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MORTALITY OF WORKERS AT TWO ASBESTOS-CEMENT PLANTS IN POLAND

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Abstract. To assess mortality rate among workers occupationally exposed to asbestos, cohort studies were carried out in two asbestos cement plants operating since the 1960s. Asbestos cement sheets for roofing and siding have been manufactured there, using mostly chrisotile, and since 1985 also crocidolite for pressure pipes. In all, the cohort comprised 3,220 workers, including 2,616 male workers. Subject to consideration were the workers employed for at least three months in the period between the onset of the production and 1980. The vital status of the subjects was traced up to 31 December 1991. The availability of the cohort was 96.8%. Workers' mortality was analysed using standardized mortality ratio (SMR). The reference group was the general population of Poland. In the male cohort, 385 cases of death were recorded. Statistically significant excess of mortality from large intestine cancer (7 cases, SMR = 264) and pleural mesothelioma (5 cases, SMR = 2846) was found. In male workers who died from pleural mesothelioma the work history ranged from 12 to 26 years. An excess mortality from pleural mesothelioma was also noted among the female workers (2 cases, SMR = 11,275). No malignant neoplasms of other locations produced significant excess mortality either in the male or female workers.

INTRODUCTION

In Poland, the manufacture and distribution of asbestos and asbestos cement products was banned in 1998. Therefore, the problems of monitoring asbestos concentrations as well as the health effects of exposure in asbestos workers have practically ceased to exist. However, of much concern are still the delayed effects of occupational exposure to asbestos which may not be manifested until 30 – 40 years since the first exposure. Besides, the environmental contamination in the vicinity of asbestos and asbestos cement plants, where high air concentrations of asbestos fibres are persistent, poses a serious hazard to the population. In view of the common use of asbestos cement products in the building industry and the resulting environmental exposure, the health effects of exposure to asbestos cement dust are among the priorities of the public health policy.

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In Poland, asbestos products started to be commonly used as the building material in the 1960s when four large asbestos cement plants were founded. (Two of these plants were considered in our study – one was opened in 1959, the other in 1965). They were roughly similar in respect of the annual average number of employees, but differed in the proportion of female to male workers – 15% and 42%, respectively. Both manufactured a wide range of asbestos cement products, including sheets, profiles, ridge tiles and pressure pipes. Asbestos cement was used mainly as a roofing material due to its high resistance to fire, corrosion and decay, low weight, good isolation and soundproof properties, and relatively low cost of manufacture. The prevailing type of asbestos processed in the two plants was chrisotile, imported mainly from Russia, but also from Canada and China. To a lower extent, crocidolite imported from South Africa, and amosite, were also used (Table 1).

Table 1. Characteristics of the asbestos-cement plants under study

Characteristics	Plant I	Plant II
Year of foundation	1959	1965
Annual employment	450	405
of which: female workers	15%	42%
Annual mean use of asbestos (tonnes)	11000	12000
Total use of asbestos until 1991 (tonnes)	350000	276000
of which: crocidolite	65000	6500

In both plants similar technologies were applied. At first, raw asbestos in jute or paper sacks was transported by rail to the plant. In the early years of production, asbestos in sacks was transferred to the store room on hand-operated trucks. These were later replaced with battery-powered trucks carrying asbestos sacks placed on wooden pallets. This improvement, however, did not reduce workers' exposure to asbestos dust. Particularly high asbestos concentrations were noted at the unloading of damaged sacks.

Until 1970, the 'dry' method was used in the manufacture of asbestos cement materials. Asbestos was defibrinated and then shovelled into the beater. This process brought about the exposure to high concentrations of asbestos dust. During the 1970s amphibole asbestos, crocidolite and amosite, were also used for pressure piping manufacture, making up 20% of the total asbestos content in the product. The production employing crocidolite and amosite was terminated in the middle 1980s. Since the 1970s the 'wet' method has been in use, which consists in the mixing of asbestos, cement and water to obtain the intermediate material to be further processed. Asbestos cement is then defibrinated in the beater with an addition of 15% of water. The defibrinated asbestos goes next to a hydropulper when it is centrifuged in copious amounts of water to obtain single fibres which make the structural framework of the building material.

The preparatory steps, including asbestos transportation, charging into beater and defibrination involve workers exposure to high concentrations of airborne asbestos fibres. Over more than 20 years, asbestos had been charged manually to the beater. The only protective measure was the use of face half-masks, not very effective protection against dust exposure. High concentration levels of asbestos dust were also recorded in the grinding of pressure pipes endings where the dry method was used.

In both plants under study, very high concentrations of asbestos dust were noted. Until the middle 1980s they were measured in mg/m^3. The measurements of the number of respirable asbestos fibres in the workplace atmosphere were seldom carried out. One may assume that during the period when the dry method was used, the fibre concentration would exceed several fibres per cm^3, and at some workposts it could even reach several dozen fibres. The introduction of the wet method has contributed largely to the reduction of asbestos dust concentration. However, in 1991, over the beater-floor and charging areas, asbestos concentrations as high as 8 fib/cm^3 could still be found.

The automatic charging now in use makes the concentration levels not to exceed the maximum admissible value of 0.5 fib/cm^3 for chrisotile asbestos, which was thought to be the safety threshold. Values lower than this are not likely to produce an increased risk of lung cancer (6,7,16,26).

METHODS

In order to assess the risk of death from asbestos-induced malignant neoplasms among industrial workers, a cohort study was performed on workers at two large asbestos cement plants. The criterion of classification for the cohort was at least a three-month employment during the period between the establishment of the plant (1959 – Plant I, and 1965 – Plant II, respectively) and 1980. For each worker meeting this criterion, the personal data regarding the date and place of birth, residence, employment period and workpost were elicited. Based on the civil statistics, the vital status of the workers was traced. The last day of the cohort's observation was 31 December 1991. At that time, the workers who had completed 80 years of age during the period under study were regarded as living; the end-point for the person-years calculation for these workers was the age of 80. The deceased workers had the date and place of death recorded. Then the cause of death was enquired about in relevant institutions and coded according to the 9th Revision of the International Classification of Diseases, (ICD-9). Death risk by causes was analysed using standardised mortality ratios (SMRs) calculated by the person-years (PYRS) method. For the cause of death for which two or more cases of death were recorded in the cohort, a 95% confidence interval (95% CI) for the SMR was calculated using Poisson distribution. The reference group for the assessment of death risk in the cohort was the general population of Poland. The calculations were performed using the PYRS package obtained from the International Agency for Research on Cancer (IARC). The analysis was conducted separately for male and female workers in the entire cohort and in subcohorts according to the length of employment.

RESULTS

The cohort comprised 3,220 persons (2,616 males and 604 females). The vital status was traced for 3,116 persons (2,525 males and 591 females), thus the cohort availability was 96.8% (Table 2).

One of the key parameters in the assessment of the cumulated lifetime dose is the duration of exposure. The cohort's characteristics by work history revealed that in 65% of male workers the length of employment was not longer than 10 years, and those employed below one year made up less than 25% of the workers examined.

Table 2. The cohort vital status and follow-up to 31 December 1991

	Males	Females
Potential study cohort	2616	604
Traced	2525 (96.5%)	591 (97.8%)
Alive	2140	567
Dead	385	24
Lost to follow-up	91 (3.5%)	13 (2.2%)

Table 3. Characteristics of the cohort of workers employed in the asbestos-cement plants

Characteristics	Workers			
	Male		Female	
	number	%	number	%
Employment (yrs)				
up to 1	581	23.0	72	12.2
1–4	717	28.4	168	28.4
5–9	338	13.4	100	16.9
10–19	583	23.1	163	27.6
20–	306	12.1	88	14.9
Workers' age on admission to the plant (yrs)				
–29	1580	62.6	406	68.7
30–39	561	22.2	131	22.2
40–49	289	11.4	48	8.1
50–	95	3.8	6	1.0
Period since the first employment in the plant (yrs)				
–9	73	2.9	4	0.7
10–19	952	37.7	222	37.6
20–29	1237	49.0	334	56.5
30–	263	10.4	31	5.2
Cohort in total				
persons	2525	100.0	591	100.0
person-years	54129		12664	

The female workers were characterised by a higher stability of employment, almost twice as low percentage of workers with a less than one year work history and a slightly higher proportion of workers with over 20 years of employment.

In both male and female groups, the workers who started working below the age of 30 constituted the largest population. It is worth noting that for almost 60% of males, and 62% of females from the cohort, the observation time was at least 20 years (Table 3).

Male cohort

The mortality rate in the male cohort was found to be lower than that in the general population (385 deaths, SMR = 85). Compared to the expected values, an increased number of deaths was noted for the following medical causes: mental

disorders (8 cases, SMR = 274), and genitourinary diseases (11 cases, SMR = 157), including nephritis and nephrosis (7 cases, SMR = 143). Only the excess of deaths from mental disorders was statistically significant ($p < 0,05$). In this group the main cause of death was the alcoholic disease.

Generally, mortality from malignant neoplasm, appeared to be at a lower level than in the reference population (88 cases, SMR = 87). A higher than expected number of deaths was found for cancer of the following sites: stomach (16 cases, SMR = 107), large intestine (7 cases, SMR = 264) and pleura (5 cases, SMR = 2846). Statistically significant was the standardized mortality ratio from colon and pleura cancers (Table 4).

Table 4. Mortality from selected causes in 2525 male asbestos-cement workers

No.	Cause of death (ICD-9)	Observed	Expected	SMR	95% CI
1.	All causes (001-999)	385	451.2	85	77–94
2.	Infectious and parasitic diseases (001-139)	6	11.5	52	19–113
3.	Malignant neoplasms (140-208)	88	101.5	87	70–107
4.	– oesophagus (150)	1	2.6	38	
5.	– stomach (151)	16	14.9	107	61–174
6.	– colon (153)	7	2.6	264	106–544
7.	– rectum and anus (154)	4	3.7	108	29–277
8.	– liver (155)	3	3.7	81	17–237
9.	– pancreas (157)	3	4.1	73	15–213
10.	– larynx (161)	4	4.0	101	28–259
11.	– trachea, branchus and lung (162)	29	34.6	84	56–121
12.	– pleura (163)	5	0.2	2846	924–6642
13.	– prostate (185)	1	2.7	37	
14.	– kidney (189)	1	2.6	39	
15.	– brain (191)	3	3.3	92	19–269
16.	– myeloid leukaemia (205)	1	1.5	68	
17.	Endocrine, nutritional and metabolic diseases (240-279)	2	5.2	39	5–141
18.	Diseases of the blood and blood-forming organs (280-289)	1	0.6	158	
19.	Mental disorders (290-319)	8	2.9	274	118–540
20.	Diseases of the nervous system and sense organs (320-389)	6	5.3	113	41–246
21.	Diseases of the circulatory system (390-459)	148	177.4	83	78–98
22.	– hypertensive disease (401-405)	5	7.5	67	22–156
23.	– ischaemic heart disease (410-414)	65	67.1	96	74–122
24.	– cerebrovascular disease (430-438)	17	22.1	77	45–123
25.	– atherosclerosis (440)	15	32.2	47	26–78
26.	Diseases of the respiratory system (460-519)	15	21.3	70	39–115
27.	Diseases of the digestive system (520-579)	9	19.0	47	21–89
28.	– chronic liver disease and cirrhosis (571)	7	8.0	88	35–181
29.	Diseases of the genitourinary system (580-629)	11	7.0	157	78–281
30.	– nephritis, nephrotic syndrome and nephrosis (580-589)	7	4.9	143	57–295
31.	Symptoms, signs and ill-defined conditions (780-799)	13	21.8	60	32–103
32.	– senility (797)	2	1.8	111	13–401
33.	Injury and poisoning (800-999)	78	74.5	102	83–131

Table 5. Mortality from malignant neoplasms in 2525 male asbestos-cement workers according to length of exposure

Cause of death (ICD-9)	Length of exposure (years)											
	< 9				10–19				20 <			
	Observed	Expected	SMR	95% CI	Observed	Expected	SMR	95% CI	Observed	Expected	SMR	95% CI
Malignant neoplasms (140-208)	52	58.0	90	67–119	25	33.4	75	49–111	11	10.1	109	54–195
– stomach (151)	12	8.8	136	70–238	4	4.9	82	22–210	–	1.2	0	
– colon (153)	4	1.5	261	71–668	2	0.9	229	28–827	1	0.2	412	
– rectum and anus (154)	1	2.1	49		1	1.3	79		2	0.4	545	66–1969
– liver (155)	3	2.1	142	29–415	–	1.2	0		–	0.3	0	
– pancreas (157)	3	2.3	128	26–374	–	1.4	0		–	0.4	0	
– larynx (161)	3	2.2	135	28–395	1	1.3	77		–	0.4	0	
– trachea, bronchus and lung (162)	15	18.9	79	44–130	11	11.7	94	47–168	3	3.9	77	16–225
– pleura (163)	–	0.1	0		2	0.1	3606	437–13206	3	0.0	16646	34209–48606
– brain (191)	2	2.0	98	12–354	–	0.9	0		1	0.3	327	

Pleural mesothelioma was the cause of death of 5 workers whose employment length ranged from 12 to 26 years ($\bar{x}$ = 19.8, SD = 6.8). The latency varied from 20 to 27 years ($\bar{x}$ = 24.4, SD = 3.0) and the workers' age at the time of death ranged between 46 to 68 years ($\bar{x}$ = 56.4, SD = 9.3).

An analysis of the death risk in relation to selected cancer sites (according to the prevalence in the cohort), and duration of exposure revealed a significant tendency only in the case of pleural neoplasms ($c^2 = 8.802$; $p < 0.05$) (Table 5).

Female cohort

During the cohort observation, 24 deaths were recorded in the group of female workers (SMR = 72). An analysis by causes revealed a higher than expected number of deaths from several groups of diseases but this referred to a very small number of cases. Mortality from malignant neoplasms was lower than in the general population (10 cases, SMR = 87). Compared to the expected value, an excess number of deaths was found for lung cancer (3 cases, SMR = 382) and pleural mesothelioma (2 cases, SMR = 11,275; $p < 0.01$). The female workers who died from the latter cause were found to be exposed for 19–20 years. The latency exceeded 20 years. In the female cohort the increased mortality was found to be due to cervical cancer (3 cases, SMR = 231) and liver cancer, however, the latter was noted only in 1 person (Table 6).

Table 6. Mortality from selected causes in 591 female asbestos-cement workers

No.	Cause of death (ICD-9)	Observed	Exposed	SMR	95% CI
1.	All causes (001-999)	24	33.5	72	46–107
2.	Malignant neoplasms (140-208)	10	11.3	89	43–164
3.	– liver (155)	1	0.5	213	
4.	– trachea, bronchus and lung (162)	3	0.8	382	79–1116
5.	– pleura (163)	2	0.0	11275	1368–40714
6.	– cervix uteri (180)	3	1.3	231	48–675
7.	Diseases of the nervous system and sense organs (320-389)	1	0.7	133	
8.	Diseases of the circulatory system (390-459)	8	11.6	69	30–136
9.	– cerebrovascular disease (430-438)	2	2.3	85	10–307
10.	– atherosclerosis (440)	3	1.9	162	33–473
11.	Diseases of the respiratory system (460-519)	1	1.1	92	
12.	Diseases of the digestive system (520-579)	1	1.5	67	
13.	Injury and poisoning (800-999)	3	2.9	102	21–298

DISCUSSION

Considering the industrial applications of asbestos, the most prevalent one seems to be the manufacture of asbestos cement products. Thus, many reports focus on the risk of cancer development among workers in this particular industry (1,3,4,6,7–12,14–16,18–21,24,25,27,28). A number of reports dates from the 1980s when comprehensive projects on health risk from exposure to asbestos, in common use at that time, were carried out in highly developed countries. Their findings, indicating adverse health effects of exposure, gave rise to a public discussion which

resulted in the reduced manufacture of asbestos cement goods, their limited use, and eventual total ban on both their production and use. Consequently, the research on the health effects of exposure in asbestos workers has become of minor interest in these countries. However, this problem remains essential in Central and Eastern Europe where asbestos manufacture reached its peak level between the 1960s and 1980s. The first effects of massive occupational exposure during that period may have appeared not earlier than in the 1990s.

The findings of the studies on cancer risk cited above are, however, contradictory. In the cohorts of workers from the plants employing chrisotile asbestos no excessive cancer risk was found (6,7,16,22). In the plants where crocidolite prevailed, an excess was noted in the number of cases of malignant neoplasms generally and of the following sites: lung, larynx, pleura, mediastinum, colon and rectum, liver, kidney and male genitals (10,11,15,18,19,23). In view of the fact that no clear evidence of excessive cancer risk among workers of asbestos cement plants has been found, in 1993 the Supreme Court of the United States of America cancelled the ban on the use of asbestos cement products (5).

In the plants considered in our study, high levels of asbestos dust were noted for a period of over 20 years. They have been measured in mg/m^3, according to the former Polish standard. However, this measurement bears no indication of the fibre concentration. The method of calculating the number of respirable asbestos fibres in one cm^3 was not implemented until the1980s and even since then it has been rather rare. It is thus impossible to make use of the exposure data in the retrospective analysis of the dose-response relationship in respect of individual assessment of asbestos fibre concentration.

Considering the above issues as well as the generally high level of asbestos dust in both the plants (particularly in the 1970s when the dry method was used), the mortality rate was analysed in the cohort comprising all the workers directly involved in the manufacture. The general mortality rate in the cohort, both male and female, was found to be lower than expected, thus revealing the 'healthy worker effect'.

The high level of risk of pleural mesothelioma and the relatively low number of deaths are associated with the very low death rate from this cause in the general population of Poland (21). The prevalence of this neoplasm in the cohort can be attributed to the wide use of crocidolite in these plants (13). Considering a 30-year latency and over a10-year period of employment under exposure to high asbestos levels, an excess of mortality from pleural mesothelioma can be expected in the cohort up to the year 2020.

The increased risk of colon cancer was also noted in the Swedish cohorts of workers exposed to asbestos cement (11). The excessive risk of stomach cancer reported in the American and Danish studies appeared to be much lower in our cohort. In case of stomach cancer, contrary to lung cancer, exposure to cement dust may be the major cause (2,10,18). Almost fourfold increase in the risk of lung cancer among the female workers was not statistically significant owing to just three cases of death. These results are consistent with the findings of other cohort studies regarding occupational exposure to asbestos cement (7,9,26). The authors explain their observations by the use of only chrisotile asbestos, low fibre concentration, manufacture technology preventing fibre damage and a rather large number of workers with short exposure periods (in some studies even up to 50%) (7,18).

The negative findings of this first survey concerning the cohorts of workers employed in the asbestos cement plants founded in the 1960s, should be interpreted with caution in view of a relatively long, 30–40 years, latency of asbestos-induced neoplasms (20). The observed cohort is rather 'young' and it is still too early to draw any definite conclusions concerning the mortality in this group. The ultimate results are usually obtained after tracing a historically prospective cohort for about 40 years.

It should be stressed, however, that the substantial improvement in the conditions of work and the change of technologies employed in the plant have contributed largely to the reduction in the asbestos fibre concentration at the workpost. This will undoubtedly result in a decreased cumulated lifetime dose of asbestos dust. It is expected that the limitation of asbestos use, enacted in 1997, and finally the prohibition of the manufacture of asbestos cement goods after 30 September 1998, will considerably diminish the health effects of occupational and environmental exposure to asbestos.

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