Radiological changes in asbestos cement workers

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Abstract

Objective—To explore associations between exposure to asbestos cement dust and radiographic findings in lung parenchyma and pleura.

Methods—Radiographs from 174 blue collar workers and 29 white collar workers from an asbestos cement plant formed one part of the study. Progression of small opacities was further studied in those 124 blue collar workers, for whom two radiographs taken after the end of employment were available. The median readings from five readers who used the full ILO 1980 classification were used. As exposure indices, times since start of employment, duration of employment, cumulative exposure, and average intensity of asbestos exposure were used. The influence of age and smoking was also considered in multiple logistic regression analyses.

Results—Small opacities (profusion ≥1/0) were closely correlated with time related exposure variables, and showed weaker association with intensity based exposure variables. The odds ratio (OR) for small opacities was equal to 2-8 (90% CI 1-2, 6-7) in the >30 f(fibre)-y/ml group, compared with those in the 0-10 f-y/ml group. Progression of at least two minor ILO categories after the end of employment was seen in 20%. Also, pleural thickening was closely related to time. By contrast, costrophenic angle obliterations were not associated with the time related variables, but closely associated with the intensity of asbestos exposure, and tended to occur during employment. The OR was 4-5 (90% CI 1-3, 15) in the >2 f/ml group, compared with those in the 0-1 f/ml group.

Conclusions—In these workers, exposed mainly to chrysotile but also to small amounts of amphibole, the risk of radiographically visible parenchymal abnormality was substantially increased and strongly dependent on time related exposure variables. Progression was found long after the end of exposure. The findings on costrophenic angle obliterations, supposed to be sequelae of benign pleural effusions, were consistent with an immediate reaction triggered by intense asbestos exposure.

Materials and methods

SUBJECTS AND RADIOGRAPHS

The study base comprised 249 male blue collar workers with at least 10 years of employment at an asbestos cement plant, born in 1900 or later, and still alive on 1 January, 1978. This group represents 60% of all male blue collar workers, ever employed for at least 10 years at the plant. A limited number of workers with certain dusty tasks at the plant had been compulsorily examined at the Department of Radiology, Lund University Hospital, before and during employment since 1962, and all workers had been offered examinations in 1971 and 1974. Moreover, all ex-workers from the plant had been invited to health examinations in 1978 and 1984. Also a small number of men, compensated for asbestosis, had been examined for medicolegal reasons. As the plant was situated in the local catchment area of this hospital, radiographs taken for clinical reasons were also available, but,
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unfortunately, a reduction of the archives has somewhat reduced the number of available radiographs. Radiographs were found for 174 men. A maximum of four radiographs for each person was included. We selected radiographs primarily from the years of the health examinations, and tried to get series with about four-year intervals between the films. Also, radiographs from 29 white collar workers examined in 1978 were included to provide information on men with no or minor asbestos exposure. There were 53 men with only one, 33 with two, and 117 with three or more radiographs.

We used the earliest available radiograph, taken after the start of available radiograph, for the prevalence study; 12% of the blue collar workers had at that time not yet been employed for 10 years. In the follow up study 124 blue collar workers with at least two radiographs after the end of employment were included. Table 1 shows group characteristics. For 96% of the radiographs in the prevalence study and for all pairs in the follow up study, high kV technique had been used. For 89% of the pairs in the follow up study, both examinations had been performed with the air gap technique.

EXPOSURE
Asbestos
The asbestos cement plant operated between 1917 and 1977. The asbestos handled was mainly crocidolite (> 95%), but small amounts of crocidolite and amosite had been used before 1966. Silica was not added, and Portland cement with low quartz content (< 0.1% crystalline quartz in the respirable fractions) was used. Details on the asbestos dust levels in the air of the production area, and the calculation of estimates of individual exposure have been reported elsewhere. Cumulative exposure up to the date of each radiograph was calculated. The average exposure intensity from the start of employment until each examination was found simply by dividing cumulative exposure by duration of employment. Table 2 shows the distribution of exposure indices in the study group. There was no systematic variation between the calendar year of a radiograph and estimated average intensity.

Lifetime occupational histories up to 1978 were available for 95% of the asbestos cement workers. No exclusions were made, as only two workers indicated any notable asbestos or quartz exposure at any other workplace.

Tobacco
For the asbestos cement workers, detailed smoking histories were obtained in the 1978 and 1984 health examinations. Only 17% were lifelong non-smokers. Among the others, 98% had started to smoke before, or within the first two years of the start of employment at the plant.

READING PROCEDURE
Posteroanterior radiographs were classified independently by five readers according to the 1980 International Labour Organisation (ILO) classification of radiographs of pneumoconiosis. The radiographs were read in random order without knowledge of exposure history, age, or date of examination. Eighty-four trigger films from the same laboratory were irregularly interspersed among the radiograph series. The median reading from a previous trial, in which the present readers had participated, was used as a gold standard.

Table 1 Characteristics of workers from an asbestos cement plant

<table>
<thead>
<tr>
<th>Study based on first available radiograph of asbestos cement workers</th>
<th>Blue collar (n = 174)</th>
<th>White collar (n = 29)</th>
<th>Follow up study* (n = 124)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age†</td>
<td>58 (25-79)</td>
<td>49 (31-66)</td>
<td>70 (38-85)</td>
</tr>
<tr>
<td>Smoking category (%):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-smoker</td>
<td>29 (17)</td>
<td>6 (21)</td>
<td>19 (15)</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>41 (23)</td>
<td>11 (38)</td>
<td>38 (31)</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>104 (60)</td>
<td>12 (41)</td>
<td>67 (54)</td>
</tr>
<tr>
<td>Work related variables:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed as investigation (%):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time since start of exposure (y)†</td>
<td>120 (69)</td>
<td>2 (7)</td>
<td>—</td>
</tr>
<tr>
<td>Duration of employment (y)†</td>
<td>23.5 (2-3-59)</td>
<td>16-7 (3-2-50)</td>
<td>31-3 (18-63)</td>
</tr>
<tr>
<td>Average intensity (fcm)†</td>
<td>1.0 (0-1-6)</td>
<td>0 (0-0-6)</td>
<td>0.9 (0-1-4)</td>
</tr>
<tr>
<td>Cumulative dose (fcm)†</td>
<td>19 (1-6-1956)</td>
<td>0.0 (0-0-1-4)</td>
<td>14 (2-1-127)</td>
</tr>
<tr>
<td>Time since end of employment (y)†</td>
<td>—</td>
<td>—</td>
<td>8-9 (4-39)</td>
</tr>
<tr>
<td>Follow up time (between radiographs, y)†</td>
<td>—</td>
<td>—</td>
<td>6-2 (2-12)</td>
</tr>
</tbody>
</table>

*Only blue collar asbestos cement workers for whom at least two radiographs after the end of employment were available.
†Median (range).
‡With respect to last available radiograph in the follow up study.

Table 2 Chest x ray film findings (median readings from first available radiographs) in workers from an asbestos cement plant

<table>
<thead>
<tr>
<th>Asbestos cement workers</th>
<th>Blue collar (n = 174)</th>
<th>White collar (n = 29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small opacities, profusion:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0/0</td>
<td>114 (66)</td>
<td>23 (79)</td>
</tr>
<tr>
<td>0/1</td>
<td>22 (13)</td>
<td>4 (14)</td>
</tr>
<tr>
<td>1/0</td>
<td>16 (9)</td>
<td>2 (7)</td>
</tr>
<tr>
<td>1/1-1/2</td>
<td>18 (10)</td>
<td>8 (6)</td>
</tr>
<tr>
<td>2/1+</td>
<td>2 (1)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Chest wall thickening:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any thickening</td>
<td>84 (48)</td>
<td>5 (17)</td>
</tr>
<tr>
<td>Hard thickening</td>
<td>44 (25)</td>
<td>2 (7)</td>
</tr>
<tr>
<td>Soft thickening</td>
<td>33 (19)</td>
<td>1 (3)</td>
</tr>
<tr>
<td>Any diffuse thickening</td>
<td>25 (14)</td>
<td>2 (7)</td>
</tr>
<tr>
<td>Diaphragmatic plaque</td>
<td>25 (14)</td>
<td>1 (3)</td>
</tr>
<tr>
<td>Costophrenic angle obliteration</td>
<td>14 (8)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Pleural strands</td>
<td>15 (9)</td>
<td>1 (3)</td>
</tr>
</tbody>
</table>
Small opacities of profusion 0/1 were seen on
20% and profusion ≥1/0 on 7% of the trigger
films, sometimes with some chest wall thickening.
The median reading for each item in the
protocol was determined if at least three read-
ers found it possible to classify (which for the
various items was possible in 97–100% of
the films). The median readings were used
when comprehensive indices were calculated.
Of 596 radiographs read, 91% were of good
or acceptable quality, and 8% were of poor
quality.

The individual readings for profusion of
small opacities were in agreement with the
median reading in 70% of the radiographs,
and only 8% of all individual readings
differed more than one minor category from
the median. As hardly any rounded opacities
were noted (95% of all median readings
positive for small opacities were either "s"
or "t") only the category of profusion was
considered. Parenchymal abnormality was
defined as profusion ≥1/0. In the follow-
up study of progression after the end of
employment an increase of one minor
category of profusion was seen in 30 out of
124 blue collar asbestos cement workers.
Regression of one minor category was,
however, found in 15 men. An increase of at
least two minor categories was seen in 24
men, and there were no regressions of this
magnitude. We defined progression as a
change of at least two minor categories, and
thus reduced misclassification of defined
progression (assuming that no true regression
exists; however, it has to be remembered
that random misclassification can only go
in the direction of progress in the lowest
profusion categories).

The presence of diffuse and circumscribed
chest wall thickenings were separately noted,
but the protocol did not distinguish width and
extent for each feature. The individual read-
ings were in agreement with the median
reading in 89% of all radiographs for the presence
of both diffuse and circumscribed changes.
In our analysis, any chest wall thickening
and bilateral chest wall thickening denote
circumscribed or diffuse wall changes regard-
less of size. Definite thickening was defined as
the presence of any chest wall thickening with
a width >5 mm and extent >1/4 of the chest
wall, or a diaphragmatic plaque. Further
restrictions, which excluded firstly all cases
with coexisting costophrenic obliteration, and
secondly all cases with any diffuse thickening,
were intended to represent cases with only
parietal plaques denoted as definite circums-
cribed thickening only. Any diffuse thickening
denotes the presence of diffuse chest wall
thickening, regardless of its size or the coexis-
tence of other pleural changes.

Costophrenic angle obliteration denotes an
obliteration, defined as in the ILO standard
radiographs, regardless of the presence or
absence of other chest wall or diaphragmatic
thickenings. The agreement between individ-
ual and median readings for costophrenic
angle obliteration was 98%.

The ILO reading protocol was extended,
according to a recent proposal for revision of
the ILO classification.8 The presence of
pleural strands, radiating from the pleura into
the parenchyma, either separate or confluent
(crow's feet), or in connection with rounded
atelectases were noted. Apical pleural thicken-
ings were noted if the width was ≥1·0 cm, the
contour was downwardly convex and uneven,
and if there was an asymmetry. A thickening of
the interlobar fissure of ≥1 mm was noted.
Additional standard radiographs for these
features were not provided. This may partly
explain the considerable variation between
the readers of the frequency of notation of these
extensions on all 596 radiographs read;
3–15% for fibrous strands, 1–8% for apical
thickenings, and 0·5–10% for interlobar
fissure thickening.

STATISTICS
We decided to analyse the impact of exposure
on findings from the first available radiograph
for each asbestos cement worker, because all
workers were then included in the analysis
and, also, the individual exposures had a rela-
tively low variability when assessed at the
time of the first radiographs. White collar
asbestos cement workers were also included in
the analysis to obtain a greater variation for
exposure variables that incorporated a mea-
sure of intensity.

Usually, prevalence refers to a certain point
of time; here it is defined with respect to the
radiographs. The odds ratio (OR) was used as
the effect measure for comparison between
different categories of workers with respect to
variables related to exposure. An odds equals
P/(1-P), where P denotes the probability of a
particular finding on the first available radi-
ograph. Hence, it should be noted that the
corresponding onset of abnormality occurred
at some time before the first radiograph was
taken. Logistic regression was used for estima-
tion of ORs adjusted for age and smoking.9 Age
was trichotomised (finer stratification implied
no substantial residual confounding by age), and
smoking was coded as non-smoker, ex-smoker,
and current smoker; these factors were regarded as potential
confounders. When such a factor was included in a
multivariate model, we tested whether there
was evidence of interaction in the data; how-
ever, no significant interactions were found.

For blue collar workers, who had two
radiographs after the end of employment, we
considered progression (at least two minor
categories of profusion) of small opacities. Let
T1 and T2 denote the times of those radi-
ographs (T1 < T2). The determinants consid-
ered in logistic regression analyses were: time
between T and T', age at T', smoking habit
at T', average intensity, duration of employ-
ment, cumulative exposure, time between the
end of employment and T', and profusion
(≤0/1 v ≥1/0) at T1.

Results
SMALL OPACITIES
Table 2 shows the distributions of ILO minor
categories for profusion of small opacities in the first available radiographs from asbestos cement workers. Among the blue collar asbestos cement workers, 20% had profusion ≥ 1/0. Among the white collar asbestos cement workers the corresponding figure was 7%. As the age distribution and smoking habits of the white collar workers differed from those of the blue collar workers (table 1), this prevalence merely indicates the reading level of the panel, and cannot be directly compared with that of the blue collar asbestos cement workers.

Among the 38 blue collar asbestos cement workers with parenchymal abnormality, 18 had also definite chest wall thickenings, and one had an obliterated costophrenic angle.

The time related exposure variables, after controlling for age and smoking, were each associated with the presence of small opacities with profusion ≥ 1/0 (table 3). These time related variables were clearly intercorrelated. Time since start of employment was most evidently associated with the presence of small opacities, with an OR of 7.3 (90% CI 1.9–29) in the ≥ 30 year group, compared with those in the < 15 year group. The average intensity did not influence the probability of profusion ≥ 1/0. The details of a model are exemplified with cumulative exposure as the exposure estimate (table 4). Alternatively when age was handled as a continuous variable (not in table) the result was similar. The effect of exposure was further emphasised when category 1/1 was used as a more stringent cut off point (table 4).

Progression after the end of employment was seen in 20% of the men. The probability of progression increased with intensity and duration of employment, and was indicated also for time since the end of employment (table 5). Neither smoking and age, nor the initial

<table>
<thead>
<tr>
<th>Table 4</th>
<th>Final multivariate logistic regression model, obtained in the analysis of the relation between the prevalence of small opacities and potential determinants (data from 203 asbestos cement workers)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor</td>
<td>OR (90% CI)</td>
</tr>
<tr>
<td>---------</td>
<td>-------------</td>
</tr>
<tr>
<td>Model I (category of profusion ≥ 1/0):</td>
<td></td>
</tr>
<tr>
<td>Cumulative dose:</td>
<td></td>
</tr>
<tr>
<td>≤ 10 f-y/ml</td>
<td>1.0</td>
</tr>
<tr>
<td>&gt;10–30 f-y/ml</td>
<td>1.0</td>
</tr>
<tr>
<td>&gt;30 f-y/ml</td>
<td>2.8</td>
</tr>
<tr>
<td>Age:</td>
<td></td>
</tr>
<tr>
<td>≤ 54 y</td>
<td>1.0</td>
</tr>
<tr>
<td>&gt;55–64 y</td>
<td>7.0</td>
</tr>
<tr>
<td>≥ 65 y</td>
<td>4.5</td>
</tr>
<tr>
<td>Smoking:</td>
<td></td>
</tr>
<tr>
<td>Non-smoker</td>
<td>1.0</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>3.0</td>
</tr>
<tr>
<td>Smoker</td>
<td>3.9</td>
</tr>
<tr>
<td>Model II (category of profusion ≥ 1/1):</td>
<td></td>
</tr>
<tr>
<td>Cumulative dose:</td>
<td></td>
</tr>
<tr>
<td>≤ 10 f-y/ml</td>
<td>1.0</td>
</tr>
<tr>
<td>&gt;10–30 f-y/ml</td>
<td>1.0</td>
</tr>
<tr>
<td>&gt;30 f-y/ml</td>
<td>1.0</td>
</tr>
<tr>
<td>Age:</td>
<td></td>
</tr>
<tr>
<td>≤ 54 y</td>
<td>1.0</td>
</tr>
<tr>
<td>&gt;55–64 y</td>
<td>10</td>
</tr>
<tr>
<td>≥ 65 y</td>
<td>6.3</td>
</tr>
<tr>
<td>Smoking:</td>
<td></td>
</tr>
<tr>
<td>Non-smoker</td>
<td>1.0</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>0.93</td>
</tr>
<tr>
<td>Smoker</td>
<td>1.6</td>
</tr>
</tbody>
</table>

Table 3 | Impact of work related factors on chest x ray film findings in 203 workers from an asbestos cement plant, obtained from logistic regression analyses based on median readings from first available radiographs |
<table>
<thead>
<tr>
<th>Exposure category</th>
<th>Average intensity</th>
<th>Time related factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small opacities (profusion ≥ 1/10):*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Medium</td>
<td>1.4 (0.69, 2.6)</td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>0.55 (0.19, 1.6)</td>
<td></td>
</tr>
<tr>
<td>Definite chest wall thickening:†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Medium</td>
<td>2.1 (1.1, 4.0)</td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>1.8 (0.77, 4.2)</td>
<td></td>
</tr>
<tr>
<td>Definitive circumscripted thickening only:*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Medium</td>
<td>2.2 (1.1, 4.6)</td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>1.8 (0.65, 5.1)</td>
<td></td>
</tr>
<tr>
<td>Any diffuse chest wall thickening:*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Medium</td>
<td>1.3 (0.58, 2.8)</td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>1.1 (0.39, 3.3)</td>
<td></td>
</tr>
<tr>
<td>Costrophrenic angle obliteration:†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Medium</td>
<td>2.1 (0.68, 6.2)</td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>4.5 (1.3, 15)</td>
<td></td>
</tr>
<tr>
<td>Pleural strands:‡</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Medium</td>
<td>1.8 (0.69, 4.8)</td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>1.9 (0.55, 6.2)</td>
<td></td>
</tr>
</tbody>
</table>

Low = intensity ≤ 1 f-y/ml (n = 117); duration ≤ 14 y (n = 84); cumulative dose ≤ 10 f-y/ml (n = 81); time since start of employment ≤ 14 y (n = 57).
Medium = intensity 1–2 f-y/ml (n = 61); duration 15–29 y (n = 78); cumulative dose 10–30 f-y/ml (n = 72); time since start of employment 15–29 y (n = 84).
High = intensity >2 f-y/ml (n = 25); duration >30 y (n = 41); cumulative dose >30 f-y/ml (n = 50); time since start of employment >30 y (n = 62).
*Adjusted for age and smoking category.
†Adjusted for age, no confounding effect of smoking category.
‡Adjusted for smoking category; no confounding effect of age.
§The model is presented in detail in table 4 (model I).
degree of profusion, were significant determinants for progression. Small opacities \( \geq 1/0 \) were found in 40% of the last radiographs (median 31 years after the start of employment).

PLEURAL THICKENINGS

The prevalence of any chest wall thickening on the first radiograph was 48% among the asbestos cement blue collar workers (table 2). Bilateral changes were seen in 30%. Of those with chest wall thickening, 67% had circumscribed thickenings only, 15% had diffuse thickenings only, and 19% had both types of changes. Among the white collar workers the prevalence of any chest wall thickening was 17%.

The time related variables, after controlling for age, were each associated with the presence of definite chest wall thickening (table 3). Smoking category had no impact. In a further analysis (not in table) the intensity of exposure, with age and duration of employment included in the model, contributed significantly, with an OR of 2:1 (90% CI 1:1, 4:1) in the 1–2 f/ml group, and OR 2:2 (90% CI 0:90, 5:2) in the >2 f/ml group.

The findings for definite circumscribed thickenings were similar. In a further analysis (not in table) the intensity of exposure contributed to an OR of 2:3 (90% CI 1:1, 4:8) in the 1–2 f/ml group, and an OR of 2:2 (90% CI 0:76, 6:5) in the >2 f/ml group after adjustment for age, duration of employment, and smoking (for which a very small confounding effect was found).

The presence of any diffuse chest wall thickening showed weak or no relations with the exposure variables examined.

OBLITERATION OF THE COSTOPHRENIC ANGLE

Among the blue collar asbestos cement workers, the prevalence of obliteration of the costophrenic angle was 8% (table 2). Of the 14 radiographs, two showed bilateral obliterations, and 10 showed a diffuse ipsilateral chest wall thickening. On one radiograph there were small opacities \( \geq 1/0 \). Among the white collar workers there were no obliterations.

Among the 14 asbestos cement workers with obliterated costophrenic angles, one case of malignant mesothelioma was diagnosed 15 years after the appearance of the obliteration. No lung cancers have been found. Thus, we assume that all obliterations represent benign effusions. For none of these 14 radiographs did the readers comment on suspected tuberculosis or fractured ribs, and only one had a noted enlargement of the cardiac outline.

In contrast with the findings for parenchymal changes and other pleural changes, duration of employment and time since start of employment showed no association with the presence of obliterated costophrenic angles after adjustment for age (table 3). The average age intensity was, however, clearly associated with obliterations with ORs of 4:5 (95% CI 1:3–15) in the >2 f/ml group and 2:1 (90% CI 0:68–6:2) in the intermediate group, compared with the \( \leq 1 \) f/ml group. Smoking had no influence. We further studied all obliterations found before the age of 65. Obliterations occurred in 116 men during employment, whereas obliterations occurred in only one man out of 45 after the end of employment (Fisher's exact test; \( P \) (two sided) = 0:28).

PLEURAL STRANDS

Strands were seen on 9% of the first radiographs among the blue collar workers (table 2), and were associated with other pleural changes in all but three of the 15 radiographs. Nine radiographs also showed small opacities \( \geq 1/0 \). There was no radiograph with a median reading that indicated a rounded atelectasis. Only one of the 29 white collar workers had visible strands.

Neither time since the start of employment nor duration of employment were associated with strands (table 3). For the highest cumulative exposures, the risk was significantly increased. The point estimate also increased with increasing intensity (not significantly; in the combined group \( \geq 1 \) f/ml an OR of 1-8, 90% CI 0.76, 4.4 was found).

OTHER FEATURES

Only two asbestos cement workers had apical pleural changes on the first radiograph available. With the last radiograph instead, there were 14 out of 124 blue collar asbestos cement workers with apical pleural changes. On all but one of these radiographs there were other chest wall thickenings, of which seven also included obliterated costophrenic angles. One radiograph showed small opacities in the upper zones of the lungs with an overall profusion of 2/3, and eight others had small opacities \( \geq 1/0 \) elsewhere. Apical thickenings were not found among the white collar workers.

A thickened interlobar fissure on the first radiograph was noted in three asbestos cement blue collar workers. With the last radiograph instead, there were seven workers with fissural thickenings. In six of them, other chest wall thickenings were present. Three of them had profusion of small opacities \( \geq 1/0 \). Fissural thickenings were not found among the white collar workers.

Table 5  Final multivariate logistic regression model, obtained in the analysis of the relation between progression of small opacities after the end of employment and potential determinants (data were from 123 blue collar asbestos cement workers (one missing))

<table>
<thead>
<tr>
<th>Factor</th>
<th>OR (90% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average intensity (f/ml):</td>
<td></td>
</tr>
<tr>
<td>( \leq 1 ) (n = 68)</td>
<td>1:0 —</td>
</tr>
<tr>
<td>1–2 (n = 44)</td>
<td>1-4 (0:54, 3:4)</td>
</tr>
<tr>
<td>&gt;2 (n = 11)</td>
<td>6:7 (1-8, 25)</td>
</tr>
<tr>
<td>Duration of employment (y):</td>
<td></td>
</tr>
<tr>
<td>( \leq 14 ) (n = 35)</td>
<td>1:0 —</td>
</tr>
<tr>
<td>15–29 (n = 53)</td>
<td>3-2 (0-75, 14)</td>
</tr>
<tr>
<td>( \geq 30 ) (n = 35)</td>
<td>19 (4-1, 84)</td>
</tr>
<tr>
<td>Time between end of employment and last radiograph (y):</td>
<td></td>
</tr>
<tr>
<td>( \leq 7-5 ) (n = 28)</td>
<td>1-0 —</td>
</tr>
<tr>
<td>7-5–14 (n = 73)</td>
<td>1-1 (0-39, 3-3)</td>
</tr>
<tr>
<td>( \geq 15 ) (n = 22)</td>
<td>3-6 (0-91, 14)</td>
</tr>
</tbody>
</table>
Discussion
In previous studies from this asbestos cement factory we have reported data on mortality and cancer morbidity,1 lung function,10 histopathological findings,11 and asbestos fibre content in lung tissue.12 Our present study adds complementary information on radiographical findings. The study base—men alive in the beginning of 1978—represents about 60% of all blue collar workers employed in the factory for at least 10 years. Radiographs were available for 70% of the men in the study base, however, only for three of the 16 men (19%) who died during 1978 and 1979. Hence, the study investigates survivors. Although it is difficult to evaluate the overall selection bias, we believe that our results tend to underestimate, rather than overestimate the effects of exposure.

Independent randomised reading according to the ILO classification has been advocated whenever an estimate of prevalence is required from serial films because of the bias that may occur in side by side reading.13 Independent randomised reading was also used for determination of progression for feasibility reasons. The readers in our present study were jointly trained in the use of the ILO classification, which may explain the relatively small between observer variability. We used median readings in all analyses to reduce the random variations in the outcome classification. The over or under reading level seems reasonable, as judged from the 7% prevalence for small opacities of profusion ≥ 1/0 found in the white collar workers, compared with the reported 4% prevalence from a sample of men from the general population with a mean age of 50.14

The median cumulative asbestos exposure among the blue collar workers was 19 (25, 75 percentiles 8.6, 47) f/g/ml. Thus, we are investigating men with low to moderate asbestos exposure on a group level. The validity of exposure estimates is crucial, when exposure-response relations are evaluated. Our estimates of airborne levels of asbestos fibres in the plant are substantially lower than those presented from some asbestos cement plants,15-17 but in the same range as in others.18 19 Both differences in the interpretations of results from early stationary sampling and real differences in the production technology between the plants are likely to contribute to these discrepancies. Detailed occupational histories were taken at a personal interview and from company data for most subjects. Thus, should we have underestimated exposure levels, we still assume that we have assigned the workers to the right category.

A further problem in any prevalence study, but not always recognised, is that the time of onset of an abnormality is not known. Duration of employment, cumulative exposure, and time since the start of employment to the onset of an abnormality will thus be overestimated. This might result in an underestimation of the slopes of these exposure-response relations that assume fixed exposure categories. In contrast, average intensity would tend to be underestimated, assuming that exposure levels gradually diminished with time. In the follow up study, these drawbacks were reduced for the time related exposure variables, and disappeared for intensity.

PARENCHYMAL ABNORMALITY
Asbestosis was, in the days of intense exposures at the beginning of the century, a rapidly progressive disease.20 With the considerably lower exposure levels encountered later on, it has become evident that time since the start of exposure is an important predictor of the risk for onset and progression of radiologically detectable parenchymal abnormality.21-24 As in several other studies on the prevalence25-27 and progression22-24 28 29 of small opacities, time related variables such as duration of employment and cumulative exposure showed positive associations. Our findings for small opacities thus agree well with the present knowledge.

With 1/0 as a cut off point for parenchymal abnormality, the estimated smoking effect was of the same magnitude as the effect in the highest exposure group. The point estimate for smoking agrees with estimates from other workforces exposed to asbestos.30 The use of 1/1 as a cut off point for parenchymal abnormality instead of 1/0 indicated a more pronounced impact of asbestos exposure, and considerably reduced the OR associated with smoking relative to the estimated effects of exposure—a finding that is consistent with the assumption that cigarette smoking in itself may cause mild, but not considerable fibrosis.31 Thus, the higher degrees of profusion probably reflect a more specific response to asbestos exposure.

The average intensity of exposure was not a determinant for the presence of small opacities in the prevalence study. Indeed, as exposure levels fall, personal characteristics may become more important in the assessment of exposure, including not only the amount of fibres delivered to the lung tissue and retained there, but also the nature of the response. Workers with high average intensity had, however, an increased risk for progression after the end of employment, when duration of employment was taken into account. One can speculate that this finding actually reflects exposure of such a degree as to cause alveolar macrophage overload that resulted in diminished clearance and subsequently a higher tissue dose after exposure, which is not accurately represented by a cumulative exposure measure.32

In workers from the asbestos cement plant studied, fibrosis in histopathological slides from the lungs of deceased workers was more common and more pronounced than among controls.33 In groups of workers with symptoms or signs suggestive of respiratory disease, functional changes were also found that reflected lung fibrosis34 and increased lung density in computerised chest radiographs.35 The mean estimated cumulative exposures in those study groups was about 20-25 f/g/ml. In our present study, the risk for small opacities ≥ 1/0 was increased to almost threefold at >30 f/g/ml.
PLEURAL ABNORMALITY

Different types of pleural reactions are seen in association with asbestos exposure. The main reactions are parietal plaques on the one hand, and acute pleurisy and diffuse pleural fibrosis on the other. The original ILO 1971 classification did not allow for a clear distinction between circumscribed plaques and diffuse chest wall thickenings, and although an amendment was made in the 1980 revision, it is evident that a posteroanterior chest radiograph cannot always distinguish the two entities. In a longitudinal study of asbestos workers and controls further investigations in people with diffuse thickening, from reviews of clinical records, additional histories, oblique views, and other diagnostic procedures, showed that these thickening were actually the result of multiple confluent plaques in 25%.[24] Such thickenings rarely extended to the costophrenic angle. Recent experience with high resolution computerised tomography scans have further shown the common coexistence of plaques and visceral pleural lesions,[30] also indicated in our study.

The difficulties of distinguishing between parietal plaques and visceral pleural lesions, with the ILO codes, are probably part of the explanation for the similarity in exposure-response relation patterns found between the wider definition of pleural thickenings and the one intended to represent pleural plaques only.

Our findings on the exposure-response relations for pleural thickenings are consistent with present knowledge, with emphasis on the long period that is necessary for the development of visible thickenings, and the very small impact or absence of a smoking effect.1 The common experience that even minor asbestos exposure may cause pleural plaques, provided the follow up period is long enough, was shown in this study, as a few cases occurred among the white collar workers.

Apical pleural changes have been ascribed to asbestos exposure,[10] but may also be present in the general population.41 The prevalence of such changes as we defined them was low, and they were usually associated with other pleural changes. The prevalence of interlobar thickening was also low and, as previously found, seldom the only abnormal finding.38 More subtle definitions than those we used, combined with the use of lateral views, may be more sensitive for the diagnosis of thickening of the interlobar fissure,3 but with our robust criteria, such thickening was not an early sign of asbestos related disease.

In one longitudinal study, 31% of the cases with diffuse thickenings had had a previous benign pleural effusion, not related to trauma or infection.42 Resolution of an effusion that was ascribed to asbestos exposure usually (>90%) resulted in an obliterated costophrenic angle. Resulting diffuse pleural thickenings were less frequent, about 50%.2 Thus, involvement of the costophrenic angle seems to be a more sensitive indicator of a previous episode of effusion than the presence of diffuse chest wall thickenings. For benign visceral pleural abnormalities the epidemiological evidence of a gradient in pathogenic potential by fibre type is scarce.41 In our study, all but one of the workers with involvement of the costophrenic angle had been working during the time when exposure to crocidolite or amosite might have occurred. The group of workers first employed after the disappearance of amphiboles in the production is, unfortunately, too small to evaluate the effects of chrysotile exposure only.

Pleural effusions appear suddenly, in contrast with the slowly developing parietal plaques. The biological mechanisms behind the pleural responses to asbestos fibres are, however, not elucidated. It is evident from necropsy studies that the tissue fibre burden is greater in patients with diffuse pleural fibrosis than in patients with plaques alone.[39] In our study, the exposure variables related only to time were not associated with an increased risk of obliterations, but average intensity, and to a lesser degree cumulative exposure, obviously were. That the risk is related to exposure intensity has also been indicated by previous findings.[40] Our findings are in accordance with reports that benign effusions in workers exposed to asbestos occur with short latency after the onset of asbestos exposure.40,41 The exposure-response patterns for obliterated costophrenic angles were thus generally different from those we found for parenchymal and chest wall abnormalities. This may indicate differences in the underlying mechanisms of these changes.

The radiographs were classified by Gerd Lindgren, Nils Stjernberg, and Göran Tomling. MA and KJ were trained by Charles E Rossiter. Bev Embrey handled all radiographs and the data collecting, for which Cases Hallstrom gave financial programming aid. Financial support was given by the Faculty of Medicine, Lund University, and the Swedish Work Environment Fund.


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