

# The effect of blood lead level on packed cell volume in a sample of Iraqi population.

Hanan L. Sudek \*      PhD Physiology,  
Israa F. Jaffar\*      PhD Physiology

## Summary:

**Background:** Low hemoglobin levels and lead exposure remain significant health issues in many parts of the world, often occurring together, however most of the researches concentrate on the effect of high levels of lead. This study aimed to demonstrate the effect of blood lead level on hemoglobin and packed cell volume (PCV).

**Patients and Methods:** Fifty eight adult volunteers were enrolled in this study with age range between 23 and 55 years, working in the different departments in medical college of Baghdad University. Whole blood was withdrawn in EDTA tubes. Hemoglobin and PCV were measured and blood lead level was estimated using blood lead testing system including lead care kit and lead care analyzer.

**Results:** After measuring blood lead level and correlate it to PCV in all volunteers results revealed a significant negative correlation between them which is obvious in both smokers and non-smokers groups. Age was found to correlate positively and significantly with blood lead level in non smokers group. Comparing the blood levels of lead and PCV in both smokers and non-smokers shows non-significant difference between them although blood lead level was higher in smokers group.

**Conclusion:** Blood lead levels is increased with age. There is a significant decrease in PCV with the increase in blood lead level which is noticed in both smokers and non smokers. There is a significant increase of blood lead levels in smokers group.

**Key words:** blood lead, packed cell volume, age, smoking.

*Fac Med Baghdad*  
2014; Vol.56, No.1  
Received: Sept., 2013  
Accepted Jan., 2014

## Introduction:

Lead (Pb) is a non-essential heavy metal widely distributed in the environment(1).It is ubiquitous, persists indefinitely, and can be found at low levels in almost all living organisms (3); chronic exposure to low levels of this agent is one of health problems, due to its toxicity. Source of lead exposure may include air, water, food, and soil (1), including internal combustion engines, oil burners, smelters, lead pipes, glass and alloy processing plants, incinerators, industrial effluents, and smokestack fallout (4). Lead produces toxicological and pathological effects on central nervous system, peripheral nerves, kidneys and hematopoietic system (2).

Although lead has been shown to cause variety of disorders such as anemia, hypertension, kidney disorders, reduction in intelligence quotient (IQ), and various types of cancers ,but little is known about the role of quantitative levels of metal in physiological fluids, at which these effects occur (6).

In spite of that, a number of countries have recently phased out lead from gasoline. Lead exposure at the population level in some locations, particularly in developing countries, remains high. Mean blood lead levels are reported to be 13 µg/dL in China(7), and 11 µg/dL among urban children in India(8). In contrast, developed countries, such as the United States,

reported mean blood lead levels of 1.9 µg/dL among children(9). It has been found that even blood Pb concentrations below 10 µg/dL (0.48 µmol/L) may be associated with negative outcomes in children (10).

Lead can create reactive radicals which damage cell structure including DNA and cell membrane (11).

Several studies have demonstrated that clinical and sub clinical effects of lead toxicity considered as safe, i.e., below 30 µg/dl in adults and 10 µg/dl in children .Since major part of lead exists in the soluble form in erythrocytes therefore it is important to expect that its effects occur even at very low levels without showing any apparent clinical manifestations (5), but usually blood often shows pathological changes before the external signs of poisoning become apparent. In fact blood Pb concentrations reflect recent exposure to Pb, and Pb in bone reflects chronic exposure to this heavy metal (12).The objective of the study is to evaluate the effect of blood lead level on hemoglobin and packed cell volume in a sample of Iraqi people.

## Patients and methods:

This study included 58 adult volunteers, from which 24 subjects are smokers. Their age ranged from 23-56 years. Data were collected from volunteer staff working in different departments in the medical college of Baghdad University in the period between December 2012 and May 2013.

\*Physiology Department /College of  
Medicine/Baghdad University.

Blood samples (2 ml) were taken from volunteers, in tubes containing EDAT for hemoglobin concentration and for blood lead level determination later on. Packed cell volume (PCV) was measured using heparinized capillary tubes sealed at one end and centrifuged at 1500 RPM for 5 minutes in microcentrifuge. Then using hematocrit scale, PCV was determined.

Blood Lead level measurement was done using lead care analyzer, which includes the lead care kit and the lead analyzer device, version 3.3 (ESA, INC, USA).

Fifty micro liter of the whole blood in the EDTA tubes was drawn to be added to the treatment reagent in a special tubes supplied by the kit, cover them and mix thoroughly until the color of the mixture became brown. Then the tube was left to stand upright for a minute allowing the mixture to drain down to the bottom of the tube. Using the pipette a 35 micro liter of the mixture was drawn and applied to the sensor of the device after calibration and the process of analyzing was started (figure 1).



**Figure (1): The lead analyzer device.**

**Statistical analysis:** Continuous variables were presented as mean and standard deviation. Comparisons of blood lead levels and PCV in smokers and non smokers were done using student t -test, the differences were considered statistically significant when the P value was less than 0. 05. Correlation coefficient was used to demonstrate the relation between age and lead level, also between lead and PCV values (13).

#### **Results:**

Measuring blood lead level in this sample of Iraqi population revealed relatively high level than in other parts of the world (13.9±5.9). Since even blood lead concentrations well below 10µg/dL may cause adverse effects (14, 15) Correlation between blood lead level

and PCV in the study group reveals a significant negative correlation between them (table1).

**Table (1): Correlation between blood lead level and PCV in the studied groups.**

	Lead level	
	R	P
PCV	-0.371	<0.05

Comparing lead and PCV in the blood of 34 adult non-smokers and 24 adult smokers, shows that there is a significant difference between the two groups regarding the lead levels while there is no significant difference in PCV between both groups (table 2).

**Table (2): Comparison of blood lead level and PCV between smokers and non- smokers.**

	Non – Smokers	Smokers	P
Lead	11.65± 3.73	20.33± 6.32	<0.05
PCV	40.15±2.84	41.67± 1.27	>0.05

A correlation was done between blood lead level and the age of the non- smoker group which showed a significant positive correlation between them i.e. with increasing age the level of lead increases in the blood. The same significant positive correlation was also noticed in the smoker group as shown in table 3.

**Table (3): Correlation between blood lead level and age in non-smokers & smoker.**

	Lead level	
	R	P
Age non-smokers	0.858	<0.05
Age Smoker	0.576005	<0.05

Blood lead level was also correlated to PCV in non - smokers and it was obvious that the correlation was negative, in which there is a significant decrease in PCV whenever there is increase in the blood lead level. This significant negative correlation was also observed in the smoker group as shown in table 4.

**Table (4): Correlation between blood lead level and PCV in non- smokers & smokers.**

	Lead level	
	R	P
PCV non-smokers	-0.97478	<0.05
PCV smokers	-0.97986	<0.05

**Discussion:**

Lead-induced toxic manifestations in blood, kidney and brain (16). Lead was found to correlate negatively and significantly with PCV values i.e the increase in lead level causes a decrease in PCV in both smokers and nonsmokers. These results go along with other researches which document that the lower Pb concentrations or more appropriately trace amounts of lead in blood have greater potential to induce anemia. This correlation is explained by the fact that lead disturbs the pathway of heme synthesis at either ferrochelatase stage or inhibiting the aminolevulinatase activity (17). The pattern of variations in anemia indicators, i.e., PCV, erythrocyte count and hemoglobin corresponding to Pb levels below 44 µg/dl and those above this level clearly demonstrate the differences in the kinetics of process (18). Anemia is one of the most characteristics symptoms that indicate high and chronic exposure to lead, which is due to inhibition of number of enzymes that are involved in heme-synthesis, and as result heme synthesis is impaired and anemia is developed. Lead also affects iron carrier system through cell membrane of red blood cell (19). Studies on animals shows that lead causes fragility and break red blood cells. These facts were demonstrated by many studies performed on humans (20, 21)

Blood Lead level was found to be significantly higher in smokers than in non smoker group ( $20.33 \pm 1.30$  vs  $11.65 \pm 3.73$ ), which agrees with a study done by Ana N. et al., who states that smoking is an important source of exposure to lead (22,23). Even passive smoking is associated with increased blood lead levels (24). Actually The increase of blood lead level for non-occupationally exposed people indicates the presence of other source of lead that reaches the human body than occupationally exposed (25).

Packed cell volume is found to be slightly higher in smokers than in non smokers, although it was expected to be less in smokers because of their high blood lead levels. This could be explained by the fact that smokers have higher number of erythrocytes than nonsmokers which masks the lead effect on PCV unapparent (26, 27). Some studies revealed that the increase in blood lead level started in the bone before appearing in the blood as they found that patella bone lead levels are associated with decreased hematocrit and hemoglobin levels despite the presence of low blood lead levels. This conclusion may reflect a subclinical effect of bone lead stores on hematopoiesis and is the first epidemiological evidence that bone lead may be an important biological marker of ongoing chronic toxicity (23)

With increasing age lead is found to increase significantly both in smokers and nonsmokers, which agrees with the work of other researcher, who found that lead levels were higher in older subjects, in those with lower educational levels, and in smokers (28,29).

**Conclusion:**

Blood lead level is increased with age. There is a significant decrease in PCV with the increase in blood lead level which is noticed in both smokers and non smokers. There is a significant increase of blood lead levels in smokers group.

**Author's contribution:**

The subject and the aim of the study was designed by dr Israa F Jaffar. Collection of samples and processing of data was done by dr. Hanan luay.

**References:**

- 1- Jassim H. M and Hassan A. A (2011): *Changes in some blood parameters in lactating female rats and their pupsexposed to lead: effects of vitamins C and E. Iraqi Journal of Veterinary Sciences, Vol. 25, No. 1, (1-7).*
- 2-Ettinger AS, Tellez – Rojo MM, Amarasiriwardena C, Gonzalaz – Avila M (2004). : *Levels of lead breast milk and their relation to maternal blood and bone lead levels at one month postpartum. Environmental health perspectives; 112 (8):926 –931.*
- 3-Darrell W. Trampel, Paula M. Imerman, Thomas L. Carson, Julie A. Kinker, Steve M. Ensley(2003): *Lead contamination of chicken eggs and tissues from a small farm flock. J Vet Diagn Invest 15:418–422.*
- 4-Sharma RP, Street JC: 1980, *Public health aspects of toxic heavy metals in animal feeds. J Am Vet Med Assoc 177:149–153.*
- 5-Fahamida Memon, Vasandani AGM, Seehar GM, Bhangar MI (2009): *effect of low blood lead levels on anaemia indicators and creatinine clearance rate of workers occupationally exposed to lead. Pak J Physiol; 5(2),p 31-33.*
- 6-Tong S, Yasmin E, Schirnding V, Prapamontol T. *Environmental lead exposure: a public health problem of global dimensions. Bull World Health Org 2000; 78:1068–77.*
- 7-Ye X, Fu H, Guidotti T (2007): *Environmental exposure and children's health in China. Arch Environ Occup Health.; 62(2):61–73.*
- 8-Roy A, Bellinger D, Hu H, Schwartz J, Ettinger AS, Wright RO, et al (2009): *Lead exposure and behavior among young children in Chennai, India. Environ Health Perspect; 117:1607–1611.*
- 9-Jones RL, Homa DM, Meyer PA, Brody DJ, Caldwell KL, Pirkle JL, et al. (2009): *Trends in blood lead levels and blood lead testing among US children aged 1 to 5 years, 1988–2004. Pediatrics; 123(3):e376–e385.*
- 10-Patricia H C Rondó; Maria de Fátima H Carvalho; Miriam C Souza; Flávio Moraes (2006): *Lead, hemoglobin, zinc protoporphyrin and ferritin concentrations in children. Rev. Saúde Pública vol.40 no.1.p.12-15*
- 11- Flora Sj, Mittal M and Mehta A. (2008): *Heavy metal induced oxidative stress & its possible reversal*

- by chelation therapy. *The Indian Journal of Medical Research* 128 (4): 501–23.
- 12-Isha Barber, Ragini Sharma, Sheetal Mogra, Khushbu Panwar and Umesh Garu (2011): Lead induced alterations in blood cell counts and hemoglobin during gestation and lactation in Swiss albino mice. *Journal of Cell and Molecular Biology* 9(1):69-74.
- 13-Wayne W. Daniel (1987): *Biostatistics: a foundation for analysis in the health sciences*. John Wiley and Sons. p367-443.
- 14-Bellinger D.C. (2006) :Lead neurotoxicity in children: *Human developmental neurotoxicology*. Taylor & Francis, New York, p 67.
- 15-Lanphear B.R., Hornug R., Khoury J., Yolton K., Baghurst P. (2005): Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environ Health Perspect.*:113, 894.
16. Vidhu Pachauri, Mohit Dubey, Abhishek Yadav, Pramod Kushwaha, S.J.S. Flora: *Food and Chemical Toxicology*. Volume 50, Issue 12, December 2012, Pages 4449–4460. IVSL
17. S. G. Suradkar, D.J. Ghodasara, Priti Vihol, Jatin Patel, Vikas Jaiswal and K.S. Prajapati : acetate toxicity in Wistar Rats induced by lead. *Haemato-Biochemical Alterations . Veterinary World*, Vol.2 (11):429-431. IVSL.
- 18-Hawad, A. F. Eman, M. A. Amnia, B. F. Elssaidi, M. A.(): *Lead and Hemoglobin Level in Blood of Occupationally Exposed Workers at Brack Alshatti Area, Libya* Piomelli, S. (1981). *Chemical toxicity of red cells*. *Environ. Health. Prespect.*, 39, 65-70.
- 19-Guer, H., Ozgunes, H., Neal; R., Sptiz, D.R. and Ereal, N. (1998). *Antioxidant effect of N-acetylcystein and succimer in red blood cells from lead\_exposed rats toxicology*: 128, 59-69.
- 20-Montiro, H.P., Abdalla. D.S.P. , Arcuri, A.S. and Bechara, E.H. (1989). *Free radical generation during delta -amiolevulinic acid autoxidation induction by hemoglobin and connections with prophyriochem. Biophys.* 271, 206-216.
- 21- Doumouchtsis, S.A., Martin, N.S. and James, R.B. (2006). "Veterinary" diagnosis of lead poisoning in pregnancy. *BMJ*. 333:1302-1303.
- 22-Ana N., Elizabeth S., A. Richey S., Emma C., Ellen S., Eliseo G. (2004): *Lead, Cadmium, Smoking, and increased risk of peripheral arterial disease*. *Circulation*, 109: 3196-3201.
- 23-Apostolou, A, E Garcia-Esquinas, JJ Fadrowski, P McClaine, VM Weaver and A Navas-Acien. 2011. *Secondhand tobacco smoke: a source of lead exposure in U.S. children and adolescents*. *American Journal of Public Health* 5(3):.300-161.
- 24-Mannino DM, Homa DM, Matte T, Hernandez-Avila M ( 2005): *Active and passive smoking and blood lead levels in U.S. adults: data from the Third National Health and Nutrition Examination Survey*. *Nicotine Tob Res.* Aug; 7(4):557-64.
- 25-Howard H., ScD; Hirokastsu W.; Marinelle P.; Susan K.; Andrea R.: *The Relationship Between Bone Lead and Hemoglobin*. *JAMA*. 1994; 272(19):1512-1517.
- 26-Helman N and Rubenstein LS. ( 1975): *The effects of age, sex, and smoking on erythrocytes and leukocytes*. *Am J Clin Pathol. Jan*; 63(1):35-44.
- 27- Tirlapur V G, Gicheru K, Charalambous B M, Evans P J, and Mir M A (1983): *Packed cell volume, haemoglobin, and oxygen saturation changes in healthy smokers and non-smokers*. *Thorax.*; 38(10): 785–787.
- 28-Bellinger D.C. (2006) :Lead neurotoxicity in children: *Human developmental neurotoxicology*. Taylor & Francis, New York, p 67.
- 29-Lanphear B.R., Hornug R., Khoury J., Yolton K., Baghurst P. (2005): Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environ Health Perspect.*:113, 894.