Coll. Antropol. 39 (2015) 4: 915–922
Original Scientific Paper

Health Impact of Elevated Levels of Lead Encountered in the Manufacture of Crystal Glass

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ABSTRACT

Lead is known to cause harmful effects in the haematopoietic, nervous, digestive, renal, and other organ systems, inhibiting a number of enzymes in the biosynthesis of haem, as well as other enzymes with haematological significance. Our study involved 151 employees involved with the cutting of crystal, i.e. leaded glass, who had been found using eco-monitoring to have been exposed to above normal levels of lead. Our bio-monitoring process followed the values of lead, delta-ALAD and EPP.The highest level of lead detected was 276 µg/L, the lowest level of delta-ALAD was 99 nkat/L), and the highest level of EPP was 14.2 nmol/gHb). We had found that contrary to expectations, lead levels were not correlated to haemoglobin levels, or to gender or age, but were instead based only on the post of the employee and their time spent working at the glassworks. The levels of haematopoiesis were directly proportional to the levels of lead, however, the correlation was not statistically significant or had perhaps been masked by the exposure due to the employee's post and gender. We had also found a significant correlation of lead levels to the levels of renal function. The study had indicated some health impacts of lead on the exposed glass workers, but also at least partly diverged from the results of previous studies, prompting us to continue our research.

Key words: *lead, crystal glass, health impact, anaemia, renal function*

Introduction

The risk for lead exposure is present in numerous industries and various occupations[1], e.g. in lead mining, battery and car battery production, ammunition manufacture, production of crystal glass, china, ceramics, lead paint, in foundries, in cutting and lathing of lead-painted metals, in the smelting of lead, in antique and automobile restoration, as well as in construction[2–5]. In normal use, most solid lead products are relatively safe to use and rarely come to represent a significant health risk. However, the danger increases with various processing activities, such as heating, crumbling, grinding, lathing, spraying or burning of metal surfaces. In short, any process of heating or melting lead or its compounds, as well as work involving lead dust, poses a risk for lead poisoning[6]. The toxicity of lead compounds is directly proportional to their solubility in water. Organic compounds are generally more toxic than inorganic ones (PbO_4)[7–8].

The principal effects of lead absorption into the body are the result of an increased affinity for sulfhydril groups (i.e. the inhibition of enzymes with such groups), which causes disruption of protein- and enzyme-related functions[9–10]. Anaemia caused by lead ingestion is the result of the inhibition of haemosynthesis and aggravated failure of erythrocytes. In the case of minor build-up, changes in the kidneys are quite reversible, as early effects are mostly concentrated on the proximal tubules. A higher degree of exposure, however, leads to interstitial fibrosis and advanced renal failure[11–13]. It is posited that the lead affects the renin-angiotensin system, thus causing increased blood pressure[14].

The levels deemed borderline for such effects have shifted during the years. For a long time, the upper acceptable level, as set by WHO, used to be 300 µg/L, as this is the limit at which first clinical symptoms begin to appear. However, we realize today that subclinical effects appear at levels much lower, particularly those connected to brain development and function of the cardio-vascular system and the kidneys[15–17]. Cognitive deficits have been found in children at levels as low as 20 µg/L, and latest findings indicate that there is actually no safe lower limit as pertaining to the exposure of children and infants[18–22]. In our laboratory, the reference value was set at 150 µg/L, which

Received for publication June 2, 2013

is also the level indicated by the currently accessible data as the point where signs of enzyme inhibition in the process of haemoglobin synthesis (delta-ALAD) begin to appear, as well as consequently the effects on other organ systems.

Crystal glass as a term is actually misleading, as the chemical structure of glass is non-crystalline. The term gained traction mostly for commercial purposes, to accentuate the beauty and special properties of lead glass – which is capable of amazing refraction and seems »pseudo-crystal«. Lead glass is a type of glass in which lead takes the place of calcium in the traditional potash used e.g. in glass manufactories to lower the melting point of silica, which otherwise stands at 1723°C. The cleaner and whiter the potash, the cleaner the glass that is manufactured with it[23–24]. Because of the later-discovered health effects many glass manufacturers have discontinued production of lead glass or have modified their production to so-called unleaded crystal glass, in which lead oxide is substituted by other oxides (barium oxide, potassium oxide, zinc oxide). In the EU, the term »leaded crystal glass« is reserved for those products that contain at least 24% of lead oxide. If the product does not contain enough, or if other types of oxides are used, the product must be labelled »crystalline glass« or »crystal glass«. The glassworks we investigated uses 3 furnaces for glass melting. A pottery furnace is almost exclusively used to melt coloured crystalline glass that contains no lead oxides (it does, however, contain other oxides, which give it its colour). The KP3 furnace is used to melt crystalline glass with 2–5% lead content, and the EP2 furnace to melt leaded crystal glass (lead content of 24%).

Blood levels of lead are estimated, taking into account our current knowledge, to be the most accurate indicator of ongoing (current) lead absorption, as well as potentially an indicator of prolonged past exposure to lead (cumulative exposure)[25]. The time of day when samples are taken does not affect lead levels in blood; urine levels of lead are used to estimate ongoing (current) exposure[25–26]. With regard to sampling time, it is advisable to use either a single sample or urine collected over a 24-hour period[26]. Bio-response tests, such as measuring the amounts of free protoporphyrin, zinc protoporphyrin, and δ-aminolevulinic acid dehydratase (delta-ALAD) in erythrocytes, are quite useful to determine the biological effects connected to lead[25]. Coproporphyrin in urine is a non-specific bio-indicator, whose use as a bio-monitoring agent to determine exposure has recently been discouraged[25]. Delta-aminolevulinic acid dehydratase (delta-ALAD) in urine is a bio-indicator particularly suited for the monitoring of persons with occupational exposure to lead (at >400 µg Pb/L of blood); it is, however, most useful at the group level[25]. Delta-ALAD in erythrocytes is the most sensitive indicator of both acute and chronic effects of lead, with its activity decreasing as blood levels of lead increase[5]. However, although delta-ALAD determination is a sensitive method, its tendency towards instability limits its use for the monitoring of exposed persons[25]. In this case, the time of sampling is not important. Delta-ALAD becomes less active as the blood levels of lead reach around 50–199 µg/L[4]. Free porphyrin and zinc-porphyrin in erythrocytes are bio-indicators suggested for screening purposes in cases of occupational and environmental exposure, and are equally effective in monitoring of individuals as well as groups[25,27]. Levels of zinc protoporphyrin begin to increase as blood levels of lead reach around 200–500 µg/L[25]. Biological level limits in the Republic of Slovenia: monoatomic lead and its inorganic compounds: 400 µg/L in men, 300 µg/L in women; blood levels of delta-aminolevulinic acid dehydratase: 15 U/I E; blood levels of erythrocyte protoporphyrin: 2.67 µmol/I E[26].

Eco-monitoring (analysis of inhalable dust for lead content using the method of electro-thermic atomic absorption spectrometry (ETAAS), carried out by IJS in Ljubljana) found lead values exceeded in the room for dry cleaning using a grinder (1.7-times), and in the grinding room for wet grinding, where lead levels exceeded acceptable limits at every place of measurement (in the middle of the room by 1.8 times, at the 1st machine by 6.1 times, and at the 2nd machine by 2.0 times). In the cells with grinding machines, which employees only enter occasionally, the levels of lead in inhalable dust exceeded the acceptable limit of 0.1 mg/m^3 [27] for 8-hour exposure by 4 times[28].

Materials and Methods

At the glassworks premises, blood samples were taken and stored in test tubes used for trace elements; sodium heparin was then added, and the tubes were transported to the UKC Ljubljana Institute of Clinical Biochemistry. In addition to the testing for trace elements, blood samples were also taken for standard screening [haemogram, biochemistry (urea and creatinine)].

Blood content of lead was analysed using the method of electro-thermic atomic absorption spectrometry (ETAAS – Zeeman) and the SpectrAA-800, Varian apparatus in a filtered-air room. Calibration was carried out using Pb Tritisol Standard Merck; the experimental control was the Seronorm Whole Blood (SERO, L1-21 µg/L and L2-396 µg/L).

Catalytic concentration measurements of delta-ALAD were carried out on a UV-1601 Shimadzu spectrometer using the colorimetric method (perchloric acid, triton x-100, a phosphate buffering agent, substrate of delta-ALAD, Ehrlich's reagent, trichloroacetic acid).

We used the same reference values as the laboratory where the tests were carried out. The upper limit for normal lead levels is 150 µg/L, and elevated levels were parsed as follows:

– 150–200 µg/L SLIGHTLY ELEVATED
– 200–250 µg/L MODERATELY ELEVATED
– >250 µg/L HIGHLY ELEVATED

With regards to delta-ALAD, the normal catalytic activity is >500 nkat/L; its lower values were categorised as follows:

– 400–500 nkat/L SLIGHT DECREASE
– 300–400 nkat/L MODERATE DECREASE
– <300 nkat/L SIGNIFICANT DECREASE

K-erythrocytes: 4.5–6.3 (men); 4.2–5.4 (women) 10^{12}/L
Haemoglobin: 140–180 (men); 120–160 (women) g/L
Urea: 2.8–7.5 mmol/L
Creatinine: 44–97 µmol/L

Biological monitoring of exposure worker is in accordance with legislation of preventive health examination of workers as a part of obligatory monitoring.

Data analysis was carried out using both descriptive and review-oriented statistical methods, as well as tests to confirm our hypotheses. Arithmetic mean is used to present the central data values, while the spread is presented as standard deviation.

Variance analysis and the F-test were used to test the hypothesis that arithmetic means of various groups did not differ to a statistical significance.

The significance of connections between variables of the discrete type was determined using the chi-squared test, which is a probability-based measure of the difference between empirical and theoretical spread of two variables.

Pearson's Correlation Coefficient was used to estimate the degree of connection between pairs of continuous variables. The Coefficient can take values from –1 to 1.

We had also used Ward's hierarchic method of unit arrangement that measures the criteria function, which is the distance between two units defined as a square of Euclidean distance.

TABLE 1
CONNECTIONS BETWEEN VARIABLES OF BIO-MONITORING

		K-Hb g/L	Pb µg/L	Eryth-protopor. nmol/gHb	Eryth-ALAD nkat/L	Urea mmol/L
Pb µg/L	r	.329**				
	p	.000				
	N	151				
Eryth-protopor nmol/gHb	r	–.167*	.190*			
	p	.041	.020			
	N	151	151			
Eryth-ALAD nkat/L	r	–.078	–.531**	–.030		
	p	.343	.000	.716		
	N	151	151	151		
Urea mmol/L	r	.337**	.209*	.128	–.120	
	p	.000	.010	.119	.143	
	N	151	151	151	151	
Creatin µmol/L	r	.446**	.296**	.010	–.193*	.369**
	p	.000	.000	.905	.017	.000
	N	151	151	151	151	151

Cluster analysis with classification of cases using the hierarchical method and the method of leaders with Ward's method

The cluster analysis was used to check whether the employees had any common characteristics in terms of the results of bio-monitoring. The following variables were included in the process of worker classification according to bio-monitoring results: K-erythro, K-Hb, Urea, Creatinin, Eryth-ALAD, Lead, Eryth-protoporphyrin, with their various statistically significant connections, whether direct or indirect (Table 1).

Prior to classification the categories were standardised, i.e. their various measures were equalized. The results of classification yielded three typical groups of workers in terms of bio-monitoring results. Our hypothesis was that three groups of workers would emerge, such that they would differ – again, in respect of bio-monitoring results – from each other with statistical significance, and that further analysis of such classification's accuracy using other data on the same employees, such as time in employment at the glassworks, age, working post and gender, would yield a meaningful interpretation of each of the groups (Table 2).

Results

The study involved 151 employees aged 21 to 53, 81 (53.7%) of which were women with the average age of 43.6 (± 2.8 years), and 70 (46.3%) of which were men with the average age of 42.8 (± 6.3 years). The greatest share of study individuals belonged to the 41–45 age bracket (58.9%), followed by the brackets 46–50 and 36–40. 23 (15.2%) have been working in a lead-heavy environment for less than 10 years, 27 (17.9%) from 11 to 20 years, 46 (30.5%) from 21 to 25 years, 52 (34.4%) from 26 to 30 years, and 3 (2%) individuals for more than 30 years. 62 study subject worked at the »glass decoration – grinding« post, 39 at the »glass decoration – dry cleaning« post, 17 at rough grinding, while the remaining individuals worked at the electric furnace in automatic production or as glassblowers, in mixture preparation, smelting or other jobs. The majority of study subjects worked as glass grinders (decorators) – 41.1%, followed by grinders (glass decorators) in dry grinding (25.8%) and rough grinding (11.3%).

Average lead concentration: 145.9 µg/L (normal levels in a healthy population are below 150 µg/L), with the maximum measurement of 276 µg/L and a minimum of 20 µg/L.

Average values of delta-ALAD: 592.1 nkat/L (normal values in a healthy population are above 500 nkat/L), with the lowest measurement being 99 nkat/L and the highest one being 1565 nkat/L.

Average value of erythrocyte protoporphyrin: 2.0 nmol/gHb (normal values in a healthy population are lower than 9 nmol/gHb), with the maximum measurement of 14.2 nmol/gHb and a minimum of pa 0.3 nmol/gHb as seen in Table 3.

TABLE 2
RESULTS OF WORKER CLASSIFICATION INTO TYPES BY BIO-MONITORING RESULTS

		Group 1 (N = 36, 23.8%)	Group 2 (N = 45, 29.8%)	Group 3 (N = 70, 46.4%)	Total	F	p
K-erythro10^{12}/L	Average (standard deviation)	4.6 (0.3)	4.4 (0.3)	5.1 (0.3)	4.8 (0.4)	73.838	.000
K-Hb g/L	Average (standard deviation)	134.1 (10.,2)	138.2 (7.8)	154.9 (8.0)	145.0 (12.6)	90.906	.000
Urea mmol/L	Average (standard deviation)	4.8 (1.0)	4.7 (1.0)	5.9 (1.1)	5.3 (1.2)	21.076	.000
Creatin µmol/L	Average (standard deviation)	62.9 (18.8)	68.0 (7.7)	87.1 (11.3)	75.6 (16.7)	55.086	.000
Eryth-ALAD nkat/L	Average (standard deviation)	777.3 (284.5)	490.8 (215.9)	562.0 (232.0)	592.1 (262.8)	15.152	.000
Pb µg/L	Average (standard deviation)	109.0 (35.4)	143.6 (44.4)	166.4 (54.7)	146.0 (52.7)	17.304	.000
Eryth-protoporph. nmol/gHb	Average (standard deviation)	4.1 (13.2)	2.3 (1.6)	1.9 (1.8)	2.5 (6.6)	1.402	.249

The strongest correlation – a negative one – was discovered between Eryth-ALAD and Pb (moderate relation). Eryth-protoporphyrin was found to be very weakly correlated to other variables (Table 1). In our sample, workers with higher blood levels of lead had significantly higher values of K-Hb, which was indirectly also a function of gender. That is – men, who were generally found with higher K-Hb values, also had higher blood levels of Pb because they were assigned to posts with greater Pb-exposure. Women, on the other hand, whose average values of K-Hb were lower, also had lower blood levels of Pb as they had been employed at posts with lower presence of Pb. The male group showed no statistically significant correlation between K-Hb and blood values of lead ($r = .098$, $p = .419$), and neither did the female group ($r = .104$, $p = .356$). We also find that age is not significantly correlated to lead values; however, the same does not hold true for the time spent in employment at the glassworks, as this was found to be significantly correlated to lead values determined through bio-monitoring.

Blood levels of lead were significantly related neither to the workers' age of the whole sample ($r = –.073$, $p = .372$) nor to the age of men or women separately. Men who had been employed at the glassworks for a longer time were found with higher blood levels of lead ($r = .258$, $p = .031$). No such statistically significant connection was found in the women's group ($r = .183$, $p = .102$).

The first group (cluster analysis with Wards method) consisted of workers with an average of 109 µg/L Pb and the highest average values of Eryth-ALAD and Eryth-protoporphyrin (23.8% of workers). The second group consisted of workers with an average value of Pb at 143.6 µg/L. In terms of average Pb and its standard deviation of 44 µg/L, part of this group is already at risk due to Pb (29.8% of workers). The third group, which was the largest (70 workers, i.e. 46.4%), had average levels of Pb at 166.0 µg/L and a standard deviation of 54.6 µg/L and thus consisted of workers at highest risk due to Pb presence (46.4% of workers) (Table 2).

The third group, i.e. one at highest Pb risk, consisted of men only, who generally have the highest values of Hb (however, compared to Hb values in the population, these are barely normal) and erythro, which is simply the result of gender differences that put all the women in the first and second groups.

Somewhat surprisingly, our classification by bio-monitoring results put all the female workers in the first two groups with lower average values of Pb. Furthermore, the first two groups contained no male workers. Women in both groups had lower K-erythro and K-Hb values, as well as lower urea and creatinin than men. (With lower values of K-erythro and K-Hb being normal characteristics of their gender, and lower values of creatinin and urea the results of lower Pb exposure.) (Figure 1 and Table 3).

Bio-monitoring variable variance analysis was used on the present sample to estimate the percentage of their variability that could be attributed to either the employment post of the worker, their gender, or an interaction of both factors. From the significantly diverging values of Pb and Eryth-ALAD between employment post groups shown by variance analysis (see Table 4) we can infer that the amounts of Pb and Erith-ALAD are very likely to be dependent on this factor. However, the hypothesis that values of Pb and Eryth-ALAD differ between men and women in the same employment post category appears unlikely to be true (Table 4).

TABLE 3
BREAKDOWN OF EMPLOYMENT POST CATEGORIES BY WORKER TYPE CLASSIFICATION IN TERMS OF BIO-MONITORING RESULTS

Employment post category	Group		K-eryth. 10^{12}/L	K-Hb g/L	Urea mmol/L	Creatin µmol/L	Eryth-ALAD nkat/L	Pb µg/l	Eryth-protopor. nmol/gHb
Mixture preparation and melting	Group with Pb levels of 166.0 g/L (± 52.7)	$\overline{X}$	5.1	156.0	6.4	89.0	437.4	179.3	2.0
		SD	.2	3.0	.7	11.0	146.6	44.7	.7
		N	7	7	7	7	7	7	7
Hot manufacturing (automatic and manual)	Group with Pb levels of 166.0 g/L (± 52.7)	$\overline{X}$	4.9	155.3	5.5	85.6	385.6	180.0	1.3
		SD	.3	7.6	1.5	14.6	117.2	24.8	.4
		N	9	9	9	9	9	9	9
Cold glass processing, basic	Group with Pb levels of 109.0 g/L (± 35.4)	$\overline{X}$	4.5	134.8	5.6	51.4	1026.2	79.8	1.3
		SD	.3	10.1	.7	28.7	318.7	24.2	.4
		N	4	4	4	4	4	4	4
	Group with Pb levels of 143.6 g/L (± 44.4)	$\overline{X}$	4.6	149.0	5.3	65.0	630.0	122.0	.5
		SD	.	.	.	.	.	.	.
		N	1	1	1	1	1	1	1
	Group with Pb levels of 166.0 g/L (± 52.7)	$\overline{X}$	5.1	151.5	6.3	82.8	687.0	142.1	1.9
		SD	.3	9.7	.8	8.7	164.4	53.2	1.5
		N	12	12	12	12	12	12	12
	Total	$\overline{X}$	4.9	147.4	6.1	74.4	763.5	126.2	1.6
		SD	.4	11.7	.8	19.9	245.7	52.8	1.3
		N	17	17	17	17	17	17	17
Cold glass processing, decoration	Group with Pb levels of 109.0 g/L (± 35.4)	$\overline{X}$	4.6	134.2	4.7	63.8	749.6	113.5	2.2
		SD	.3	10.5	1.0	17.4	273.2	35.3	1.7
		N	31	31	31	31	31	31	31
	Group with Pb levels of 143.6 g/L (± 44.4)	$\overline{X}$	4.4	138.0	4.7	68.0	487.6	144.1	2.3
		SD	.3	7.7	1.0	7.7	217.4	44.8	1.6
		N	44	44	44	44	44	44	44
	Group with Pb levels of 166.0 g/L (± 52.7)	$\overline{X}$	5.1	154.6	5.7	86.5	587.0	181.7	2.2
		SD	.4	8.5	.9	10.6	235.2	48.8	2.4
		N	31	31	31	31	31	31	31
	Total	$\overline{X}$	4.7	141.7	5.0	72.2	593.3	146.2	2.2
		SD	.4	12.2	1.1	15.2	261.5	50.5	1.9
		N	106	106	106	106	106	106	106
Maintenance and servicing	Group with Pb levels of 166.0 g/L (± 52.7)	$\overline{X}$	4.9	160.2	6.3	92.3	476.7	158.2	1.6
		SD	.2	5.2	1.9	14.0	150.4	66.1	.8
		N	6	6	6	6	6	6	6
Head employees	Group with Pb levels of 109.0 g/L (± 35.4)	$\overline{X}$	4.2	128.0	4.7	79.0	641.0	84.0	2.1
		SD	.	.	.	.	.	.	.
		N	1	1	1	1	1	1	1
	Group with Pb levels of 166.0 g/L (± 52.7)	$\overline{X}$	4.9	156.2	5.9	95.2	701.6	97.8	1.5
		SD	.2	7.2	1.3	11.1	408.4	77.4	.8
		N	5	5	5	5	5	5	5
	Total	$\overline{X}$	4.8	151.5	5.7	92.5	691.5	95.5	1.6
		SD	.3	13.2	1.3	11.9	366.1	69.4	.8
		N	6	6	6	6	6	6	6

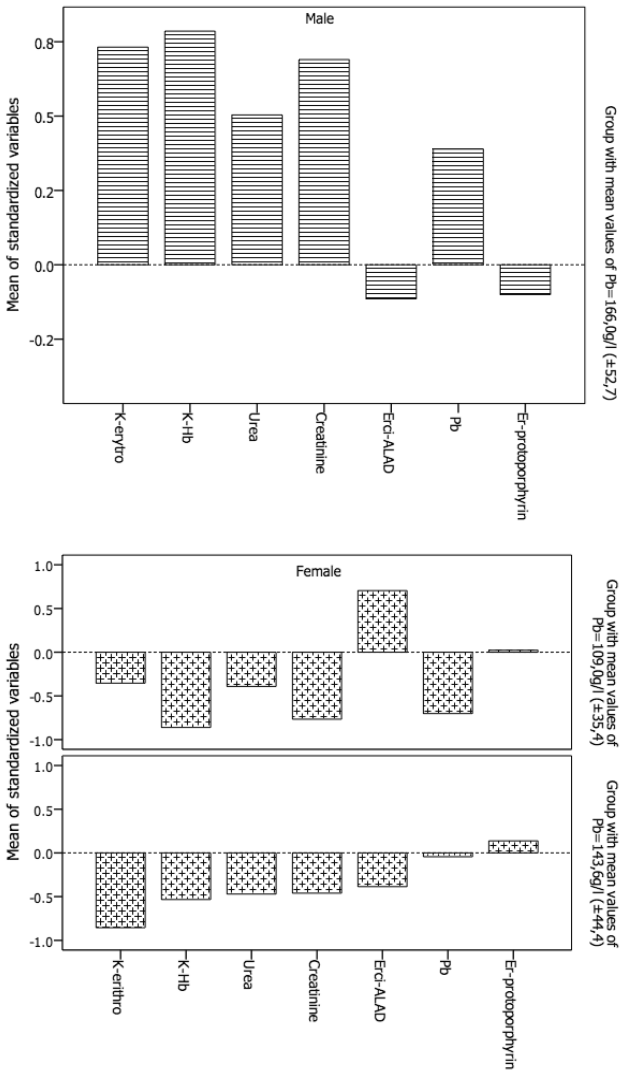

Fig. 1. Correlation of values of standardized bio-monitoring variables to classification into types with significantly different blood values of Pb in men and in women.

TABLE 4
BIO-MONITORING VARIABLE VARIANCE ANALYSIS FOR FACTORS OF GENDER AND EMPLOYMENT POST

Factor	Dependent variable	F	p	Percentage of independent variable variance explainable by the factor (i.e. the square of Eta-coefficient)
Gender	Pb µg/L	4.819	.030	.038
	Eryth-ALAD nkat/L	1.129	.290	.009
	Eryth-protopor. nmol/gHb	.004	.947	.000
	K-erythro10^{12}/L	23.262	.000	.159
	K-Hb g/L	27.670	.000	.184
	Urea mmol/L	4.914	.028	.038
	Creatinin µmol/L	18.115	.000	.128
Workplace	Pb µg/L	2.997	.014	.109
	Eryth-ALAD nkat/L	3.789	.003	.133
	Eryth-protopor. nmol/gHb	.661	.654	.026
	K-erythro10^{12}/L	.796	.555	.031
	K-Hb g/L	.324	.897	.013
	Urea mmol/L	1.769	.124	.067
	Creatinin µmol/L	2.086	.072	.078
Gender * Workplace	Pb µg/L	.108	.898	.002
	Eryth-ALAD nkat/L	2.294	.105	.036
	Eryth-protopor. nmol/gHb	.405	.668	.007
	K-erythro10^{12}/L	.161	.852	.003
	K-Hb g/L	.947	.391	.015
	Urea mmol/L	.128	.880	.002
	Creatinin µmol/L	.342	.711	.006

Highest explainable variance in comparison to other variables of bio-monitoring was found in K-Hb (18%) and K-erythro (16%) regardless of gender, while the employment post category had no statistically significant influence on K-Hb and K-erythro values. The value of Eryth-ALAD had been showed to vary significantly between the six analyzed employment post categories (i.e. »mixture preparation and melting,« »hot manufacturing (automatic and manual),« »cold processing, basic,« »cold processing, decoration,« »maintenance and servicing,« »head employees«), with 13% of its variability from lowest in »mixture »preparation and melting« to highest in »cold basic processing« attributable to the employment post category (see Table 4). In the present sample, the values of results showing blood levels of Pb and Eryth-ALAD were dependent on the employment post category but not on gender (it should however be noted that the sample was gender-selected by default, as no women were employed at posts with highest Pb exposure, thus making it impossible to determine the values of Pb and Eryth-ALAD in women, had they been employed evenly at all available posts).

Discussion

Toxic effects of lead comprise a broad spectrum of laboratory and clinical manifestations, varying from subtle, sub-clinical biochemical anomalies to serious states of clinical emergency. Lead causes damage to the haematopoietic,

nervous, digestive, renal, cardio-vascular, and reproductive organ systems[3,5,25]. Lead poisoning is known nowadays to be primarily a chronic disorder, brought upon by gradual accretion of lead in the human body[5,25]. Accumulation of lead in the body means that the acute symptoms are usually a manifestation of (sub)chronic poisoning[29].

Lead has a strong inhibitory effect on a number of enzymes contributing to biosynthesis of haem, as well as other haematologically important enzymes[3]. The classic symptom of lead poisoning is anaemia, which occurs due to the reduction of haemoglobin synthesis, the inhibition of erythropoietin (erythrocyte regulator) production in the kidneys and a reduced life-span of erythrocytes[4]. The inhibition of haem biosynthesis is primarily a consequence of the inhibition of the delta-ALAD cytoplasm enzyme. The effect is tied to the dosage and begins to appear at the lead blood content of 100–200 μg/L[4]. The ferrochelatase mitochondrial enzyme is the next most vulnerable enzyme in the biosynthesis of haem, its role being the catalysis of the transfer of iron from ferritin to protoporphyrin to assist in haemosynthesis. Inhibition of said enzyme thus causes increased secretion of coproporphyrin in urine and the accumulation of protoporphyrin (zinc protoporphyrin) in the erythrocytes. Another consequence of chronic exposure to lead is the inhibition of erythropoietin synthesis in the renal tubules; erythropoietin is a glycol-protein hormone that plays a role in the regulation of erythrocyte production[4]. At minor levels of excessive lead exposure, levels of haemoglobin often remain within normal boundaries[4], so anaemia cannot be the criterion for proof of occupational exposure. Furthermore, our study was not concerned with significant cases of anaemia, but was rather interested in finding decreased haemoglobin values, which were, however, at least partly occluded by the sample itself, as values of lead were found to have been highest in male subjects, which, however, also characteristically have higher default haemoglobin values than women, who also suffer from other causes of potential anaemia in their reproductive age[30].

Kidneys represent the main path of elimination of lead from the body. Furthermore, renal tissue is among those soft tissues with highest levels of lead[5]. Nephrotoxicity is one of the earliest described toxic effects of lead[31]. Lead nephropathy primarily involves the malfunction and disorder of proximal renal tubules. Malfunction of proximal tubules is most frequently encountered in workers exposed to lead and in serious cases of lead poisoning in children, presenting as the Fanconi syndrome, with characteristic aminoaciduria, glycosuria, and phosphaturia[3,31]. Subtler renal tubular changes manifest themselves through increased secretion of enzymes such as N-acetyl-ß-glucosaminidase (NAG) and low-molecular-mass proteins such as $ß_2$-microglobulin in urine[3]. While tubular malfunction is regarded as an early defect in the framework of a clinically-manifested lead poisoning and a potentially reversible one, prolonged occupational exposure to lead may cause progressive renal disease, which manifests itself through glomerular sclerosis, diffuse interstitial fibrosis and renal failure[3]. As had been the case with previous research, our study found a statistically significant connection between lead exposure and renal function disorder[32].

Our project studied 151 employees of R. glassworks (81 women, aged 43.6 years on average, and 70 men with the average age of 42.8 years). The greatest part of the employees (62) were posted at the section for grinding – glass decoration, 39 at dry cleaning, 17 at rough grinding, while the remaining workers (exclusively male) were working at the electric furnace, as glassblowers, in the smelter and at other posts (maintenance and leadership jobs). Measurements were taken to determine that the values of lead at many of the posts exceeded acceptable levels (measurements were done in direct atmospheric contact, i.e. without accounting for the use of personal protective gear). Highest average levels of lead were found in employees posted at mixture preparation (206), followed by glassblowers and machinists, while head employees exhibited the lowest levels (95); highest values of delta-ALAD were found in rough cleaning (763.5), followed by head employees and smelter workers, while values in workers posted at mixture preparation were the lowest (238.3).

In the first phase of our study, we were interested in the dependence of bio-monitoring values on gender, post, time employed at a workplace with lead exposure, and accompanying effects on renal function and haematopoiesis. We had discovered that the values of bio-monitoring were negatively correlated to levels of renal function with a high statistical significance, while values of haematopoiesis (particularly haemoglobin levels) showed a statistically insignificant decrease that was inversely proportional to lead values – however, the statistical insignificance was perhaps the result of the fact that lead values were highest in males who also have higher haemoglobin, while they were significantly lower in women due to other factors.

Lead levels showed a statistically significant correlation to the post of employment; however, this is again a case where we find the posts with highest risk of exposure to be staffed exclusively by men.

We shall continue with similar research, as the present study has on the one hand highlighted a health risk in employees, and on the other hand (perhaps also due to a small sample) exhibited a slight divergence from the results of previously published studies, which mandates further and more focused research.

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UTJECAJ POVIŠENIH KONCENTRACIJA OLOVA PRI PROIZVODNJI KRISTALNOG STAKLA

S A Ž E T A K

Olovo uzrokuje štetne efekte na hematopoetskom, nervnom, digestivnom, urinarnom i drugim organskim sistemima. Inhibira nekoliko enzima koji regulišu biosintezu hema, kao i druge enzime, koji su od značaja za hematopoezu. Zbog puteva eliminacije olova iz organizma je disfunkcija proksimalnih tubula bubrega najčešće opisivana kod radnika izloženih olovu. U istraživanje smo uključili 151 radnika, koji rade na brušenju kristalnog olovnog stakla i gdje smo s ekološkim monitoringom utvrdili prekoračene granične vrijednosti. Biološkim monitoringom smo pratili vrijednosti koncentracije olova, ALAD-D i EPP. Najviša vrijednost koncentracije olova je bila 276 µg/L (prosjek 145,9 µg/L), najniža ALAD-D je bila 99 nkat/L (prosjek 592,2 nkat/L) i najveća vrijednost koncentracije EPP 14,2 nmol/L 14,2 nmol/gHb (prosjek 2,0 nmol/gHb). Utvrdili smo da vrijednosti koncentracije olova ne koreliraju očekivano sa vrijednostima koncentracije hemoglobina, niti sa polom i uzrastom, nego samo u zavisnosti od radnog mjesta i radnog staža u staklari. Nije uočena statistički značajna razlika u hematopoezi u zavisnosti od vrijednosti koncentracije olova, odnosno nepostojanje statistički značajne razlike je prikriveno izloženošću na radnom mjestu i polom. Takođe smo utvrdili da vrijednosti koncentracije olova značajno koreliraju sa vrijednostima parametara bubrežne funkcije. Istraživanje je ukazalo na zdravstvene efekte olova kod izloženih staklara, istovremeno takođe i djelimična razilaženja sa rezultatima već napravljenih studija. Istraživanje ćemo iz tih razloga nastaviti.