Health survey of former workers in a Norwegian coke plant: part 2. Cancer incidence and cause specific mortality

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Abstract

Objectives—A Norwegian coke plant that operated from 1964 to 1988 was investigated to ascertain whether the male workers in this plant had increased morbidities of cancer or increased mortality from specific causes, particularly associated with specific exposures at the coke plant.

Methods—Personal data on all the employees of the plant were obtained from the plant's archives. With additional data from the Norwegian Bureau of Statistics we identified 888 male former workers at the plant. Causes of death were obtained from the Norwegian Bureau of Statistics, and cancer diagnoses from the Norwegian Cancer Registry. The results were compared with national averages adjusted for age. Specific exposures were estimated with records of actual measurements done at the plant and interviews with former workers at the plant.

Results—A significant excess of stomach cancer (standardised incidence ratio (SIR) 2.22, 95% confidence interval (95% CI) 1.01 to 4.21) was found. Mortality from ischaemic heart disease and sudden death was positively associated with work in areas which entailed peak exposures to CO. When considering work in such areas the past 3 years before death, the association was significant ($p=0.01$). The last result is based on only two deaths.

Conclusions—Considering the short follow up time and the small size of the cohort the results should be interpreted with a certain caution. The positive results would justify a re-examination of the cohort at a later date.

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Keywords: coke production; polycyclic aromatic hydrocarbons; cancer of the stomach

The present study concerns a Norwegian coke plant which operated from 1964 to 1988. Coke is produced by heating coal in nearly anaerobic conditions. The process produces not only coke, but also a range of gaseous and solid substances with known or suspected deleterious effects on human health. This ranges from the long term carcinogenicity of polycyclic aromatic hydrocarbons (PAHs) to the acute toxicity of carbon monoxide (CO).

As the relation between tobacco smoking and lung cancer became generally recognised in the 1960s, it was also known among the coke

plant workers that the same components as in tobacco smoke (notably PAHs) were present in the air in the coke plant. Reports in the press in the 1970s compared the exposure during one day at the top of the batteries with smoking 6000 cigarettes, based on measurements of PAHs.

Much of the work in the plant was also physically demanding, and the working conditions were far from optimal. Dust and heat were common. There were incidents when workers fainted because of exposure to CO. Some jobs were perceived as so strenuous that some of the workers during some periods refused to perform them.

The workers' perception of their own working conditions gave rise to a rich lore of stories. Some were based on facts—such as washing the floors with benzene, whereas others were probably apocryphal—such as when all the green plants in the administration building allegedly died overnight due to poisoning from the environment.

The coke plant was closed down in 1988 at short notice, and more than 300 workers were made redundant.

This situation accentuated the workers' fear of the possible consequences of their exposures during the employment at the coke plant on their health. The local trade union campaigned for a health screening of the former employees of the coke plant, and in 1993 we were engaged by the last owner of the plant, the Norwegian Ministry of Oil and Energy, to conduct a health survey and exposure assessment of the coke plant workers. The health survey and this study were financed by the Norwegian Ministry of Oil and Energy.

Previous studies of coke plant workers evaluated by the International Agency for Research on Cancer have shown increased risks of lung cancer and cancers of the kidney.¹ More recent studies have also reported increased risk of cancer of the prostate,² gastrointestinal cancer,³ and increased mortality from non-malignant respiratory disease and ischaemic heart disease.^{4,5}

The objectives of this study were to investigate whether the former workers at the coke plant had a higher overall and cause specific mortality and a higher morbidity from any cancer. Also we wanted to investigate whether any morbidity or mortality could be linked to specific exposures at the coke plant.

Specifically we wanted to investigate the following associations: between exposure to PAHs or CO and mortality for ischaemic heart

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Table 1 Observed and expected deaths among 888 male coke plant workers between 1962 and 1993

Cause of death (ICD-9 codes)	Obs	Exp	SMR	(95% CI)
All causes	122	139.7	0.87	(0.72 to 1.04)
Cancer (140–208)	27	33.7	0.80	(0.53 to 1.17)
Ischaemic heart disease (410–14)	47	43.0	1.09	(0.80 to 1.45)
Cerebrovascular disease (431–8)	3	9.1	0.33	(0.07 to 0.97)
Arteriosclerosis (440, 444, 785)	0	0.9	0.00	(0.00 to 3.99)
Sudden death (798)	4	4.5	0.88	(0.24 to 2.26)
Chronic obstructive lung disease (490–3, 496)	3	3.5	0.85	(0.18 to 2.48)
Violent death (800–999)	9	15.7	0.57	(0.26 to 1.09)
All other causes	29	29.3	0.99	(0.66 to 1.42)

disease, and between cumulative exposure to carbonaceous dust and chronic lung disease. We also wanted to investigate whether men employed in rotation shift schedules had a different mortality from ischaemic heart disease than men with no employment in such work.

The study of cancer incidence aimed at investigating associations between exposure to PAHs and the occurrence of cancers of the lungs and urinary tract, and between exposure to asbestos and respiratory cancers. We also wanted to investigate whether workers exposed to arsenic or benzene might have contracted cancers associated with these agents.^{6,7}

Methods

The investigation was designed as a cohort study of cause specific mortality and cancer incidence between 1962 and 1993. During this period the Cancer Registry of Norway gives a complete coverage of the population for cancer sites other than basal cell carcinoma of the skin. The coke plant's personnel files contained name, date of birth, and work histories for all people employed since the first workers were hired in 1962. As noted earlier, the plant did not become operational before 1964, but some of the workers were employed during the construction period.

Job histories were obtained from personnel records and validated through the records of the company's occupational health service, personal interviews, and questionnaires. A preliminary individual job history was constructed from information in the personnel files. This history was mailed, together with a questionnaire, to each worker for correction and completion. The job histories for deceased and non-responding people were checked and corrected against the company's occupational health service records.

Completion of the work histories has been described in greater detail in another paper.⁸

Table 2 Observed (Obs) number of cancers and standardised incidence ratios (SIRs; 95% CIs) among 888 male coke plant workers 1962–93

Cancer site (ICD-7 codes)	Obs	Exp	SIR	(95% CI)
All cancers (140–208)	53	61.1	0.87	(0.65–1.13)
Stomach (151)	9	4.1	2.22	(1.01–4.21)
Colon	6	4.9	1.21	(0.44–2.63)
Rectum	2	3.4	0.60	(0.07–2.15)
Lung (162)	7	8.5	0.82	(0.33–1.70)
Prostate (177)	5	9.6	0.53	(0.17–1.24)
Testes (178)	2	1.7	1.15	(0.14–4.16)
Kidneys (180)	2	2.4	0.82	(0.10–2.96)
Bladder (181)	5	4.5	1.11	(0.36–2.60)
Malignant melanoma of the skin (190)	3	3.2	0.94	(0.19–2.75)
Central nervous system (193)	3	2.3	1.29	(0.27–3.77)
Haematopoietic tissue (200–4)	5	5.3	0.95	(0.31–2.22)
All other sites	4	11.4	0.35	(0.10–0.90)

The study included all men with at least one year of employment. The Central Bureau of Statistics provided personal identification and the date of all emigrations from 1962. Of the 889 men who satisfied the inclusion criteria, a personal identification number could not be obtained for one man who thus was lost to follow up. This left 888 men in the final cohort.

Each man was investigated for the event of cancer or death from 1 January 1962 or the time of hire if later. Observation continued until 31 December 1993 or the time of death or emigration. Cancer cases were identified from the Cancer Registry through each cohort member's personal 11 digit identification number assigned to every person in Norway since 1961. The mortality analysis was based on the underlying cause of death (3 digit code, international classification of diseases (ICD)) as registered by the Central Bureau of Statistics. This institution has revised and coded all medical death certificates and necropsy reports according to the current 9th revision of the ICD-9.⁹ The observation period spanned the 7th, 8th, and 9th revision of the ICD. Up to and including 1992 tumour topography (primary site) was coded according to a slightly modified version of ICD-7. Since 1993, ICD-O (ICD for oncology, 2nd ed, 1990) has been the basis for topography coding.¹⁰ In this work all cases are presented in the ICD-7 format.

Because most cases of sudden death are due to ischaemic heart disease,¹¹ we show results for these two causes both separately and combined.

EXPOSURE DATA

A job exposure matrix was developed as described previously.⁸ Individual job histories and the job exposure matrix for the plant were used to determine individual exposure. The job histories were obtained from personnel records and validated by means of personal interviews, questionnaires, and records from the former occupational health service of the plant.

The following agents of interest to the study were considered: PAHs, asbestos, carbonaceous dust, quartz, arsenic, benzene, CO, and heat.

For PAHs and carbonaceous dust, time weighted average exposures (TWA) were estimated based on previous personal measurements. The exposures to other agents were estimated qualitatively and semiquantitatively with both measurements and other indicators of exposure.

Time spent at the oven top was used to indicate heat exposure. For benzene and arsenic the categories were divided into exposed and not exposed respectively.⁸ Exposure to asbestos was estimated relative to the time handling the material. When evaluating exposure to CO the categories were divided into exposed and not exposed with CO surveillance as an indicator of exposure. Faintings related to CO were used to define peak CO exposure categories.

The range of non-zero estimates was 2–300 µg/m³ for PAHs and 1–16 mg/m³ for carbonaceous dust. The content of quartz in the dust

Table 3 Deaths from ischaemic heart disease and sudden death 1962–93 in 888 male coke plant workers by cumulative exposure to PAHs, carbon monoxide (CO), and rotation shift work

Type of exposure	Latency period (y)	Diagnosis	Cumulative exposure to PAHs (SMR (n))*				p Value for trend
PAH ($\mu\text{g}/\text{m}^3\text{y}$):			Unexposed	< 50	50–149	≥ 150	
	0	Ischaemic heart disease	1.06 (29)	1.18 (9)	1.04 (4)	1.19 (5)	> 0.3
	0	Sudden death	1.04 (3)	1.22 (1)	0.0 (0.4)	0.0 (0.4)	> 0.3
	0	Combined	1.06 (32)	1.29 (10)	0.95 (4)	1.08 (5)	> 0.3
	15	Combined	0.96 (35)	1.76 (11)	0.71 (2)	1.38 (3)	> 0.3
CO (y):			Unexposed	< 2.0	2.0–5.9	≥ 6.0	
	0	Ischaemic heart disease	1.08 (18)	1.20 (7)	1.06 (8)	1.09 (14)	> 0.3
	0	Sudden death	0.57 (1)	1.50 (1)	0.0 (0.9)	1.61 (2)	> 0.3
	0	Combined	1.03 (19)	1.23 (8)	0.95 (8)	1.13 (16)	> 0.3
Rotation (y) shift work:			Unexposed	< 5.0	5.0–9.9	≥ 10.0	
	0	Ischaemic heart disease	1.26 (26)	1.03 (11)	1.21 (6)	0.67 (4)	> 0.3
	0	Sudden death	0.89 (2)	0.0 (1.2)	1.84 (1)	1.84 (1)	> 0.3
	0	Combined	1.18 (28)	0.93 (11)	1.27 (7)	0.77 (5)	> 0.3

* If SMR=0.0, the expected number is shown in italics instead of the observed number.

was estimated to be <3%. Benzene measurements suggested a TWA exposure between 0.1 and 3 ppm. The arsenic TWA exposure was estimated to be <5 $\mu\text{g}/\text{m}^3$.

Rotation shift work, which was common at the plant, has been suggested as a risk factor for ischaemic heart disease.¹² We therefore added rotating shift work to the job exposure matrix. Work involving two shifts was not regarded as rotating shift work.

ANALYSIS OF DOSE-RESPONSE RELATIONS

We used the standardised mortality ratio (SMR) as an effect measure in the mortality analysis, and the standardised incidence ratio (SIR) in the analysis of incidence of cancer. These measures were calculated as the ratio between the observed and expected number of deaths or cancer cases. The expected numbers were calculated from the biannual national mortality or incidence among men by 5-year age groups. The 95% confidence intervals (95% CIs) were calculated assuming a Poisson distribution for the observed numbers.

Dose-response relations were investigated by stratified analysis of SIRs with the calculated cumulative exposure as an indicator of individual dose. This was calculated for each person-year under observation as the product of exposure intensity and duration summed for all jobs held. Thus, each person could contribute person-time to more than one exposure category in each analysis. Unexposed person-time constituted one exposure category, and the exposed person-time was divided into three exposure categories with roughly equal amounts of person-years. The dose-response relations were also analysed by allowing for a latency period of 5, 10, 15, or 20 years. This approach assumes that a sufficient dose to contribute to the disease process is received during a limited induction period which may be followed by a period when the particular exposure does not influence the disease process.¹³

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The significance of each association was assessed with the Poisson trend statistic (two sided),¹⁴ and all analyses were carried out with the Epicure software package.¹⁵

Results

During the study period the cohort contributed a total of 19 430 person-years. Table 1 shows mortality from selected causes. Total mortality did not differ significantly from that of the general population and the only significant finding was a deficit of deaths from cerebrovascular disease. There were also fewer deaths than expected from external causes. Specifically, the mortality from chronic obstructive lung disease did not differ significantly from the expected value.

As shown in table 2, the cohort's incidence of cancer did not differ significantly from the expected values, with the exception of a significant excess of stomach cancers.

Table 3 shows that there was no association between cumulative exposure to PAHs or CO and mortality from ischaemic heart disease and sudden death. When the data were pooled for all three groups of workers exposed to PAHs and both causes of death, the SMR was 1.09 (19 deaths *v* 17.3 expected). However, when allowing for a latency period of 15 years this rose to 1.42 (16 deaths *v* 11.2 expected; NS). We found no association between employment in rotation shift work and mortality from these causes.

As an alternative to cumulative exposure, we investigated the association between employment that entailed peak exposure to CO and mortality from ischaemic heart disease and sudden death. Table 4 shows that mortality from these causes was positively associated with employment in such work, particularly when the exposure variable was restricted to peak exposure to CO during the three years before observation ($p=0.01$).

Table 5 shows the analysis of dose-response relations for those cancer sites which were of interest according to the hypotheses. A positive association was found between cumulative exposure to PAHs and cancers of the stomach and lungs. The association between PAHs and

Table 4 Deaths from ischaemic heart disease and sudden death 1962–93 in 888 coke plant workers by peak exposure to CO, either for total exposure (T) or for exposure the last three years before observation (L)

Cause of death	Exposure period	Peak exposure (y)			p Value for trend
		Unexposed (SMR (n))*	< 2.0 (SMR (n))*	≥ 2.0 (SMR (n))*	
Ischaemic heart disease	T	1.07 (43)	0.0 (0.9)	3.11 (4)	0.10
Sudden death	T	0.93 (4)	0.0 (0.1)	0.0 (0.1)	> 0.30
Combined	T	1.94 (47)	0.0 (1.0)	2.80 (4)	0.14
Ischaemic heart disease	L	1.06 (45)	0.0 (0.2)	7.39 (2)	0.01
Sudden death	L	0.90 (4)	0.0 (0.0)	0.0 (0.0)	> 0.30
Combined	L	1.04 (49)	0.0 (0.2)	6.39 (2)	0.01

* If SMR=0.0, the expected number is shown in italics instead of the observed number.

Table 5 Standardised incidence ratios of selected cancers 1962–93 in 888 male coke plant workers by cumulative exposure to PAHs and dust

Type of exposure	Cancer site	Latency period (y)	Cumulative exposure (SIR (n))				p Value for trend
PAH ($\mu\text{g}/\text{m}^3\text{y}$)	Stomach	0	Unexposed	< 50	50–149	≥ 150	
		20	1.94 (5)	0.0 (0.7)	5.61 (2)	5.15 (2)	0.16
	Colon and rectum	0	1.72 (6)	2.74 (1)	7.40 (1)	12.61 (1)	0.02
		0	1.12 (6)	0.72 (1)	1.38 (1)	0.0 (0.8)	> 0.3
	Lungs	0	0.72 (4)	0.0 (1.4)	0.0 (0.7)	3.60 (3)	0.08
		15	0.76 (5)	0.0 (1.1)	0.0 (0.5)	4.82 (2)	0.12
	Prostate	0	0.68 (4)	0.60 (1)	0.0 (0.9)	0.0 (0.9)	0.30
	Bladder	0	1.38 (4)	1.33 (1)	0.0 (0.4)	0.0 (0.4)	> 0.3
	Lymphatic and haematopoietic tissue	0	0.91 (3)	2.11 (2)	0.0 (0.5)	0.0 (0.6)	> 0.3
Carbonaceous dust ($\text{mg}/\text{m}^3\text{y}$)	Stomach	0	Unexposed	< 10.0	10.0–29.9	≥ 30.0	
		20	1.88 (4)	0.0 (0.6)	3.65 (2)	3.70 (3)	0.28
	Colon and rectum	0	1.49 (5)	0.0 (0.3)	16.49 (4)	0.0 (0.1)	0.02
		0	1.29 (6)	0.97 (1)	1.02 (1)	0.0 (1.7)	0.17
	Lungs	0	0.61 (3)	0.0 (1.0)	0.0 (0.9)	2.43 (4)	0.08
		15	0.65 (4)	0.0 (0.9)	0.0 (0.7)	3.95 (3)	0.04

* If SMR=0.0, the expected number is shown in italics instead of the observed number.

stomach cancer was significant when allowing for a latency period of 20 years ($p=0.02$). Analysis of the incidence of these cancers by cumulative dust exposure showed similar results. We found no association between any of these dose variables and cancers of the colon, rectum, prostate, or bladder. There were no cases of lung cancer among men with exposure to asbestos, and no case of lymphatic or haematopoietic cancers among those exposed to benzene.

Discussion

The reduced overall mortality and cancer morbidity found, although non-significant, can probably be viewed as a so called healthy worker effect. As mentioned, the work was physically demanding, and both a selection in of workers in good health and a selection out of workers unable to stand the working conditions can be envisaged.

This study has two main drawbacks, a small cohort and a short follow up time. This gives in general small expected values for incidence and mortality. Also, our study contains few cases. This makes it vulnerable to misclassification of exposures as well as the outcomes.

We decided to compare our results with data from the general population. An internal comparison between exposed and non-exposed groups would have reduced the power of an already low powered study. An internal comparison would reduce the impact of possible confounders, but any selection within the study population cannot be entirely excluded in this way.

For the trend analyses note should be taken of the fact that all the analyses except one contained at least one exposure category with no cases, and in the case of stomach cancer and exposure to PAHs with 20 years latency, the exposed categories contained only one case each.

We have data on tobacco consumption for 641 members of the cohort. These data do not differ from data on tobacco consumption from a steel making plant in the same community or from data from the general working population in the same community. When we compared our data on tobacco consumption with data

published earlier,¹⁶ we found that the workers at the coke plant smoked less than the general population 8 years earlier. The workers' consumption ranged from 24% to 56% of that of the general population, depending on the age group. Unfortunately we have no data on tobacco consumption for the deceased workers and for only a few of those with a diagnosis of cancer. The mortality and morbidity rates where thus compared with those of the general population adjusted for age under the assumption that tobacco consumption of coke plant workers did not differ substantially from the general population.

In studies of this kind the ideal would be to relate the received dose to incidence of disease. We decided to use the estimated cumulative exposure as an indicator of received dose. The exposure assessment has enabled us to measure the exposure in different periods and thus compute a cumulative exposure instead of using employment time as an indicator of received dose. These computations were based on the presumptions that the concentrations of airborne compounds were relatively stable over a period of time and that the working routines did not change in the same period. In spite of the uncertainties we are reasonably sure that the exposure assessments give a sound background for stratifying the workers according to exposure. However, when assessing the dose-response relations, the figures should be regarded with caution.

Studies among coke plant workers and workers exposed to combustion products show conflicting evidence of the relations between such exposures and cause specific mortalities as well as cancer morbidities. Some studies have found increased morbidities and mortalities from malignant as well as non-malignant causes,^{2 17–20} whereas others have found no such increases.^{21 22} The finding of an increased incidence of stomach cancer is in accord with a German study of workers in a gas producing plant which showed increased incidences of stomach cancer as well as cancer of the large intestine and rectum.³ Another study found a correlation between exposure to PAHs and death from ischaemic heart disease.²² In this last study no positive correlation between

exposure to CO and ischaemic heart disease was found.

The strength of our study lies in the exposure assessment and the possibility of a reliable computation of cancer morbidity. Even though the results are not conclusive they warrant a follow up study in a few years time. A longer follow up time would give a considerably better statistical power.

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