Lung and bladder cancer among workers in a Norwegian aluminium reduction plant

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Abstract

***Objective*—To investigate the relation between exposure to polycyclic aromatic hydrocarbons (PAHs) and the incidence of lung and bladder cancer among aluminium production workers.**

***Methods*—The cohort comprised 1790 men employed for more than 5 years at a Norwegian aluminium plant contributing 36 587 person-years to the study. Historical exposure to PAHs was estimated by the use of industrial hygiene measurements and by a panel of three people familiar with the industry. Cancer incidence was investigated from 1953 to 1995. The observed cases of cancer among men were compared with expected numbers calculated from national rates for men, and dose-response relations were investigated by internal comparison by Poisson regression with age, period, smoking, and cumulative exposure included in the models. The effect of lagging exposure by 10, 20, and 30 years was also investigated.**

***Result*—The present study showed no increased risk of urinary bladder cancer or lung cancer with increasing cumulative exposure to PAHs. No significant changes in risk were found for different lag times.**

***Conclusions*—Due to the small size of this study, a minor increase in risk could not be excluded.**

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Aluminium is produced by the electrolysis of alumina (Al_2O_3) dissolved in molten cryolite (Na_3AlF_6) in several carbon lined steel shells (potline). During the process, alumina dust, solid fluoride salts, and hydrogen fluoride gas are emitted from the bath in the pots. The anodes liberate carbon dioxide (CO_2), carbon monoxide (CO), and sulphur dioxide (SO_2). The Søderberg anode is regularly supplied with unbaked anode paste at the top. The heating of the uncarbonised paste in the Søderberg anode leads to a considerable emission of coal tar pitch volatiles containing polycyclic aromatic hydrocarbons (PAHs). From prebaked anodes, on the contrary, only negligible amounts of coal tar pitch volatiles are emitted during electrolysis.

Epidemiological studies have indicated an increased risk of lung and urinary bladder cancer among workers exposed to coal tar pitch volatiles for long periods in the aluminium industry.[1–4] Norwegian studies have, however, shown conflicting results for lung cancer. The first Norwegian study found an increased risk of lung cancer,[4] whereas two recent Norwegian studies found no association between exposure to PAHs among aluminium process workers and lung cancer.[5 6] They found, however, a positive association between exposure to PAHs and bladder cancer when the cumulative exposure index was restricted to exposure received $\geqslant$30 years before observation.[5 6] The purpose of the present study was to investigate the relation between cumulative exposure to coal tar pitch volatiles, measured quantitatively as PAHs, and the incidence of lung and bladder cancer in an aluminium plant not previously investigated. We used different lag times and controlled for the potential confounding effects from smoking.

Materials and methods

The study plant (Hydro Aluminium Høyanger) is situated by a fjord in the western part of Norway and started production in 1920 with unhooded prebaked anodes. In 1939 the plant, as the first in Norway, changed to horizontal stud Søderberg technology, which was used at this plant until 1978. From 1958 the plant expanded with a potline carrying vertical stud Søderberg anodes, and more recently in 1981 the plant started another potline that used "hooded centre worked prebaked pots" with prebaked anodes.

Information on each employee was obtained from the company records giving name, date of birth, departments, jobs, and dates for all job changes. Since 1964 all inhabitants in Norway have a unique identification number, and these numbers were used for linkage between data sources. Dates of death or emigration were found by linkage to the population registry of Norway. Before 1964, the cohort was linked to the death and cancer registry data by name and date of birth only. After these linkages, we were able to identify 2437 men. About 2% (52 men) were not identified, and 88 were found to have died before the start of cancer registration in 1953.

Only the 1790 men who had worked at the study plant for >5 years were included in the present analyses due to difficulties with job assignments and scarce smoking data for workers with less experience.

Total particulate PAHs ($\mu g/m^3$) was used as the measure of exposure. The quantitative estimation of exposure to PAHs was based on personal measurements, stationary measurements, and descriptions of changes in the technological process over time. In periods with sufficient personal measurements the

Table 1 Job exposure matrix for the study plant

Type of potroom and job	*Period*	*PAH ($\mu g/m^3$)*	*Measurements (n)*
Open prebaked:			
All workers	1919–41	—	—
HS Søderberg:			
Potroom worker	1939–60	*150*	—
	1961–78	*100*	*3**
Supervisor	1939–60	*120*	—
	1961–78	*80*	*3**
Stud puller	1939–60	*300*	—
	1961–78	*200*	*3**
Day maintenance	1939–60	*300*	—
	1961–78	*200*	*3**
VS Søderberg:			
Potroom worker	1958–69	*200*	—
	1970–79	150	14†
	1980–85	75	60†
	1986–95	**25**	**170**
Stud puller	1958–69	*1500*	—
	1970–79	1200	14†
	1980–84	900	60†
	1985–95	**100**	**50**
Day maintenance	1958–69	*300*	—
	1970–79	200	14†
	1980–85	120	60†
	1986–95	**40**	**108**
Hooded prebaked:			
All workers	1981–95	**<2**	**31**
Anode production	1918–19	*50*	—
	1920–38	*125*	—
	1939–78	*100*	*1‡*
Anode mounting	1981–95	**5**	**38**
Potlining	1981–95	**75**	**178**

*The same three stationary measurements in the potrooms.
†The same 14 and 60 stationary measurements in the potroom.
‡One stationary measurement.
Bold=estimates based directly on personal measurements; normal=estimates based on relative changes in stationary measurements; italics=estimates based on assignment from a panel of three people familiar with the industry.

arithmetic mean was calculated as a direct measure of exposure. Stationary measurements were used to indicate relative changes in exposure for periods when only these had been taken. For periods with no or very few measurements (n<5), a panel of three people (occupational hygienists) agreed on subjective estimates based on changes in technology along with estimates and measurements from plants with comparable technology.[5 7] The results were summed in a job exposure matrix for PAHs (table 1). Based on 16 measurements of parallel sampling of PAH and benzene soluble material (BSM) at the plant, the PAH fraction of benzene soluble material was calculated to be 16%. The content of benzo(a)pyrene in total PAHs at Søderberg potrooms is typically 5%.[7]

Maintenance personnel working in the process departments were assigned one third of the average exposure estimated for the corresponding process workers based on estimates of time spent by the maintenance workers in the same departments.

Cumulative exposure was used as an indicator of individual dose, and calculated for each person-year under observation as the product of the exposure intensity and duration summed for all jobs held. Unexposed or low exposed person-time constituted one exposure category, and the exposed person-years were divided into two or three exposure categories based on the number of expected cases in each exposure category. For all the dose-response analyses we used a minimum lag time of 3 years, which means that exposure was disregarded during the last 3 years before each year of observation. The lagging of exposure was performed to avoid accumulating exposure in periods where exposure potentially had no effect on development of cancer.[8 9] We also performed analyses lagging exposure by 10, 20, and 30 years.

Due to uncertainties in the exposure estimates for periods before 1970, we used the time (years) employed in departments with exposure to PAHs and years of employment in potrooms (Søderberg and prebaked) as alternative dose measures.

Individual smoking habits were abstracted from medical files at the health department of the smelter, and independently collected from three long term employees who assigned smoking habits for people they knew from personnel lists. The data were categorised into never, current, and former smokers, and people with unknown smoking habits. Smoking habits were ascertained for about 74% of the workers employed for >5 years. Of these, 16% were never smokers, 60% were current smokers, and 24% were ex-smokers. Due to lack of information on date of stopping smoking among the former smokers, these were treated as former smokers throughout the analyses.

Because of the inclusion criterion of at least 5 years of employment, the follow up of cancer incidence started after 5 years of employment (net employment time) or from 1 January 1953 if the 5 year date of employment was reached before that date. Observation continued until 31 December 1995 or the time of death or emigration. For this time span, the Cancer Registry of Norway gave complete coverage of the population for all cancer sites except basal cell carcinoma of the skin, which was omitted in the present analysis. The registration system was built on multiple reporting from pathological laboratories and hospital departments and was based on compulsory reporting by physicians. The coding of cancers was based on a modified version of the seventh revision of the international classification of diseases code (ICD-7). Standardised incidence ratios (SIRs) were calculated as ratios between the observed and the expected numbers of cancer cases, the expected numbers were calculated from national incidences in 5 year periods by 5 year age groups. The 95% confidence intervals (95% CIs) were calculated assuming a Poisson distribution of the observed numbers. The cohort contributed 36 587 person-years to the study.

Poisson regression analysis was used for the investigation of internal dose-response relations and for the exploration of potential confounding effects from smoking. Age was included in the models as five or six age groups, and period of diagnosis was included as two calendar periods. Trend tests for the investigation of potential dose-response relations were performed by assigning scores (1, 2, 3 . . .) to the exposure categories.

The calculations of the SIRs and the Poisson regression analyses were performed with the program package EPICURE.[10]

Table 2 Observed (Obs) and expected (Exp) numbers of cases of different types of cancer and standardised incidence ratio (SIR) among 1790 male aluminium smelter workers in the follow up period 1953–95 (36 587 person-years)

Cancer site	*ICD-7 codes*	*Obs*	*Exp*	*SIR*	*95% CI*
All sites	140–204	286	278.5	1.0	0.9 to 1.2
Upper respiratory organs	141, 143–8, 161	8	7.6	1.1	0.5 to 2.1
Lip	140	1	4.0	0.3	0.0 to 1.4
Oesophagus	150	1	3.7	0.3	0.0 to 1.5
Stomach	151	26	30.0	0.9	0.6 to 1.3
Colon	153	31	22.3	1.4	0.9 to 2.0
Rectum	154	20	14.2	1.4	0.9 to 2.2
Liver	155.0	4	1.9	2.2	0.6 to 5.5
Pancreas	157	6	10.4	0.6	0.2 to 1.3
Nose and sinuses	160	2	0.9	2.2	0.3 to 8.0
Lung	162	27	30.6	0.9	0.6 to 1.3
Pleura	163	0	0.8	0.0	0.0 to 4.8
Prostate	177	69	60.5	1.1	0.9 to 1.4
Testes	178	1	2.1	0.5	0.0 to 2.6
Penis	179	3	1.1	2.9	0.6 to 8.4
Kidney	180	10	9.0	1.1	0.5 to 2.0
Bladder	181	23	18.1	1.3	0.8 to 1.9
Malignant melanoma	190	3	6.1	0.5	0.1 to 1.4
Other skin	191	8	9.6	0.8	0.4 to 1.6
Brain and nervous system	193	5	5.6	0.9	0.3 to 2.1
Thyroid	194	4	1.3	3.1	0.9 to 8.0
Unspecified	199	13	12.1	1.1	0.6 to 1.8
Lymphatic	200–2	4	7.9	0.5	0.1 to 1.3
Haematopoietic	203–4	13	12.6	1.0	0.6 to 1.8
All other sites	—	4	6.3	0.6	0.2 to 1.6

Results

The observed number of cancers at various sites did not differ significantly from the expected values (table 2). However, the overall SIR for bladder cancer was slightly increased (SIR 1.27), whereas the number of cases of lung cancer was lower than expected (SIR 0.88).

We found no indications of an increased risk of bladder or lung cancer with increasing cumulative exposure to PAHs, and the risk estimates for bladder and lung cancer did not rise when we used different lag times in the SIR analyses (tables 3 and 4).

No indications of positive dose-response relations were found in the internal analyses (tables 5 and 6). As there were no cases of lung or bladder cancer among the never smokers, these were categorised together with the former smokers in the internal analyses (tables 5 and 6). The relatively wide confidence intervals and the p value for the trend test (p=0.2) suggested that the indication of a negative trend in the dose-response analyses should be attributed to random variation (tables 5 and 6).

Table 3 Observed (Obs) and expected (Exp) numbers of cases of bladder cancer and standardised incidence ratio (SIR) by cumulative exposure to PAHs (μg/m³×year)

Cumulative exposure to PAHs (μg/m³×year)	*Obs*	*Exp*	*SIR*	*95% CI*
Lag 3 y:				
<50	13	8.2	1.6	0.8 to 2.7
50–1500	6	4.7	1.3	0.5 to 2.8
>1500	4	5.2	0.8	0.3 to 2.0
Lag 30 y:				
<50	16	10.7	1.5	0.9 to 2.4
50–1500	6	5.3	1.1	0.4 to 2.5
>1500	1	2.1	0.5	0.0 to 2.7

Table 4 Observed (Obs) and expected (Exp) numbers of cases of lung cancer and standardised incidence ratio (SIR) by cumulative exposure to PAHs (μg/m³×year)

Cumulative exposure to PAHs (μg/m³×year)	*Obs*	*Exp*	*SIR*	*95% CI*
Lag 3 y:				
<50	12	13.5	0.9	0.5 to 1.6
50–1000	11	6.3	1.7	0.9 to 3.1
1000–3000	2	5.8	0.4	0.0 to 1.3
>3000	2	5.0	0.4	0.1 to 1.4
Lag 20 y:				
<50	14	15.6	0.9	0.5 to 1.5
50–1000	11	7.2	1.5	0.8 to 2.7
1000–3000	1	5.6	0.2	0.0 to 1.0
>3000	1	2.2	0.5	0.0 to 2.5

Table 5 Poisson regression analysis of risk of bladder cancer by cumulative exposure to PAHs (μg/m³×year), age, calendar period of diagnosis, and smoking

Variable	*Rate ratio*	*95% CI*	*p Value test for trend*
Age:			
<60	1.0	—	
60–69	5.9	1.2 to 29.4	
70–74	8.4	2.2 to 32.7	
75–79	16.0	3.8 to 67.9	
>80	7.9	1.5 to 42.7	
Period of diagnosis:			
1953–74	1.0	—	
1975–95	3.7	1.3 to 10.6	
Smoking:			
Never and former smoker*	1.0	—	
Smoker	1.3	0.4 to 4.0	
Unknown	1.4	0.4 to 4.5	
Cumulative exposure to PAHs (μg/m³×year), lag 3 y:			
<50	1.0	—	
50–1500	0.7	0.3 to 2.0	0.2
>1500	0.5	0.2 to 1.5	

*No cases among the never smokers.

Table 6 Poisson regression analysis of lung cancer risk by cumulative exposure to PAH (μg/m³×year), age, calendar period of diagnosis, and smoking

Variable	*Rate ratio*	*95% CI*	*p Value test for trend*
Age:			
<60	1.0	—	
60–64	14.5	1.7 to 125.3	
65–69	30.8	3.6 to 266.0	
70–74	69.1	8.7 to 546.1	
75–79	26.3	2.7 to 257.7	
>80	27.6	2.8 to 273.8	
Period of diagnosis:			
1953–74	1.0	—	
1975–95	2.5	1.0 to 6.0	
Smoking:			
Never and former smokers*	1.0	—	
Smokers	13.2	1.8 to 100	
Unknown	3.7	0.4 to 32.4	
Cumulative exposure to PAHs (μg/m³×year), lag 3 y:			
<50	1.0	—	
50–1000	2.0	0.9 to 4.6	0.2
1000–3000	0.4	0.1 to 1.9	
>1000	0.5	0.1 to 2.1	

*No cases among never smokers.

Application of the alternative dose measure for PAHs, time employed in departments with exposure to PAHs, also showed a lack of association with bladder or lung cancer, as did the quantitative measure of PAHs (table 7). When years employed in potrooms (both Søderberg and prebaked) was used as the alternative dose measure, there was still no sign of an association between employment in potrooms and risk of bladder and lung cancer.

Discussion

The present study showed a lack of association between bladder and lung cancer and exposure to PAHs among aluminium production workers, and no significant change in risk was found with different lag times.

A large case-control study in Quebec, Canada showed a strong association between employment in Søderberg potrooms, or

Table 7 Observed (Obs) and expected (Exp) numbers of cases of bladder cancer and lung cancer, and standardised incidence ratio (SIR) by cumulative employment (y) in departments with exposure to PAHs and by cumulative emplyoment (y) in potroom departments*

	Obs	Exp	SIR	95% CI
Cumulative employment in departments* with exposure to PAHs (y), lag 3 y				
Bladder cancer:				
<0.1	18	10.3	1.7	1.0 to 2.8
0.1–5	2	1.9	1.1	0.1 to 3.8
5–20	1	3.1	0.3	0.0 to 1.8
>20	2	2.8	0.7	0.1 to 2.6
Lung cancer:				
<0.1	17	17.4	1.0	0.6 to 1.6
0.1–5	5	3.2	1.5	0.5 to 3.6
5–20	4	5.3	0.8	0.2 to 1.9
>20	1	4.6	0.2	0.0 to 1.2
Cumulative employment in potroom departments (y), lag 3 y				
Bladder cancer:				
<0.1	14	10.0	1.4	0.8 to 2.3
0.1–5	3	1.3	2.3	0.5 to 6.7
5–20	3	3.4	0.9	0.2 to 2.6
>20	3	3.4	0.9	0.2 to 2.6
Lung cancer:				
<0.1	14	16.7	1.0	0.5 to 1.4
0.1–5	5	2.4	1.5	0.7 to 4.9
5–20	3	5.81	0.7	0.1 to 1.5
>20	5	5.7	0.9	0.3 to 2.1

*The following departments were assumed to be related to exposure to PAHs: Søderberg potrooms, anode production, and potlining.

exposure to coal tar pitch volatiles, and bladder cancer.[1] A smaller cohort study of cancer incidence among workers in an aluminium plant in British Colombia, Canada also indicated an association between exposure to coal tar pitch volatiles and bladder cancer.[2] In that cohort study, the excess seemed to be confined primarily to millwrights, as six of the 16 cases observed had this job for >5 years.[11] Although both studies showed associations between exposure to coal tar pitch volatiles and bladder cancer, the risks seemed to be linked to different departments and jobs. Two recent Norwegian studies showed a positive association between exposure to PAHs and bladder cancer when exposure was lagged by 30 years, suggesting a latency period of ⩾30 years for bladder cancer in this industry.[5 6] In the Quebec case-control study, however, the authors reported that there was little evidence for latency.[1]

The lack of associations in the present study corroborates earlier findings in that the risk of bladder cancer after work in Norwegian Søderberg plants may be considerably lower than the risk reported from the Canadian studies.[1 2 5 6]

The absence of an increased risk of lung cancer in this study is not in accordance with a case-cohort study from Quebec, Canada where an increased risk was found for workers in the Søderberg departments.[3] The risk of lung cancer among aluminium production workers has, however, not been consistent among epidemiological studies.[2–6 12 13] The duration and intensity of exposure, as well as differences in smoking habits and diet could be possible explanations for the discrepancy. Asbestos has also been mentioned as a potential confounder.[3] Exposure to asbestos (mainly chrysotile) was assessed qualitatively in the present cohort based on the frequency and duration of dusty work that involved handling asbestos. Maintenance workers and potliners were considered to be exposed to asbestos, and employment (years) was used as a cumulative index. No signs of association were found, so negative confounding due to asbestos exposure seemed unlikely.

The use of individual smoking data gives considerable strength to this study. Although the smoking variable was crude, known associations between smoking and bladder cancer and lung cancer were found. However, some residual confounding or modifying effect from smoking cannot be ruled out.

As we had no cases of lung or bladder cancer among the never smokers, these was grouped together with former smokers in the internal regression analyses. The rate ratios for smokers do not therefore correspond to the relative risk between smokers and never smokers, which in such an analysis would go towards infinity. We also performed analyses restricted to smokers, which showed essentially the same lack of association between exposure to PAHs and the incidence of lung and bladder cancer.

Although the percentage of ever smokers in this group of workers was similar to other industrial populations, the tobacco consumption among smokers seems to have been considerably lower at the study plant. A survey of smoking habits among the employees at the plant in 1973 indicated that the mean number of cigarettes smoked a day was 5–10. Among employees in three other Norwegian aluminium plants the calculated mean was 12–15 cigarettes a day, and the respective mean in one of the Canadian studies was reported to be 18 cigarettes a day (mean value for controls).[3] These differences could be important, as the combined effect from smoking and exposure to PAHs may be synergistic.

A possible, although unlikely, explanation of our negative results could be that some cancer patients were missing from the personnel files. The plant is situated in a small community by a fjord in the western part of Norway, and has been the only workplace within the chemical industry in the area. Men living in this municipality and with professions within the chemical industry were abstracted from census data from 1960 and 1970 and linked to the cancer registry. Among these, a total of eight cases of bladder cancer and 12 cases of lung cancer were identified throughout 1995. As all of these cases were found in our material, we were confident that the personnel files were complete.

The estimates of exposure were partly based on industrial hygiene measurements and partly on estimation of exposure by a panel of three people familiar with the industry. For the period before 1970 no measurements were available, leading to a higher degree of uncertainty in the exposure estimates. Thus, misclassification of exposure was a potential threat to this study as it may have led to attenuation of potential dose-response relations and loss of power. However, misclassification probably does not explain the lack of association between exposure to PAHs and cancers of the bladder and the lung, as the investigation using alternative exposure indicators showed similar results, and the

dose-response trend tended to be more negative than positive.

The relatively small size of the present study is probably a more serious problem as it well may lead to inconclusive results. Epidemiological methodology can unfortunately not distinguish between low risk and no risk. Nevertheless, the results are important as they contrast the strong associations between exposure to PAHs and the risk of bladder cancer found elsewhere.[1]

An explanation for both the positive and negative findings in the various studies can be related to differences in the intensity and duration of exposure to PAHs, differences in the composition and bioavailability of PAHs and related compounds, and differences in lifestyle factors—such as smoking and diet. Methodological differences and limitations are also important.

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