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Small airways function in aluminium and stainless steel welders

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Summary. The effect of welding fumes on small airways was studied in 25 male subjects who welded in aluminium (Al) and to some extent also in stainless steel (SS). Despite a low exposure to welding fumes as compared to the permissible exposure limits, excretion of Al in urine was found to be increased in all subjects (median value: 0.29 mmol/mol creatinine on Friday afternoon, as compared to an upper reference level of 0.10 mmol/mol creatinine). In addition, the welders displayed increased prevalences of work-related eye and airways (pharyngitis and non-specific bronchial hyperreactivity) symptoms, as compared to 25 matched controls. Short-term welders (≤ 2.5 years) had more symptoms related to the upper airways than did long-term welders, which may indicate a selection. Spirometry, closing volume and volume of trapped gas (VTG) did not deviate. However, after methacholine inhalation, the long-term welders had a significantly steeper slope of the alveolar plateau on the single-breath nitrogen wash-out test, and a slight increase in VTG, as compared to the short-term welders and the controls. These findings may indicate a welding fume-induced increase in the reactivity of the small airways. Because Al welding was far more frequent than SS welding, an association with the former seems likely.

Key words: Volume of trapped gas – Closing, volume – Hyperreactivity – Immunoglobulin A – Aluminium

Introduction

In the welding process, a number of air pollutants such as ozone, nitrogen oxides and respirable particles are produced. Since many of these components have irritating properties [28], one should expect development of epithelial damage at different levels of the respiratory tract at high exposure. Accordingly, numerous studies have been carried out on groups of welders with regard to non-malignant respiratory disease. Although the results have been conflicting, some studies have shown that welding operations may cause airways symptoms, especially cough and phlegm production [7, 18, 28].

Lung function testing has also yielded conflicting results. In some studies, a reduction in lung function has been shown by the use of conventional spirometry, while in others no reduction has been demonstrated. However, small airways may be more sensitive. Thus, a reduction in lung function has been found by the use of methods to test small airways function (expiratory flow rates and closing volume) [10–12, 14–16, 20, 23].

Little is known about exposure to welding fumes and airways hyperreactivity. However, in one study, welders showed slightly greater bronchial reactivity than control subjects [19], although only large airways were tested (by forced expiratory volume in 1 s: FEV_1). A newly published study in non-smoking shipyard arc welders showed an increase in the volume of trapped gas in the lungs (VTG) after methacholine inhalation, indicating hyperreactivity of the small airways [11].

In this field study, we have explored the hypothesis that workers welding in aluminium (Al) and stainless steel (SS) suffer from hyperreactivity in the small airways, as tested by two different methods before and after methacholine inhalation.

Materials and methods

The plant. The plant produced tanks (volume 20–50 m^3) for trucks. Most of the tanks were made from Al, some from SS. The welders used the metal inert gas and the tungsten inert gas methods, with argon as the shielding gas. Manual metal arc (MMA) welding was also applied for the SS tanks. Tanks in Al and SS were produced in different workshops, so the welders were to a certain degree specialized in either Al or SS welding. It was estimated by the company that welding accounted for approximately 40% of welders' working time. Grinding was another dust-generating work task. All welders had access to local exhaust ventilation systems, and some of them had personal respiratory protective devices for use when welding inside tanks.

Air sampling and anaylsis. Thirty-six samples of welding fumes were collected on membrane filters (Millipore AAWP 0.8 μm pore

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size; diameter 37 mm) in the breathing zones of 19 welders. The total sampling time was 131 h. The workshift time-weighted concentration of total dust was determined gravimetrically for all welders. The concentrations of Al in air were meausred for 14 Al welders by inductively coupled argon plasma atomic emission spectrometry after sample digestion in hydrofluoric acid. The concentrations of water-soluble chromium in air (Cr_s) were measured for five SS welders by atomic absorption spectrometry after extraction [31].

The levels of ozone and nitrous oxides were determined through random sampling by Dräger tubes (Ozone 0.05/a, Nitros Fumes 0.5/a; Drägerwerk AG, Lübeck, Germany) in the breathing zone of all welders.

Subjects. Twenty-five male subjects who had regularly welded, mainly in Al and to a lesser extent in SS, were studied. Their median exposure time was 2.5 (0.1–13) years. According to their time of welding in Al and SS, welders were divided into two groups: long-term (above the median time of exposure) and short-term (below the median time of exposure) welders.

The control group consisted of 25 males employed in a warehouse for wines. They were not exposed to any significant extent to potentially harmful dust or smoke. Exposed workers and controls were matched for age and smoking habits. The control subjects had no previously known lung disease.

Medical examination. The subjects were interviewed by a physician (J.N.) about their occupational and medical history, including symptoms of conjunctivitis, rhinitis, pharyngitis and asthma (attacks of wheezing breath, dyspnoea and cough), and whether the symptoms were work related (association with work, better during weekends and holidays [6]). Furthermore, the interview included questions about chronic bronchitis (daily productive cough more than 3 months a year during the last 2 years [2]) as well as questions about non-specific bronchial hyperreactivity (attacks of dyspnoea, wheezing and/or chest tightness upon exposure to strong odours, cold, dust or exercise) and attacks of "flu-like" symptoms after work. In addition, information obtained included atopic symptoms during childhood and adolescence (allergic rhinitis, asthma, urticaria or atopic eczema) and smoking habits (modified from Rose and Blackburn [25]). A physical examination was performed, including pulmonary auscultation.

Skin-prick test and laboratory analysis. Skin-prick test with 13 common allergens (Allergologisk laboratorium, Copenhagen, Denmark) was performed. A prick test was scored as positive when the area of the wheal was at least half that induced by histamine (1 mg/ml).

Blood serum was analysed for total serum IgE (S-IgE) using the PRIST-test (Pharmacia Diagnostics AB, Uppsala, Sweden) for IgG, IgA and IgM, as well as for α_1-antitrypsin, orosomucoid, haptoglobin and ceruloplasmin.

Aluminium in urine. Urine samples from 19 welders were collected in acid-washed polyethylene bottles for the measurement of Al in urine (U-Al) by electrothermal atomic absorption spectrometry [22]. A sample was collected from each welder on Friday after the workshift (3 p.m.) and on the following Monday morning before work (6 a.m.). An upper reference limit was assessed from a study in farmers from the same area but without occupational exposure to aluminium.

Lung function tests. Vital capacity (VC), FEV_1 and the maximum expiratory flow at 75% of forced expiratory volume (MEF_{25}) were measured by a Vicatest-5 spirometer (Siemens-Elema, Mijnhardt, Holland) according to the guidelines of the American Thoracic Society [6].

The small airways were studied by two methods: (1) determination of VTG in the lungs, and (2) determination of closing volume (CV); in both cases the airways were studied before and after bronchial provocation with methacholine.

VTG was measured by a nitrogen wash-out technique, as described earlier [9], and expressed as a percentage of total lung capacity (TLC; VTG_0/TLC). Then, for a period of 2 min, the subjects inhaled an aerosol of methacholine chloride dissolved in saline, at a concentration of 0.01% [9]. If, after further spirometry, FEV_1 did not decrease by more than 15% of the baseline value, a methacholine chloride solution of 0.1% was inhaled. Following this procedure, the VTG measurement was repeated.

CV and the slope of the alveolar plateau (phase III) was measured by a single-breath nitrogen wash-out test modified after Anthonisen et al. [1, 3]. CV was expressed in percentage of VC (CV/VC). The CV measurements were then repeated after inhalation of a 0.1% solution of methacholine chloride, as described above.

All volumes were expressed at body temperature and pressure and water vapour saturated.

Statistical methods. For comparison of distributions between different groups, the Mann-Whitney U-test was used. For comparison of symptoms in the exposed groups and the control groups, Fisher's test was applied. Comparisons of differences between pairs were made by the Wilcoxon matched-pairs signed-rank test. All stated P values involved two-tailed analysis. Differences were considered to be statistically significant at $P < 0.05$.

Results

Exposure

The median time-weighted air concentrations of total dust, Al and Cr_s were 2.8, 1.4 and 0.008 mg/m^3 respectively (Table 1). The time-weighted air concentrations of gases were low: ozone < 0.01 ppm and nitrous oxides < 0.1 ppm.

The U-Al in the welders was elevated compared to the upper referent level of 0.10 mmol Al/mol creatinine for both Friday after work and Monday morning before work (Table 1). There was a numerical decrease in U-Al from Friday to Monday, but this difference was not statistically significant.

Symptoms

The welders in the short-term group were younger than the welders in the long-term group (Table 2). Further-

Table 1. Air levels of total dust, aluminium (Al), water-soluble chromium (Cr_s), ozone, nitrous oxides (NO_x) and urinary levels of Al on Friday after work and on the following Monday morning before work, in 19 Al and SS welders

	Samples (*n*)	Median	Range	Welder group
Air levels				
Total dust (mg/m^3)	19	2.8	0.7–19	All
Ozone (ppm)	33	<0.01	<0.01–0.7	All
NO_x (ppm)	13	<0.1	<0.1–0.2	All
Cr_s (μg/m^3)	5	8	5–380	SS
Al (mg/m^3)	14	1.4	0.2–6.1	Al
Urinary Al levels (mmol/mol creatinine)[a]				
Friday	19	0.29	0.08–1.1	Al
Monday	19	0.16	0.07–0.16	Al

[a] Upper reference limit: 0.10 mmol/mol creatinine

Table 2. Exposure, smoking, work-related symptoms, non-specific bronchial hyperreactivity, chronic bronchitis, spirometry, closing volume, and volume of trapped gas in short- and long-term Al and SS welders and controls. For abbreviations see text (Materials and methods)

Parameter	Welders		Controls
	Long-term (> 2.5 years)	Short-term (≤ 2.5 years)	
Number	12	13	25
Age (years; median, range)	50 (34–62)	35 (19–49)	44 (24–59)
Time of welding (Al and SS; median, range)	6.0 (3–13)	0.7 (0.1–2.5)	0
Smokers (*n*, %)	1 (8)[a]	7 (54)	7 (28)
Symptoms (*n*, %)			
Conjunctivitis	3 (25)[b]	3 (23)[c]	0[d]
Nasal secretion	2 (17)	2 (15)	0
Nasal congestion	1 (8)	4 (31)[c]	0
Pharyngitis	2 (17)	5 (38)[c]	0[d]
Asthma	1 (8)	1 (8)	0
Dry cough	0	2 (15)	0
Non-specific bronchial hyperreactivity	5 (42)[b]	4 (31)[c]	1 (4)[d]
Chronic bronchitis	1 (8)	2 (15)	1 (4)
Spirometry (median, range)			
VC (l)	4.9 (3.5–6.4)	5.7 (3.8–6.8)	5.0 (3.5–7.0)
FEV_1 (l)	3.9 (2.5–4.4)	4.6 (2.8–5.1)	4.1 (2.9–5.3)
MEF_{25} ($l \times s^{-1}$)	1.5 (1.0–3.0)	2.0 (1.0–3.3)	1.8 (0.7–3.6)
Closing volume (median, range)			
CV/VC (%)			
Baseline	28 (14–37)	20 (11–37)	24 (7.9–40)
After methacholine	23 (12–40)	24 (11–40)	26 (13–44)
Phase III (% $N_2 \times l^{-1}$)			
Baseline	0.8 (0.6–1.9)	0.6 (0.5–1.5)	0.8 (0.5–2.2)
After methacholine	1.1 (0.9–2.2)[a, b]	0.7 (0.3–1.0)	0.8 (0.3–1.6)[e]
Volume of trapped gas			
VTG/TLC (%; median, range)			
Baseline	1.1 (0.6–2.0)	1.4 (0.6–2.1)	1.8 (0.6–7.2)
After methacholine	1.5 (0.9–9.6)	1.6 (0.9–5.2)	2.0 (0.8–8.0)

[a] Long-term welders different from short-term welders, $P < 0.05$
[b] Long-term welders different from controls, $P < 0.05$
[c] Short-term welders different from controls, $P < 0.05$
[d] Controls different from all welders, $P < 0.05$
[e] Controls different from all welders, $P = 0.01$

more, the control group and the long-term welder group contained less smokers than the short-term welder group ($P < 0.05$). Thus, when these groups were studied separately, correction for age and smoking habits was made. There were no differences with regard to height or atopic status.

The total group of exposed subjects had significantly more work-related eye symptoms than the controls, but there was no difference between short-term and long-term welders (Table 2). There were no differences between the groups with regard to symptoms of nasal secretion, while the short-term welders displayed significantly more symptoms of nasal congestion than the controls. The total group of welders had a significantly greater frequency of work-related pharyngitis than the controls. When the welders in the two exposure groups were studied separately, only the short-term ones were significantly different from the controls.

Two welders suffered from work-related asthma (one in each exposure group), and two short-term welders had dry cough related to work (Table 2). Compared with the controls, the welders in both exposure groups more often showed symptoms of non-specific bronchial hyperreactivity. No difference was found with regard to chronic bronchitis. Nobody had suffered from attacks of "flu-like" symptoms after work.

Serum analysis

There were no differences between the welders and the controls with regard to total serum immunoglobulins of types IgE, IgG or IgM. However, the welders displayed

significantly lower levels of IgA than the control subjects (1.6 vs 2.1; $P < 0.001$). No differences were observed between the groups for α_1-antitrypsin, orosomucoid, haptoglobin and ceruloplasmin.

Lung function tests

Spirometry. There were no differences between the groups for spirometry parameters (Table 2).

Closing volume. No differences between the groups were recorded for CV/VC%, either before or after inhalation of methacholine (Table 2). Moreover, no differences were obtained between the groups for phase III readings before methacholine. However, phase III level after methacholine was found to be significantly ($P = 0.01$) increased in the welders compared to the controls. Furthermore, following methacholine inhalation, long-term welders had significantly higher phase III readings than short-term welders and controls.

Volume of trapped gas. Although VTG was found to be the lowest in the long-term welders and highest in the controls, these differences were not statistically significant (Table 2). Following methacholine inhalation, VTG increased in all groups, with the highest, though not significant ($P = 0.05$ compared to controls), increase observed in the long-term group.

Discussion

The welders were exposed to dust and gases at levels generally not exceeding the current Swedish standards [29]. However, peaks exceeding the standard were sometimes found for ozone. The exposure level was in the same order of magnitude as found in other studies of welding dust [30, 33], whereas the levels of ozone and nitrous oxides were surprisingly low [30]. Since the local exhaust-ventilation system has been improved in recent years, it is possible that the exposure levels had been higher in the past.

The welders showed an increase in U-Al, indicating a significant uptake. Interestingly, there was no significant decrease during the weekend. This is in accordance with previous observations reporting increased excretion even in retired welders [27], as well as in workers retired from exposure to aluminium-flake powders [17]. These observations suggest the presence of an Al compartment in the body with a slow elimination rate.

Welders in the present study had frequent work-related eye and upper airway symptoms, in accordance with previous studies [13, 32, 33]. The prevalence of upper airway symptoms was higher in short-term than in long-term welders. This may be due to adaptation or to a healthy worker selection. Interestingly, the smoking prevalence was low in the long-term group. Thus, the combination of smoking and welding fume exposure could have accentuated the selection process.

The welders also showed an increased prevalence of non-specific symptoms suggesting bronchial hyperreactivity. The prevalence was even higher than in workers exposed to polyvinyl chloride thermal degradation products [21]. However, only two cases of clear-cut work-related asthma were recorded. Furthermore, the prevalence of chronic bronchitis was not increased. A higher frequency of chronic bronchitis in welders was found by Sjögren et al. [26], but not by Mur et al. [19] and Kalliomäki et al. [13], whereas in the latter study there was a higher frequency of other respiratory symptoms. The bronchitis was related to ozone exposure in welders working with Al [26]. Thus the lack of such an effect on the bronchi in the present study could be due to the relatively low exposure to welding fumes.

No significant differences were found between the different groups of welders and controls in the simple spirometry and the various more advanced lung function tests performed before methacholine inhalation. Using CV examinations, Oxhöj et al. [23] found an effect on the small airways in non-smoking shipyard welders who, however, had a heavier exposure than the welders in the present study. Kalliomäki et al. [13] also found an effect in SS welders using the same technique, but the effect was associated with the MMA welding method, which was used to only a small extent by our subjects.

However, after inhalation of methacholine, the long-term welders had a somewhat steeper alveolar plateau (phase III) than either the short-term welders or the controls. When phase III is increased the lungs are more likely to have an uneven gas distribution. In addition, following methacholine administration, VTG in the long-term welders was found to be increased more than in the controls. These observations indicate that exposure to welding fumes may induce a slight increase in the reactivity of the small airways. Recently, similar observations were recorded in shipyard welders [11].

The small airways effect may be due to inflammation of the airways, as has already been suggested after exposure to ozone [4] or isocyanates [5]. Because the present welders were exposed to both Al and SS welding fumes, we cannot with certainty ascribe the effect to either of these operations. However, in light of the fact that Al welding was far more frequent than SS welding, an association with the former seems likely.

Pierre et al. [24] found a decrease in serum ceruloplasmin in Al welders. We could not confirm this finding. However, we found that welders had a significant decrease in serum IgA as compared to the controls. A similar effect was found in smokers compared to non-smokers in a study of Gerrard et al. [8]. The reason for this observation remains unclear.

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