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Exposure and urinary excretion of aluminum during welding

by Bengt Sjögren, MD, Vitauts Lidums, Marianne Håkansson, Lars Hedström, MSc¹

SJÖGREN B, LIDUMS V, HÅKANSSON M, HEDSTRÖM L. Exposure and urinary excretion of aluminum during welding. *Scand J Work Environ Health* 11 (1985) 39–43. The exposure and urinary excretion of aluminum was studied among three previously unexposed volunteers and six welders exposed to welding fumes containing aluminum. The aluminum concentrations in air and urine were determined. The urinary aluminum concentrations rose rapidly in volunteers exposed only for 1 d and returned to the preexposure levels with an estimated half-time of about 8 h. The welders were monitored for one workweek. During the subsequent weekend a decrease in the urinary concentrations occurred in the three welders exposed for two years or less, but such a decrease was not observed among welders exposed for more than 15 years. The urinary concentrations of aluminum were dependent partly on the level of current exposure and partly on the duration of exposure. The data suggest that welders exposed to welding fumes containing aluminum may retain some of the inhaled metal fume for extended periods of time.

Key terms: air concentrations, urinary concentrations of aluminum.

Aluminum has been the focus of considerable interest toxicologically ever since encephalopathy among dialysis patients was associated with it. A relationship has been observed between the cumulative aluminum exposure of dialysis patients and the attack rate of encephalopathy (11). In a previous study of workers exposed to aluminum in different types of industries, welders were found to be one of the most exposed groups, with significantly higher blood concentrations of aluminum than unexposed referents. However, their aluminum blood levels were lower than those of mentally healthy dialysis patients (12).

The purpose of the present study was to investigate exposure to and excretion of aluminum among subjects exposed to welding fumes containing aluminum. Exposure was assessed by determinations of the aluminum content in the air and by information on exposure periods. Excretion was determined from the urinary concentrations of aluminum.

Subjects

Three male volunteers, previously unexposed to aluminum, were exposed to aluminum-containing welding fumes during one workday. Urine samples were taken before and during the exposure and 4 d thereafter. The mean age of the volunteers was 40 (range 27–58) years.

Three male welders exposed to aluminum-containing welding fumes for relatively short periods (1 month, 3 months, and 2 years) and another three male welders exposed for relatively long periods (18,

19, and 20 years) participated in the study. Air and urine sampling was performed during one week. All welders in the first group were 19 years of age, and the mean age of the welders in the second group was 47 (range 40–56) years.

Aluminum welding was performed with the gas-shielded technique. Metal inert-gas (MIG) welding generates more particles than tungsten inert-gas (TIG) welding (13). All participants were exposed to fumes from the MIG welding of aluminum. One welder with long-term exposure worked with the MIG method as well as with the TIG method.

None of the exposed subjects had taken any aluminum-containing drugs, such as antacids, during the previous six months.

Methods

Air samples

Samples of the welding fume were collected on cellulose ester membrane filters (Millipore filter AAWP, diameter 37 mm, mean pore size 0.8 μ m) in the breathing zone of the subjects. The amounts of welding fume particles were determined gravimetrically, and the particle concentrations in air were calculated by means of the air volumes sampled.

The aluminum content on the filters was determined by electrothermal atomic absorption spectrometry with a Perkin-Elmer 403 instrument (12). Reagents, concentrated sulfuric acid, and 40 % hydrofluoric acid were of the quality "Suprapur." A solution of ammonia (10 M) was produced by isothermal distillation (15).

The filters were moistened with 0.15 ml of ammonia (10 M) and transferred to platinum crucibles. Sulfuric acid (0.05 ml) (1 + 1) was added, and the crucibles were placed in a special holder of acidproof

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steel, which was provided with an arrangement for the influx of filtered air. The holder was placed in an electric furnace, and the temperature was gradually raised to 600°C.

Hydrofluoric acid (0.2 ml), water (about 0.5 ml), and sulfuric acid (0.2 ml) (1 + 1) were added to the residue in the crucibles. The mixture was warmed until heavy fumes evolved. After the mixture cooled, some milliliters of water were added, and the mixture was warmed until a clear solution was obtained. The solution was diluted to a suitable volume (not more than 140 µg of aluminum per liter) in volumetric flasks of polypropylene.

Sample aliquots of 25 µl were introduced to the graphite furnace and analyzed with the following program: drying, 100°C, for 30 s; ashing, 1 250°C, for 40 s; atomizing, 2 680°C, for 7 s and at gas stop. Background correction was not needed.

The results were calculated by means of a standard curve. Blanks, consisting of unused filters and all added reagents, were analyzed in each series. The detection limit was 1 µg/filter.

Urine samples

Urine was voided into 500-ml bottles of polyethylene with lids of the same material. The bottles and lids were carefully cleaned, soaked in 5 % RBS-25 solution (detergent from Robert Boygrafs no 25, Belgium) for at least 5 h and in 1 % ethylenediaminetetraacetate solution overnight, and carefully rinsed with ion-free water.

At the laboratory the urine samples were divided into two portions, a small amount being preserved for the assessment of creatinine. To the remainder 1.5 % of 9 M sulfuric acid was added. The samples were stored at -20°C until analysis. Aluminum was determined by electrothermal atomic absorption spectrometry in a previously described way (12). The detection limit was 3 µg/l (0.1 µmol/l).

Creatinine was determined by a colorimetric method on a reaction rate analyzer (LKB 8600).

Statistical methods

Multiple regression analysis was used to study the relationship between the urinary concentration of aluminum, the air concentration of aluminum, and exposure time.

Results

Altogether 57 air samples and 204 urine samples were studied.

The mean aluminum content of the welding fume was 39 % with a standard deviation of 4 %. The welding fume was assumed to consist mainly of aluminum oxide (Al₂O₃). The welders were exposed to a mean 8-h time-weighted average aluminum level of 2.4 mg/m³. The exposure varied between 0.3 and 10.2 mg/m³.

Air and urine concentrations for one individual representing each of the exposed groups (volunteers and short-term and long-term exposed welders) are presented in figures 1-3.

The urinary concentrations of aluminum among the previously unexposed volunteers increased from less than 3 µg/l (0.1 µmol/l) before exposure to between 15 and 414 µg/l (0.6 and 15 µmol/l). Within a few days the urinary concentrations had decreased to the preexposure levels. The half-time of the first phase of excretion was calculated to be about 8 h for these three volunteers.

Figure 1. Concentrations of aluminum in air and urine in a previously unexposed volunteer (BS).

Figure 2. Concentrations of aluminum in air and urine in a welder (JH) exposed for one month.

Figure 3. Concentrations of aluminum in air and urine in a welder (BJ) exposed for 19 years.

Table 1. Mean urinary concentrations of aluminum during exposure and nonexposure. Urine samples from Monday morning and unexposed days are referred to as the period of nonexposure.

Welder	Exposure time (years)	Mean concentrations during exposure				Mean concentrations during nonexposure			
		µg/g	µmol/l	µg/g creatinine	mmol/mol creatinine	µg/l	µmol/l	µg/g creatinine	mmol/mol creatinine
JH	0.08	44	1.6	31	0.13	16	0.6	12	0.05
MJ	0.25	9	0.3	6	0.03	5	0.2	3	0.01
TG	2	43	1.6	36	0.15	44	1.6	17	0.07
BI	18	355	13.2	352	1.48	341	12.6	400	1.68
BJ	19	321	11.9	251	1.05	366	13.6	240	1.01
NS	20	295	10.9	203	0.85	296	11.0	217	0.91

One welder with short-term exposure could only be followed with air measurements for 1 d due to tonsillitis, and his urine concentrations decreased after some days to the reference level below 3 µg/l (0.1 µmol/l). Among the other short-term exposed welders the mean urinary concentrations of aluminum decreased during the weekend (table 1). For the welder exposed for two years this decrease was observed only when the urinary concentrations were adjusted to creatinine. Among the long-term exposed welders the urinary concentrations did not change despite the cessation of exposure. The variation of the urinary concentrations which were observed during the unexposed weekend could not be explained by any regular circadian variation.

The following relationship between the urinary concentration of aluminum, the air concentration,

and the exposure time was calculated according to a multiple linear regression model as follows:

$$Y_1 = 42.0 X_1 + 7.42 X_2 + 6.60 \quad R^2 = 85.5 \%$$

$$R = 0.92$$

$$Y_2 = 1.56 X_1 + 0.275 X_2 + 0.245 \quad N = 9$$

where

Y_1 = the urinary concentration of aluminum (µg/l) after the shift the last day in an exposure period of 1 to 5 d. The collected urine had been generated during the last 3–4 h of the work-shift.

Y_2 = the same as Y_1 but expressed as micromoles per liter.

X_1 = the 8-h time-weighted average of aluminum in air (mg/m^3) during an exposure period of 1 to 5 workdays.

X_2 = the number of years the person had been exposed to aluminum welding fumes.

R^2 = the percentage of explained variance for the Y variable.

R = the coefficient of correlation between the estimated and observed value.

N = the number of individuals.

If the urinary excretion is calculated as micrograms of aluminum per hour after the shift the last day in an exposure period of 1 to 5 d (Y_3) or expressed as nanomoles per hour (Y_4), the equations will be:

$$\begin{aligned} Y_3 &= 1.45 X_1 + 0.60 X_2 + 1.79 & R^2 &= 78.9 \% \\ & & R &= 0.89 \\ Y_4 &= 53.7 X_1 + 22.2 X_2 + 66.3 & N &= 9 \end{aligned}$$

If the urinary excretion is calculated as micrograms of aluminum per gram of creatinine after the shift the last day in an exposure period of 1 to 5 d (Y_5) or expressed as micromoles per mole of creatinine (Y_6), the equations will be [Due to missing values for the three volunteers the number of observations are reduced to six.]:

$$\begin{aligned} Y_5 &= 7.96 X_1 + 10.5 X_2 + 14.2 & R^2 &= 99.6 \% \\ & & R &= 0.99 \\ Y_6 &= 33.4 X_1 + 44.0 X_2 + 59.5 & N &= 6 \end{aligned}$$

Because of lack of knowledge of the distribution of the aluminum concentrations in urine for different combinations of concentrations in air and length of exposure, the regression equations should only be regarded as a fit of the data to planes in a three-dimensional space according to the method of least squares.

Discussion

The following discussion is structured according to reference levels and the absorption, distribution, and excretion of aluminum.

Urinary concentrations of aluminum among industrially unexposed persons have been reported to vary from 2 $\mu\text{g}/\text{l}$ (0.07 $\mu\text{mol}/\text{l}$) to about 30 $\mu\text{g}/\text{l}$ (1 $\mu\text{mol}/\text{l}$) (4, 10, 12, 14). The levels of aluminum in urine might be influenced by contamination. In this study pretreatment of the samples and the use of additives were avoided so far as possible because of the risks of contamination at such steps in the analysis. All three volunteers in this study had urinary concentrations of aluminum below 3 $\mu\text{g}/\text{l}$ (0.1 $\mu\text{mol}/\text{l}$) prior to the welding fume exposure.

Food and certain drugs contain aluminum, which is absorbed via the gastrointestinal tract. The normal daily intake is calculated to be about 7 mg per person (5). Cooking in aluminum vessels slightly increases the level of aluminum in food. The total increase might be 29 mg per portion when acid food, such as rhubarb, is prepared. In most cases, however, the increase in aluminum levels produced by cooking in aluminum saucepans does not influence the daily intake (8). The urinary concentrations have been reported to increase about threefold when the total amount of aluminum in food is increased from 5 mg/d to 125 mg/d (3). A total intake of 2.2 g of aluminum-containing drugs during a 3-d period increased the urinary aluminum concentration by 4–50 times, depending on the chemical composition of the drug (4). However, none of the participants in the present study had taken any aluminum-containing drugs within the previous six months.

The particles generated in electric-arc welding are generally respirable (13). The mass median diameter is about 0.4 μm in the MIG welding of aluminum and somewhat smaller in TIG welding (7). The rapid increase of urinary aluminum among persons not previously exposed to aluminum indicates significant pulmonary absorption. The urine levels of the short-term exposed subjects were in agreement with recently published results from Italian welders (10). Among the long-term exposed welders the urinary concentrations were about 100 times the level of the unexposed subjects. According to our previous data (12) the corresponding increase in the blood concentrations of aluminum would be on the order of 10–20 times the normal level.

Among industrially unexposed persons the lung tissue has been reported to have higher concentrations of aluminum than any other organ, expressed as the amount of aluminum per amount of dry weight of tissue. The lung is the only organ in which the concentrations of aluminum increase with age (2). Dialysis patients are exposed to aluminum via the gastrointestinal tract and via the water used for dialysis. In dialysis patients without signs of encephalopathy the mean concentrations of aluminum increase to 1.6 times the normal level in the lung tissues, 64 times the normal level in the spleen, 40 times the normal level in the liver, 35 times the normal level in the bones, and 4 times the normal level in the gray matter of the brain (2). There is only one case of encephalopathy which has been associated with occupational exposure to aluminum. This man was exposed to aluminum powder for 13 years, and his lungs had 18 times the normal amount of aluminum and his liver had 120 times the normal level (9). These results indicate that several organs, eg, the lungs, liver, spleen, and bones, can store aluminum after long-term exposure.

Among the previously unexposed volunteers the urine concentrations decreased to reach their pre-exposure level a few days after exposure. The cal-

culated half-time was about 8 h. Human studies of excretion from a single exposure have not been reported earlier, but dogs excreted one-third of a given intravenous dose of aluminum chloride within 2 h (6). In our study the volunteers performed very light physical work during exposure. If their pulmonary ventilation was estimated to be about 20 l/min (1), the urinary excretion of aluminum was 0.1–0.3 % of the total inhaled dose within the next 2 d after exposure. Thus a very small proportion of the inhaled dose was excreted in the urine among these subjects. Among the short-term exposed welders the urinary concentrations of aluminum decreased during the weekend, but no such decrease was observed among the long-term exposed welders. In four Italian welders exposed for six months, the urinary levels of aluminum decreased to 10–20 % two weeks after exposure (10). These differences in excretion between short-term and long-term exposed welders might be explained by a slowly releasing compartment being more influential to the urinary excretion of aluminum in the workers exposed long-term.

In our previous study a linear relationship was observed between the exposure time and the urinary concentrations of aluminum in welders (12). According to the present study recent exposure, as well as the number of years of exposure, were important for the urinary concentrations of aluminum, and this finding suggests at least a two-compartment excretion model.

Aluminum is inhaled when the metal is welded. The present study demonstrates that one part is excreted rapidly in the urine. Another part is excreted slowly, perhaps after being redistributed from the lungs to some other organ. Further studies are needed for a better understanding of the absorption, distribution, and excretion of aluminum in the human body.

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