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Mortality study of asbestos cement workers

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Abstract The present study describes cause-specific mortality of asbestos cement workers in the Emilia Romagna region of Italy. The cohort included workers in ten factories, most of which started operating between 1955 and 1965. Asbestos, mainly chrysotile, constituted 10%– 20% of the dry component of the mixture. Crocidolite range between 5% and 50% of total asbestos. Asbestos concentrations up to 44 ff/cc were reported prior to 1975, while in recent years they have usually been below 0–1 ff/cc. The cohort included 3341 workers who had at some time been employed in the ten factories under study. Their mortality experience was compared with that of the population resident in Emilia Romagna. Vital status was ascertained at 1989. Seventy-three subjects were lost to follow-up (2.2%). Mortality from all causes and from all types of cancer was increased in the cohort. Malignant neoplasms of the respiratory tract showed a significant increase (SMR: 134; 90% confidence interval: 101–175; 40 observed) due to lung cancer (SMR: 124; 90% confidence interval: 91–166; 33 observed) and neoplasms of the pleura, mediastinum, and other parts of the respiratory tract (SMR: 602; 90% confidence interval 237–1267; 5 observed). The discrepancy between observed and expected mortality mainly concerned subjects with at least 20 years of employment in the factories. Five more cases of histologically confirmed mesothelioma occurred after the end of follow-up.

Key words Asbestos · Cohort study · Lung cancer
Pleural mesothelioma · Occupational mortality

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Introduction

The asbestos cement industry has been the subject of a number of epidemiologic investigations, mainly cohort studies. In Canada, Finkelstein [7, 8] reported an increased risk of all cancer (especially respiratory and digestive tract neoplasms) and the occurrence of both pleural and peritoneal mesothelioma in asbestos cement workers. In France, Alies-Patin et al. [3] reported an increased risk for all cancer and lung cancer Hugues et al. [11] described an excess of lung cancer and the occurrence of some cases of mesothelioma in a factory in the United States. No excess mortality was found by Gardner et al. [10] in the United Kingdom. Increased risk for mesothelioma, but not for lung cancer (after correction for smoking), was reported in Austria by Neuberger and Kundi [17]. No major increase in lung cancer mortality was reported in Sweden by Ohlson and Hogstedt [18], while Albin et al. described a cohort with increased mortality and incidence of all cancer and respiratory cancer, showing a dose-response relationship for pleural mesothelioma [2]. In Denmark, an increased incidence of cancer of the lung, pleura, and mediastinum was reported, together with an increased mortality; the digestive tract and other sites were also at risk [20]. In Italy, a study by Magnani et al. [14] described an increased risk of all cancer, lung cancer, and pleural and peritoneal mesothelioma in a facility located in Piedmont; the same authors reported an increased risk for pleural mesothelioma among the population resident in the area, even in the absence of occupational exposure to asbestos [13].

The second major settlement of the asbestos cement industry in Italy (after the aforementioned facility in Piedmont) is located in Emilia Romagna, the region around Bologna; ten factories and many craftsmen's shops have operated in this region since the mid-1950s.

The purpose of the present study was to investigate the mortality of the asbestos cement workers in Emilia Romagna in order to clarify further the cancer risk associated with this industrial activity. Notwithstanding the existence of the aformentioned previous epidemiologic investigations, it was felt that this was a valuable opportunity for the implementation of a new study, given the fair size of the study population, the good quality of record keeping and death registration in the area, and the active cooperation of health authorities, companies, and trade unions.

Materials and methods

The asbestos cement industry in Emilia Romagna includes ten factories and many craftsmen's shops; the latter were not included in the study. Six factories started between 1952 and 1962, while the other four started between 1965 and 1973. The number of workers employed in these factories was about 800 around 1970 and is now close to 400 in the six plants still operating. Pipes, slabs, tanks, chimneys, and other products for the construction industry were manufactured in these factories.

Table 1 reports some information on the historic patterns of asbestos occurrence in the factories under study. Asbestos fibers, mainly chrysotile, constituted 10%–20% of the dry component of the mixture. Crocidolite ranged between 5% and 50% of total asbestos, and its use was discontinued between 1982 and 1986. Mixture preparation and finishing were the activities implying higher asbestos exposure; bags were manually opened and emptied until the 1980s. The operation causing the highest fiber release was probably asbestos grinding, which was performed in four factories only.

Table 1 Use of crocidolite and grinding of asbestos in the ten plants enrolled in the study

Plant	Year of starting work	Use of crocidolite	% of asbestos in the blend	
A	1965	1965–1980 1981–1982	Mixture[a] 30%	–
B	1968	–	–	–
C	1961	1961–1977	30%	Open air grinding Only one worker Exposed until 1979
D	1957	1957–1963 1964–1983	Mixture[a] 4%–22%	–
E	1955	1955–1979 1980–1986	Mixture[a] 4%–30%	Grinding in protected conditions since 1977
F	1952	1952–1984	30%	Open air grinding from 1956 to 1970
G	1962	1962–1985	15%–50%	Open air grinding from 1962 to 1966
H	1969	1970–1979	5%	–
I	1954	1954–1982	15%	–
L	1973	1973–1985	20%	–

[a] Commercial mixture not prepared by the plant, unknown percentage

Table 2 Median asbestos concentration in air (ff/cc) for different activities and in different calendar years (from Poletti et al. [19])

	1983–84				1975–76[a]
	No. of samples	Median	Min.	Max.	Median
Mixture preparation					
Manual	23	0.99	0.30	9.70	4.20
Automatic	23	0.81	0.51	2.23	ND
Manufacturing					
Pipes, slabs	16	0.20	0.18	0.35	0.50
Other products	27	0.24	0.12	0.60	ND
Trimming or finishing	20	0.52	0.35	0.72	2

ND, not determined

[a] Based on about 20 samples

Asbestos concentrations were measured in different work stages and time periods [19]; median values are shown in Table 2. No systematic hygienic monitoring had been performed prior to 1975, but up to 40 ff/cc had been reported with respect to manual opening of asbestos bags.

Measurements performed in 1988–91 constantly showed asbestos concentrations below 0.5 ff/cc, in most instances below 0.1 ff/cc.

The cohort included all subjects who had ever been employed in the factories under study between the start of their operation (or, alternatively, from the earliest year for which company files were available) and 1987. Vital status was ascertained at 30 June 1989. Causes of death were coded according to the VIIIth and IXth Revisions of the International Classification of Diseases, depending on whether death occurred before or after 1980.

Standardized mortality ratios (SMRs) were computed using the Life Table Analysis System Computer Program [24] and their 90% confidence intervals (CIs) were computed according to Poisson distribution. The choice of using 90% confidence was taken a priori in order to decrease the impression of falsely negative results [1]. The number of expected deaths was obtained using cause-, age-, sex-, and calendar time-specific mortality rates of the population resident in Emilia Romagna.

Results

At the end of follow-up, 2994 subjects were alive, 274 were deceased, and vital status was unknown for the remaining 73 subjects, corresponding to 2.2% of the total cohort. Lost subjects were regarded as being alive at the end of follow-up. It was not possible to ascertain the cause of death for five subjects, corresponding to 1.8% of the deceased.

Table 3 shows observed and expected mortality in the cohort. Mortality from all causes and from all cancers was increased. Malignant neoplasms of the respiratory tract showed a significant increase (SMR: 134; 90% CI: 101–175) due to lung cancer (SMR: 124; 90% CI: 91–166) and to neoplasms of pleura, mediastinum, and other parts of the respiratory tract (SMR: 602; 90% CI: 237–1267). A significant increase in "other malignant neoplasms", including nine cases of ill-defined neoplasms, was detected as well.

Table 3 Observed and expected mortality in the cohort

	Obs.	Exp.	SMR	90% CI
All causes	274	255.66	107	97– 118
All malignant neoplasms	103	91.87	112	95– 132
Mouth and pharynx	0	2.18	0	0– 137
Digestive tract and peritoneum	28	30.80	91	65– 125
Peritoneum	1	0.63	159	8– 753
Respiratory tract	40	29.81	134	101– 175
Larynx	2	2.43	82	15– 259
Trachea, bronchi, and lungs	33	26.54	124	91– 166
Pleura, mediastinum, and other parts of respiratory tract	5	0.83	602	237–1267
Prostate	0	2.45	0	0– 122
Urinary tract	5	4.81	104	41– 219
Hematopoietic system	7	6.88	102	48– 191
Others	23	14.95	154	105– 218
Diabetes mellitus	1	3.37	30	2– 141
Circulatory diseases	75	88.14	85	70– 103
Respiratory diseases	13	11.10	117	69– 186
Hepatic cirrhosis	4	12.18	33	11– 75
Ill-defined and missing causes	8	2.05	390	194– 704
Accidents	45	29.76	151	116– 194
Other causes	25	17.19	145	101– 203

Table 4 Mortality from respiratory cancer over time

	–1974	1975–79	1980–84	1985–89	1955–89
Obs.	3	5	8	24	40
Exp.	2.61	5.50	9.62	12.08	29.81
SMR	115	91	83	199	134
90% CI	31–297	36–191	41–150	137–279	101–175

Mortality from cancer of the digestive tract was not increased, even if a case of peritoneal neoplasm was reported.

Circulatory disease and hepatic cirrhosis showed a deficit with respect to expected figures. There were 13 deaths due to respiratory disease versus 11 expected. One of these cases was certified as being affected by asbestosis, which was reported as a concurrent cause on three other certificates. Finally, significant increases in mortality from ill-defined and missing causes, accidents, and other causes were detected.

The increased mortality from respiratory cancer was further investigated. Table 4 shows that the number of observed cases significantly exceeded the expected number only in the most recent years. The epidemic curve thus shows that a significant excess of respiratory cancer occurred about 30 years after the start of operations and thus the beginning of exposure to asbestos. This finding is supported by the fact that the discrepancy between observed and expected cases essentially concerned subjects who had worked in the factories for at least 20 years (Table 5).

Table 5 Mortality from respiratory cancer by duration of exposure and latency time

Latency (years)	Exposure (years)				Total
	≤ 10	11–20	21–30	≥ 30	
≤ 10					
Obs.	7	0	0	0	7
Exp.	6.68	0	0	0	6.68
11–20					
Obs.	9	4	0	0	13
Exp.	8.79	4.71	0	0	13.5
21–30					
Obs.	7	8	3	0	18
Exp.	4.87	2.61	1.52	0	9.01
≥ 30					
Obs.	0	2	0	0	2
Exp.	0.03	0.46	0.1	0.03	0.62
Total					
Obs.	23	14	3	0	40
Exp.	20.37	7.78	1.63	0.03	29.81

Table 6 Observed (O) exprected (E) deaths from respiratory cancer in the different plants by latency time

Plant	Latency ≤ 20 years		Latency > 20 years	
	O	E	O	E
A	1	0.45	–	–
B	2	0.36	–	–
C	3	3.61	3	1.94
D	3	1.87	2	0.44
E	5	5.47	4	2.99
F	4	3.54	8	3.37
G	1	1.41	3	0.50
H	1	1.39	–	–
I	–	1.69	–	0.36
L	–	0.36	–	–
All	20	20.17	20	9.63

Table 6 shows observed and expected cases in the individual subcohorts, taking into account time since start of work. Notwithstanding low statistical power due to the limited sample size, it was possible to evidence some points of interest. When the four factories characterized by open air grinding (Table 1) were grouped, the SMR rose to 149 (90% CI: 108–201), while in the remaining factories nine cases were observed versus 8.98 expected. Two cases of pleural tumor, one case of pericardial tumor, and one case of peritoneal tumor came from the same factory, (factory F), in which, furthermore, nine cases of lung cancer were observed versus six expected.

Five more cases of histologically confirmed pleural mesothelioma were reported after the end of follow-up, and at least ten more deaths occurred due to respiratory cancer.

Discussion

The present study does not appear to be affected by major biases. The study population was derived from company files and it is reasonable to assume that it was complete; the proportion of subjects lost to follow-up (2.2%) is in agreement with current standards of epidemiologic research.

The issues concerning quality of death certification are crucial, especially when dealing with asbestos-induced diseases. Misclassification is known to affect the reporting of pleural mesothelioma on death certificates: the proportion of misclassified cases in Italy has been estimated by Delendi et al. [4] as close to 50%. Such misclassification is a random phenomenon and does not appear to be conditional on asbestos exposure, so that it may presumably result in an underestimation of the SMR. In the present study, the excess mortality from ill-defined cancer is consistent with the occurrence of unreported cases of mesothelioma.

The reference population whose mortality rates were employed in the computation of expected figures is that of the Emilia Romagna region. This choice ensures overall comparability, while the observed deficit in circulatory disease and hepatic cirrhosis may be indicative of a "healthy worker effect".

No data on smoking habits at an individual level were available in this study. This problem is commonly met in occupational cohort studies, and has been the object of several studies (for a review, see Steenland et al. [23]). In order to evaluate whether smoking habit may have been an actual confounding variable, some points should be considered. First of all, the excess in lung cancer mortality that was reported occurred together with the detection of an excess mortality from pleural neoplasms, and an etiologic role of asbestos exposure on both cancer sites is well known. Furthermore, in the experience of the occupational physicians who were in charge of medical surveillance in the factories studied, the smoking habits of asbestos cement workers seemed to be comparable to those of other working populations of the same areas. Finally, if the excess of lung cancer had been due to the presence of a higher cigarette consumption among cohort members in comparison with the regional population, one would have expected increases in mortality from other smoking-related causes, such as respiratory disease, circulatory disease, and laryngeal and bladder cancer, which in fact did not occur. It can thus be suggested that smoking habits did not play an appreciable confoudning role in the present cohort study.

The present findings seems to be consistent with those of other authors who have reported increased mortality from lung and pleural cancer in the asbestos cement industry, and with those on other asbestos-related industries, especially allowing for induction-latency time [3, 7, 8, 11, 15, 16, 21]. The situation was considerably less severe than in the other Italian study, which concerned the Piedmont asbestos cement industry, where deaths from pleural and peritoneal neoplasms had numbered, respectively, 28 and 10 in men (2 expected) and 15 and 4 in women (0.6 expected) [14].

On the other hand, no excess was detected in respect of some other neoplasms whose association with asbestos exposure is still being debated. Mortality from laryngeal cancer was not increased in this cohort, analogously to reports in other cohorts of asbestos workers (see reviews by Liddell and Edelman [6, 12], even if a causal role of asbestos exposure in the induction of laryngeal neoplasms has recently been suggested [22]. Furthermore, no excess was seen with regard to gastrointestinal tract neoplasms, in agreement with the findings reviewed by Edelman [5]; it should be noted, though, that other authors, such as Frumkin and Berlin [9], suggest that elevated exposures to asbestos are associated with an increased risk of digestive tract cancer, implying, indicatively, exposures causing a twofold increase in lung cancer.

In conclusion, the asbestos cement workers of Emilia Romagna show an increased mortality from respiratory cancer. The discrepancy between expected and observed mortality started in the mid 1980s, and there have been several new cases of mesothelioma subsequent to the end of follow-up. Most of the cases have so far been detected in the factories that were the first to start work, and in which dusty operations such as open air grinding of asbestos and dry cutting of pipes took place. The detection of an increased occurrence of respiratory cancer in the study population and the study of its trend over time are of interest in the evaluation of cancer risk associated with asbestos cement facilities. On-going epidemiologic surveillance will provide further information on the occurrence of respiratory cancer in the cohort and on the role of various etiologic agents, including smoking habits, thereby supplementing the body of knowledge provided by the present investigation.

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References

1. Ahlbom A, Axelson O, Hansen ES, Hogstedt C, Jensen UI, Olsen J (1990) Interpretation of "negative studies" in occupational epidemiology. Scand J Work Environ Health 16:153–157
2. Albin M, Jakobsson K, Attewell R, Johansson L, Welinder H (1990) Mortality and cancer morbidity in cohorts of asbestos cement workers and referents. Br J Ind Med 47:602–610
3. Alies-Patin AM, Valleron AJ (1985) Mortality of workers in a French asbestos cement factory 1940–1982. Br J Ind Med 42:219–225
4. Delendi M, Riboli E, Peruzzo P, Stanta G, Cocchi A, Gardiman D, Sasco AJ, Giarelli L (1991) Comparison of diagnoses of cancers of the respiratory system on death certificates and autopsy. In: Riboli E, Delendi M (eds) Autopsy in epidemiology and medical research. International Agency for Research on Cancer, Lyon, Scientific Publication 112:55–62
5. Edelman DA (1988) Exposure to asbestos and the risk of gastrointestinal cancer: a reassessment. Br J Ind Med 45:75–82
6. Edelman DA (1989) Laryngeal cancer and occupational exposure to asbestos. Int Arch Occup Environ Health 61:223–227

7. Finkelstein MM (1983) Mortality among long-term employees of an Ontario asbestos cement factory. Br J Ind Med 40:138–144
8. Finkelstein MM (1984) Mortality among employees of an Ontario asbestos cement factory. Am Rev Respir Dis 129:754–761
9. Frumkin H, Berlin J (1988) Asbestos exposure and gastrointestinal malignancy review and meta-analysis. Am J Ind Med 14:79–95
10. Gardner MJ, Winter PD, Bannett B, Powell CA (1986) Follow-up study of workers manufacturing chrysotile asbestos cement products. Br J Ind Med 43:726–732
11. Hughes JM, Weill H, Hammad YY (1987) Mortality of workers employed in two asbestos cement manufacturing plants. Br J Ind Med 44:161–174
12. Liddell FDK (1990) Laryngeal cancer and asbestos. Br J Ind Med 47:289–291
13. Magnani C, Borgo G, Betta GP, Botta M, Ivaldi C, Mollo F, Scelzi M, Terracini B (1991) Mesothelioma and non-occupational environmental exposure to asbestos. Lancet 330:50
14. Magnani C, Terracini B, Bertolone GP, Castagneto B, Cocito V, De Giovanni D, Paglieri P, Botta M (1987) Mortalità per tumori e altre malattie del sistema respiratorio tra i lavoratori del cemento amianto a Casale Monferrato. Uno studio di coorte storico. Med Lav 78:441–453
15. McDonald AD, Fry JS, Woolley AJ, McDonald JC (1983) Dust exposure and mortality in an American chrysotile textile plant. Br J Ind Med 40:361–367
16. McDonald JC, Liddell FDK, Gibbs GW, Eysen GE, McDonald AD (1980) Dust exposure and mortality in chrysotile mining, 1910–75. Br J Ind Med 37:11–24
17. Neuberger M, Kundi M (1990) Individual asbestos exposure: smoking and mortality – a cohort study in the asbestos cement industry. Br J Ind Med 47:615–620
18. Ohlson CG, Hogstedt C (1985) Lung cancer among asbestos cement workers. A Swedish cohort study and a review. Br J Ind Med 42:397–402
19. Poletti M, Vecchi G, Ziccardi A (1987) Un'esperienza di prevenzione nel comparto dei manufatti in cemento-amianto della Provincia di Reggio Emilia. In: Parolari G, Gherson G, Cristofolini A, Merler E (eds) Il rischio neoplastico da amianto nei luoghi di lavoro e nell'ambiente di vita. Bi & Gi Editori, Verona, pp 171–179
20. Raffn E, Lynge E, Juel K, Korsgaard B (1989) Incidence of cancer and mortality among employees in the asbestos cement industry in Denmark. Br J Ind Med 46:90–96
21. Selikoff IJ, Seidman H (1991) Asbestos-associated deaths among insulation workers in the United States and Canada, 1967–1987. Ann NY Acad Sci 643:1–14
22. Smith AH, Handley MA, Wood R (1990) Epidemiological evidence indicates asbestos causes laryngeal cancer. J Occup Med 32:499–507
23. Steenland K, Beaumont J, Halperin W (1984) Methods of control of smoking in occupational cohort studies. Scand J Work Environ Health 10:143–149
24. Waxweiler RJ, Beaumont JJ, Henry JA (1983) A modified life table analysis system for cohort studies. J Occup Med 25:115–124