Cancer mortality among man-made graphite electrode manufacturing workers: results of a 38 year follow up

I Mori

Background and Aims: To examine the risk for cancer mortality among workers exposed to coal tar and coal tar pitch volatiles in a man-made graphite electrode factory. The risk for cancer mortality in this type of factory is still inconclusive, although coal tar and coal tar pitch are recognised as human carcinogens.

Methods: The study cohort consisted of 332 male employees who served more than five years in the period 1951–74. The cohort was traced until 1988. Analyses used standardised mortality ratios (SMRs) to compare cause specific mortality with that in the general and local population. Effect of smoking was estimated based on the information collected from the subgroup of the cohort. SMRs for leading causes of death were compared among different job titles, duration of employment, time since first employment, and observation subperiods. Exposure level for tar and benzo[a]pyrene (BaP) in the factory was also discussed, based on measurements done by other researchers in the past.

Results: During the study period, 52 deaths were identified (SMR 0.68), including 22 cancer deaths (SMR 1.01). The SMR for lung cancer was significantly increased in comparison with the general population (SMR 2.62). It was slightly decreased in comparison with the local population, but remained significant (SMR 2.35). Excess deaths were also observed for lymphatic and haematopoietic cancers (SMR 3.46). Smoking habits in the subgroup were similar to those in the general population; thus the increased SMR for lung cancer was unlikely to be explained by smoking.

Conclusion: Previous environmental measurements suggested that considerable exposure to tar and BaP had existed in the factory. The results suggest a possible risk for lung cancer among the cohort, but the limitations of the study, such as the small study population and insufficient information on exposure, indicate that further study is required.

Materials and Methods

Design of the study

The subjects were selected from workers employed by a man-made graphite electrode manufacturing plant which had been in operation since 1934 in Nishinomiya City (Hyogo prefecture, Japan), a heavy industry area next to Osaka. The cohort was constructed based on the membership registration of the plant’s labour union for 1961 and thereafter, and the list of retired workers which the labour union had prepared for this study. Among the retired workers, the earliest hiring was in 1946 and some of these workers had retired before 1961. Identified were 336 male workers who were employed or newly hired as of 1 January 1951 and thereafter, and had worked for at least five years in any of the manufacturing, transportation, and maintenance divisions before the plant was relocated in 1974. After excluding four workers with uncertain birth dates, 332 workers remained in the cohort. Two cancer cases had filed a compensation claim, which as described above, was already known when the study began, and were included in the cohort according to the criteria. Data on the cohort were traced for the 38 years from 1 January 1951 to 31 December 1988. The vital status of each worker was verified from the resident registry and the records of the Koseki

Abbreviations: BaP, benzo[a]pyrene; CTPV, coal tar pitch volatile; PAH, polycyclic aromatic hydrocarbon; SMR, standardised mortality ratio
were submitted as documentary evidence for a compensation lawsuit. Since the plant was relocated into a modern facility in 1974 (after Nakaminami’s measurement) and the exposure to CTPVs was assumed to have dropped significantly, the exposure of this cohort to CTPVs was considered to end virtually in 1974.

Data analysis
Statistical analysis
Person-years, standardised mortality rates (SMRs), and their 95% confidence intervals (95% CI) were computed using Epi-Plus.10 Expected numbers of deaths were calculated based on age and cause specific male mortality rates in Japan for the period 1951–88. Every five years from 1955 to 1985, mortality rates based on national census data were applied and estimated mortality rates from Vital Statistics of Japan for other calendar years were used.11 As most of the cohort members lived in a heavily industrialised area, the expected number of deaths from cancer should be calculated based on vital statistics in the local reference population. Age, sex, and cause specific death rates calculated from vital statistics of Hyogo prefecture, which includes Nishinomiya city and surrounding municipalities, were used for this purpose.12 For the period 1951–64, statistics were not available and those of 1965 were applied for these calendar years.

In order to examine the association between exposure and cancer risks, the author tried two different approaches. Firstly, SMRs were computed by job title. The list of cohort members included job titles and the length of time each title was held. Since beginning and ending dates were not given, the author had no knowledge of what job title a worker had at a given point in time. The longest held job title before 1974 was determined by such information and was used as the proxy of intensity of exposure. Therefore, person-years may be falsely attributed to a job before it becomes the longest title held, which could lead to underestimation of the results. In the case of a worker being assigned to different job titles for the same period of time, the job title that was assumed to impose the greatest exposure to CTPVs was chosen for analysis. Secondly, the duration of employment was used as the proxy of cumulative exposure under the assumption that all workers were equally exposed. SMRs by time since the first employment were also computed to consider the latency factor.

As the cohort came from different lists of active and retired workers provided by the union, and included two known cancer cases, SMRs for major causes of death were also computed by different observation periods to examine for a selection bias. The entire observation period was divided into three sub-periods: 1951–68, 1969–78, and 1979–88. As there were very few deaths in the early years, the initial eight years were included in the first subperiod.

Smoking habit
Information on smoking habits for the entire cohort was not available. However, information on the habit and daily cigarette consumption among 65 skilled workers engaged in the crushing, shaping, and impregnation processes was obtained from the records of a dermatological survey conducted in 1973. This information was used to estimate the effect of smoking habit on the association between exposure to CTPVs and lung cancer mortality using the method described by Axelson and Steenland.13 For this estimation, relative risks for lung cancer according to daily cigarette consumption proposed by Hirayama8 and smoking rates in the Japanese male population in 1970 (77.5%)14 and the proportion of the population who consumed cigarettes at various daily rates among male smokers in...
1.13 (95% CI: 0.46 to 2.33).

The value obtained was considered to be representative of the entire observation period.

RESULTS
SMRs for major causes of death
A total of 8043 person-years were included in this study. Table 1 shows the distribution of the population by age and calendar year. Fifty-two deaths were observed during the study period. Table 2 shows the SMRs for major causes of deaths. Compared to the national male population, the SMR for all causes of death was 0.68 (95% CI: 0.51 to 0.89). There were 22 deaths from all types of cancer; SMR was 1.01 (95% CI: 0.63 to 1.53). Nine of these deaths were from lung cancer, with an SMR of 2.62 (95% CI: 1.20 to 4.98). The SMR for lung cancer remained high in comparison with the local reference population (SMR 2.35, 95% CI: 1.07 to 4.46). Although there were only four deaths from haematopoietic cancers, the SMR was high at 3.19 (95% CI: 0.87 to 8.17). Cerebrovascular disease (430–438) 2 14.6 0.14 0.02 to 0.50. All deaths from cardiovascular disease were expected deaths for all causes, all cancers, lung cancer, and haematopoietic cancers by observation subperiods. The SMR for lung cancer was significantly high in the maintenance division (SMR 5.90, 95% CI: 1.22 to 17.26). Although the value was not significant, the SMR for lung cancer for all manufacturing processes combined (crushing, shaping, baking, impregnating, grafting, and finishing) was high (SMR 2.14, 95% CI: 0.79 to 4.46). Haematopoietic cancers were noted for the job title of crushing, shaping, and maintenance.

Table 4 shows lung cancer by duration of employment and time since the first employment. In the first 10 years of follow up (time since the first employment: 5–13 years), only one case of lung cancer was observed. Eight of nine cases were observed after 15 years of follow up. SMRs tended to decrease with duration of exposure, but the decrease was not statistically significant (p = 0.37 for trend).

Table 5 summarises the distribution of observed and expected deaths for all causes, all cancers, lung cancer, and haematopoietic cancers by observation subperiods. The SMR for all causes of death was lower than unity for any subperiod and, in particular, as low as 0.61 (95% CI: 0.40 to 0.91) for the period 1979–88. The SMR for all cancers was greater than unity for the first two subperiods but was only 0.84 (95% CI: 0.42 to 1.51) for the last subperiod. The SMR for lung cancer

<table>
<thead>
<tr>
<th>Cause of death (ICD-9th codes)</th>
<th>Observed</th>
<th>Expected</th>
<th>SMR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>All causes (140–208)</td>
<td>52</td>
<td>76.3</td>
<td>0.68</td>
<td>0.51 to 0.89</td>
</tr>
<tr>
<td>All cancers (140–208)</td>
<td>22</td>
<td>21.8</td>
<td>1.01</td>
<td>0.63 to 1.53</td>
</tr>
<tr>
<td>Oesophagus (150)</td>
<td>1</td>
<td>1.0</td>
<td>0.96</td>
<td>0.02 to 5.33</td>
</tr>
<tr>
<td>Stomach (151)</td>
<td>6</td>
<td>7.5</td>
<td>0.80</td>
<td>0.29 to 1.73</td>
</tr>
<tr>
<td>Liver (155)</td>
<td>2</td>
<td>2.7</td>
<td>0.74</td>
<td>0.09 to 2.69</td>
</tr>
<tr>
<td>Lung (162)</td>
<td>9</td>
<td>3.4</td>
<td>2.62</td>
<td>1.20 to 4.98</td>
</tr>
<tr>
<td>Lymphatic and haematopoietic (200–208)</td>
<td>4</td>
<td>1.2</td>
<td>3.46</td>
<td>0.94 to 8.86</td>
</tr>
<tr>
<td>Leukaemia (204–208)</td>
<td>2</td>
<td>0.5</td>
<td>4.02</td>
<td>0.49 to 14.52</td>
</tr>
<tr>
<td>Multiple myeloma (203)</td>
<td>2</td>
<td>0.2</td>
<td>13.37</td>
<td>1.62 to 48.29</td>
</tr>
<tr>
<td>Other forms of heart disease (420–438)</td>
<td>7</td>
<td>6.2</td>
<td>1.13</td>
<td>0.46 to 2.33</td>
</tr>
<tr>
<td>Cerebrovascular disease (430–438)</td>
<td>2</td>
<td>14.6</td>
<td>0.14</td>
<td>0.02 to 0.50</td>
</tr>
<tr>
<td>Pneumonia (480–486)</td>
<td>2</td>
<td>2.8</td>
<td>0.73</td>
<td>0.09 to 2.63</td>
</tr>
<tr>
<td>Bronchitis, emphysema, and asthma (490–493)</td>
<td>4</td>
<td>1.3</td>
<td>3.19</td>
<td>0.87 to 8.17</td>
</tr>
<tr>
<td>Chronic liver disease and cirrhosis of the liver (571)</td>
<td>2</td>
<td>3.0</td>
<td>0.67</td>
<td>0.08 to 2.44</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>13</td>
<td>26.6</td>
<td>0.49</td>
<td>0.26 to 0.84</td>
</tr>
</tbody>
</table>
Effect of smoking habits

Daily cigarette consumption was divided into four categories: non-smoker; ≤9 pieces/day; 10–19 pieces/day; and ≥20 pieces/day. Within these four categories, estimated smoking rates according to daily cigarette consumption were 0.38, 0.02, 0.20, and 0.40, respectively, for the cohort; and 0.22, 0.03, 0.20, and 0.55, respectively, for the general population. Relative risks of lung cancer by daily cigarette consumption for Japanese males were 1.00, 2.06, 4.00, and 6.24, respectively, for each category. Based on these data, the risk ratio of lung cancer in the cohort compared to the national male population was determined to be 0.82, suggesting that the effect of smoking can be neglected for the analysis of the mortality rate for lung cancer in this cohort.

DISCUSSION

Review of previous studies

Epidemiological studies have reported on the risk of cancer for graphite electrode workers from four countries. To evaluate their "population based corporate wide mortality surveillance system", Teta et al traced 2219 male workers for 10 years in several plants which manufactured graphite electrodes and other carbon products. In their study, the SMR for respiratory cancers was 0.85 and no excess death was detected. The authors suggested that their surveillance strategy may not have been sensitive enough to detect excess deaths which may have existed in small subgroups of the population, and that because of the lower temperatures to which coal tar pitch was heated in the carbon industry, concentration, and therefore exposure to suspect carcinogens may have been low.

Moulin et al conducted a cohort study and a nested case–control study in graphite electrode workers in two different plants: a cancer incidence study in one cohort (A, 1302 male workers) and a mortality study in another cohort (B, 1115 male workers). In cohort A, the standardised incidence rate (SIR) of tracheal, bronchial, and lung cancer was 79. The SMR was slightly higher, 1.18, in cohort B, although this was not statistically significant. In each nested case–control study, the odds ratio for lung cancer was 3.42 in plant A, showing a recognisable but insignificant relation to the duration of exposure. In plant B, the odds ratio was low for lung cancer with no relation to the duration of exposure. However, the authors pointed out that the SIR was possibly underestimated as a result of the lack of a local cancer registry, and that 12% of the cohort members were lost to follow up. They did not deny the presence of a selection bias to explain the discrepancy between the results of the cohort studies and the nested case–control study in plant A.
Boffetta et al investigated 901 Swedish graphite electrode workers for 21 years, with an elaborate effort directed towards exposure assessment. Two lung cancer cases were observed, whereas 1.2 were expected (SMR 1.68; 95% CI: 0.20 to 6.07). As the observation period was relatively short for such an assessment and the observed number was also small, they stated that they could not draw a definite conclusion on lung cancer risk in the cohort.

Donato et al recently published the results of a 41 year follow-up of 1006 male workers. Thirty four lung cancer cases were observed, whereas 44.2 were expected (SMR 0.77; 95% CI: 0.53 to 1.08). Although they failed to identify a high risk group among the cohort because of lack of information on job title, they argued against lung cancer risk from graphite electrode manufacturing based on the negative relation between the duration of employment, a proxy for cumulative exposure to PAHs, and the relative risk for lung cancer.

Overall, no reliable conclusions can be drawn from these previous studies with respect to the risk of lung cancer for graphite electrode workers.

**Lower SMR for all causes of death**

In the present study, the SMR for all causes of death was 0.68, which suggests the presence of the healthy worker effect; that is, the effect of selective employment on mortality. Apart from the present study, a review of documents submitted by 200 workers who resigned from the plant from 1965 to 1969 revealed that 121 (60%) left the plant by the end of the second year of employment. Of the 200 workers who had resigned, 166 were involved in graphite electrode manufacturing when they left the company; 38 (23%) of these listed an improper working environment and serious concern about health conditions as reasons for their resignation. These facts may support to a certain extent the significantly low SMR for all causes of death being a result of the healthy worker effect.

On the other hand, with a somewhat high percentage of subjects lost to follow up, the possibility of missing deaths in those subjects and consequently underestimating the overall SMR should also be taken into consideration. If the same risk ratio of the total cohort is applied to the 21 subjects who were lost to follow up, five or six extra deaths may be expected. In addition, four workers who were excluded from the study because of the lack of information on their date of birth, three for whom a compensation claim had been filed and who was considered to represent the entire cohort on the grounds that the physical examination for the subgroup was considered to represent the entire cohort on the grounds that the physical examination for the subgroup was performed; and physical examinations screening for dermatological changes were unlikely to cause serious selection and information bias for smoking rate. Using the method of Axelsson and Steenland, confounding from smoking was found to be negligible.

The SMR for lung cancer was high over all study subperiods. Consistently high SMRs over time can provide support for increased lung cancer risk in the cohort to a certain extent, even though the SMRs were calculated based on a small number of observed/expected number of deaths. In particular, it is important that the SMR in the last subperiod (1979–88) was 2.00 since this period is assumed to be free from serious selection bias as mentioned previously. The lung cancer case for whom a compensation claim had been filed and who was known to have cancer before the beginning of the study died in 1974. Therefore, this case has no impact on lung cancer risk in the last subperiod.

These findings suggest that excess deaths from lung cancer possibly exist in the study population, and thus the subjects might be exposed to certain agents which can contribute to increasing the risk of lung cancer.

**Cause of excess deaths from lung cancer**

In the present study the exposure to CTPVs would be considered as a possible cause of excess mortality from lung cancer. There is no other information available on CTPVs from this
### Table 6  Results of working environment measurement of tar and benzo[a]pyrene (BaP) in the air by workshop in the plant, 1973 (by Japan Industrial Safety and Health Association (JISHA)) and 1974 (by Nakaminami)*

<table>
<thead>
<tr>
<th>Workshop/ measurement method†</th>
<th>JISHA</th>
<th>Nakaminami</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Tar in the air (mg/m³)</td>
<td>BaP in the air (µg/m³)</td>
</tr>
<tr>
<td></td>
<td>Number of samples‡ Mean Range</td>
<td>Number of samples‡ Mean Range</td>
</tr>
<tr>
<td>Crushing A sampling</td>
<td>6 (3) 1.52 0.26–4.69</td>
<td>4 (2) 40 Tr§–82</td>
</tr>
<tr>
<td></td>
<td>2 (1) 0.79 0.77–0.82</td>
<td></td>
</tr>
<tr>
<td>Blending A sampling</td>
<td>6 (3) 0.57 0.31–1.15</td>
<td>1 (1) 65 –</td>
</tr>
<tr>
<td></td>
<td>4 (2) 1.54 0.15–2.96</td>
<td></td>
</tr>
<tr>
<td>Kneading A sampling</td>
<td>4 (2) 0.30 0.23–0.46</td>
<td>2 (2) 51 Tr§ 102</td>
</tr>
<tr>
<td></td>
<td>9 (5) 1.06 0.29–4.10</td>
<td></td>
</tr>
<tr>
<td>Shaping A sampling</td>
<td>– – –</td>
<td></td>
</tr>
<tr>
<td></td>
<td>12 (6) 0.38 0.15–1.08</td>
<td>2 (1) 2.38 1.90–2.86 4.4</td>
</tr>
<tr>
<td>Impregnation A sampling</td>
<td>2 (1) 0.14 0.09–0.18</td>
<td></td>
</tr>
<tr>
<td>Graphitisation A sampling</td>
<td>– – –</td>
<td>1 (1) Tr§ –</td>
</tr>
<tr>
<td></td>
<td>– – –</td>
<td></td>
</tr>
<tr>
<td>All workshops</td>
<td>18 (9) 0.81 0.09–4.69</td>
<td>5 (3) 32.2 Tr§–82</td>
</tr>
<tr>
<td></td>
<td>27 (14) 0.81 0.15–4.10</td>
<td>3 (3) 55.7 Tr§–102</td>
</tr>
</tbody>
</table>

*These data were submitted as evidence of the plaintiff (measured by Nakaminami) and of the defence (measured by JISHA) to the trial on occupational cancer cases.
†“A sampling” means area sampling at randomly selected points in the unit work area. “B sampling” means area sampling at close proximity to workers whose exposure is considered to be maximum.‡
‡Number in parentheses is number of sampling points.
§Tr, traceable—that is, the concentration of BaP less than 0.00 mg/m³ but not zero. It was assumed to be zero for calculation of the means.
**Mean BaP concentration.
workplace except for the data measured by the Japan Industrial Safety and Health Association and Nakaminami. They independently measured concentrations of tar and benzo[a]pyrene (BaP) in the air at selected sites of the plant in June 1973 and March 1974, respectively, in connection with legal actions brought against the plant. As table 6 shows, both studies had similar results regarding the level of tar in the plant, which was much higher than 0.2 mg/m³, the Administrative Control Level in Japan. BaP in the air was mostly in the order of 10 μg/m³, although the number of samples was quite limited and the data were scattered over a wide range (from traceable to 102 μg/m³).

With regard to reports from studies of other graphite electrode plants with similar facilities in Japan during the same period, Numata et al monitored concentrations of tar and BaP in the air for four years beginning in 1971, and reported that the average concentration of tar in the air was 0.745 mg/m³ in the plant that they studied, slightly lower than the level in the plant that we studied.¹ The level of BaP in the air ranged from 0.44 to 72.3 μg/m³, similar to the factory in the present study. Shibata et al also studied BaP concentrations in a graphite electrode plant and reported a range of 0.2–17.2 μg/m³ (45th Annual Meeting of Japan Association of Industrial Health, Abstract in Japanese). Thus, it may be possible to speculate that the air concentrations of tar and BaP in these plants may be considered to be in the same category as those in the plant we examined.

In a study of a graphite electrode plant conducted by Lindstedt and Sollenberg in Sweden from 1974 to 1976, the BaP level was highest, or 19–40 μg/m³, at the site of impregnation.¹ This result suggests that levels of CTPV air concentration may have occurred in graphite electrode plants in Europe similar to those in Japan.

Moulin et al measured concentrations of BaP in two plants from 1983 to 1984. The average BaP level in the air was 1.90 μg/m³ (area sampling: ranging from 0.43 to 12 μg/m³), and 2.70 μg/m³ (personal sampling: ranging from 0.59 to 6.20 μg/m³) in one plant, whereas the respective values were much lower in the other plant, 0.46 μg/m³ (area sampling: ranging from 0.18 to 0.74 μg/m³) and 0.17 μg/m³ (personal sampling: ranging from 0.015 to 0.57 μg/m³).¹ These results indicate that the exposure level to BaP differed considerably between the two graphite electrode plants. Thus, it may be misleading to group these two plants together in evaluating the risk of lung cancer for graphite electrode workers.

Some researchers have focused on the polycyclic aromatic hydrocarbons (PAH) contained in CTPVs and attempted to quantify PAH exposure by means of measuring its metabolites in urine. Buchet et al measured 1-hydroxypyrene in urine collected from graphite electrode workers and reported that the exposure to PAH among those workers was similar to that in coke oven workers.¹¹

In considering these observations as a whole, it can be assumed that graphite electrode workers, including this study population, were often exposed to significant levels of CTPVs in their workplace in the past. The average concentration of BaP in the air in the plant investigated was of the order of 10 μg/m³ and classified as “very high BaP exposure,” according to the classification by Lindstedt and Sollenberg.¹ Top side workers in gasification and coke production plants and pin setters, potmen, and cranemen in aluminium refineries are included in this category and are identified by IARC as jobs in which workers are placed at risk of cancer. This is in parallel to the excess mortality of lung cancer observed in this study.

In the present study, to compare cancer risks among different exposure levels, the author attempted to use job title as a proxy of CTPV exposure. This provided limited information because person-years in each subgroup were too small for a statistical analysis; insufficient measurement data and incomplete job histories did not allow the construction of a cumulative exposure index. Interpretation of the results needs careful consideration since, as explained previously, person-years prior to the longest held job are not excluded from calculation and may have caused underestimation of the results. As a result, however, the SMR of 2.14 for lung cancer was high, if not statistically significant, for the manufacturing workers, suggesting a possible association with manufacturing of graphite electrodes. With regard to maintenance workers, two of three lung cancer cases were welders. Although the exposure to metal fumes during welding should be taken into account,²⁸ these workers were also likely to be exposed to considerable concentrations of CTPVs because maintenance workers in this plant were reported to have been engaged in welding and repairing machines caked with raw materials without proper personal protective equipment.

Duration of employment as a proxy of cumulative exposure and time since the first employment as a latency factor were also considered and changes in SMR for lung cancer across these two factors were examined. With longer follow up periods (longer than or equal to 15 years), SMRs were increased in each duration of employment category but tended to decrease rather than increase. One important and possible reason is misclassification of cumulative exposure because of neglect of the exposure level by job. Thus, it is not appropriate to consider that the result does not support overall lung cancer risk from electrode manufacturing.

Risk of lymphatic and haematopoietic malignancies

In the present study, the risk of lymphatic and haematopoietic cancers was also found to be higher in the cohort than in the national male population. In particular, the SMR for multiple myeloma was significantly greater than unity. As there were only two cases each of leukaemia and multiple myeloma, they may merely represent a chance cluster. However, other researchers have recognised higher risks for leukaemia, multiple myeloma, and other lymphatic and haematopoietic malignancies in those who are exposed to CTPVs, such as aluminium reduction workers,²⁶ coke production workers, stokers,²⁷ and chimney sweeps.²⁵ The association between CTPV exposure and lymphatic and haematopoietic malignancies needs further investigation.

Conclusions

In the present study, excess deaths from lung cancer were observed. The SMR for lung cancer remained high, even though smoking habits among the cohort members, regional characteristics, and selection bias that might have occurred in the registration process of the cohort were taken into account. Processes involved in graphite electrode manufacturing are reported in the literature to be associated with substantial exposure to CTPVs and this was also true for some parts of the plant studied. A dose–response relation could not be established in this study because of limited exposure data and incomplete records of job histories for the subjects. Further studies with a larger population and a precise exposure assessment are expected.

ACKNOWLEDGEMENTS

The author would like to thank Dr Shin'ya Watanabe for his extended support, and Ms Kayo Tanaka for supporting data management.

REFERENCES


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*Occup Environ Med* 2002 59: 473-480
doi: 10.1136/oem.59.7.473

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