

STUDIES ON BATTERY REPAIR AND RECYCLING WORKERS OCCUPATIONALLY EXPOSED TO LEAD IN KARACHI

Muhammad Jamal Haider, Naeemullah Qureshi

Federal Urdu University for Arts, Sciences & Technology, Gulshan-e-Iqbal campus, Karachi, Pakistan

ABSTRACT

Objective. The present study was carried out to investigate the effects of occupational lead exposure on the hematological and biochemical parameters in occupationally exposed and non exposed inhabitants of Karachi.

Material and methods. In 100 lead exposed subjects recruited from automobile workshops, lead battery repair and recycling units located in Karachi and in 100 control subjects the general health status, hematological parameters and exposure markers for lead were measured.

Results. Results indicated that the mean values of blood lead level and δ -aminolevulinic acid were significantly higher ($P<0.05$) while the activity of δ -aminolevulinic acid dehydratase were significantly decreased ($P<0.05$) among battery repair and recycling workers as compared to controls. The abnormalities in the blood lead level, δ -aminolevulinic acid and δ -aminolevulinic acid dehydratase were more frequent in lead exposed battery repair workers when compared with control subjects. The blood lead levels and δ -aminolevulinic acid were positively correlated while δ -aminolevulinic acid dehydratase was found to be negatively correlated with age, years of exposure and years of employment. Blood lead level was positively correlated with hemoglobin and RBC count while δ -aminolevulinic acid dehydratase was negatively correlated with hemoglobin concentration. The work related symptoms, droopiness, nasal symptoms and muscular pain were more frequent among battery repair workers as compared to control group. The findings of present study confirmed that occupational exposure to lead is associated with deviation in important hematological parameters and biological markers of exposure to lead among lead exposed workers, and also confirms the impact of lead exposure in the development of adverse effects among lead exposed workers.

Conclusions. The study provides the data for risk assessment in lead battery repair workers of Karachi and suggests the need for preventive measures for battery repair workers and improvements to reduce occupational lead exposures to protect them from lead toxicity. It is suggested that hematological and physical examinations of lead exposed workers should be carried out periodically to prevent future health hazards.

Keywords: *blood lead level, occupational exposure, hematological parameters, Karachi*

STRESZCZENIE

Cel. Badania przeprowadzono w celu określenia wpływu zawodowego narażenia na ołów na hematologiczne i biochemiczne parametry u zawodowo narażonych w porównaniu do nienarażonych mieszkańców Karachi.

Material i metody. U 100 osób narażonych na ołów spośród pracowników zatrudnionych w zakładach naprawy i przetwarzania akumulatorów w Karachi i u 100 osób kontrolnych badano ogólny stan zdrowia, parametry hematologiczne i markery narażenia na ołów.

Wyniki. Wyniki wskazują, że średnie poziomy ołowiu i kwasu δ -aminolewulinowego we krwi były znacząco wyższe ($P<0.05$), podczas gdy aktywność dehydratazy kwasu δ -aminolewulinowego była znacząco niższa ($P<0.05$) u zatrudnionych przy naprawie i przetwarzaniu akumulatorów w porównaniu do osób z grupy kontrolnej. Poziomy ołowiu we krwi i kwasu δ -aminolewulinowego były skorelowane pozytywnie podczas gdy w przypadku aktywności dehydratazy kwasu δ -aminolewulinowego stwierdzono negatywną korelację do wieku i mierzonego w latach czasu narażenia. Stężenie ołowiu we krwi było pozytywnie skorelowane z hemoglobina i liczbą erytrocytów a aktywność dehydratazy kwasu δ -aminolewulinowego była negatywnie skorelowana ze stężeniem hemoglobiny. Objawy związane z wykonywaną pracą, takie jak osłabienie, objawy ze strony nosa i bóle mięśniowe częściej występowały u osób narażonych niż z grupy kontrolnej. Wyniki przeprowadzonych badań potwierdziły, że zawodowe narażenie na ołów wiąże się ze zmianami istotnych parametrów hematologicznych i występowaniem markerów narażenia na ołów u narażonych pracowników, a także występowaniem szkodliwych skutków zdrowotnych.

Corresponding author: Naeemullah Qureshi, Hematological and Hematoparasitic Diseases Research Unit, Federal Urdu University for Arts, Sciences & Technology, Gulshan-e-Iqbal campus, Karachi, Pakistan, phone: +92-333-2286319, e-mail: profnaeem52@yahoo.com

Wnioski. Wyniki badań dostarczają danych do oceny ryzyka u pracowników naprawy i recyklingu akumulatorów i sugerują potrzebę zastosowania środków zaradczych w celu obniżenia zawodowego narażenia na ołów u tej grupy pracowników i ochrony przed toksycznym oddziaływaniem ołowiu. Sugeruje się, że badania parametrów hematologicznych i medycznych u pracowników narażonych na ołów powinny być wykonywane okresowo w celu zapobieżenia przyszłym zagrożeniom zdrowia.

Słowa kluczowe: poziom ołowiu we krwi, narażenie zawodowe, parametry hematologiczne, Karaczi

INTRODUCTION

Lead is a naturally occurring element found in earth's crust, which becomes very toxic when used into manmade products [10]. Once dispersed through spray paints, paint removers, gasoline exhaust and battery repairers, lead can never be completely removed from the environment posing a constant risk for human health [19]. After coming in contact with lead production processes, or environmental lead or through soil lead via drinking water and eating food can result in acute, sub-acute or chronic lead poisoning [7]. Blood lead level in the general population has decreased since the use of lead in paints and petroleum products and food containers have been curtailed. Nevertheless, other sources of lead continue to make lead an issue to public health [12]. The presence of large number of lead battery repair units in the Liaquatabad Saddar and Keamari towns of Karachi city gave rise to the issues related to lead poisoning among battery repair workers and general populations. The risk of occupational lead exposure exists where workers are exposed to lead and its compounds in the form of lead dust, lead fumes especially in lead smelting plants, battery repairs and recycling units. Lead is an important toxic metal with multistage toxic effects at very low levels of exposure on cardiovascular, nervous, urinary, gastrointestinal, reproductive and hemopoetic systems [1, 25]. Lead in human specimens is a bioindicator for lead body burden, actual and previous or recent lead exposure. The biological markers of lead include increased levels of coproporphyrin and aminolevulinic acid in urine; pirimidine-5-nucleotidase, protoporphyrin (FEP) and activity δ -aminolevulinic acid dehydratase (ALAD) in erythrocytes [17, 18]. The lowest observed adverse effect level of lead is 10 $\mu\text{g}/\text{dL}$ in children and 30 $\mu\text{g}/\text{dL}$ in adult [1]. Lead exposure inhibits δ -aminolevulinic acid dehydratase, ferrochelataze and coporphyrinogen oxidase in heme synthesis but its most significant effects are observed on ALAD [13]. As the threshold for erythrocytes ALAD inhibition lays below 10 $\mu\text{g}/\text{dL}$ the levels of δ -aminolevulinic acid in the blood and urine increased significantly. Similarly, the elevated levels of coproporphyrine in blood where the lead levels were 40 $\mu\text{g}/\text{dL}$ have been reported by Jaffe et al. [11].

The exposure to environmental lead is an issue of great importance in lead battery repair and recycling

industries and certain aspects of lead toxicity are yet to be elucidated. The aim of present study was to investigate the relationship between some biomarkers of lead effects and occupational lead exposure in battery repair and recycling workers, also to determine the role of work-related lead poisoning in the progression of lead related health hazards with particular emphasis on hemopoetic system in lead exposed workers.

MATERIAL AND METHODS

Present study was carried out from October 2008 to September 2010 to assess lead exposure and its toxic effects aiming to detect health impact caused by lead poisoning among general populations and lead battery repair workers at Liaquatabad, Sadda, and Keamari towns of Karachi. Data about demography, work history, hours of daily lead exposure, duration of employments, smoking status, prevailing disease, work related symptoms and preventive measures were obtained by using a structured questionnaire filled in by the subjects. The subjects with poor literacy were assisted to fill the questionnaire. An informed oral consent of each subject was obtained and study protocol was approved by the ethical review committee. The study group consisted of 100 lead exposed male workers of 30 battery repair and recycling units with an average of 32.4 ± 6.9 duration or years of exposure of exposure at current work place 11.4 ± 5.2 and duration of employment 14.6 ± 8.1 years. The control groups were the employees of other than lead battery repair industries but working in the vicinities of lead emitting industries. The control groups also consisted of 100 male subjects with an average age of 33.6 ± 4.3 and duration of employment of 15.3 ± 7.3 years. The demographic characteristics, smoking habits duration of employment and environmental lead exposure was not significantly different in the two groups studied. After taking a brief history, height and weight of each subject was recorded. Height was measured by using a fix stadia rod and an electronic scale was used to record the weight of each subject. Height and weight were used to calculate the body mass index (BMI) of each subject. General and work related symptoms of each subject were noted prior to the blood sampling. 2 ml blood of each subject was drawn from a large antecubital vein using a 20 G needle attached to a plastic syringe

and immediately transferred to a glass vial containing K₂EDTA 1.5 mg/ml of blood sample was stored at +4°C until tested for blood lead level on the same day and lead biomarkers. Blood lead level was determined using an atomic absorption spectrophotometer (Paar Physica Perkin Elmer 4100 HGA 700) with a sensitivity of 4.4×10^{-10} g for lead.

To determine the activity of δ -aminolevulinic dehydratase 0.3 ml heparinised blood was used. The hemolysed blood was added to aminolevulinic acid substrate and resulting porphobilinogen was determined spectrophotometrically at 555 nm after the addition of P-dimethyl amino benzaldehyde [5]. The urine samples from subjects were collected in clean plastic bottles. The concentration of ALA in urine was determined spectrophotometrically at 553 nm using the method of Grabecki *et al.* [9]. To determine coproporphyrin concentration in urine, the coproporphyrin was extracted from urine by ether and measured by spectrophotometer absorption measurement at 400 nm [6]. Reticulocyte and erythrocyte counts with basophilic stippling were determined by the method of Tasevski *et al.* [24] respectively while hemoglobin level, erythrocyte count and leukocyte count were determined using coulter model S-IV. Descriptive and inferential statistical methods were used to analyze the data using SPSS 12.0. The values of continuous variables were presented as mean \pm SD while nominal variables were expressed as percentages and numbers. The difference in frequencies was tested by Fisher's exact test and *Chi*² test and *P* < 0.05 were considered significant.

RESULTS

The demographic characteristics of study subjects are shown in Table 1. There was no significant difference in the smoking habits, years of employment duration of exposure in the two groups studied.

Table 1. Characteristics of the study subjects

Parameters	Controls (n=100)	Battery workers (n=100)
Age (years)	33.6 \pm 4.3	32.4 \pm 6.9
BMI (kg/m ²)	25.2 \pm 1.3	24.2 \pm 2.2
Years of employment	15.3 \pm 7.3	11.4 \pm 5.2
Years of exposure	not relevant	14.6 \pm 8.1
Current smokers	52 (52%)	48 (48%)
Years of smoking	Range: 0-40; Median: 9.3	Range: 0-50; Median: 9.3
Cigarettes/day	Range: 0-50; Median: 6.4	Range: 0-30; Median: 6.5
Ex-smokers	16 (16%)	12 (12%)
Passive smokers	18 (18%)	20 (20%)

Data expressed as mean \pm SD, frequencies expressed absolute numbers and percentages of subjects

Table 2 shows the history of non-work related symptoms during previous one year among the study subjects. Except for the higher prevalence of droopiness and muscle pain among exposed groups, there was no significant difference in the non-work related symptoms of lead workers when compared with controls. The occurrence of work related symptoms during last one year among lead battery repair and recycling workers and controls are depicted in Table 3. Among battery workers the work related symptoms in skin, lungs, eyes and nose were reported as 8%, 80%, 23% and 30% respectively. The work related symptoms in skin and eyes were not significantly different in the two groups studied. The nasal symptoms of battery repair workers showed increasing tendency as compared to controls which however was not statistically significant.

Table 2. Non work related symptoms among study subjects during the last one year

Symptoms	Controls (n=100)	Battery workers (n=100)	P values
Grumpiness	10	4	0.06
Gastrointestinal disorders	18	10	0.32
Droopiness	25	8	0.01
Weight loss	4	1	0.12
Muscular pain	24	10	0.04
Low appetite	10	5	0.21
Insomnia	9	6	0.54
Headache	18	14	0.61

Data expressed as numbers of subjects, tested by *Chi*² test.

Table 3. Work related symptoms among study subjects during the last one year

Symptoms	Controls (n=100)	Battery workers (n=100)	P values
Skin	6	8	0.07
Lungs	10	18	0.03
Nose	11	30	0.06
Eyes	8	23	0.43

Data expressed as numbers of subjects tested by *Chi*² test.

Table 4. Biological markers in the blood of occupationally exposed and non-exposed workers

Biomarkers	Controls (n=100)	Battery workers (n=100)	P values
BLL (μ g/dL)	10	4	0.06
RBC (10 ⁶ / μ L)	18	10	0.32
Hb (g/dL)	25	8	0.01
Reticulocytes %	4	1	0.12
ALAD (/ncat)	24	10	0.04
EBS count	10	5	0.21
ALA (μ mol/L)	9	6	0.54
Coproporphyrine (μ mol/L)	18	14	0.61

BLL= Blood lead levels; Hb= Hemoglobin; ALAD= δ -aminolevulinic acid dehydratase; ALA= δ -aminolevulinic acid; EBS= Erythrocytes with basophilic stipplings

The mean values of biological markers of lead exposure among study groups are depicted in Table 4. There was a significant increase in the blood lead level (BLL) and δ -aminolevulinic acid (ALA) and a significant decrease activity of δ -aminolevulinic acid dehydratase (ALAD) was observed in lead battery repair workers when compared with controls. Although equal or lower percentages for RBC count, reticulocytes, hemoglobin coproporphyrine and erythrocyte count with basophilic stipplings (EBS) higher rate of blood lead level, ALA and ALAD were observed in lead battery repair workers but the difference was statistically significant only for BLL and ALAD when compared with controls (Table 5).

Table 5. Prevalence of deviations from the mean levels of biological markers in the study subjects

Biomarkers	Controls (n=100)	Battery workers (n=100)	P values
BLL ($\mu\text{g/dL}$)	15	68	0.001
RBC ($10^6/\mu\text{L}$)	3	4	0.128
Hb (g/dL)	1	1	0.217
Reticulocytes %	12	12	0.125
ALAD (/ ncat)	15	45	0.001
EBS count	8	9	0.652
ALA ($\mu\text{mol/L}$)	2	7	0.213
Coproporphyrine ($\mu\text{mol/L}$)	1	2	0.559

BLL=Blood lead levels; Hb= Hemoglobin; ALAD= δ -aminolevulinic acid dehydratase; ALA= δ -aminolevulinic acid; EBS= Erythrocytes with basophilic stipplings

Table 6. Correlation coefficients for the relationships between BLL, ALA and ALAD in study subjects

Biomarkers	BLL	ALAD	ALA
Blood lead level	1	-0.465**	-0.005
δ -aminolevulinic acid dehydratase	-0.465**	1	-0.064
δ -aminolevulinic acid	-0.005	-0.065	1
Age	0.293**	-0.194*	-0.035
Duration of employment	0.425**	-0.245*	0.031
Duration of exposure	0.345**	-0.291*	0.125
BMI	-0.067	-0.074	0.041
Blood pressure	0.134	-0.241*	-0.036
Smoking habit	-0.003	-0.065	0.043
Duration of smoking	0.085	-0.258	-0.210
Daily cigarettes smoked	0.035	-0.062	0.031
RBC count	0.334**	-0.234	-0.125
Hemoglobin	0.321**	-0.355*	0.062
Hematocrit	0.123	0.310	-0.356
WBC count	0.021	-0.125	-0.124

Level of significance: *P<0.05; **P<0.01

Table 6 shows the results of correlation analysis between the studied parameters and blood lead level, ALA and ALAD activity in battery repair workers and controls. Blood lead level and ALAD showed a significant inverse correlation ($r = -0.621$, $P < 0.01$) while blood lead level was positively correlated with age (0.298, $P < 0.01$) years of exposure ($r = 0.328$, $P <$

0.01) and years of employment ($r = 0.39$, $P < 0.01$). The activity of ALAD was found to be inversely correlated with age ($r = -0.194$, $P < 0.05$), years of exposure ($r = -0.245$, $P < 0.05$) and years of employment ($r = -0.291$, $P < 0.05$) and systolic blood pressure ($r = -0.241$, $P < 0.05$), while BMI and smoking characters were not significantly correlated either with blood lead level or the activity of ALAD. Blood lead level was positively correlated with RBC count ($r = 0.334$, $P < 0.01$) and hemoglobin ($r = 0.321$, $P < 0.01$), while the activity of ALAD was inversely correlated with hemoglobin ($r = -0.298$, $P < 0.05$). On the other hand no significant correlation was found between blood lead level and activity of ALAD with hematocrit and WBC count or between blood lead level and systolic blood pressure.

DISCUSSION

In the present study the blood lead level in battery repair and recycling workers (42 $\mu\text{g/dL}$) was found to be significantly higher ($P < 0.001$) when compared with controls (13 $\mu\text{g/dL}$). Since control subjects were selected from general population of Karachi city, it therefore seems that the BLL in the general population of Karachi are higher than other cities such as 2.5 $\mu\text{g/dL}$ in general population of Sweden [3], 3.2 $\mu\text{g/dL}$ in Thailand [22]; 3.6 $\mu\text{g/dL}$ in Germany [4]. The elevated blood lead levels among general population of Karachi may be attributed to the frequent use of illegal leaded petrol in the public transport. *Kelada et al.* [14] reported that the blood lead level and activity of ALAD also depends on the genotype variants of the subjects. Out of eight δ -aminolevulinic acid dehydratase gene variants ALAD 1 and 2 are codominant and significantly higher blood level among people having ALAD 1- and ALAD 2 genotypes as compared to people having ALAD 1-1 genotypes have been reported previously by *Wetmur et al* [26]. Therefore it is hypothesize that ALAD 1-2 and ALAD 2-2 are at risk genotype at higher levels of lead exposure.

In the present study the lead battery repair workers had significantly decreased ALAD activity which was also significantly correlated with blood lead levels. *Telisman et al.* [23] have reported the activity of ALAD is highly sensitive to blood lead level and it is comparatively highly specific to blood lead level than the other biological markers of lead exposure. The results of present study showed no correlation between smoking characteristics and the activity of ALAD while a strong positive correlation between years of exposure and years of employment with the blood lead level was observed. On the other hand a negative correlation between these parameters and the activity of ALAD was also observed. In consistence with other studies our data showed the

effects of lead exposure on the prevalence of respiratory tract symptoms [21].

In agreement with the previous reports, the results of this study showed the prevalence of microcytic type anemia accompanied by reticulocytosis among lead exposed subjects [8]. In consistence with the results of Kelada et al. [14] present study also showed an inverse correlation between the activity of ALAD and hemoglobin and a strong positive correlation between hemoglobin and blood lead levels. Although Kuo et al. [15] studies have reported a decreased immune response in lead exposed workers but duration of exposure was not found to be correlated with WBC count and hematocrit in the present study. In the present study age and blood lead level showed a positive correlation, age and the activity of ALAD showed an inverse correlation, while body mass index showed no correlation with the markers of lead exposure, which is in consistence with the previous findings Apostoli et al. [2]. Present data showed that the activity of ALAD was inversely correlated with systolic blood pressure while systolic blood pressure was not correlated with blood lead levels. In contrast, Pocock et al. [20] reported a correlation of increased diastolic blood pressure with BLL in lead exposed workers.

CONCLUSIONS

In conclusion, present study provides the data for risk assessment in lead battery repair workers of Karachi. It was also examined and confirmed the association between specific biomarkers of lead effect and occupational lead exposure. This study also suggests the need for preventive measures in battery repair workers and improvements to reduce occupational lead exposures to protect them from lead toxicity.

REFERENCES

1. Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for Lead. Draft for public comment. US Department of Health and Human Services. Atlanta, US. 2005.
2. Apostoli P., Baj A., Bavazzano P., Ganzi A., Neri G., Ronchi A., Soleo L., Di L.L., Spinelli P., Valente T., Minoia C.: Blood lead reference values: the results of an Italian polycentric study. *Sci Total Environ.* 2002, 287:1-11.
3. Bárány E., Bergdahl I.A., Bratteby L.E., Lundh T., Samuelson G., Schütz A., Skerfving S., Oskarsson A.: Trace elements in blood and serum of Swedish adolescents: relation to gender, age, residential area, and socioeconomic status. *Environ Res.* 2002, 89, 72-84.
4. Becker K., Kaus S., Krause C., Lepom P., Schulz C., Seiwert M., Seifert B.: German Environmental Survey 1998 (GerEs III): environmental pollutants in blood of the German population. *Int J Hyg Environ Health* 2002, 205, 297-308.
5. Bonsignore D., Calissano P., Cartasangna C.: A simple method to determine blood δ -aminolevulinic acid dehydratase, in Italian. *Med Lav.* 1965, 56, 199.
6. Cvetanov V., Stikova E., Karad`inska-Bislimovska J.: Health condition and work ability. Skopje: Institute of Occupational Health 1989, 5, 25-30.
7. Eisinger J.: Sweet poison. Episodic outbreaks of colic, or 'wine disease,' plagued Europe for many centuries, even after an obscure German physician traced the cause to lead. *Nat Hist.* 1996, 105, 48-53.
8. Goyer R.A., Clarkson T.W.: Toxic effects of metals. In: Casarett & Doull's Toxicology. New York: McGraw-Hill, 6th edition 2001, 811-867.
9. Grabecki J., Haduch T., Urbanowicz H.: Die einfachen Bestimmungsmethoden der δ -Aminolavulinsäure im Harn. [Simple regulation methods of the aminolevulinic acid in the urine, in German], *Int Arch Gewerbepathol Gewerbehyg.* 1967, 23, 226
10. Hernandez-Avila, M., Cortez-Lugo, M., Munoz, I., Tellez, M.M., Rojo, S.: Lead exposure in developing countries. Studies and findings 1999 [displayed 10 November 2011]. Available at <http://www.leadpoison.net/general.htm>.
11. Jaffe E.K., Bagla S., Michini P.A.: Reevaluation of a sensitive indicator of early lead exposure: measurement of porphobilinogen synthase in blood. *Biol Trace Elem Res.* 1991, 28, 223-231.
12. Kocubovski M.: Influence of lead from ambient air and evaluation of health condition in school children, in Macedonian. [PhD Thesis]. Skopje: Medical faculty, University Sts. Cyril and Methodius 2004.
13. Kappas A., Sassa S., Galbraith, R.A.: The porphyrias. In: Scriver CR, Beaudet AL, Sly WS, editors. The metabolic and molecular basis of inherited disease. 7th ed. New York (NY): McGraw-Hill Book Company 1995, 2103-2159.
14. Kelada N.S., Shelton E., Kaufmann R.B., Khoury J.M.: δ -Aminolevulinic acid dehydratase genotype and lead toxicity: A HuGE review. *Am J Epidemiol.* 2001, 154, 1-13.
15. Kuo H.W., Hsiao T.Y., Lai J.S.: Immunological effects of long-term lead exposure among Taiwanese workers. *Arch Toxicol.* 2001, 75, 569-573.
16. Kelada N.S., Shelton E., Kaufmann R.B., Khoury J.M.: δ -Aminolevulinic acid dehydratase genotype and lead toxicity: a HuGE review. *Am J Epidemiol.* 2001, 154, 1-13.
17. Mahaffey K., McKinney J., Reigart J.R.: Lead and compounds. In: Lippmann M, editor. Environmental toxicants, human exposures and their health effects. 2nd ed. New York (NY): John Wiley and Sons, Inc. 2000, 481-521.
18. McElvaine M.D., Orbach H.G., Binder S., Blanksma L.A., Maes E.F., Krieg R.M.: Evaluation of the erythrocyte protoporphyrin test as a screen for elevated blood lead levels. *J Pediatr.* 1991, 119, 548-550.
19. Needleman L.H.: Clamped in a straitjacket: The insertion of lead into gasoline. *Environ Res.* 1997, 74, 95-103.

20. Pocock S.J., Shaper A.G., Ashby D.T., Whitehead T.P.: Blood lead concentration, blood pressure, and renal function. *Br Med J.* 1984, 289, 872-874.
21. Rastogi S.K., Gupta B.N., Husain T., Mathur N., Srivastava S.: Spirometric abnormalities among welders. *Environ Res.* 1991, 56, 15-24.
22. Sirivarasi, J., Kaorjaren, S., Wananukul, W., Srisomerg, P.: Non-occupational determinants of cadmium and lead in blood and urine among a general population in Thailand. *Southeast Asian J Trop Med Public Health* 2002, 33, 180-187.
23. Telisman S., PrpiMaji D., Ke S.: In vivo study on lead and alcohol interaction and the inhibition of erythrocyte delta-aminolevulinic acid dehydratase in man. *Scand J Work Environ Health* 1984, 10, 239-244.
24. Tasevski S., Velkov S., Kostoski D.: Determination of basophil punctuated erythrocytes count as a diagnostic method for occupational saturnism. *Laboratory* 2002, 4, 13-16.
25. Vidakovic A.: Lead. In: Vidakovi A., editor. *Occupational toxicology*. Belgrade. Society of Toxicologists of Yugoslavia 2000, 316-29.
26. Wetmur J.G.: Influence of the common human δ -aminolevulinate dehydratase polymorphism on lead body burden. *Environ Health Perspect* 1994, 102, Suppl 3, 215-219.

Received: 13 August 2012

Accepted: 16 December 2012