Survival in cohorts of asbestos cement workers and controls

M Albin, V Horstmann, K Jakobsson, H Welinder

Abstract

Objectives—To measure the impact on survival of being exposed to asbestos cement dust.

Methods—Survival of 866 asbestos cement workers and 755 controls was studied with Cox's proportional hazards regression models with age as the basic time variable. The effect of cumulative exposure up to the age of 40 was investigated in an internal analysis of 635 asbestos cement workers who had dose estimates.

Results—The death risk was higher for the asbestos cement workers than for the controls with a hazard ratio (HR) of 1.15 (95% confidence interval was 1.00 to 1.31). The increased risk found seemed to be confined to the period 20–40 years from start of employment. The estimates of the cohort effect were almost unaffected by adjustment for smoking habits. The estimates of the exposure effect rose with increasing dose (< 4 fibre-years/ml (f-y/ml): HR = 1.00, 4–9.9 f-y/ml: HR = 1.06, ≥ 10 f-y/ml: HR = 1.35, for workers with at least five years of employment), and were higher when restricted only to deaths from malignant or non-malignant respiratory disease. However, none of the point estimates were significantly increased. Median age at death was two years lower in the high than in the low, exposure group.

Conclusions—The results indicate that even a moderate asbestos exposure may shorten the median duration of life in an exposed population. Compared with the estimated effect on duration of life from ever being a smoker, that of ever being an asbestos cement worker was less, although that of having a high exposure was similar.

(*Occup Environ Med* 1996;53:87–93)

Keywords: asbestos cement workers; survival; dose

Several studies of asbestos workers have reported an increased mortality both from all causes and from non-malignant respiratory diseases and different malignant neoplasms. In spite of its obvious importance, the impact on duration of life from these increased risks has not been studied for the total worker popula-

tions, except by Elmes.¹ Several studies have, however, investigated survival among workers compensated for asbestosis.^{2–6} We have studied survival in cohorts of asbestos cement workers and controls.

Material and methods

STUDY POPULATIONS

A cohort was set up from the personnel records of an asbestos cement plant operating in 1907–77, including workers employed for at least three months but starting employment no later than January 1976.⁷ The study group was originally defined as all male blue collar workers ($n = 2898$).

A control cohort from the same region was also created, consisting of 1522 male industrial workers, employed for at least three months, from plants requiring similar professional skills and with similar work loads. Thus, workers from a slaughter house, a fertiliser plant, a sugar refinery, a textile plant, and a metal industry were included. Workers with former employment at the asbestos cement plant ($n = 13$), and workers with job titles indicating probable asbestos exposure were excluded ($n = 142$).

Analysis of the end of employment by calendar year indicated that the personnel records were complete from 1907 for the asbestos cement plant. For the control plants this was the case only from 1942 for the slaughter house, 1945 for the fertiliser plant, 1944 for the sugar refinery, 1948 for the textile plant, and 1952 for the metal industry (all considerably later than formerly assumed⁷).

Analyses of the comparability between the two cohorts showed a substantially higher proportion of immigrants among the asbestos cement workers than among the controls (33% *v* 11%). Follow up of the immigrant workers was poor in both cohorts (19% lost to follow up, compared with 1% among native Swedes). Further, restricting the analysis to native Swedes, a higher proportion of the asbestos cement than the control workers had a short duration of employment (45% *v* 23% employed < 1 year).

Thus, to make the two cohorts comparable, the study populations were restricted to male Swedish workers employed for at least one year, alive at the age of 40 (see discussion), and in employment after February 1946, although they might have started earlier (but not later than January 1975). Table 1 shows

Department of
Occupational and
Environmental
Medicine, University
Hospital, S-221 85
Lund, Sweden
M Albin
V Horstmann
K Jakobsson
H Welinder

Correspondence to:
Dr Maria Albin, Department
of Occupational and
Environmental Medicine,
University Hospital, S-221
85 Lund, Sweden.

Accepted 3 September 1995

Table 1 Descriptive data for asbestos cement workers and controls

	Asbestos cement workers		Controls
	Internal analysis	External analysis	
Number	635*	866	755
Year of birth†	1915 (1907, 1933)	1914 (1905, 1929)	1914 (1903, 1929)
Vital status:			
Alive	313	416	373
Dead	321	450	382
Employment:			
Age at start†	25.5 (21.4, 32.1)	33.9 (22.9, 43.7)	35.5 (23.8, 46.5)
Calendar year at start†	1942 (1935, 1959)	1953 (1944, 1961)	1952 (1944, 1962)
Duration (y)†	5.8 (2.1, 20.8)	7.9 (2.3, 19.9)	10.3 (4.2, 20.0)
Smoking:			
Information available (%)	88	87	38
Ever smokers (%)‡	85	83	79
Pack-years‡§	28.4 (15.0, 45.8)	29.0 (15.0, 45.0)	26.5 (15.0, 41.5)

*Including one subject lost to national registration; †median (upper, lower quartiles); ‡percentage of subjects with available information on smoking habits; §distribution among current and former smokers: information on pack-years available for 464 asbestos cement workers in the internal analysis, 610 asbestos cement workers in the external analysis, and 226 controls.

group characteristics of the 866 asbestos cement workers and 755 controls.

In the internal analysis within the asbestos cement cohort at least one year of employment and being alive at the age of 40 was still required, and having started employment before the age of 40. Further, workers with missing dose estimates ($n = 121$) were excluded. No restriction was used on calendar year of employment. The study group in the internal analysis comprised 635 asbestos cement workers (table 1).

Vital status was determined on 30 June 1991. Information on underlying cause of death transformed to international classification of diseases (ICD-8) was obtained from the Statistics Sweden. Information as to the cause of death was not yet available for three asbestos cement workers and one control.

SMOKING

Information on smoking habits was assembled in a clinical study in 1978–9 for 33% of the 866 asbestos cement workers in the external, and 45% of the 635 workers in the internal analyses. For asbestos cement workers in the unrestricted cohort, not participating in the clinical study, and for controls from four out of five plants, information was obtained by a postal inquiry (sent to the next of kin of deceased workers). The response rate was 79% for the asbestos cement, and 74% for the control workers. No information at all was available for the 345 workers from the fifth control plant.

Data were collected for the year smoking started and stopped, and average daily consumption. Table 1 shows the distributions of

available information on smoking.

Subjects with missing information on smoking were tentatively assigned first to the non-smokers, and then to the smokers. With age as the basic time variable Cox's regression models were fitted to the data, first based only on workers with known smoking habits and then including all workers within the two definitions of smokers (table 2). The assumption that all subjects with missing information on smoking were non-smokers seemed unjustified (as smoking in this model has a clearly protective effect), whereas the estimate of the smoking effect when all subjects with missing smoking information were assumed to be smokers, seemed reasonable. Thus, this last assumption was considered to be best justified, and was used in the subsequent analyses where smoking was taken into account.

In the asbestos cement cohort, 95% of the known smokers or former smokers were smokers when they started employment; the same applied to 87% of the known control smokers. The others usually started smoking shortly after the start of employment. Thus, we assumed that ever smokers were smokers already at the start of employment.

EXPOSURE DATA

Exposure assessment was based on job titles, dust measurements (available from 1956 onwards), changes in production, and dust control. Individual estimates based on job title were made for each worker by an experienced industrial hygienist (HW), as described earlier.⁷ Chrysotile was the major asbestos type, but small amounts of amosite and crocidolite were also used.

The median estimated intensity of asbestos exposure was 1.1 (lower quartile = 0.6, higher quartile = 1.6) f/ml; the distribution was skewed, with an arithmetic mean of 1.4 f/ml.

STATISTICS

Follow up started at the age of 40, but not earlier than one year after the start of employment. However, in the comparison between asbestos cement workers and controls, follow up did not start earlier than February 1946 (1952 for the control workers employed at the plant for which the personnel records were not complete until then), and continued until the first date of either death, emigration, declared lost in the national registration ($n = 1$), or 30 June 1991.

Table 2 Cox's regression models for the mortality in cohorts of asbestos cement workers ($n = 866$) and controls ($n = 755$) workers by age, with different adjustments for smoking

Covariates	Known smoking habits* HR (95% CI) ($n = 1041$)	Smoker unless otherwise stated† HR (95% CI) ($n = 1621$)	Non-smoker unless otherwise stated† HR (95% CI) ($n = 1621$)
Deaths (n)	(474)	(832)	(832)
Model 1 (cohort effect only): Asbestos cement worker	1.25 (1.02, 1.55)	1.15 (1.00, 1.31)	1.15 (1.00, 1.31)
Model 2 (cohort and smoking effects included): Asbestos cement worker	1.24 (1.01, 1.53)	1.16 (1.01, 1.33)	1.23 (1.06, 1.43)
Smoker	1.15 (0.91, 1.45)	1.26 (1.01, 1.57)	0.84 (0.72, 0.98)

*Based only on workers with known smoking habits; †all workers included, both with and without known smoking habits.

One part of the analyses concerns the comparisons between the exposed workers and the controls. With age as the basic time variable we calculated Kaplan-Meier survival curves with 95% confidence intervals (95% CIs).⁸ Further comparisons were performed by means of Cox's regression models that included an indicator variable for each cohort (0 for the control and 1 for the asbestos cement workers). Also an indicator variable for smoking (0 for non-smokers, 1 for smokers), and a time dependent variable measuring time since first exposure and categorised into 0–19.9 years (included all controls), 20–39.9 years, and ≥ 40 years, were considered in the models. Other explanatory variables were calendar year at start of employment (given in years, coded as (start of employment—1950)/10), age at start of employment (given in years, coded as (age–35)/10), plus several interaction terms.

We divided the observations twice: firstly into those starting employment before or after the age of 30; secondly, into those starting employment before or after 1946. However, since the estimates for workers starting employment before and after 1946 were similar, they are not presented here.

Similar analyses were performed when the event of interest was natural death (diseases only) instead of all deaths.

In the internal comparison the asbestos cement workers were divided into three groups according to their cumulative dose at the age of 40. So the doses were factorised into indicator variables. The factorised dose variable, and the smoking variable, were used as covariates in a regression model. A test for trend was performed by comparing the overall score for this model with the score for the model where exposure of the dose variable was the median dose for the corresponding group (low, medium, or high).

The covariates for calendar year of birth and calendar year at the start of employment were also considered as covariates in the regression model.

The appropriateness of the proportional hazard models was in each case investigated by including in the model, for each covariate, a time dependent interaction of this covariate multiplied by the log of the basic time variable. The interaction term was not significant (at the 5% level) in any of the models considered, indicating that the proportional hazard model seemed appropriate.

We used the computer package EGRET (graphics version 26.06) to obtain estimates of the hazard ratios (HRs), with 95% CIs (based on Walds test⁹). Data are described with a median, upper and lower quartiles, and 95% CIs.

Results

ASBESTOS CEMENT WORKERS *v* CONTROLS

There were twice as many deaths from malignant and non-malignant (infectious and chronic obstructive) respiratory diseases among the 866 asbestos cement workers than

among the 755 controls (85 *v* 39). Pneumoconiosis, or lung fibrosis, was given as the underlying cause of death for four asbestos cement workers and two controls. The higher number of deaths among the asbestos cement workers than the controls from non-malignant respiratory diseases was apparent mainly 20–40 years from the start of employment (26 *v* 9 deaths). Deaths from respiratory cancers were more numerous among the asbestos cement workers for both the periods 20–40 years (16 *v* 7) and > 40 years (19 *v* 3) from first employment. Seven of the deaths among the asbestos cement workers were due to mesothelioma according to the death certificates, another three were found when tissue from respiratory cancers were submitted for histopathological review.⁷ Five of the mesotheliomas occurred 20–40 years, and five 40 years or more, from first employment. Thus, for the period 40 years or more from first exposure, excess lung cancers were still numerous among the asbestos cement workers.

The number of deaths from external causes was slightly higher among the asbestos cement workers than among the controls (38 *v* 29). To evaluate possible differences in lifestyle factors between the two cohorts the number of deaths with a strong empirical link with alcohol¹⁰ was computed by combining deaths from alcoholism, alcohol psychosis, alcohol intoxication, cirrhosis of the liver, and pancreatitis (ICD-8: 291, 303, 571, 577, and 860). Three deaths from these causes were stated on the death certificate for the asbestos cement cohort and two for the controls.

Kaplan-Meier estimates for the two cohorts showed that survival was dependent both on smoking category and cohort. Median age at death was 77.9 (95% CI 75.8 to 80.5) for non-smoking asbestos cement workers, and 78.9 (95% CI 73.2 to 86.3) for non-smoking controls, 75.8 (95% CI 75.0 to 77.0) for asbestos cement workers who smoked, and 76.9 (95% CI 75.9 to 77.8) for controls who smoked.

Cohort and smoking category were included in a Cox's regression model with age as the basic time variable. Both were significant at the 5% level. We found a higher death risk for the asbestos cement workers than for the controls, in the entire cohorts (HR = 1.16, 95% CI 1.01 to 1.33; table 2, fig 1) and in those with known smoking habits (HR = 1.24, 95% CI 1.01 to 1.53). A model without the variable on smoking provided similar estimates of the HRs. Median age at death was estimated to be 78.2 for non-smoking asbestos cement workers, 79.9 for non-smoking controls, 76.3 for asbestos cement workers who smoked, and 77.3 for controls who smoked (not in table).

The effect of age at first employment on the estimated cohort effect was evaluated by subdividing the two cohorts according to age at first employment. The point estimate of the cohort effect was slightly higher for asbestos cement workers employed before the age of 30 (HR = 1.27, 95% CI 0.96 to 1.66), compared with those employed after 30 (HR =

Table 3 Hazard ratios for different exposures among the asbestos cement workers for all deaths, and deaths from respiratory (malignant and non-malignant) respiratory diseases, derived from Cox's regression analyses by age

	All causes		Respiratory diseases			
	Employed $\geq 1y$ (n)	HR (95% CI)	Employed $\geq 5y$ (n)	HR (95% CI)	Employed $\geq 1y$ HR (95% CI)	Employed $\geq 5y$ HR (95% CI)
Model 1						
0-3.9 f-y/ml	(322)	1.00	(78)	1.00	1.00	1.00
4-9.9 f-y/ml*	(155)	0.96 (0.73, 1.27)	(100)	1.04 (0.68, 1.58)	0.95 (0.53, 1.70)	1.71 (0.66, 4.47)
≥ 10 f-y/ml*	(158)	1.16 (0.90, 1.50)	(153)	1.33 (0.92, 1.94)	1.12 (0.65, 1.92)	1.89 (0.73, 4.43)
Model 2						
0-3.9 f-y/ml		1.00		1.00	1.00	1.00
4-9.9 f-y/ml*		0.97 (0.74, 1.28)		1.06 (0.70, 1.61)	0.97 (0.54, 1.72)	1.75 (0.67, 4.58)
≥ 10 f-y/ml*		1.16 (0.90, 1.50)		1.35 (0.93, 1.97)	1.11 (0.65, 1.90)	1.81 (0.73, 4.48)
Smoking		1.75 (1.24, 2.47)		1.56 (1.01, 2.42)	2.25 (1.03, 4.92)	2.14 (0.75, 6.07)

*Cumulative dose up to the age of 40. Subjects with dose up to 4 f-y/ml are the reference group.

1.12, 95% CI 0.96 to 1.32; both estimates adjusted for smoking).

Several other explanatory variables were explored in further Cox's regression models. Inclusion of the categorised time dependent variable, time since first exposure, indicated that the excess mortality was confined to the period 20-40 years from first exposure (HR = 1.27, 95% CI 0.99 to 1.62). For the 40 years or more since first exposure group the point estimate was slightly above one (HR = 1.14), but far from significant (95% CI 0.86 to 1.51). The cohort effect disappeared when time since first exposure was included (HR = 0.99, 95% CI 0.79 to 1.26).

A late calendar year of employment was estimated to be slightly worse than an early one (HR = 1.02 for employment 10 years later). However, this variable, and its interaction term with the cohort variable, were not significant. Further, they did not alter the estimates for the other covariates. Thus, we did not include calendar year in the further analyses.

A high age at first employment was estimated to be weakly protective (HR = 0.97 for an increase of 10 years; NS).

INTERNAL COMPARISONS

The asbestos cement workers were subdivided into three categories according to cumulative dose at the age of 40 (< 4, 4-10, > 10 f-y/ml) with the medians 1.5, 6.3, and 16.8 f-y/ml. The corresponding medians for total cumulative dose were 2.2, 7.3, and 31.6 f-y/ml. For those employed for five years or more the median cumulative dose at the age of 40 was 1.5, 6.8, and 16.9 f-y/ml, with corresponding medians for total cumulative doses of 8.0, 8.9, and 31.7 f-y/ml.

In the Cox's regression analysis with age as the basic time variable, we found that in comparison with the workers with a dose < 4

f-y/ml, those with > 10 f-y/ml had consistently higher point estimates, both among those employed for at least one year and those employed for at least five years (table 3, fig 2). A comparison of overall scores for models in which smoking was included and dose is either factorised (X2 (3) = 7.5, P = 0.057), or coded by the relevant median (X2 (2) = 7.4, P = 0.025) indicated that the hypothesis of a trend cannot be rejected. The point estimates for the dose-effect on deaths from respiratory diseases only were higher than for all causes, indicating a causal relation with the asbestos exposure.

The estimate of the smoking effect was numerically higher than in the external analyses, and as expected higher for respiratory diseases only than for all causes. The estimates of the dose-effect (all causes and respiratory diseases only) were however virtually unchanged after adjustment for smoking.

Inclusion of the categorised time dependent variable time since first exposure, provided similar results to the external analysis.

An interaction term for smoking and cumulative dose was not significant.

Calendar year at the start of employment, and calendar year of birth had no significant effect on total mortality, and inclusion of these variables did not improve the fit of the model.

Median age at death as estimated from Kaplan-Meier analysis (table 4) was 75.1 in the high and 78.8 in the low exposure group, among workers with at least five years of exposure. The differences were slightly less when stratified also by smoking. From the Cox's regression models median age at death was estimated to be 77.4 for a smoker or former smoker in the low exposure group, and 75.1 in the high exposure group. Being a smoker, or former smoker, was estimated to give a 2.7 year lower median age at death in the Kaplan-Meier and 3.4 years lower median age at death in the Cox's regression analysis.

Table 4 Median age at death among 331 asbestos cement workers with at least five years of employment by category of cumulative dose (up to the age of 40) and smoking: estimates are from Kaplan-Meier analyses, and a Cox's regression model (table 3, model 2)

Cumulative dose	Kaplan-Meier (median 95% CI)			Cox's regression (median)	
	Non-smoker	Smoker	All	Non-smoker	Smoker
0-3.9 f-y/ml	80.3 (—)*	78.4 (71.5, 80.8)	78.8 (71.5, 80.8)	80.8	77.4
4-9.9 f-y/ml*	89.3 (—)*	76.3 (75.0, 78.7)	76.9 (75.3, 79.3)	80.5	76.1
≥ 10 f-y/ml*	78.4 (68.3, 81.6)	75.0 (69.3, 77.4)	75.1 (70.0, 77.4)	78.4	75.1
All	78.3 (75.2, 82.6)	75.6 (74.7, 77.6)			

*Not obtained due to few deaths.

Figure 1 Estimated baseline survival functions by age, for cohorts of asbestos cement workers and controls, based on Cox's regression models with smoking included (table 2).

Figure 2 Estimated baseline survival functions by age, for different levels of cumulated asbestos dose (up to the age of 40) among asbestos cement workers with at least five years of employment, based on Cox's regression models with smoking included.

Discussion

We have in a previous study found a higher overall mortality in the asbestos cement workers than in the controls (relative risk = 1.2).⁷ Increased risks were found for deaths from non-malignant and malignant respiratory diseases and—for the highest exposure category—colorectal cancer. The implications of an increased relative risk are difficult to understand. The overall risk can alternatively be described as a shorter survival.

In a study which compares death risk without restrictions to specified diagnoses between exposed workers and controls, comparability between the two groups for extraneous factors is vital for the validity of the results. The two study populations were selected to be comparable for socioeconomic factors, and thereby, indirectly, also lifestyle factors. Still, we found a higher proportion of short term (< 1 year) workers in the exposed than the control population. The standardised mortality ratio (SMR) for deaths from poisoning and violence was increased for short term workers in both cohorts, but more so in the exposed group (not in results). After restriction of the two

cohorts to workers employed for at least one year, a higher risk was still found among young exposed workers than among controls. Finally, after further restriction of follow up to deaths at the age of 40 or later, we found a similar incidence of deaths from alcohol related and external (violence and poisoning) causes. The separate risk estimates for deaths from all diseases (external causes excluded) were similar to the ones obtained for all causes. Thus, no major bias should have invalidated our estimates.

To start observations at the age of 40 has two further advantages: (a) the induction latency time for asbestos related diseases is generally assumed to be around 20 years, and thus the excluded person-years are not considered relevant in this context. (b) The problem of late entry is reduced, as most workers had started employment by the age of 40.

Our adjustment for smoking habits in the comparisons between the two cohorts has two major limitations. Most importantly, smoking habits were known for only 40% of the control workers. Further we adjusted only for smoking or non-smoking and not for the quantity of tobacco consumed. Restriction of the analysis to workers with known smoking habits was not justified, as the availability of the information was related to the outcome. The mortality, compared with the general population in the region, was substantially higher among asbestos cement workers without smoking information (all causes: SMR 2.0, external causes SMR 6.6) than among those with such information (all causes: SMR 1.0, external causes: SMR 0.94; not in results). Workers with unknown smoking habits were thus likely to constitute a particular unhealthy subset of all workers, and an analysis excluding these subjects would have a low validity. The same pattern appeared among the controls, but was less pronounced, partly because smoking information was never assembled for the fifth control plant. Restriction of the analysis to workers with information on smoking would thus have different effects in the two cohorts.

Thus, we chose to evaluate models in which workers with missing information on smoking were included with arbitrarily assigned smoking habits. We decided to use the model in which workers with missing information on smoking were treated as smokers, as it yielded reasonable estimates for the smoking related risk.

Our estimates of median age at death were two years shorter for a smoker, or former smoker, than for a non-smoker. Current cigarette smokers were recently estimated to have a relative risk (RR) for death from all causes of 1.78, corresponding to a 7.5 years shorter median survival than those who never smoked regularly.¹¹ Our smoker category included former smokers, pipe smokers, and some misclassified never smokers, who all have a lower death risk than current cigarette smokers. Applying the risk estimates by Doll *et al.*¹¹ to our distribution of former *v* current smokers, and cigarette *v* pipe smokers, would give a relative risk of 1.44 for being an ever smoker *v*

being a never smoker. Our estimate of the relative risk was 1.26 in the external study, and 1.56 in the internal analysis (for workers with at least five years of employment). It is thus likely that our arbitrary classification of those with missing information on smoking induces some underestimation of the effect of smoking in the external analysis.

In the internal analyses, the probability of socioeconomic confounding should be less than in the external analyses. Further, the quality of the information on smoking was better, and missing for only 12%, but detailed information on smoking was hampered by small numbers. A comparison with the risk estimates by Doll *et al.*¹¹ does not indicate a systematic bias. Cumulative dose and duration of employment were similarly distributed in workers with and without known smoking habits (not in results).

The assessment of exposure in this study had three major difficulties: incomplete work histories (only the first assignment was known for many workers), conversion between particle and fibre counts, and the lack of dust measurements for the period 1907–55. Briefly, we estimated that the assigned dust concentrations in 1942–77 were on average accurate to within a factor of two. We have previously found that, compared with the entire cohort, separate analyses of workers who started employment in 1942 or later did not indicate disparate dose-response patterns.⁷

We did not adjust for the fact that present rules of filter preparation and fibre counting are likely to give about 30%–50% (Cherilyn Tillman, personal communication) higher concentrations than fibre counts made before 1978.

Mortality increased slightly with calendar time in both cohorts. This may indicate that the overall increase of life expectancy in the general population is not homogeneous. This is in accordance with a rising mortality among Swedish industrial workers,¹² and among lower class middle aged men in Britain.¹³

The difference in survival that we found between the asbestos cement workers and the controls, seemed to be restricted to the period 20–40 years from the start of employment. The lower limit of this span, 20 years, is in agreement with previous findings of a latency time of at least 20 years, often 25–30 years, for asbestosis and malignant diseases among North American insulators.¹⁴

It has been suggested that the increased risk found among asbestos workers should be expected to tail off, as the fibre burden in lung tissue (assumed to be an important determinant for risk) decreases with time since the end of exposure.^{15,16} The pattern we found for non-malignant respiratory disease is consistent with this hypothesis. We still found high numbers of mesothelioma and lung cancer 40 years or more from the start of exposure. However, this finding does not exclude a decreasing risk with time since the end of exposure as this was not analysed separately. The occurrence of mesothelioma with this long latency time accords with the previous findings in this

cohort that asbestos exposure of 40 years or more before the diagnosis of mesothelioma had a significant effect.⁷

The adjusted risk estimates were, as expected from the findings on latency time, somewhat higher for workers employed at under 30 compared with over 30 years of age.

Our results in the internal analysis support the hypothesis of a higher death risk for workers with higher doses and longer durations of exposure. Causality is supported by the finding of higher risk estimates for deaths from respiratory diseases only, among workers with at least five years of exposure.

In this study the estimated death risk from being a former or current smoker was higher than the estimated effect of being an asbestos cement worker. In the internal analysis, when low exposure was contrasted with high exposure, the estimated effect on median age at death is similar for being a current or former smoker (three to four years lower), and having a dose of ≥ 10 f-y/ml up to the age of 40 (median total dose 31.7) and durations of five years or more (two to three years lower). This decrease is similar to the estimate for workers employed at an arsenic smelter (two years).¹⁷ Elmes¹ estimated that insulation workers heavily exposed to asbestos lost 11 years of their life expectancy. The present asbestos exposure was less, even in the high exposure group. In a study of workers compensated for asbestosis, Berry³ estimated that the life expectation for a man awarded a 10% benefit would be reduced by three years.

For comparison, it could further be mentioned that Doll and Peto¹⁸ estimated that a maximal exposure of 0.5 f/ml for chrysotile would, for 20–30 years of exposure, shorten life expectancy by about one month. This corresponds approximately to the life time doses and durations of exposure in our intermediate dose category. The CIs are, however, wide in our internal analysis, and do not permit any firm conclusions.

This project was supported by grants from the Swedish Work Environment Fund, and the Medical Faculty, University of Lund. We also acknowledge valuable help in tracing the cohorts and assembling information on smoking habits and exposure from Ms Elisabeth Jonsson, Ms Maria Wikman, Ms Katarina Sjöland, Ms Viveka Englander, and the late Mr Gunnar Nilsson, and statistical help from Lars Rylander, BSc. Mr Lars Gunnar Hörte, Statistics Sweden made the transformations to ICD-8.

- 1 Elmes PC. Relative importance of cigarette smoking in occupational lung disease. *Br J Ind Med* 1981;38:1–13.
- 2 Huuskonen MS. Clinical features, mortality and survival of patients with asbestosis. *Scand J Work Environ Health* 1978;4:265–74.
- 3 Berry G. Mortality of workers certified by pneumoconiosis medical panels as having asbestosis. *Br J Ind Med* 1981;38:130–7.
- 4 Finkelstein M, Kusiak R, Suranyi G. Mortality among workers receiving compensation for asbestosis in Ontario. *Can Med Assoc J* 1981;125:259–62.
- 5 Cookson WOC, Musk AW, Glancy JJ, de Klerk NH, Yin R, Mele R, *et al.* Compensation, radiographic changes, and survival in applicants for asbestosis compensation. *Br J Ind Med* 1985;42:461–8.
- 6 Coutts II, Gilson JC, Kerr IH, Parkes WR, Turner-Warwick M. Mortality in cases of asbestosis diagnosed by a pneumoconiosis medical panel. *Thorax* 1987;42:111–6.
- 7 Albin M, Jakobsson K, Attewell R, Johansson L, Welinder H. Mortality and cancer morbidity in cohorts of asbestos cement workers and referents. *Br J Ind Med* 1990;47:602–10.
- 8 Kalbfleisch JD, Prentice RL. *The statistical analysis of failure time data*. New York: Wiley, 1980.
- 9 Rao CR. *Linear statistical inference and its application*. 2nd

- edition. New York: Wiley, 1973.
- 10 Ågren G. *Alcohol-related mortality in relation to social factors in the county of Stockholm and in the whole of Sweden*. Sundbyberg: Karolinska Institute, 1989. (Doctoral dissertation.)
 - 11 Doll R, Peto R, Wheatley K, Gray R, Sutherland I. Mortality in relation to smoking: 40 years observations on male British doctors. *BMJ* 1994;309:901-11.
 - 12 Diderichsen F. "Dödlighetsutvecklingen—ett mått på hälsopolitikens resultat?" In: Smedby B, Isacson S-O, Westring C-G, eds. *Folkhälsans förändring och hälsoarbets konsekvenser*. Stockholm: Medicinska forskningsrådet 1991. (in Swedish.)
 - 13 Wilkinson RG. Income distribution and life expectancy. *BMJ* 1992;304:165-8.
 - 14 Selikoff IJ. Historical developments and perspectives in inorganic fiber toxicity in man. *Environ Health Perspect* 1990;88:269-76.
 - 15 Berry G. Prediction of mesothelioma, lung cancer, and asbestosis in former Wittenoom asbestos workers. *Br J Ind Med* 1991;58:793-802.
 - 16 Albin M, Pooley FD, Strömberg U, Attewell R, Mitha R, Johansson L, Welinder H. Retention patterns of asbestos fibres in lung tissue among asbestos cement workers. *Occup Environ Med* 1994;51:205-11.
 - 17 Wall S. Survival and mortality pattern among Swedish smelter workers. *Int J Epidemiol* 1980;9:73-87.
 - 18 Doll R, Peto J. *Asbestos. Effects on health of exposure to asbestos. A report to the Health and Safety Commission*. London: HMSO, 1985.

Vancouver style

All manuscripts submitted to *Occup Environ Med* should conform to the uniform requirements for manuscripts submitted to biomedical journals (known as the Vancouver style.)

Occup Environ Med, together with many other international biomedical journals, has agreed to accept articles prepared in accordance with the Vancouver style. The style (described in full in the *BMJ*, 24 February 1979, p 532) is intended to standardise requirements for authors.

References should be numbered consecutively in the order in which they are first mentioned in the text by Arabic numerals above the line on each occasion the reference is cited (Manson¹ confirmed other reports²⁻⁵ . . .). In future references to papers submitted to *Occup Environ Med*

should include: the names of all authors if there are seven or less or, if there are more, the first six followed by *et al*; the title of journal articles or book chapters; the titles of journals abbreviated according to the style of *Index Medicus*; and the first and final page numbers of the article or chapter. Titles not in *Index Medicus* should be given in full.

Examples of common forms of references are:

- 1 International Steering Committee of Medical Editors, Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1979;1:532-5.
- 2 Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *N Engl J Med* 1976;294:687-90.
- 3 Weinstein L, Swartz MN. Pathogenic properties of invading micro-organisms. In: Sodeman WA Jr, Sodeman WA, eds. *Pathologic physiology, mechanisms of disease*. Philadelphia: W B Saunders, 1974:457-72.