Asbestos: Update and assessment of the airborne exposure data used in the Hodgson and Darnton meta-analysis and its effects on the estimates of the cumulative exposure

**Aim and background**

The aim of this work was to re-examine and update the airborne sampling data for some of the pivotal epidemiological studies that contribute to the dose response relationships in the quantitative meta-analyses of risk from airborne asbestos exposures. The analysis by Berman and Crump (2004) and Hodgson and Darnton (2000), in particular, looked at the sampling data available and combined this to calculate the cumulative exposure. In the case of the Hodgson and Darnton (H&D) analysis, this was derived as a single figure for the cohort in terms of fibres-years/ml by a number of ways depending on the information available. This included multiplying the estimated arithmetical average airborne fibre concentration based on phase contrast microscopy (PCM) fibre count index by the average duration of the exposure, the mid-point of the range of exposures or by weighing separate exposure groups by the expected deaths from lung cancer. Berman and Crump (2004) assigned a risk to the quality of the data available and consistently the airborne fibre concentrations came out as by far the highest level of uncertainty. This and subsequent discussions at the WATCH meeting in November 2007 lead to a work item to look further into the H&D meta-analysis to determine the robustness of the key points. Examination of the air concentration data was one of the assessments to be undertaken.

It should be noted that very little updating of any of the cohort outcomes have appeared since the H&D paper was published, and those that have been produced (e.g. Berry et al., 2004 and Hein et al., 2007) have not made a significant difference to the previous rates.

**Method**

Giving the time and limited resource it was decided to determine which cohorts in the meta-analyses were most important. In discussion with H&D it was clear that cohorts with some quantitative airborne exposure data to a single asbestos fibre type, were the key data points to revisit. There were eleven such cohorts in H&D, (three crocidolite, two amosite and six chrysotile). It was also considered that amphibole exposures, which gave the highest dose-response, were particularly important and any further quantitative data would be particularly helpful for establishing the shape and slope of the dose response curves.

Chrysotile has lower risk and is often more problematic, as most chrysotile cohorts have also been exposed to small but potentially significant amounts of amphibole fibres. Most asbestos cement processes had a mixture of chrysotile and amphiboles asbestos used and the classification of the cohorts and the amount of mixed exposure is discussed in H&D (2000). The presence of tremolite asbestos impurities in some chrysotile mines has also been suggested as the cause of the risks observed. Conversely, the introduction of small amount of chrysotile into a predominantly amphibole fibre situation will have much less effect on the estimate of the risk. The studies that had predominantly mixed exposures of asbestos types have not been looked at in this paper, as their contribution to a meta-analysis of the risk by fibre type is limited. The chrysotile risk is dominated by the experience of the South Carolina cohort which have much higher lung cancer rates compared to the other chrysotile “only” cohorts and especially the mining and milling cohorts. These have been looked at in detail as they represent the worst and best case for chrysotile exposure.
The air concentration data available is reviewed and discussed. The average cumulative exposure based on the air measurement is also given. This is usually derived from an exposure matrix based on the type and duration of work carried out and job information, from the employee records, to produce an average cumulative exposure estimate. This approach treats each cohort as a single exposure experience so any systematic biases in the air concentration data will have a direct effect on the mean cumulative exposure. Some H&D cohorts had adjustments to the mean cumulative exposure to account for variations in the methodologies used.

To simplify the process three types of biases were looked for in the air concentration data:
Extent of data used for estimating air concentration over time,
Representative ness of the data to historical conditions,
Conversion of data to PCM equivalent fibre counts.

To avoid confusion a fourth bias, the relationship between the PCM fibre count at the date of sampling and its conversion to a current day PCM fibre counts is discussed separately, in appendix 2. The reason for this is firstly, that converting all the data used for the H&D risk estimates to account for what a modern day PCM measurement would give will require extensive recalculation of the data and it is far easier to give an estimated additional protection factor that might be expected from a current workplace or environmental exposure measurement. Secondly, as several cohorts did not have any PCM data it is difficult to apply this process uniformly. These estimated conversions are however, given in the summary tables for convenience.

**Limitations**

There are of course many limitations on the extent of the data available and although the original workers often went to substantial lengths to assess the cumulative exposures they were often working from very limited measurements of the airborne concentration. Although it is at best ambitious to think that this can be done better with a less intimate understanding of the conditions: the focus on the air concentration measurements can highlight inconsistencies or supporting data from lung burden studies, which have come to light since. It is also possible to compare the measurements across the sectors to see if the estimates of the air concentrations that were made are consistent. One additional point is that nobody has updated the fibre estimates to take account of what a current PCM analyst would have counted.

**Overview of airborne asbestos fibre measurement for estimating the risk**

Airborne asbestos fibres have a range of dimensions with lengths from <1 – several hundred micrometres (µm) and widths from 0.01 - > 3 µm. The current index of exposure used for epidemiology and risk assessment is based on optical phase contrast microscopy (PCM) which counts visible fibres > 5 µm long and less than 3 µm width. A fibre is broadly defined as a particle with an aspect ratio of >3:1. Due to the limited resolution of light, PCM will not be able to see fibres below about 0.2 µm in width but the actual performance depends on the way the microscope is set up, the quality of the illumination and optics, the visual acuity of the operator and other subjective influences.

Only the much higher resolution from transmission electron microscopy (TEM) can give an accurate fibre size distribution and the PCM index will be constricted to a few percent of the total numbers of asbestos fibres present. Mechanistically it has long been argued that fibres with lengths great enough to give incomplete phagatosis (approximately 15 - 20 µm) are responsible for the adverse health effects. Various animal experiments (e.g. Stanton and Wrench, 1972; Pott et al, 1974; Davies et al., 1986, Berman et al., 1996) have shown the
importance of long durable fibres for carcinogenesis and arguably much greater clarity of the risk would have been obtained had >15 - 20µm long fibres been used as the index of exposure, as proposed by Merryweather, (1938) instead of >5 µm long definition adopted by the Asbestosis Research Council some 25 years later (see Walton, 1982).

Even though most of the risk resides in the longer fibres, the >5 µm long PCM index does not exclude them unless they are too thin to be visible. This undercounting by PCM of long thin fibres may in part (or full) account for the potency differences observed in cohorts for different types of amphibole fibres (e.g. crocidolite and amosite). Some authors contend that very thin fibres not visible by PCM are related to specific asbestos related diseases such as mesothelioma (e.g. Lippman, 1990). Again if this is the case, the use of the PCM fibre count will only be an indirect assessment of their concentration. In practice there is little that can be done about this unless TEM data is available, and this is reviewed in appendix 1.

Use of the current PCM index of risk is further complicated by the fact that many of the cohorts had limited or no measurements made before 1960 and if they were done it was usually in terms of all particles rather than fibres using different methods of sampling and a range of microscopy techniques. However, there were exceptions and these studies are of particular value.

Membrane filter sampling and PCM analysis was first described in 1959 and developed in the UK in the early 1960’s by the asbestos industry / asbestosis research council (ARC) (see Addingley, 1964; Holmes, 1964 and Roach, 1964). The use of the membrane filter sampling was significant as it heralded a change from mostly area (static) sampling to personal sampling, which in may in indoor industrial environments give increased concentrations of up to an order of magnitude higher. Also membrane filter sampling cemented the use of an index of exposure based on fibre concentrations rather than particle concentrations, which had been introduced for the LRTP sampling in some UK factories and SA mines. The uniformity of the PCM analysis did not come immediately and several further improvements were added over the next 20years (Walton, 1982) to improve the PCM fibre counting. During this period of initial and further development, many of the cohorts used in the meta-analyses were sampled to compare the historic method for exposure assessment with the new PCM method. Undoubtedly the period when this was carried out and the experience of the persons involved would affect the conversion ratios as the current PCM count would give significantly higher counts due to a number of improvements. Although some authors have attempted to roughly quantify the effect of the various improvements (e.g. Rickards, 1984) not all of these would apply depending on the set up used at the time as well as asbestos fibre type being assessed. Thus there is no simple bespoke solution to looking at the robustness of the estimates of the fibre concentrations; this is looked at in more detail in appendix 2.

Alongside the technical issues of the assessment methods and conversion factors, the areas and activities sampled, and more importantly not sampled, plus the plant conditions at the time will all affect the arithmetic average calculated. From the point of the H&D analysis the use of a single cumulative exposure to characterise the whole cohort, the precision and confidence of the exposure estimate will be dependent on the number of data points and the biases and systematic errors present in the data.
Crocidolite exposure results

Wittenoom Occupational

Blue asbestos was mined and milled at Wittenoom in Western Australia between 1943 and 1966. Some 7000 male workers who worked at the Wittenoom mine and mill have been followed up (75% of cohort traced). To the end of 2000 (Musk et al., 2007), there have been 190 cases of pleural and 32 cases of peritoneal mesothelioma, a raised mortality from lung cancer (SMR=1.52) and particularly pneumoconiosis (SMR=15.5). The workforce was young, (57% aged less than 30 years) and transient with the average duration of exposure was 4 months. The estimated cumulative exposures are low due to the short period of employment and Armstrong et al., 1988 estimated 55.8% of the workforce had <10 f year/ml, 29.1% had 10-100 f year/ml and only 4.7% exceeded 100 f years /ml. Although the rates of mesothelioma are slowing the long lag time to disease means that the numbers will continue to rise.

Exposure estimates are complicated as various mines and mills are involved. Low level “pick and shovel” mining activity started in 1936 and the Wittenoom Gorge mine and mill operated from 1943 –1958 and also hired previously exposed workers from the Yampire Gorge crocidolite mine and mill which operated from 1937 –1946. A new mill was constructed in 1949 and various modifications and attempts at dust control were made until its closure in 1958. The colonial Gorge mine started and mill commenced operation in 1953 and ran to its closure in 1966 for economic reasons.

The only attempt at fibre level monitoring see (Major, 1968 and Rogers and Major, 2001) was made in 1966 at the Colonial Gorge mine about 2 months before it closed, 38 long running thermal precipitator (LRTP) static samples were collected. The results were reported only in very general terms based on some measurement over 12 shifts with the number of fibres expressed as “about 10%” for the mine samples. The results have been summarized in table 1.

<table>
<thead>
<tr>
<th>Process</th>
<th>P/cc &gt;0.5 µm</th>
<th>All fibre lengths s/cc</th>
<th>&gt;5 µm long fibres f/ml</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Colonial Mine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Miner and scraper operator</td>
<td>1500</td>
<td>120</td>
<td>20</td>
</tr>
<tr>
<td>Ross ore feeder</td>
<td>100</td>
<td>10</td>
<td>~1 –3</td>
</tr>
<tr>
<td>Picking belt and drier area</td>
<td>200</td>
<td>20</td>
<td>~1</td>
</tr>
<tr>
<td><strong>Colonial Mill</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plant operator control platform</td>
<td>3000</td>
<td>670</td>
<td>270</td>
</tr>
<tr>
<td>Hand bagging</td>
<td>3000</td>
<td>600</td>
<td>100</td>
</tr>
<tr>
<td>Mechanical bag press operator</td>
<td>2000</td>
<td>330</td>
<td>80</td>
</tr>
</tbody>
</table>

The use of static sampling undoubtedly underestimates the personal exposure and has to be taken into account and the way the ore was mined suggests that the personal exposure to the miner and scraper operator would have been at least double the static sample result, similarly without knowledge of where the LRTP was placed, personal exposures would normally expect to be at least double for those actively involved in the process operation and only the Plant control operator result may be representative of the actual personal exposure. The relationship between LRTP fibre count and the early PCM method was taken as 1:1 by the ARC (See Walton, 1982) comparison in the S African mines also produced a lower conversion value. Therefore it could easily be argued that the average >5 µm long airborne
concentrations in the mines were around 15 - 40 f/ml and the mills were 100 - 250 f/ml, with the higher values towards the earlier dates.

Other sampling carried out prior to 1966 were particle counts using a konimeter by the Mines Dept of Western Australia between 1948 – 58, as the vast majority of the results recorded were 1000+ p/cc, representing overloaded samples, these results were of limited value, other than showing airborne concentrations were consistently high (Rogers and Major, 2001). Reid et al (2008) reports that between 1948 –51 the results were considered to be some 6-8 times safe levels but also mentions that anecdotal evidence suggests that operations were shut down before inspections commenced.

Further attempts to verify the airborne concentration was undertaken by measuring the lung burden of fibres from deceased workers (de Klerk et al., 1996). A log v /log plot of some 90 cases showed an overall trend between estimated cumulative exposure and lung burden but the points on a similar estimate of cumulative fibre exposure were generally spread over some two – four orders of magnitude, and it was perhaps an over interpretation to suggest the lung data showed that the previous airborne exposure estimates were reasonably reliable. Based on various assumptions the residence time in the lung was estimated to have a half–life of 92 months. The constant removal of fibre was the reason for a reduction in risk for lung cancers long after exposure had ceased. One interesting point of the (de Kerk, 1996) paper was that the intensity of exposure was expressed in terms of a geometric mean of 20 f/ml and the cumulative exposure was given as geometric mean of 20.9 f/ml. Given the high rates of pneumoconiosis reported from Wittenoom workers the lung burdens may be affected by the reduced ability for lung clearance to take place for fibrotic areas of the lung.

Wittenoom Environmental

The original town of Wittenoom was established 1 km from the original Wittenoom Gorge mine and mill but was moved 12 Km away in 1957 closer to the site of the Colonial Gorge mine and mill. Mine tailings containing crocidolite were widely used for roads playgrounds, backyards etc. giving rise to widespread environmental sources of airborne crocidolite. The environmental conditions when the mine was operating, due to traffic movements, transport of tailings and fugitive emissions, were very different from the situation after closure from December 1966 onwards. Also, most non-workers had para-occupational exposures from family members. In an early study (Hansen et al. 1997), 24 of the 27 cases of mesothelioma occurred in people who lived in the same house as a Wittenoom crocidolite worker and only one case has been diagnosed in a resident who first went to Wittenoom after the mining operations ceased. Latest information (Reid et al., 2008) for women residents (non-workers) showed that 219 women (72%) of those who developed a cancer lived with a worker at the mines.

A total of 4,768 residents of the town of Wittenoom have now been followed up to the end of 2002 where 67 cases and 64 deaths with mesothelioma have occurred (Reid et al. 2007). The mortality rate with mesothelioma increased with increasing residence duration, time since first exposure, and estimated cumulative exposure. The mesothelioma mortality rate was consistently lower for female subjects when compared with male subjects, but the cumulative exposures were estimated at 5.9 and 4.9 f.years/ml respectively, which produced a steeper dose-response curve for female subjects. The rate was lower in those first exposed as children compared with those first exposed at > 15 years of age. The dose-response slope for asbestos exposure and mortality from mesothelioma was not different between those who were first exposed as children than those who were first exposed at > 15 years of age.

In a study of the 2,608 females (Reid et al., 2008) covering the period 1960 –2005 about 2.5% of the workers compared to 1% of the resident had mesothelioma (total 47) based on a median duration of exposure of 1.3 years. These mesothelioma rates were ~77 times higher than the
background rate for Western Australia Careful separation of this cohort is necessary as 11 female workers contracted mesothelioma but only one worked in production (bagging), the four others had clerical and canteen jobs < 1 km from the mill and four other worked in the town. Average duration of residence for those developing mesothelioma was 4.5 years for women non-workers, compared to 2.2 years for women workers. Of the 600 women residents who lived in Wittenoom since the mine closed only one has developed mesothelioma. Estimates of cumulative exposures for women were given as 1% >40, 1% 30 – 40, 3% 20 – 30, 11% 10 – 20 and 84% <10 f.years/ml, with significantly greater exposures for residents compared to workers, 12.2 and 3.6 f.years/ml, based on airborne concentrations estimated at 3.1 and 1.7 f/ml, respectively

Environmental measurement data were collected on several occasions, the first being collected by LTRP in 1966 (Major, 1968), which found concentrations of 2 and 0.5 f/ml some one hundred metres outside the mill. Other environmental data was obtained from membrane filter sampling and PCM analysis of personal and static samples were taken infrequently in 1973, 1977, 1978, 1980, 1984, 1986 and 1992. For the epidemiology studies people not working directly with asbestos were assigned an intensity of exposure of 1.0 PCM (f/ml) of air from 1943 to 1957 (when the new mill was commissioned), and then 0.5 f/ml between 1958 and 1966, when the mining operations ceased. Interpolation between surveys using personal monitors assigned exposures from 0.5 f/ml in 1966 to 0.01 f/ml in 1992. Exposures were measured and assessed using occupational methods (average over an 8-h working day, 5 d/wk) and the cumulative exposure figures were multiplied by 4.2 (ie, 168/40) to represent 24 hour, 7 day a week residential exposure.

As personal PCM measurements were only taken in 1977, 1978, 1980 and a high percentage of these were overloaded with dust, it is very likely that the mean is an underestimate. The static samples gave much lower results than the personal samples with the highest personal sample being 0.88 f/ml. These results were taken over 10 years after mining ceased and the lack of any environmental PCM data when the mine and mill were working means that the early exposures are guestimates. As ~80% of the environmental cohort started their exposure when the mine was operating the assignment of cumulative exposure during the mine operation period were not based on airborne measurements. There are no measurements of the para-occupational exposures at Wittenoom.

The contributions from the mine when working to the environmental and para-occupational exposures was likely to be considerable, as shown by the accounts of pilots flying into Wittenoom by the blue haze visible from a long distance, to the clerical staff in the company office well away from the production areas being able to write in the fibrous dust that settled on their desks (Reid, 2008).

South African mines and mills

Commercial amphibole asbestos production in South Africa started in the 1890’s. The Cape crocidolite and the Transvaal crocidolite andamosite mines and surface processing mills were sampled infrequently from about 1940. Most of the employees were non-white and they were mainly itinerant workers who worked for 1 year or less and no epidemiological or medical follow up is available. Therefore the only epidemiology relates to a minority of white workers who often had the less dusty jobs. Only <1% the cohort reported by Sluis-Cremer et al., 1992 had exposures before 1940, and 6.4% were exposed before 1950 when the air levels were much higher. Over the next three decades about one third of the remaining cohort was first exposed in the 50’s, 60’s and 70’s when air measurements were consistently lower (see figure 1). The follow up in 1980 represents a relatively short period since first exposure for the majority of the cohort and no follow up of this cohort has been done since, to establish the true disease potential from the exposures. An adjustment was used to account for the limited follow up in the original H&D paper.
What occupational measurements were made had their own inherent problems; they were often averaged for the mine and the mill, and then for groups of mines, or over time. At varying times measurements were made using two types of konimeters and two types of thermal precipitators. Furthermore, it was not always clear what was being measured, viz. particles and/or fibres. The definition of a fibre is determined by the ratio of its length to diameter and the measurement of the diameter itself. This definition was not constant; the fibre length: diameter ratio was changed from two to three in 1965, and the diameter from five to three microns in 1970 (du Toit, 1989). Also as different optics were also used for the evaluation (e.g. x150 dark field and x1000 light field) and various sample treatments (e.g. acid washing and incineration) were used at different times, it is difficult to establish a single reliable index of exposure. The PCM method (ARC, 1971) was introduced for health surveillance in 1975, although reference is made to a large PCM survey in 1970. A summary of the early measurements is given in figure 1.

Figure 1: Summary of the historical airborne particle concentrations made in the main South African Amphibole mining areas (Cape and Transvaal) using the konimeter (1940 –55) and thermal precipitator methods (1965 – 71). (Note: blue lines = crocidolite)

Du Toit (1977) concluded that in general the limit of visibility for particles in a x150 dark field count with a konimeter could be equated to a thermal precipitator count of >0.5 µm using x1000 light field microscopy.

Given that about 94% of the cohort were exposed in the 1950 –70’s it should be possible to give a reasonable estimate their average of >5 µm long PCM fibre exposure from side-by-side comparisons of the monitoring techniques. Also on some occasional fibre counting took place from the 1950s. Although comparing particle counts to a fibre counts by separating out the fibres should be relatively straightforward process, the attempts to do this for the PCM membrane filter method in the South African mines are difficult to reconcile. Du toit and Gilfillan (1977, 1979) originally used 8 µm pore size filters instead of the recommended 0.8 µm pore size. The conversion factors are summarized in table 2, along with a later set of values produced in co-operation with the Asbestos International Association (AIA) (Du toit et al., 1983) which after detailed statistical analysis were found to be around five times lower. However, this last set of values was extremely dependent on one high data point and the slope could change from 1.09 to 2.68 without this point (see Burdett, 1998).
Table 2: Summary of South African mine conversion factors in terms of the expected Membrane filter fibre count from a concentration of 1 f/ml measured by the konimeter and the (standard thermal precipitator).

<table>
<thead>
<tr>
<th>Pore size (µm) (publication)</th>
<th>Cape Crocidolite</th>
<th>Transvaal Crocidolite</th>
<th>Chrysotile</th>
<th>Amosite</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 (D&amp;G, 1977)</td>
<td>0.75 (0.9)</td>
<td>2.2 (1.9)</td>
<td>4.6 (7.3)</td>
<td>4.4 (5.7)</td>
</tr>
<tr>
<td>0.8 (D&amp;G, 1979)</td>
<td>4.3 (4.5)</td>
<td>6.8 (5.7)</td>
<td>8.1 (9.8)</td>
<td>7.9 (8.8)</td>
</tr>
<tr>
<td>0.8 (D et al., 1981)</td>
<td>1 (0.53)</td>
<td>1 (0.53)</td>
<td></td>
<td>1 (0.53)</td>
</tr>
</tbody>
</table>

The estimates of the actual fibre concentrations used for the epidemiology are given in table 3. However, given the uncertainties between conversion between indices and methods it is perhaps only possible to conclude that recorded levels of dust in the Cape crocidolite mines and mills, and the Transvaal Amosite mills fell by about a factor of 3 between 1940 – 45 to 1950 – 55; but over the same period the Amosite mines increased by about a half. A similar trend was seen from 1965 – 1971 when levels in both the Cape and Transvaal crocidolite mines and mills fell usually by around one half. However, these falls did coincide with an increase in production and a much greater number of people being exposed (see figure 2). The crocidolite cohort of 3430 men had an average exposure of 9.6 f/ml, which was multiplied by the number of years employed to give the cumulative exposure.
Table 3: Overall estimated average in South African mines (underground) and mills (surface) (Sluis-Cremer et al., 1992) in terms of PCM f/ml.

<table>
<thead>
<tr>
<th>Year</th>
<th>Amosite surface</th>
<th>Amosite underground</th>
<th>Crocidolite surface</th>
<th>Crocidolite underground</th>
</tr>
</thead>
<tbody>
<tr>
<td>1945</td>
<td>150</td>
<td>14</td>
<td>30 – 160</td>
<td>2 – 60</td>
</tr>
<tr>
<td>1960</td>
<td>56</td>
<td>6</td>
<td>10 – 50</td>
<td>2 – 6</td>
</tr>
<tr>
<td>1970</td>
<td>40</td>
<td>4</td>
<td>8 – 30</td>
<td>2 – 6</td>
</tr>
</tbody>
</table>

By the early 1980s when membrane filter PCMs were being collected, the results from two laboratories for 10 crocidolite samples from the mills ranged from 0.5 – 57.2 f/ml and three amosite mill samples were 20.4 – 41.2 f/ml. Only one mine / underground sample was available from each site with the highest laboratory count being 0.75 and 0.3 f/ml respectively (Du toit et al., 1983). This suggests air concentrations in the mills remained high.

**Massachusetts cigarette filter manufacturing**

There were one set of air measurements from this small facility in 1952 (Talcott et al., 1989) and the likely airborne concentrations were estimated in an appendix in the Hodgson and Darnton paper. This involved the conversion of impinger measurements and the experience of historic measurements in a crocidolite textile factory were used to estimate a modern PCM count. Further evidence for exposure can be found from the reported lung burdens from two workers who processed the crocidolite containing filters made in at the Massachusetts facility at another plant in Kentucky (Dodgson et al. 2002). Both of the workers developed mesothelioma.

One person who worked at the factory during 1953-54 cut the lengths of filter plugs received to the correct size. The work area was described as very dusty and the lung burden of 77 million fibres /g of dried lung (note: 1 million fibres /g of dried lung is usually taken as evidence of an occupational exposure) showed that this must have been the case. Using estimates of clearance based on Wittenoom crocidolite cases suggests that the initial exposure during this period was ~540 million fibres /g of dried lung assuming a 7 year clearance half-life. The second mesothelioma case was from a machine mechanic who worked mostly in the room where the filters were attached to the cigarettes. His exposure lasted from 1951 – 83 and his burden was 3 million fibres dry /g. Nearly all fibres found were high magnesium crocidolite typical of the Bolivian crocidolite used specifically by the Massachusetts cigarette filter manufacture. The results therefore suggest that the estimates of the exposure were approximately correct but were estimating what would be counted at present.

**UK gas mask workers**

No air concentration measurements were available from the facilities in Leyland, Nottingham, Blackburn and Birmingham, which were operating under difficult condition before and during the second world war. Military gas masks used a mixture of crocidolite and chrysotile and incidences of mesothelioma were found in the workforce. The only exposure data comes from lung analysis of mesothelioma victims and therefore represents a selective group of diseased workers. The only use that this data may have is that the factories used Wittenoom crocidolite and the lung burden study carried out by de Klerk to bolster the very limited airborne fibre data from Wittenoom may allow a broad order of magnitude estimate to be attempted.

The concentrations of fibres in the lungs of the gas-mask factory workers as counted by PCM ranged from 0-07 to 216 million fibres / dry gram (mfpdg). Among the Nottingham workers,
the one with the longest recorded exposure had the greatest fibre concentration, but there was no clear correlation between concentration and duration of exposure for the remainder. Workers who were not employed on the assembly line still had significant fibre concentrations. The geometric mean fibre concentration for the Leyland workers (8.6 mfpdg) was about twice that for the Nottingham workers (4.6 mfpdg). This is consistent with the report that only at the latter factory were effective precautions taken to control dust levels, but the average duration of exposure may have been longer for the Leyland workers so that these figures cannot be used to draw conclusions regarding the relative magnitude of airborne dust levels. Blackburn factory workers had lower lung burdens (0.5 – 6.5 mfpdg).

Concentrations of crocidolite fibres in the lungs of two of the Wittenoom miners approached 1000 mfpdg. The high concentrations observed are consistent with the high incidence of asbestosis reported among the Wittenoom miners (McNulty) but in contrast only three of the gas-mask factory workers had asbestosis as a complicating factor and they were among those with the highest fibre counts. Jones et al, using the electron microscope, have also measured the concentrations of various types of asbestos in the lungs of 22 Nottingham gas-mask factory workers and found that crocidolite fibres predominated with a median of 40 mfpdg, which is equivalent to around 5 mfpdg by PCM analysis.

**Crocidolite Summary**

Table 4 summarises the crocidolite exposure of various cohorts. Overall the average cumulative exposure at Wittenoom seems low, given the airborne concentrations and conditions. However, this reflects the very short time many people worked at the site and is not dissimilar from the white South African experience. The very limited information from the small Massachusetts cigarette filter cohort does not allow any real understanding of the risk from crocidolite use in textiles and asbestos cement, which produce vastly different outcomes for chrysotile. Yet there are a number of cohorts as pointed out in H&D where some epidemiological information exists. Given the increased disease rates in these crocidolite cohorts and there would be some merit in trying to assess the best estimates of likely concentrations from lung burden or other data (semi-quantitative estimates) to get further confirmation of the high risks associated with crocidolite. At the present time all that can be added is the Wittenoom environmental/para-occupational exposure cohort, whose exposure is also poorly described by limited number of airborne measurements and no measurements of the early exposures when the mine was active from which all but one of the “environmental” mesotheliomas have been derived. Other mixed cohorts (e.g. gas masks and asbestos cement manufacture) may yet provide further useful data for better defining the risk from mesothelioma.
### Table 4: Summary of crocidolite cohort exposure and mesothelioma rate

<table>
<thead>
<tr>
<th>Facility and dates of operation with asbestos</th>
<th>Average airborne concentration (f/ml)</th>
<th>Average cumulative exposure from H &amp; D (f.years/ml)</th>
<th>H&amp;D Mesothelioma risk. (Number of mesotheliomas / cohort size.)</th>
<th>Change due to current fibre counting method.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wittenoom Workers 1943 - 66</td>
<td>Mines 15-40 and mills 150-250</td>
<td>23*</td>
<td>0.48</td>
<td>Assumed 1:1 for TP:PCM but x2-2.5 higher</td>
</tr>
<tr>
<td></td>
<td>No PCMs but 28 LTPTs in 1966.</td>
<td></td>
<td>(222/7000)</td>
<td></td>
</tr>
<tr>
<td>-South African mines and mills 1893 - 1992</td>
<td>1945 mills 30-160 and mines 2-60.</td>
<td>16.4*</td>
<td>0.59</td>
<td>X2 – 2.5</td>
</tr>
<tr>
<td></td>
<td>1960 mills 10-50 and mines 2-6</td>
<td>9.6</td>
<td>(20 / 3430)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1970 mills 8-30 and mines 2-6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PCMs from 1975.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wittenoom domestic / environmental 1943-1993</td>
<td>1943-57 1 1958–66 0.5 1966–94 0.1</td>
<td>Not given</td>
<td>Not given</td>
<td>Not relevant to high early exposures when mine open.</td>
</tr>
<tr>
<td></td>
<td>PCMs from 1973–92</td>
<td>5.4 mean h 98 15.2 median</td>
<td>(27 / 4659)</td>
<td></td>
</tr>
<tr>
<td>Massachusetts</td>
<td>No PCM’s estimated at 60</td>
<td>120*</td>
<td>0.68</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(5 /55)</td>
<td></td>
</tr>
</tbody>
</table>

* From H&D
**Amosite**

**South African Amosite Mining**

South Africa was the sole commercial source of grunerite (Amosite) asbestos with the Penge, Weltevrede and Kromellemboog mines in (Transvaal) Limpopo region being the main source of production along with some smaller mills operated in the near vicinity. The ease of mining (only Penge mine had underground shafts) meant that amosite was the main fibre produced from South Africa up to 1960.

However any epidemiological investigations are hampered by the fact that crocidolite asbestos was also present in this region and at some places occurred alongside or interwoven with the amosite deposits. Also many of the workers at the mines were transient, staff turnover approach 100% per annum among Penge miners. Due to this, white miners are the only group studied; yet in 1963 Penge had 360 white and 6500 black workers (McCulloch, 2002).

In addition to the two mills at Penge mine, there were many small mills in the Penge area. They were of basic design and caused extensive environmental pollution in the valleys (McCulloch, 2002). The workers themselves were exposed to extremely high fibre levels, as sweepers, sorters and packers. Cobbing, whereby the fibre was loosened from the rock, was done primarily by women and children at Penge, as well as at the smaller operations. In 1940, 25% of the Penge workforce were boys younger than 16 (McCulloch, 2002). The employment of children younger than 16 years of age was only prohibited in 1973 and hand cobbing was reported to have continued to this date.

Despite the limited number of exposure measurements (see figure 1 table 2) anecdotal evidence and the amount of non-malignant lung disease clearly indicates that the levels of asbestos fibres in and around the Penge mining area were high (Murray and Nelson, 2007). Davies et al. (2001) studied the prevalence of asbestos disease in 770 women who had worked on asbestos mines in the Pietersburg asbestos field from 1929 to 1980; 80% were cobbers. A diagnosis of pleural and/or parenchymal asbestosis was made in 96% of cases. They ascribed the high prevalence to exposure to high levels of amphibole asbestos dust, predominantly amosite. Widespread environmental contamination has been described around the mines and downstream. In 1988, some 12 years after the closure Felix (1997) reported environmental concentrations, with children in the area having the highest exposures (mean 0.02 f/ml; range 0.002–0.090 f/ml).

In 1947, workplace static sampler measurements in the Penge mills ranged from 162 to 720 p/ml, and underground, from 80 to 228 p/ml: levels appeared to increase in the 1950s, probably due to mechanisation, when 30% of the counts in the mills were above 780 p/ml (Sluis-Cremer, 1965). Rendall (1971) using personal samplers, recorded counts of up to 327 f/ml in the mills and more than 100 f/ml in a range of other surface jobs: underground levels were <5 f/ml. In the 1980s the fibre counts at Penge ranged from 0.1 to 6.5 f/ml in the mills, and from 0.2 to 1.0 f/ml underground (Murray and Nelson, 2007).

Although, higher incidences of non-malignant pleural abnormalities (Irwig et al. 1979) have been found in amosite workers compared to crocidolite workers with more than 15 years of service (34% v 17%): case control studies (Rees et al., 1999) for mesothelioma the higher risk from crocidolite are apparent with only three mesotheliomas from amosite workers and of the 22 environmental mesotheliomas, only two (9%) were attributable to amosite exposure. This trend is further confirmed by Murray and Nelson (2007) where in fifty recent case of mesothelioma only one (2%) was attributable to amosite exposure.
Amosite Manufacturing

There are three main cohorts that have been described: Patterson, Tyler and Uxbridge which used mainly amosite asbestos (Uxbridge also used chrysotile) to manufacture insulation products: they have recently been reviewed by Ribak and Ribak, (2008). There is no record of measurements from Patterson and the other facilities have no airborne measurement data of their early operations. However, by using the epidemiological data it was hoped that some further insight to the likely exposures could be gained. The epidemiological data is summarised in table 5. Other than an update from Ribak in 1989, for the Patterson cohort where some 740 of the cohort have now died, the mesothelioma deaths have remained unchanged and represent 2.3 % of the total mortalities. As no follow up of the cohorts has taken place for some 20 years, there is a considerable knowledge gap on the outcomes of the amosite manufacturing cohorts.

Table 5: Mortality outcomes from the amosite manufacturing cohorts

<table>
<thead>
<tr>
<th>Cohort and last main update</th>
<th>Number in cohort</th>
<th>Number of deaths</th>
<th>Number and (%) of lung cancers</th>
<th>Number and (%) of mesotheliomas</th>
<th>Number and (%) of asbestosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patterson Siedman, 1986</td>
<td>820</td>
<td>593</td>
<td>111 (18.7)</td>
<td>17 (2.9)</td>
<td>31 (5.2)</td>
</tr>
<tr>
<td>Tyler Levin, 1988</td>
<td>1130</td>
<td>222</td>
<td>35 (15.8)</td>
<td>6 (2.7)</td>
<td>3 (1.4)</td>
</tr>
<tr>
<td>Uxbridge Acheson, 1984</td>
<td>4820</td>
<td>333</td>
<td>57 (17.1)</td>
<td>5 (1.5)</td>
<td>9 (2.7)</td>
</tr>
</tbody>
</table>

Patterson, New Jersey

The first study to establish the carcinogenicity of amosite was conducted on a cohort of 820 workers, from a plant in Paterson, NJ. The plant operated from 1941 –54 has been reported to have utilized almost exclusively amosite asbestos. The exposure period for the cohort was limited to the workers who started work between June 1941 and December 1945 (encompassing the entire period the United States was engaged in World War II). The work force was made up almost entirely of white males, who were older than would be usual for such a work force, as the younger men were in the armed forces. Only workers hired up to 1945 were included in the cohort, so by 1975 the latency period had been a minimum of 30 years. Workers in the cohort who had asbestos exposure besides the Paterson factory were omitted, so the health hazard would relate specifically to the amosite exposures in the Paterson, NJ factory. Every death in the cohort was traced, and relevant clinical and pathological information was sought (Selikoff et al., 1972, Seidman et al., 1979, Seidman et al., 1986, Selikoff and Seidman, 1980, and Ribak et al., 1989).

In the latest update (where 740 deaths had occurred in the Paterson cohort of 820 men), cases of mesothelioma were diagnosed: eight were pleural and nine peritoneal mesotheliomas. The fibre concentrations of airborne asbestos have been estimated from measurement conducted some 25 years later in another factory using some of the same machinery (Tyler). A range of between 14 and 75 f/ml was estimated by Levin et al., (1998), with an average exposure of 50 f/ml estimated by Seidman et al., (1986). For a risk assessment, Nolan et al. (1999) used 30 f/ml, as a lower limit for the average exposure.
Tyler, Texas

The Paterson factory closed in 1954 and moved to Tyler, Texas where a cohort of 1130 workers fabricated grunerite of asbestos (amosite) containing products until February 1972 when it was closed after failing to meet occupational hygiene controls (Levin et al., 1998). Exposures at the factory are thought to be similar to those of Paterson as both facilities were operated by the same company and used, in some cases, the very same machinery and processes. Unlike Paterson, where no airborne asbestos concentrations were ever recorded, in Tyler 170 personal air samples were collected and analysed by Massachusetts than 70% of the air samples collected in 1971). The concentration range was reported to be from 15.9 to 91.4 f/ml (Levin et al., 1998). More detailed exposure data specific to various tasks the workers performed are available for 1967, 1970, and 1971, and are shown in table 6 (see Hurst et al, 1979 and Johnson et al., 1982). Since these air samples were taken in the last phase of operation at the plant where it was carrying out modifications to meet the new industrial hygiene limits (12 f/ml) the figures in table 6 represent the lowest value achievable and the mean airborne concentrations over the previous 13 years would have been significantly higher.

Although the airborne concentrations are high there were three asbestosis deaths (representing 1.4% of the total mortality) from the 18 years it operated, there was some 3.7-fold less asbestosis mortality than in the Paterson cohort which had a maximum exposure duration of 13 years.

Table 6: Results of personal air sampling for five different functions at the grunerite asbestos (amosite) factory in Tyler, Texas.

<table>
<thead>
<tr>
<th>Operation</th>
<th>PCM air concentrations f/ml</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1967</td>
</tr>
<tr>
<td>Milling/fiberising</td>
<td>163.5</td>
</tr>
<tr>
<td>Forming</td>
<td>33.3</td>
</tr>
<tr>
<td>Curing</td>
<td>2.5</td>
</tr>
<tr>
<td>Finishing</td>
<td>44.6</td>
</tr>
<tr>
<td>Packing</td>
<td>16.7</td>
</tr>
</tbody>
</table>

In the latest update (where 740 deaths had occurred in the Paterson cohort of 820 men), cases of mesothelioma were diagnosed: eight were pleural and 9 peritoneal mesotheliomas.
Uxbridge

Amosite use at the factory began in 1947 and ended when the factory closed in 1979. Between 1946-1973 chrysotile asbestos was also used but generally constituted <3% of the board material being manufactured compared to ~20% Amosite. Exposures prior to 1964 were thought to be as high as 100 f/ml, particularly on the “beater floor” where the raw asbestos was unpacked and opened. This process was removed to a new building and enclosed in 1966, which resulted in significant dust reduction. Four of the five mesothelioma cases were exposed to the dustiest operations such as milling or fiberising of asbestos before 1960.

A cohort of 4820 men were known to have been involved in the manufacture of asbestos insulation board but only 14% of these started work prior to 1960. The other 1689 workers made non-asbestos building materials in the same building complex. In a later follow up no additional mesotheliomas were reported besides the five reported earlier (Acheson et al., 1981). One of these mesotheliomas was arguably due to prior exposure to crocidolite elsewhere (Gibbs et al., 1994).

A 2-fold excess of lung cancer was report with 57 observed while only 29 were expected with increased lung cancer mortality for those starting work both before and after 1960. The asbestos exposed cohort also had 9 asbestosis deaths an intermediate number between Paterson and Tyler (Table 5). All of the nine workers with the asbestosis had started work prior to the dust reduction in 1964, and seven of them started before 1960 when the cohorts of asbestos workers and non-asbestos workers were 654 and 236 respectively. Most of the work force were employed and only exposed over a short period from 1965, with 67.6% exposed for < 1 year 38% for < 3 months.

PCM air measurements were made from 1969 after the improvements had been made, and levels of around 30 f/ml were reported (Acheson, 1994). Evidence provided to the Advisory Committee on asbestos showed that for the first half of 1976 the average fibre concentration was 1.26 f/ml with 10.1 % between 2 - 4 f/ml and 1.3% above 4 f/ml. There were also 11 respirator areas in the 90 stations monitored, showing that meeting the 2 f/ml limit was difficult.

The early exposure estimates of Acheson et al. (1984) for opening and fibrising are similar to the average of 91.4 f/ml reported by Hurst et al. (1979) for milling/fiberising at the Tyler, factory. In Uxbridge, employment histories of the mesothelioma cases were reported, and three of the five mesotheliomas occurred among individuals whose job included milling/fiberising. This is also the case for the two workers who developed asbestosis and either pleural or peritoneal mesothelioma, after working for 8 years and 6 years, respectively, at milling/fiberising. Of the three pleural mesotheliomas with less than 4 years’ exposure, two worked in milling/fiberising and the third case with 4 months of exposure had both asbestosis and a pleural mesothelioma indicating a significant cumulative exposure during his brief period in the factory.

Gibbs et al., 1994 reported the lung burdens from 43 autopsy cases from Cape Uxbridge. Amosite was the predominant fibre found with a mean of 784.9 mfpdg with two subjects having no exposure and the highest being 7852.9 mfpdg. These are high exposures and show that high airborne fibre concentrations existed in the factory. Crocidolite the next most common fibre with a mean of 22 mfpdg with 19 subjects with no exposure and the highest 72.7 mfpdg. The chrysotile mean was 12.1 mfpdg with 14 subjects with no exposure and the highest being 24.3 mfpdg. Cases of lung cancer and mesothelioma had by far the highest lung burdens, 1434 and 1001 mfpdg and accounted for 14 and 5 of the deaths respectively. Other cancers have 24 associated deaths but lung burdens were an order of magnitude lower (297 mfpdg). There were strong correlations between the lung burden and the grade of asbestosis.
Amosite general

All three factories are similar in that they produced high airborne fibre exposures during their early use, but the Uxbridge factory was the most successful in reducing airborne concentrations significantly after 1966. Overall, the cancer mortality rates between the three manufacturing cohorts are similar but the asbestosis rate is significantly different: with the Uxbridge cohort having double the rate, and Patterson quadruple the rate associated with the Tyler cohort. If these were indicative of the exposures this would suggest that Tyler cohort had the lowest exposures and the Patterson cohort the highest.

Table 7: Summary of amosite cohorts exposure and mesothelioma risk

<table>
<thead>
<tr>
<th>Facility and dates of operation with asbestos</th>
<th>Average airborne concentration (f/ml)</th>
<th>Average cumulative exposure from H &amp; D* or this estimate (f.years/ml)</th>
<th>H&amp;D Mesothelioma risk. (Number of mesos / cohort size.)</th>
<th>Change due to current fibre counting method. (see appendix 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SA mines and mills 1914-92</td>
<td>1945 mills 150 and mines 14. 1960 mills 56 and mines 6 1970 mills 40 and mines 4</td>
<td>23.6* 15.2 (Note: 41% had &gt;1 and &lt;10% had &gt;30.)</td>
<td>0.06 (4/3212)</td>
<td>X1.5 -2</td>
</tr>
<tr>
<td>Patterson 1941-54</td>
<td>50 No measurements were made.</td>
<td>65 *</td>
<td>0.12 (17/820)</td>
<td>X1.5 -2</td>
</tr>
<tr>
<td>Tyler 1954 - 72</td>
<td>(1954 – 66) 60 (1966-72) 40 170 PCMs 1967-71</td>
<td>50 35</td>
<td>Not done (6/753**)</td>
<td>X1.5 -2</td>
</tr>
<tr>
<td>Uxbridge 1946-79</td>
<td>(1947-65) 60 (1965-78) 2 ~100 PCMs 1969 &amp;1976</td>
<td>50 1</td>
<td>Not done (4/654 early) (0/4166 late)</td>
<td></td>
</tr>
</tbody>
</table>

* From H&D. ** 753 with records and >10 years from first employment
Chrysotile mining and milling

The Quebec and Balangero mining and milling cohorts are the two quantitative studies reviewed by H&D but these rely on measurement records from 1948 onwards for Quebec and from 1969 for Balangero, so the historic exposure in these cohorts rely on reconstruction estimates. Both mining areas have fibrous amphibole impurities (~0.5 - 1%), which are often associated with the relatively small rates of mesotheliomas (Yarborough, 2006). However, these are estimated by H&D at 0.0009 % and 0.0025 %, respectively, in terms of the total mortality per f.year/ml. The Quebec estimate also appears to apply to “non-occupationally” exposed residents in the mining areas, as well as the workers (Camus et al., 1998).

Lung cancers in Quebec were raised SMR=1.21 but the general conclusion was that cumulative exposures of <1000 f.years/ml were innocuous (Liddell et al., 1997) and would never be reached under current practices, which were estimated to be several orders of magnitude lower than the historic conditions. The lung cancer rates at Balangero were not raised. The comparison of chrysotile mining and milling to chrysotile textile manufacturing is difficult in that textile manufacture has a 50 times higher dose response slope for lung cancer. Overall the evidence is for a much lower rate of lung cancer and mesothelioma than for amosite and crocidolite.

Airborne concentrations

These Quebec concentrations rely on impinger measurements made since 1948 and fell from an average at about 75 mppcf to about 10 mppcf in 1968 when the PCM conversions were made (equivalent to 30 f/ml, Gibbs and LaChance, 1972). This conversion was based on highly scattered data on log v log graphs and has severe limitations in its ability to predict a conversion factor. Therefore the historic exposure in 1948 of 225 f/ml can be seen as highly debatable and Balangero exposures based on PCM measurements of reconstructions of the conditions, give values between five to ten times lower (Rubino et al., 1979) for the pre 1950 period and about half as low in the late 1960s. Therefore given the uncertainties in the conversion for the Quebec data a significant part of the difference disappears if the Balangero reconstructions are taken as more representative of the historic airborne concentrations of the chrysotile miners and millers. This also overcomes the issues raised as how the Canadian chrysotile mines and mills were so much more dusty than the South African crocidolite mines and mills (and to a lesser extent the Australian crocidolite) given the much dry climate, the much greater propensity for crocidolite and amosite fibres to become airborne and their greater visibility under the microscope.

Chrysotile mining environmental

Although not in the H&D analysis the exposures of women residents in three main Quebec mining towns have been estimated using a variety of methods (e.g. annual tonnage, residential questionnaires, various air monitoring data and an expert panel.) In brief the cumulative exposure in f/ml years were estimated as shown in table 8.

The only fibre concentration data available was from annual measurements in the three major asbestos towns by the asbestos companies from 1974 and two TEM surveys by Sebastien et al. at various locations. Environmental levels were also assessed on the lung burdens of 22 residents and the para-occupational household exposure on the lung burdens of 10 residents who lived with asbestos workers.
Table 8: Summary of Quebec environmental exposures

<table>
<thead>
<tr>
<th>Type of exposure 1970 - 1989</th>
<th>Cumulative* exposure in f.years / ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neighbourhood</td>
<td>16</td>
</tr>
<tr>
<td>Household</td>
<td>7.8</td>
</tr>
<tr>
<td>Occupational</td>
<td>1.2</td>
</tr>
<tr>
<td>Total</td>
<td>25</td>
</tr>
<tr>
<td>Plausible range</td>
<td>5 – 125</td>
</tr>
</tbody>
</table>

* Exposures adjusted for round the clock exposure by a factor of x 4.2

The high reliance on static sampling means that the personal exposures were likely to be higher (see Wittenoom environmental estimates) but no very little personal exposure sampling appears to have been carried out (values of 0.1 – 6 f/ml reported by Gibbs et al. 1980).

Chrysotile textiles

Textile exposures produce high rates of lung cancer than other manufacturing uses of chrysotile so represent a worst-case estimate for the risks from chrysotile. A general overview of best practice in the UK chrysotile textile industry is summarised in table 9 from various sources. Obviously not all measurements met the best practice and problem areas such as opening and fibrising and carding often considerably exceeded these levels, relying on the use (or misuse) of respirators and exhaust ventilation to reduce exposures.

Table 9: Overview of UK best practice that should have been achieved with controls in terms of modern PCM measurements.

<table>
<thead>
<tr>
<th>Approximate date of measurement</th>
<th>Process</th>
<th>Modern PCM TWA airborne concentration (f/ml)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1930’s</td>
<td>Spinning and doubling</td>
<td>20 –50</td>
<td>Burdett, 1998</td>
</tr>
<tr>
<td>1960s</td>
<td>Spinning and weaving</td>
<td>5 – 25*</td>
<td>Ogden, 2003</td>
</tr>
<tr>
<td>1976</td>
<td>All with controls</td>
<td>&lt;2</td>
<td>HSE (1976)</td>
</tr>
<tr>
<td>1983</td>
<td>All with controls</td>
<td>&lt;1</td>
<td>CEC, 1983</td>
</tr>
<tr>
<td>2006</td>
<td>All with controls</td>
<td>&lt;0.1</td>
<td>EU, 2003, CAR, 2006</td>
</tr>
</tbody>
</table>

* Originally quoted as 2 –10 f/ml adjusted for early PCM counting methods

South Carolina textiles

South Carolina is exceptional in that the exposure response for lung cancer is some 50 times greater than observed in Canadian miners and millers who supplied the vast bulk of the chrysotile fibre to the factory. The reason for these differences has been the subject of intense scrutiny and debate.
The latest update cohort of 3072 workers exposed to chrysotile in a South Carolina asbestos textile plant (1916-77) has been followed up for mortality through 2001 (Hein et al., 2007). This facilities gives the highest asbestos related disease for a relatively chrysotile only exposure. Standardised mortality ratios (SMRs) were computed using US and South Carolina mortality rates. A job exposure matrix provided calendar time dependent estimates of chrysotile exposure concentrations. Poisson regression models were fitted for lung cancer and asbestosis. Covariates considered included sex, race, age, calendar time, birth cohort and time since first exposure. Cumulative exposure lags of 5 and 10 years were considered by disregarding exposure in the most recent 5 and 10 years, respectively.

A majority of the cohort was deceased (64%) and 702 of the 1961 deaths occurred since the previous update. Mortality was elevated based on US referent rates for a priori causes of interest for lung cancer (SMR 1.95, 95% CI 1.68 to 2.24 and pneumoconiosis and other respiratory diseases (SMR 4.81, 95% CI 3.84 to 5.94). Mortality remained elevated for these causes when South Carolina referent rates were used. Three cases of mesothelioma were observed among cohort members.

Exposure-response modelling for lung cancer, using a linear relative risk model, produced a slope coefficient of 0.0198 (fibre-years/ml) (standard error 0.00496), when cumulative exposure was lagged 10 years. Poisson regression modelling confirmed significant positive relations between estimated chrysotile exposure and lung cancer and asbestosis mortality observed in previous updates of this cohort. It was reported previously there was no significant evidence for a threshold in models of either the lung cancer or asbestosis. The excess lifetime risk for white men exposed for 45 years at the recently revised OSHA standard of 0.1 fibre/ml was predicted to be about 5/1000 for lung cancer, and 2/1000 for asbestosis.

**Airborne concentrations**

A detailed study of plant processes and dust control methods over the period 1930-1975 was conducted in an asbestos textile plant processing chrysotile. Linear statistical models for reconstructing historic dust exposure levels, taking into account textile processes, dust control measures, and job assignments, were developed (Dement et al., 1993). Parameters of these statistical models were estimated using 5,952 industrial hygiene sampling measurements covering the period 1930-1975. For most textile operations, exposure levels were significantly reduced by about 1940, when most engineering dust control measures were in place. Results of the exposure estimates indicated "precontrol" exposure levels to range from 3 to 78 f/ml with typical levels well above 10 f/ml. After textile operations were provided with dust control measures, estimated exposure levels ranged from 3 to 17 f/ml and were usually in the range of 5 to 10 f/ml. The Job exposure matrix was broken down into 10 areas and table 10 gives an example for the fibre preparation zone.

Air concentrations were monitored from 1930–39 on five occasions by the Metropolitan Life insurance company using an impinger, with results in terms of particle counts (mppcf) visible at x100 magnification at the bottom of a sedimentation cell. The company also monitored on an infrequent basis from the 1930’s and routinely from 1956. The US public health service also monitored in 1968 and 1971 using both PCM and impingers. Therefore the conversion from impinger measurements to membrane filters is crucial and was based on the PCM method described by Edwards and Lynch, 1965.

Mc Donald et al. 1983 considered various conversion factors for the impinger to PCM conversions and reported levels of between 1.3 – 10 f/ml = 1 mppcf with an average of about 6 f/ml for 1 mppcf. This is close to the ratio (5.9) reported by the US PHS studies (Ayer et al, 1965) and twice the ratio used by Dement et al., 1994 (3) except for the zone 1 where 8 was used.
Although McDonald et al., (1983) felt the impinger results were extraordinarily low compared to the Canadian mines and mills, they found the levels comparable or lower than with other textile plants in the US (e.g. Connecticut) and in the UK they had used a conversion factor of 3.64 and another independent study of the mines and mills had used about 3 (Nicholson et al., 1971).

The use of the higher conversion factor for zone 1 is puzzling as the fibre is still being separated from the non-fibrous particles, so it would be expected that the particle fibre conversion ratios are lower rather than higher.

Table 10: Estimated historic exposures in the fibre preparation area (Zone 1).

<table>
<thead>
<tr>
<th>Uniform Job Category</th>
<th>Mean PCM Fiber Concentration (Fibers &gt; 5 μm/cc) and 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1930-1944</td>
</tr>
<tr>
<td>A: General Area Personnel</td>
<td>26.2 (12.1-40.2)</td>
</tr>
<tr>
<td>B: Machine Operators</td>
<td></td>
</tr>
<tr>
<td>Fly machine operator</td>
<td>78.0 (38.1-117.8)</td>
</tr>
<tr>
<td>Crusher operator</td>
<td>78.0 (38.1-117.8)</td>
</tr>
<tr>
<td>Waste machine operator</td>
<td>45.9 (21.0-70.8)</td>
</tr>
<tr>
<td>C: Cleanup Personnel</td>
<td>54.4 (10.7-98.1)</td>
</tr>
<tr>
<td>D: Raw fiber handling</td>
<td>35.0 (17.0-53.0)</td>
</tr>
</tbody>
</table>

Another area where some of the differences in the estimates of exposure may arise are the exclusion of several very dusty practices which took place as “overtime” activities and were not included in the air monitoring. The exhaust ventilation system from the preparation area and the carding department during 1937 –1953 was based on filtering the air through burlap bags (the bags in which the asbestos was delivered from the mines) stretched across a wooden frame. These were beaten with buggy whips on a daily basis to dislodge the layer of fibre collected and then shovelled up at the end of the week into containers and returned for reuse in the preparation area. No mention is made of where the exhaust air from the crude filters was vented to air or back into the factory.

During 1954 – 64 the mixing of fibres in the preparation department was transferred to a mezzanine floor where the asbestos was opened using pitchforks with no dust suppression. This meant that some people were exposed to extremely high exposures (500+ f/ml) that went unrecorded and have not been included in the analysis. Mc Donald et al., 1983 felt that these high exposure interludes were unlikely to exceed a two fold underestimate of the cumulative exposure estimates and overall a factor of 10 difference would still exist in the dose response relationship for lung cancer between the manufacturing and Quebec mining and milling operations.

**Chrysotile Summary**

As explained in the introduction the wide variation between the mines and mills and the South Carolina textiles represent the two extremes in the lung cancer dose-response relationships (about a factor of 50). Small numbers of mesotheliomas have been reported in the chrysotile mining and textile manufacture but the influence of the small amphibole components cannot be entirely ruled out. There is some evidence that the Quebec cohort conversions from impinger measurements to fibre concentration may be too high, while for South Carolina is too low. This can bring the dose-response relationships closer but is unlikely to account for the factor of 50. Variations in the percentage of long fibres may be another area to explain the differences and the recent TEM studies should answer this shortly. Overall there is enough
uncertainty in the measurements and their conversions to put the chrysotile risk estimates between the Quebec and South Carolina dose–response relationships.

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26


Appendix 1: TEM Fibre size distribution at mines and mills

Gibbs and Hwang (1981) reported on surveys of the airborne fibre size distributions at the South African and Canadian mines. They used TEM micrographs of x 15,000 to size the fibres but due to the small size of the TEM grid openings (45 µm) and the micrograph magnification, the maximum length of fibre that could be measured was truncated. Also as all fibre sizes were calculated, the numbers of >10 µm long fibres counted were limited so do not have good precision. However, using their published data for >5µm and >10 µm long fibres, it can be seen in table 1 that there are substantial differences between fibre types in what will be counted by phase contrast microscopy.

In the first instance the same limit of visibility (> 0.2µm width) has been applied to the mining data for three main commercial asbestos types. Table A1 shows that due to the finer fibres present only about a quarter of the >5 µm long crocidolite fibres and half of the chrysotile fibres will be visible to a well adjusted PCM, compared to the amosite fibres. Therefore a PCM count of crocidolite will underestimate the numbers of long fibres the person was exposed to. In a direct comparison on different areas of the same filter a direct comparison with the PCM, estimated that ~34% of the TEM >5 µm long crocidolite fibres were counted.

If we follow current trends that the longer fibres (e.g. >10 or >40 µm long) are the most carcinogenic, the percentage of > 10 µm long fibres in mine bagging samples quickly reduces to ~1% of the total numbers of fibres except for amosite (see table 1). This reduction in fibres >5 µm long to fibres >10 µm long is a factor of 8, 3.3 and 4 for crocidolite, amosite and chrysotile respectively. For >40 um long fibres the TEM data cannot be used but the light microscope data showed that only 2% of the >5 um long crocidolite fibres were >40 um long.

Table A1: Summary of fibre size distribution data from asbestos mines and mills (Hwang and Gibbs, 1981).

<table>
<thead>
<tr>
<th></th>
<th>Crocidolite</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% &gt;5</td>
<td>% &gt;10</td>
<td>% &gt;5</td>
<td>% &gt;10</td>
<td>% &gt;5</td>
<td>% &gt;10</td>
</tr>
<tr>
<td>Length (µm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All dia.</td>
<td>7.1</td>
<td>0.89</td>
<td>24.5</td>
<td>7.5</td>
<td>4.12</td>
<td>1.04</td>
</tr>
<tr>
<td>Dia &gt;0.2</td>
<td>1.85</td>
<td>0.22</td>
<td>20.89</td>
<td>6.8</td>
<td>2.01</td>
<td>0.55</td>
</tr>
<tr>
<td>Ratio All/&gt;0.2</td>
<td>3.84</td>
<td>4.05</td>
<td>1.17</td>
<td>1.10</td>
<td>2.05</td>
<td>1.89</td>
</tr>
<tr>
<td>Percentage of</td>
<td>26</td>
<td></td>
<td>85</td>
<td></td>
<td>49</td>
<td></td>
</tr>
</tbody>
</table>

Substantial differences between the mining and bagging were found in terms of the >5 µm long fibres as a percentage of the TEM fibre size distribution (see table A2). The relative increase in the percentage of long fibres with increasing processing has
been suggested as the reason for the observation of greater carcinogenicity in manufacturing cohorts than with some mining cohorts and a meta-analysis has confirmed this (Lash et al., 1997).

**Table A2: Percentage of > 5 µm long fibres in the TEM size distributions at mines and Mills (Hwang and Gibbs, 1981).**

<table>
<thead>
<tr>
<th></th>
<th>Crocidolite</th>
<th>Amosite</th>
<th>Chrysotile</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mining</td>
<td>Bagging</td>
<td>Mining</td>
</tr>
<tr>
<td>% of &gt; 5 µm long fibres</td>
<td>4.1</td>
<td>7.1</td>
<td>12.4</td>
</tr>
</tbody>
</table>

**TEM size distributions in Factories**

At the present time it is very difficult to find any historic studies using TEM analysis of airborne concentrations of crocidolite in manufacturing. This is mainly due to crocidolite use being phased out as membrane filter sampling was starting to be used. This is also the case for amosite and most of the TEM data in the literature relates to chrysotile, which continues to be used. Table A3 shows the results from a chrysotile textiles factory (Rochdale) which shows that the initial process (e.g. carding) have a coarse diameter distribution. Overall, under half of the of >5 µm long fibres are <0.2 µm and are unlikely to be visible by PCM. Increased microscope performance will alter the minimum fibre visibility from >0.4 µm to >0.2 µm wide will increase the count by a factor of two.

**Table A3: Examples of the effect of fibre visibility on counts of >5 µm long fibres from HSE data from monitoring different processes at a chrysotile textile factory.**

<table>
<thead>
<tr>
<th>Process</th>
<th>Percentage of &gt;5 µm long fibres with diameters &gt; (µm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.1</td>
</tr>
<tr>
<td>Carding</td>
<td>77</td>
</tr>
<tr>
<td>Spinning</td>
<td>69</td>
</tr>
<tr>
<td>Weaving</td>
<td>78</td>
</tr>
</tbody>
</table>

Dement (2007) has recently analysed by TEM 84 archived membrane filters collected between 1964 – 68 at the chrysotile textile manufacturer at Charleston S. Carolina. Based on a stratified count of 18,824 fibres and fibre bundles only ~ 6.5% were PCME (>5 length and >0.25 diameter) and only 1% of fibres of any diameters exceeded 40 µm length (which according to Berman and Crump contains 97% of the carcinogenicity). The fibre counts were divided into 10 production processes. Between 4.5 – 11.4% of the fibres in different zones were > 5µm long but too thin to be visible by PCM. The main purpose of the analysis was to use the TEM size distribution to apply adjustment factors to the biologically active fibres as defined by size parameters (e.g. Stanton fibres and >40 µm long fibres etc.). The results for these have been extracted from the data and are summarised in Table A5. It can be seen that
that >40 µm long fibres represented only 1% of TEM size distribution and only half of these would be counted by PCM.

Table A4: Results from TEM analysis of eighty four filters collected at South Carolina chrysotile textile factory for general area personnel in 1965 (Dement et al, 2007) and calculated adjustment factors.

<table>
<thead>
<tr>
<th>Exposure Zone</th>
<th>Plant Department Associated with Exposure Zone</th>
<th>Mean PCM Exposure (fibers &gt; 5 µm/cc)</th>
<th>PCM Adjustment Factor</th>
<th>Mean TEM Exposure (fibers &lt; 0.25 µm in diameter and &gt; 5 µm in length /cc)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Preparation</td>
<td>5.8</td>
<td>1.02</td>
<td>5.92</td>
</tr>
<tr>
<td>2</td>
<td>Carding</td>
<td>2.4</td>
<td>1.15</td>
<td>2.76</td>
</tr>
<tr>
<td>3</td>
<td>Ring Spinning</td>
<td>8.2</td>
<td>1.31</td>
<td>10.74</td>
</tr>
<tr>
<td>4</td>
<td>Mule Spinning</td>
<td>6.3</td>
<td>1.33</td>
<td>8.38</td>
</tr>
<tr>
<td>5</td>
<td>Foster Winding</td>
<td>4.2</td>
<td>1.32</td>
<td>5.54</td>
</tr>
<tr>
<td>6</td>
<td>Twisting</td>
<td>5.4</td>
<td>2.17</td>
<td>11.72</td>
</tr>
<tr>
<td>7</td>
<td>Universal Winding</td>
<td>4.1</td>
<td>1.13</td>
<td>4.63</td>
</tr>
<tr>
<td>8</td>
<td>Heavy Weaving</td>
<td>2.0</td>
<td>1.34</td>
<td>3.46</td>
</tr>
<tr>
<td>9</td>
<td>Light Weaving</td>
<td>2.7</td>
<td>1.69</td>
<td>4.56</td>
</tr>
<tr>
<td>10</td>
<td>Finishing Operations</td>
<td>0.2</td>
<td>2.15</td>
<td>0.43</td>
</tr>
</tbody>
</table>

Table A5 Summary of percentage of various categories of biologically active fibre sizes in the TEM analysis of eighty four filters collected at South Carolina chrysotile textile factory for general area personnel in 1965 (see Dement et al, 2007).

<table>
<thead>
<tr>
<th>Fibres lengths (µm)</th>
<th>Width (&lt;0.25)</th>
<th>&gt;0.25 - &lt;3</th>
<th>&gt;3</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;5</td>
<td>9.2</td>
<td>6.6</td>
<td>0.7</td>
</tr>
<tr>
<td>&gt;15</td>
<td>2.5</td>
<td>2.0</td>
<td>0.4</td>
</tr>
<tr>
<td>&gt;40</td>
<td>0.4</td>
<td>0.5</td>
<td>0.1</td>
</tr>
<tr>
<td>Total</td>
<td>16.5</td>
<td>4.9</td>
<td>1</td>
</tr>
</tbody>
</table>
Appendix 2: Relationships between historic fibre counts and a modern day PCM membrane filter count.

There are a number of sampling and analytical issues when looking at the relationship between the quantitative asbestos cohort data and a modern day measurements even when a membrane filter count was carried out. These include variations in:

- Sampling;
- Sample preparation;
- Microscope equipment and set up;
- Fibre definitions;
- Counting performance.

These issues have been discussed in some detail by Walton, 1982 and Rickards, 1984. This appendix briefly reviews the main sources of difference before looking at how specific studies may have under or overestimated the fibre count. Again this has been looked at in terms of asbestos fibre type as for the H&D (2000) analysis. As for all fibre counting there is a mixture of human, systematic and random errors and poor statistical precision. Although these may be reduced by developing standard procedures it ultimately depend on adherence and attention to detail.

**Sampling**

Key issues for sampling are whether personal or area (static) samples were collected. Area samples are usually between 2-10 times lower than personal samples depending on distance from the emission source and the amount of dust being raised by the person. Even if personal sampling is used, depending on which shoulder/lapel the filter is placed; a factor of two differences is common. Large fluctuations in concentrations can occur during the day so time weighted average sampling is important but is only possible in relative low dust concentrations to avoid over-sampling, unless multiple short term samples are taken and average calculated (rarely done). Flow rate will also determine the particle loading on the filter and the size of particles that can reach the filter. This became more important when cowled samplers were introduced to prevent damage to the filter (e.g. thumb prints). As with any sampling operation accurate measurement of the flow rate against a calibrated method is important and significant errors of the flow rate can occur from the use of inline or uncalibrated rotameters. Also much overlooked is the effectiveness of the seal of the filter in the holder and a percentage of filters are under sampled due to inconsistencies in the seal of the filter in the holder.

The process of unloading the filter is very important and loss of the fibrous deposit may occur due to over-sampling, careless handling, the use of fixative sprays or the use of highly static containers to hold the sampled filters for return to the laboratory. Some studies mention many filters being too overloaded to count, giving an immediate low bias to the measured airborne concentration.

**Sample preparation**

The choice of filter type and pore size will also have an influence. Membrane filters made from cellulose nitrate and/or a mixture with cellulose acetate (MCE) was the normal filter formulations used for asbestos sampling. These have a sponge like
texture where interconnecting pores or a framework gives a nominal pore size. The size of the pores will determining the filtration efficiency and how far particles penetrate into the ~160 thick filter, therefore there will be significant differences between using a 0.8 um pore size filter instead of an 8 m pore size filter as was used in some early measurements in the South African mines and mills.

The filter material has a refractive index (RI) of around 1.51 (very close to chrysotile asbestos) and appears white in air due to the air filled pores. To render it transparent for counting, a number of liquids of similar RI were used to fill the pores. In the USA this was usually a mixture dimethylpthalate and diethyl oxalate plus dissolved filter material, this became codified as the NIOSH P&CAM 239 method. In Europe and the UK “triacetin” was used and was used and codified in the DEP, 1970, ARC (1971) methods. These pore filling – semi soluble methods produced samples that were stable for only a short period as liquid and particles would migrate towards the edges of the slide and fibres would be buried deep inside the filter requiring the microscopist to rack the focal plane down into the filter to find the fibres for each field of view. More stable mounting methods which collapsed the pore structure instead of filling the pores, had the advantage of producing a permanent mount, aligning the fibres horizontally to the filter/slide in a much narrower plane. A mount with the coverslip was still needed and a thermoset resin (Euparal) or triacetin was used. The final RI of the mount is very important for chrysotile, which has RIs close to the membrane filter, hence the need for phase contrast microscopy to enhance the very low contrast between the chrysotile fibre and the filter background. Gonzalez-Fernandez and Martin (1986) reported that the filter collapsing methods gave higher mean values than the NIOSH mount by almost 2:1.

**Microscope equipment and set-up**

The type and quality of the optics, the power of the light source and how well set up the microscope is will have a large effect on the fibre count. Bright field, oil-immersion, phase contrast and dark field microscope have all been used for fibre counting with magnifications from x100 to x2000. The importance of using phase contrast microscopy for counting chrysotile fibres has already been noted and early counting comparisons showed these were particularly likely to be undercounted by inexperienced counters (e.g. a factor of 3 (Becket and Attfield, 1974)). Until a standard test slide was introduced (LeGuen et al., 1981) and widely used to set up equipment to a known performance, it was difficult to know how much the count was influenced. As discussed in appendix 1, the width distribution of asbestos fibres and in particular chrysotile and crocidolite is spread around the limit of visibility so a microscope capable of seeing 0.2 um width fibres will give a much higher fibre count (factor of 2-3) than one only seeing 0.4 um width fibres. The intensity of the light source was also important for PCM and modern halogen illumination sources offer a significant advantage over tungsten bulbs.

Given the higher concentrations being measured during the early use of the membrane filter method for asbestos analysis the use of full field counting was particularly responsible for the undercounting fibres (Becket and Attfield, 1974). Becket et al. 1976 concluded on industrial samples with small graticule grids, fibre counts were increased by a factor of about 1.5 for amosite and 2.5 for chrysotile as compared with full-field counting. The adoption of a standard (Walton-Becket) graticule for asbestos
significantly increased fibre counts compared to full field counting but many of the early counts may have been done with graticules. Gonzalez-Fernandez and Martin (1986) reported results from using different graticules were in descending order Porton: Walton-Beckett: Patterson in the approximate ratios 1.6:1.2:1

**Fibre definitions and counting performance**

Although since the advent of membrane filter -PCM fibre counting the basic definition of a fibre has been similar except for relative unimportant changes in the upper fibre width and length counted, the way bundles and split fibres were counted has varied. Also the practise of whether fibres attached to non-fibrous particles has been an important issue. Previously samples attached to particles >3 m width were not counted and only in 2006 was the counting of all fibres in the sample obligatory in the EU. This was particularly important when the asbestos was used in a matrix, which includes most commercial uses of asbestos except textiles. However even textiles often contained cotton as well and the use of subjective discrimination was commonplace as reported by Walton (1982) that, “In practice up to the present day, some laboratories in the ARC member companies and elsewhere attempt such discrimination while other do not”.

Although some subjective assessment is inevitable with human counters there were large differences between laboratories that performed the membrane filter counts and this became apparent when standard methods started to be produced and inter-laboratory counting exercises took place (Becket and Attfield, 1974). The substantial differenced in counts between different laboratories on the same slides were sometimes very large and what started as casual inter-laboratory comparisons between government and asbestos manufacturing industry laboratories spread to become proficiency testing schemes (eg RICE and AFRICA) and later expanded into full laboratory accreditation (e.g. UKAS).

**Crocidolite and Amosite PCM counts**

As discussed in the body of the paper, no membrane filter samples were ever taken of the Wittenoom miners and millers or the Massachusetts industrial cohort. Some environmental samples were taken in Wittenoom as early as 1973 but most were collected during and after 1977, over a decade after the mines and mills had closed. Although no details are available of the method used given the early date it is likely that the PCM crocidolite counts would need to be adjusted (around a factor of two higher) as for the S. African crocidolite results discussed below.

Although the South African mines were still active, the first membrane filter sampling did not appear to occur until the late 70’s and conversions to PCM f/ml were made results from samples taken in 1977 (see Du toit et al., 1983). These conversions were based on an early form of the PCM method based on pore filling and the use of a large graticule (250 x 250 µm) by one laboratory and the Patterson and Cawood graticule by the other. No test slide was available to check the performance and adjustment of the microscopes but both institutes were experienced at fibre counting. This suggests that a modern PCM count would have been higher by around a factor of 1.5 –2 for amosite and around 2 –2.5 for crocidolite based on the mounting method, the influence of the different size of the graticules and the fibre width distribution.
Other than the South African mines and mills amosite PCM counts are only available from Tyler and it appears these were carried out using an early form of the NIOSH P&CAM 239 method by the US Public Health Service (Edwards and Lynch, 1968) and later by NIOSH. A PCM magnification of X430 was used to count the pore filled membrane filters. A modern day PCM membrane filter method would be about a factor of 1.5 - 2 higher, based on the type of sample preparation used and the lack of a test slide to help check the adjustment. This takes account that amosite fibres are more readily visible by PCM than for the finer crocidolite and chrysotile fibres.

Chrysotile PCM counts

Several of the main chrysotile cohorts were in the US (South Carolina, Pennsylvania and Connecticut) and the conversion was made using the US PHS method. However, for chrysotile where the refractive index difference is small the visibility of fibres is much more dependent on the mounting method and adjustment of the phase contrast microscope. At the present time it is only possible to make guestimate as there was no test slide in use what the performance the microscopist achieved with the modern PCM method. Given the number of fine fibres present it is probable that a current analysis may count around 2.5 – 3 times more >5 um long fibres than the method used for conversion of the impinger counts.