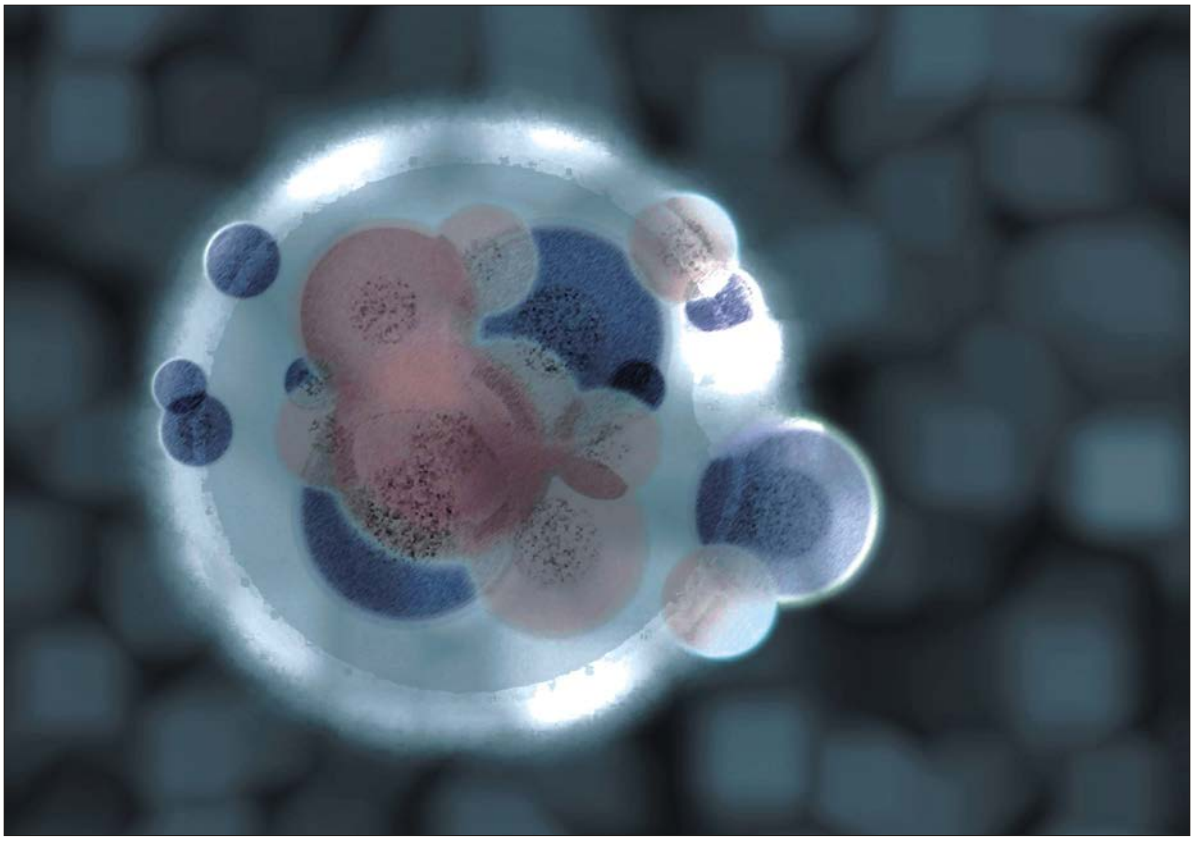

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The Relationship Between Blood Pressure and Lead Exposure in Battery Recycling Workers

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Abstract: The effect of lead on blood pressure is still controversial in spite of the numerous studies which have been conducted in the recent years. The aim of this study was to evaluate the effects of exposure to lead on blood pressure among workers in a battery recycling factory in Iran in 2008. In this cross sectional study, 165 male workers were enrolled. Their blood pressure, blood lead level and chelatable blood lead were measured for all subjects. Mean age of the participants was 39.41±7.23 years. The mean systolic and diastolic blood pressure was 121.57±7.23 and 81.84±8.73 mmHg, respectively. Blood lead level ranged from 15.6 to 85.6 μg dL^{-1} with a mean level of 44.04±16.05 μg dL^{-1}. There was no significant difference in blood pressure between subgroups with different blood lead levels. After adjusting for potential confounders (age, work duration, cholesterol, triglyceride, HDL, hemoglobin, creatinine, smoking and BMI) by multiple regression analysis, a borderline significant ($p = 0.057$) association was found between systolic blood pressure and blood lead concentration. There was no association between diastolic blood pressure and blood lead. Body mass index and smoking were positive predictors of blood pressure. Hemoglobin concentration and cholesterol were predictors of diastolic blood pressure. Systolic blood pressure showed an increase of 4 mmHg in patients with higher chelatable lead levels ($p = 0.05$). Present study showed that blood lead might not serve as a good predictor of blood pressure changes and there was no statistically significant association between blood lead and blood pressure. Present finding added to existing body of knowledge that blood lead possibly does not affect blood pressure.

Key words: Lead, poisoning, blood pressure, hypertension, chelatable lead

INTRODUCTION

Lead is a metal and despite its numerous industrial uses, it has no biologic role in the human body (Bernard and Becker, 1988). It affects multiple systems and causes a wide range of health insults. The effects of lead on the metabolic state and hematological, neurological and reproductive systems have been confirmed (Goyer, 1993). Because of the widespread use of lead, it is a major public health concern, especially in developing countries. Several studies have been performed in the recent years to determine cardiovascular effects, especially blood pressure changes, due to lead toxicity in both the general population and the occupational setting. There is experimental and epidemiological evidence that lead predisposes individuals to hypertension (Schwartz and Stewart, 2000; Fenga *et al*., 2006; Schwartz, 1988; Menditto *et al*., 1994). On the other hand, many studies show no or a weak relationship. Wu *et al*. (1996) reported lead did not affect blood pressure in workers who were exposed to lead. In a population-based study, only a weak association between systolic blood pressure and blood lead level was found in males after adjusting for potential confounders (Chu *et al*., 1999). According to a meta-analysis, there is a weak correlation between blood pressure and blood lead. A two fold increase in blood lead level is associated with a rise of one mmHg in systolic and 0.6 mmHg in diastolic blood pressure (Nawrot *et al*., 2002). Data shows that body lead burden is more relevant to blood pressure elevation. Lee *et al*. (2001) showed that blood pressure correlated with chelatable lead as an indicator of soft tissue bioavailable lead burden although there is controversy in this issue. With these considerations, the aim of this study was to assess the relationship between lead exposure and lood pressure in battery

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recycling workers with considering potential confounders associated to blood pressure and blood lead.

MATERIALS AND METHODS

We conducted this cross-sectional study at a battery recycling factory in Iran in 2008. Participation in this study was voluntary and an informed consent was taken from each participant before enrolment. Also, all subjects were informed of the fact that they could leave the study at any time. Ethical committee of Tehran Medical School approved this study. The eligible study population was male workers with at least six months of work experience. The exclusion criteria were history of hypertension before employment, diabetes, coronary heart disease, cerebrovascular disease, renal insufficiency, use of medications and any medical condition affecting blood pressure. Two hundred male workers were enrolled but 35 workers were subsequently excluded because of diabetes, coronary heart disease, thyroid disorders and use of medications. As a result 165 workers were recruited as participants. Socio-demographic data, disease history, cigarette smoking, occupational history and family history were obtained by a physician. Body weight and height were measured using a standard scale and a ruler attached to it; then, body mass index (BMI was calculated and expressed as (kg m^{-2}).

The participants did not use caffeine or cigarettes at least half hour before blood pressure measurement. After 20 min of rest in the sitting position, blood pressure was measured twice with a 10 min interval by a well trained physician. The average of these two readings was used for data analysis. A standard mercury Sphygmomanometer (Reister-NOVA model was used for measurement). The pressure at the first and fifth Korotkoff sounds was recorded as systolic and diastolic blood pressures, respectively. All measurements were taken between 7-8 am for reducing the diurnal variation effect.

Venous whole blood was sampled by a trained phlebotomist, collected in lead free heparinized vacutainer and stored at 4°C for 2 weeks for analysis. We used a flameless atomic absorption spectrophotometer for blood lead measurement. All samples were analyzed at a clinical laboratory supervised by Tehran Medical University three times and the mean reading was recorded. After 12 h fasting, serum levels of cholesterol, High Density Lipoprotein cholesterol (HDL, triglycerides, Hemoglobin) and creatinine were determined within 24 h of collection. Also, we used a statistical technique (regression equation) to estimate the chelatable lead as an indicator of current bioavailable lead. Predictors of chelatable lead in this model were age, smoking status, body mass index, creatinine clearance rate, blood lead and blood lead squares. In a study on 779 Korean lead workers, these variables predicted 81.6% of chelatable lead changes (R^2 = 81.6%; using this equation, we estimated the chelatable lead in 164 workers (Todd *et al*., 2001).

The results were expressed as mean and standard deviation. The study participants were divided into tree subgroups according to the blood lead level: <25 (mild intoxication), 25-50 (moderate intoxication) and >50 μg dL^{-1} (severe intoxication). Mean systolic and diastolic blood pressures were compared between groups using analysis of variance (ANOVA). We also used independent sample T test for evaluating mean differences of blood pressure in participants with estimated chelatable lead. Pearson's correlation coefficient was calculated to evaluate the relationship between study parameters. Backward multivariate regression was performed to calculate interrelationship of the explanatory factors with respect to systolic and diastolic blood pressures. The p-values less than or equal 0.05 were considered significant. All statistical analyses were performed using SPSS (version 14).

RESULTS

Our eligible participants were 165 male battery recycling workers. In our study population, the Mean±SD blood lead level was 44.04±16.05 μg dL^{-1} and mean systolic and diastolic blood pressures were 121.57±12.62 and 81.84±8.073 mmHg, respectively. The average of estimated chelatable lead was 628.61±153.28 μg. Sociodemographic and biological characteristics of the participants are shown in Table 1.

We compared mean differences of systolic and diastolic blood pressure in three subgroups with different blood lead levels but no significant differences were noted in sociodemographic and biological characteristics. The results showed no significant increase in blood pressure among workers with higher blood lead levels (Table 2).

Table 1: Characteristics of the workers (n: 165)

Item	Mean±SD
Age (year)	39.41±7.23
BMI (kg m^{-2})	26.32±3.56
Work duration (year)	14.63±7.00
Blood lead level (μg dL^{-1})	44.04±16.05
Systolic blood pressure (mmHg)	121.57±12.62
Diastolic blood pressure (mmHg)	81.84±8.073
Estimated chelatable lead (μg)	628.61±153.28
Creatinine (mg dL^{-1})	001.08±0.17
Hemoglobin (g dL^{-1})	014.71±.03
Cholesterol (mg dL^{-1})	191.8±0.99
HDL (mg dL^{-1})	157.23±90.18
Triglyceride (mg dL^{-1})	039.09±8.32
Smoking status	
Yes	55(33.5%)
No	109(66.5%)

To evaluate the association between blood pressure and soft tissue bioavailable lead, we categorized the estimated chelatable lead level into two subgroups; Table 3 shows mean differences of systolic and diastolic blood pressures in the two subgroups. The mean systolic blood pressure of the group with the higher chelatable lead level was 4 mmHg higher than the group with the lower level of chelatable lead ($p = 0.05$, but the mean difference of diastolic blood pressure in these two subgroups had a borderline significance ($p = 0.06$).

Table 4 shows Pearson correlation coefficients between age, BMI, blood lead level, work duration, cholesterol, triglyceride, creatinine, hemoglobin and blood pressure in our workers. BMI, cholesterol and triglyceride were positively correlated with both systolic and diastolic blood pressures ($p<0.05$). Age, work duration and hemoglobin concentration were significantly correlated only with diastolic blood pressure.

Backward multiple regression was performed to evaluate the association between the blood lead level and blood pressure after adjusting for potential confounders which could mask this association. Variables included in the model were age, work duration, cholesterol, triglyceride, HDL, hemoglobin, creatinine, smoking and BMI. Increased systolic blood pressure was significantly associated with BMI and smoking. A positive association with borderline significance ($p = 0.057$) was detected between the blood lead level and systolic blood pressure (Table 5). BMI, smoking, hemoglobin and cholesterol were significant predictors of diastolic blood pressure ($p<0.05$. No significant changes in diastolic blood pressure were seen in association with the blood lead level (Table 5).

Table 2: Blood pressure in different blood lead groups (n =165)

Blood lead	<25 μg dL^{-1}	25-50 μg dL^{-1}	>50 μg dL^{-1}
N	19	102	44
SBP (mmHg)	121.1±13.6	121.05±13.2	122.4±11.5
DBP (mmHg)	83.7±7.3	82.7±0.9	80.8±9.1

SBP: Systolic blood pressure, DBP: Diastolic blood pressure

Table 3: Blood pressure in the two estimated chelatable lead groups (n = 164)

Chelatable lead	≤625 μg	>625 μg
N	91	73
SBP (mmHg)	119.8±12.04	123.9±13.05
DBP (mmHg)	80.7±8.1	83.3±9.3

Table 4: Pearson's Correlations Coefficient and the level of significance for association of blood pressure with potential confounders

Blood pressure	Age	BMI	BLL	WD	Choles	TG	Cr	Hb	Ch lead
Systolic	0.126	0.299*	0.06	0.04	0.16*	0.152*	0.116	0.058	0.127
Diastolic	0.225*	0.322*	-0.06	0.162	0.315*	0.253*	0.130	0.207*	0.103

WD: Work duration, Chols: Cholesterol, TG: Triglyceride, Cr: Creatinine, Hb: Hemoglobin, Ch Lead: Chelatable lead, * $p<0.05$

Table 5: Multiple regression results for the interrelationship of potential confounders

Variable	Regression Coefficient (β)	Standard error of β	p-value
Systolic blood pressure			
BMI	1.001	0.267	<0.05
Smoking	6.584	1.965	<0.05
Blood lead	0.114	0.059	0.057
Cholesterol	4.211	0.023	0.073
Diastolic blood pressure			
BMI	0.614	0.179	<0.05
Smoking	3.240	1.309	<0.05
Hemoglobin	1.542	0.608	<0.05
Cholesterol	5.626	0.016	<0.05

DISCUSSION

We observed no significant differences in both systolic and diastolic blood pressure between groups with different blood lead concentrations. After adjusting for potential confounders, a borderline significant association was found between systolic blood pressure and blood lead concentration but no association was detected for diastolic blood pressure. In the present study, BMI and cigarette smoking were positive predictors of systolic and diastolic blood pressures but blood lead did not serve as a good predictor of blood pressure changes. These findings are in concordance with other studies which did not reach the level of significance or showed only a weak association between blood pressure and lead exposure (Tepper *et al*., 2001; Telisman *et al*., 2004; Ademuyiwa *et al*., 2005; Sharp *et al*., 1988). However, many studies, especially population-based surveys, have showed the association (Nash *et al*., 2003; Muntner *et al*., 2005; Rahman *et al*., 2006; Martin *et al*., 2006; Kaewboonchoo *et al*., 2007). The significant dose response relationship in population groups with lower lead exposure signifies that the most blood pressure rising occurs at relatively low blood lead levels and leveling out at higher blood lead concentrations (Goyer, 1993, Telisman *et al*., 2004). Animal studies support this hypothesis, too (Khalil-Manesh *et al*., 1993). Most of the participants in our study had high lead exposure and their mean blood lead level was more than advised by OSHA in the occupational setting which may possibly explain lack of correlation in our results. Also, race and genetic polymorphisms are factors that may change the effect of lead on the human body, leading to different outcomes in various population groups. Our knowledge regarding the role of genetic variations in developing lead-related high blood pressure is little. In particular, two polymorphic genes known to alter the effects of lead-vitamin D receptor (VDR and σ -aminolevulonic acid dehydratase (ALAD-could influence the effects of lead on blood pressure and cardiovascular outcome (Lee *et al*., 2001).

Also, a study that examined the relationship between blood pressure and lead exposure only found this correlation in black people but not in white men or women (Vupputuri *et al*., 2003). Another reason for detecting no correlation in our study may be because blood lead only signifies recent exposure and evidence suggests an increase in blood pressure with respect to bone lead concentration or chelatable lead excreted in the urine (Cheng *et al*., 2001). Similar to our findings, chelatable lead –a marker of current soft tissue bioavailable lead- has a significant association with systolic blood pressure among workers. Our findings were similar to other studies reporting that chelatable lead correlates with blood pressure changes (Lee *et al*., 2001; Batuman *et al*., 1983). The lead mobilization test was used in the past to assess the body lead burden and is now being replaced with *in vivo* X-ray fluorescence which is less invasive but it is still a practical method from the clinical point of view.

CONCLUSION

In conclusion, present findings indicated that blood lead concentration failed to be a good predictor of blood pressure changes as there was no significant association between blood lead and blood pressure. Present findings added to the existing body of knowledge that blood lead may not affect blood pressure obviously. However, we can not rule out the possible effects of lead, especially the total burden of lead, on the blood pressure.

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