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Prevalence of respiratory disorders among aluminium potroom workers in relation to exposure to fluoride

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Abstract

In a survey of 370 aluminium potroom workers in western Norway, bronchial responsiveness, lung function, and respiratory symptoms were studied in relation to occupational exposure to air contaminants in the potroom. Increased prevalences of respiratory symptoms, work related asthmatic symptoms, and abnormal lung function were found in subjects exposed to total fluorides above 0·5 mg/m^3 when compared with workers exposed to total fluorides at concentrations of less than 0·5 mg/m^3. No significant association between bronchial responsiveness and exposure to fluoride was found and the prevalence of respiratory symptoms was independent of the degree of dust exposure. These findings indicate that work related asthmatic symptoms in potroom workers may be related to exposure to fluorides.

Aluminium is produced by electrolysis of alumina (Al_2O_3). During the process alumina is reduced by carbon (from the anode) to form CO_2 and aluminium. The alumina is dissolved in cryolite (Na_3AlF_6). Two different technologies are used ("prebaked" and "Soderberg"). The air contaminants emitted from the electrolytic pots of these two types of technology are principally the same. Various fluorides escape to the potroom atmosphere. Also, the anode is contaminated by sulphur, which is oxidised to SO_2 and emitted into the workplace air. The potroom air contains dust mainly composed of alumina, aluminium trifluoride, and cryolite as well as minor amounts of metals such as nickel, chromium, and vanadium.[1 2] Thus the potroom atmosphere contains many compounds that are potentially harmful to the respiratory tree.

Asthma among aluminium potroom workers has been reported in several studies[3–7] although the causative agents are unknown. Few studies exist in which the levels of pollution in the potrooms have been related to respiratory effects.[8] In a cross sectional study of respiratory symptoms among potroom workers in seven Norwegian aluminium plants a positive association between exposure to fluoride and work related asthmatic symptoms was found among pot operators (490 subjects) but this association was not found in the total population of 1679 workers.[9] The lack of association in the total workforce could arise from incorrect job classification between plants that would dilute the association.

The objective of the present cross sectional study was to examine if the association between work related asthmatic symptoms and exposure to fluoride was reproducible in the total workforce in one plant and if any association existed between exposure to fluoride, and methacholine responsiveness and lung function.

Methods

STUDY POPULATION

The survey was carried out in an aluminium smelter in western Norway. The plant was situated in a small town of 6500 inhabitants and employed 50% of the workforce in the municipality. The study population consisted of all the subjects (380) employed in the potrooms at the plant. Of those, 370 (97%; 39 women and 331 men) participated in the survey. The median age was 32·8 (range 18·5–66·5) (table 1). Bronchial challenge was carried out on 337 subjects and four subjects were excluded from challenge because of low forced expiratory volume in one second (FEV_1 < 60% of predicted). The remaining 29 workers refused to participate in the bronchial challenge. All subjects gave their informed consent to take part in the study, which had approval from the ethics committee.

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Table 1 Distribution of sex, smoking habits, atopy, familial asthma, and age according to degree of exposure to fluoride and dust

	Total fluorides $< 0{\cdot}5$ *mg/m³*	*Total fluorides* $\geq 0{\cdot}5$ *mg/m³*	*Dust* $< 0{\cdot}5$ *mg/m³*	*Dust* $\geq 0{\cdot}5$ *mg/m³*
Sex:				
No of men (No (%))	192 (91·9)	139 (86·3)	155 (91·2)	176 (88·0)
No of women (No (%))	17 (8·1)	22 (13·7)	15 (8·8)	24 (12·0)
Smoking habits:				
Current smokers (No (%))	132 (63·2)	117 (72·7)	112 (65·9)	137 (68·5)
Ex-smokers (No (%))	19 (9·1)	13 (9·1)	17 (10·0)	15 (7·5)
Never smokers (No (%))	58 (27·8)	31 (19·3)	41 (24·1)	48 (24·0)
Atopy:				
Negative (No (%))	166 (79·4)	112 (69·6)	134 (78·8)	144 (72·0)
Minor reaction (No (%))	25 (12·0)	22 (13·7)	20 (11·8)	27 (13·5)
Positive reaction (No (%))	18 (8·6)	27 (16·8)	16 (9·4)	29 (14·5)
Familial asthma (yes) (No (%))	34 (16·3)	33 (20·5)	34 (20·0)	33 (16·5)
Age:				
Mean	37·3	33·2	39·1	32·5
Median	36·7	28·6	38·5	28·2
Lower quartile	26·8	22·8	29·0	22·3
Upper quartile	46·3	42·1	46·3	41·1

QUESTIONNAIRE

All subjects completed a self administered, validated questionnaire that has been discussed in detail elsewhere.[10] Information about respiratory symptoms (dyspnoea, wheeze, and cough) within the last year, familial asthma, smoking habits, years of employment in potrooms, and use of respiratory protection mask were recorded. Those who reported dyspnoea and wheeze, or cough were interviewed by trained interviewers using a questionnaire based on the British Medical Research Council questionnaire supplemented by questions concerning the relation between work and symptoms and whether symptoms improved on rest days and during vacations. This procedure has also been described by Kongerud and coworkers.[10] Work related asthmatic symptoms (WASTH) were defined as dyspnoea and wheezing apart from colds with improvement on rest days or during vacations and absence of pre-employment asthma.

LUNG FUNCTION TESTS

Baseline FEV_1 was measured with a dry bellow spirometer (Jones Pulmonaire). The FEV_1 was taken as the highest value from the first three technically satisfactory readings (the difference between the best and the next best value should not exceed 0·3 l). The predicted spirometric values were taken from a survey of an urban symptom free population that consisted of 65% smokers.[11] Spirometry was expressed as standardised residuals of FEV_1 ($SFEV_1$) calculated as the difference between recorded FEV_1 and predicted FEV_1 divided by the residual standard deviation (SD) taken from the regression equation used to predict lung function.[11] Abnormal spirometry was defined as $SFEV_1$ less than or equal to $-1{\cdot}5$.

Methacholine challenge was carried out by a shortened version of the method described by Cockroft *et al.*[12] Briefly, during two minutes of tidal breathing the subjects inhaled methacholine solution increasing from 2·0 mg/ml (0·125 mg/ml if asthma was suspected or if FEV_1 was less than 80% of predicted) to 32·0 mg/ml. If FEV_1 decreased by more than 10% from one concentration to the next, or FEV_1 was less than 85% of the pre-test value, a doubling dose was given. Otherwise, a fourfold increment of the dose was given. The concentration required to produce a 20% fall in FEV_1 (PC_{20}) was taken from the log dose-response curve by linear interpolation of the last two points. Bronchial hyperresponsiveness (BHR) was defined as PC_{20} less than or equal to 8 mg/ml. Those who had PC_{20} less than or equal to 32 mg/ml were classified as responders whereas a fall of FEV_1 of less than 20% of the pre-test value at 32 mg/ml was regarded as normal bronchial responsiveness.

SKIN TESTS

Skin prick tests were performed with five common aeroallergens—namely, dog epithelium, common silver birch, timothy grass, mugwort, and house mite; saline and histamine references were used. The weal size was recorded after 15 minutes as the mean of the long axis and its perpendicular and scored as follows: positive—larger than histamine weal; minor—larger than 1 mm and less than or equal to histamine weal; and negative—less than or equal to 1 mm.

ESTIMATES OF WORK EXPOSURE

Hygienic measurements of total dust, gaseous fluorides, particulate fluorides, and total fluorides have been carried out routinely in the potrooms with

personal sampling equipment. Job specific exposure measurements were collected during the six months preceding the survey. The measurement procedures are described elsewhere.[13] The smelter consisted of three separate potroom departments for each worker and information on the job of each worker was obtained from the questionnaire. An estimate of current exposure to dust and fluorides was made. For workers rotating to more than one job, exposure for each separate job was averaged.

DATA ANALYSIS

The associations between continuous variables were analysed by simple linear regression.[14] Categorical outcome variables were analysed in two steps: firstly, crude prevalences of respiratory symptoms, WASTH, bronchial responsiveness (BR), and abnormal spirometry in relation to the exposure level were calculated. Secondly, adjusted odds ratios (ORs) of these associations were estimated by logistic regression (GLIM).[15] The logit was not linear in the continuous exposure variates. Thus exposure to dust and to total fluorides were dichotomised resulting in two exposure groups of about equal size. Similarly, duration of employment and age were divided into three categories. Atopy, smoking habits, age, duration of employment, and familial history of asthma were included in the model as potential confounders. Second degree product terms involving exposure to fluoride were included in the logistic model and interaction was assessed by backward elimination.

Results

Table 1 shows the distribution of sex, smoking habits, atopy, age, and presence of familial asthma by the degree of exposure to fluoride and to dust. The prevalence of current smoking was 67·3%, 24·1% of the subjects were never smokers, and 8·6% of the workers were ex-smokers. A positive reaction to the skin test was found in 12·2% of subjects and 75·1% showed no reaction. A history of familial asthma was obtained from 18·1% of the subjects. In the high fluoride exposure group we found higher prevalences of current smoking, atopy, and history of familial asthma. Regression analysis indicated an inverse relation between duration of employment and degree of both fluoride and dust exposure ($r = -0·24$; $p < 0·001$).

Table 2 Distribution of various air contaminants among potroom workers

Type of exposure	*Mean*	*Median*	*Lower quartile*	*Upper quartile*
Total particulates (mg/m³)	3·80	4·05	1·56	4·95
Particulate fluorides (mg/m³)	0·35	0·26	0·16	0·48
Gaseous fluorides (mg/m³)	0·18	0·17	0·12	0·23
Total fluorides (mg/m³)	0·54	0·45	0·27	0·73

Table 2 shows the distribution of work exposure. Exposure to fluoride and dust were positively correlated ($r = 0·81$, $p < 0·001$). The median of dust exposure was 4·05 mg/m³, interquartile range 1·56–4·95 mg/m³, and the corresponding values of total fluorides were 0·45 mg/m³ and 0·27–0·73 mg/m³. The hygienic threshold values in Norway for dust and total fluorides are 5·0 mg/m³ and 2·5 mg/m³, respectively. The hygienic threshold value for fluorides was not exceeded in any job category, and the limit to dust exposure was exceeded in three of the job categories. The calculation of dust exposure in two of these groups, however, was based on only two measurements and the results should be interpreted with caution.

Table 3 shows the prevalences of respiratory symptoms and lung function abnormalities. When dividing the population into two exposure categories regarding total fluorides a higher prevalence of respiratory symptoms (range 17·4%–27·3%) in the high exposure group was found compared with the low exposure group (range 9·6%–15·8%). This finding could result from uneven distribution of sex, smoking habits, atopy, age, or presence of familial asthma across the exposure categories as indicated in table 1.

Table 3 Prevalences of respiratory disorders in subjects with high and low exposure to fluorides and dust

	Total fluorides $< 0·5$ *mg/m³*	*Total fluorides* $\geq 0·5$ *mg/m³*	*Dust* $< 0·05$ *mg/m³*	*Dust* $\geq 0·5$ *mg/m³*
Symptoms:				
Dyspnoea (No (%))	33 (15·8)	44 (27·3)	37 (21·8)	40 (20·0)
Dyspnoea and wheezing (No (%))	30 (14·4)	40 (24·8)	25 (14·7)	23 (11·5)
Cough (No (%))	20 (9·6)	28 (17·4)	25 (14·7)	23 (11·5)
WASTH (No (%))	12 (5·7)	19 (11·8)	16 (9·4)	15 (7·5)
Total (No)	209	161	170	200
Spirometry:				
Abnormal (No (%))	43 (20·6)	38 (23·6)	35 (20·6)	43 (21·5)
Total (No)	209	161	170	200
Bronchial responsiveness:				
Hyper (BHR) (No (%))	10 (5·5)	7 (4·5)	6 (4·0)	11 (5·9)
Responders (No (%))	22 (12·0)	19 (12·3)	14 (9·4)	10 (5·3)
Total (No)	183	154	149	188

Table 4 Adjusted ORs (95% confidence intervals (95% CIs)) of respiratory disorders in the high exposure group estimated by logistic regression. Sex, smoking habits, atopy, familial asthma, age, respiratory protection, and duration of employment were included in the model

	Fluoride exposure OR (95% CI)	*Dust exposure OR (95% CI)*
Symptoms:		
Dyspnoea	2·9 (1·5–5·7)	0·6 (0·3–1·2)
Dyspnoea and wheezing	2·9 (1·3–6·5)	0·5 (0·2–1·1)
Cough	2·4 (1·0–5·6)	0·7 (0·1–3·5)
WASTH	3·7 (1·4–9·6)	0·4 (0·1–1·0)
Spirometry:		
Abnormal	1·2 (0·7–2·2)	1·0 (0·6–1·8)
Bronchial responsiveness:		
Responders	1·0 (0·4–2·4)	0·6 (0·3–1·4)

The prevalence of respiratory symptoms was not higher in the high dust exposure group than in the low dust exposure group.

The adjusted ORs of these respiratory variables across the degrees of exposure to fluoride estimated by the logistic model indicated an increased risk in the high exposure group ranging from 2·4 to 2·9 for each respiratory symptom and reaching 3·7 for WASTH (table 4). No significant effect on lung function tests was found, however. Positive associations between these symptoms and particulate fluorides and gas fluorides were also found. The effect of particulate fluorides was of almost the same magnitude as total fluorides and of borderline significance, whereas the effect of gaseous fluorides was weaker. The ORs of respiratory symptoms across the degree of dust exposure were lower than unity, and the upper limit of the 95% confidence interval of WASTH was also lower than unity (0·98).

Table 5 lists the effects on respiratory symptoms and WASTH of smoking, duration of employment, and age. The adjusted ORs of the different respiratory symptoms in current smokers compared with never smokers ranged from 1·6 to 2·2. Except for WASTH a dose-response relation between respiratory symptoms and duration of employment was indicated although no effect of age was found. Dyspnoea, dyspnoea with wheezing, cough, and WASTH were also positively associated with atopy (OR = 1·0–2·3), presence of familial asthma (OR = 1·0–1·8), and use of respiratory protection (OR = 1·5–2·6) although none of these reached significant values.

Interactions between exposure to fluoride and any of the other variables included in the model were not significant. The regression of SEV_1 did not indicate any association with exposure to fluoride or dust.

Discussion

In the present study the prevalence of respiratory symptoms and WASTH among potroom workers was associated with the degree of total exposure to fluoride. Bronchial responsiveness and spirometry were not, however, associated with exposure to fluorides or dust.

EXTRANEOUS FACTORS

The associations between exposure to fluoride and respiratory symptoms and WASTH could possibly be spurious. They were most pronounced with respect to respiratory symptoms, whereas the objective tests did not show any significant association with the degree of exposure to fluoride. Such a result could be explained by over-reporting of symptoms among the subjects in the high exposure group. The finding that the prevalence of symptoms did not show any association with the degree of exposure to dust makes this explanation unlikely. Neither the workers nor the interviewers were aware of the exposure values before the survey. Thus the result should not arise from this type of information bias. An increased prevalence of respiratory symptoms among potroom workers who spend more than 50% of their time at work in the potroom compared with controls has also been found by Chan-Yeung and coworkers[16] although they did not relate their findings to contaminants in the potroom atmosphere.

The associations between these respiratory disorders and exposure to fluoride could conceivably arise from confounders not included in the analyses. Such a confounder might be of occupational origin—for example, SO_2, which is known to be a potent airway irritant in both asthmatic[17] and healthy subjects[18] and which is emitted from the pots. A similar effect of SO_2 on silicon carbide workers exposed to SO_2 concentrations above 1 ppm was found by Osterman and

Table 5 Adjusted ORs (95% CIs) from multiple logistic regression for various symptoms on current smoking, duration of employment, and age

		Duration of employment (y)		
	Current smoking habits OR (95% CI)	*5–10 OR (95% CI)*	*⩾10 OR (95% CI)*	*Age > 40 y OR (95% CI)*
Dyspnoea	2·2 (1·3–6·4)	2·3 (1·0–5·3)	3·0 (1·2–7·8)	0·96 (0·9–1·0)
Dyspnoea and wheezing	2·0 (0·8–5·2)	2·6 (0·9–7·2)	3·5 (1·1–10·8)	0·96 (0·9–1·0)
Cough	1·6 (0·6–4·0)	2·0 (0·7–6·9)	2·2 (1·0–5·1)	0·97 (0·9–1·0)
WASTH	1·7 (0·6–4·8)	2·6 (0·8–8·0)	2·6 (0·7–9·4)	0·95 (0·9–1·0)

coworkers.[19] Although this possibility cannot be excluded, one would expect SO_2 to correlate with exposure to dust and to contribute to a positive association between respiratory symptoms and exposure to dust. Exposure to SO_2, however, probably had a higher correlation with gas fluorides than dust and hence confounding by SO_2 cannot be totally ruled out. It is possible that this problem cannot be solved by epidemiological methods even if measurements of SO_2 are available, because of problems of colinearity.

MISCLASSIFICATION OF EXPOSURE

Several sources of misclassification of exposure are possible. The subjects were classified according to their self reported jobs. Within each job department some jobs were rotated resulting in some misclassification that cannot be quantified. Furthermore, the estimates of exposure may not give a representative picture of the exposure within each job category and this results in a distorted estimate among those subjects who were "correctly" classified. Finally, if shorter periods of high peak exposures are the most relevant hazard, then eight hour sampling would not be the most optimal parameter of exposure. In a previous report including seven aluminium plants in Norway[9] the prevalence of respiratory symptoms was independent of the degree of exposure to fluoride in the total group whereas a positive association was found in pot operators. Differences between plants regarding job routines, sampling of exposure measurements, and differences in laboratory procedures would increase misclassification. Pot operators were thought to be the most comparable subgroup between plants in the population and the least prone to misclassification.

All these sources of misclassification are random and as neither the workers nor interviewers were aware of the degree of work exposure, the association between work exposure and outcome will be distorted towards zero.[20]

SELECTION OF POTROOM WORKERS

Even though an increased prevalence of respiratory symptoms could be shown in the high exposure group, the overall prevalence of respiratory symptoms in this population is not higher than reported from general population studies[21 22] and a population of students.[23] The prevalence of BHR was even lower than among the general population of Norway.[24] This finding can probably be explained by a selection bias that has also been described by others in connection with dusty jobs.[25–27] Such selection was also indicated by the data in the present study. The prevalence of no reaction to the skin test in the study population was 75%. In two surveys of a general adult population the similar prevalence was 49%[22] (three aeroallergens) and 47%[21] (fourteen aeroallergens) respectively. The inverse relation between degree of exposure to fluoride and duration of employment ($r = -0{\cdot}24$) indicates a selection within the workforce towards cleaner jobs and is of the same magnitude as that found by Kongerud *et al*[9] ($r = -0{\cdot}21$).

EFFECTS OF SMOKING AND DURATION OF EMPLOYMENT

The ORs of respiratory symptoms in current smokers compared with never smokers were in the range 1·6–2·2 (table 5) (again of the same magnitude as those found by Kongerud and coworkers[9]). The effect of duration of employment was also of the same magnitude as that described earlier[9] and indicated a dose-response relation in all symptoms except for WASTH. Between these two surveys 29 subjects reporting WASTH have been relocated to other parts of the plant. In accordance with Kongerud and coworkers[9] no effect of age was found.

The influence of familial asthma and atopy was of minor importance whereas those reporting daily use of respiratory airway protection had an increased prevalence of respiratory symptoms. This finding could be explained by an increased use of respirators among those who had symptoms and was also found by Kongerud and coworkers.[9]

BRONCHIAL RESPONSE AND EXPOSURE TO FLUORIDE

Our data indicated a poor association between exposure to fluoride and BHR, and makes a causal interpretation of the association between fluoride exposure and asthma less likely. The prevalence of BHR was low, resulting in imprecise estimates. This low prevalence of BHR could be explained by a survival effect that is likely to occur under such industrial settings[27] where irritating gases are found. Within each job category great fluctuation of work exposure might occur. Even in the low exposure groups, workers might experience days with high peak exposure. It is possible that such episodes can cause epithelial damage resulting in a transient BHR that returns to normal during days or weeks with "normal" exposure and rest days. Transient airway inflammation and BHR have also been reported after upper respiratory tract infection.[28] Association between BHR and exposure to agents containing aluminium fluoride has been reported elsewhere.[29 30] Thus if high peak exposure to fluorides induces BHR a misclassification of the cases is more likely to occur regarding prevalence of BHR than of symptoms when geometric mean values of fluorides are used as determinants of exposure. This is because the subjects probably still remember such episodes when the BHR returned to normal. These fluctuations could also result in misclassification of exposure as discussed earlier. The relation between BHR and WASTH should be studied using a longitudinal design.

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