Environmental Asbestos Exposure and Cancer Mortality

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ABSTRACT. From 1970 to 1980, mortality from cancer of the lung and stomach was analyzed in a town with asbestos deposits and in a town with asbestos processing. The populations of the entire country, the province, the district, and of all communities of the same size and agricultural index were used as references. In the town with asbestos contamination of air and water from natural tremolite deposits and an endemic occurrence of pleural plaques, no increased risk for lung or stomach cancer was found. In the town with asbestos cement production lung cancer rates were lower in males and higher in females, and stomach cancer rates were lower than expected. No significant differences could be attributed to environmental asbestos exposure.

IN COHORT STUDIES on dust workers[1] and asbestos workers,[2] evidence for increased mortality from lung and stomach cancer was found. Extrapolations of dose-response curves from occupational studies and theoretical considerations questioning thresholds for carcinogenic substances have induced investigations on public health risks resulting from low level asbestos exposures.[3-5] They focus on lung cancer as the most important cancer risk from asbestos inhalation. Some consideration was also given to gastrointestinal cancer in relation to environmental asbestos exposure from drinking water.

METHODS

Two study populations in Austria were selected for the investigation of cancer risks from environmental asbestos exposure. One is located in an area with natural asbestos deposits. Tremolite was mined there until 1945, and today asbestos still is found in the soil of the fields and vineyards, in the atmosphere (from natural erosion), and in the drinking water. In this community an increased prevalence of pleural plaques was detected[6]; present levels of asbestos exposure from air and water have been reported.[7,8] The other study area is located around the oldest asbestos cement factory in the world, which now processes about 90% of all asbestos imported to Austria. Present atmospheric asbestos concentrations in the near vicinity of this plant have been reported.[7]

We analyzed mortality data from official death certificates from 1970 to 1980. Table 1 shows the size of the populations studied, migration,[9] and the minimal relative risks to be detected in the two towns and corresponding districts, calculated from expected numbers in Austria.[10,11] In the smallest community studied, a twofold or higher risk for lung or stomach cancer must show up after the observation period of 11 years.

Table 1.—Populations under Study and Detectable Relative Risks (see text)

	Population 1971	Population 1981	Migration 1971-1981	Minimal Relative Risk to be Detected in 1970-1980 Lung Cancer	Minimal Relative Risk to be Detected in 1970-1980 Stomach Cancer
Town with asbestos deposits	3.412	3.425		2.04	2.03
District	53.471	54.172	+ 195 (+0.4%)	1.21	1.22
Province	272.119	272.274	- 1.855 (-0.7%)		
Town with asbestos processing	10.627	11.039		1.45	1.49
District	109.663	114.378	+ 553 (+0.5%)	1.16	1.16
Province	1,223.444	1,270.426	+ 7.981 (+0.6%)		
Austria	7,456.403	7,555.338	+ 73.710 (+1.0%)		

Table 2.—Lung Cancer Mortality (1970-1980) in a Town with Asbestos Deposits and a Town with Asbestos Processing

		Austria		Province		District		Community Size		Community Size and Agric. Index	
	OBS	EXP	SMR	EXP	SMR	EXP	SMR	EXP	SMR	EXP	SMR
Town with asbestos deposits											
Male	9	11.8	76	12.0	75	10.7	84	11.5	76	11.9	76
Female	3	2.9	105	2.0	147	1.5	200	3.8	79	4.1	73
Total	12	14.6	82	14.1	78	12.2	98	15.3	78	16.0	75
Town with asbestos processing											
Male	30	56.2	53†	53.8	56†	51.8	58†	47.0	64*	47.1	64*
Female	11	8.6	128	7.0	158	5.2	211*	7.5	146	7.7	142
Total	41	64.8	63†	60.8	67*	57.0	72*	54.5	75	54.9	75

NOTES: Observed deaths (OBS), expected values (EXP), and standard mortality ratio (SMR) calculated from different reference populations.

* $P < .05$.

† $P < .01$.

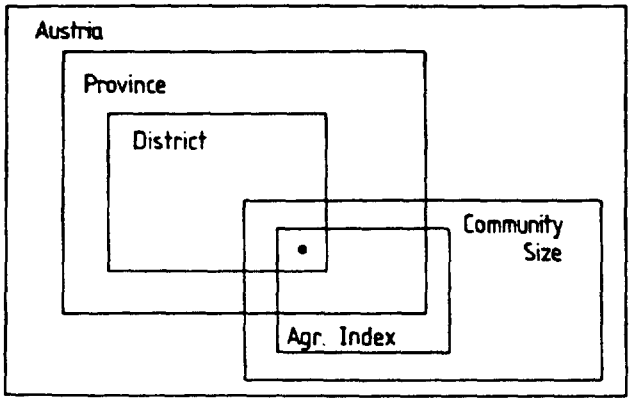

Fig. 1. Reference populations used for calculating expected mortality.

Standard mortality ratios (SMRs) were calculated[12,13] on the basis of population census results from 1961, 1971, and 1981 (corrections for births, deaths, and mobility were used for the intervening years), and annual mortality data, both grouped according to sex and age (5-yr age groups). Expected numbers of deaths from lung and stomach cancer were calculated using five populations as references: (1) the Austrian population, (2) the respective province population to which the study population belongs, (3) the population of the respective district, (4) the subpopulation of Austria living in communities belonging to the same community size class, and (5) the subpopulation living in communities with the same agricultural index as the communities under study (Fig. 1).

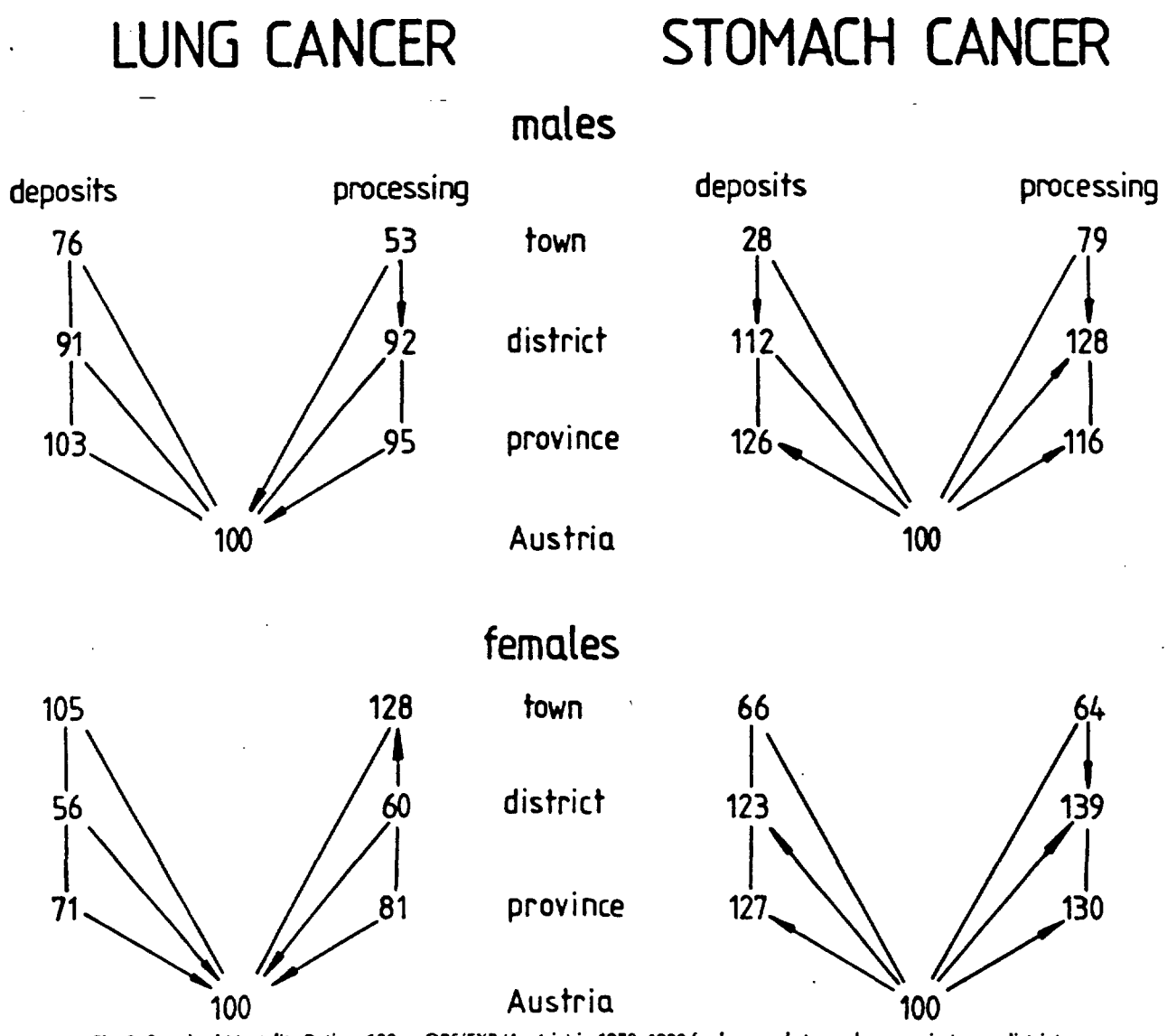

Fig. 2. Standard Mortality Ratios: 100 × OBS/EXP (Austria) in 1970–1980 for lung and stomach cancer in town, district and province with asbestos deposits or asbestos processing. Arrows indicate significant differences ($P < .05$)

RESULTS

Lung cancer mortality in districts and towns with asbestos deposits or asbestos processing was lower than expected (Table 2). In the town with asbestos deposits, the SMR was 76 in males and 105 in females (Fig. 2). After adjusting for community size and agricultural index, both males and females showed lower, but not statistically significant, SMRs (Table 2). In the town with asbestos processing, the SMR was significantly lower in males and insignificantly higher in females. This result remained essentially unchanged after adjusting for community size and agricultural index (Table 2).

Stomach cancer mortality for both males and females (Fig. 1) was higher in the districts and lower in the towns with asbestos deposits or asbestos processing (Table 1 and Fig. 1). In the town with asbestos deposits, the SMR (with or without adjustments for community size or agricultural index) was significantly lower in males, and in the town with asbestos processing the SMR was significantly lower in females (Table 3).

DISCUSSION

While we found an increase in risk from cancer of the lung and stomach in highly exposed asbestos workers,[14] no evidence of cancer excess from environmental asbestos exposure could be detected in this study. Similarly, the U.S. Cancer Institute reported no increase of cancer mortality rates in U.S. counties with asbestos deposits, but did not draw definite conclusions from these findings because of lack of exact information on exposure and because of the dilution of exposure in studying the population of counties.[15] Our study covered smaller populations, but was based on better data on exposure and cause of death. In the town with asbestos deposits, measurements in 1975–1979 provided evidence that practically all inhabitants there are

exposed to tremolite asbestos via air and drinking water, so that a dilution of the effect from dilution of exposure can be disregarded. Because of the small size of this community, however, a small excess in cancer deaths could not be ruled out, and only relative risks greater than 2 (for lung or stomach cancer) would have been detected.

As a population risk marker for asbestos cancer we have also looked for the geographical distribution of 161 malignant mesotheliomas in Austria[3] and found no clustering of this tumor around asbestos deposits or processing. In a nationwide survey on pleural mesothelioma in 1969-1974,[14,16] no case was detected in the district with asbestos deposits (only 0.9 cases, however, would have been expected there under the assumption of no increased risk). Since then no case of mesothelioma has been reported from the town with asbestos deposits. In the population of this town, however, we found an increased prevalence of pleural plaques. Pleural plaques have been suggested as risk markers for asbestos cancer.[17] Our findings suggest that low levels of (environmental) asbestos exposure can lead to an endemic occurrence of pleural plaques without increasing the cancer rates. This hypothesis is consistent with the results of Kiviluoto et al.,[18] who found no lung cancer excess in persons with pleural plaques from low level exposure to another type of amphibole asbestos. Only when plaques, combined with lung fibrosis, were detected as a sign of higher (occupational?) exposure, were lung cancer rates increased, too.

In the town with asbestos cement processing the outdoor concentrations have been measured near the vicinity of the plant only.[7] A dilution of exposure in the population under study can lead to an underestimation of cancer risk related to environmental asbestos exposure. Conversely, occupationally exposed persons included in the population under study can lead to an overestimation of this risk. But lung cancer rates in males, though partly occupationally exposed, were lower than expected. Lung cancer rates in females were slightly higher than expected; however, this could be a chance finding and cannot be related to environmental asbestos exposure (overadditive combined effects with smoking should lead to increased lung cancer rates in men earlier than in women).

There is evidence that smoking habits for males in the town with asbestos deposits are not different from those in the reference population, since relative frequencies of male smokers show only marginal variations according to estimations from microcensus data:[19] 41.1% smokers in the general male population, 39.5% smokers in the male population of the province, and 39.7% smokers in the population living in communities with the same size and agricultural index.

The variations in the female population seem to be of importance, however, since only 9.1% of female smokers would be estimated from the province population, whereas 16% are inferred from the population living in communities with the same size and agricultural index. Unfortunately, no data are available on smoking habits of the communities under study so that no clear conclusions could be drawn. It might be, however, that the slightly increased lung cancer mortality in females is due to smoking habits.

For the town with asbestos processing, estimations of smoking habits according to different stratification criteria reveal for both males and females only slightly different values, which are well within the sampling error.

The microcensus data on smoking habits in 1972 and 1979 showed no essential differences, but major changes during the past decades which might affect regional lung cancer differences cannot be ignored.

Table 3.—Stomach Cancer Mortality (1970-1980)

		Austria		Province		District		Community Size		Community Size and Agric. Index	
	OBS	EXP	SMR	EXP	SMR	EXP	SMR	EXP	SMR	EXP	SMR
Town with asbestos deposits											
Male	2	7.3	28*	9.1	22*	8.2	24*	9.5	21*	9.5	21*
Female	5	7.6	66	9.9	51	9.4	53	11.1	45	10.4	48
Total	7	14.9	47*	19.0	37†	17.6	40*	20.6	34†	20.0	35†
Town with asbestos processing											
Male	27	34.2	79	39.7	68*	43.3	62*	32.9	82	32.4	83
Female	14	21.9	64	28.5	49†	30.1	47†	26.9	52*	27.4	51*
Total	41	56.1	73*	68.2	60†	73.4	56†	59.7	69*	59.8	69*

NOTES: Observed deaths (OBS), expected values (EXP), and standard mortality ratio (SMR) calculated from different reference populations.

* $P < .05$.

† $P < .01$.

Nutrition as a confounding factor for stomach cancer has changed essentially during the past decades[20] and could not be accounted for in this investigation. A bias from population mobility seems to be negligible (Table 1). Occupation as a confounding factor would have been expected to increase, rather than decrease the cancer rates in the towns under study.

In the study on pleural mesothelioma[14,16] in 1969–1974, no case was detected in the district with asbestos cement processing (1.6 expected); since that time 2 occupationally exposed mesotheliomas have been found. This contrasts with the observations in the surroundings of shipbuilding and other industries, where not only occupational but also environmental asbestos exposure was related to mesothelioma incidence.[21] We conclude that asbestos concentrations not only within these industries, but also in their vicinities, must have been higher than in our study (or that occupational and paraoccupational cases in the vicinity of shipyards, etc. could not be distinguished precisely from environmental ones).

Because we did not find any indication of cancer excess from environmental asbestos exposure in our study, we cannot extrapolate this result to other countries with higher levels of environmental asbestos exposure. Recently, Siemiatycki[22] reported that cancer mortality in the comparably high-exposed general population of the Canadian mining areas was not higher than expected. In this study an increased lung cancer rate in men was attributed to occupational exposure rather than to combined effects of smoking and environmental exposure.

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