Relationship of Lead Exposure with Workers' Blood Pressure and Blood Components: A Case Study

Salman Torabi Goodarzi[1], Farideh Golbabaei[2], Bahram Harati[3], Robabeh Chang[4], Vahid Ahmadi[1], Mohsen Sadeghi Yarandi[1*]

[1]MSc Student of Occupational Health Engineering, School of Public Health, Tehran University of Medical Sciences, Tehran, Iran• [2]Professor of Occupational Health Engineering, Department of Occupational Health, School of Public Health, Tehran University of Medical Sciences, Tehran, Iran• [3]MSc of Occupational Health Engineering, School of Public Health, Tehran University of Medical Sciences, Tehran, Iran• [4]MSc of Occupational Health Engineering, School of Public Health, Hamedan University of Medical Sciences, Hamedan, Iran•
*Correspondence author email: Eng.Sadeghi1995@Gmail.Com, Tel: +98-912-0872898

Abstract

Background: Lead is one of the most widely used metals in the industries and is the amplest metal element on the Earth's crust. This metal disrupts the physiological processes of the body due to its accumulation in various tissues. The present study aimed to determining the association of lead exposure with blood pressure and blood components of the lead mine employees in Isfahan City, Iran. **Methods :**In this cross-sectional study, 187 workers were investigated; they worked in the lead mines of Isfahan from January to April 2016. The staff was divided into two groups: one was exposed to lead and the other was the control group. The participants' general information was collected in worksheets, including age, work experience, work shift, body mass index, blood pressure, and smoking status. Sampling was carried out by active sampling using the NIOSH 7300 method. Blood samples were taken from all participants and their hematological parameters were evaluated, including red blood cell, platelet count, percent of lymphocytes, and volume of red blood cells. **Results**: The participants' age means were 34.06 (8.8) and 37.04 (11.48) years in the case and control groups, respectively. The time-weighted average concentration of lead in the breathing zone air of workers was 0.0533 mg/m3. The average systolic blood pressure was 12.01 (1.3) mmHg in exposed workers, while it was 11.78 (1.1) mmHg in the control group. The average diastolic blood pressure was 7.84 (0.71) mmHg in exposed workers and 7.73 (0.54) mmHg in control group. Statistical test results showed a significant difference between the case and control groups with regard to their systolic and diastolic blood pressure ($P<0.05$). Furthermore, no significant difference was observed between the lead-exposed and non-exposed groups with regard to the hematocrit and hemoglobin levels. **Conclusion :**According to the results, lead exposure can cause metabolic changes in blood pressure and some of its features. Considering the importance of this issue, some preventive measures should be taken to maintain and improve the worker's health and well-being, such as increasing the workers' rest time, performing periodic examinations for them, as well as conducting safety workshops and training sessions.

Keywords :Lead; Blood pressure; Blood components; Lead mine

Introduction

Metals long-term exposure can lead to significant problems.[1] Some metals are beneficial to health, while others can cause toxicity and occupational diseases.[2] Lead is one of the most commonly used metals in industry and the most abundant heavy metal element on the Earth's crust.[3] although exposure to lead has declined

dramatically during the last two decades, it is still present in industrial and urban areas as a toxic environmental pollutant.[4,5] Lead is scattered in working environments due to its extensive use in industrial areas.[3] Generally, increased concentrations of pollutants in the work environment can lead to their increase in the human body.[5] Employees' exposure to pollutants in industrial environments may increase absorption of toxins in their body. In turn, this disrupts their body's physiological processes and leads to occupational poisoning as well as chronic diseases.[6-8] Lead and its components are toxic substances that cause toxicity in peripheral and central nervous systems, kidney, gastrointestinal system, cardiovascular, internal glands, and hematopoietic systems due to their accumulation in various tissues of the body.[9] Anemia and encephalopathy are common complications caused by lead exposure.[10,11] Reduction of oxygen supply into tissues, weakens and reduces the body function, decreases blood hemoglobin levels, and causes anemia.[12]

Decreased blood hemoglobin level (lower than 13.7 mg/dl for women and lower than 12.6 mg/dl for men) and increased blood hemoglobin level (greater than 14.1 mg/dl for women and greater than 15.6 mg/dl for men) increase the risk of mortality rate by 17%.[12] Junco-Munco conducted a study on 469 Monterry residents in 1996 and showed a significant correlation between the lead levels in the air of four urban areas and the levels of blood lead in individuals.[13] Exposure to lead metal in mines and workplaces enters lead into the human blood plasma.[3] Most researchers have focused on the relationship between toxic effects of lead and blood pressure in recent years.[14-16] According to the statistics, 8% of adults in the United States have hypertension, which is a major risk factor for stroke and heart attack.18 Some studies reported a correlation between lead exposure and changes in blood pressure.[9,17] Navas-Acien et al. conducted a study in 2008 and found that exposure to lead and increased lead accumulation in the blood could increase the blood pressure, although these changes were low.16 Cheng et al.'s study at Harvard University showed that respiratory exposure to lead increased blood pressure even at low concentrations.[19]

Most researchers believe that by doubling the level of lead in the blood, systolic blood pressure rises from 0.8 to 1.25 mmHg.[16] Occupational exposure limit for 8-hour respiratory exposure to lead and its inorganic components is 50 micrograms per cubic meter according to the Iran Occupational Health Technical Committee. The International Agency for the Research of Cancer classified lead and its inorganic derivatives under the category 2A (possibly carcinogenic to humans).[20] Given the high exposure of workers in lead mines, the impact of lead on blood pressure and many blood parameters, the idea that changes in blood pressure are among the risk factors for cardiovascular problems, anemia[19], and increased death rates[21], and the fact that no study has ever been conducted in this area, this study was carried out. The aim was to monitor the respiratory exposure status of lead mine workers and investigate the effect of lead exposure on workers' blood pressure and blood components. This study aimed to determining the association of lead exposure with blood pressure and blood components in lead mine employees in Isfahan province in 2016.

Methods

Study population

This cross-sectional study was carried out among 187 employees of a lead mine in Isfahan in 2016. Among the participants, 164 were exposed to lead and 23 were selected as the control group. All exposed workers were selected from the operational section of the mine and all participants present in the non-exposed (control) group were employed in the administrative department. Participants were fully informed about the study goals and procedures; later, written consent forms were collected from them. The Helsinki Declaration principles were taken into account to carry out the study. The demographic information as well as medical and occupational information was collected using case study method and direct interviews, in some cases. The interviews were recorded in the information sheet designed to conduct this study. Next, the medical records of the workers who had no history of hypertension and specific diseases were collected during the examination. The participants' age was divided into four groups of 20-29, 30-39, 40-49, and 50≤ years. Work experience was classified into five groups of< 5, 5-10, 11-15, 16-20 years and, above 20 years. According to education level, participants were divided into four categories of high school, diploma, associated, and bachelor or above.

Determination of lead concentration levels in the respiratory zone of personnel

Based on the data obtained from the mean and standard deviation of previous studies and by dividing workers into similar exposure groups (SEGs), the number of samples needed for the present study was determined as 10.

According to the working hours of the study area, sampling was performed from 10 am to 4 pm. Furthermore, 10 blank samples were considered to determine the possible contamination levels at the time of sampling and transfer the samples into the laboratory. NIOSH 7300 method was used to measure lead concentration in the respiratory area of the staff.[22] Sampling was performed using cellulose ester membrane filter with 0.8μm pore size and by personal sampling pumps (SKC model) calibrated at 2 L/min using a soap bubble flow meter in the staffs' respiratory zone. The sampling lasted about 20 to 40 minutes. After sampling, filters were transferred to the laboratory and digested in a mixture of 65% nitric acid and 70% perchloric acid at a ratio of 4:1 during the sample preparation phase. Moreover, a flame atomic absorption spectrometer (Varian Spectra AA-220FS) was used to analyze the samples.

Determination participants' blood parameter

Subsequently, blood samples were collected by a relevant expert in the workplace. The CBC vials containing EDTA (anticoagulants) were used to prevent coagulation of the samples. Later, the samples were transferred to the laboratory quickly by following the standards and the cold chain for analysis. Blood cells were measured and counted in the laboratory by a cell counter. In addition, blood hemoglobin was measured using an optical absorption photometer.

Determine participants' blood pressure

A mercury barometer was applied to determine blood pressure. The systolic and diastolic blood pressure was measured two times for each participant in the sitting position and after a 10-minute rest; then, their mean was calculated.

Data analysis

Finally, the data were analyzed using SPSS V22. The normality of the data distribution was checked using the Kolmogorov – Smirnov test. One-way ANOVA, two-sample independent t-test and multiple linear regression were run for data analysis. Significance level of tests was considered at 0.05.

Results

The results showed that the mean age in the lead-exposed and non-exposed workers were 34.06 (8.8) and 37.04 (11.48) years, respectively. The 20-29 and 50 years and above age groups had the highest and lowest frequency with 38% and 9.1%, respectively. The mean work experience for the exposed and non-exposed workers was 3.3(3.5) and 3.7(3.8) years, respectively. In addition, the group with less than five years of work experience had the highest frequency with 86.6%. As it can be seen from Table 1, most participants (58.8%) had a diploma degree.

Table 1. Demographic characteristics of the study population (187 persons)

Parameters	Category	Frequency (%)	
		Exposed(%)	Non-exposed(%)
Age (years old)	20-29	63(38.4)	8(34.8)
	30-39	56(34.1)	5(21.7)
	40-49	31(19.5)	6(26.1)
	50≤	13(7.9)	4(17.4)
Work Experience (years)	≤5	143(87.2)	19(82.6)
	5-10	14(8.5)	1(4.3)
	11-15	5(3.0)	3(13.0)
	16-20	1(0.6)	0(0.0)
	20<	1(0.6)	0(0.0)
Education Level	High school	43(26.2)	1(4.3)
	Diploma	95(57.9)	15(65.2)
	Associate	6(3.7)	2(8.7)
	Bachelor's degree or above	20(12.2)	5(21.7)
Smoking	Yes	78(47.6)	8(34.8)
	No	86(52.4)	15(65.2)

The mean time-weight average (TWA) concentration of lead in the staffs' respiratory air was 0.0533 mg/m³ and above the 8-hour occupational exposure limit (50 μg/m³) (Table 2). Mean systolic blood pressure was 12.01 (1.3) mmHg in exposed workers and 11.78 (1.1) mmHg in non-exposed workers. Mean diastolic blood pressure for exposed and non-exposed workers was 7.84 (0.71) mmHg and 7.73 (0.54) mmHg, respectively (Table 3). The age group of above 50 years had the highest mean systolic blood pressure (13.1 (0.15) mmHg) in comparison to other age groups exposed to lead (Table 4).

The results of statistical tests revealed a significant correlation between the mean of systolic blood pressure in lead-exposed and non-exposed workers ($P= 0.040$). Furthermore, mean diastolic blood pressure was significantly different between the exposed and non-exposed groups ($P= 0.049$). A significant relationship was found between the systolic blood pressure and age ($P= 0.02$). The correlation between the other demographic variables and the recorded blood pressure values was not significant ($P> 0.05$). The simultaneous effects of age and respiratory exposure to lead on blood pressure were also examined using multiple linear regression. After adjusting for the confounding variables of age, work experience, education, and smoking, a significant relationship was observed between exposure to lead during a work shift and systolic blood pressure ($P<0.05$).

The results showed that the average hemoglobin level for exposure workers was 15.16(2.84) Gm/dl and 16.1(2.2) Gm/dl in non-exposed staff. Despite the difference, the results of statistical tests showed no significant difference between the level of hemoglobin in the exposed and non-exposed persons ($p= 0.079$). The mean hematocrit of the exposed and non-exposed workers was 47.4 (4.5) and 47.7 (7.6) respectively. The results of statistical tests showed no significant difference in this regard ($p= 0.147$) in spite of the small differences between the two groups (Table 5). A significant relationship was observed between work experience and hemoglobin and hematocrit values. However, no meaningful relationship was found between the participants' age and the level of hemoglobin and hematocrit ($p = 0.08$).

Table 2. Results of the lead concentration evaluation in the respiratory area of the participants (10 samples)

Sample number	Average concentration(mg/m³)	Standard Deviation	Assessment*
1	0.0511	0.019	More than OEL
2	0.0460	0.016	Lower than OEL
3	0.0320	0.026	Lower than OEL
4	0.0670	0.021	Lower than OEL
5	0.0900	0.029	More than OEL
6	0.0010	0.011	More than OEL
7	0.0530	0.013	More than OEL
8	0.0490	0.008	Lower than OEL
9	0.0630	0.023	More than OEL
10	0.0700	0.028	More than OEL
Average concentration	0.0533	0.021	More than OEL

*The occupational exposure limit is set at 0.05 mg/m³.

Table 3. Average systolic and diastolic blood pressure (BP) in study groups

Variable	Exposed			Non-exposed			P-Value*
	Average	Min	Max	Average	Min	Max	
Systolic BP (mm/Hg)	12.01 (1.30)	10	16	11.78 (1.10)	9	14	0.040
Diastolic BP (mm/Hg)	7.48 (0.71)	5	9	7.73 (0.54)	6	8	0.049

*Two-Independent Sample T-Test

Table 4. Mean and standard deviation of systolic and diastolic blood pressure of case and control workers based on different age groups

Blood pressure	Age categories (year)	Exposed	Non-exposed	P-Value*
Systolic BP (mm/Hg)	20-29	11.87 (1.40)	11.50 (1.06)	0.02
	30-39	11.89 (1.05)	11.20 (0.83)	
	40-49	12.09 (1.39)	12.33 (1.75)	
	50≤	13.00 (1.15)	12.25 (1.50)	
Diastolic BP (mm/Hg)	20-29	7.87 (0.65)	7.75 (0.46)	0.53
	30-39	7.75 (0.79)	8.00 (0.00)	
	40-49	7.9 (0.58)	7.83 (0.40)	
	50≤	7.9 (0.95)	7.25 (0.95)	

*One Way ANOVA

Table 5. Values of blood parameters based on the study groups

Variable	Exposed			Non-exposed			P-Value*
	Average	Min	Max	Average	Min	Max	
Hemoglobin (Gm/dl)	15.16 (2.84)	11.80	18.00	16.10 (2.20)	13.80	18.30	0.079
Hematocrit (Ratio)	47.40 (4.5)	13.90	54.90	47.70 (7.60)	15.50	56.20	0.147
Red Blood Cells RBC (Mil/cumm)	5.00 (4.52)	4.21	6.28	5.38 (4.61)	4.40	7.22	0.660
Mean Cell Hemoglobin MCH (Pg)	28.30 (3.02)	3.10	33.40	30.10 (2.46)	27.80	33.40	0.290
Mean Cell Hemoglobin Concentration MCHC (g/dl)	30.22 (2.38)	29.70	31.90	34.00 (6.87)	31.70	35.40	0.890

*Two-Independent Sample T- Test

Discussion

This study aimed to investigate the relationship of exposure to lead with workers' blood pressure and blood components in a lead mine in 2016. The mean concentration of lead in the respiratory zone of mine workers was 0.053 mg/m3 in winter, which is approximately 1.07 times higher than the permissible limit recommended by ACGIH. The mean systolic blood pressure was 12.01 (1.3) mmHg in exposed workers and 11.78 (1.1) mmHg in those with no exposure. The mean diastolic blood pressure in the exposed and non-exposed persons was 7.84 (0.71) mmHg and 7.73 (0.54) mmHg, respectively. Moreover, a meaningful relationship was observed between mean systolic and diastolic blood pressure in both exposed and non-exposed groups (p <0.05) (Table 3). In the present study, the mean systolic and diastolic blood pressure of the lead-exposed group was higher than the unexposed. Nowadays, most participants focused on the toxic effects of lead on the cardiovascular system, Therefore, it is required to study the lead effect on blood pressure and anemia.[3] Navas et al. (2007) revealed that respiratory exposure to lead increased the risk of hypertension in persons.[23] Zota et al. found that exposure to lead caused changes in blood pressure.[24] Landrigan et

al. reported that hypertension increased due to exposure to high concentrations of lead.[25] The results of a cohort study conducted by Gambelunghe et al. in 2016 showed that exposure to lead had a significant relationship with elevated systolic and diastolic blood pressure. Furthermore, continued exposure to lead and lead content eventually led to increased risk of hypertension.[26] Moreover, Boukerma et al. investigated 275 workers at an acid-lead battery factory and showed was a significant relationship between exposure to lead and elevated systolic and diastolic blood pressure17, which is consistent with the results of the present study.

In our study, systolic and diastolic blood pressure was higher in the 50-year-old group compared to other age groups. Results of statistical tests showed a significant difference between systolic blood pressure and age, while no significant difference was seen between diastolic blood pressure and age. Vupputuri et al. in 2003 showed that systolic blood pressure increased with age.[27] In the present study, a slight difference was observed in the levels of hematocrit, hemoglobin, mean hemoglobin concentration, and red blood cells between the lead-exposed workers and non-exposed workers, so that these parameters were higher in non-exposed individuals. While

statistical tests did not show any significant differences between the two groups (Table 5). The hematology system of the body is one of the most sensitive systems due to the interference of lead metal in the synthesis of hemoglobin.[28] A study by Nasiri et al., aimed at investigating the biological and environmental monitoring of lead and its effects in the car industry. They showed that the prevalence of anemia and bone metabolism disorder was higher in lead-exposed workers than the control group.[29] A study by Liu et al., (2014) on preschool children, found a significant relationship between blood lead levels and mean hemoglobin levels.[30] A study by Tutuarima et al. in 2018 also showed that lead exposure had a significant relationship with mean hemoglobin and hematocrit and lead exposure decreased hemoglobin, red blood cells, and hematocrit levels, [31] which is consistent with the results of the present study. Chwalba et al. in 2018 aimed at investigating the relationship between occupational exposure to lead and blood parameters. They found a significant relationship between chronic exposure to lead during one's employment and the number of red blood cells, hemoglobin, and hematocrit ,[32] which is similar to our results.

No significant relationship was found among education level, exposure to lead, blood pressure, and blood parameters. In this study, unlike other studies, no significant difference was found between the age of exposure group and the level of their blood hemoglobin and hematocrit. Increase of age and work experience in lead mines increased respiratory exposure to this metal, increased lead accumulation in the bones, and thus made changes in blood components more evident. Therefore, lack of correlation of blood components with variables of age and work experience can be attributed to the low work experience of lead exposed workers. Previous studies have shown that at least two years of work experience can lead to physiological symptoms of lead exposure.[32]

Conclusion

According to the results, exposure to lead metal can cause changes in blood pressure and some metabolic properties. We observed that employees' exposure to lead in the studied mine increased by non-compliance with the hygiene principles at work, increased work experience, and smoking in such environments. This changed the participants' systolic and diastolic blood pressure,

hematocrit, and hemoglobin and caused cardiovascular diseases. Therefore, taking control measures such as increasing staff break time, conducting periodic examinations, training courses, and other health care practices in such workplaces prevents changes in blood pressure and blood parameters.

Acknowledgment

References

1. Spierenburg TJ, De Graaf GJ, Baars AJ, Brus DHJ, Tielen MJM, Arts BJ. Cadmium, zinc, lead, and copper in livers and kidneys of cattle in the neighborhood of zinc refineries. Environmental Monitoring and assessment. 1988;11(2):107-14
2. Shariati S. Measurement of the lead, zinc, cadmium, and arsenic in Cattle's blood and hair Angouran's factories. Earth and resources. 2010;2(5):43-54.
3. Fiorim J, Júnior RFR, Silveira EA, Padilha AS, Vescovi MVA, de Jesus HC, et al. Low-level lead exposure increases systolic arterial pressure and endothelium-derived vasodilator factors in rat aortas. PLoS One. 2011;6(2):e17117.
4. Fadrowski JJ, Abraham AG, Navas-Acien A, Guallar E, Weaver VM, Furth SL. Blood lead level and measured glomerular filtration rate in children with chronic kidney disease. Environmental health perspectives. 2013;121(8):965-70.
5. Vaziri N, Gonick HC. Cardiovascular effects of lead exposure. 2008;426-35.
6. Lyn P. Lead toxicity, a review of the literature. Part I: exposure, evaluation, and treatment. Alternative medicine review. 2006;11(1):2-22.
7. Clougherty JE, Kubzansky LD. A framework for examining social stress and susceptibility to air pollution in respiratory health. Environmental health perspectives. 2009;117(9):1351-8.
8. Gee GC, Payne-Sturges DC. Environmental health disparities: a framework integrating psychosocial and environmental concepts. Environmental health perspectives. 2004;112(17):1645-53.
9. Campbell C, Gracely E, Tran M, Starkey N, Kersten H, Palermo P, et al. Primary prevention of lead exposure—blood lead results at age two years. International journal of environmental research and public health. 2012;9(4):1216-26.
10. Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, et al. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. Environmental health perspectives. 2005;113(7):894-9.
11. Wilhelm M, Heinzow B, Angerer J, Schulz C. Reassessment of critical lead effects by the German Human Biomonitoring Commission results in suspension of the human biomonitoring

values (HBM I and HBM II) for lead in the blood of children and adults. International journal of hygiene and environmental health. 2010;213(4):265-9.

12. Peters R, Burch L, Warner J, Beckett N, Poulter R, Bulpitt C. Haemoglobin, anemia, dementia and cognitive decline in the elderly, a systematic review. BMC geriatrics. 2008;8(1):18.

13. Muñoz PJ, Ottman R, Lee JH, Barton SA. Blood lead concentrations and associated factors in residents of Monterrey, Mexico. Archives of medical research. 1996;27(4):547-51.

14. Glenn BS, Bandeen-Roche K, Lee B-K, Weaver VM, Todd AC, Schwartz BS. Changes in systolic blood pressure associated with lead in blood and bone. Epidemiology. 2006;17(5):538-44.

15. Glenn BS, Stewart WF, Links JM, Todd AC, Schwartz BS. The longitudinal association of lead with blood pressure. Epidemiology. 2003;14(1):30-6.

16. Navas-Acien A, Schwartz BS, Rothenberg SJ, Hu H, Silbergeld EK, Guallar E. Bone lead levels, and blood pressure endpoints: a meta-analysis. Epidemiology. 2008;19(3):496-504.

17. Boukerma Z, Behlouli A-L, Terra A. Cumulative lead exposure, blood pressure, and pulse pressure: a longitudinal case-control study. Italian journal of occupational and environmental hygiene. 2018;9(3):146.

18. Ostchega Y, Yoon S, Hughes J, Louis T. Hypertension awareness, treatment, and control--continued disparities in adults: United States, 2005-2006. NCHS data brief. 2008(3):1-8.

19. Cheng Y, Schwartz J, Sparrow D, Aro A, Weiss ST, Hu H. Bone lead and blood lead levels in relation to baseline blood pressure and the prospective development of hypertension the normative aging study. American journal of epidemiology. 2001;153(2):164-71.

20. Inorganic and Organic Lead Compounds: International Agency for Research on Cancer; 2006.

21. Sandhu A, Soman S, Hudson M, Besarab A. Managing anemia in patients with chronic heart failure: what do we know? Vascular health and risk management. 2010;6:237-52.

22. Safety NIfO, Health. Elements by ICP: Method 7300. Pub. No. 98-119, US Department of Health and Human Services, NIOSH ...;

1994.

23. Navas-Acien A, Guallar E, Silbergeld EK, Rothenberg SJ. Lead exposure and cardiovascular disease: a systematic review. Environmental health perspectives. 2006;115(3):472-82.

24. Zota AR, Shenassa ED, Morello-Frosch R. Allostatic load amplifies the effect of blood lead levels on elevated blood pressure among middle-aged US adults: a cross-sectional study. Environmental health. 2013;12(1):64.

25. Landrigan PJ. Current issues in the epidemiology and toxicology of occupational exposure to lead. Environmental health perspectives. 1990;89:61-6.

26. Gambelunghe A, Sallsten G, Borné Y, Forsgard N, Hedblad B, Nilsson P, et al. Low-level exposure to lead, blood pressure, and hypertension in a population-based cohort. Environmental research. 2016;149:157-63.

27. Vupputuri S, He J, Muntner P, Bazzano LA, Whelton PK, Batuman V. Blood lead level is associated with elevated blood pressure in blacks. Hypertension. 2003;41(3):463-8.

28. He K, Wang S, Zhang J. Blood lead levels of children and its trend in China. Science of the total environment. 2009;407(13):3986-93.

29. Dehghan-Nasiri M, Golbabaii F, Koohpaii AR, Rahimi-Forooshani A, Shahtaheri SJ. Biological and environmental monitoring of lead and exposure in the automobile industry. Iran occupational health. 2012;8(4):1-8.

30. Liu C, Huo X, Lin P, Zhang Y, Li W, Xu X. Association between blood erythrocyte lead concentrations and hemoglobin levels in preschool children. Environmental science and pollution research. 2015;22(12):9233-40.

31. Tutuarima J. Lead Level Correlation (Pb) in Blood on Hb Level, Hematocrit, Cystatin C Serum, SGOT and SGPT Serum On Car Paint Workshop in Rungkut, Surabaya. Health notions. 2018;2(3):387-97.

32. Chwalba A, Maksym B, Dobrakowski M, Kasperczyk S, Pawlas N, Birkner E, et al. The effect of occupational chronic lead exposure on the complete blood count and the levels of selected hematopoietic cytokines. Toxicology and applied pharmacology. 2018;355:174-9.