

EPIDEMIOLOGICAL INVESTIGATIONS OF THE FIBRE CEMENT INDUSTRY IN THE FEDERAL REPUBLIC OF GERMANY (1981–1986)

E. G. BECK • R.-H. Bödeker • P. Schmidt

Institute of Hygiene and Institute for Medical Information
University of Giessen (FRG)

INTRODUCTION

The present study is based on an analysis of death causes in 9 plants of the Association of the Fibre Cement Industries in the Federal Republic of Germany. These represent the entire West-German fibre cement industry. It is a continuation of the epidemiological investigations conducted in 1976–1980.^{1,2,3} In order to extend the conclusions drawn from the results of the first study phase (1976–1980) it was followed by a second phase (1981–1986) for comparison and crosschecking;⁴ in this case the registered death cases were already considered following a minimal exposure period of 5 years as compared to 10 years in the first study. Of the workers exposed for at least 5 years, 325 cases of the death were registered by the plants of the Association of the Fibre Cement Industries between January 1, 1981, and December 31, 1986. A total of 290 cases were recorded. The remaining 35 cases could not be included in the investigation due to the lacking consent of the next of kin until the beginning of the evaluation. Thus a response rate of 89% was obtained. Furthermore, not all criteria of interest could be ascertained leading to different sample sizes for the individual criteria. As there was only one female worker among the deceased, as 4 cases were already reported in 1980 and as the duration of exposure was less than 5 years in 5 cases, only 280 cases could be included in the evaluation.

METHODS

In the present communication it was attempted to analyse the proportional mortality rates (SPMR) with regard to the ICD classification of the observed death causes in order to derive indications concerning the health hazards due to asbestos exposure of the employees in the asbestos industry. In addition this allows a comparison with the results of the study executed in 1976–1980. The SPMR was determined according to the method of Rao and Marsh.⁸ The mortality of the total male population (German Federal Office of Statistics) was adjusted according to the age of the test population. For the calculation of the probability that the various diagnoses or diagnostic groups occurred at the observed rate or at more extreme frequencies, it was assumed that the observed instances followed a Poisson distribution.

RESULTS

Of the registered 280 death cases 160 died in hospital and 118 at home, of 2 cases the location was unknown. The dates were taken from the following documents or records: 280

death certificates, 156 medical records (56%), 73 pathological reports (26%) and 81 histological reports (34%); an occupational disease was recorded in only 10 cases (3.6%). The average age at entry into employment of the employees of the first phase was 50 years and of the second phase $x = 42.5$ years (median = 44 years).

The average age at death is $x = 69.5$ with a median of 72 years; the exposure time $x = 18.7$, median = 17 years, and the survival time (time from start of exposure to death) $x = 27.2$, median = 27 years. The employment and survival times of the registered deceased correspond approximately to earlier data on the actual exposure time and also confirm the results of the first phase of our study. With regard to the distribution of the death causes according to the ICD classification the diseases of the cardiovascular system (ICD 460–519) are in the foreground, followed by malignant neoplasms of all organs (ICD 140–239) and the diseases of the respiratory organs (ICD 460–519). Firm distinctions between the diseases of the circulatory and respiratory systems cannot be drawn. This corresponds to expectation and does not differ essentially from the most frequent diseases of the total male population of the FRG and thus confirms the results of the first study period as well.^{1,2,3}

Malignant neoplasms of the respiratory system (ICD 160165) were registered in 29 deceased, among them pleural mesotheliomas (ICD 163) in 7 and malignancies of the digestive tract and peritoneum (ICD 150159) in 9 cases. In 21 of the deceased workers asbestosis was diagnosed. This agrees with previous experience concerning the incidence of this disease in the asbestos cement industry during the time without sufficient dust protection measures.

For the evaluation of the incidence of the observed death causes the SPMR was calculated. A clear excess incidence (SPMR 1.7, $p < 0.01$) compared to the general population was noted with regard to respiratory diseases excluding cancer (ICD 460–519). The incidence of bronchitis, bronchial asthma and lung emphysema (ICD 490–493) is even higher (SPMR 2.1, $p < 0.001$). This is typical of industries with high dust concentrations (Table I). Malignancies of the respiratory organs (ICD 160–165) are more frequent in the exposed group than in the general male population (SPMR 1.36, $p = 0.09$). Less frequent are the total malignant neoplasms (ICD 140–239) in asbestos workers; this lesser incidence leads to a SPMR of 0.77 and $p = 0.05$. Similarly there is a clearly less frequent incidence of cancer of the

Table I
Analysis of the Proportional Mortality (n=262) 1981-1986

ICD-Classification	Observed*	Expected	SPMR	(chi) ²	p
390-459 Diseases of the cardiovascular system	103	125.2	0.82	3.9	0.048
140-239 Total malignant neoplasms	55	71.5	0.77	3.8	0.050
160-165 Malignant neoplasms of the respiratory organs	29	21.3	1.36	2.8	0.095
160-162, 164-165 Malignant neoplasms of the respiratory organs without mesotheliomas	22				
163 Pleural mesotheliomas	7				
150-159 Malignant neoplasms of the digestive tract and peritoneum	9	23.1	0.39	8.6	0.004
150-157, 159 Malignant neoplasms of the digestive tract	9				
158 Peritoneal mesotheliomas	-				
460-519 Diseases of the respiratory organs without malignant neoplasms	33	18.7	1.7	10.9	<0.001
490-493 Bronchitis, emphysema, asthma bronchiale	25	11.9	2.1	14.4	<0.001
501 Asbestosis	5				
800-999 Accidents	4				
303, 570-577 Alcoholism, hepatic cirrhosis	12	46.6	1.52	12.7	<0.001
Other death causes	55				

The calculation of the expected values is based on the distribution of age at death in 1981 - 1986. * 18 cases are excluded due to lacking data concerning age at death or year of death; p is stated in a two-tailed manner.

digestive organs and the peritoneum (ICD 150-159; SPMR 0.39, $p = 0.48$). The same behaviour can be observed in diseases of the circulatory system (ICD 390-459) with a SPMR of 0.82, $p = 0.48$.

DISCUSSION

The average age at death (72 years) of the deceased employees in our study does not essentially differ from the average age at death of the general population of the FRG. The mean age at entry into employment of the group of the first study period was 50 years, 50% of these had an age of 40-55 years. It was 42.5 years in the employees of the second phase of the study with a 50% interquartile of 36-50 years.⁴ Why the mean age at entry of the employees in the asbestos cement industry is relatively high, especially in the first phase, could not be elucidated. There was a critical discussion that this elevated age at entry impairs the significance of the results of the first phase in the sense that the tumour expectancy would be significantly greater with an earlier age at entry. However, the age correlated expected value has been used for the calculation of the SPMR. In addition, this argument can be countered with results of other authors. In the study by Neuberger et al.⁷ the age at the beginning of employment was higher in asbestos-associated cancer death cases than in the not asbestos-associated death cases. In their evaluation of the results of 11 international epidemiological studies of lung cancer mortality in employees of the asbestos cement industry, Gardner and Powell⁵ stated latency periods from first exposure to tumour manifestation

of at least 5 and maximally 20 years. This is in accordance with the concept of the present study where a minimal exposure time of 5 years has been used as criteria of inclusion and in which an exposure time of 20 years could be observed.

The present investigations of the first as well as the second study phase confirm essentially the results of Lacquet et al.⁶ and of Weill et al.¹¹ Thomas et al.¹⁰ examined a cohort of comparable size of deceased employees of the British asbestos cement industry and obtained results largely in accordance with those of the present study, particularly with regard to the expected and observed cases of all malignant neoplasms and of cancer of the respiratory organs and the digestive system. The studies by Ohlson and Hogstedt⁸ and Gardner and Powell⁵ are also in agreement.

Neuberger et al.⁷ conducted a recent investigation of ca. 2800 employees of the Austrian asbestos cement industry using the general population as controls just as in the present study. Based on official death certificates death from lung cancer was diagnosed in 535 cases exceeding the mortality of the age and sex adjusted general population by 1.7. This excess mortality was explained by the authors as due to the higher tobacco consumption of the employees as compared to the general population. In the same study 5 cases of mesothelioma were demonstrated and attributed to the earlier use of crocidolite in the production of asbestos cement pipes. The findings of the present study also point to a possible correlation between mixed dust exposure with crocidolite and chrysotile and the incidence of 7 pleural mesotheliomas

($p = 0.08$). In the review by Gardner and Powell⁵ cited above this is also stressed with regard to the SPMR of lung cancer which lies in the range of 5.2–0.8 in plants with previous mixed dust exposure (chrysotile and crocidolite) and 1.5–0.9 in those with pure chrysotile exposure, while the SPMR of the present investigation is 1.4. Of the three mesotheliomas listed in the review two are attributed to mixed exposure with chrysotile and crocidolite and one to exposure with chrysotile and amosite.

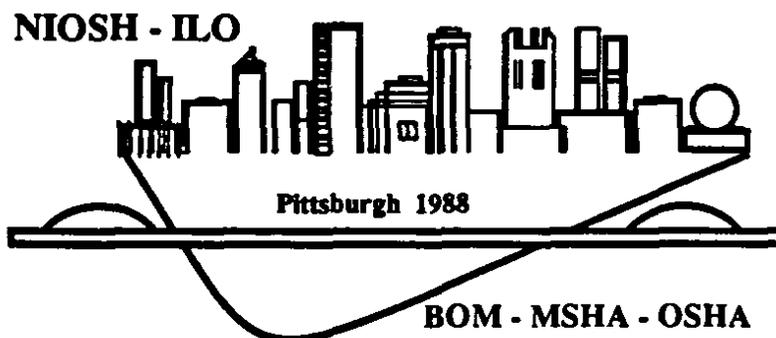
According to these results and those of the authors cited above a distinction should be made between crocidolite and chrysotile asbestos with respect to their carcinogenic effects. Thus inhalable crocidolite fibres possess a stronger oncogenic potential than the corresponding chrysotile fibres. The higher durability of crocidolite in the reacting tissue may explain its pathogenetic importance for the development of pleural or peritoneal mesothelioma. Under present-day working conditions and with the considerably restricted use of crocidolite only for exceptional technical products (pipes) a reduction of the increased mesothelioma hazard appears possible. With the sole exposure to chrysotile the relative risk of mesothelioma is expected to be low (see also Neuberger et al.)⁷

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