of lead, which itshalf-lifein these tissuesis between 20 and 30 years (Gordon et al., 2002). Blood lead level (BLL) is the best indicator for exposure to lead and for the biological monitoring of the occupational population, this metal is used. There are a lot of experiments available to investigate exposure of lead to workers, but the BLL test has been accepted as the most appropriate method for determining therapeutic measures. According to ACGIH, the biological exposure index (BEI) is 30 µg/dl. Also, according to Occupational Safety and Health Administration Occupational Safety and Health Administration (OSHA), the levels of blood lead in workers requiring medical treatment are 40µg/dl and the amount required to be taken for releasing, is 60µg/dl. Because of the high importance of the subject, The United States Centers for Disease Control and Prevention (CDC) has changed the level of blood lead level requiring individual management, including clinical interventions, from 60 µg/dl to 35 µg/dl in 1975, and in 1985, it changed to 25 µg/dl, and in 1991, it decreased to 10 µg/dl (Health and Services, 1995, ABDOLLAHI et al., 1996a). Oxidative stress occurs when the total antioxidant capacity of the body is less than the amount of oxidative compounds which forms during the metabolic activity of the cell. Active oxygen species (ROS), including hydroperoxides, superoxide, radical hydroxyl, radical oxygen, hydrogen peroxide,

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are active molecules and radicals that have high redox potential and attack cell macromolecules, and if there is not enough antioxidant defense, they can lead to oxidative stress, which eventually can result in problems like cardiovascular disease or cancer (Valko et al., 2005). Heavy metals and, in particular, lead can contribute to their toxicity through oxidative stress. Lead can disable glutathione and result in the depletion of antioxidants in the cellby attacking the thiol groups of enzymes (Flora et al., 2012). Contrary to acute toxicity of lead, chronic toxicity occurs in serum concentrations of lead below the known limits. Oxidative stress is one of the main causes of chronic lead-induced toxicity (Ahamed and Siddiqui, 2007). A study of 20 male workers working at a battery plant and 16 as control group, by measuring the levels of malondialdehyde (MDA) and decreasing glutathione levels, have shown a significant correlation between oxidative stress parameters and clinical indexes in comparison with the control group. Atomic absorption spectrophotometric technique was used to measure the level of lead in the blood and the average serum levels of lead that lead to oxidative stress in the workers was 55 ± 17 μg/dl (Gurer-Orhan et al., 2004).

Given the importance of the subject and the possibility of exposure of workers which works in gas stations, welding, battery plants, painting and printing and duplicating to lead, and possibility of high levels of lead in the blood higher than standards and guidelines, the purpose of this study was to determine the blood lead level in Sanandaj city's workers and measuring oxidative stress and determining their relationship with parameters which can be indicative of oxidative stress in these individuals. In this study, blood lead levels were measured in a city, one season and one time interval. Blood lead levels, oxidative stress parameters, and other blood parameters were compared in different occupations.

Materials and Methods

The present study is a descriptive-analytic study in various workers of Sanandaj (non-exposed people, gas station staff, welding, battery plant workers, mechanical, painting and printing and duplicating). Due to the type error of 5% and the study's strength of 90%, for the non-exposed group, 64 people and for the exposed group, 214 were selected. Of the exposed people, 64 were gas station's staff (because more people work at gas station) and for each of the other occupations, 30 were considered. The entry criteria for the case group included male gender, with the age of 25 to 50, and at least two years' work experience. The exclusion criteria included workers who used chelating agents such as sucrose, $CaNa_2EDTA$, BAL or D-penicillamine, alcohol, cigarettes and drugs for 6 months before taking the blood sample. The control group consisted of non-exposed individuals or indicators of causing oxidative stress, which based on demographic data in terms of age, gender, ethnicity and daily activity were similar to those in the case group. To perform this study, a demographic questionnaire containing information about age, smoking, history of disease, work experienceand etc. was completed. To provide a sample of blood from workers, we used sterile disposable syringe and about 7 ml of blood of each person in heparin tubes was collected by a nurse and then transferred to the lab.

BLL measurement: To determine the amount of lead in blood, 1 ml of the blood sample was transferred to a 10 ml polystyrene tube and then digested with 0.5 ml of concentrated nitric acid and kept at 30 °C to dry and after drying, mixed with distilled water and then filtered with Wattman filter paper. The AA6800 atomic absorption spectrophotometer was used to estimate blood lead levels.

Lipid peroxidation measurement: Measuring lipid peroxidation was measured by Thiobabitoric Acid method (TBA). In this way, 0.2 ml of serum was added to 0.1 ml of the TBA reagent including tricloroacetic acid, and after incubation n-butanol was added and then centrifuged and the absorbance was measured by a spectrophotometer at 532 nm (Kei, 1978).

Glutathione measurement: Trichloroacetic Acid 2% was added to one ml of serum and after centrifugation, two ml of disodium hydrogen phosphate and 0.5 ml Alman solution (DTNB) was added to one ml of supernatant, and after 15 minutes of incubation, the absorbance was measured at 412 nm. The glutathione concentration was obtained from the standard glutathion curve in terms of nanomol/ml (Ellman, 1959).

Total antioxidant capacity in serum: Total antioxidant capacity in serum was measured using FRAP method. In summary, the ability of plasma to recover ferric ions in the presence of a specific agent and the formation of a blue complex is the basis of this method, which can be reported by reading at 539 nm in micromole per milliliter of blood (Benzie and Strain, 1996).

Malondialdehyde Measurement: Measuring malondialdehyde was performed using modified Aust and Buege methods. In this method, at first one ml of serum was added to one ml trichloroacetic acid 20% w/v in 0.6 M HCl, after mixing, centrifuged in 2000 g for 15 minutes. Then 0.3 ml of 0.12 M Thiobarbitoric Acid solution in 0.26 M Tris with pH = 7 was added to 5 ml of the supernatant, and incubated for 15 minutes at 100 °C. After cooling, the tubes were removed and their absorption was measured at wavelength of 535 nm (Shahsavari et al., 2015, Buege and Aust, 1978).

Activity of catalase Measurement: The activity of catalase in serum was measured according to the Aebi method. This method is designed based on the decomposition of hydrogen peroxide by catalase. A reaction was initiated by adding 0.5 ml of H_2O_2 at a concentration of 50 mM to the mixture. Reduction of absorption due to the peroxide decomposition at 240 nm, which is proportional to the activity of catalase at 1, 2 and 3 minutes, was measured by the average catalase activity absorption per minute in mU/L (Kharazi-Nejad et al., 2014, Aebi, 1984).

blood parameters Measurement: For measurement of hemoglobin, the red blood cells and white blood cells, we used the SYSMEX XT 4000i cell counter (Malekirad et al., 2011). For

measuring blood pressure, we used a portable Blood Pressure Monitor. Sampling of air was performed using a SKC-224 filtered pump connected to a Teflon tube. Adjustment of the flow between the sampler and the pump was achieved using a flow meter. An AA 6800 atomic absorption spectrophotometer was used to measure the amount of lead in the environment (Bahrami et al., 2002). After collecting the results, the information was entered to the SPSS software version 22, and according to the objectives of the study, for the descriptive-qualitative purposes the frequency and their percentages were calculated. For descriptive-quantitative variables, mean and standard deviation were calculated. For analytical purposes, after reviewing normal and non-normal of quantitative data, for normal data, appropriate parametric tests (T-test, ANOVA) and for non-normal data non-parametric tests were used (Mann-Whitney, Kruskal-Wallis). Chi-square test was used to analyze the two-quality qualitative data.

Results

Due to the type error of 5% and the study strength of 90%, for the non-exposed group, 64 people and for the exposed group, 214 were selected. Of the exposed group, 64 of them were gas station's staff, and of each of the welding, battery plant, mechanic, printing and duplication and paintings professions, 30 subjects were selected as samples. The age range of participants was between 25 and 50 years. All participants were male and 43% of exposed group and 37% of non-exposed group had higher education than diploma. For non-exposed group, characteristics such as blood lead, as well as blood and environmental parameters have been shown in table 1.

Table 1: Blood lead levels, lead levels in the air and blood parameters

parameters	group	minimum	maximum	mean	Std. deviation
Blood lead	1*	9.0000	21.0000	14.7500	3.61654
	2*	29.0000	45.0000	37.4375	3.93952
	3*	56.0000	71.0000	63.3500	4.60912
	4*	24.0000	77.0000	49.6000	19.27407
	5*	26.0000	43.0000	35.6167	5.14226
	6*	7.0000	35.0000	20.3167	8.75577
	7*	32.0000	71.0000	49.3833	11.70005
Air lead	1	11.0000	26.0000	16.5156	4.02765
	2	31.0000	50.0000	39.5938	4.18887
	3	58.0000	76.5000	66.4500	5.34556
	4	27.0000	85.0000	52.3667	18.43625
	5	28.0000	46.0000	37.7667	4.39187
	6	9.0000	36.0000	22.4333	8.31257
	7	34.0000	92.0000	56.7000	16.78906
Time of activity	1	25.0000	50.0000	35.7344	6.92417
	2	34.0000	67.0000	49.5000	7.64074
	3	48.0000	108.0000	73.5667	15.33912
	4	35.0000	109.0000	63.5333	22.81822
	5	36.0000	123.0000	77.6000	28.55316
	6	30.0000	94.0000	57.4000	19.32053
	7	38.0000	138.0000	84.8000	31.25137
Blood pressure	1	8.5000	12.5000	10.2187	1.19813
	2	8.5000	12.5000	10.8516	1.18080
	3	8.5000	12.5000	10.6333	1.3456
	4	8.5000	12.5000	10.1500	1.16818
	5	8.5000	11.5000	9.6500	0.82158
	6	8.5000	10.5000	9.6333	0.58624
	7	8.5000	12.000	10.5667	1.14269
Number of pulses	1	49.0000	68.0000	55.2344	4.27893
	2	49.000	68.0000	58.6094	4.77300
	3	49.000	68.0000	54.7000	4.41119
	4	49.000	62.0000	55.2667	3.52267
	5	49.000	68.0000	59.1333	5.02225
	6	49.000	59.0000	53.9000	3.65164
	7	49.000	61.0000	54.3667	3.24285
Number of red cell	1	5.5000	6.0000	5.7578	0.25185
	2	4.0000	5.0000	4.6250	0.25198
	3	4.1500	4.4700	4.3133	0.10087
	4	4.1500	5.4500	4.8337	0.52366
	5	4.5000	5.5000	4.7917	0.32776
	6	5.0000	6.0000	5.5517	0.35172
	7	4.1500	5.2000	4.5520	0.28432
Number of white cell	1	5.0000	10.0000	7.4135	1.71760
	2	4.0000	8.0000	5.8133	1.08709
	3	4.0700	5.1000	4.4237	0.36035
	4	4.1500	9.0000	5.6150	1.43424
	5	4.5000	9.0000	6.3250	1.06194
	6	5.6000	10.0000	7.8500	1.35156
	7	4.2000	6.5000	4.9333	0.65183
hemoglobin	1	12.5000	17.0000	15.1972	1.21215
	2	12.0000	17.0000	13.9934	1.19636
	3	12.5000	17.0000	14.8860	1.18472
	4	14.0000	17.0000	15.5667	1.07265
	5	13.5000	17.0000	15.1180	1.16403
	6	12.5000	17.0000	15.2167	1.18673
	7	12.5000	17.0000	14.6527	1.12148
glutathione	1	600.000	730.000	673.5938	27.27678
	2	700.000	850.000	782.4219	31.77291
	3	800.000	940.000	884.8333	30.41334
	4	720.000	955.000	847.000	73.09182
	5	700.000	820.000	759.766	28.42932

	6	640.000	780.000	704.8333	49.76641
	7	760.000	910.000	831.3333	43.44901
catalase	1	130.000	210.000	164.0625	21.80132
	2	230.000	260.000	243.2812	8.78395
	3	265.000	325.000	294.8333	20.53103
	4	220.000	335.000	262.1667	34.45845
	5	220.000	255.000	239.5000	9.03537
	6	130.000	240.000	187.3333	38.83505
	7	235.000	320.000	263.3333	21.34823
Malone De Aldehyde	1	8.000	20.000	13.2109	3.40145
	2	25.000	41.000	33.0156	3.89339
	3	40.000	45.000	42.9000	1.29588
	4	20.000	46.000	34.4000	9.27585
	5	22.000	39.000	30.8333	5.35681
	6	8.000	30.000	16.5667	7.19523
	7	28.000	44.000	37.7000	4.83629
Lipid peroxidation	1	2.500	3.900	3.1797	0.44630
	2	3.900	5.500	4.5797	0.49093
	3	5.600	6.200	5.9100	0.20401
	4	4.000	6.500	4.9200	0.90493
	5	3.000	4.000	3.4933	0.24344
	6	2.200	3.600	2.8233	0.48115
	7	2.800	6.100	4.1300	0.93298
Total antioxidant	1	1.200	1.400	1.3406	0.07064
	2	1.500	1.800	1.6250	0.07766
	3	1.800	2.200	1.9767	0.13566
	4	1.400	2.500	1.7767	0.35300
	5	1.400	1.700	1.5667	0.10613
	6	1.200	1.500	1.3700	0.09523
	7	1.500	2.100	1.7367	0.15196

1 * = no exposure, 2*=gas station, 3*=welding, 4*=car repair shop, 5*=battery manufacturing, 6*=printing and duplicating, 7*=painting

The findings of the study showed a significant relationship between blood lead level and lead concentration in air ($p < 0.05$). There was a significant relationship between blood lead level and all blood parameters except pulses ($p < 0.05$), the relationship between blood lead levels and the pulse rate was meaningless ($p = 0.084$). Also, the relationship between blood lead level and oxidative stress parameters was significant ($p < 0.05$) (Table 2).

Table 2: Relationship between blood lead levels and other parameters

Blood lead	Correlation Coefficient	Sig. (2-tailed)	N
Lead in air	0.979	p<0.05	278
Blood pressure	0.226	p<0.05	278
pulses	0.104	p>0.05	278
hemoglobin	-0.206	p<0.05	278
White cell	-0.884	p<0.05	278
Red cell	-0.959	p<0.05	278
glutathione	0.952	p<0.05	278
catalase	0.991	p<0.05	278
Malone De Aldehyde	0.994	p<0.05	278
Lipid peroxidation	0.878	p<0.05	278
Total antioxidant	0.984	p<0.05	278

All parameters were measured in downtown and slum areas of the city. 143 points of slum areas of city and 135 points of downtown were selected and measurments were carried out at these points. All parameters measured on the downtown and slum areas of the city are given in Table 3.

According to Table 4, there was a significant relationship between blood lead levels in different occupations and sampling site (slum areas and downtown) ($p < 0.05$).

Table 3- Different parameters in the slum areas of city and downtown

City's area	parameters	number	minimum	maximum	mean	Std. deviation
Slum areas	Blood lead	143	7	64	31.0245	15.80485
	Lead in air	143	9	66	34.1014	15.66682
	Time of exposure	143	25	95	47.2937	15.41850
	Blood pressure	143	8.5	12.50	10.2552	1.23172
	pulses	143	49	68.00	56.1678	4.95328
	hemoglobin	143	12.5	17.00	15.0331	1.28129
	White cell	143	4.22	10.00	6.9152	1.74992
	Red cell	143	4.31	6.00	5.1278	0.57565
	glutathione	143	70.00	890.00	747.4476	94.22939
	catalase	143	130.00	300.00	216.0839	48.17858
	Malone De Aldehyde	143	8.00	43.00	25.3182	11.45698
	Lipid peroxidation	143	2.20	6.00	3.7524	1.04804
	Total antioxidant	143	1.20	2.00	1.5189	0.20520
downtown	Blood lead	135	9.00	77.00	40.3815	18.99079
	Lead in air	135	11.00	92.00	42.8593	21.38407
	Time of exposure	135	27.00	138.00	69.6222	27.40679

	Blood pressure	135	8.50	12.50	10.3778	1.13701
	pulses	135	49.00	68.00	56.1111	4.31358
	hemoglobin	135	12.00	17.00	14.6790	1.24895
	White cell	135	4.00	10.00	5.4414	1.16332
	Red cell	135	4.00	6.00	4.8333	0.60673
	glutathione	135	600.00	955.00	793.0741	83.84640
	catalase	135	130.00	335.00	241.3704	47.58115
	Malone De Aldehyde	135	8.00	46.00	31.1852	11.15842
	Lipid peroxidation	135	2.50	6.500	4.4319	1.04240
	Total antioxidant	135	1.20	2.500	1.6696	0.27651

Table 4- Relationship between Blood lead level with the sampling site (slum areas and downtown)

Sampling site	Study groups	Number of sample	Blood lead	sig
Slum areas	No exposure	30	11.9667	p<0.05
	Gas station	28	35.8929	
	welding	17	59.9706	
	Battery maker	17	34.3529	
	mechanic	17	32.0249	
	Printing and duplicating	17	13.8529	
	painting	17	40.5294	
downtown	No exposure	34	17.2059	
	Gas station	36	38.6389	
	welding	13	67.7692	
	Battery maker	13	69.5385	
	mechanic	13	40.3077	
	Printing and duplicating	13	26. 7692	
	painting	13	60.9615	

Discussion

In the present study, the average blood lead level in non-exposed groups, gas station workers, welder, battery plant, mechanics, painter, printing and duplicating was 14.7500 µg/dl, 37.4375 µg/dl, 63.3500 µg/dl, 49.6000 µg/dl, 35.6167 µg/dl, 20.3167 µg/dl, 49.3833 µg/dl, respectively. Therefore, the level of lead in non-exposed group, gas station, mechanics and printing industry was so high that it requires individual management, such as clinical measures. According to the OSHA standard, the level of lead in the battery plants and the painter is so much that needs medical treatment, and the lead level of the welder requires actions such as releasing from the job (Health and Services, 1995). According to a review study conducted by Azami et al in 2017, blood lead levels in workers at the gas station, welder, battery plant, mechanic, printing and duplicator were 30.05 µg/dl, 67.02 µg/dl, 43.3 µg/dl, 38 µg/dl, 36.6 µg/dl, 47.84 µg/dl, respectively. Therefore, the blood lead level in various occupations with blood lead levels in the study by Sayehmiriet al. were approximately similar and there was little difference (Sayehmiri et al., 2016).

In a study conducted by Orhan et al., blood lead levels in a non-exposed group were 11.8 µg/dl, which is in accordance with blood lead levels in this study (Gurer-Orhan et al., 2004).
In other studies, blood lead levels in the battery plants, printing and duplicating and painting occupations were 96.7 µg/dl, 75 µg/dl, and 27.76 µg/dl, respectively, which have obvious contradictions with the blood lead level of occupations in this study (Sayehmiri et al., 2016, Kermani and Niktab, 2005, ABDOLLAHI et al., 1996b). For this discrepancy, we can represent different reasons such as various working hours, using materials with a different levels of lead, using personal protective equipment etc.

In Iran, the blood lead level of all workers is 42.8 µg/dl. There are several reasons why there is a high blood lead level in Iran, which may include the inappropriate use or misuse of personal protective equipment, high levels of lead in the materials used by the staff, food's exposure, exposure time and age, while the average exposure of all workers with lead in Korea is 4.35 µg/dl (Golpayegani and Khanjani, 2012, Wrońska-Nofer et al., 2015, Gebrie et al., 2014, Basit et al., 2015). Among the exposed industries mentioned in this study, the average blood lead in the weld industry was highest and in printing and duplicating industry was the lowest. The high level of lead in the welding can be due to metallic vapours from galvanized sheet welding, welding of sheets with silicate zinc coatings, indoors places, and lack of suitable ventilation (Shahrabi Farahani et al., 2006).

There was a significant relationship between blood lead level and air lead level ($p < 0.05$), so that, the more lead in the air, the more blood lead levels, too. In a study conducted by Bahrami et al., a significant relationship was observed between the concentration of lead and blood lead levels ($p < 0.05$) (Bahrami et al., 2002).

There was a significant relationship between blood lead level and blood pressure level, which means, as the blood lead level increased, blood pressure increased, too ($P < 0.05$). In a study by Cheng et al., there was a positive significant relationship between blood lead and blood pressure levels. Also, in a study conducted by Nash et al., this relationship was positivly significant ($p < 0.05$) (Cheng et al., 2001, Nash et al., 2003).
In a study carried out by Malekirad et al., there was no significant relationship between blood lead level and blood pressure and this was due to the high level of antioxidants and zinc, which prevented the effect of lead on blood pressure (Malekirad et al., 2011).

No significant relationship was found between blood lead level and pulse rate ($p < 0.084$). In a study carried out by Malekirad et al., there was no significant relationship between blood lead level and pulse rate (Malekirad et al., 2011).

There was a significant negative relationship between blood lead level and white blood cell, red blood cell, and hemoglobin levels ($p < 0.05$); as blood levels increased, levels of these blood parameters decreased. A study by Apostoli et al. revealed the high levels of lead destroyed the red blood cell membrane (Apostoli et al., 1988). Another study by Mishra et al showed a significant negative relationship between blood lead levels and lymphocytes ($p < 0.05$) (Mishra et al., 2010). High blood lead level can cause erythrocyte and reticulocyte in the blood and ultimately reduce hematocrit. Therefore, increasing blood lead level can lead to reduction of hemoglobin levels. Lead can also inhibit sodium-potassium pump, which reduces the life span and results in reduction of red blood cell number (Mitema et al., 1980, Ding et al., 2001, Aminipour et al., 2008).

There was a significant positive relationship between blood lead and total antioxidant levels ($p < 0.05$), so that the level of total antioxidant of plasma increased with increasing blood lead level. A study carried out by Arinola et al. showed that here was a significant positive relatioship between blood lead level and total membrane antioxidant (Arinola and Akiibinu, 2006). In another study by Fani et al., this relationship was significantly positive (Malekirad et al., 2011). The reason for increase in total antioxidants is the increase in free radicals due to lead toxicity and finally, more antioxidants are produced to neutralize free radicals. In a study by Payal et al., increased lead levels reduced total membrane antioxidants (Payal et al., 2009).

In the present study, the relationship between blood lead level and glutathione levels was significantly positive ($p < 0.05$), so that glutathione increased with increasing blood lead level.In a study carried out by Gurer et al., a significant positive relation was found between blood lead level and glutathione levels ($p < 0.05$). With high blood lead level, glutathione level increases to protect RBC against oxidative stress (Gurer-Orhan et al., 2004).The relation between blood lead level and catalase levels was significantly positive ($p < 0.05$). A study carried out by Cohen et al. showed that increasing the blood lead level leads to an increase in catalase level. Increased catalase levels are all for H_2O_2 decomposition in red blood cells and indeed, catalase production is a reaction to protect RBC against H_2O_2 through the process of oxidative stress (Cohen and Hochstein, 1963).

Lipid peroxidationwas increased significantly with increasing blood lead level ($p < 0.05$). In a study by Moro et al., the level of lead in staff that are exposed to color, lipid peroxidation and enzymatic antioxidants was significantly increased (Moro et al., 2010).

Increasing blood lead level causes a significant increase in malondialdehyde level ($p < 0.05$). In a study conducted by Orhan et al., malondialdehyde concentration as an index for lipid peroxidation with blood lead level have a significant relation and there was also a significant relationship between malondialdehyde concentration and other clinical indicators related to lead toxicity (Gurer-Orhan et al., 2004). In another study by Guror et al., the mice F344 exposed to lead which led to an increase in the level of melandidealdehyde (Gürer et al., 1998).

There was a significant relationship between sampling site and blood lead level, and blood lead level in the employees of downtown was higher than the level of blood's lead in the slum area's staff ($p < 0.05$). In downtown, the number of vehicles and other factors producing heavy metals, including lead, is more than the slum areas of city, therefore, the concentration of lead in the air is increased, exposure level of people and staff will be more with lead and consequencely results in more level of lead in their blood. But on the slum areas of the city, due to the small number of cars and the flow of more airflow, staff are exposed to less lead. In a study by Bahrami et al., there was a significant relationship between the concentration of lead in air in the gas station in downtown and in the slum areas of the city. Also, there was a significant relationship between blood lead level and place of sampling (slum areas or downtown) ($p < 0.05$) and the concentration of lead in air and blood lead level in downtown was more than the slum areas. The reason is also the difference in the number of vehicles (Bahrami et al., 2002).

Conclusion

In professions which faced a lot with chemicals and metals, blood lead levels are significantly increased. With regard to lead losses and effects on the blood circulation system and other organs, it is necessary to take the necessary protective measures to reduce exposure to lead including: 1- Control measures in the origin, such as reducing the amount of heavy metals, including lead in chemicals used by different industries and replacing less toxic substances, 2-Controlling in the end, such as reducing exposure to heavy metals, including lead, through suitable personal protective equipment such as chemical agents, masks, etc.

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