Available online at www.sciencedirect.com
SCIENCE DIRECT
Toxicology Letters 162 (2006) 239–245
Toxicology Letters
www.elsevier.com/locate/toxlet

Biological monitoring of welders exposed to aluminium

Bernd Rossbach[a,*], Mark Buchta[a], György A. Csanády[b], Johannes G. Filser[b], Wolfgang Hilla[c], Klaus Windorfer[c], Joachim Stork[c], Wolfgang Zschiesche[d], Olaf Gefeller[e], Annette Pfahlberg[e], Karl-Heinz Schaller[f], Ellen Egerer[a], Luis Carlos Escobar Pinzón[a], Stephan Letzel[a]

[a] *Institute for Occupational, Social, and Environmental Medicine, University of Mainz, Obere Zahlbacher Straße 67, 55131 Mainz, Germany*
[b] *Institute of Toxicology, GSF-National Research Center for Environment and Health, Ingolstaedter Landstraße 1, 85764 Neuherberg, Germany*
[c] *Occupational Health Center of AUDI AG, 85045 Ingolstadt, Germany*
[d] *Berufsgenossenschaft der Feinmechanik und Elektrotechnik, Gustav-Heinemann-Ufer 130, 50968 Koeln, Germany*
[e] *Department of Medical Informatics, Biometry, and Epidemiology, University of Erlangen-Nuremberg, Waldstrasse 6, 91054 Erlangen, Germany*
[f] *Institute and Outpatient Clinic for Occupational, Social, and Environmental Medicine, University of Erlangen-Nuremberg, Schillerstrasse 27, 91054 Erlangen, Germany*

Available online 8 November 2005

Abstract

To evaluate an adequate strategy for biological monitoring of aluminium (Al), a group of 62 Al welders (age in 1999: 23–51 years, median 35 years) was surveyed annually from 1999 to 2003 by determination of pre- and post-shift Al in urine and plasma. Biomonitoring was supplemented by personal air measurements of the total dust concentration. The welders' internal exposure was compared to the exposure of 60 non-exposed assembly workers (age in 1999: 21–51 years, median: 36 years) who were surveyed in 1999, 2001 and 2003.

Having a nearly constant dust exposure, median concentrations of Al in urine (Al in plasma) of the welders decreased from 40.1 μg/g to 19.8 μg/g creatinine (8.7 to 4.6 μg/l). For the control group the median levels of Al in urine (plasma) ranged from 4.8 μg/g to 5.2 μg/g creatinine (2.4–4.3 μg/l) indicating a higher sensitivity for the marker Al in urine. No systematic differences have been found between pre- and post-shift internal exposure. This might be caused by the slow elimination kinetics and low systemic bioavailability of Al. A correlation analysis did not yield close relationships between dust exposure, Al in plasma and Al in urine underlining the importance of biomonitoring for assessment of Al exposure.
© 2005 Elsevier Ireland Ltd. All rights reserved.

Keywords: Aluminium; Aluminium welders; Biomonitoring; Welding fumes

1. Introduction

With a share of approximately 8.1%, aluminium (Al) is the most abundant metal of the earth's crust. Next to this high natural abundance the industrial use of Al has increased due to its advantageous material properties like light weight, high durability, and high electric and thermal conductivity. Al can be found ubiquitously in our environment and is also present in drinking water, food and some pharmaceuticals. Hence, an uptake of Al by the general population is inevitable (Yokel and McNamara, 2001).

Next to background exposure which mainly can be ascribed to an oral Al uptake at particular work places,

* Corresponding author. Tel.: +49 6131 3930279; fax: +49 6131 3930280.
E-mail address: rossbach@uni-mainz.de (B. Rossbach).

0378-4274/$ – see front matter © 2005 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.toxlet.2005.09.018

Table 1
Median (bold) and range of urinary Al excretion of welders found in several studies in the past

n	Age (years)	Total exposure time (month)	Al in urine (μg/g creatinine)	Reference
25[a]	– (19–62)	**30** (1–156)	**69** (19–262)	Nielsen et al. (1993)
17	**37**[b] (24–48)	**48**	**65** (24–164)	Hanninen et al. (1994)
38	**39**[b] (26–56)	**>60**	**24** (4.5–162)	Sjogren et al. (1996)
16	**32** (23–55)	**17** (11–51)	**118** (44–244)	Letzel et al. (1999)
29[c]	**36** (24–54)	**56** (0–198)	**49**[e] (8–154)	Riihimaki et al. (2000)
30[d]	**44** (22–58)	**165** (44–275)	**192**[e] (86–745)	

[a] Post-shift results for 19 workers out of this group.
[b] Mean.
[c] Low-exposure group.
[d] High-exposure group.
[e] Concentrations in μg/l urine.

for example, in Al production, in foundries or in the Al powder industry, an additional occupational exposure occurs. As Al sheets are increasingly used in construction of vehicles also, an increasing number of welders can be found among Al-exposed workers. In case of occupational Al exposure, inhalation is the main route of uptake. Therefore, bioavailability is dependent on the particular Al species and particle size of the inhaled material.

After absorption, Al distributes unequally in all tissues. In normal human beings the skeletal system and the lung account for about 50% and 25% of the body burden, respectively (ATSDR, 1999). Within the blood Al is approximately equally distributed between plasma and cells. In plasma the majority of Al is bound to transferrin and citrate (Yokel and McNamara, 2001).

Al is eliminated mainly by renal excretion. Data on Al half-times in the urine of occupationally exposed persons varies widely in literature from days to months or years depending on the duration of the exposure and Al species (Letzel et al., 1999). As Al in the organism is stored in compartments with slow elimination kinetics (i.e. bones) urinary Al excretion can be increased for years after termination of occupational exposure (Ljunggren et al., 1991). While for non-occupationally exposed persons a preliminary reference level of <15 μg/l in urine has been published in Germany (HBM-Kommission, 1998) several studies showed an increased excretion of Al in exposed welders (see Table 1).

Studies regarding the Al powder industry showed that long-term inhalative exposure to Al can induce pulmonary fibrosis (Kraus et al., 2000). A neurotoxic potential of Al is discussed, since elevated levels of Al were found in brains of patients who died of dialysis-related encephalopathy (Alfrey et al., 1976). Some epidemiological studies showed a poorer performance in cognitive tests or a higher abundance of neurological symptoms for workers occupationally exposed to Al (Rifat et al., 1990; Sjogren et al., 1996).

These possible adverse effects should give reason to conduct a continuous surveillance of occupational Al exposure which can be carried out by ambient or biological monitoring.

In Germany, airborne Al exposure is limited by the occupational exposure limit for alveolar fine dust (MAK) which is 1.5 mg/m^3. For biological monitoring the determination of the urinary Al concentration is recommended which should be lower than threshold limit of 200 μg/l (BAT-value) (DFG, 2004).

Within our longitudinal study Al exposure of welders was followed up for 5 years. Among other things it was the intention of the study to evaluate an adequate strategy for biological monitoring in Al welders. By comparing the study population with non-exposed controls, the sensitivity of the alternative biomarker Al in plasma should be investigated. For both markers of internal exposure, the influence of the sampling time and possible correlations with external total dust exposure should be assessed.

2. Material and methods

Within a 5-year longitudinal study from 1999 to 2003, the internal Al exposure of $n = 143$ Al welders in the automobile industry and in train body and truck trailer construction was followed up by an annual biological monitoring, including the determination of the Al concentration in pre- and post-shift plasma and urine samples. In every cross sectional study a questionnaire referring to working conditions, working hours, and non-occupational Al exposure was applied. External occupational exposure to total dust was measured by personal air sampling at selected work places.

We report here the results for $n = 62$ workers (exposed group) out of this study population. For all of them at least

one complete data set with data from ambient and biological monitoring was obtained within the 5-year study. Altogether 123 data sets could be analysed comprising $n = 39/23/23/19/19$ measurements in 1999/2000/2001/2002 and 2003, respectively. At the beginning of the study the participants' age ranged from 23 to 51 years (median: 35.0). At that time the workers had been welding Al materials for 7–180 months (median 60) using predominantly the MIG method.

The control group consisted of $n = 60$ assembly workers aged from 21 to 51 years (median: 36.0) in 1999 without any exposure to Al-containing welding fumes. For all members of this group the pre-shift Al concentration in plasma and urine was determined at least once in 1999, 2001 or 2003 resulting in 152 data sets altogether (1999: $n = 54$; 2001: $n = 51$; 2003: $n = 47$).

In the exposed group personal air sampling was carried out in the breathing zone of the workers. For collection of welding fumes a membrane filter cassette was mounted underneath the forced-air-ventilated helmets. Using an Alpha-1 sampler (DuPont®) the median sampling time ranged from 120 min to 240 min. The filter membranes were analysed gravimetrically for total dust.

To avoid pre-analytical Al contaminations, post-shift biological material was collected after sufficient body cleaning and change of working clothes into street clothes, while pre-shift material was collected before changing of clothes into working clothes.

The quantitative determination of the Al concentration in plasma and urine was carried out by graphite furnace atomic absorption spectrometry (GF-AAS) using the standard addition technique for calibration according to the method of Fleischer and Schaller (2000). The limits of detection (LOD) were 1.4 μg/l for Al in plasma and 1.0 μg/l for Al in urine, respectively. All determinations were performed under a strict internal and external quality-assessment scheme (Schaller et al., 2002).

To minimise diuretic dilution effects in the spot urine samples, the urinary creatinine concentrations were determined by the Jaffé-method (Taussky, 1956). For analysis only samples with a creatinine concentration between 0.5 g/l and 2.5 g/l were considered, and the measured Al concentration was related to the creatinine level of the sample.

For statistical analysis the SPSS package (release 11.0.1., SPSS Inc., Chicago, USA) was used. Biological monitoring data lower than the limit of detection were replaced by half of the detection limit for calculations. Pre- and post-shift Al concentrations in biological materials were compared by the Wilcoxon matched-pairs signed rank test. For comparison of internal exposure of welders and controls the Mann–Whitney test was used. In the exposed group, correlations between Al in plasma and Al in urine and the external dust concentration were examined nonparametrically by the Spearmans procedure. For this analysis the individual pre- and post-shift measurements of Al in plasma and urine were averaged. For statistical tests and correlation analysis p-levels ≤ 0.05 were considered as significant.

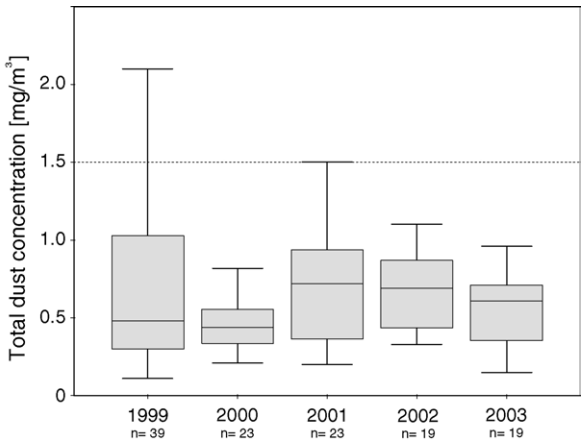

Fig. 1. External exposure to total dust in the welders' group, determined by annual personal air sampling (dotted line: German MAK value for respirable dust).

3. Results

3.1. Ambient monitoring

The concentrations of respirable dust measured in the five consecutive samplings from 1999 to 2003 are compared in Fig. 1. Total dust exposure of the Al welders ranged from 0.11 mg/m^3 to 15.60 mg/m^3. According to the annual median values which ranged from 0.44 mg/m^3 to 0.72 mg/m^3 only minor fluctuations in external exposure occurred. The median exposure was well below the German MAK-level (1.5 mg/m^3). Dust concentration higher than the MAK-level were observed in 13 samplings (10.5% of all measurements).

3.2. Biological monitoring

The concentrations of Al in the welders' plasma samples ranged from 0.7 μg/l (1/2 LOD) to 51.0 μg/l, including two samples < LOD. Pre- and post-shift results of the five annual samplings are compared in Fig. 2. From 1999 to 2002, the median concentrations decreased from 8.1/8.0 μg/l (pre-/post-shift) to 3.6/3.5 μg/l. However, in 2003, a slight increase was observed (5.0/5.7 μg/l). As a comparison of the individual pre- and post-shift, results by a Wilcoxon test did not show a significant influence of the sampling time on the Al concentration; for further calculations the averaged value from pre- and post-shift sampling was used.

In the control group Al concentrations ranged from 0.7 μg/l to 31.0 μg/l plasma, including eight samples < LOD. In 1999, 2001 and 2003, the median values were 4.3 μg/l, 2.4 μg/l and 3.7 μg/l, respectively, show-

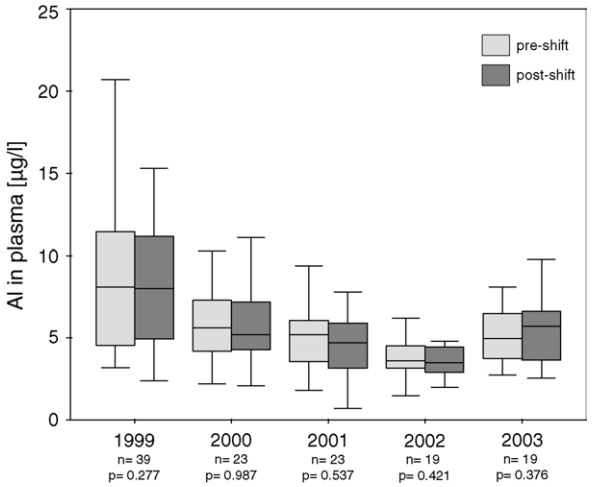

Fig. 2. Pre- and post-shift levels of Al in plasma determined over 5 years in the exposed group (*p*: significance levels of the Wilcoxon signed rank test for comparison of pre- and post-shift concentrations).

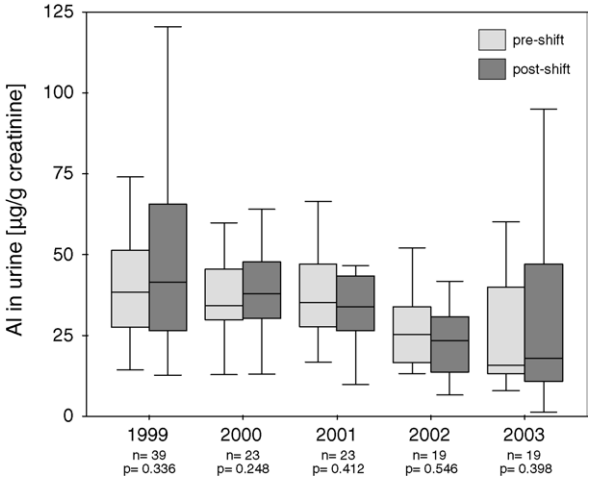

Fig. 4. Pre- and post-shift creatinine-related urinary Al excretion determined over 5 years in the exposed group (*p*: significance levels of the Wilcoxon signed rank test for comparison of pre- and post-shift concentrations).

ing a minor fluctuation of internal Al exposure in this group.

A statistical significant difference between welders and controls was found for each cross sectional analysis (Mann–Whitney test, see Fig. 3).

The urinary Al excretions in the welders ranged from 1.4 μg/g to 355.4 μg/g creatinine. A comparison of pre- and post-shift results from 1999 to 2003 is shown in Fig. 4. Within the 5-year period the median Al concentration steadily decreased from 38.3/41.5 μg/g to 15.8/18.0 μg/g creatinine (pre-/post-shift). Al concentrations higher than the German BAT-level (200 μg/l) have been measured in 14 samples overall (pre-shift: 6, post-shift: 8). Comparable to the situation for plasma, an individual comparison of pre- and post-shift results did not reveal any significant influence of the sampling time on creatinine-related Al excretion.

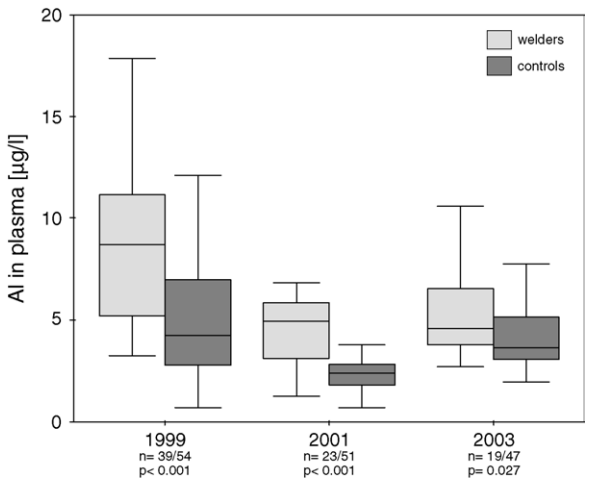

Fig. 3. Comparison of Al concentrations in plasma of Al welders and controls (*p*: significance levels of the Mann–Whitney test for comparison of exposure in both groups).

Including three samples with Al concentrations below LOD, the urinary Al concentrations in the control group ranged between 0.2 μg/g and 30.3 μg/g creatinine. In 1999, 2001 and 2003, the median values were 4.8 μg/g, 5.0 μg/g and 5.2 μg/g creatinine, respectively, indicating a rather constant background exposure. Although Al excretion of welders clearly decreased from 1999 to 2003, statistically significant differences between welders and controls were observed for every cross sectional analysis (Mann–Whitney test, see Fig. 5).

3.3. Correlation analysis

The Spearman correlation coefficients between the total dust concentration in air as a marker of external exposure, and the concentration of Al in plasma and urine as markers of internal exposure are listed in Table 2. For the parameters total dust/Al plasma an individual analysis of the annual samplings resulted only for 2001 in a significant correlation ($r = 0.449$). For total dust/Al urine in 1999 and 2003, significant correlations ($r = 0.617$ and 0.712) were observed. An analysis of personal averaged data from 1999 to 2003 revealed a significant relationship only for total dust/Al urine ($r = 0.367$).

Significant correlations between both markers of internal exposure (Al plasma, Al urine) were found for the samplings in 2001, 2002 and 2003, as well as for the

Table 2
Spearman correlation coefficients for correlations between ambient total dust concentration, Al in plasma and Al in urine (related to creatinine) calculated for the individual annual samplings and for averaged personal data from 1999 to 2003

Year	*n*	Correlation coefficient		
		Total dust/Al plasma	Total dust/Al urine	Al plasma/Al urine
1999	39	0.165	**0.617**	0.221
2000	23	0.262	0.137	0.103
2001	23	**0.449**	0.391	**0.463**
2002	19	0.417	0.300	**0.708**
2003	19	0.436	**0.712**	**0.681**
Mean (1999–2003)	62	0.120	**0.367**	**0.460**

Bold: $p < 0.05$.

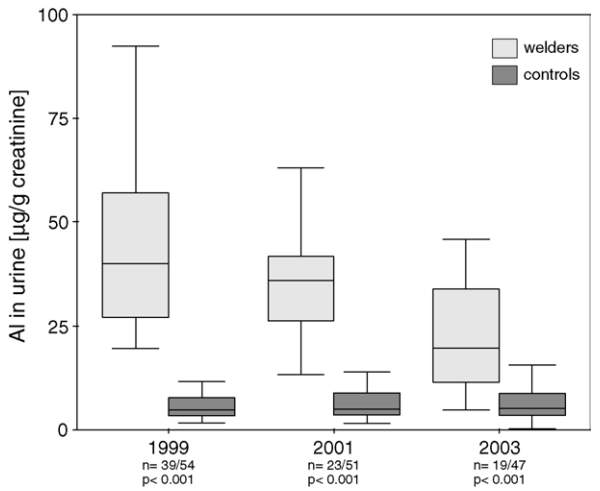

Fig. 5. Comparison of creatinine-related Al concentrations in urine of Al welders and controls (*p*: significance levels of the Mann–Whitney test for comparison of exposure in both groups).

averaged data with correlation coefficients ranging from 0.460 to 0.708.

4. Discussion

To evaluate an adequate strategy for sampling, we determined internal Al exposure of welders by analysing pre- and post-shift samples of plasma and urine annually over 5 years. As it is shown in Figs. 2 and 4, neither for plasma nor for urine systematic changes of the Al concentration in pre- and post-shift samples occurred. These results which are in line with data published by Gitelman (1995) and Gitelman et al. (1995) suggest that in case of prolonged occupational exposure the sampling time seems to be of minor importance for determination of the Al body burden. For explanation, the long half-life of Al in the human body has to be considered. After inhalative deposition of Al containing particles in the lungs, a slow resorption might contribute to the long elimination half-life of Al ranging from days to month or years (Letzel et al., 1999).

By comparing Al concentrations in plasma and urine of welders and controls, differences according to the particular marker of internal exposure become apparent (Figs. 3 and 5). While the median Al concentrations in plasma for both groups differed by a factors of 2.0, 2.1 and 1.3 for the years 1999, 2001 and 2003, respectively, the same factors for Al in urine were 8.4, 7.2 and 3.8. Considering these differences, an occupational exposure to Al is indicated more sensitively by the urinary Al excretion than by the Al concentration in plasma. As the same effect was reported by other authors for the Al concentrations in serum and urine of exposed workers our findings seem to be typical for occupational Al exposure (Hanninen et al., 1994; Gitelman et al., 1995; Riihimaki et al., 2000). A reasonable explanation of these findings is still missing.

In our study Al in urine of welders steadily decreased from 1999 (median 40.1 µg/g creatinine) to 2003 (19.8 µg/g creatinine (Fig. 4)). According to Table 1, the Al excretions found were comparable to the results published by other authors at the beginning of the study but have to be considered as rather low in 2003.

Except for the sampling in 2003, a decrease of the Al concentration was also found in plasma (Fig. 2). This reduction of internal exposure can be explained by improvements in occupational hygiene and changes in the production process rising the degree of automation in the course of the study. The contrary shifting of Al in plasma and urine found from 2002 to 2003 (Figs. 2 and 4) may be due to the increase of the analytical imprecision occurring, when Al concentrations slightly higher than the LOD have to be determined in plasma.

To evaluate the exposure measured for the control group, the 95th percentile data of this group can be compared with proposed reference values in literature.

According to several authors, Al concentrations <5 μg/l (serum) and <15 μg/l (urine) can be expected in the general population (Minoia et al., 1990; Wang et al., 1991; Valkonen and Aitio, 1997; HBM-Kommission, 1998). Investigations of Fleischer and Schaller showed that the Al concentration in plasma and serum are well comparable. Therefore, the proposed value for serum should also apply for plasma (Fleischer and Schaller, 2000). Except for the sampling in 2001 (4.4 μg/l) the 95th percentiles for Al in plasma in our control group exceeded this value (1999: 13.1 μg/l; 2003: 7.6 μg/l) and showed a clear variability. To our opinion this may be ascribed to changes in the composition of the control group on the one hand, and to the increased analytical imprecision emerging at low Al plasma levels on the other hand.

Compared to plasma the 95th percentiles for Al in urine in our control group showed only minor fluctuations. The values, which were 26.8 μg/l, 28.0 μg/l and 23.2 μg/l in the years 1999, 2001 and 2003, respectively, also exceeded the proposed reference value for Al in urine. Due to the consistent information yielded from the determination of Al in plasma and urine in our control group, a slightly increased background exposure can be assumed.

As it is shown in Fig. 1 personal air sampling revealed a rather constant exposure of the welders to total dust during the study. Unlike external exposure Al concentrations in plasma and urine decreased from 1999 to 2003. Due to these results close correlations between external and internal exposure cannot be expected. In fact an explorative nonparametric correlation analysis only yielded for particular samplings a significant relationship with partly very low correlation coefficients (Table 2) indicating that internal Al exposure is not reflected well by external exposure to total dust. In the past significant correlations between external Al exposure and Al in plasma and urine have been found (Mussi et al., 1984; Sjogren et al., 1988). The lack of close correlations in our study may be caused to some extent by the fact that for assessment of external exposure the total dust concentration instead of the Al concentration in the air was measured. Therefore, the use of this parameter for estimation of external Al exposure seems questionable.

To our opinion also the use of the ambient Al concentration for the assessment of Al uptake should be considered critically. Because of the accumulation of Al in particular body compartments on the one hand and the retarded resorption of inhaled Al from the lungs on the other hand, an estimation of the actual body burden seems difficult by this parameter.

In the analysis of correlations between both markers of internal exposure also, no close relationship could be found. Significant correlations were observed for three out five annual samplings and for the averaged exposure over 5 years (Table 2). A comparable correlation between Al in serum and urine was also observed by Riihimaki et al. (2000). The closer relationship found in this study might be explained by the higher internal Al exposure of the study population compared to the welders surveyed in our study.

Regarding the results of our study, we conclude that for the assessment of occupational Al exposure an ambient monitoring of total dust or Al concentration in the air has to be regarded critically. If possible internal exposure should be determined by continuous biological monitoring. For biological monitoring we recommend the determination of Al in urine due to the higher sensitivity and robustness of this marker compared to Al in plasma. However, to correct for diuretic dilution effects the concentration of Al in urine should be evaluated related to creatinine.

Acknowledgements

We thank the German Hauptverband der gewerblichen Berufsgenossenschaften (HVBG) and Vereinigung der Metallberufsgenossenschaften (VMBG) for financial support of the study.

References

Alfrey, A.C., LeGendre, G.R., Kaehny, W.D., 1976. The dialysis encephalopathy syndrome. Possible aluminum intoxication. N. Engl. J. Med. 294, 184–188.

ATSDR (Agency for Toxic Substances and Disease Registry), 1999. Toxicological profile for aluminium (update), U.S. Department of Health and Human Services, Public Health Service.

DFG (Deutsche Forschungsgemeinschaft), 2004. Commission for the investigation of health hazards of chemical compounds in the work area. MAK and BAT values 2004 Report no. 40. Wiley–VCH, Weinheim.

Fleischer, M., Schaller, K.-H., 2000. Aluminium. In: Angerer, J., Schaller, K.-H. (Eds.), Analysis of Hazardous Substances in Biological Materials, Deutsche Forschungsgemeinschaft, vol. 7. Wiley–VCH, Weinheim.

Gitelman, H.J., 1995. Aluminum exposure and excretion. Sci. Total Environ. 163, 129–135.

Gitelman, H.J., Alderman, F.R., Kurs-Lasky, M., Rockette, H.E., 1995. Serum and urinary aluminium levels of workers in the aluminium industry. Ann. Occup. Hyg. 39, 181–191.

Hanninen, H., Matikainen, E., Kovala, T., Valkonen, S., Riihimaki, V., 1994. Internal load of aluminum and the central nervous system function of aluminum welders. Scand. J. Work Environ. Health 20, 279–285.

HBM-Kommission (Kommission “Human-Biomonitoring”), 1998. Stellungnahme der Kommission “Human-Biomonitoring” des Umweltbundeamtes—Aluminium. Bundesgesundhbl. 41, 271.

Kraus, T., Schaller, K.H., Angerer, J., Letzel, S., 2000. Aluminium dust-induced lung disease in the pyro-powder-producing industry: detection by high-resolution computed tomography. Int. Arch. Occup. Environ. Health 73, 61–64.

Letzel, S., Schaller, K.H., Hilla, W., Windorfer, K., Kraus, T., 1999. Untersuchungen zur biologischen Halbwertszeit der renalen Aluminiumausscheidung bei Aluminiumschweißern. Arbeitsmed. Sozialmed. Umweltmed. 34, 456–460.

Ljunggren, K.G., Lidums, V., Sjogren, B., 1991. Blood and urine concentrations of aluminium among workers exposed to aluminium flake powders. Br. J. Ind. Med. 48, 106–109.

Minoia, C., Sabbioni, E., Apostoli, P., Pietra, R., Pozzoli, L., Gallorini, M., Nicolaou, G., Alessio, L., Capodaglio, E., 1990. Trace element reference values in tissues from inhabitants of the European community. I. A study of 46 elements in urine, blood and serum of Italian subjects. Sci. Total Environ. 95, 89–105.

Mussi, I., Calzaferri, G., Buratti, M., Alessio, L., 1984. Behaviour of plasma and urinary aluminium levels in occupationally exposed subjects. Int. Arch. Occup. Environ. Health 54, 155–161.

Nielsen, J., Dahlqvist, M., Welinder, H., Thomassen, Y., Alexandersson, R., Skerfving, S., 1993. Small airways function in aluminium and stainless steel welders. Int. Arch. Occup. Environ. Health 65, 101–105.

Rifat, S.L., Eastwood, M.R., McLachlan, D.R., Corey, P.N., 1990. Effect of exposure of miners to aluminium powder. Lancet 336, 1162–1165.

Riihimaki, V., Hanninen, H., Akila, R., Kovala, T., Kuosma, E., Paakkulainen, H., Valkonen, S., Engstrom, B., 2000. Body burden of aluminum in relation to central nervous system function among metal inert-gas welders. Scand. J. Work Environ. Health 26, 118–130.

Schaller, K.-H., Angerer, J., Drexler, H., 2002. Quality assurance of biological monitoring in occupational and environmental medicine. J. Chromatogr. B Analyt. Technol. Biomed. Life Sci. 778, 403–417.

Sjogren, B., Elinder, C.G., Lidums, V., Chang, G., 1988. Uptake and urinary excretion of aluminum among welders. Int. Arch. Occup. Environ. Health 60, 77–79.

Sjogren, B., Iregren, A., Frech, W., Hagman, M., Johansson, L., Tesarz, M., Wennberg, A., 1996. Effects on the nervous system among welders exposed to aluminium and manganese. Occup. Environ. Med. 53, 32–40.

Taussky, H.H., 1956. A procedure increasing the specificity of the Jaffe reaction for the determination of creatine and creatinine in urine and plasma. Clin. Chim. Acta 1, 210–224.

Valkonen, S., Aitio, A., 1997. Analysis of aluminium in serum and urine for the biomonitoring of occupational exposure. Sci. Total Environ. 199, 103–110.

Wang, S.T., Pizzolato, S., Demshar, H.P., 1991. Aluminum levels in normal human serum and urine as determined by Zeeman atomic absorption spectrometry. J. Anal. Toxicol. 15, 66–70.

Yokel, R.A., McNamara, P.J., 2001. Aluminium toxicokinetics: an updated minireview. Pharmacol. Toxicol. 88, 159–167.