

## Assessment of Lead Exposure Risk in Locksmiths

Vladislav Kondrashov<sup>1</sup>, Joseph L. McQuirter<sup>1</sup>, Melba Miller<sup>1</sup>, Stephen J. Rothenberg<sup>1,2\*</sup>

<sup>1</sup>Departments of Oral and Maxillofacial Surgery (J.L.M.), and Anaesthesiology (S.J.R. V.K.), Clinical Research Center (MM), Charles R. Drew University of Medicine and Sciences, Los Angeles, California, USA

<sup>2</sup>The Center for Research in Population Health, National Institute of Public Health, Cuernavaca, Morelos, Mexico

\*Correspondence to Dr. Stephen Rothenberg, Email: drlead@prodigy.net.mx

Received: 15 November 2004 / Accepted: 06 February 2005 / Published: 30 April 2005

**Abstract:** Exposure to lead has been well recognized in a number of work environments, but little is known about lead exposure associated with machining brass keys containing lead. The brass that is widely used for key manufacturing usually contains 1.5% - 2.5 % of lead. Six (6) licensed locksmiths and 6 case-matched controls successfully completed the pilot study to assess the prevalence of increased body lead burden of professional locksmiths. We measured both Blood Lead (atomic absorption spectrometry), bone-lead (KXRF) and had each subject complete a health and lead exposure risk questionnaire. One locksmith had not cut keys during the past two years, therefore this subject and case-matched control was excluded from the blood lead analysis only. The average blood-lead concentration ( $\pm$ SEM) for the 5 paired subjects was 3.1 ( $\pm$  0.4)  $\mu$ g /dL and 2.2 ( $\pm$  0.3)  $\mu$ g /dL for controls. Bone measurements, including all 6 paired subjects, showed tibia lead concentration ( $\pm$ SEM) for locksmiths and controls was 27.8 ( $\pm$  2.3)  $\mu$ g /g and 13.7 ( $\pm$  3.3)  $\mu$ g /g, respectively; average calcaneus lead concentration for locksmiths and controls was 31.9 ( $\pm$  3.7)  $\mu$ g /g and 22.6 ( $\pm$  4.1)  $\mu$ g /g, respectively: The t-test shows a significantly higher tibia lead ( $p < 0.05$ ) and blood lead ( $p < 0.05$ ) for locksmiths than for their matched controls, but no significant difference for calcaneus lead ( $p > 0.10$ ). Given that the mean tibia bone lead concentration was 13.1  $\mu$ g/g higher in locksmiths than in their matched controls, this average difference in the two groups would translate to an OR of increased hypertension in locksmiths of between 1.1 and 2.3, based on the published literature. Even with the very small number of subjects participating in this pilot study, we were able to demonstrate that locksmiths had significantly higher current exposure to lead (blood lead concentration) and significantly higher past exposure to lead (tibia lead concentration) than their age, sex and ethnically matched controls. Additional research is needed to fully identify the prevalence and associated risk factors for occupational exposure of lead in this previously understudied profession.

**Key Words:** lead, elevated blood lead, lead poisoning, occupational health, brass keys

### Introduction

Adverse health effects of lead exposure in the workplace are well-known for both workers and their families [1, 2]. More than 140 at-risk occupations for lead exposure have been identified [3] and the National Institute of Occupational Safety and Health (NIOSH) estimates that more than 3 million workers in the United States are potentially exposed to lead in the workplace [4]. Among these are workers of brass foundry and brass product manufacturing industries. A major route of exposure to lead and other toxic substances in these workers is inhalation of fumes and dust particles [5]. Workers exposed to lead fumes and dust particles may inadvertently contaminate their homes and expose family

members with lead dust particles transported on their clothes, skin, hair, tools and in their vehicles [6, 7].

Brass that is widely used for key manufacturing usually contains 1.5% - 2.5 % of lead [8]. Even the brass alloy manufactured with especially reduced lead content (so-called “reduced lead brass”) of American Society for Testing and Materials (ASTM) specification (B-121) has a nominal 2% lead. Another brass product, the so-called “free cutting brass”, (ASTM B-16/B-219) has a nominal 3.25% lead [9, 10].

Concerns about this exposure prompted the Attorney General for the State of California to sue 13 key manufacturers and distributors for allegedly failing to warn that their products expose consumers to the toxic chemical lead in violation of Proposition 65 [11].

Proposition 65 – otherwise known as the California Safe Drinking Water and Toxic Enforcement Act of 1986 – was passed by the voters to protect the public from exposure to toxic substances known to cause cancer or be harmful to reproductive health. State conducted studies found the average exposure level for the lead residue on the hands of consumers handling brass keys was 19 times the Proposition 65 “No Significant Risk Level” of 0.5 µg per day limit [11].

Although the lawsuit focused on protecting the end user of this product, the consumers, nothing was said about the locksmiths who actually spend more time than anybody else handling, grinding, and polishing lead-containing keys. Some keys are coated with a thin layer of metal such as nickel or chromium to minimize exposure to the underlying lead toxin, but this provides no protection for the locksmiths who must cut through the coating to the underlying metal. As a result of the grinding and polishing, lead-containing dust is dispersed throughout the workplace environment. The fine fraction of airborne dust may be inhaled directly, or during reentrainment after settling. Lead deposited on worksite surfaces may be ingested as a result of hand to mouth activity or contamination of open food or beverage containers. Lead deposited on work clothes may be translocated to the home environment, resulting in secondary, or “take-home” exposure to family members, particularly young children [6, 7, 12, 13].

The facts on which this lawsuit was based provide critical insight into the potential occupational hazard for exposure to lead for locksmiths. Because they handle and grind keys on a daily basis, they sustain thousands of exposures during their entire active careers. The chronic low level exposure to lead spanning a 10 to 40 year career may produce significant cumulative total body burden of this well-known toxin. This is the first study to show a risk for lead exposure from yet another occupational source not previously examined.

## Materials and Methods

### Subjects

We recruited six locksmiths and six case-matched controls from volunteers residing in Los Angeles, California. Locksmiths selected for the study were at least 18 years of age, worked in his/her profession for at least one year and possess a valid California license as a locksmith or recent retiree. The six volunteer control subjects were matched to each locksmith by age, sex and ethnicity.

Excluded from the study were unlicensed individuals who duplicate keys, individuals with known histories of exposure to lead from other sources (i.e. painters, battery workers, metal foundry workers, firearm instructors, subjects with retained bullets in their bodies, etc.), pregnant women in the first two trimesters, children less than 18 years of age and those unable to give informed consent. Subjects with paraplegia, quadriplegia, movement disorder or individuals without at least one natural leg free of metal plates, screws or other objects were excluded because such conditions would interfere with bone lead determinations. All data collection was

completed in a single visit on or after the date the subject consented to participate.

The Institutional Review Board of the KDMC approved the protocol and a written informed consent was given by each study participant.

### Blood Lead Measurements

Blood lead was measured by atomic absorption spectrometry, using Zeeman background correction and graphite furnace. All blood lead measurements were performed by Westcliff Medical Laboratories, Inc. of Garden Grove, California. Westcliff fulfills the requirements for CLIA 1988 laboratory testing and participates in proficiency testing with the American Association of Bioanalysts and the College of American Pathologists. Abnormal test results are confirmed by repeating the test in duplicate, to ensure its reproducibility and accuracy

### Bone Lead Measurements

In vivo bone lead concentration (bone lead) was measured in study subjects by a K-shell X-ray fluorescence (KXRF) measurement system [14]. Bone lead measurements were made at the mediolateral and proximodistal midpoint of the anterior right tibia diaphysis and at the lateral surface of the right calcaneus. Because of measurement error, estimates of bone lead are sometimes negative, especially when true bone lead approaches zero. Bone lead slowly accumulates during the life of a person [15], with residence times for calcaneus and tibia bone lead estimated at 11 to 29 years (95 percent CI) and 16 to 98 years (95 percent CI), respectively [16]. Measurement of bone lead provides an estimate of cumulative past exposure of the subject. Estimates of bone lead were calculated from KXRF spectra using the recently corrected formulation [17, 18].

### Health and Risk Questionnaires

During the bone lead measurements, a trained project staff member administered a structured questionnaire to capture demographic and medical information and to determine risk factors (occupational, avocational, life style, and habits) for lead exposure [19].

## Results

The data were obtained from 6 professional locksmiths and 6 control volunteers matched to each locksmith by age, sex and ethnicity. The data are shown in the Table 1. Bone lead concentrations (tibia lead and calcaneus lead) are given in micrograms per gram in bone minerals, blood lead is given in micrograms per deciliter.

The t-test for all 6 subject pairs showed significantly higher tibia ( $p < 0.05$ ,  $t = 3.62$ ,  $df(\text{degrees of freedom}) = 10$ ) and no difference for calcaneus ( $p > 0.10$ ,  $t = 1.78$ ,  $df = 10$ ). We excluded from the blood t-test subject number 4 and his case matched-control because the locksmith reported that he had not practiced his trade during within the past two years. The results of his blood lead did not reflect

**Table 1:** Bone and Blood Lead Measurement for Locksmiths and Case Matched Controls Charles R. Drew University of Medicine and Sciences, Los Angeles, California, December 2003 to January 2004.

Study Number	Bone Lead Measurements				Blood Lead( $\mu\text{g/dL}$ )	Age(years)	Sex	Ethnicity	Years in Profession (years)
	Tibia ( $\mu\text{g/g}$ )	Error( $\pm$ )	Calcaneus( $\mu\text{g/g}$ )	Error( $\pm$ )					
<i>Locksmiths</i>									
1	18.7	9.9	22.2	10.3	3.9	31	M	W	18
2	29.3	11.6	30.7	14.6	3.9	67	M	AA	30
3	23.5	8.2	45.7	9.7	3.9	59	M	AA	28
4*	35	9.2	26	11.3	1.3	56	M	H	38
5	31.1	6.9	26.1	10.3	3.1	60	M	AA	23
6	26	10	40.4	13.6	2.5	45	M	AA	20
Average	27.8	( $\pm 2.3$ )	31.9	( $\pm 3.7$ )	3.1	( $\pm 0.4$ )			26
<i>Controls</i>									
1	7.6	6.9	12.5	8.6	3.2	37	M	W	
2	24.4	7.6	36.2	11.6	1.5	61	M	AA	
3	20.3	7.3	30	8.6	2.8	59	M	AA	
4*	12.8	6.3	18.3	10.1	1.6	51	M	H	
5	1.6	8.6	28	9	2	66	M	AA	
6	15.3	7.3	10.6	8.3	1.9	44	M	AA	
Average	13.7	( $\pm 3.3$ )	22.6	( $\pm 4.1$ )	2.2	( $\pm 0.3$ )			

\* Data from subject #4 and case-matched control were not included in blood lead statistical analysis – locksmith had not practiced his trade for over two years. All 6 subject pairs were included in bone lead data analysis.

W = White; AA = African American; H = Hispanic

recent exposure to lead, a finding that corresponds with his work history. The t-test for the remaining 5 subject pairs showed significantly higher blood lead ( $p < 0.05$ ,  $t = 3.14$ ,  $df = 8$ ) for locksmiths than for their matched controls.

Even with the very small number of subjects in this pilot study, we were able to demonstrate that locksmiths had significantly higher current exposure to lead (blood lead concentration) and significantly higher past exposure to lead (tibia lead concentration) than their age, sex and ethnically matched controls.

## Discussion

Following the establishment of general industry lead standards in 1978, monitored data on permissible exposure limits have shown significant decreases in lead exposure for these facilities. Such decreases have not occurred in the construction industry since the enactment of construction industry standards in 1993 [4, 20]. Because permissible limits are based on monitored blood and air specimen standards that were established a quarter century ago (see Table 2), they have not taken advantage of current data that shows adverse health consequences also occurs at lower exposure limits [21-23].

Until the State of California lawsuit, lead exposure from handling brass keys and in locksmiths cutting them has been essentially overlooked. This is not surprising since most of the exposure may be “low level”, resulting in blood lead concentrations that generally do not produce overt signs or symptoms during the active course of employment. There is only one case study in the English literature where the potential for lead exposure from grinding keys has even been evaluated.

Ferguson reported that industrial hygiene monitored air samples were well within permissible Occupational Safety and Health Administration (OSHA) limits for three hardware store employees who cut an average of 5 keys per day [24].

In cases where overt lead exposure does exist, it is often unrecognized or misdiagnosed due the nonspecific nature of lead toxicity, particularly in settings where lead exposure is not traditionally expected. The first report of serious lead poisoning occurring in the California plastic industry was uncovered when a worker requested his personal physician to perform a lead test on him after seeing “Lead” printed on bags of powder that he routinely handled at work. The worker with a blood lead level of  $164 \mu\text{g/dl}$  was suffering from abdominal pain, fatigue and constipation. Several of his co-workers also had elevated blood lead levels and two were found with blood leads  $108$  and  $114 \mu\text{g/dl}$  [25].

Adding lead to brass, typically about 2% lead, makes it easier to cut the keys [11]. Although the dose appears low, inhaled lead particles that penetrate to the unciliated airways (100% for particles less than  $< 1 \mu\text{m}$  and up to 50% of particles that are  $< 50 \mu\text{m}$ ) are completely absorbed; particles cleared from the ciliated airways and those settling in the area above the trachea are typically ingested with 10% to 15% being absorbed in adults and up to 50% in children [5, 26, 27]. Even though most of the lead exposure among locksmiths may be “subclinical”, recent research has nonetheless indicated that chronic low level lead exposure to adults poses significant risks for hypertension, adverse reproductive outcome, and possibly renal dysfunction [21-23, 28, 29].

Bone lead as a biomarker suggests the impact of low level lead exposure may not occur until a certain

**Table 2:** OSHA Lead Standards for Air and Blood

<i>Focus</i>	<i>Level</i>	<i>Comments</i>
Air	50 µg per m <sup>3</sup>	Permissible exposure limit (PEL): the employer shall assure that no employee is exposed to lead at concentrations >50µg per m <sup>3</sup> of air averaged over an 8 hour period.
Air	30 µg per m <sup>3</sup>	Action level: initiate medical surveillance for all employees exposed to levels above 30µg per m <sup>3</sup> for more than 30 days per year (regardless of respiratory protection).
Blood	>=60 µg per dL (2.90µmol per L) or average of last three levels is >=50 µg per dL (2.40µmol per L)	Medical removal from exposure

STAUDINGER, K. C.; ROTH, V. S.: Occupational. Lead Poisoning. American Family Physician, 1998. Adapted with permission from Occupational exposure to lead: final standard. U.S. Department of Labor, Occupational Safety and Health Administration. Federal Regist 1978; no. 29 CFR 1910.1025.

cumulative exposure has taken place [30]. In the locksmith industry, where biomonitoring for past lead exposure by blood lead or industrial hygiene data is practically nonexistent, KXRF offers the optimal tool for assessing the magnitude (and clinical impact) of long term cumulative exposure. KXRF measurement of lead in bone identifies a tissue compartment that may serve as a potential source of increased lead exposure during pathological or physiological states associated with high bone lead turnover, such as osteoporosis, hyperthyroidism, bone fractures, metabolic bone disease, cancer, nutritional deficiency, etc.

Our particular concern with this presumably moderate environmental exposure to lead in the work place is an endogenous factor that may contribute to substantial health risk in locksmiths during their lifespan. Constant exposure to lead, even in small amounts, leads to accumulation of lead in bones [31-35]. Lead can be actively mobilized from bone to blood during periods of normal homeostasis and periods of demineralization, such as long-term bed rest, osteoporosis, pregnancy and etc [36-38]. The ability of the skeleton to act as an endogenous source of lead to the blood has been shown in studies of the kinetics of lead after cessation of the occupational exposure [39-41]. Bone lead concentration is significantly related to health risk, especially in elevating blood pressure and increasing risk of hypertension. In these studies odds ratios (OR) for increased incidence of hypertension range from 1.1 to 1.9 for every 10µg/g increase in bone lead concentration, depending on the age, sex, and physiological status of the subjects studied [28, 29, 42, 43]. Applied to the present results, these studies suggest that the locksmiths in our study have odds ratios for increased risk of hypertension between 1.1 and 2.3 compared to the matched controls.

Lead dust and particles are known to be a hazard to the family of workers who transported these particles in their clothing from the workplace to their homes. Children and women of childbearing age are especially at-risk from the adverse affects of lead exposure. Skeletal body stores from chronic lead exposure are mobilized during pregnancy at an accelerated rate and contribute significantly to blood lead levels during this

period [44]. Since lead readily crosses the placenta and the developing blood brain barrier, lead exposure of the fetus and young child during pregnancy has potential implications for significant neurobehavioral disorders [44]. Exposure to high doses of lead during pregnancy has also been associated with an increased frequency of miscarriages and stillbirths among women working in the lead trades [45]. There is increasing evidence indicating that lead not only affects the viability of the fetus, but development as well. Developmental consequences of prenatal exposure to low levels of lead include reduced birth weight and premature births [46]. Lead is an animal teratogen, though most studies in human have failed to show a relationship between lead levels and congenital malformations [46]. Though regulatory limits on blood lead levels suggest there are levels of lead exposure without significant health risks, an increasing body of evidence indicates that there may be no lower limits of safe exposure to lead.

Data from the Department of Consumer affairs for the State of California show over 1300 licensed locksmiths located in Los Angeles County alone. There are thousands key makers that are not licensed locksmiths but exposed to lead from this occupational source, such as workers in hardware stores, home improvement stores, discount outlets and other merchants providing key duplication services.

This pilot study has identified an occupationally-related exposure to lead from yet another source that has not been previously examined. Additional research is needed to identify appropriate prevention measures and establish the relationship between chronic low level occupational lead exposure and body lead burden and corresponding health effects.

**Acknowledgements:** Supported in part by the the Research Centers for Minority Institutions Grant No. RR03026 and the National Center for Research Resources Grant No. RR11145.

## References

- Gerson, M.; Van den Eeden, S. K.; Gahagan, P.: Take-home lead poisoning in a child from his

- father's occupational exposure. *Am. J. Ind. Med.* **1996**, *29*(5), 507-508.
2. Sathaye, A. U.; Javadekar, B. B.: A presumptive case of lead poisoning in a brass-worker's child. *J. Indian Med. Assoc.* **2000**, *98*(8), 457-458.
  3. Needleman H. Human Lead Exposure. *CRC Press, Boca Raton*, **1992**, pp. 154
  4. Staudinger, K. C.; Ross, V. S. Occupational Lead Poisoning. *Am. Fam. Physician*, **1998**, *57*(4), 19-26.
  5. Cherrie, J. W.; Aitken, R. J.: Measurement of human exposure to biologically relevant fractions of inhaled aerosols. *Occup. Environ. Med.*, **1999**, *56*, 747-752.
  6. U.S. Department of Health and Human Services, National Institute of Occupational Safety and Health. Report to congress on workers' home contamination study conducted under the Workers' Family Protection Act (29 U.S.C. 671A). *Washington, DC*, **1995**.
  7. Grandjean, P.; Bauh, E.: Indirect exposures: the significance of bystanders at work and at home. *Am. Ind. Hyg. Assoc. J.*, **1986**, *47*, 819-824.
  8. State of California, Department of Justice. News Release: Major manufacturers agree to reduce amount of lead in door keys under settlement of proposition 65 lawsuits. <http://ag.ca.gov/newsalerts/2001/01-042.htm>. Last accessed, **9/17/2004**.
  9. Metal Supplier Online: Material property data: Copper alloys C35300, specifications. [www.supplieronline.com/propertypages/C35300.asp](http://www.supplieronline.com/propertypages/C35300.asp) Last accessed August 29, **2004**.
  10. C36000 Free Cutting Brass [www.setonresourcecenter.com/dtSearch/dtisapi6.dll?cmd=getdoc&DocId=197898&](http://www.setonresourcecenter.com/dtSearch/dtisapi6.dll?cmd=getdoc&DocId=197898&).
  11. State of California, Department of Justice. News Release: Attorney General Lockyer sues key manufacturers over failure to warn consumers about exposure to lead from keys in violation of Proposition 65. <http://caag.state.ca.us/newsalerts/1999/99-079>. Last accessed **2004**.
  12. Roscoe, R. J.; Gittleman, J. L.; Deddens, J. A.; Peterson, M. R.; Halperin, W. E.: Blood lead levels among children of lead-exposed workers: a meta-analysis. *Am. J. Ind. Med.*, **1999**, *36*, 475-481.
  13. Whelan, E. A.; Piacitelli, G. M.; Gerwel, B.; Schnorr, T. M.; Mueller, C. A.; Gittleman, J.; Matte, T. D.: Elevated blood lead levels in children of construction workers. *Am. J. Pub. Health*, **1997**, *87*(8), 1352-1355.
  14. Fernandez, F.; Hilligoss, D.: An improved graphite furnace method for the determination of lead in blood using matrix modification and the L'vov platform. *At. Spectrosc.* **1982**, *3*, 130-131.
  15. Heard, M. J.; Chamberlain, A. C.: Uptake of Pb by human skeleton and comparative metabolism of Pb and alkaline earth elements. *Health Phys.* **1984**, *47*, 857-865.
  16. Gerhardsson, I.; Attewell, R.; Chettle, D. R.; et al. In vivo measurements of lead in bone in long-term exposed lead smelter workers. *Arch. Environ. Health*, **1993**, *48*, 147-156.
  17. Kondrashov, V. S.; Rothenberg, S. J.: How to calculate lead concentration and concentration uncertainty in XRF in vivo bone lead analysis. *Appl. Radiat. Isot.*, **2001**, *55*, 799-803.
  18. Chettle, D. R.; Arnold, M. L.; Aro, A. C.; Fleming, D. E.; Kondrashov, V. S.; McNeill, F. E.; Moshier, E. L.; Nie, H.; Rothenberg, S. J.; Stronach, I. M.; Todd, A. C.: An agreed statement on calculating lead concentration and uncertainty in XRF in vivo bone lead analysis. *Appl. Radiat. Isot.*, **2003**, *58*, 603-5.
  19. Rothenberg, S. J.; Manalo, M.; Jiang, J.; et. al.: Maternal blood lead level during pregnancy in south central Los Angeles. *Arch Environ. Health*, **1999**, *54*, 382-389.
  20. Okun, A.; Cooper, G.; Bailer, J.; Bena, J.; Stayner, L. Trends in occupational lead exposure since the 1978 OSHA lead standards. *Am. J. Ind. Med.*, **2004**, *45*(6), 558-572.
  21. Muntner, P.; He, J.; Vupputuri, S. et. al.: Blood lead and chronic kidney disease in the general United State population: results for NHANES III. *Kidney Int.*, **2003**, *63*, 1044-1050.
  22. Lin, J. L.; Lin-Tan, D. T. Hsu, K. H.; et. al.: Environmental lead exposure and progression of chronic renal disease in patients without diabetes. *N. Engl. J. Med.*, **2003**, *348*, 277-286.
  23. Vupputuri, S.; He, J.; Muntner, P.; et. al. Blood lead level is associated with elevated blood pressure in blacks. *Hypertension*, **2003**, *41*, 463-468.
  24. Ferguson, J.: A case of "reasonable grounds". *Appl. Occup. Environ. Hyg.*, **1992**, *7*(12), 808-809.
  25. California Department of Health Services, Occupational Health Branch. Lead in the workplace: interesting case; General Industry - plastic compounders. 2002, pp 3. [www.dhs.ca.gov/ohb/OLPPP/regshort.pdf](http://www.dhs.ca.gov/ohb/OLPPP/regshort.pdf). Last accessed September 12, **2004**.
  26. Zigler, E. E.; Edwards B. B.; Jensen, R.L.; et. al.: Absorption and retention of lead by infants. *Pediatr. Res.*, **1978**, *12*, 29.
  27. Rabinowitz, M. B.; Kopple, J. D.; Wetherill, G, W.: Effects of food intake and fasting on gastrointestinal lead absorption in humans. *Am. J. Clin. Nutr.* **1980**, *33*, 1784.
  28. Korrick, S. A.; Hunter, D. J.; Rotnitzky, A.; Hu, H.; Speizer: Lead and hypertension in a sample of middle-aged women. *American Journal of Public Health*, **1999**, *89*, 330-335.
  29. Rothenberg, S. J.; Kondrashov, V.; Manalo, M.; Jiang, J.; Cuellar, R.; Garcia, M.; Reynoso, B.; Reyes, S.; Diaz, M.; Todd, A. C.: Increases of hypertension and blood pressure during pregnancy with increased bone lead. *American Journal of Epidemiology*, **2002**, *156*(12), 1079-1087.
  30. Hu, H.; Milder, F. L.; Burger, D. E. The use of K x-ray fluorescence for measuring lead burden in epidemiological studies: High and low lead burdens and measurement uncertainty. *Environ. Health Perspect.*, **1991**, *94*, 107-10.
  31. Somervaille, L. J.; Chettle, D. R.; Scott, M. C.; Tennant, D. R.; McKiernan, M. J.; Skilbeck, A.; Trethowan, W. N. In vivo tibia lead measurements as an index of cumulative exposure in

- occupationally exposed subjects. *British Journal of Industrial Medicine*, **1988**, *45*(3), 174-81.
32. Roels, H.; Konings, J.; Green, S.; Bradley, D.; Chettle, D.; Lauwerys, R.: Time-integrated blood lead concentration is a valid surrogate for estimating the cumulative lead dose assessed by tibial lead measurement. *Environ. Res.*, **1995**, *69*(2), 75-82.
  33. Hu, H.; Aro, A.; Rotitzky, A.: Bone lead measured by x-ray fluorescence: epidemiological methods. *Environmental Health Perspectives*, **1995**, *103*, 105-110.
  34. Schwartz, B. S.; Stewart, W. F.; Bolla, K. I.; Simon, M. S.; Bandeen-Roche, K.; Gordon, B.; Links, J. M.; Todd, A. C.: Past adult lead exposure is associated with longitudinal decline in cognitive function. *Neurology*, **2000**, *55*, 1144-1150.
  35. Kosnett, M. J. 2001 Lead: In Clinical Toxicology. *Saunders: New York*. **2001**, pp. 723-736.
  36. Landrigan, P. J.: Lead in the modern workplace (Editorial). *Am. J. Public Health*, **1990**, *80*, 907-908.
  37. Landrigan, P. J.; Baker, E. L.; Himmelstein, J. S.; Stein, G. F.; Wedding, J. P.; Straub, W. E. Exposure to lead from the Mystic River Bridge. The dilemma of deleading. *N. Engl. J. Med.*, **1982**, *306*, 673-676.
  38. Mariano, P. E.; Frantzblau, A.; Lilis, R.; Landrigan, P. J. Acute lead poisoning in construction workers – The failure of current protective standatds. *Arch. Environ. Health*, **1989**, *44*, 140-145.
  39. Roy, H. M.; Gordon, C. L.; Beaumont, L. F.; Chettle, D. R.; Webber, C. E.: Further experience with bone lead content measurements in residents of Southern Ontario. *Appl. Radiat. Isot.*, **1997**, *48*, 391-396.
  40. Tell, I.; Somerville, L. J.; Nillsson, U.; Bensryd, I.; Schutz, A.; Chettle, R. D.; Scott, M. C.; Skerving, S. Chelated lead and bone lead. *Scand. J. Work Environ. Health*, **1992**, *18*, 113-119.
  41. Ehrlich, R.; Robins, T.; Jordan, E.; Miller, S.; Mbuli, S.; Selby, P.; Wynchank, S.; Cantrell, A.; De Broe, M.; O' Haese, P.; Todd, A.; Landrigan, P.: A lead absorption and renal dysfunction in South African battery factory. *Occup. Environ. Med.*, **1998**, *55*, 453-460.
  42. Hu, H.; Aro, A.; Payton, M.; Korrick, S.; Sparrow, D.; Weiss, S.T.; Rotnitzky, A. The relationship of bone and blood lead to hypertension. *J. of Am. Med. Assoc.*, **1996**, *275*(15), 1171-1176.
  43. Glenn, B. S.; Stewart, W. F.; Links, J. M.; Todd, A. C.; Schwartz, B. S.: The longitudinal association of lead with blood pressure. *Epidemiology*, **2003**, *14*(1), 30-6.
  44. Gulson, R. L.; Jamerson, C. W.; Mahaffey, K. R.; Mizon, K. J.; Korsch, M. J.; Vimpani, G. Pregnancy increases mobilization of lead from maternal skeleton. *J. of Lab. and Clin. Med.*, **1997**, *130*(1), 51-62.
  45. Somervaille, L. J.; et. al.: In vivo measurements of bone lead- a comparison of two x-ray fluorescence techniques used at three different bone sites. *Phys. Med. Biol.*, **1989**, *34*(12), 1833-45.
  46. Todd, A. C.; Chettle, D. R. In vivo x-ray fluorescence of lead in bone: Review and current issues. *Environ. Health Perspec.* **1994**, *102*(2), 127-7.