Mesothelioma in Sweden: Dose–Response Analysis for Exposure to 29 Potential Occupational Carcinogenic Agents

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A B S T R A C T
Background: There is little information on the dose–response relationship between exposure to occupational carcinogenic agents and mesothelioma. This study aimed to investigate this association as well as the existence of agents other than asbestos that might cause mesothelioma.

Methods: The Swedish component of the Nordic Occupational Cancer (NOCCA) study consists of 6.78 million individuals with detailed information on occupation. Mesothelioma diagnoses recorded in 1961–2009 were identified through linkage to the Swedish Cancer Registry. We determined cumulative exposure, time of first exposure, and maximum exposure intensity by linking data on occupation to the Swedish NOCCA job-exposure matrix, which includes 29 carcinogenic agents and corresponding exposure for 283 occupations. To assess the risk of mesothelioma, we used conditional logistic regression models to estimate hazard ratios and 95% confidence intervals.

Results: 2,757 mesothelioma cases were identified in males, including 1,416 who were exposed to asbestos. Univariate analyses showed not only a significant excess risk for maximum exposure intensity, with a hazard ratio of 4.81 at exposure levels 1.25–2.0 fb/ml but also a clear dose–response effect for cumulative exposure with a 30-, 40-, and 50-year latency time. No convincing excess risk was revealed for any of the other carcinogenic agents included in the Swedish NOCCA job-exposure matrix.

Conclusion: When considering asbestos exposure, past exposure, even for short periods, might be enough to cause mesothelioma of the pleura later in life.

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1. Introduction

Mesothelioma is a very aggressive malignancy that occurs in the peritoneum or pleura. It is mainly linked to occupational asbestos exposure and was classified as an occupational disease in the 1950s [1]. Mesothelioma has a poor prognosis, with most cases dying within 1 year of diagnosis [2], and a long latency period of up to 40 years [3]. The incidence of mesothelioma varies; incidence in developed countries such as Belgium, Britain, and Australia exceeds 30 cases per 1 million inhabitants [4], and in Sweden, about 12 cases are diagnosed per 1 million inhabitants (approximately 120 cases/year) [5]. Between 1994 and 2008, 92,253 mesothelioma deaths were reported in 83 countries, 54% of them in Europe [6].

The risk of mesothelioma is strongest with exposure to asbestos types from the amphibole family. However, chrysotile (serpentine type) was the most common asbestos fiber used in Sweden [7]. In some industries, such as asbestos cement production, asbestos types from both the serpentine and amphibole families were used.
Asbestos was banned in Sweden in 1982 [9], and there are strict precautions and security requirements for occupations that include the handling of or exposure to asbestos, e.g., asbestos removal [10]. Despite the fact that asbestos has been absent from the Swedish labor market for 35 years, the total annual number of new mesothelioma cases did not show any evidence of decline until the year 2014 [11].

Only a limited number of occupations have been reported to confer a significant excess risk of mesothelioma [12], and no connection between mesothelioma and any occupational exposure besides asbestos, such as air pollution [13], has been scientifically proven. In the United Kingdom and Finland, it has been estimated that 97% [14] and 90% [15] of mesothelioma cases, respectively, are related to asbestos exposure, mostly occupational asbestos exposure.

A recent report on the Nordic Occupational Cancer (NOCCA) cohort covered the period 1961–2005 and is the largest study of occupational cancer published so far in the Nordic countries. It included 2.8 million diagnosed cases of cancer in the five Nordic countries (Denmark, Finland, Iceland, Norway, and Sweden) and reported mesothelioma cases in occupations where asbestos exposure is not usually considered to be present [16]. A total of 40.6% of the mesothelioma cases in the NOCCA cohort were from Sweden (2,521 men and 548 women), and in 12 of the 53 occupations considered in this population, there was a statistically significant excess risk of mesothelioma among men [16].

There is little information about substances other than asbestos that can cause mesothelioma. Authors have found mesothelioma outcomes in animal studies after inhalation of refractory ceramic fibers [17], but no such effect on humans has been reported in the literature. Moreover, there is a lack of studies on the dose–response relationship between agents other than asbestos and mesothelioma, and the studies that do exist reported conflicting results [18–22].

We used the Swedish NOCCA-job exposure matrix (JEM) [23] to identify 29 potential occupational carcinogenic agents. Twenty-five of these agents are chemical agents, and four are non-chemical agents. In the present study, we linked the Swedish NOCCA-JEM with mesothelioma outcomes in the Swedish NOCCA study [16] to determine the existence of a dose–response relationship between occupational exposures and mesothelioma of the pleura.

2. Materials and methods

All people who participated in the 1960, 1970, 1980, and 1990 censuses in Sweden and were still alive and living in the country on 1 January of the year following the census were included in the study cohort. The individual data from these censuses are centrally computerized at the Swedish National Statistics office [24] and include information on economic activity, occupation, and industry. Personal identification numbers were used to link census data to the Swedish Cancer Registry to identify incident cases of mesothelioma, and the studies that do exist reported conflicting results [18–22]. When estimating cumulative exposure, we assumed that the occupation that cases and controls reported in the 1960 census also applied up to 45 years prior to that census. To quantify cumulative exposure, we assigned a value of the product of the proportion and level of exposure (P × L) from the Swedish NOCCA-JEM for each occupational category. This value was then multiplied by employment period, i.e., the time (T) in years during which the individual was in that occupation. This procedure was repeated for all agents in the Swedish NOCCA-JEM. Employment period was assumed to start at age 20 years and end at 65 years. When individuals had more than one occupation during the study period, the individual exposure history consisted of more than one P × L × T value. Thus, cumulative exposure was estimated by summing the P × L × T values over an individual’s entire working career. We estimated hazard ratios (HRs) and 95% confidence intervals (CIs) for each occupational carcinogenic agent by conditional logistic regression.

We selected values corresponding to the 50th and 90th percentiles of cumulative exposure distribution among all exposed case/control individuals as cut-off points for categorization. Exposure values in the 0–50th percentile were categorized as “low,” the 50–90th percentile as “moderate,” and >90th percentile as “high”. Indiviudals with zero exposure were used as the reference group. A test for trend was performed for a dose–response relationship between cumulative exposure and mesothelioma.

For asbestos, we present analyses among unexposed and ever-exposed individuals, with ever exposure distributed in categories of low, moderate, and high. Cumulative exposure to each occupational carcinogenic agent is reported in unit-years. Assuming that mesothelioma has a long latency period, i.e., the time between the beginning of asbestos exposure and mesothelioma diagnosis, and that recent exposures are less relevant than those which took place in the past, we performed additional analyses excluding all exposures that occurred 20–50 years before the index date of the cases and controls. This latency allowed us to compare our results to those of other studies [27]. Information on other potential confounders, such as smoking and alcohol consumption, were not available. Asbestos exposure <1.78 f/ml was categorized as low, 1.79–15.2 f/ml as moderate, and >15.2 f/ml as high and used in analysis for 0–50 years latency.
Analysis for asbestos was also done for time since first exposure, divided in 0—9 years, 10—19 years, 20—29 years, 30—39 years, 40—49 years, and 50 + years. HRs and 95% CIs were used.

HRs and 95% CIs for pleural mesothelioma among men and women were analyzed in four categories: 0 fb/ml, 0.2 fb/ml, 0.8 fb/ml, and 1.25 fb/ml. HRs were also analyzed for maximum exposure intensity, defined by exposure index (P x L), were analyzed in four categories: 0 fb/ml, 0—0.1 fb/ml, 0.1—0.8 fb/ml, and 0.8—1.8 fb/ml. HRs were also analyzed for maximum exposure intensity (L) in four categories: 0 fb/ml, 0—0.2 fb/ml, 0.2—1.25 fb/ml, and 1.25—2 fb/ml.

4. Results

During follow-up, 2,757 mesothelioma cases and 27,570 controls were identified (Table 1). In the univariate model, 17 of the 29 occupational carcinogenic agents were statistically significantly associated with the risk of mesothelioma. Our study indicates that risk of mesothelioma is increased even at low levels of asbestos exposure. Cumulative doses of less than 1.78 f-y/ml led to an HR of 2.3. We found a dose—response relationship between cumulative asbestos exposure and mesothelioma of the pleura using a 30-year, 40-year, and 50-year lag time (i.e., latency period). We also found a clear relationship between increased exposure intensity (exposure index) and for maximum exposure intensity and increased HRs (Tables 4 and 5). We found a clear relationship between maximum exposure intensity and increased HRs, with an HR of 4.81 for asbestos exposure levels 1.25—2.0 fb/ml. The asbestos part from the NOCCA-JEM [23] describing level (L) and probability (P) of exposure for occupations in 1945—1984 is shown in Table 6.

Nine other occupational carcinogenic agents had p <.01 after adjustment for asbestos exposure (Table 7). The other 18 occupational carcinogenic agents did not show any excess risk or trend for mesothelioma of the pleura after adjustment for asbestos exposure. Moreover, after adjustment for asbestos exposure we found no significant excess risk for exposure to animal dust, benzene, chlorinated hydrocarbon solvents, crystalline silica, formaldehyde, methylene chloride, other organic solvents, toluene, wood dust, ionizing radiation, or perceived physical workload. Aliphatic and alicyclic hydrocarbon solvents, diesel exhaust, gasoline,
We studied the dose–response relationship between exposure to 29 occupational agents classified as carcinogens by the International Agency for Research on Cancer [28] and the risk of mesothelioma of the pleura. Our important findings were a clear relationship between maximum exposure intensity and increased HR, with an HR of 4.81 for asbestos exposure levels 1.25–2.0 fb/ml. We found a dose–response relationship in univariate analyses between asbestos exposure and mesothelioma of the pleura with a 30-, 40-, and 50-year latency period. Our study indicates that risk of mesothelioma is increased even at low levels of asbestos exposure. Cumulative doses of less than 1.78 f/ml led to an HR of 2.3. The threshold limit value (TLV) for asbestos in 1976 was 2 f/ml, which means that 1 year of exposure around the TLV was considered a risk. In 1982, the Swedish Agency of Working Life dramatically decreased the TLV to 0.5 f/ml and again in 1987 to 0.2 f/ml [29–31].

Previous studies on the dose–response relationship between asbestos exposure and the risk of mesothelioma have shown mixed results. Some found no clear relationship [20–22], whereas Rodgers et al. [18] and Lacourt et al. did find a dose–response relationship [19]. A later case–control study of mesothelioma and cumulative asbestos exposure found that the impact of a given increase in dose depended on when the dose was received [32]. Järvholm et al. [33] observed a significant excess risk of mesothelioma among construction workers exposed to multiple carcinogenic agents, including asbestos and bitumen. We were unable to control for other potential confounding factors or effect modifiers such as smoking.

Nine occupational carcinogenic agents showed an excess risk of mesothelioma of the pleura after adjustment for asbestos exposure, with a $p < 0.01$. However, welding fumes, trichloroethylene, lead, aromatic hydrocarbon solvents, and benzo[a]pyrene showed a positive significant trend, but this was probably due to misclassification. Most of the 10 agents in Table 7 occur in industries in which asbestos was handled, but the Swedish NOCCA-JEM is not sensitive enough to separate those exposures, as its occupational classification is unspecific in many groups e.g., mechanics. Asbestos exposure has probably occurred as background exposure at low levels in many mechanical industries, but the JEM just classify occupations where asbestos have been handled.

5. Discussion

Table 4

| Maximum intensity | Number of individuals | HR | 95% CI |
|-------------------|-----------------------|----|--------|
| 0 fibres/ml       | 1341 | 19674 | 1.00  | Ref.  |
| >0–0.2 fibres/ml  | 672  | 4391  | 2.27  | 2.06–2.51 |
| >0.2–1.25 fibres/ml | 543  | 2886  | 2.26  | 2.48–3.07 |
| >1.25–2.0 fibres/ml | 201  | 619   | 4.81  | 4.06–5.70  |

* Maximum intensity is categorized based on the 50th and 90th percentiles of exposure distribution among exposed cases and controls.

Table 5

| Maximum intensity | Number of individuals | HR | 95% CI |
|-------------------|-----------------------|----|--------|
| 0 fibres/ml       | 1341 | 19674 | 1.00  | Ref.  |
| >0–0.1 fibres/ml  | 665  | 4348  | 2.27  | 2.06–2.51 |
| >0.1–0.8 fibres/ml | 724  | 3516  | 3.02  | 2.74–3.33 |
| >0.8–1.8 fibres/ml | 27   | 32    | 12.7  | 7.58–21.4 |

* Maximum intensity is categorized based on the 50th and 90th percentiles of exposure distribution among exposed cases and controls.

perchloroethylene, sulfur dioxide, 1,1,1-trichloroethane, and night-work had $p > 0.01$ and were also omitted.

Just 55.3% of the cases of mesothelioma of the pleura in our study were rated for asbestos exposure in the Swedish NOCCA-JEM. The number of women was too few to permit an analysis by gender.

Table 6

| NYK | Title | 1945–1959 | 1960–1974 | 1975–1984 | 1985–1994 |
|-----|-------|-----------|-----------|-----------|-----------|
|     | P     | L         | P         | L         | P         | L         |
| 631 | Railway engine drivers and assistants | 80 | 0.20 | 80 | 0.20 | 0 | 0.00 | 0 | 0.00 |
| 731 | Furnacemen | 5 | 0.05 | 5 | 0.05 | 5 | 0.01 | 5 | 0.01 |
| 751 | Machinery fitters, machine assemblers | 51 | 0.20 | 53 | 0.20 | 25 | 0.05 | 2 | 0.01 |
| 753 | Sheet metal workers | 30 | 2.00 | 30 | 1.00 | 30 | 0.20 | 0 | 0.00 |
| 754 | Plumbers and pipe fitters | 56 | 0.50 | 56 | 0.30 | 30 | 0.20 | 1 | 0.10 |
| 755 | Welders and flame cutters | 40 | 2.00 | 40 | 1.00 | 25 | 0.20 | 0 | 0.00 |
| 761 | Electrical fitters and wiremen | 40 | 0.03 | 50 | 0.04 | 10 | 0.02 | 2 | 0.01 |
| 769 | Nonspecified electrical and electronics work | 10 | 0.03 | 20 | 0.05 | 15 | 0.02 | 0 | 0.02 |
| 771 | Construction carpenters and joiners | 40 | 0.50 | 70 | 1.00 | 30 | 0.40 | 2 | 0.10 |
| 781 | Painters | 10 | 0.05 | 25 | 0.14 | 10 | 0.05 | 2 | 0.02 |
| 791 | Bricklayers | 40 | 0.30 | 50 | 0.31 | 30 | 0.10 | 5 | 0.05 |
| 794 | Insulators | 90 | 2.00 | 89 | 1.50 | 70 | 2.00 | 50 | 0.08 |
| 793 | Concrete and construction workers | 40 | 1.00 | 80 | 1.00 | 25 | 0.40 | 1 | 0.02 |
| 799 | Nonspecified other building and construction work | 10 | 1.00 | 21 | 1.25 | 10 | 0.40 | 2 | 0.06 |
| 811 | Glass formers and cutters | 20 | 0.20 | 20 | 0.20 | 15 | 0.10 | 0 | 0.00 |
| 836 | Paper and paperboard workers | 20 | 0.01 | 20 | 0.01 | 10 | 0.01 | 0 | 0.00 |
| 838 | Chemical and cellulose processing work n.e.c. | 5 | 0.08 | 5 | 0.06 | 5 | 0.05 | 0 | 0.00 |
| 872 | Crane and hoist operators | 25 | 0.10 | 25 | 0.10 | 25 | 0.10 | 0 | 0.00 |
| 873 | Riggers and cable splicers | 41 | 0.08 | 41 | 0.08 | 20 | 0.02 | 0 | 0.00 |
| 883 | Store and warehouse workers | 0 | 0.00 | 3 | 0.02 | 0 | 0.02 | 0 | 0.00 |
| 913 | Chimney sweeps | 80 | 0.10 | 80 | 0.10 | 80 | 0.02 | 80 | 0.00 |

NYK – Nordisk Yrkesklassificering.
Exposures are categorized based on 50th and 90th percentile of cumulative exposure distribution among exposed mesothelioma cases and controls.

The high HR we observed in the highest exposure category of bitumen fumes was unexpected. Bitumen exposure is common among asphalt workers, but this group is generally exposed to multiple agents, as are workers in many other occupations. In some occupations, individuals were classified for both bitumen and asbestos exposure, e.g., insulators and chemical and cellulose process workers; thus, we cannot exclude the possibility of misclassification.

The association we found between exposure to trichloroethylene and the risk of mesothelioma of the pleura was also unexpected, and the mechanism behind it is unclear. Simultaneous exposure to asbestos and other occupational carcinogenic agents or misclassification of occupations/exposures cannot be entirely ruled out. Another limitation of the Swedish NOCCA-JEM is that occupation/exposure was only available every 10 years. Moreover, a JEM is a rather crude tool for exposure assessment, as occupations are only surrogates of exposure. Some occupations have high exposure prevalence, e.g., painters for solvent exposure during the 1960s and 1970s, and are a good fit for the JEM, whereas others are more complicated. Mechanic or process workers are large occupational groups with different or multiple exposures, which can lead to lower exposure prevalence and an increased risk of misclassification. Ship building workers and dock workers are also included in this group. A proportion of the mechanics in the mechanical industry have been exposed to trichloroethylene through degreasing and to asbestos fibers through other activities. There are 283 occupations in the NOCCA-JEM, but only 21 of them had a relevant prevalence of asbestos exposure. Each occupational category consists of a large number of job titles, some of which could be exposed to asbestos but were diluted in the occupational category and did not fulfill our definition of exposure in the Swedish NOCCA-JEM. Other occupations had broad definitions. This misclassification can bias HRs towards the null.

Asbestos was banned in Sweden in 1982 [9], after which exposure levels and the number of exposed individuals decreased dramatically. We chose a 50-year latency period in our analysis due to the exposure pattern in the Swedish NOCCA-JEM, which ended in 1994. The latency time for mesothelioma is up to 40 years [33,34].

We used rather simple rules: The variable must be either statistically significantly associated or consistently associated with mesothelioma, and it could not be highly correlated with other variables. Consistency means the levels of exposure should show a dose–response relationship, i.e., variables should be rational considering their effect on cancer.

The main limitation of our study is the inevitable potential for exposure misclassification, which may arise from two sources. First, JEMs cannot account for exposure heterogeneity within jobs in an occupational category [35]. Second, because work history in our study was based on census records, we had information on profession only every 10 years; we did not know about every change in profession only every 10 years; we did not know about every change in occupational category and did not fulfill our definition of exposure in the Swedish NOCCA-JEM. Other occupations had broad definitions. This misclassification can bias HRs towards the null.

We found the pattern in time/years since first exposure to asbestos; the highest group (aged 50 + years) had a weaker relationship. That may be due to the insufficient nature of older information. Maximum exposure intensity was the analysis that confirmed a dose–response relationship between asbestos exposure and pleura mesothelioma. However, this study supports the existence of a dose–response relationship between asbestos exposure with risk of mesothelioma of the pleura.

6. Conclusions

We observed a significant, dose–response relationship between maximum intensity asbestos exposure and mesothelioma of the
pleura and cumulative asbestos exposure with 30-, 40-, and 50-years lag time. Cumulative exposure to asbestos, even at low levels, entailed an increased risk of mesothelioma of the pleura, indicating that even short periods with cumulative doses <1.78 f-g/ml can increase the risk of mesothelioma. Time since first exposure did not show any sufficient dose—response relationship in the longest lag period (>50 years).

Conflicts of interest

The authors have no competing interests to declare.

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