The Public Health Significance of Asbestos Exposures from Large Scale Fires

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ABSTRACT

Large scale fires involving asbestos containing materials are a relatively common occurrence in the UK and can cause significant public concern. The potential public health consequences of such incidents have been explored. Literature reviews were undertaken to identify available information on both the level of asbestos exposures that might result from fires and the potential health impact of such exposures. Considering the available information on asbestos exposure levels in the context of the epidemiological evidence, from both occupational and environmental exposure studies, it is clear that, if appropriate clean-up procedures are followed, there is no significant public health risk resulting from asbestos exposures from large scale fires.

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EXECUTIVE SUMMARY

Asbestos is the name given to a small group of naturally occurring silicate minerals that can be readily separated into thin, strong fibres. These are divided into two sub-groups: serpentine (chrysotile), which is the most commonly used form of asbestos, and the amphiboles (amosite, tremolite, actinolite, anthophylite, and crocidolite), of which crocidolite is the most commonly used. Asbestos is defined in UK legislation as one, or a mixture of, any of the following: chrysotile (white), crocidolite (blue), amosite (brown), fibrous anthrophylite, fibrous tremolite and fibrous actinolite. Asbestos fibres are flexible, very strong and resistant to heat and chemicals. These properties have been recognised for thousands of years and asbestos was used by the ancient Egyptians, Greeks and Romans. It is only, however, in the relatively recent past that asbestos use, and therefore potential exposure, became widespread. About six million tonnes of asbestos have been imported to the UK since the late 19th Century with a peak of around 195,000 tonnes in 1973.

The importation, supply and use of asbestos was banned in 1999. However, due to its extensive use in the building industry it is still found in many products including: sprayed coatings/lagging, insulating boards, ropes, cloth, millboard, asbestos-cement sheets, coated metal, textured paints and reinforced plastics etc.

Because of its extensive use large scale fires involving asbestos containing materials (ACM) are a relatively common occurrence in the UK and can cause significant public concern. In addition, the HPA is responsible for ensuring that public health responses to such incidents are appropriate and consistent. It was therefore considered important to investigate the potential public health consequences of such incidents and explore actions that can be taken to minimise their impact.

To this end, a systematic literature review has been undertaken to identify available information on both the level of asbestos exposures that might result from fires (Section 3) and the potential health impact of such exposures (Section 4). The key elements of plans for and responses to such incidents that can minimise their impact have also been briefly explored (Section 5).

The conclusions and recommendations of this study are summarised below:

- Large scale fires involving asbestos containing materials (ACM) are a relatively common occurrence in the UK and can cause significant public concern (Section 1).
- A number of factors mitigate against significant exposures of members of the public following a fire involving ACM. These include the following (Section 3):
    - not all the ACM present may be involved in the fire;
    - fibres may be entrapped, in larger pieces of material etc.;
    - respirable fibres will be a fraction of the total released;
    - some fibres may be ‘denatured’ at the temperatures involved;

  - atmospheric dispersion and deposition (particularly as a result of rain) will reduce concentrations; and
  - the duration of exposure will be short.

- The available evidence indicates that asbestos exposures of members of the public following fires involving ACM will be very small if appropriate clean-up operations are undertaken. (Section 3)
- There is no direct evidence of long-term health risks from fires involving ACM, although the literature in this area is limited. Considering the available evidence on asbestos exposures from fires involving ACM in the context of the results of epidemiological studies of occupational and environmental asbestos exposures it is concluded that the risks of long-term health risks (mesothelioma and lung cancer) are minimal if appropriate clean-up occurs. It is recognised that this analysis involves the extrapolation of exposure response models developed from occupational studies of populations exposed for longer periods at significantly higher asbestos concentration levels. However, it is considered that this approach is reasonable and unlikely to underestimate the risks. This conclusion is in agreement with other similar studies in this area. (Section 4)
- The majority of asbestos encountered in such incidents will be chrysotile. The type of asbestos is a major consideration as the exposure specific risk of mesothelioma is broadly in the ratio 1:100:500 for chrysotile, amosite and crocidolite respectively. Identification of the asbestos type is, therefore, of great importance. (Section 4)
- It is recommended that all Local Authorities have a written policy for dealing with large scale fires involving asbestos. This might be a full and detailed asbestos fire specific plan or simply further guidance in addition to a generic incident plan covering only those issues pertinent to asbestos. (Section 5).
- Some members of the public perceive a greater risk from large scale fires involving asbestos than is actually the case, and this needs to be taken into consideration when devising and issuing public warnings. (Section 5).

CONTENTS

1 INTRODUCTION

Large scale fires involving asbestos containing materials (ACM) are a relatively common occurrence in the UK (HPA, 2005) and can cause significant public concern. It was therefore considered important to investigate the potential public health consequences of such incidents and explore actions that can be taken to minimise their impact.

To this end, a systematic literature review has been undertaken to identify available information on both the level of asbestos exposures that might result from fires (Section 3) and the potential health impact of such exposures (Section 4). The key elements of plans for and responses to such incidents that can minimise their impact have also been briefly explored (Section 4). The conclusions and recommendations of this study are summarised in Section 5. This study builds upon earlier work in this area (Saunders, 1996; 1999).

1.1 Asbestos in building materials

Asbestos is the name given to a small group of naturally occurring silicate minerals that can be readily separated into thin, strong fibres. These are divided into two sub-groups: serpentine (chrysotile), which is the most commonly used form of asbestos, and the amphiboles (amosite, tremolite, actinolite, anthophylite, and crocidolite), of which crocidolite is the most commonly used. Asbestos is defined in UK legislation as one, or a mixture of, any of the following: chrysotile (white), crocidolite (blue), amosite (brown), fibrous anthrophylite, fibrous tremolite and fibrous actinolite (CAR, 2006). Asbestos fibres are flexible, very strong and resistant to heat and chemicals. These properties have been recognised for thousands of years and asbestos was used by the ancient Egyptians, Greeks and Romans. It is only, however, in the relatively recent past that asbestos use, and therefore potential exposure, became widespread.

About six million tonnes of asbestos have been imported to the UK since the late 19th Century with a peak of around 195,000 tonnes in 1973. Imports of crocidolite and amosite ceased in 1972 and 1980 respectively (DoE, 1991) and imports of chrysotile had ceased by 1999. The Asbestos (Prohibitions) Regulations 1992 (AR, 1992) banned the importation, supply and use of blue and brown (amphibole) asbestos in the UK and also the supply and use of any product to which amphibole asbestos had been intentionally added. A number of products and uses of white (chrysotile) asbestos were also prohibited. The regulations were amended in 1999 (AR, 1999) to ban the importation, supply and use of chrysotile asbestos to implement EC Directive 1999/77/EC (EU, 1999) and to extend the prohibitions on importation to include any product containing asbestos. As a result of EC Directive 1999/77/EC (generally referred to in this context as the Marketing and Use Directive) the importation, supply and use of asbestos was banned throughout the EU from 1st January 2005.

Due to its extensive use asbestos is still found in buildings in some 3,000 products including sprayed coatings/lagging, insulating boards, ropes, cloth, millboard, asbestos-cement sheets, coated metal, textured paints and reinforced plastics etc. (DoE, 1991).

The amount and type of asbestos found in the fabric of buildings depends on the product. Sprayed coating was a mixture of hydrated asbestos-cement containing up to 85% asbestos fibre, mainly amosite but also crocidolite and chrysotile to a limited extent. Crocidolite and amosite were used for lagging up to the mid-sixties. Some lagging millboard and paper may contain up to 100% asbestos. Asbestos-cement products contain 10-15% asbestos fibre, generally chrysotile. Examples of asbestos materials used in buildings are given in Table 1. Examples of asbestos containing products encountered in fires include roof tiles and asbestos-bitumen roof coatings.

1.2 Frequency of large scale fires involving asbestos products

A review of information on chemical incidents reported to the Health Protection Agency (HPA) over the five year period 1999 - 2004 indicated that asbestos was one of the most commonly identified chemicals (12%), with a typical event identified as a factory fire where the roof contained asbestos (HPA, 2005).

The number of incidents involving asbestos generally increased over the five year period with a peak of around 100 such incidents in 2002/3 and 70 in 2003/4 (Jeffery and Saunders, 2005). It is recognised that the conclusions of the review are limited by data variability, under-reporting and regional reporting bias, and thus there is a degree of uncertainty around the total number of incidents involving asbestos. It is clear from the available information, however, that a significant fraction of the total number of incidents reported involved asbestos. A review of reported chemical incidents for 2005 indicates that asbestos was again one of the most commonly identified chemicals, representing 5% of all incidents (n=54) (Figure 1) (HPA, 2007).

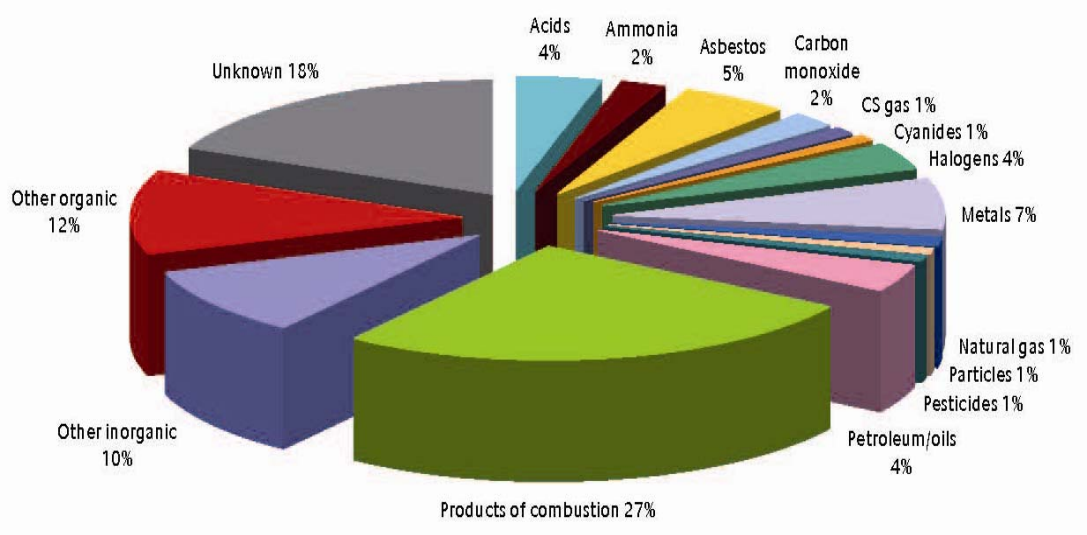

Figure 1 Chemicals involved in chemical incidents for 2005 (HPA, 2006).

Table 1 Examples of asbestos materials used in buildings (DoE, 1991)

Asbestos Product	Use	Asbestos content	Remarks
Sprayed asbestos coatings	Thermal and acoustic insulation. Fire and condensation protection.	Sprayed coatings contain up to 85% asbestos. A mixture of types was used until 1974. Crocidolite was used for the thermal insulation of steam turbines until 1970. Amosite was used for fire protection of structural steel condensation protection and acoustic control. Chrysotile, mixed with mineral wool and binder, was used until 1974. Chrysotile was also used as a coating on top of other sprayed asbestos.	Potential for fibre release unless sealed. Potential increases as the materials age or become friable and disintegrate. Dust released may then accumulate. Removal of sprayed coating is a licensed activity.
Asbestos lagging	Thermal insulation of pipes, boilers, pressure vessels etc. Includes preformed pipe sections, slabs, tape, rope, corrugated paper, quilts, felts and blankets.	All types of asbestos have been used. Content varies (e.g.. 6-8% in calcium silicate slabs, 100% in blankets, felts, etc.).	Friability depends on the nature of the lagging. Potential for fibre release unless sealed. Potential increases as the materials age or become friable and disintegrate. Dust released may then accumulate. Removal of lagging is a licensed activity.
Insulating boards	Fire protection, thermal and acoustic insulation, resistance to moisture movement and general building board. Used in ducts, firebreaks, infill panels, partitions and ceilings (including ceiling tiles), roof underlays, wall lining, bath panels, external canopies and porch linings.	Crocidolite used for some boards up to 1965. 16-40% amosite and chrysotile.	Likely to cause a dust hazard if very friable, broken, abraded, sawn or drilled.
Insulating board cores and linings of composite products.	Acoustic attenuators cladding infill panels, domestic boiler casings, partition and ceiling panels, oven linings and suspended floor systems.		
Ropes and yarns.	Lagging. Jointing and packing materials. Heat/fire resisting gaskets and seals. Caulking in brickwork. Boiler and flue sealing. Plaited asbestos tubing in electric cable.	All types of asbestos where used until about 1970. Since then only chrysotile has been used. Asbestos content approximately 100%.	Fibre may be released when large quantities of unbonded material are stored or handled. Caulking etc. in situ is not likely to release fibre.

2 ASBESTOS REGULATIONS

The Control of Asbestos Regulations (CAR) came into force on 13 November 2006. These regulations bring together the three previous sets of regulations covering the prohibition of asbestos, the control of asbestos at work and asbestos licensing. The regulations also implement EU Council Directive 83/477/EEC (EU, 1983) on the protection of workers from the risks related to exposure to asbestos at work, as amended in 2003 (2003/18/EC) (EU, 2003). The Regulations are supported by two Approved Codes of Practice – *Work with materials containing asbestos* (HSE, 2006a); and *The management of asbestos in non-domestic premises* (HSE, 2006b).

The regulations require mandatory training for anyone liable to be exposed to asbestos fibres at work. This includes maintenance workers and others who may come into contact with or who may disturb asbestos (eg cable installers) as well as those involved in asbestos removal work.

When work with asbestos or which may disturb asbestos is being carried out, the regulations require employers and the self-employed to prevent exposure to asbestos fibres. Where this is not reasonably practicable, they must make sure that exposure is kept as low as reasonably practicable by measures other than the use of respiratory protective equipment (RPE). The spread of asbestos must be prevented. The Regulations specify the work methods and controls that should be used to prevent exposure and spread.

Worker exposures must be below the airborne exposure limit (Control Limit). The asbestos regulations have a single Control Limit for all types of asbestos of 0.1 fibres per ml[*]. A Control Limit is a maximum concentration of asbestos fibres in the air (averaged over any continuous 4 hour period) that must not be exceeded. In addition, short term exposures must be strictly controlled and worker exposures should not exceed 0.6 fibres per ml of air averaged over any continuous 10 minute period, using RPE if exposure cannot be reduced sufficiently using other means (HSE, 2006a). These limits are summarised in Table 2.

Table 2 Occupational limits for asbestos exposure

	f/ml of air averaged over any continuous period of:	
	4 hours (CAR, 2006)	10 mins (HSE, 2006a)
All types of asbestos	0.1	0.6

The Health and Safety Executive (HSE) has produced extensive guidance on working with asbestos (www.hse.gov.uk/asbestos/index.htm). This includes guidance on how asbestos removal work should be carried out, the asbestos exposure limits and how they are used, and when and where air monitoring is necessary during asbestos

[*] Concentrations of asbestos is air are generally expressed as fibres per ml of air (f/ml). Cumulative exposures as f/ml-hours or f/ml-years.

removal and disturbance work, and how it should be carried out (eg HSE, 2005; 2006a, 2006b).

The Approved Code of Practice (ACOP) *Work with materials containing asbestos* (HSE, 2006a) defines, for licensable activities, the 4-stage clearance procedure required for production of a certificate for reoccupation of a building when asbestos removal work has finished. The third stage of the procedure involves air sampling, and the manner in which this should be carried out is defined in the ACOP. It is stated that in most cases it will be reasonably practicable to clean the area thoroughly enough that the airborne fibre concentration after final clean would be less than 0.01 f/ml. If measurements of 0.01 f/ml or more are found then investigations would need to be carried out to find the cause and, in general, additional cleaning activities undertaken. The ACOP states that *'the threshold of less than 0.01 f/ml should be taken only as a transient indication of site cleanliness, in conjunction with visual inspection, and not as an acceptable permanent environmental level*'. It is indicated in the ACOP that for licensable asbestos removal work performed out of doors there is no requirement for air sampling.

There are currently no UK standards for asbestos in the environment that are directly relevant to asbestos exposures to members of the public following fires. However, the Environment Agency (Grosso, 2007) is currently developing guidance on remediation standards for asbestos contaminated land, which may be of interest in this area.

There are also no relevant international standards. World Health Organisation (WHO) guidance on air quality (WHO, 2000) states that asbestos is a proven carcinogen for which no safe air concentration level can be proposed because a threshold is not known to exist. For contaminants of this type it is indicated that risk managers need to regulate at levels that result in an acceptable degree of risk and generally to keep exposures as low as possible (or prohibit). However, the WHO report does note that a number of groups have proposed that limiting the concentration of asbestos in air to 0.0005 f/ml would provide adequate health protection. A lifetime exposure at this level is said to equate to a lifetime mesothelioma risk of the order of 10^{-5} to 10^{-4} and a lung cancer risk (assuming population 30% smokers) of the order of 10^{-6} to 10^{-5} (WHO, 2000). Any such a standard would, however, relate to long-term exposures from controlled processes, and as such would also not be directly relevant to exposures following fires.

Although asbestos is a known human carcinogen by the inhalation route, WHO has concluded that epidemiological studies do not support the hypothesis that an increased cancer risk is associated with the ingestion of asbestos in drinking water. They therefore concluded that there is no need to establish a guideline for asbestos in drinking water (WHO; 2003, 2004).

3 ASBESTOS EXPOSURES FROM FIRES

The potential for fibre release from ACM depends on three principal factors:

- the type of material/asbestos;
- the integrity of the material and any sealant or enclosure; and

- the position of the material.

If the ACM is intact and well maintained, there is no cause for concern. However, in the event of a fire there is considerable potential for fibre release.

Fire can change the mineral structure and mechanical strength of asbestos, fixing the fibres. While this transformed fabric is not as hazardous, the process will generally only affect the outer layers leaving most fibres intact within the material.

Fires can disrupt the structure of a building leading to the break-up of any asbestos material and the release of fibres into the atmosphere. Fires may burn off the coating from buildings clad or roofed with asbestos-bitumen, and asbestos-cement can explode releasing fibres over a wide area. Asbestos sheets offer no fire resistance and will crack in building fires (Hoskins and Brown, 1994). There are anecdotal reports that these violent events massively increased the level of airborne fibres over a considerable area in war zones such as Bosnia. During the Los Angeles riots of 1992 there were numerous fires involving asbestos materials and there were general concerns regarding asbestos in burned debris and in the atmosphere (Evans, 1993). Public health officials found that over one third of fire damaged sites requiring clean-up were contaminated with asbestos. These sorts of reports clearly heighten the public's concern following a fire.

During a fire most asbestos will be deposited as large pieces. Many fibres will also be trapped in the fabric of the building. The temperatures involved in large scale fires may also result in the 'denaturing' of some of the asbestos present[*] (Jeyaratnam and West, 1994).

All the above factors mean that the respirable fraction of the released fibres will be small - large fibres above 100 μm in length or 3 μm in diameter are not respirable and the smallest <0.01 μm are not retained in the lungs (Hoskins and Brown, 1994). Airborne fibres of between 5 and 100 μm in length, with diameters less than 1.5 - 2 μm and ratios of length to diameter of more than 5 to 1 are most hazardous (Doll and Peto, 1985).

Whilst fragments will settle out relatively quickly, fibres, including respirable fibres, may travel considerable distances in some circumstances. The concentration of respirable asbestos fibres in air at locations around the site of the fire will depend upon a number of factors including: the quantities of respirable asbestos released; the heat generated by the fire; the distance from the fire; the meteorological conditions; and the type of surfaces onto which fall-out occurs. Rain will have a particularly significant effect as it enhances deposition of the fibres onto the ground and hence removal (to drains etc).

The main potential exposure pathways are direct inhalation of asbestos in the original plume and the inhalation of asbestos fibres resuspended into the air (e.g. wind-driven or a result of mechanical processes) following deposition on the ground or other surfaces.

[*] The degree of denaturing will depend upon the asbestos type, the material matrix, the temperature reached and the time at elevated temperature. Temperatures will vary significantly throughout a large building on fire and also through any ACM involved. It is therefore not possible to make general statements about the fraction of the asbestos involved that would be denatured.

Another potential exposure route is through ingestion of local produce but this pathway is not considered a significant risk (Bridgman, 2001). The International Agency for Research on Cancer (IARC) judged that there was no evidence of an increased cancer risk from fibres in water, beverages or food (Becklake, 1976).

The exposures received by members of the public will clearly depend upon the concentration of asbestos in the air (either directly from the plume during the fire or as a result of resuspension following the fire), and the subsequent actions of the public and authorities. Individuals indoors will, for example, receive lower exposures than those outside at similar locations. Rapid removal of significant fall-out will also reduce the potential for significant resuspension exposures of members of the public although may result in exposures to the staff involved in the clean-up.

The public health impact of such fires will clearly depend upon the levels of public exposure. A systematic review of the literature on public exposure to asbestos from fire incidents was undertaken for this report (see Section 4 for details). This search covered the period from 1996 to February 2005[*]. The search indicated that that the literature on human exposures to asbestos from fires during this period was limited, i.e. only two papers were identified (Bridgman, 2001; Landrigan et al, 2004). An earlier search had identified an additional paper (Lewis and Curtis, 1990) and some unpublished information in this area was also obtained (Adams 1996; Matthews 1996).

A study following a major fire in a large ordnance warehouse with an asbestos cement roof in 1988 revealed no significant levels of fibres in the air 1-18 days after the fire, see Tables 3 and 4 (Lewis and Curtis, 1990). This included exposure of individuals involved in the manual clean-up operation including the use of a lawn mower. Phase contrast microscopy (PCM) was used to analyse the fibres and some caution is needed in interpreting the results as this method will not detect very thin fibres (<0.1μm in diameter) and counts all fibres including non-asbestos. However, it gives results very quickly and PCM is considered adequate for occupational and epidemiological purposes (DoE, 1991). The warehouse covered 40,000 m^2 and was roofed in corrugated asbestos-cement sheeting containing 10% chrysotile. This would contain approximately 3500 kg of asbestos.

A major factory fire in Tranmere, Merseyside, England on 22 September 1994 deposited asbestos containing fallout in an urban area. Fallout from the fire consisted of both large paper-like material and fine particles. It was later reported that the fallout contained both white (chrysotile) and brown (amosite) asbestos. A clean up operation was carried out over the following two days. The public were informed by the Local Authority and Health Authority that the dangers of such an incident should be minimal. Despite this advice, publicity and controversy over health risks continued. Therefore Wirral Health Authority commissioned a study of the health consequences and lessons learnt from the incident, some months after the acute phase of the incident had been dealt with. As part of the study, conservative estimates of the exposure of the population were made. No air samples were taken during the fire or in the immediate aftermath. The exposures were

[*] This period was chosen as it represented an update to an earlier literature search (Saunders, 1996) that identified only one paper on human exposures to asbestos from fires (Lewis and Curtis, 1990).

therefore estimated on the basis of measurements of the asbestos content of fallout from the fire. Some air samples were taken during demolition. These were used to determine the fibre content of the air sampled. Unfortunately this did not allow for differentiation between asbestos and non-asbestos fibres. These measurements were interpreted as indicating that during demolition air asbestos fibre concentrations did not exceed normal background levels. The study concluded that the predominant source of asbestos deposited in the surrounding urban area was asbestos bitumen paper from the factory roof. It was estimated that the roof of the factory contained 240 kg of chrysotile. The assumption made in the study was that exposure reached the industrial control limit, which was 0.5 fi/ml at that time, for two days. It was stated that this was likely to greatly overestimate the actual exposures of members of the public (Bridgman; 1996, 2001).

Table 3 Environmental asbestos monitoring 1-18 days after fire (Lewis and Curtis, 1990)

	No. of samples < 0.01 f/ml air	No. of samples > 0.01 f/ml air
Static sampling	83	3*
Personal sampling of contractors clearing up	4	4†

Notes:

* On the day after the fire one value of 0.02 f/ml and two values of 0.01 f/ml were found at sites immediately adjacent to the warehouse.

† Positive values of personal monitoring of contractors consisted of: two values of 0.02 f/ml (one occurring whilst lawn mowing); one value of 0.03 f/ml; one value of 0.4 f/ml. Further examination of the 0.4 value (including interviewing the worker whose sample gave the result) suggested it was an artefact.

Table 4 Asbestos monitoring inside warehouse following fire (Lewis and Curtis, 1990)

	No. of samples < 0.01 f/ml air	No. of samples > 0.01 f/ml air
Static sampling (including 10 contact tests)	34	7*
Personal monitoring (surveying and working)	22	0

Note:

* The following positive values were found in the vehicle workshop: one value of 0.03 f/ml, two values of 0.02 f/ml and four values of 0.01 f/ml. These were almost certainly due to pre-existing contamination due to previous roof maintenance work.

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Air sampling carried out in the immediate aftermath of fires involving substantial asbestos cement roofs in the West Midlands did not reveal any significant levels of asbestos fibres (Adams, 1996; Matthews, 1996).

Measurements were made of dust arising from the destruction of the World Trade Center (WTC) in 2001 (Landrigan et al, 2004). The dust was found to contain a number of chemical toxins including asbestos. Asbestos, primarily chrysotile, was used for fire insulation in the construction of the north tower up to the 40th floor. Because of its known carcinogenic potential asbestos became a major health concern. Samples of airborne material were taken, commencing on 14th September. By this stage the predominant sources of pollution were smouldering fires, with occasional flare-ups, and resuspension of settled dust and smoke. More than 10,000 ambient air samples from lower Manhattan were tested for asbestos by the US Environmental Protection Agency (EPA) using phase-contrast light microscopy (PCM) to identify fibres > 5 µm in length; more than 8,000 of these samples were also examined by transmission electron microscopy (TEM) to identify fibres of ≥ 0.5 µm in length. Twenty-two of the air samples analysed by the US EPA were found to contain asbestos at levels above the clearance standard of 70 fibres/mm^2 established under the US Asbestos Hazard Emergency Response Act (US EPA, 1986). This standard uses the TEM measurement technique. Most of the elevated asbestos levels in air were observed in the earliest days after the destruction. There were no 8 hour time-weighted average asbestos exposures to workers above the Occupational Safety and Health Administration standard (US Department of Labor, 2003), which uses the PCM measurement technique, of 0.1 fibre/cm^3, although workers undoubtedly had short term peak exposures when they disturbed asbestos-containing rubble at Ground Zero. The ambient air samples showed that asbestos exposures were initially elevated but fell to within US EPA standards after the first few days (US EPA, 2004). Asbestos was found in settled dust at ground zero in concentrations ranging from 0.8 – 3%. Asbestos was found in dust in nearby apartments and other buildings, sometimes at higher levels than in the outside environment.

In addition to direct inhalation of asbestos fibres in the plume or inhalation of fibres resuspended following deposition on the ground, inhalation of asbestos fibres resuspended following deposition on clothing is another possible exposure route, especially for first responders and those involved in clean-up. However, this pathway is not significant. Analysis of the tunics worn by emergency personnel attending the fire at the British Leather Factory in Tranmere in 1994 revealed – '*an insignificant number of ..... fibres in the weave .... consistent with normal background levels arising from building dust emanating from general fire incidents. There was no evidence to suggest that dangerous asbestos contamination of the garment had occurred at the time of the fire'* (Bridgman, 2001).

The studies reported above indicate that asbestos exposures of members of the public during and in the immediate aftermath of a fire involving asbestos material are expected to be minimal and that in the longer term, as long as appropriate clean-up has taken place, the exposures will also be minimal.

For the purposes of this study, a conservative estimate of the maximum asbestos exposure of a member of the public following a fire has been made. Landrigan et al (2004) indicated that measurements taken commencing three days after the destruction of the World Trade Center indicated that there were no 8 hour time-averaged asbestos exposures to workers above 0.1 f/ml. Exposures following such an event are expected to be at the extreme end of any expected from a large scale fire in the UK and are certainly higher than those indicated by Lewis and Curtis (1990), Adams (1996) and Matthews (1996). If it is assumed that exposure at a level of 0.1 f/ml lasts for a period of 2 days, again a conservative assumption, and that following this period, as a result of appropriate clean-up activities taking place, exposures are around ambient background levels then this would result in a cumulative exposure of < 0.0006 f/ml-years.

3.1 Ambient asbestos concentrations

To place the potential asbestos exposures from fires in context it is useful to consider ambient background levels. Mineral fibres, including asbestos, are widespread contaminants of the environment and everybody will have been exposed at some stage.

In the literature a wide range of background levels are reported. The Ontario Royal Commission estimated levels of asbestos fibres in buildings containing asbestos to be of the order of 0.001 f/ml (Ontario Royal Commission, 1984), while the then UK Department of the Environment (DoE) estimated a level of regulated fibres of 0.0005 f/ml above background (DoE, 1991). The Health Effects Institute estimated mean concentrations of 0.00051, 0.00019 and 0.0002 f/ml in schools, homes and public buildings respectively (HEI, 1991). A study of a closed crocidolite mine in South Africa found ambient levels of 0.0002 - 0.0007 f/ml in the immediate vicinity (Reid et al, 1990) which is reflected by Bignon's estimate of 0.0001 f/ml for remote rural areas and 'asbestos free' buildings (Bignon, 1989). Bignon goes on to estimate ambient levels of 0.0002 - 0.011 f/ml for residential areas, 0.001 - 0.003 f/ml on roads and 0.001 - 0.04 f/ml in buildings. Other reviews have indicated that outdoor ambient levels of respirable asbestos fibres may range from 0.000001 to 0.0001 f/ml and that most indoor concentrations are below 0.0002 f/ml (IEH, 1997). A summary of asbestos in air measurements indicates the following concentrations levels, WHO (2000):

- Rural areas (outdoors - remote from asbestos emission sources) - below 0.0001 f/ml
- Urban areas (outdoors) – general levels may vary from below 0.0001 f/ml to 0.001 f/ml
- In buildings without specific asbestos sources – generally below 0.001 f/ml

WHO (2000) provides estimates of typical lifetime cumulative asbestos exposures of members of the public in industrialised countries from ambient outdoor concentrations.

For an urban population with moderate exposure an average exposure of 0.00003 f/ml is assumed for 70 years, resulting in a cumulative exposure of 0.0021 f/ml-years and the inhalation of approximately 15 million fibres. For a rural population a fibre concentration of 0.00001 f/ml is assumed, resulting in an estimated cumulative exposure of 0.0007 f/ml and the inhalation of approximately 5 million fibres. Values for indoor air exposure have not been included in the above estimates because only a few are available. However, it is noted that one study in the USA indicated that median concentrations range from 0.0004 – 0.0005 f/ml. If these estimates were correct, they would result in a lifetime fibre burden of up to 200 million fibres (implying a cumulative exposure of 0.035 f/ml-years). In this case outdoor exposure would be of minor importance to most of the US population.

4 THE PUBLIC HEALTH IMPACT OF ASBESTOS EXPOSURES FROM FIRES

4.1 Acute health effects

Potential acute medical problems from large scale fires are generally those from thermal injury or inhalation of products of combustion (smoke inhalation). Asbestos is not acutely toxic. It may produce irritation of the skin, eyes and respiratory tract due to the mechanical action of the fibres but only at very high air concentration levels (Fielder, 2006), well beyond those that members of the public would encounter from a fire. Such acute effects have only ever been found in mining and processing workers. Asbestos fibres released in a fire would therefore not be expected to cause acute medical problems (Bridgman, 2000). However, the potential for health impacts due to anxiety as a result of the presence of asbestos should not be ignored (Bridgman, 2000).

4.2 Long-term health effects

The principal diseases known to be caused by exposure to asbestos are asbestosis, non-malignant pleural disease, lung cancer, and malignant mesothelioma. Mesothelioma is a formerly rare cancer* that principally affects the pleura and the peritoneum (Greenberg and Lloyd Davies, 1974) and is almost always caused by asbestos exposure. The disease is rapidly fatal with most of those affected dying within a year of diagnosis (Peto et al, 1995). There is a long latent period between first

* The annual number of mesothelioma deaths in Great Britain has increased from 153 in 1968 to 1848 in 2001. Models derived by matching the changing mesothelioma mortality rates over this period with the rise and fall in asbestos exposure during the 20th century have been used to make predictions of future rates of the disease. Such studies indicate that the death rate is expected to peak at around 1950 per year between 2011 and 2015 (Hodgson et al, 2005). Following this peak, the number of deaths is expected to decline rapidly reflecting significant reductions in occupational exposures many decades previously.

exposure to asbestos and diagnosis of mesothelioma that is seldom less than 15 years and often exceeds 60 years (Bianchi et al 1997).

There is also some evidence linking asbestos and laryngeal cancer (Doll and Peto, 1985). In addition, there has also been some concern regarding asbestos and breast cancer with some studies showing weak links (Cantor et al, 1995; Goldberg and Labreche, 1996). However, no significant associations have been reported. There is no current clinical evidence of asbestosis in the general public (Doll and Peto, 1985). The focus of this study was therefore on mesothelioma and lung cancer. Peritoneal mesothelioma is less prevalent than pleural mesothelioma and thus most epidemiological studies concentrate on the latter and, although peritoneal mesothelioma has not been excluded from this study, the focus here is also on the pleural form.

The potential long-term impact on health of asbestos exposures from fires can be explored by considering information on the likely level of asbestos exposures in the context of the results of epidemiological studies on the risks from asbestos exposure. A number of epidemiological studies to consider the risks from both occupational and environmental exposures have been undertaken and the results of these are explored in the following sections.

4.2.1 Occupational epidemiology

A large number of studies of the health impact of occupational asbestos exposures have been undertaken and are reported in the literature. These indicate in general that the incidence of mesothelioma and lung cancer increases as exposure to asbestos increases. On the basis of such studies a number of exposure-response models have been developed over the years.

For example, Peto et al (1982) showed that the incidence of mesothelioma was dependent on time since first exposure, but not dependent on age at first exposure, nor smoking habit nor gender. The exposure-response model proposed by Peto et al (1982), which is still widely used (WHO, 2000; Jones et al, 2007), has incidence as a function of time expressed in terms of asbestos fibre concentrations in air, duration of exposure and time since first exposure. For continuous exposure this is of the form:

$$I_m (t,f) = k_m \times f \times (t-t_1)^n$$

Where, $I_m(t,f)$ is the mesothelioma incidence at time t for asbestos exposure concentration f (f/ml), k_m is a constant expressing the mesothelioma risk per unit of exposure, $(t-t_1)$ is the time since first exposure and n is a parameter expressing the relative importance of elapsed time since start of exposure. Modified forms of the equation can be used to estimate incidence after exposure has ceased. Modified versions have also been developed to include a lag effect of, for example, 10 years to allow for the latency of disease development after exposure. Values of k_m and n have been derived from a number of studies. Values for k_m in the range $1\ 10^{-8}$ to $3\ 10^{-8}$ have been proposed (HEI, 1991). Peto et al (1982) recommended using a value for n of 3.5, although many studies have used values of 3.0 or 3.2. Such a model is very flexible in that it allows for the determination of risks with time and for various levels of exposure.

The situation for lung cancers is rather more complex than for mesothelioma in that any excess cases of lung cancer appear against a background of a relative high incidence of lung cancers from other causes, in particular smoking. This makes the epidemiology more complex. Many studies have shown that smokers have a higher risk of developing lung cancer than non-smokers when exposed to asbestos. Early studies suggested an interaction between asbestos and smoking that appeared to be multiplicative, however, in general it is considered that, although more than additive, the effect is probably less than multiplicative and not simple (McDonald, 2000).

In general, therefore, the exposure-response models developed express the excess lung cancer risk relative to that in a similar population (age, smoking habits etc) without asbestos exposure. Such models predict a percentage increase on the existing risk, where the existing risk is much higher for a smoker than a non-smoker. For example, Doll and Peto (1985) developed a linear no-threshold model to predict lung cancer from asbestos exposure. This defines the relative risk as follows:

Relative risk = O/E = 1 + b x cumulative exposure

Where, O is the number of cases observed, E is the number of cases expected in the absence of asbestos exposure, b is a constant and the cumulative exposure is in f/ml-years. There has been some debate about the most valid value for the constant, b in the model. Doll and Peto (1985), on the basis of an analysis of a cohort of chrysotile textile workers, proposed a value of 0.01, however, Hughes (1994) argues that for a population exposed to non-textile chrysotile, the available data suggests a constant of 0.0006 is more appropriate.

The models discussed above are still widely used. However, it is always important to note that they were derived on the basis of high occupational exposure and extrapolating to lower exposures introduces further uncertainty.

A brief review of the predicted risks of mesothelioma and lung cancer from a number of asbestos studies has been carried out by WHO (2000), with the objective of estimating risks from background environmental asbestos exposure levels. The risks were based on evidence from epidemiological studies concerning occupational exposure, including Doll and Peto (1985) and Peto et al (1982). Data from these studies were conservatively extrapolated to the much lower concentrations found in the general environment. Although WHO acknowledge the evidence that chrysotile is less potent than amphiboles, as a precaution chrysotile was attributed the same risk in these estimates. The study concluded that a best estimate of the lifetime mesothelioma risk resulting from lifetime exposure to asbestos at a level of 0.0001 f/ml was $2\ 10^{-5}$. A review of lung cancer risks indicated a predicted lifetime risk for the same level of lifetime exposure of $2\ 10^{-5}$ for smokers and $2\ 10^{-6}$ for non-smokers.

A recent, more substantial review by Hodgson and Darnton (2000) summarises information on the risks of lung cancer and mesothelioma (both pleural and peritoneal) for various occupational exposure levels. This is an extensive review of quantitative risks from asbestos exposure undertaken by staff in the Health and Safety Executive's Epidemiology and Medical Statistics Unit. Mortality studies on asbestos exposed cohorts that gave information on exposure levels from which (as a minimum) a cohort average

cumulative exposure could be estimated were systematically reviewed (17 such studies were identified).

The reviewed cohorts all had high (> 10 f/ml-years) cumulative asbestos exposures. To use the evidence from such studies to estimate risks at much lower exposures the standard assumption made is of dose-linearity (ie risk proportional to dose). However, if the true relationship is not linear, the impact on low dose extrapolations could be significant. The studies were therefore examined in detail to explore any possible deviations from dose linearity. The authors concluded that there is some indication in the data suggesting a non-linear exposure response, particularly for peritoneal mesothelioma.

Hodgson and Darnton (2000) consider that the mesothelioma risk is best expressed using the following model:

$P_M = A_{pl}X^r + A_{pr}X^t$

Where P_M is the percent excess mortality, r and t are the slopes of, respectively, the pleural and peritoneal responses on a log-log scale, A_{pl} and A_{pr} are constants of proportionality for the respective elements of the risk, and X is the cumulative exposure. On the basis of the analysis of the occupational studies best estimate values for r and t are given as, respectively, 0.75 and 2.2 (a value of unity would indicate linearity). Thus the model implies that the risk of pleural mesothelioma rises more steeply at low exposures than at high exposures and that each additional unit of exposure will add progressively less risk for pleural tumours and more for peritoneal. The point at which risks for tumours at the two sites are predicted to be equal is around 90 f/ml-years for crocidolite and 55 f/ml-years for amosite. Below these values pleural tumours are more common and vice versa. The plausible range on r is stated to be 0.6 to 1.0 (linearity). The equivalent range on t is 1.7 to 2.5.

For lung cancer the following model is proposed:

$P_L = A_lX^r$

Where the parameters are defined equivalently to those above. The best estimate for r is 1.3 with a range from 1.0 (linearity) to 1.6.

The authors note that although these non-linear relationships provide the best fit to the data, statistical and other uncertainties mean that a linear relationship remains arguable for pleural mesothelioma and lung tumours (but not for peritoneal tumours).

Using the above models, Hodgson and Darnton generated estimates of risks for various cumulative exposures, including exposures outside the range for which direct observations were available. Under such circumstances there are two primary sources of uncertainty in the estimated risks. Firstly there is the usual statistical uncertainty of inferring underlying risk from observations in particular groups. This uncertainty can to some extent be quantified. Such uncertainties are typically expressed as a CI. The second type of uncertainty relates to whether the relationship between exposure and outcome seen in the observed range is also valid outside that range. This uncertainty cannot be quantified statistically. Uncertainty about the slopes of exposure-response lines has an increasing impact with increasing distance from the observed range. For

these reasons Hodgson and Darnton considered that simply presenting a table of risk estimates for different cumulative exposures was not appropriate, as this would not capture the changing balance of the different types of uncertainty. They therefore produced a table (reproduced below in Table 5) giving a numerical and qualitative assessment of lifetime risk at a range of cumulative exposures. No estimates are given for lifetime risks lower than 1 in 100,000, and this level is referred to as 'insignificant'.

The statements on lifetime risks relate to exposures accumulated over 'short' (up to 5 years) periods from age 30. Guidance is given on extrapolating to different ages at first exposure and for longer exposure durations. The results confirm that exposure to amphibole fibres is appreciably more hazardous than exposure to chrysotile.

The results indicate that the exposure specific risk of mesothelioma is broadly in the ratio 1:100:500 for chrysotile, amosite and crocidolite respectively. For lung cancer the conclusions are less clear with a risk differential between chrysotile and the two amphibole fibres of between 1:10 and 1:50.

The estimated risks from the WHO review (WHO, 2000) are for lifetime exposures and don't differentiate between asbestos types, it is therefore difficult to compare directly with those in Hodgson and Darnton (2000). However, making reasonable assumptions about exposure durations and asbestos types the results from the two reviews are broadly consistent.

Table 5 Quantitative cancer risks from asbestos exposure at different levels of cumulative exposure[a,b,c,d] (from Hodgson and Darnton (2000))

Fibre	Mesothelioma	Lung cancer
Risk summaries for cumulative exposures between 10 and 100 f/ml-years		
Crocidolite	Best estimate about 400 deaths per 100 000 exposed for each f/ml-year of cumulative exposure. Up to 2-fold uncertainty.	Rising from about 150 (range 100 to 250) excess lung cancer deaths per 100 000 exposed for each f/ml-year of cumulative exposure at 10 f/ml-years to 350 (range 250 to 550) at 100 f/ml-years.
Amosite	Best estimate about 65 deaths per 100 000 exposed for each f/ml-year of cumulative exposure. 2-fold to 4-fold uncertainty.	
Chrysotile	Best estimate about 2 deaths per 100 000 exposed for each f/ml.year of cumulative exposure. Up to 3-fold uncertainty	Best estimate about 5 excess lung cancer deaths per 100 000 exposed for each f/ml-year of cumulative exposure. Cautious estimate 30. In exceptional circumstances (see note c) it is arguable that an estimate of 100 might be justified
Risk summaries for cumulative exposures of 1 f/ml-years		
Crocidolite	Best estimate about 650 deaths per 100 000 exposed. Highest arguable estimate 1500, lowest 250.	Best estimate about 85 (range 20 to 250) excess lung cancer deaths per 100 000 exposed.
Amosite	Best estimate about 90 deaths per 100 000 exposed. Highest arguable estimate 300, lowest 15.	
Chrysotile	Best estimate about 5 deaths per 100 000 exposed. Highest arguable estimate 20, lowest 1.	Best estimate about 2 excess lung cancer deaths per 100 000 exposed. Cautious estimate 30 per 100 000. In exceptional circumstances (see note c) it is arguable that an estimate of 100 per 100 000 might be justified. The case for a threshold-ie zero, or at least very low risk-is arguable
Risk summaries for cumulative exposures of 0.1 f/ml-years		
Crocidolite	Best estimate about 100 deaths per 100 000 exposed. Highest arguable estimate 350, lowest 25.	Best estimate about 4 (range <1 to 25) excess lung cancer deaths per 100 000 exposed`
Amosite	Best estimate about 15 deaths per 100 000 exposed. Highest arguable estimate 80, lowest 2.	
Chrysotile	Risk probably insignificant, highest arguable estimate 4 deaths per 100 000	Excess lung cancer deaths probably insignificant. Cautious estimate 3 per 100 000. In exceptional circumstances (see note c) it is arguable that an estimate of 10 per 100 000 might be justified. The case for a threshold – ie zero, or at least very low risk-is strongly arguable
Risk summaries for cumulative exposures of 0.01 f/ml-years		

Crocidolite	Best estimate about 20 deaths per 100 000 exposed. Highest arguable estimate 100, lowest 2.	Risk is probably insignificant (range <1 to 3 excess lung cancer deaths per 100 000 exposed). Mesothelioma is now the dominant risk, so precise estimations of the lung cancer risk is not critical.
Amosite	Best estimate about 3 deaths per 100 000 exposed. Highest arguable estimate 20, lowest insignificant	
Chrysotile	Risk probably insignificant, highest arguable estimate 1 death per 100 000 exposed	Risk of excess lung cancer very probably insignificant except in exceptional circumstances (see note c) when it is arguable that an estimate of 1 death per 100 000 might be justified. The case for a threshold-ie zero, or at least very low risk-is strongly arguable.
Risk summaries for cumulative exposures of 0.005 f/ml-years and lower		
At these levels only mesothelioma need be considered. The absolute risk is low-, but quantitative uncertainties are very considerable		
Crocidolite	Best estimate about 10 deaths per 100 000 exposed. Highest arguable estimate 55. Best estimate falls to insignificant level at 0.0002 f/ml-years, and highest arguable risk becomes insignificant at 6 10^{-6} f/ml-years.	Insignificant, possibly zero
Amosite	Best estimate about 2 deaths per 100 000 exposed highest arguable lifetime risk 15, falling to < 1 (ie insignificant) at 7 10^{-5} f/ml-years	
Chrysotile	Insignificant	Insignificant, very possibly zero

Notes:

a Exposure assumed to be accumulated over short - up to 5 yr periods starting at age 30. For exposure at other ages adjust the predicted mesothelioma figures using the factors in Table 9 of Hodgson and Darnton (2000). Estimates for longer periods of exposure can be approximated by making separate estimates for successive 5-year periods and adding the resulting risks (this will slightly overestimate risk). Estimates have been rounded to nearest 5 in second significant digit (or to make one significant digit when less than 10).

b The lung cancer risk is based on British male mortality in 1997 when 9.5% of male deaths at ages 40-79 were due to lung cancer. This represents an average for a population with a past pattern of smoking similar to that of older British men. In 1996 23% of men aged 60+ had never (or only occasionally) smoked and 25% were current smokers. For lifetime smokers the lung cancer risk will be about double the stated levels, for non-smokers about a sixth (if the interaction with asbestos is multiplicative) or about a third if the relative risk is higher than in non-smokers.

c The lung cancer risk arguable in ‘exceptional circumstances’ should only be considered where there is simultaneous exposure to textile grade (ie long fibre) chrysotile and mineral oil or some other analogous co-exposure.

d The simple pro rata formulae proposed in this table do not take account of the impact of competing causes of mortality. The impact will be trivial so long as the predicted asbestos related mortality is low, and limited for predicted (individual cause) mortality below about 30 percent. Above this level the individual asbestos related diseases (including asbestosis, which is not covered by this analysis) will reduce each other’s observed impact. In this situation all that can usefully be predicted is that the total asbestos related mortality will be very high indeed.

4.2.2 Environmental epidemiology

A systematic review of the literature on public exposure to asbestos from fire incidents and the possible health effects resulting from such exposures was undertaken for this report. The search criterion was to identify papers with at least one word from each of the following two keyword groups: Group 1: asbestos, mesothelioma, lung cancer, fibrosis; and Group 2: environment, short-term, fire, non-occupational. The review was also restricted to English Language papers and humans. The following databases were searched: BIDS; Web of Science; Medline; and Embase. The search covered the period from 1996 to February 2005[*]. The search identified 152 papers. The abstracts from all these were considered. Papers were excluded from further consideration if they related solely to occupational exposures. Papers were included if they referred to one of the following: asbestos release; exposure estimate; health assessment; fire; and environmental. Following these procedures 65 references remained. Copies of all these were obtained. At this stage it became clear that the literature on human exposures to asbestos from fires was limited, ie only two papers were identified. These two papers (Bridgman, 2001; Landrigan et al, 2004) provided some useful information on the levels of asbestos in air and in deposited material following major incidents, as discussed in Section 3.

In the absence of specific epidemiological studies on the health impact of asbestos exposure from fires, the scope of the literature review was widened to include consideration of epidemiological studies on the health impacts from any environmental exposures to asbestos, and, in particular, to review all quantitative risk data in this area. It was considered that this shift in the objective did not necessitate a new literature search, as the initial search criteria and exclusion and inclusion criteria were also valid for this objective. The 65 papers were then reviewed in turn to establish their relevance to the objective of summarising quantitative risk factors for environmental exposures to asbestos. To this end each paper was examined and put into one of the following three categories:

Category 1 - Epidemiological study relating to environmental asbestos exposures with clear quantitative asbestos exposure levels (eg in terms of f/ml)

Category 2 - Epidemiological study relating to environmental asbestos exposures with no clear quantitative asbestos exposure levels (eg exposure may be related to a surrogate, such as distance from an asbestos plant)

Category 3 - Non epidemiological study (eg case series), or epidemiological study on purely occupational or paraoccupational[*] exposures, or epidemiological study on environmental asbestos exposures with insufficient quantitative data (ie no risks provided).

The 49 papers in category 3 were excluded from further consideration. Sixteen papers were included in categories 1 and 2 (12 original articles and 4 reviews). One of these papers was excluded as it simply presented preliminary results of a study addressed in

[*] This period was chosen as it represented an update to an earlier literature search (Saunders, 1996).

[*] Domestic exposures to asbestos arising from household members' occupational exposures. Exposure can arise from asbestos dust brought home on clothing, for example.

one of the other papers. The review papers were not considered further at this stage thus a total of 11 original study papers were examined in detail.

Examination of the 16 papers in categories 1 and 2 revealed a further 12 papers that might be of interest. Although strictly outside the bounds of the original search criteria copies of these were also obtained. Of these 5 were excluded on the basis of the earlier exclusion criteria, or because they related to studies already addressed in other papers. The remaining 7 papers were examined in detail.

Although peritoneal mesothelioma was not excluded from the literature review none of the final set of papers examined in detail related to this form of mesothelioma. This reflects, in general, the much lower incidence of the disease in comparison to pleural mesothelioma.

The studies identified were either case-control or ecological and related to two general environmental sources of asbestos exposure: asbestos exposure from the presence of high concentrations of naturally occurring asbestos in the local environment; and asbestos exposure from industrial operations such as mining, shipping and product manufacture. Details of each of the papers reviewed are provided in Appendix A. Where studies derived relative risks (RRs), odds ratios (ORs) or similar quantitative risk related quantities these are summarised in Tables 6 (pleural mesothelioma) and 7 (lung cancer).

None of the studies considered included individual specific asbestos exposure levels. However, two did include broad exposure estimates for groups of individuals (Hansen et al, 1998; Camus et al, 1998). None of the studies attempted to link risks to absolute asbestos exposure levels. Some of the studies attempted to investigate how risks varied with asbestos exposure but generally used a surrogate, primarily distance from source, to represent exposure.

Summaries of the results in relation to pleural mesothelioma and lung cancer are presented separately below. The standard of the reported studies was variable, as is discussed on an individual study basis in Appendix A. There are, however, some general issues that it is useful to identify at this stage.

Environmental vs occupational exposure

One of the major methodological issues with epidemiological studies intended to explore the relationship between environmental asbestos exposures, particularly from local industrial sources, and lung cancer or mesothelioma incidence is the need to exclude those occupationally exposed from the study. This is not straightforward and can lead to errors in the resulting risks. For example, if in a population there are 100 mesothelioma cases and 25 are identified as occupationally exposed then it is assumed that the remaining 75 are environmentally or, perhaps, paraoccupationally exposed and these cases are considered further in the study. It is thus clear that any errors in the numbers identified as occupationally exposed has implications for the assessment of risks to other groups. In most of the studies cases were removed from the analysis if there was any indication of occupational exposure. In some instances this may not be a conservative approach. For example, an individual may have spent a very short period of time potentially occupationally exposed and a large part of their lives environmentally exposed but they would, under the criteria used in these studies, be removed from

further consideration. The implicit assumption is that occupational exposures are always significantly greater than environmental exposures. This may be a reasonable assumption for some cohorts but was not discussed as such in any of the studies. The potential consequences, ie that the number of environmental exposure induced cases may be underestimated, were similarly not addressed.

Use of information from proxies

One possible source of bias in the case control studies arises from the use of proxies to obtain information on cases (as in most of the studies the cases are deceased), in comparison to the controls who are generally interviewed themselves. The proxies are generally spouses or other family members. Information on residence is generally reasonably robust against this potential bias and can also be relatively easily checked against other sources, however, occupational histories may be more subject to recall error by a proxy. Cognisant of this potential problem one study looked specifically at the impact of using proxy information (Magnani et al, 2000) and found that in that case the impact was minimal. Other studies sought to confirm information by considering other sources such as factory rosters and union lists etc (eg Magnani et al, 2001).

Distance based exposure classification schemes

The classification of environmentally exposed individuals was often done using very crude criteria. For industrial sites the criterion was generally location within a certain distance of an industrial asbestos source (mine, cement factory etc.). Some studies considered different categories, eg 'high probability of exposure' vs 'low probability of exposure'. Thus in one study (Magnani et al (2000)) individuals were classified as 'high probability of exposure' if they lived within 2000 m of asbestos mines, asbestos cement plants, asbestos textile plants, shipyards or brake factories. A number of the studies also considered distance bands. Using distance bands as a surrogate for exposure is a reasonable assumption, if, firstly there is one predominant source and secondly if atmospheric dispersion, or perhaps some other generally uniform dispersion mechanism dominates. In general neither the rationale for using distance bands nor the reasoning behind the choice of bands was discussed.

Smoking and other confounding factors

One of the difficulties in undertaking epidemiological studies for lung cancer incidence is the importance of confounding factors, in particular smoking. In some of the ecological studies it was noted that the exposed and non-exposed populations had similar smoking habits but in others this was not discussed in detail. In some of the studies it was stated that the confounding factor of smoking had been taken into account in the derivation of risks but is was rarely clear how this had been done. In one ecological study on lung cancer from environmental exposures from an asbestos cement factory (Magnani and Leporati, 1998) the authors concluded that the study did not show a significant increase in mortality from lung cancer for the environmentally exposed population. However, they noted that the contribution of cigarette smoking could not be evaluated as individual information on smoking habits was not available. Any discussion of general smoking habits for the environmentally exposed and non-exposed control populations would have been very useful, without this it is not possible to draw significant conclusions from the data. For ecological studies it is important that the comparison population has similar characteristics to the study population, or that any differences are taken into account

appropriately. In general the studies considered addressed the issue of matching in relation to age and gender distributions adequately.

4.2.2.1 Pleural mesothelioma

Nine of the studies reviewed provided quantitative information on the relationship between pleural mesothelioma and environmental asbestos exposure. Information on these studies, including any relative risks (RRs), odds ratios (ORs) or similar quantitative risk related quantities is summarised in Table 6.

Two of the studies related to high concentrations of naturally occurring asbestos in local materials that were used in building materials. These were a case control study in New Caledonia (Luce et al, 2000) and an ecological study in Turkey (Metintas et al, 2002). Three of the studies related to environmental exposures resulting from residence in asbestos mining regions. Two of these were ecological studies, one considered the impact of environmental exposures on the non-occupationally exposed residents of the Australian crocidolite mining town of Wittenoom (Hansen et al, 1998) and the other the impact of exposures on female residents in two chrysotile mining areas in Quebec (Camus et al, 1998). The third mining related study was a multicentre case control study in South Africa (Rees et al, 1999). The remaining four studies concerned environmental exposures from industrial plants processing or manufacturing ACM. One of these was an ecological study considering the impact of an asbestos plant in Manville, New Jersey (Berry, 1997). The other three were case control studies. One was a multicentre study that considered various industrial sources in Italy, Spain and Switzerland (Magnani et al, 2000) and another related to an asbestos cement plant in Italy (Magnani et al, 2001). The final study considered the impact of various industrial asbestos sources in Yorkshire (Howel et al, 1997).

The number of cases in most studies was small, reflecting the generally low mesothelioma risk. The best estimate ORs or RRs derived in the studies are all greater than unity, thus implying an increased risk of mesothelioma in the exposed population. However, the reported confidence intervals (CIs) are very wide in many of the studies. For example, in the case-control study investigating risks from exposure by various industrial asbestos sources in a number of health districts in Yorkshire (Howel, 1997) the CIs intervals are so wide that reduced or greatly increased ORs could not be ruled out. In about half of the studies the CI range is between one and two orders of magnitude. That all the studies suggested an association may also be an indication of potential publication bias.

The highest estimated risks are from exposure to naturally occurring asbestos deposits that are or were used to make whitewashes and other building materials in New Caledonia and Turkey (Luce, 2000; Metintas, 2002). This would appear reasonable given the potential, under such circumstances, for relatively high exposures from an early age.

Table 6 Epidemiological studies on environmental exposure to asbestos and pleural mesothelioma (PM)

Country	Source of Exposure	Fibre Type*	Study Design+	Study details	Cases	Relative Risk or Odds Ratio#	95% CI**	Reference
Italy	Exposure from asbestos cement factory in town	UM	CC	Retrospective follow-up study. All PM cases in Local Health Authority 1987-1993. cases matched by birth date, sex and age of death to two controls, or 4 if < 60 years. Controls randomly selected from residents.				Magnani et al (2001)
				Odds ratios for residence within various distances of plant				
				< 500 m from plant	5	27.7	3.1 - 247.7	
				500 – 1499 m from plant	41	22.0	6.3 - 76.5	
				1500 – 2499 m from plant	9	21.0	4.9 - 91.8	
				> 2500 m from plant	4	11.1	1.8 - 67.2	
Italy, Spain and Switzerland	Various occupational, domestic and environmental. All environmental from industrial sources eg asbestos cement plants, shipyards etc.	UM	CC	Multicentre study. Cases diagnosed 1995 – 1996. Control group twice the number of cases. Industrial hygienists classified exposure based on questionnaire results.				Magnani et al (2000)
				Odds ratio for a high probability of environmental exposure (defined as living within 2000 m of asbestos mines, factories etc.)	17	11.5	3.5 - 38.2	
New Caledonia	Use of naturally occurring asbestos in whitewash	A	CC	Cases diagnosed 1993-1995. Control group frequency matched by sex and age to expected distribution of cases.				Luce et al (2000)
				Odds ratio for use of whitewash containing asbestos	14	40.9	5.15 – 325	
South Africa	Residence in mining region	UM	CC	Multicentre case control study. Two controls identified for each case				Rees et al (1999)
				Odds ratio for purely environmental exposure from residence in mining area	22	19.6	3.7 – 105	
England	General environmental	UM	CC	Cases of mesothelioma 1979-1991 in four				Howel et al (1997)

	exposures from various industrial sources			health districts in Yorkshire, total 185				
				Odds ratios for cases identified as solely exposed to asbestos through their residence	6	1.6 to 6.6	++	
Turkey	Use of naturally occurring asbestos in building materials, eg whitewash and plaster	UM	Ec	Observation period 10 years (1990-2000)				Metintas et al (2002)
				Relative risks				
				Men	12	88.3[##]	Not given	
				Women	12	799[##]	Not given	
Canada	Residence in asbestos mining area in Quebec	C	Ec	Mortality among women in 2 asbestos mining areas in the province of Quebec was compared with mortality among women in 60 control areas (1970-1989)				Camus et al (1998)
				Age standardised mortality ratios (SMR) for deaths from cancer of the pleura	7	7.63	3.06 - 15.73	
Australia	Background environmental exposure in mining town	A	Ec	Retrospective follow-up study. All 4659 former residents of Wittenoom who lived there between 1943 and 1993 for at least a month and were not directly employed in the industry were followed up. Exposure quantified in terms of duration of residence and estimated quantitative exposures				Hansen et al (1998)
				Relative risks				
				Duration of residence (months)				
				1-11 (comparison case)	3	1		
				12-59	10	2.6	1.4 - 4.7	
				60+	14	6.7	2.0 - 22.2	
				Exposure intensity (fibre/ml - years)				
				1-7 (comparison case)	7	1		

				7-20	11	1.9	1.2 - 3.1	
				20+	9	3.6	1.3 - 9.5	
USA	Environmental exposure from asbestos plant in Manville, New Jersey	UM	Ec	Cases of mesothelioma in period 1978-1990 in the town of Manville, Somerset County and New Jersey. Cases occupationally exposed not considered				Berry (1997)
				Relative risks				
				Population of Manville (in comparison to New Jersey population)				
				Men	16	10.1	5.8 – 16.4	
				Women	8	22.4	9.7 – 44.2	
				Population of Somerset County (in comparison to New jersey)	47	1.9	1.4 – 2.5	
				Men	11	2.0	1.0 – 3.6	
				Women				

Notes:

∗ Predominant type of fibre: A – amphiboles; C – Chrysotile; UM – unspecified and mixed.

+ Study design: CC - case control; Ec - ecological study.

Relative risk, odds ratio or other quantitative risk quantity. Details of type indicated under study design.

** Confidence interval.

++ So wide that reduced or greatly increased odds could not be ruled out.

In comparison to world background incidence rates.

Table 7 Epidemiological studies on environmental exposure to asbestos and lung cancer

Country	Source of Exposure	Fibre Type*	Study Design+	Study details	Cases	Relative Risk or Odds Ratio#	95% CI**	Reference
New Caledonia	Use of naturally occurring asbestos in whitewash	A	CC	Cases diagnosed 1993-1995. Control group frequency matched by sex and age to expected distribution of cases				Luce et al (2000)
				Odds ratios for us of whitewash containing asbestos				
				Men	35	0.89	0.51 - 1.54	
				Women	21	2.51	1.01 - 6.22	
South Africa	Residence in areas where asbestos shipped (moderately polluted asbestos area) and residence in mining area (termed heavily polluted asbestos area)	UM	CC	Cases between 1993 and 1995. 288 men and 60 women with lung cancer and 183 male and 197 female controls were interviewed. Asbestos exposure classified according to residence near asbestos distribution areas (termed moderately polluted) and mining areas (termed heavily polluted)				Mzileni et al (1999)
				Odds ratios for residence in areas with varying asbestos pollution levels				
				Moderately polluted		2.1		
				Men	38	1.1	1.0 - 4.4	
				Women	4		0.3 - 3.9	
				Heavily polluted		2.8		
				Men	10	5.4	0.7 - 10.4	
				Women	6		1.3 - 22.5	
China	Exposure from naturally occurring asbestos in the environment and use in manufacture of stoves	A	Ec	Three ecological studies in affected area. I Obsevation period 7 years (1977 – 1983) II Observation priod 9 years (1987-1995) III (subset of II)				Luo et al (2003)

				Relative risks for the three studies				
				I	4	6.67	Not given	
				II	21	2.14	1.07 - 4.28	
				III	6	3.02	Not given	
Italy	Exposure from asbestos cement factory in town	UM	Ec	Cases dying between 1989 – 1995.	168			Magnani and Leporati (1998)
				Showed no increase in mortality from lung cancer for population not occupationally or paraoccupationally exposed in the town in comparison with rates for the entire region.				
Canada	Residence in asbestos mining area in Quebec	C	Ec	Mortality among women in 2 asbestos mining areas in the province of Quebec was compared with mortality among women in 60 control areas (1970-1989)				Camus et al (1998)
				Age standardised mortality ratios (SMR) for deaths from cancer of the lung or bronchus	71	0.99	0.78 - 1.25	
Austria	Two towns considered, one where asbestos processing had been carried out and another with naturally occurring tremolite contamination	UM	Ec	Lung cancer mortality rates in the two towns were compared with those for five reference populations: the Austrian population; the population of the relevant province; the population of the relevant district; and the subpopulation of Austria living in communities belonging to the same community class size; and the subpopulation of Austria living in communities with similar agricultural characteristics.	53			Neuberger et al (1984)[++]
				No evidence of lung cancer excess detected				
USA	Exposure from amosite asbestos plant in New Jersey	A	Ec	Mortality of men in vicinity of asbestos plant compared with that in a similar sized neighbourhood several miles away. Asbestos workers excluded from study				Hammond et al (1979)[++]
				Relative risk	41	0.9	Not given	

Notes:

* Predominant type of fibre: A – amphiboles; C – Chrysotile; UM – unspecified and mixed.

+ Study design: CC - case control; Ec - ecological study.

Relative risk, odds ratio or other quantitative risk quantity. Details of type indicated under study design.

** Confidence interval.

++ References outside temporal scope of literature review, included for information

In three of the studies attempts were made to investigate the variation in risk with exposure. Magnani et al (2001) considered distance from the asbestos plant as a surrogate for exposure (ie it was postulated that the further individuals lived from the site the lower their exposure would be). This assumption is reasonable if there is, as in this case, one dominant source and also if the dominant asbestos environmental transport process results in dilution of exposure with distance (eg atmospheric dispersion). In the study ORs for non-occupationally exposed individuals living within various distance bands from the site (< 500 m, 500 – 1499 m, 1500 – 2499 m and > 2500 m) were determined. The ORs did not vary significantly up to a distance of 2500 m from the plant. The authors argue that the relatively high risk at significant distances suggests a role for sources other than simply atmospheric pollution direct from the plant. The use of residues in construction is suggested as one possible source.

In another study related to non-occupational exposures from a single asbestos plant in Manville, USA (Berry, 1997) RRs to those living in the town of Manville were compared with those to individuals living in the rest of the county. This indicated that risks to those in Manville were between about 5 and 10 times higher than those to individuals in the remainder of the county.

For the ecological study of non-occupationally exposed residents of the Australian asbestos (crocidolite) mining town, Wittenoom (Hansen et al, 1998), information on the time people lived in the town was collected. Relative risks were estimated as a function of residence duration. The results indicated that mesothelioma risk increased with duration of residence, with the risk for someone with > 5 years residence approximately 7 times that for someone with < 1 years residence. For the study attempts were also made to estimate the asbestos exposure of the non-occupationally exposed population. Subjects were assigned an intensity of exposure of 1.0 fibre/ml from 1943 to 1957 (when a new mill was commissioned and the town was moved), and then 0.5 f/ml between 1958 and 1966, when the mining operations ceased. Since then, interpolation between periodic surveys using personal monitors assigned exposures from 0.5 fibres (> 5 microns long) per ml of air in 1966 to 0.01 f/ml in 1992. Thus the assessment of exposure for each individual in the study was based on these general asbestos levels multiplied by residence durations during each of the periods identified. Relative risks as functions of asbestos exposure were then determined. These indicated an increased risk with exposure but not as strongly as with exposure duration. The interpretation of such results is complicated by the fact that most of the cases (24 out of 27) were also paraoccupationally exposed, ie their spouse or other family member was occupationally exposed, which could lead to exposure of other members of the household. This is also likely to be the case for many other members of the exposed cohort. This undermines to a significant extent the exposure estimates discussed above, as these are based on general environmental levels rather than those that might be encountered in the home of an asbestos worker.

No attempt was made in the Wittenoon study to link mesothelioma risks to absolute asbestos exposure levels. It is instructive, however, to consider, to the extent possible, whether the estimated risks are generally consistent with those from the review of occupational exposure risks (Hodgson and Darnton, 2000) discussed earlier. The estimated median cumulative exposure was 15.2 f/ml-years with a range from 0.53 to 40.4 f/ml-years. The majority of the population (76.4%) had an estimated exposure

below 7 f/ml-years. Only 247 people (approximately 5%) were estimated to have exposures above 20 f/ml-years.

Hodgson and Darnton (2000) (see Table 5) indicate that the best estimate mesothelioma risk for cumulative crocidolite exposure levels between 10 and 100 f/ml-years is about 400 deaths per 100,000 exposed for each f/ml-year of cumulative exposure; with up to a two fold uncertainty on this value. For a cumulative exposure of 1 f/ml-years the risk estimate is about 650 deaths per 100,000 exposed with the highest estimate 1500 and the lowest 250.

Assuming the 247 individuals with exposures above 20 f/ml-years each have an exposure of 30 f/ml-years (conservative estimate assuming a uniform distribution between 20 f/ml-years and the maximum level of 40 f/ml-years) then, using the Hodgson and Darnton best estimate risk levels, the predicted number of mesothelioma cases is 30. The actual number of cases with exposures above 20 f/ml-years was 9 (Hansen et al, 1998), which is more than a factor of 3 lower. Assuming the entire exposed population is exposed at a level of 1 f/ml-years the Hodgson and Darnton risk values predict approximately 29 mesothelioma cases. This exposure level is somewhat lower than the estimates given above but the results compare well with the actual number of cases, 27. Assuming the entire population is exposed at a level of 5 f/ml-years would result in a predicted number of cases approximately 5 times higher. Without additional information on the distribution of exposures within the population it is not possible to make judgements on which would be a more appropriate estimate. The exposure estimates clearly have some level of associated uncertainty but as this is not discussed within the paper it is not possible to comment on the significance of these to the above analysis. As indicated above there is also some uncertainty regarding the estimated exposures of the cases as these are complicated by paraoccupational exposures. It is therefore difficult to arrive at any conclusions on the basis of the above analysis given the uncertainties in the exposures.

A Canadian study was undertaken to investigate the risk of lung cancer from non-occupational exposure to chrysotile asbestos (Camus et al, 1998). Mortality among women in two chrysotile asbestos mining areas of the Canadian province of Quebec was compared with mortality among women in 60 control areas. The study was restricted to women in order to exclude most asbestos workers. It used data from death certificates. This included information on pleural in addition to lung cancers. The analysis of information on death certificates revealed 7 deaths from pleural cancer[*] giving a RR of 7.63.

For the study it was estimated that the average cumulative lifetime chrysotile exposure to the women in the two chrysotile areas was 25 f/ml-years; with an associated subjective range of 5 – 125 fibre/ml-years. It is instructive, to consider, to the extent possible, whether the estimated risks are generally consistent with those from the

[*] Camus et al state that the instances of pleural cancer (term recorded on the death certificates) suggest an excess risk of mesothelioma. However, they note that historical death certificates may reflect the incidence of mesothelioma poorly, and so intend to investigate this in more detail using hospital records.

review of occupational exposure risks (Hodgson and Darnton, 2000) discussed earlier. Hodgson and Darnton (2000) indicate that the best estimate occupational mesothelioma risk for cumulative chrysotile exposure levels between 10 and 100 f/ml-years is about 2 deaths per 100,000 exposed for each f/ml-year of cumulative exposure; with up to a three fold uncertainty on this value. An exposure of 25 f/ml-years would therefore imply a risk of 50 deaths per 100,000 exposed. An indication of the effective number of exposed individuals in the study was obtained by dividing the quoted person-years during the reference period by the duration of the reference period (ie 221,375 person-years / 19 years). This gave a total in the region of 10,000. Using the above risks from Hodgson and Darnton the predicted number of deaths from pleural mesothelioma would be in the region of 5. The number of pleural cancer cases identified in the study was 7. Assuming the pleural cancers are mesothelioma, the number of mesothelioma deaths is therefore broadly consistent with those predicted using the occupational risk factors. However, the significance of the uncertainties should not be underestimated. In addition to the uncertainties quoted above in relation to the risk factors and the exposure estimates, Camus et al (1998) also indicate that they used information from death certificates in the study, which they considered was adequate for lung cancer, but which they considered were not sufficient for the study of mesothelioma due to difficulties in diagnosis. Others have also noted that some of the mesothelioma cases identified by Camus may have arisen as a result of occupational exposure (Churg, 1998).

The results of the two studies discussed above can, with the above caveats, be considered to be very broadly consistent with Hodgson and Darnton (2000).

4.2.2.2 Lung cancer

Seven of the studies reviewed provided information on the relationship between lung cancer and environmental asbestos exposure. Information on these studies, including any RRs, ORs or similar quantitative risk related quantities is summarised in Table 7.

Three of the studies considered areas with high concentrations of naturally occurring asbestos in the local environment. These included a case control study in New Caledonia (Luce et al, 2000), where naturally occurring ACM were used in building materials, notably whitewash, and two ecological studies, one in China (Luo et al, 2003) and one in Austria (Neuberger et al, 1984). Two studies related to environmental exposures resulting from residence in asbestos mining or shipping regions. One of these was an ecological study considering the impact of environmental exposures on female residents in two chrysotile mining areas in Quebec (Camus et al, 1998) and the other a case control study considering exposures in mining areas and areas where asbestos was shipped in South Africa (Mzileni et al, 1999). The remaining studies, all ecological, investigated the impact of environmental exposures from particular industrial plants processing or manufacturing ACM in Austria (Neuberger et al, 1984), Italy (Magnani and Leporati, 1998) and the USA (Hammond et al, 1979).

The results of the lung cancer studies are less consistent than those for pleural mesothelioma. This is as expected given the importance of confounders, especially smoking. For four of the seven studies considered it was stated that no excess risk of lung cancer was detected in the exposed population (Magnani and Leporati, 1998; Neuberger et al, 1984; Hammond et al, 1979; Camus, 1998). Only three of the studies

indicated, as a best estimate, an increased risk of lung cancer in the exposed population. The highest RR of 6.7 was a result of exposure from naturally occurring asbestos present in a region of China (Luo et al, 2003). The New Caledonia study (Luce et al, 2000) identified an increased risk from the use of asbestos containing building materials for women (OR 2.51, 95% CI 1.01 – 6.22) but not for men (OR 0.89, 95% CI 0.51 – 1.54). The South African study (Mzileni, 1999) derived ORs ranging from 1.1 (95% CI 0.3 – 3.9) to 5.4 (95% CI 1.3 – 22.5) for individuals in asbestos mining or shipping areas, with risks higher in the more heavily polluted mining areas.

Only one of the studies, Camus et al (1998), included, albeit limited, asbestos exposure estimates for the exposed populations. This study of women living in two chrysotile mining areas in Quebec (Camus et al, 1998) generated an age standardised mortality ratio for the exposed population, in comparison to the unexposed, of 0.99 (ie no excess cancer risk) with a range of 0.78 to 1.25. For the study an average cumulative lifetime chrysotile exposure of 25 f/ml-years was estimated; with an associated subjective range of 5 – 125 fibre/ml-years. Hodgson and Darnton (2000) indicate that the best estimate lung cancer risk for cumulative chrysotile exposure levels between 10 and 100 f/ml-years is about 5 excess deaths per 100,000 exposed for each f/ml-year of cumulative exposure. An exposure of 25 f/ml-years would therefore imply a risk of 125 deaths per 100,000 exposed. An indication of the effective number of exposed individuals in the study was obtained by dividing the quoted person-years during the reference period by the duration of the reference period (ie 221,375 person-years / 19 years). This gave a total in the region of 10,000. Using the above risks from Hodgson and Darnton the predicted number of excess deaths from lung cancer would be in the region of 13. Camus et al (1998) indicate that the estimated number of excess deaths in the population studies is between 0 and 6.5. The number of cases is therefore significantly lower than that predicted using the standard best estimates from Hodgson and Darnton. However, Hodgson and Darnton indicate that the best estimate excess lung cancer risks represent an average for a population with a past pattern of smoking similar to that of older British men and that for non-smokers the risk would be between a third and a sixth of those quoted. Camus et al indicate that smoking levels within the exposed population were slightly lower than those in the non-exposed populations. This factor may go some way to explaining the difference.

4.2.3 Summary

The studies reviewed in Section 3 indicate that asbestos exposures of members of the public during and in the immediate aftermath of a fire involving asbestos material are expected to be minimal, certainly below industrial control levels. The studies indicate that in the longer term, as long as appropriate clean-up has taken place, the exposures will return to effectively typical background levels.

As noted in Section 3, a study of the levels of asbestos in air following the destruction of the World Trade Center that commenced three days after the event when the dominant pollution sources were smouldering fires with occasional flareups and resuspension of dust, indicated that there were no 8 hour time-averaged asbestos exposures to workers above 0.1 f/ml. On the basis of the information in Section 3, this can be considered as a very conservative upper estimate of the level of exposure of a member of the public

following a fire involving ACM in the UK. Assuming the exposure lasts for a period of 2 days, again a conservative assumption, this would result in a cumulative exposure of < 0.0006 f/ml-years.

Estimating the mesothelioma and lung cancer risks from this low level of exposure is not straightforward. The review of environmental epidemiological studies (Section 4.2.2) does not provide an appropriate exposure response model for this. It should be noted that the limited available information on exposures for these studies indicated that exposures were relatively high, approaching and exceeding occupational exposure levels in some cases. The environmental exposures were also generally received over long time periods.

As discussed in Section 4.2.1, exposure response models have been developed from occupational studies, but these are based on groups with longer exposures (months to years) at significantly higher asbestos levels. However, in the absence of any other more directly relevant models, the levels of exposure from a fire are discussed below in relation to the extrapolation of risks from occupational studies to lower levels of exposure. In particular, the exposures are discussed in relation to the analysis provided by Hodgson and Darnton (2000) as this is one of the most recent reviews and also attempts to address uncertainties arising from both the original study data and the extrapolation process.

Hodgson and Darnton (2000) indicate that for cumulative exposures of 0.005 f/ml-years (ie approximately an order of magnitude higher than the level of the above conservative upper estimate of exposure) and lower, only risks of mesothelioma need be considered as those for lung cancer as insignificant[*] (possibly zero). They note that the absolute levels of mesothelioma risk are low but the quantitative uncertainties are considerable. The mesothelioma risks from chrysotile are also considered insignificant at this exposure level. Mesothelioma risks for crocidolite at 0.005 f/ml-years are quoted as best estimate 10 deaths per 100,000 exposure (highest arguable estimate 55) with the best estimate falling to insignificant levels at 0.0002 f/ml-years. For amosite the best estimate risk at 0.005 f/ml-years is about 2 deaths per 100,000 exposed, with the highest arguable lifetime risk 15 per 100,000. On the basis of this analysis it can be concluded that the health risks from asbestos exposures from large scale fires are minimal if appropriate clean-up operations are undertaken. It is clear, however, that because of the different levels of risk it is important to establish the types of asbestos present following any fire.

The uncertainties in the above approach must, however, be recognised. Hodgson and Darnton's analysis is based upon the extrapolation of estimated risks from industrial exposures at relatively high levels (> 10 f/ml-years) for periods greater than a month. There is some evidence that mesothelioma can result from short term exposures. A study of the incidence of malignant mesothelioma in Australia indicated that the shortest exposure duration of any of the cases was 16 hours (waterside worker loading crocidolite fibres which would have resulted in a very high exposure) and that 3% of the cases had asbestos exposures of less than three months (Leigh and Driscoll, 2003).

[*] Hodgson and Darnton (2000) define an insignificant risk as a lifetime risk of less than 1 in 100,000.

Another study investigating the link between short-term amosite exposure and mesothelioma reported a case occurring with only one months exposure (Seidman, 1979). The study of non-occupationally exposed residents of Wittenoom (residence > 1 month) indicated that cases of mesothelioma had arisen in subjects with durations of crocidolite exposure as short as 2 months and estimated cumulative exposures as low as 0.53 f/ml-years. In general it is expected that the use of such a model extrapolating from occupational exposures will not significantly underestimate the risk presented

Other studies have been undertaken to estimate the risks of asbestos exposures from fires and other serious incidents potentially giving rise to short term asbestos exposures. These also indicate minimal risks.

Following a major factory fire in Tranmere, Merseyside on 22 September 1994, which deposited asbestos containing fall-out in an urban area, Wirral Health Authority commissioned a study of the health consequences and lessons learnt from the incident (Bridgman, 1996). Bridgman concluded that the risk was almost entirely due to fire fallout of chrysotile in asbestos bitumen paper covering the factory roof. No measurements of asbestos in air concentrations were made, but a number of estimates were made on the basis of details of the incident. The maximum estimated cumulative exposure was 0.008 f/ml-years - a level of 0.5 f/ml (the industrial control level at the time) for two days. Using a linear non-threshold model (Doll and Peto, 1985) for lung cancer from chrysotile exposure a lifetime risk of lung cancer of less than one in a million was estimated. Bridgman also concluded that the risk of mesothelioma was low. This analysis clearly required the use of an exposure response model extrapolated to exposure levels below those of the occupational data from which they were derived. Bridgman notes the uncertainties this approaches involves and that some authors have argued that there is a threshold for exposure, however, in the absence of confirmation of a threshold the appropriate public health position to take is that there is no threshold and that furthermore there is some agreement that extrapolating such models possibly overestimates risks from low cumulative exposures but is unlikely to underestimate them (Weill et al, 1986).

A study to estimate the lifetime risk of asbestos related cancer for residents of Lower Manhattan attributable to asbestos released into the air by the 9/11 attack on New York City's World Trade Center has been undertaken (Nolan et al, 2005). A worst case cumulative chrysotile exposure of 0.065 f/ml-years was estimated on the basis of samples of settled dust taken during the six days following 9/11 and air sampling information. Assessments of the risk were undertaken using two risk models: Hodgson and Darnton (2000) and EPA's aggregate risk model (as defined in Nolan et al (2005)). On the basis of the study the authors concluded that the exposure to asbestos in ambient air has resulted in no more than a negligible increase in the risk of cancer for the residents of Lower Manhattan.

On the basis of the above it is concluded that the risks from asbestos exposures from fires are minimal if appropriate clean-up occurs.

It should be noted that the issue of risks from low levels of asbestos exposure, including the extrapolation of occupational epidemiological study results down to low levels, is of current interest in the UK. For example, the Health and Safety Commission's Working

Group on Action to Control Chemicals (WATCH) are currently exploring this issue (WATCH, 2007). The Environment Agency is also considering risks from low level asbestos exposures in relation to the derivation of appropriate guidance on the clean-up of land contaminated with asbestos (Grosso, 2007).

5 INCIDENT RESPONSE AND PLANNING

Actions taken by Local Authorities and other organisations when responding to large scale fires involving asbestos will be very similar to those taken for other acute chemical incidents. Similarly, plans for responding to asbestos fires will have much in common with those for other chemical incidents.

Guidance on the key elements for preparing emergency response plans for chemical incidents and responding to them are available from a large number of sources. Such guidance covers many issues, such as communications, structures, authorities, organisation of responders etc., which are beyond the scope of this document. The guidance in this section concentrates on those aspects that are either specific to asbestos incidents (eg, arrangements for clean-up of asbestos debris), or relate to aspects of incident response that may require more focus when asbestos is involved (eg, the need for appropriate and timely public information, advice and reassurance, given general public concerns regarding asbestos). These aspects of planning and response are discussed below.

Following a major factory fire in Tranmere, Merseyside on 22 September 1994, which deposited asbestos containing fall-out in an urban area, Wirral Health Authority commissioned a study of the health consequences and lessons learnt from the incident (Bridgman, 1996). This very thorough study made a number of recommendations relating to the response to such incidents and, a decade on from publication, it was considered appropriate to revisit those recommendations relevant within the context defined above.

One of Bridgman's main recommendations was that '*There should be a written district policy on handling asbestos incidents, and staff should receive training on the policy'.* It is considered that this recommendation is still valid, and that written plans and training are important in minimising the potential impact of such incidents. It is therefore recommended that Local Authorities have a written policy for dealing with asbestos fires. This might be a full and detailed asbestos fire specific plan or simply additional guidance in addition to a generic incident plan covering only those issues pertinent to asbestos. Unfortunately the limited information available tends to suggest that the development by Local Authorities of plans for dealing with large scale fires involving ACM, if it happens at all, is not common practice (Camborne, 2006; Kowalczyk, 2007).

HPA staff, including those from precursor organisations, have been involved in the public health response to a number of large scale fires involving ACM (eg Hodgson, 2006; Kirkpatrick et al, 2006; Packard and Welch, 2002; Sedgwick, 2003; Stanton, 2002;

Stewart and MacDonald, 2002; van den Bosch, 2005). Experience from such incidents is a useful input to the development and implementation of appropriate incident response plans.

5.1 Clean-up and waste disposal

Following a large scale fire involving asbestos there will be a requirement to clean-up the asbestos debris. Examples of asbestos containing products encountered in fires include roof tiles and asbestos-bitumen roof coatings (see examples in Figure 2). The clean-up will generally involve manual pick-up of larger fragments and possibly other processes such as wet mechanical clean-up using road sweepers.

The Health and Safety Executive (HSE) has produced extensive guidance on working with asbestos (www.hse.gov.uk/asbestos). This includes guidance on how asbestos removal and clean up work should be carried out (eg HSE, 2006a,b).

Handling asbestos materials is a specialist task requiring appropriate training and equipment, including personal protective equipment (PPE). It is important that any plans for responding to such incidents include arrangements for the provision of such staff and equipment either in-house or from specialist companies.

There is the potential for the workers involved to be exposed during the process. The clean-up process needs to be defined and documented and subject to a risk assessment. With rational precautions including protective clothing and the use of 'wet' collection processes there is no reason why clean-up should present any significant risk. Guidance on the protection of workers can be obtained from the HSE. In a well-organised response it should be possible for all significant fall-out to be removed within 48 hours.

If the clean-up is performed in an appropriate manner the level of asbestos contamination in the affected area will be minimal. Thus the potential for long-term environmental exposures and thus the associated risk will be minimal.

In the context of clean-up and disposal, it should be noted that asbestos wastes are defined are hazardous wastes under the Hazardous Waste (England and Wales) Regulations 2005 and the List of Wastes (England) Regulations 2005. These came into force on 16 July 2005 replacing the Special Waste Regulations 1996. The Regulations set out procedures to be followed when disposing of, carrying and receiving hazardous waste. Technical guidance on the interpretation of the definition and classification of hazardous waste is provided by the Environment Agency (http://www.environment-agency.gov.uk/subjects/waste/1019330/1217981/).

Figure 1 Asbestos containing fallout from large scale industrial fire (Bridgman, 1994) (Published with the permission of S Bridgman and Wirral MBC)

5.2 Sampling and air monitoring

During and following a fire in which it is suspected that ACM are involved it may be necessary to take samples and have them analysed to confirm the presence of asbestos and in some cases the type of asbestos. Chrysotile asbestos is more common and less hazardous than amphibole types such as crocidolite and amosite. If the presence of amphiboles is suspected then it may be important to establish if this is in fact the case and at what level. It needs to be recognised, however, that the analysis of samples can take some time and that it is often necessary to take action before the presence and type of asbestos is confirmed.

In general, as discussed earlier in this report, air sampling carried out following asbestos fires will not reveal significant levels of asbestos fibres. Therefore, in many cases it will not be necessary to carry out such monitoring. Monitoring may however be appropriate for large incidents for public reassurance purposes. This is a decision that needs to be taken on a case by case basis.

The Asbestos Regulations (CAR, 2006) require any analysis of the concentration of asbestos in air to be measured in accordance with the 1997 WHO recommended method.

Because of the potential need for sampling and analysis, emergency plans should identify laboratories that are competent to undertake emergency analysis.

The above recommendations are consistent with those made by Bridgman (1996) in relation to the analysis of samples. He noted that, following the fire at Tranmere the first laboratory to analyse samples required a second opinion to definitively identify asbestos and it also took many days to get quantitative results that the amosite present was only present in trace amounts. No air asbestos fibre concentration measurements were made. Bridgman made the following recommendations: emergency plans should identify twenty-four hour call-out arrangements with laboratories with the required experience and competence to undertake emergency analysis; air fibre or asbestos levels should be measured at the time of an incident if needed for public reassurance; and quantitative analysis on all samples of fallout should be received by those managing an incident as soon as possible.

5.3 Information

Providing appropriate and timely information to members of the public is a key element of the response to any chemical incident. However, given the public perception of risks from asbestos it is especially important for such incidents. Depending upon the incident this might include information on the low level of risks and guidance on what to do if individuals find pieces of asbestos materials, for example in gardens. It is important that information is provided to everyone in the potentially affected area. Also information on actions being taken, eg clean-up and contact telephone numbers.

Bridgman was critical of various elements of public communications following the Tranmere fire. He noted that nearly fourteen hours elapsed between the Fire Brigade

being called to the fire and the first press release. Six hours elapsed between the first public query to Wirral Borough Council Environmental Health Department, and the first press release. The public were not told that only trace amounts of amosite were found in fallout samples. Dedicated public phonelines were not used to inform the public, and phonelines were staffed largely during office hours. Conflicting messages on health risks were reported to the public from public and other agencies. The main recommendation made by Bridgman to reduce such problems was for the development of written district policies and plans. There are, however, examples of good practice, including, for example, the prompt issue of press releases, the use of helpline numbers and the distribution of pamphlets in the affected area for example from fire involving ACM at a school in Newhaven in 2005 (Lewes District Council, 2005).

In relation to the public perception of risk, Bridgman concluded that the determinants of public risk perception are complex and that, in general, public perception of asbestos risk is greater than the reality. He noted that public fear and anxiety may lead to ill-health but that the gap between perceived and objective risk of asbestos can only be closed by mutual communication. Bridgman recommended in this context that – 'research is needed on how best incident teams should measure and manage perceived health risk in acute environmental incidents'.

6 CONCLUSIONS AND RECOMMENDATIONS

Large scale fires involving asbestos containing materials (ACM) are a relatively common occurrence in the UK and can cause significant public concern (Section 1).

A number of factors mitigate against significant exposures of members of the public following a fire involving ACM. These include the following: not all the ACM present may be involved in the fire; fibres may be entrapped, in larger pieces of material etc.; respirable fibres will be a fraction of the total released; some fibres may be 'denatured' at the temperatures involved; atmospheric dispersion and deposition (particularly as a result of rain) will reduce concentrations; the duration of exposure will be short. (Section 3)

The available evidence indicates that asbestos exposures of members of the public following fires involving ACM will be very small if appropriate clean-up operations are undertaken. (Section 3)

There is no direct evidence of long-term health risks from fires involving ACM, although the literature in this area is limited. Considering the available evidence on asbestos exposures from fires involving ACM in the context of the results of epidemiological studies of occupational and environmental asbestos exposures it is concluded that the risks of long-term health risks (mesothelioma and lung cancer) are minimal if appropriate clean-up occurs. It is recognised that this analysis involves the extrapolation of exposure response models developed from occupational studies of populations exposed for longer periods at significantly higher asbestos concentration levels. However, it is considered that this approach is reasonable and unlikely to underestimate

the risks. This conclusion is in agreement with other similar studies in this area. (Section 4)

The majority of asbestos encountered in such incidents will be chrysotile. The type of asbestos is a major consideration as the exposure specific risk of mesothelioma is broadly in the ratio 1:100:500 for chrysotile (white), amosite (brown) and crocidolite (blue) respectively. Identification of the asbestos type is, therefore, of great importance.

To mitigate the impact of such fires it is recommended that all Local Authorities have a written policy for dealing with large scale fires involving ACM. This might be a full and detailed asbestos fire specific plan or simply additional guidance in addition to a generic incident plan covering only those issues pertinent to asbestos. (Section 5).

Some members of the public perceive a greater risk from large scale fires involving asbestos than is actually the case and this needs to be taken into consideration when devising and issuing public warnings. (Section 5).

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APPENDIX A

Risk of mesothelioma and lung cancer from environmental exposure to asbestos – a literature survey

A1 INTRODUCTION

A review of the literature on acute human exposure to asbestos from fire incidents and the possible health effects resulting from such exposure was undertaken. The search criterion was to identify papers with at least one word from each of the following two keyword groups:

Group 1: asbestos, mesothelioma, lung cancer, fibrosis

Group 2: environment, short-term, fire, non-occupational

The review was also restricted to English Language papers and humans. The following databases were searched: BIDS; Web of Science; Medline; and Embase. The search covered the period from 1996 to February 2005[*]. This identified 152 papers. The abstracts from these were considered. Papers were excluded from further consideration if they related solely to occupational exposures. Papers were included if they referred to one of the following: asbestos release; exposure estimate; health assessment; fire; and environmental. Following these procedures 65 references remained. Copies of all these were obtained. At this stage it became clear that the literature on acute human exposures to asbestos from fires was limited, ie only two papers were identified. These two papers provided useful information on the levels of asbestos in air and in deposited material following major fires, as discussed in Section 3 of the main text, but did not provide any information on actual health effects resulting from the fires.

In the absence of specific epidemiological studies on the health impact of asbestos exposure from fires, the objective of the literature review was expanded to include consideration of all epidemiological studies on the health effects from environmental exposures to asbestos, and, in particular, to review all quantitative risk data in this area. It was considered that this shift in the objective did not necessitate a new literature search as the initial search criteria and exclusion and inclusion criteria were also valid for this objective. The 65 papers were then reviewed in turn to establish their relevance to the objective of summarising quantitative risk factors for environmental exposures to asbestos. To this end each paper was examined and put into 1 of the following 3 categories:

[*] This period was chosen as it represented an update to an earlier literature search (Saunders, 1996).

Category 1 - Epidemiological study relating to environmental asbestos exposures with clear quantitative asbestos exposure levels (eg in terms of f/ml).

Category 2 - Epidemiological study relating to environmental asbestos exposures with no clear quantitative asbestos exposure levels (eg exposure may be related to a surrogate, such as distance from an asbestos plant).

Category 3 - Non epidemiological study (eg case series), or epidemiological study on purely occupational or paraoccupational* exposures, or epidemiological study on environmental asbestos exposures with insufficient quantitative data (ie no risks provided).

The 49 papers in category 3 were excluded from further consideration. Sixteen papers were included in categories 1 and 2 (12 original articles and 4 reviews). One of these papers was excluded as it simply presented preliminary results of a study addressed in one of the other papers. The review papers were not considered further at this stage thus a total of 11 original study papers were examined in detail (see Section A2.1).

Examination of the 16 papers in categories 1 and 2 revealed a further 12 papers that might be of interest. Although strictly outside the bounds of the original search criteria copies of these were also obtained. Of these 5 were excluded on the basis of the earlier exclusion criteria, or because they related to studies already addressed in other papers. The remaining 7 papers were examined in detail (see Section A2.2).

The studies examined in detail were either case-control or ecological studies and related to two general sources of environmental asbestos exposure:

- high concentrations of naturally occurring asbestos in the local environment; and
- industrial operations involving asbestos, such as mining, shipping and product manufacture.

Details of each of the papers considered are provided in the following section in reverse chronological order of publication. An analysis of the literature is provided in Section 4 of the main text. Summaries of the quantitative risks from the papers reviewed are provided in Tables 6 and 7 of the main text.

A list of all the papers identified in the original literature search and the reasons for their inclusion or exclusion is provided in Table A1.

A2 LITERATURE REVIEWED

A2.1 Papers identified by literature search

* Domestic exposures to asbestos arising from household members' occupational exposures. For example, exposures that can arise from asbestos dust brought home on clothing.

Luo S, Liu X, Mu S, Tsai S P and Wen C P (2003). Asbestos related diseases from environmental exposure to crocidolite in Da-yao, China. 1. Review of exposure and epidemiological data. Occup. Environ. Med. 60, 35-42.

In the rural county of Da-yao in southwestern China, patches of crocodilite are distributed widely in the surface soil (20% by area). Exposure of the local population occurs as a result of the wide dispersion of crocodilite dust in the area and also from the use of stoves manufactured from the asbestos material (a small local industry). This paper reports a number of studies of the health impact on the local population of asbestos exposure. In particular, it describes three ecological studies carried out in the county. The first of these considered a cohort of 5603 adults born between 1915 and 1955 for an observation period of 7 years (1977-1983). At the end of the study period information on the health status of 10% of the population could not be identified. The rates of mesothelioma and lung cancer in the study population were compared with those for neighbouring, non asbestos contaminated, counties for an earlier period 1974-1976. The size of the comparison population was more than 10 times larger than the study population. One confounding factor noted is that the neighbouring counties have lower smoking rates. Overall the numbers of cancers were a third higher in the study population than in the comparison group. Lung cancer numbers were 6.67 times higher (4 cases) than the comparison group, although no confidence interval (CI) is given so it is difficult to judge the significance of this result, in particular given the difference in smoking rates between the two groups. There were 3 cases of mesothelioma in the study population and none in the comparison population.

The second study involved an exposed population group of 4598 aged 30 years or older on 1 January 1987. The comparison population was 5641 residents of an uncontaminated town 200 km away with similar smoking patterns. The two groups had similar gender composition (60% men in study and 55% in the control) and their age distributions were virtually the same. The observation period was 9 years (1987 - 1995). The loss to follow up was less than 1.5% for both groups. The overall cancer rate was 47% higher in the study group. The lung cancer relative risk (RR) was 2.14 with 95% CI 1.07-4.28 (21 deaths in study group). There were seven cases of mesothelioma in the study population and none in the comparison group. This second study was clearly more thorough than the first. The third study was simply a subset of the second study, including only those defined as ‘peasants’, testing the hypothesis that those working on the land would have higher asbestos exposures and thus be at greater risk. The exposed population was 1610 and the comparison population 2481. The loss to follow up less than 1% for each group. The results indicate a lung cancer RR of 3.02 (6 deaths in the study group), although no CI is given so it is difficult to judge the significance of the result. Five cases of mesothelioma were seen in the study population and none in the comparison group. No details were given of the gender and age match for this subgroup. The results indicate a raised lung cancer and mesothelioma risk in the exposed population, however, no quantitative exposure levels are provided so it is of limited use in application to other exposure situations.

Metintas S, Metintas M, Ucgun I and Oner U (2002). Malignant mesothelioma due to environmental exposure to asbestos – Follow-up of a Turkish Cohort living in a rural area. Chest, 122, 6, 2224-2229.

In some areas of Turkey asbestos deposits are found close to the surface and asbestos containing soil mixtures are used as a whitewash or plaster material for walls or as insulation and waterproofing. The paper describes an ecological study undertaken to investigate the link between exposure to asbestos containing soil and mesothelioma. The authors used questionnaires to identify villages where 'white soil' was still being used. From the 126 villages identified they randomly selected 67 villages. They collected white soil samples from these villages. They found asbestos in 41 villages. The majority of the asbestos is tremolite but some other types were also identified. By random sampling they selected 11 of these 41 villages.

The study population was the 1886 villagers aged over 30 years from these 11 villages. The study population was investigated in detail over the observation period of ten years, 1990 – 2000, in a very thorough manner. This included the investigation of health records and personal interviews with families by two researchers (chest physician and epidemiologist). Information on the subjects (including: name, sex, age, length of time in villages etc) was collected in addition to any information on causes of death. All mesothelioma cases were histologically confirmed. During the observation period 377 deaths occurred in total and 24 mesothelioma cases were diagnosed (12 men and 12 women). The rates within the population were compared with general values for Turkey and the world. The results indicate that average annual mesothelioma incidence rates were 114.8 per 100,000 for men and 159.8 per 100,000 for women. Thus indicating a risk 88.3 times greater in men and 799 times greater in women in comparison to world background incidence levels. There are clearly issues regarding the match between the study population and comparison group, however, the study clearly indicates an increased mesothelioma risk for the exposed population. The authors report some limited asbestos concentration measurements but these are insufficient to allow the quantification of risk with exposure levels.

Magnani C, Dalmasso P, Biggeri A, Ivaldi C Mirabelli D and Terracini B (2001). Increased risk of malignant mesothelioma of the pleura after residential or domestic exposure to asbestos: A case-control study in Casale Monferrato. Environmental Health Perspectives 109, 9.

In Casale Monferrato, a medium sized town in northwest Italy, a large asbestos cement factory was active from 1907 to 1985. The purpose of this case control study was to investigate the link between pleural mesothelioma and residential and domestic exposure to asbestos. The plant used various types of asbestos. The study considered all histologically confirmed pleural mesothelioma cases in the Local Health Authority (LHA) covering the Casale area (48 towns and 100,000 inhabitants, of whom 40,000 live in Casale) between 1987 (1 Jan) and 1993 (30 June). Cases were retrospectively identified through periodic surveys of the pathology units of the hospitals serving the study area. Diagnosis was confirmed by a panel of pathologists. Cases were matched by birth date (±18 months) and sex, and if dead, date of death (±6 months) to two controls (four if < 60 years). Controls were selected either from the files of residents of

the LHA of Casale (if the corresponding case was alive) or from the mortality files (for deceased cases, no cause of death was excluded).

Trained interviewers conducted the interviews from 1993 to 1995 using a standardized questionnaire. When the subject had died, the closest relative was interviewed. The questionnaire included sections on occupational and residential histories as well as the occupations of the spouse and smoking habits. Interview data was checked against other sources were possible. For example, occupational histories were compared to factory rosters. One hundred and sixteen cases and 330 controls were eligible for the study and 102 cases (or relatives) (89%) and 273 (83%) controls were interviewed. There were no significant differences in age, sex or residence between responders and non-responders. Cases and controls did not differ significantly in terms of numbers of jobs or residences. The frequency of missing data was also similar for cases and controls. As with all studies of this type where relatives of deceased cases are interviewed there is the potential for errors in the information. This is of less significance in relation to residence information as this was checked with other sources. However, the risks from domestic and residential exposure were estimated using information from non-occupationally exposed individuals (75, ie 102 minus 27 occupational cases) thus any errors in identifying those occupationally exposed has implications for the assessment of risks to the other groups.

Odds ratios (ORs) and associated CIs were determined for individuals living at various distances from the asbestos plant. These were based on the closest residence address for each individual, thus an individual who had spent most of their life outside Casale but spent a brief period living very close to the factory site would be classified according to this residence period. The authors state that the model used to estimate risk adjusts for the effect of exposure of those with occupationally exposed spouses or co-habitants (23 cases in total). It is not clear how this was done. The results indicate higher ORs for individuals living within 2500 m of the plant in comparison with those further away, however, the CIs are very wide so it is difficult to judge the significance of this effect. The authors argue that the high risk at significant distances suggests a role for sources other than simply atmospheric pollution direct from the plant. The use of residues in construction is suggested as one possible source. The authors also provide some limited information on asbestos concentrations in Casale but these are insufficient to estimate asbestos exposures.

Magnani C, Agudo A, Gonzalez CA, Andrion A, Calleja A, Chellini E, Dalmasso P, Escolar A, Hernandez S, Ivaldi C, Mirabelli D, Ramirez J, Turuguet D, Usel M and Terracini B (2000). Multicentre study on malignant pleural mesothelioma and non-occupational exposure to asbestos. British Journal of Cancer 83(1), 104-111.

This is a multi-centre case-control study investigating the link between malignant pleural mesothelioma and domestic and environmental asbestos exposures arising from various industrial sources (eg asbestos cement plants, shipyards) in a number of areas in Italy, Spain and Switzerland. Six areas were considered in total; three in Italy (Torino, 13 towns in LHA of Casale Monferrato, and Firenze plus Prato) two in Spain (Barcelona and Cadiz) and one in Switzerland (Geneva). The study considered all malignant pleural mesothelioma cases diagnosed between 1 Jan 1995 and 31 December 1996. Except in

Barcelona where the study also included cases diagnosed in 1993 and 1994 and in Torino where the recruitment ended in April 1997. Controls were selected as a random sample from the population in Italian centres and Geneva. In the Spanish centres controls were randomly selected from patients discharged from all hospitals in the area, excluding those with asbestos-related conditions. This approach was taken because of problems with low participation in a pilot study. The control group was selected according to the age-sex structure expected for cases with a sample size twice the number of cases.

Cases and controls were interviewed at home or at the hospital by trained interviewers or, when the subject had died, a relative provided the information. Almost all controls (98%) were directly interviewed, while a proxy respondent was needed for one third of cases (in all but 4 cases this was the spouse or son/daughter). Information was collected for 215 histologically confirmed cases and 448 controls. The interviews lasted about 1 hour. The questionnaire included: demographic characteristics; smoking habits; radiation treatment; lifelong occupational history with specific sections for 33 industrial activities and occupations with possible asbestos use; occupations of spouse, parents and other cohabitants; and lifelong residential history, including address and description of dwellings and their neighbourhood environment.

Lifetime asbestos exposure was assessed from the questionnaire data by a panel of industrial hygienists, together with their knowledge of asbestos use in the study areas. Standardized criteria were used to assess the probability and intensity of asbestos exposure separately for occupational, domestic and environmental sources, blinded to the case-control condition of the subject. For 53 cases and 232 controls the panel found no evidence of occupational exposure to asbestos. In this group the age distribution was very similar in cases and controls, but there was a striking preponderance of females among cases and of males among controls. The assessment of risks associated with domestic and environmental exposure was carried out for subjects without occupational exposure; therefore potential exposure to asbestos at the workplace was carefully assessed by the industrial hygienists. The classification of domestic and environmental exposure was based on residence history. Evaluation of environmental exposure depended on the industrial activities in the surroundings and the distance from the subject's home. Classification was in terms of both probability and intensity. In this context a high probability of environmental asbestos exposure was defined as living at some time within 2000 m of asbestos mines, asbestos cement plants, asbestos textiles, shipyards or brakes factories. Odds ratios were calculated for each exposure category as compared to the never exposed. All the estimates were adjusted by centre, sex and age. The results indicate a statistically significant risk for individuals with a high probability of environmental exposure. Living between 2000 and 5000 m from asbestos industries or within 500 m of industries using asbestos could also be associated with an increased, but not statistically significant, risk.

The authors state that this is one of the largest studies of non-occupational exposure to asbestos. Despite this the total numbers of cases environmentally exposed are small (eg 17 cases for the high probability of environmental exposure category). The study does, however, appear to have been conducted in a very thorough manner. Cognisant of the potential problems with the use of proxies they investigated this issue carefully. This is of less significance in relation to residence, as the spouse or children are likely to

be reasonably accurate on this and there are various confirmatory sources, but is more of an issue in relation to occupational exposures, as those occupationally exposed are excluded. It is also possible that cases, being aware of the hypothesis studied may recall better than controls. Within the study a validation exercise was carried out on 18 cases from Barcelona: subjects provided direct information and, after they died, a proxy was asked to answer the same questionnaire. The overall agreement was good especially from spouses. The classification of the subjects did not change as a result of the alternative respondent. The authors conclude that the study confirms neighbourhood risk in Casale and is suggestive of corresponding risk in Barcelona and Torino. They also note that in the Barcelona cases there seems to be a preponderance of people who may be exposed as a consequence of having asbestos roofs. No quantitative asbestos exposure levels were provided in the study.

Luce D, Bugel I, Goldberg P, Goldberg M, Salomon C, Billon-Galland M, Nicolau J, Quenel P, Fevotte J and Brochard P (2000). Environmental exposure to tremolite and respiratory cancer in New Caledonia: A case-control study. Americal Journal of Epidemiology, 151, No 3.

A case-control study was undertaken in New Caledonia (South Pacific) to investigate the possible link between environmental asbestos exposures resulting from the use of material from local tremolite asbestos outcrops as whitewash and lung cancer and mesothelioma. All respiratory cancer cases diagnosed between Jan 1993 and Dec 1995 were included in the study. The study was limited to subjects over the age of 18 years and living in New Caledonia. Cases were identified from the Cancer Registry of New Caledonia. Thirteen lung cancer cases were not microscopically verified but were diagnosed by clinical, radiological and endoscopic evidence; all other cases were histologically confirmed. The total number of cases was 273 (including 15 pleural mesotheliomas, 228 lung cancers and 23 laryngeal cancers). There were 305 controls for the study. The control group was drawn at the beginning of the study and randomly selected from electoral rolls. The control group was frequency matched by sex and age (5 year age groups) to the expected distribution of the combined cases.

Interviews were conducted by trained interviewers. These were undertaken with individuals or next-of-kin, where possible, to obtain detailed information on past or present use of the whitewash, residential history, smoking, diet, and occupation. The interview lasted on average 1 h 45 minutes. For deceased cases and subjects who could not be interviewed for health reasons (total 65 cases and 2 controls) a shorter questionnaire was used to interview a next of kin. This had the same questions on residence and use of whitewash but simplified questions on smoking, diet and occupation.

For each cancer site ORs and the associated 95% CIs were obtained. All ORs were adjusted for age. For lung cancer, ORs were adjusted for lifetime smoking. The risk of mesothelioma was strongly associated with the use of whitewash, OR 40.9, but with a very wide 95% CI, 5.15 – 325 (from 14 cases). All cases had been exposed. The risk increased with duration of exposure. Among women, exposure to the whitewash was associated with an increased risk of lung cancer (OR 2.51, 95% CI 1.01 - 6.22) (from 21

cases), in contrast no association between exposure and lung cancer risk was noted for men (OR 0.89, 95% CI 0.51-1.54) (from 35 cases).

The difference between lung cancer risks for men and women may relate to differences in exposures. However, the manner in which confounders, such as, primarily, smoking, have been taken into account may have had an impact upon these results. As with all studies of this type the need to interview proxies could introduce bias. The use of a shorter questionnaire for these may have exacerbated this especially as the smoking questions were simplified. However, the authors note that reanalysis of the data with next-of-kin interviews excluded gave similar results. Odds ratios were adjusted for smoking using four categories for men (pack-years <20, 20-39, 40-59 and > 60) and two for women (never smoker, <20 and >20). It is not clear what impact this difference in approach would have on the results. The authors argue that the most likely explanation for the sex difference is that the level of exposure would be higher for women as they spend more time at home in the contaminated environment. Their studies indicate that women spend 2 more hours a day at home indoors and also that they do the cleaning and so may be exposed to higher dust concentrations. No actual exposure measurements were given in the paper.

Rees D, Myers JE, Goodman K, Fourie E, Blignaut C, Chapman R and Bachmann MO (1999). Case-control study of Mesothelioma in South Africa. Am. J. Ind. Med. 35:213-222.

The objective of this multi-centre case-control study was to estimate RRs of mesothelioma for various levels of certainty of asbestos exposure (classified as - definite, probable, possible and unlikely), for a number of categories of exposure (eg occupational and environmental) and asbestos fibre type. The study was conducted in the six major industrial centres of South Africa (Greater Bloemfontein, Cape Town, Durban, Johannesberg, Kimberley, Port Elizabeth and Pretoria – Durban was later abandoned as it didn't operate successfully). Each centre included all hospitals within 50 km of the city centre. Cases and controls were collected by the six study centres from referral hospitals. Exposure information was collected by interviewing cases and controls. The interviewers were trained, blind to case-control status, and did not know that the primary research questions related to asbestos and mesothelioma.

Research teams operated for about 16 months, from late 1988 through to early 1990. The intention was to include all cases treated or diagnosed in the study centres over the study period. Only histological confirmed diagnoses were included in the study. Cases and controls were matched by skin colour, gender and approximate age. Two controls were identified for each case, one with a medical condition and one with cancer. General exclusion criteria for both medical and cancer controls were that they should not have asbestos related disease or an undiagnosed condition. Controls with lung, pleural or peritoneal malignancy were excluded. Detailed questionnaires were used for cases and controls. The information included: residential history; time spent near dockyards, mines, mills, asbestos using factories, or stores of asbestos; parent's occupation; domestic and leisure time; exposure to dust; a complete occupational history with detailed questioning on asbestos exposure; diet and smoking. To reduce recall bias cases were interviewed as soon as diagnosis was suspected and subjects

were not told that the study related to asbestos specifically. Subjects were grouped by probability of exposure to asbestos, the likely fibre type and the nature of the exposure (eg occupational or environmental). One hundred and twenty three cases were accepted into the study and 119 cancer controls and 103 medical controls were available. Control sets were short due to inappropriate controls being selected (ie problems with inclusion criteria). A minimum of 22 cases had exclusively environmental exposure, 20 from the NW Cape crocidolite mining area. Conditional logistic regression was used to calculate ORs and 95% CIs. Odds ratios for environmental exposure were determined using only the 22 cases with solely environmental exposure. They indicate increased risk (OR 19.6) with environmental exposure but with wide CIs (95% CI 3.7-105).

This study also considered occupational and domestic (paraoccupational) exposures. One problem with this type of study is that only those with solely environmental exposures are included in determining risks from environmental exposures, which means that the numbers involved are low (22 in this case living in mining districts). This also means that errors in assigning individuals to the occupational exposure set will impact upon these ORs. The authors note that the main limitations of the study relate to representativeness of cases and possible misclassifications of exposure. There is evidence that the 123 cases in the study misrepresented South African cases in some respects; 55% of the cases were white, yet this group makes up only about 20% of South Africa's population. Poorer access to health care by black South Africans is a likely explanation but the effect of this under representation is unknown. It is possible that this series of cases under represents cases with environmental exposure. For some confirmation of classification they undertook some sputum analyses and found that no subject classified in the unlikely exposure class had fibres in sputum and 19 of the 20 subjects with fibres were classified as definite or probable exposure. No quantitative asbestos exposure levels were provided in the study.

Mzileni O, Sitas F, Steyn K, Carrara H and Bekker P (1999). Lung cancer, tobacco, and environmental factors in the African population of the Northern Province, South Africa. Tobacco Control 8, 398-401.

The aim of this case-control study was to assess the risk of developing lung cancer related to tobacco smoking, fuel use, and residential and occupational exposure to dust and asbestos in the Northern Province of South Africa. Between 1993 and 1995 a case-control study was conducted at Garankuwa hospital, which is the main tertiary referral hospital for patients with cancer in the Northern Province. Cases consisted of 288 men and 60 women with lung cancer. Controls, selected at the same time as the cases, consisted of 183 male and 197 female controls (these comprised patients newly diagnosed with cancers for the first time other than those known to be associated with smoking). Cases and controls were interviewed on basic demographic details, smoking habits, place of birth, current residence, their main occupation and fuel use at home. To investigate the importance of asbestos exposure, postal codes of places of current residence and place of birth were used to classify areas into 'heavy polluted asbestos areas' (areas where mining took place), 'moderate asbestos polluted areas' (towns where raw asbestos was transferred from the mines for shipping to other areas), and 'non asbestos polluted areas'. Subjects were not asked whether they worked with

asbestos or in dusty occupations to avoid any possible recall biases. Unmatched, unconditional logistic regression was used to calculate ORs and 95% CIs in relation to potential asbestos exposures after controlling for a number of variables including tobacco consumption.

The study found 38 male cases currently living in moderately polluted asbestos areas and 10 in heavy polluted asbestos areas (controls 11 and 3 respectively). For asbestos exposure at birth the study identified 36 male cases with moderate exposure and 8 with heavy exposure (controls 9 and 2 respectively). The equivalent data for women were: 4 cases currently living in moderately polluted asbestos areas and 6 in heavily polluted asbestos areas (controls 11 and 4 respectively) and 2 cases in moderate asbestos polluted areas at birth and 3 in heavily polluted areas at birth (controls 8 and 0 respectively). The results indicate increased risks of lung cancer for living and being born in asbestos polluted areas, although in some instances the number of cases was small and the resulting 95% CIs are high.

The study also looked at the risk of developing lung cancer related to exposure to tobacco, indoor pollution, and a dusty work environment. One problem with the study is that it is not clear how smoking was accounted for in those environmentally exposed. The method adopted is not described in detail. Another problem with the study relates to the case control match. The cases are mainly in men but there is a disproportionate number of women in the controls, so some of the ORs for men are based on a very small number of controls. There seems to be no detailed matching of controls to cases. The authors also note that the asbestos exposure categories are crude (for example, sharing the same postal code as a mine). No quantitative exposure levels are given in the paper.

Magnani C and Leporati M (1998). Mortality from lung cancer and population risk attributable to asbestos in an asbestos cement manufacturing town in Italy. Occup Environ Med 55, 111-114.

The objective of this ecological study (geographical comparator) was to consider mortality from lung cancer and estimate the risk attributable to asbestos exposure separately for asbestos cement workers and for the general (non-occupationally exposed) population in the town of Casale Monferrato, where the largest Italian asbestos cement factory had been in operation between 1907-1986. The study was based on a list of residents in Casale Monferrato dying from lung neoplasms between 1 Jan 1989 and 31 December 1995. In this period 227 deaths were attributed to lung cancer. No information on occupation is included in the list. The list was therefore linked with the lists of workers in the asbestos cement factory and of their wives, to identify the people occupationally or paraoccupationally exposed to asbestos from the cement works. The population at risk was estimated from a follow-up of the cohorts of asbestos cement workers and their spouses. The non-occupationally exposed population was estimated as the difference between the total and the occupationally exposed populations. For simplicity the subjects were defined in the study (rather unhelpfully and certainly inaccurately) as ‘exposed’ if they ever worked in the asbestos plant or were married to a worker in the factory and ‘non-exposed’ otherwise. Of the 227 lung cancer deaths (184 men and 43 women) 59 were asbestos workers or married to an asbestos

worker. Lung cancer mortality rates for the exposed and non-exposed populations were determined and compared. These were also compared with general lung cancer mortality rates. The rates were clearly higher for the occupationally exposed population. Males who did not work in the asbestos plant did not have different mortality rates to males from elsewhere in the Piedmont region. A small increase was found among women but this did not reach significance. The authors conclude that this work did not show an increase in mortality from lung cancer for the population not exposed occupationally, but a large excess was found among men and women occupationally exposed in asbestos cement production. This was in contrast to an earlier study undertaken by the same group which showed a general increase in mesothelioma in the non-exposed population.

The authors note that the contribution of cigarette smoking could not be evaluated as individual information on smoking habits was not available. It would have been useful to have some indication of general smoking rates in the town versus the region of Piedmont. They also note that they couldn't include other workers (eg construction) in the area who may have been exposed to asbestos.

The paper provides some information on asbestos concentrations in Casale. Environmental asbestos concentrations were measured only shortly before the factory shut down and afterwards. Estimates reported are the average of repeated measurements and are limited to airborne asbestos fibres with length > 5 μm and diameter 0.3 μm. In 1984 the asbestos concentration was 0.011 f/ml close to the plant and 0.001 f/ml in the city area farthest away. In 1990 and 1991 the local health authority reported annual average concentrations below 0.001 f/ml. Another study in 1991 reported a range in residential areas of Casale of 0.0022 to 0.0074 f/ml. It was noted that the differences between the two 1991 studies could be partly due to the different methods used for sampling and analyses.

Camus M, Siemiatycki J and Meek B (1998). Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. N Engl J Med 338 1565-71.

The objective of this ecological study was to investigate the risk of lung cancer from non-occupational exposure to chrysotile asbestos. Mortality among women in two chrysotile asbestos mining areas of the Canadian province of Quebec was compared with mortality among women in 60 control areas. The study was restricted to women in order to exclude most asbestos workers. It used data from death certificates, which the authors considered adequate to study the risk of lung cancer but not that of mesothelioma. Mesotheliomas among the population are being investigated in a separate study. The study considered the number of lung cancer deaths between 1970 and 1989 among women at least 30 years of age who lived in the two chrysotile asbestos mining areas or 60 reference areas in the province of Quebec.

The population within the two asbestos areas live within 10 km of a mine or mill, and 80% live within 4 km. Over the observation period, among women 30 years of age or older there were 221,375 person-years in the asbestos mining areas and 8,629,630 person-years in the reference areas. The (age) standardised mortality ratios (SMRs) and standardised proportionate mortality ratios (SPMRs) for death from selected causes

among women in the asbestos mining area as compared with women in the reference population were determined. Both measures are ratios of the numbers of observed deaths in the population under study to the expected numbers, with adjustment for age and calendar year. For the standardized mortality ratio, the expected number is based on the absolute mortality according to cause in the reference population. For the standardized proportionate mortality ratios, the expected number is based on the proportion of all deaths in the reference population that are due to each cause. They found no measurable excess risk of death due to lung cancer among women in the two chrysotile asbestos mining areas. They did note, however, the increased incidence of asbestosis and pleural cancer – age standardised mortality ratio for deaths from cancer of the pleura 7.63 (95% CI 3.06 – 15.73).

Population characteristics were similar between the exposed and not exposed groups, although there was some indication that smoking was less prevalent in the exposed group. The allocation to group was on the basis of place of residence at death (ie movement ignored). But the authors considered that movement was not a significant issue for these populations. The authors also concluded that adjusting for the different smoking rates between the populations would not have changed the results by more than 7%. Substantial bias is therefore unlikely.

In addition to the above analysis of mortality rates the study also estimated exposures. In 1989 they interviewed 817 elderly female residents of the asbestos mining areas about their lifetime residential and employment histories and those of the people they lived with. This information was used to estimate exposures. These were determined using an expert panel. They used data from continuous and annual measurement programs starting in the 1970s combined with other information on production. Average ambient levels were estimated to have peaked at 1 fibre/ml or more between 1940 and 1954 and to have been above 0.2 f/ml from about 1905 to about 1965. The panel thought that the true values were unlikely to lie below 33% or above 300% of their best estimates. The average cumulative lifetime exposure of the population in the asbestos mining areas, according to type of exposure, 1970 through 1989, was estimated as indicated in the table below.

Estimated cumulative exposures (Camus et al, 1998)

Type of exposure	Estimated cumulative exposure, f/ml-years
Neighbourhood	16
Household	7.8
Occupational	1.2
Total	25
Subjective plausible range	5 – 125

In the study the authors used the above exposure estimates with the asbestos risk model used by the US Environmental Protection Agency (EPA) (Upton et al, 1991) to determine the expected number of lung cancer deaths. They infer from the results obtained that the EPA's model overestimates the risk of asbestos-induced lung cancer by at least a factor of 10.

This study produced a lot of comment in the literature. For example, It was suggested that some of the women were occupationally exposed, even though they didn't work in the mining industry, and that they were exposed to amphiboles (Churg, 1998).

Hansen J, de Klerk N, Musk AW and Hobbs M S T (1998). Environmental exposure to Crocidolite and Mesothelioma. Am. J. Respir Crit. Care Med. 157 69-75

The aim of this retrospective follow-up study was to estimate exposure response relationships between environmental exposure to crocodilte and mesothelioma. The exposure resulted from the operation of a crocodilite mine in Wittenoom, Weastern Australia. All 4,659 former residents of Wittenoom who lived there between 1943 and 1993 for at least a month and were not directly employed in the crocidolite industry, were followed up through the Western Australia death, cancer and mesothelioma registries, electoral rolls, telephone books and other sources. The whereabouts of 71.4% of the cohort as at December 31, 1993 was known and 11.8% of the cohort remained untraced. The total person-years accumulated to December 31, 1993 was 132,986, representing an average length of follow-up of 29.5 years. In 1992 all subjects who were traced (71.4%) were sent a questionnaire. Records of deaths were used to try and identify all deaths within the cohort between 1943 and 1993. To the end of 1993, 27 cases of mesothelioma (histologically confirmed) had been diagnosed, 16 of these had occurred since 1989 (total deaths 460). Of the 27 cases (18 female and 9 male), 12 were wives, 11 were children and one was a brother of men who worked with crocidolite at Wittenoom (total 24).

Information on the duration of exposure was obtained where possible. Estimates were made of individual's exposures based on their duration of exposure. Subjects were assigned an intensity of exposure of 1.0 fibre/ml from 1943 to 1957 (when a new mill was commissioned and the town was moved), and then 0.5 f/ml between 1958 and 1966, when the mining operations ceased. Since then, interpolation between periodic surveys using personal monitors assigned exposures from 0.5 fibres (> 5 microns long) per ml of air in 1966 to 0.01 f/ml in 1992. Thus there was no individual assessment of exposure concentrations but simple general values for the population as a whole multiplied by duration of exposure.

The study aimed to investigate exposure-response relationships for mesothelioma and environmental exposure to crocidolite. The results indicated that mesothelioma cases stayed longer at Wittenoom, had a higher average intensity of exposure, and a higher cumulative exposure to crocidolite than control subjects. The authors generated RRs as a function of duration of exposure and estimated intensity of exposure. These clearly indicate that risk increased with both duration and cumulative exposure, although the link with duration was stronger. They also noted that in this cohort of Wittenoom residents, cases of mesothelioma have arisen in subjects with durations of crocidolite exposure as short as 2 months and estimated cumulative exposure as low as 0.53 f/ml.

The authors state that no previous study of environmental exposure to asbestos and the risk of mesothelioma has been able to utilize exposure levels to derive quantitative exposure-response relationships. However, they note that, because of the greater errors in intensity and cumulative exposures, the model with exposure assessed by just

duration of residence appeared to be a better fit than the one which used estimated cumulative exposure, probably because of the large error introduced into the estimate of cumulative exposure by the estimate of intensity of exposure. They also note, however, that 24 of the 27 cases lived with an asbestos worker. In this case paraoccupational exposures may have been higher than the general environmental levels used in the study. This would also be an alternative explanation why duration alone produced a better fit than exposure intensity. In general, not addressing the significant potential paraoccupational exposures is a significant shortcoming of the study.

Howel D, Arblaster L, Swinburne L, Schweiger, Renvoize E and Hatton P (1997). Routes of asbestos exposure and the development of mesothelioma in an English region. Occup Environ Med 54: 403-409.

The main purpose of this case control study was to investigate the link between asbestos exposure resulting from industrial uses and the incidence of mesothelioma. The study focussed on histologically confirmed cases of mesothelioma, where death had occurred between 1979 and 1991, in four health districts in Yorkshire. Of a total of 316 potential cases identified, 71 could not be confirmed, 15 were not mesothelioma, and 4 were possible, leaving 226 confirmed. Relatives of 133 were interviewed and information on a further 52 cases was obtained from coroner's records (total 185).

Surviving relatives were interviewed to ascertain lifetime exposure to asbestos. Necropsy records were used to identify controls for the cases. Sets of cases and controls were matched for sex, age at death (to within 10 years) and year of death (to within 2 years). The sets ranged in size from one case matched to one control, to six cases matched with five controls. The choice of matched sets rather than matched pairs was considered to make the best use of scarce subjects. Surviving relatives of all 160 controls were also interviewed. Odds ratios for mesothelioma for exposure to asbestos through occupational, paraoccupational and residential routes were calculated (adjusted for age, year of death etc.) Likely or possible occupational exposure to asbestos was more common in cases than controls. After excluding those with likely or possible occupational exposure, likely or possible paraoccupational exposure was more common in cases than controls. Only six cases of mesothelioma were identified as being solely exposed to asbestos through their residence, compared to 14 controls. Residential exposure was defined as living within 500 m of an industrial site potentially using asbestos. The OR for residential exposure to asbestos varied between 1.6 and 6.6, depending upon which potential sources were included, but the 95% CIs were so wide that reduced or greatly increased odds comparing cases with controls could not be ruled out. The study results support previous evidence that occupational and paraoccupational exposure to asbestos is associated with developing mesothelioma. Despite a rigorous search, purely residential exposure seemed to account for only 3% of identified cases. No firm conclusion can be drawn about the risks from residential exposure alone, as many of the study subjects could also have been occupationally or paraoccupationally exposed.

This is a thorough study, but was limited by the small number of residential cases and the complex confounding issues related to occupational and paraoccupational exposures. One problem with this type of study is that the subjects are deceased so the

retrospectively derived asbestos exposure histories are potentially subject to error. The time delay also introduces errors in identifying industrial sites that might have been a source of environmental asbestos pollution. It was also assumed that occupational exposures would dominate those from other sources; this may not be the case. The need for larger studies was identified if links between environmental exposures and mesothelioma are to be confirmed.

A2.2 Other papers identified from search papers

Berry M (1997). Mesothelioma incidence and community asbestos exposure. Environ. Res. 75(1) 34-40.

Manville, located in Somerset County, New Jersey, was the site of the largest asbestos products manufacturing plant (primarily chrysotile) in North America between 1912 and 1980. This ecological study considered the 143 mesothelioma cases in Somerset County reported to the NJ State Cancer Registry between 1979 and 1990. Cases were removed from the analysis when their usual employment was reported as being at the asbestos plant, as evidenced through union lists or occupational information from either the Cancer Registry or mortality records.

Standardised incident ratios (SIRs) were computed for residents of Manville and Somerset County (minus Manville) by sex. New Jersey mesothelioma rates less the Somerset county contribution, 1979-1990, were used to generate the expected number of cases. A total of 1358 newly diagnosed mesothelioma cases were reported to the NJ State Cancer Registry over the 12 year study period. Of these, 143 cases were identified as residents of Somerset County (122 men, 21 women) and 55 of these were residents of Manville (46 men, 9 women). Of the 143 cases a total of 61 were identified as persons having worked at the Manville plant. A total of 82 had no evidence of employment at the plant; 24 were residents of Manville and 58 resided elsewhere in the county. These 82 cases were used in the study. The SIRs, after removal of plant employees, for Manville males and females were, respectively 10.1 (95% CI 5.8-16.4) and 22.4 (95% CI 9.7-44.2). For Somerset County (except Manville) the equivalent values were 1.9 (95% CI 1.4-2.5) and 2.0 (95% CI 1.0-3.6).

The authors conclude that the study shows a strong relationship between past asbestos exposure from living in Manville and eventual development of mesothelioma. However, there are some problems with the study. No interviews were carried out so full occupational histories were not known. It is therefore possible that some had worked at the plant at some time, so the possibility of the number of occupational cases being underestimated exists. This would mean that risks from non-occupational exposure would be lower than indicated in the study. It is also possible that domestic exposure is making a major contribution to the risk, but this was not addressed.

Botha JL, Irwig LM and Strebel PM (1986). Excess mortality from stomach cancer, lung cancer, and asbestosis and/or mesothelioma in crocidolite mining districts in South Africa. Am J Epidemiol 1986, 123, 30-40.

The purpose of this ecological study (geographical comparator) was to investigate whether exposure to crocidolite from mining operations in the northwestern part of Cape Province is associated with excess mortality. This area is over 800 km from areas where other forms of asbestos are mined. The authors calculated standardized mortality ratios based on deaths in these mining districts from 1968 to 1980 for selected causes of death. Contiguous districts were used as controls. To take account of background geographical variability, they divided the control districts into groups with population sizes similar to those of the crocidolite mining districts.

Crocidolite mining districts in the province were selected on the basis of the average annual percentage of the male population aged 20-64 years who were employed in crocidolite mines during the 40 year period 1921-1960. Only white and coloured (of mixed racial origin) populations were considered for analysis, since the necessary information for other races was not available. Three districts had annual employment rates more than 2% of the adult white or coloured population (termed in the study as 'high crocidolite districts'). Two other districts had equivalent rates of between 0.1 and 0.7% (termed 'low crocidolite districts'). Control districts were defined in two concentric "rings" around the crocidolite districts. A total of 29 control districts were used. A few potential control districts were excluded as they had large urban populations and were therefore unlike the case districts or because the mortality data was not present or of poor quality. Mortality data for the period 1968-1980 were extracted from official death records. Until 1977 there was a single category for asbestosis and mesothelioma. After this date separate categories were introduced. Deaths were attributed to the district stated as the usual residence on the death certificate. Mortality rates were calculated for asbestosis and/or mesothelioma. For all other causes of death, age standardised mortality ratios were determined for group of districts (mining or control). The reference population chosen consisted of the combined population of all the crocidolite and control groups of districts. The authors also compared rates with those for the South African population as a whole.

The study found that crude death rates in the control districts were slightly higher than those in the rest of South Africa, below those of the low crocidolite districts and far below those in the high crocidolite districts. For other health endpoints the standardised mortality ratios for 'low crocidolite' areas were indistinguishable from those of controls for all causes of death. Standardized mortality ratios in the crocidolite mining districts were elevated for asbestosis and/or mesothelioma, and cancer of the lung and stomach.

There are a number of issues with the use of the data provided for estimating risks from environmental exposure. The main problem is that there is no identification of workers. It is likely that a large proportion of cases, especially of mesothelioma, would arise among the more exposed working population. It is not possible to use the results to imply any link with environmental exposures. There are also issues surrounding the limited population considered. Information on the smoking habits of the districts is not provided, which is clearly of importance in relation to lung cancer incidence.

Neuberger M, Kundi M and Friedl HP (1984). Environmental Asbestos Exposure and Cancer Mortality. Arch Environ Health 39, 261-265.

The purpose of this ecological (geographical comparator) study was to investigate lung cancer risks from environmental asbestos exposure. The study considered two Austrian towns. One town located in an area with natural asbestos deposits (principally tremolite), where mining had occurred until 1945, and asbestos is found in the soil, atmosphere and drinking water. The other study area is located around the oldest asbestos cement factory in the world.

The authors analysed mortality data from official death certificates from 1970 to 1980 in the two areas. Standard mortality ratios (SMRs) were calculated on the basis of population census returns for 1961, 1971 and 1981 (corrections for births, deaths, and mobility were used for the intervening years), and annual mortality data, both grouped according to sex and age (5-yr age groups). Expected numbers of deaths from lung cancer (and stomach cancer) were calculated using 5 populations as references: (1) the Austrian population, (2) the respective province population to which the study population belongs, (3) the population of the respective district, (4) the subpopulation of Austria living in communities belonging to the same community size class, and (5) the subpopulation of Austria living in communities with the same agricultural index as the communities under study. Lung cancer mortality in men (and total, ie men and women) in the two asbestos exposed populations was lower than expected in comparison with each of the 5 populations. For lung cancer mortality in women, in the asbestos processing town the rates were higher in comparison to all 5 populations. For the town with natural asbestos deposits the lung cancer mortality rates in women were higher in comparison with those for Austria as a whole, the province and district but lower than those for the community size class and same agricultural index.

The authors conclude that no evidence of lung cancer excess from environmental asbestos exposure could be detected in the study. The authors do note, however, that because of the small sizes of the communities studied a small excess in cancer deaths could not be ruled out as only RRs greater than 2 would have been detected.

There are a number of issues arising from the study. For example, the exposed population in the asbestos cement production plant town includes occupationally exposed individuals, so the exposure is not purely environmental. One important confounding issue in studies of lung cancer is clearly smoking. The authors note that for the town with asbestos deposits relative frequencies of male smoking vary only marginally between the populations considered (41.1% in general population, 39.5% in the province and 39.7% in communities with the same size and agricultural index) which would tend to indicate that smoking had been accounted for to some extent in the study for men. They do note a much larger range for women smoking, eg 9.1% for the province and 16% for populations living in communities with the same size and agricultural index. For the town with the asbestos plant it is estimated that smoking habits are little different to those in the comparison populations.

Yazicioglu S, Ilcayto R, Balci K, Sayli B S, and Yorulmaz (1980). Pleural calcification, pleural mesotheliomas, and bronchial cancers caused by tremolite dust. Thorax 35, 564-569.

Natural deposits of asbestiform minerals around the town of Cermik in southeastern Turkey are used by the local population to make whitewash and stucco for walls and roofs. The objective of this ecological (geographical comparator) study was to investigate the potential links between exposure to tremolite dust from the use of such materials and mesothelioma and bronchial cancers.

The Diyarbakir Chest Hospital serves a wide area including the asbestos contaminated area. During the period of the survey (1977 and 1978) there were 86 admissions to the Hospital for neoplasms of the lung and pleura. Those in the five asbestos districts were compared with those from seven non-asbestos contaminated districts. The two groups had approximately the same population numbers (respectively 227,000 and 218,000). Of the 86 cases 41 patients came from the two population groups. The asbestos exposed group had 22 pleural neoplasms and the comparison group 1; a statistically significant difference. There were 11 pulmonary neoplasms in the asbestos area and 7 in the non-exposed area; difference not statistically significant. The rate of pleural neoplasms was estimated as 9.67 per 100,000 inhabitants.

There is little quantitative analysis of the data, for example, no RRs were determined. There is also no discussion of potential confounding factors such smoking rates and population movement.

McDonald AD and McDonald JC (1980). Malignant mesothelioma in North America. Cancer 46, 1650-1656.

All fatal mesotheliomas in Canada (1960-1975) and the USA (1972) were considered in this case-control study. The primary purpose of the study was related to risks from occupational exposures to asbestos. There was some indication of risks from domestic exposure (6 cases) but only brief mention is made of possible risks from environmental exposures.

The authors note that, excluding those with occupational or domestic exposures, no subject but two controls had lived within 20 miles of a Canadian chrysotile mine. In the USA one subject and two controls had lived within 20 miles of a chrysotile mine in California. It was also noted that 17 subjects and 12 controls had lived for 20 through 40 years before death within 20 miles of reported zeolite deposits in Western USA, a paired analysis gave a RR of 1.83, reduced to 1.6 (75% CI 0.58 and 4.93) when allowance was made for occupational exposure. Zeolite is not one of the asbestos mineral group but is similar in form.

Hammond EC, Garfinkel L, Selikoff I J, and Nicholson W J (1979). Mortality experience of residents in the neighbourhood of an asbestos factory. Ann NY Acad Sci 330 417-422.

This ecological (geographical comparator) study looked at the mortality experience of men who lived in the vicinity of an amosite asbestos plant. The factory was located in a district known as Riverside in the town of Paterson, New Jersey. The study looked at the male population of Riverside in comparison with another neighbourhood, Totowa,

located several miles from Riverside. The authors traced residents of the two neighbourhoods over a 35 year period. They used city directories for Paterson (published annually) which list by name all adults living at each address and states the occupation of the head of the household. Only men were included in the study as it was considered that it would be much more difficult to follow-up women, especially given changes in names, for long periods. Of the total on the original list of names (7653) 5550 (alive as of January 1, 1962) were included in the analysis. They were followed for 15 years. Asbestos plant workers were excluded from the study. The age distributions of the two population groups were very similar. Lung cancer numbers were a little higher in Totowa than Riverside. There were 41 cases in Riverside (RR 0.9, no CI given), indicating negligible differences between the two populations for lung cancer. It was noted that there was only one case of pleural mesothelioma during the 15 years in the study population. That was an electrician in Riverside. The author's comment that they had undertaken analyses of dust samples in attics of houses in the Riverside area that did indicate the presence of asbestos, with lower concentrations at greater distances, although no actual measurements are given in the paper.

This is a relatively thorough study marred by no consideration or discussion of the potential confounding factors between the two populations, especially of smoking. There is no indication given that the smoking habits of the two groups are similar.

Newhouse M and Thompson H (1965). Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. Br J Ind Med 1965, 22, 261-269.

The purpose of this case-control study was to investigate the potential link between mesothelioma and occupational, domestic and neighbourhood exposures from asbestos industries. Eighty-three patients (41 men and 42 women) from the London Hospital with a confirmed diagnosis (necroscopy or biopsy) of mesothelioma were studied to gain information on their possible exposure to asbestos. These were cases diagnosed during the previous 50 years (although samples were analysed for confirmation at the time of the study). Twenty seven of the patients had peritoneal and 56 pleural tumours. For 76 of the 83 cases occupational and residential histories were obtained. Each of the 76 cases was matched with an in-patient of the same sex born in the same 5 year period, either from the London Hospital or, for some elderly cases, from a neighbouring geriatric hospital. Information on occupational asbestos exposure and place of residence was obtained for the controls. The sources of information were: ward notes; the patient's GP; the records of an asbestos factory in the area; and personal interviews with patients (the 4 surviving) or their surviving relatives.

The study showed a clear link between mesothelioma and occupational and domestic asbestos exposure. Among those with no evidence of occupational or domestic exposures, 30.6% of the cases (11) and 7.6% of the controls (5) lived within half a mile of an asbestos factory, indicating significance. However, no RRs were derived by the authors. An analysis of the basic data was undertaken by Bourdes et al (2000). They derived a RR of 7.5 and a 95% CI of 2.5-22 for residence within 0.5 miles of an asbestos factory, but it is not clear how this was determined from the basic data.

The authors note that one of the potential problems with the study is the differences between the cases and controls. The controls were all admitted to hospital in 1964. The patients were admitted to the same hospital over a period of 47 years during which there might have been a substantial change both in the residential areas and social classes of patients attending the hospital. To investigate the influence of this a further group of patients was taken from the hospital records matched by date of admission as well as birth and sex with the cases. Their places of residence were extracted for comparison with the initial control group. This showed little difference between the two 'control' groups. A general problem with this type of study, exacerbated in this case by the large timescale over which the cases arose, is the reliability of the occupational and residential history data, which was collected many years after the majority of the cases had died.

A3 REFERENCES

Bourdes V, Boffeta P and Pisani P (2000). Environmental exposure to asbestos and risk of pleural mesothelioma: review and meta-analysis. European Journal of Epidemiology 16: 411 – 417).

Churg a (1998). Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. New Eng. J of Med. 339, 14, 999.

Upton AC, Barrett JC, Becklake MR (1991). Asbestos in public and commercial buildings: a literature review and synthesis of current knowledge. Cambridge, Mass: Health Effects Institute-Asbestos Research.

Table A1 Papers identified in literature search and reasons for inclusion or exclusion

Reference*	Inclusion/Exclusion+					Comments
	Stage 1	Stage 2			Stage 3	
		1	2	3		
Abratt RP, Vorobiof DA, White N (2004). Asbestos and mesothelioma in South Africa. Lung Cancer, 45 Suppl 1:S3-6.	Y			N		One concentration measurement but not linked to study, non quantitative epidemiological study results
Aguilar-Madrid G, Juarez-Perez CA, Markowitz S, Hernandez-Avila M. Sanchez Roman FR, Vazquez Grameix JH (2003). Globalization and the transfer of hazardous industry: asbestos in Mexico, 1979-2000. International Journal of Occupational and Environmental Health, 9(3),p272-9.	N					Analysis of quantitative use of asbestos in Mexico and increased prevalence of mesothelioma
Algranti E (1998). Asbestos: current issues related to cancer and to uses in developing countries. Cadernos de Saude Publica, 14 Suppl 3, p173-6.	N					Review of lung cancer rates in Brazil, some mention of asbestos but occupational only
Algranti E, Menezes AM, Achutti AC (2001). Lung cancer in Brazil. Seminars in Oncology, 28(2), p143-52.	N					Review of lung cancer rates in Brazil and possible occupational exposure risks
Anonymous (1999). Call for an international ban on asbestos. Collegium Ramazzini. American Journal of Industrial Medicine, 36(2), p227-9	N					Call for international ban on asbestos
Anthonisen NR (2004). Trouble in Anatolia. Canadian Respiratory Journal, 11(4), p273-4.	Y			N		Brief comments on the long time taken to institute changes to use of asbestos and asbestos like minerals in Turkey for the protection of human health
Ascoli V, Carnovale-Scalzo C, Nardi F, Efrati C, Menegozzo M (2003). A one-generation cluster of malignant mesothelioma within a family reveals exposure to asbestos-contaminated jute bags in Naples, Italy. European Journal of Epidemiology, 18(2), p171-4.	Y			N		Report on cases of mesothelioma within a family in Naples using asbestos contaminated jute bags.
Ascoli V, Comba P, Pasetto R (2004). Urban mesothelioma: is there an emerging risk of asbestos in place? International Journal of Cancer, 111(6), p975-6.	Y			N		General discussion of mesothelioma risks but no relevant quantitative data
Ascoli V, Scalzo CC, Facciolo F, Martelli M, Manente L, Comba P, Bruno C, Nardi F (1996). Malignant mesothelioma in Rome, Italy 1980-1995. A retrospective study of 79 patients. Tumori, 82(6), p526-32.	N					Essentially an occupational study
Berg R (2004). When science crosses politics, I: The case of naturally occurring asbestos. Journal of Environmental Health, 66(10), p31-9.	Y			N		Paper primarily discussing the difficulty in presenting information on environmental risks focussing on asbestos. Relates to the interplay between science and public policy
Berry G, Liddell FD (2004). The interaction of asbestos and smoking in lung cancer: a modified measure of effect.	N					Effect of smoking on incidence of lung cancer from asbestos

Annals of Occupational Hygiene, 48(5), p459-62.						
Bianchi C, Brollo A, Ramani L, Zuch C (1999). Asbestos exposure in lung carcinoma: a necropsy-based study of 414 cases. American Journal of Industrial Medicine, 36(3):360-4.	N					Medical – attributing lung carcinoma to asbestos
Boffetta P (1998). Health effects of asbestos exposure in humans: a quantitative assessment. Medicina del Lavoro, 89(6), p471-80.	N					Review of models for lung cancer risk with asbestos exposure
Boffetta P (2004). Epidemiology of environmental and occupational cancer. Oncogene, 23(38), p6392-403.	Y		Y		N	Review paper
Boffetta P, Nyberg F (2003). Contribution of environmental factors to cancer risk. British Medical Bulletin, 68, p71-94.	Y		Y		N	Review paper
Bourdes V, Boffetta P, Pisani P (2000). Environmental exposure to asbestos and risk of pleural mesothelioma: review and meta-analysis. European Journal of Epidemiology, 6(5), p411-7.	Y		Y		N	Review paper
Braun L, Greene A, Manseau M, Singhal R, Kisting S, Jacobs N (2003). Scientific controversy and asbestos: making disease invisible. International Journal of Occupational and Environmental Health, 9(3), p194-205.	Y			N		General discussion of the impact of asbestos related diseases particularly in South Africa and India and the arguments about risks from different types and genetic susceptibility
Bridgman S (2001). Community health risk assessment after a fire with asbestos containing fallout. Journal of Epidemiology and Community Health, 55(12), p921-7.	Y			N		Factory fire involving ACM - no epidemiology, estimation of mesothelioma risks on basis of estimate of exposure using exposure response models
Burkart W (2001). Combined effect of radiation and other agents: is there a synergism trap? Journal of Environmental Pathology, Toxicology and Oncology, 20(1), p53-8.	N					Radiation not asbestos exposure
Camus M, Siemiatycki J, Meek B (1998). Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. **New England Journal of Medicine, 338(22), p1565-71.**	Y	Y				
Case BW (1998). Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. New England Journal of Medicine, 339(14), p1001.	Y			N		Comment on Camus et al (1998)
Castleman B, Dement J, Giannasi F, Frank AL, Frumkin H, Gochfeld M, Goldstein BD, Grandjean P, LaDou J, Lemen RA, Levy BS, Maltoni C, McDiarmid M, Silbergeld EK, Teitelbaum DT. Thebaud-Mony A, Upton AC, Wegman DH (1998). Salud ocupacional. International Journal of Occupational Medicine and Environmental Health, 11(2), p195-7.	N					Occupational
Chang HY, Chen CR, Wang JD (1999). Risk assessment of lung cancer and mesothelioma in people living near asbestos-related factories in Taiwan. Archives of Environmental Health, 54(3),	Y			N		Risk assessment for factories in Taiwan, no epidemiology

p194-201.						
Chatterjee N, Hartge P (2003). Apportioning causes, targeting populations and predicting risks: population attributable fractions. European Journal of Epidemiology, 18(10), p933-5.	Y			N		Mathematical paper commenting of statistical methods adopted for asbestos risks
Churg A (1998). Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. New England Journal of Medicine, 339(14), p999.	Y			N		Comment on Camus et al (1998)
Comba P, Di Paola M, Martuzzi M, Pirastu R (1997). Asbestos-related mortality in Italy: a geographical approach. Medicina del Lavoro, 88(4), p293-301.	N					No exposure estimates and no attempt to differentiate between occupational and environmental exposure
Cugell DW, Kamp DW (2004). Asbestos and the pleura: a review. Chest, 125(3), p1103-17.	Y			N		Medical descriptions of diseases caused by asbestos exposure
Curin K, Saric M, Strnad M (2002). Incidence of malignant pleural mesothelioma in coastal and continental Croatia: epidemiological study. Croatian Medical Journal, 43(4) p498-502.	N					Occupational exposure study
Cvitanovic S, Znaor L, Konsa T, Ivancevic Z, Peric I, Erceg M, Vujovic M, Vukovic J, Beg-Zec Z (2003). Malignant and non-malignant asbestos-related pleural and lung disease: 10-year follow-up study. Croatian Medical Journal, 44(5), p618-25.	Y			N		Follow up of patients with lung and pleural changes to look at correlation between asbestos exposure and disease progression
De Vuyst P, Dumortier P, Gevenois PA (1997). Analysis of asbestos bodies in BAL from subjects with particular exposures. American Journal of Industrial Medicine, 31(6), p699-704.	N					Medical – analysis of asbestos bodies in lungs
Demiroglu H (1998). Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. New England Journal of Medicine, 339(14), p999-1000.	Y			N		Comment on Camus et al (1998)
Demiruglu H (1998). Hazards of white asbestos. Lancet, 352(9124), p322-3.	Y			N		Brief note commenting on carcinogenic potential of chrysotile
Dodson RF, O'Sullivan M, Brooks DR, Hammar SP (2003). Quantitative analysis of asbestos burden in women with mesothelioma. American Journal of Industrial Medicine, 43(2), p188-95.	N					Asbestos tissue burden of women who died of mesothelioma
Du Plessis H (2003). Asbestos's sorrowful legacy: a photoessay. International Journal of Occupational and Environmental Health, 9(3), p236-43.	Y			N		A series of photographs of individuals with mesothelioma in South Africa
Dumortier P, Coplu L, Broucke I, Emri S, Selcuk T, de Maertelaer	N					Medical – lung fibre burden

V, De Vuyst P, Baris I (2001). Erionite bodies and fibres in bronchoalveolar lavage fluid (BALF) of residents from Tuzkoy, Cappadocia, Turkey. Occupational and Environmental Medicine, 58(4), p261-6.						
Dumortier P, Gocmen A, Laurent K, Manco A, De Vuyst P (2001). The role of environmental and occupational exposures in Turkish immigrants with fibre-related disease. European Respiratory Journal, 17(5), p922-7.	Y			N		Asbestos related diseases in Turkish immigrants in Belgium, no epidemiology
Emri S, Akbulut H, Zorlu F, Dincol D, Akay H, Gungen Y, Icli F (2001). Prognostic significance of flow cytometric DNA analysis in patients with malignant pleural mesothelioma. Lung Cancer, 33(2-3), p109-14.	N					Medical – prognosis
Emri S, Demir A, Dogan M, Akay H, Bozkurt B, Carbone M. Baris I (2002). Lung diseases due to environmental exposures to erionite and asbestos in Turkey. Toxicology Letters, 127(1-3), p251-7.	Y			N		Quantitative risks only given for exposure to erionite (asbestos like mineral)
Emri S, Demir AU (2004), Malignant pleural mesothelioma in Turkey, 2000-2002. Lung Cancer, 45 Suppl 1, p17-20.	N					Comparison of characteristics of mesothelioma associated with erionite and asbestos
Emri S, Kocagoz T, Olut A, Gungen Y. Mutti L. Baris YI (2000). Simian virus 40 is not a cofactor in the pathogenesis of environmentally induced malignant pleural mesothelioma in Turkey. Anticancer Research, 20(2A), p891-4.	N					Investigation of SV40 virus as co-factor in relation to mesothelioma
Fano V, Michelozzi P, Ancona C, Capon A, Forastiere F, Perucci CA (2004). Occupational and environmental exposures and lung cancer in an industrialised area in Italy. Occupational and Environmental Medicine, 61(9), p757-63.	Y			N		Asbestos only considered in relation to occupational exposures
Feinstein MB, Bach PB (2000). Epidemiology of lung cancer. Chest Surgery Clinics of North America, 10(4), p653-61.	N					General review of lung cancer incidence in USA
Filiberti R, Montanaro F (2004). Epidemiology of pleural mesothelioma in Italy. Lung Cancer, 45 Suppl 1, p25-7.	Y			N		General discussion of mesothelioma risks in Italy but no relevant quantitative data
Galani V, Constantopoulos S, Manda-Stachouli C, Frangou-Lazaridis M, Mavridis A, Vassiliou M, Dalavanga Y (2002). Additional proteins in BAL fluid of Metsovites environmentally exposed to asbestos: more evidence of "protection" against neoplasia? Chest. 121(1), p273-8.	N					Medical – inidence of mesothelioma in those with and without lung calcifications
Godleski JJ (2004). Role of asbestos in etiology of malignant pleural mesothelioma. Thoracic Surgery Clinics, 14(4), p479-87.	N					Biological study of effects of asbestos on cells
Guidotti TL (2002). Apportionment in asbestos-related disease for purposes of compensation. Industrial Health, 40(4), p295-311.	N					Occupational – workers compensation schemes

Gulmez I, Kart L, Buyukoglan H, Er O, Balkanli S, Ozesmi M (2004). Evaluation of malignant mesothelioma in central Anatolia: a study of 67 cases. Canadian Respiratory Journal, 11(4), p287-90.	Y			N		Consideration of incidence of mesothelioma in Antolia, no quantitative epidemiological results
Gustavsson P, Jakobsson R, Nyberg F, Pershagen G, Jarup L, Scheele P (2000). Occupational exposure and lung cancer risk: a population-based case-referent study in Sweden. American Journal of Epidemiology, 152(1), p32-40.	N					Occupational lung cancer risk factors
Hansen J, de Klerk NH, Musk AW, Hobbs MS (1998). **Environmental exposure to crocidolite and mesothelioma: exposure-response relationships. American Journal of Respiratory and Critical Care Medicine, 157(1), p69-75.**	Y	Y				
Hansen KS, Lauritsen JM, Skytthe A (1996). Cancer incidence among mild steel and stainless steel welders and other metal workers. American Journal of Industrial Medicine, 30(4), p373-82.	N					Not asbestos
Harris LV, Kahwa IA (2003). Asbestos: old foe in 21st century developing countries. Science of the Total Environment, 307(1-3), p1-9.	N					Use of asbestos in developing countries and analysis of research gaps
Heineman EF, Bernstein L, Stark AD, Spirtas R (1996). Mesothelioma, asbestos, and reported history of cancer in first-degree relatives. Cancer, 77(3), p549-54.	N					Cancer in first degree relatives of mesothelioma cases
Hillerdal G (1999). Mesothelioma: cases associated with non-occupational and low dose exposures. Occupational and Environmental Medicine, 56(8), p505-13.	Y			N		Interesting review with summary of background and issues, gives some air concentration values but no useful quantitative risk values
Hiraoka T, Ohkura M, Morinaga K, Kohyama N, Shimazu K, Ando M (1998). Anthophyllite exposure and endemic pleural plaques in Kumamoto, Japan. Scandinavian Journal of Work, Environment and Health, 24(5), p392-7.	N					Prevalance of pleural plaques, no exposure assessment or analysis of occupational vs environmental exposure
Howel D, Arblaster L (2000). Identifying industrial sites with potential for residential exposure to asbestos. Journal of Public Health Medicine, 22(2), p146-8.	N					Identification of industrial sites with potential for residential exposure to asbestos (no exposure data)
Howel D, Arblaster L, Swinburne L, Schweiger M, Renvoize E, Hatton P (1997). Routes of asbestos exposure and the development of mesothelioma in an English region. **Occupational and Environmental Medicine, 54(6), p403-9.**	Y		Y			
Howel D, Gibbs A, Arblaster L, Swinburne L, Schweiger M, Renvoize E, Hatton P, Pooley F (1999). Mineral fibre analysis and routes of exposure to asbestos in the development of	N					Concentrations of fibres in the lungs of cases of mesothlioma and controls

mesothelioma in an English region. Occupational and Environmental Medicine, 56(1), p51-8.						
Hubbard R (1997). The aetiology of mesothelioma: are risk factors other than asbestos exposure important? Thorax, 52(6), p496-7.	N					Not asbestos
Ilg AG, Bignon J, Valleron AJ (1998). Estimation of the past and future burden of mortality from mesothelioma in France. Occupational and Environmental Medicine, 55(11), p760-5.	N					Analysis of mesothelioma rates and projection to future for France
Iscovich J, Fischbein A, Witt-Kushner J, Ginsberg G, Richter E, Tulchinsky T (1999). Malignant mesothelioma in Israel, 1961-1992. International Journal of Occupational and Environmental Health, 5(3), p157-63.	N					Temporal trends in mesothelioma rates in Israel
Isik R, Metintas M, Gibbs AR, Metintas S, Jasani B, Oner U, Harmanci E, Demircan S, Isiksoy S (2001). p53, p21 and metallothionein immunoreactivities in patients with malignant pleural mesothelioma: correlations with the epidemiological features and prognosis of mesotheliomas with environmental asbestos exposure. Respiratory Medicine, 95(7), p588-93.	N					Medical – investigating different immunoactivities for mesotheliomas for occupational vs environmental asbestos exposure
Jockel KH, Ahrens W, Jahn I, Pohlabeln H, Bolm-Audorff U (1998). Occupational risk factors for lung cancer: a case-control study in West Germany. International Journal of Epidemiology, 27(4), p549-60.	N					Occupational lung cancer risk factors
Johansen C (2004). Electromagnetic fields and health effects--epidemiologic studies of cancer, diseases of the central nervous system and arrhythmia-related heart disease. Scandinavian Journal of Work, Environment and Health, 30 Suppl 1, p1-30.	N					Not asbestos
Joshi TK, Gupta RK. Asbestos in developing countries: magnitude of risk and its practical Implications. International Journal of Occupational Medicine and Environmental Health, 17(1), p179-85.	N					Use of asbestos in developing countries
Karakoca Y, Emri S, Bagci T, Demir A, Erdem Y, Baris E, Sahin AA (1998). Environmentally-induced malignant pleural mesothelioma and HLA distribution in Turkey. International Journal of Tuberculosis and Lung Disease, 2(12), p1017-22.	N					Medical – presence of human leukocyte antigens in mesothelioma patients
Kielkowski D, Nelson G, Rees D (2000). Risk of mesothelioma from exposure to crocidolite asbestos: a 1995 update of a South African mortality study. Occupational and Environmental Medicine, 57(8), p563-7.	N					Determination of mesothelioma rates in a South African asbestos mining town – follow up to an original study, no quantitative risks
Kjaergaard J, Andersson M (2000). Incidence rates of malignant mesothelioma in Denmark and predicted future number of cases among men. Scandinavian Journal of Work, Environment and	N					Investigation of temporal changes in mesothelioma rates in Denmark

Health. 26(2), p112-7.						
Kottek M, Kilpatrick D (2002). Malignant mesothelioma from neighborhood exposure to anthophyllite Asbestos. American Journal of Industrial Medicine, 41(6), p514.	Y			N		Brief comment on where anthophyllite asbestos is mined.
Kreienbrock L, Kreuzer M, Gerken M, Dingerkus G, Wellmann J, Keller G, Wichmann HE (2001). Case-control study on lung cancer and residential radon in western Germany. American Journal of Epidemiology, 153(1), p42-52.	N					Not asbestos
Lam WK, White NW, Chan-Yeung MM (2004). Lung cancer epidemiology and risk factors in Asia and Africa. International Journal of Tuberculosis and Lung Disease,8(9), p1045-57.	N					Review of occupational asbestos exposure
Landrigan PJ (1998). Asbestos--still a carcinogen. New England Journal of Medicine, 338(22), p1618-9.	Y			N		Editorial comment on Camus et al (1998)
Landrigan PJ, Lioy PJ, Thurston G, Berkowitz G, Chen LC, Chillrud SN, Gavett SH, Georgopoulos PG, Geyh AS, Levin S, Perera F, Rappaport SM, Small C (2004). NIEHS World Trade Center Working Group. Health and environmental consequences of the world trade center disaster. Environmental Health Perspectives, 112(6), p731-9.	Y			N		World Trade Centre dust measurements including asbestos content
Lange JH (2004). "Mesothelioma trends in the United States: an update based on surveillance, epidemiology, and end results program data for 1973 through 2003". American Journal of Epidemiology, 160(8), p823.	N					Trends in mesothelioma rates
Langer AM (1998). Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. New England Journal of Medicine, 339(14), p1000-1.	Y			N		Comment on Camus et al (1998)
Larsen RI (2003). An air quality data analysis system for interrelating effects, standards, and needed source reductions: Part 13--Applying the EPA Proposed Guidelines for Carcinogen Risk Assessment to a set of asbestos lung cancer mortality data. Journal of the Air and Waste Management Association, 53(11), p1326-39.	N					Developing mathematical model to express asbestos mortality data as function of asbestos concentrations to determine air quality standards
Lash TL, Crouch EA, Green LC (1997). A meta-analysis of the relation between cumulative exposure to asbestos and relative risk of lung cancer. Occupational and Environmental Medicine, 54(4), p254-63.	N					Occupational exposures, meta-analysis of risks
Lauritsen JM (1996). Hansen KS. Lung cancer mortality in stainless steel and mild steel welders: a nested case-referent study. American Journal of Industrial Medicine, 30(4), p383-91.	N					Not asbestos

Lee BW, Wain JC, Kelsey KT, Wiencke JK, Christiani DC (1998). Association of cigarette smoking and asbestos exposure with location and histology of lung cancer. American Journal of Respiratory and Critical Care Medicine, 157(3 Pt 1), p748-55.	N					Medical – location of tumours and histology
Leigh J, Driscoll T (2003). Malignant mesothelioma in Australia, 1945-2002. International Journal of Occupational and Environmental Health, 9(3), p206-17.	Y			N		General epidemiological study of mesothelioma in Australia, the only quantitative information on risks for occupational exposure.
Lippmann M (2003). An air quality data analysis system for interrelating effects, standards, and needed source reductions. Journal of the Air and Waste Management Association, 53(11), p1298-300.	N					Comment on paper on air quality standards
Liu Y, Zhang P, Yi F (2001). Asbestos fiber burdens in lung tissues of Hong Kong Chinese with and without lung cancer. Lung Cancer, 32(2), p113-6.	Y			N		Asbestos fibres in lung tissues of Hong Kong Chinese with and without lung cancer
Luce D, Bugel I, Goldberg P, Goldberg M, Salomon C, Billon-Galland MA, Nicolau J, Quenel P, Fevotte J, Brochard P (2000). Environmental exposure to tremolite and respiratory cancer in New Caledonia: a case-control study. American Journal of Epidemiology, 151(3), p259-65.	Y		Y			
Luo S, Liu X, Mu S, Tsai SP, Wen CP (2003). Asbestos related diseases from environmental exposure to crocidolite in Da-yao, China. I. Review of exposure and epidemiological data. Occupational and Environmental Medicine, 60(1), p35-41.	Y		Y			
Magnani C, Agudo A, Gonzalez CA, Andrion A, Calleja A, Chellini E, Dalmasso P, Escolar A, Hernandez S, Ivaldi C, Mirabelli D, Ramirez J, Turuguet D, Usel M, Terracini B (2000). Multicentric study on malignant pleural mesothelioma and non-occupational exposure to asbestos. British Journal of Cancer, 83(1), p104-11.	Y		Y			
Magnani C, Dalmasso P, Biggeri A, Ivaldi C, Mirabelli D, Terracini B (2001). Increased risk of malignant mesothelioma of the pleura after residential or domestic exposure to asbestos: a case-control study in Casale Monferrato, Italy. Environmental Health Perspectives, 109(9), p915-9.	Y		Y			
Magnani C, Ivaldi C, Botta M, Terracini B (1997). Pleural malignant mesothelioma and environmental asbestos exposure in Casale Monferrato, Piedmont. Preliminary analysis of a case-control study. Medicina del Lavoro, 88(4), p302-9.	Y		Y		N	Preliminary analysis of study reported in full in Magnani et al (2001)

Magnani C, Leporati M (1998). Mortality from lung cancer and population risk attributable to asbestos in an asbestos cement manufacturing town in Italy. Occupational and Environmental Medicine, 55(2), p111-4.	Y		Y			
Magnani C, Mollo F, Paoletti L, Bellis D, Bernardi P, Betta P, Botta M, Falchi M, Ivaldi C, Pavesi M (1998). Asbestos lung burden and asbestosis after occupational and environmental exposure in an asbestos cement manufacturing area: a necropsy study. Occupational and Environmental Medicine, 55(12), p840-6.	Y			N		Study of presence of histological asbestosis, lung burden and asbestos fibres and asbestos bodies in necropsy samples
Manda-Stachouli C, Dalavanga Y, Daskalopoulos G, Leontaridi C, Vassiliou M, Constantopoulos SH (2004). Decreasing prevalence of pleural calcifications among Metsovites with nonoccupational asbestos exposure. Chest, 126(2), p617-21.	Y			N		Pleural calcifications related to asbestos exposure, no quantitative risks
Marchevsky AM, Wick MR (2003). Current controversies regarding the role of asbestos exposure in the causation of malignant mesothelioma: the need for an evidence-based approach to develop medicolegal guidelines. Annals of Diagnostic Pathology, 7(5), p321-32.	N					Review of current controversies regarding role of asbestos exposure in causation of mesothelioma
Marinaccio A, Nesti M (2003). Regional Operational Centers. Analysis of survival of mesothelioma cases in the Italian register (ReNaM). European Journal of Cancer, 39(9), p1290-5.	N					Mesothelioma survival rates
Marsh GM, Stone RA, Esmen NA, Gula MJ, Gause CK, Petersen NJ, Meaney FJ, Rodney S, Prybylski D (1997). A case-control study of lung cancer mortality in six Gila Basin, Arizona smelter towns. Environmental Research, 75(1), p56-72.	N					Study of impact of smelter emissions on lung cancer mortality, only mention of asbestos for occupational exposure
Martuzzi M, Comba P, De Santis M, Iavarone I, Di Paola M, Mastrantonio M, Pirastu R (1998). Asbestos-related lung cancer mortality in Piedmont, Italy. American Journal of Industrial Medicine, 33(6), p565-70.	N					Association between pleural neoplasm mortality (as possible proxy for asbestos exposure) and lung cancer mortality
Maynard C (2004). Asbestos problems in Montana and California. Environmental Science and Technology, 38(3), p46A.	Y			N		Brief comments on the situation regarding the clean-up of asbestos in Libby Montana and El Dorado, California
Melato M, Rizzardi C (2001). Malignant pleural mesothelioma following chemotherapy for breast cancer. Anticancer Research, 21(4B), p3093-6.	N					Medical – mesothelioma not related to asbestos
Metintas M, Metintas S, Ucgun I, Gibbs AR, Harmanci E, Alatas F, Erginel S, Tel N, Pasaoglu O (2001). Prognostic factors in diffuse malignant pleural mesothelioma: effects of pretreatment clinical and laboratory characteristics. Respiratory Medicine, 95(10), p829-35.	N					Medical - effects of pre-treatment clinical and laboratory characteristics on survival

Metintas M, Ozdemir N, Hillerdal G, Ucgun I, Metintas S, Baykul C, Elbek O, Mutlu S, Kolsuz M (1999). Environmental asbestos exposure and malignant pleural mesothelioma. Respiratory Medicine, 93(5), p349-55.	Y			N		Study of environmentally exposed population, no useful quantitative risks presented
Metintas S, Metintas M, Ucgun I, Oner U (2002). Malignant mesothelioma due to environmental exposure to asbestos: follow-up of a Turkish cohort living in a rural area. Chest. 122(6), p2224-9.	Y		Y			
Mizuki M, Yukishige K, Abe Y, Tsuda T (1997). A case of malignant pleural mesothelioma following exposure to atomic radiation in Nagasaki. Respirology, 2(3), p201-5.	N					A single mesothelioma case with radiation and asbestos exposure
Montanaro F, Bray F, Gennaro V, Merler E, Tyczynski JE, Parkin DM, Strnad M, Jechov'a M, Storm HH, Aareleid T, Hakulinen T, Velten M, Lef'evre H, Danzon A, Buemi A, Daur'es JP, Menegoz F, Raverdy N, Sauvage M, Ziegler H, Comber H, Paci E, Vercelli M, De Lisi V, Tumino R, Zanetti R, Berrino F, Stanta G, Langmark F, Rachtan J, Mezyk R, Blaszczyk J, Ivan P, Primic-Zakelj M, Martinez AC, Izarzugaza I, Borras J, Garcia CM, Garau I, Sanchez NC, Aicua A, Barlow L, Torhorst J, Bouchardy C, Levi F, Fisch T, Probst N, Visser O, Quinn M, Gavin A, Brewster D, Mikov M. ENCR Working Group. (2003) Pleural mesothelioma incidence in Europe: evidence of some deceleration in the increasing trends. Feb;15(1):103]. Cancer Causes and Control, 14(8), p791-803.	N					Review of pleural mesothlioma rates in Europe: geographical distribution and temporal trends
Morgan RW, Goodman M (1998). Non-occupational exposure to chrysotile asbestos and the risk of lung cancer. New England Journal of Medicine, 339(14), p1001.	Y			N		Comment on Camus et al (1998)
Muller KM, Fischer M (2000). Malignant pleural mesotheliomas: an environmental health risk in southeast Turkey. Respiration, 67(6), p608-9.	Y			N		Brief description of environmental health risk from naturally occurring asbestos in Southeast Turkey
Musti M, Cavone D, Aalto Y, Scattone A, Serio G, Knuutila S (2002). A cluster of familial malignant mesothelioma with del(9p) as the sole chromosomal anomaly. Cancer Genetics and Cytogenetics, 138(1), p73-6.	N					Medical – genetics of familial cluster of mesothelioma
Mzileni O, Sitas F, Steyn K, Carrara H, Bekker P (1999). Lung cancer, tobacco, and environmental factors in the African population of the Northern Province, South Africa. Tobacco Control, 8(4), p398-401.	Y		Y			
Nesti M, Marinaccio A, Chellini E (2004). Malignant mesothelioma in Italy, 1997. American Journal of Industrial Medicine, 45(1), p55-62.	Y			N		Mesothelioma cases in various Italian areas – looking at general trends but no useful quantitative epidemiological information

Niklinski J, Niklinska W, Chyczewska E, Laudanski J, Naumnik W, Chyczewski L, Pluygers E (2004). The epidemiology of asbestos-related diseases. Lung Cancer, 45 Suppl 1, S7-S15.	Y			N		Review of occupational and environmental epidemiological studies (with some data for Poland) but no relevant quantitative results
Oksuzoglu B, Yalcin S, Erman M, Dagdelen S (2002). Leptomeningeal infiltration of malignant mesothelioma. Medical Oncology, 19(3), p167-9.	Y			N		Report on the treatment of a patient with mesothelioma
Ordonez NG (2000) . Epithelial mesothelioma with deciduoid features: report of four cases. American Journal of Surgical Pathology, 24(6), p816-23.	N					Medical – morphological variants in mesothelioma
Orenstein MR and Schenker MB (2000). Environmental asbestos exposure and mesothelioma. Current Opinion in Pulmonary Medicine, 6(4), p371-7.	Y		Y	N		Review
Ozer N, Shehu V, Aytemir K, Ovunc K, Emre S, Kes S (2000). Echocardiographic findings of pericardial involvement in patients with malignant pleural mesothelioma with a history of environmental exposure to asbestos and erionite. Respirology, 5(4), p333-6.	N					Medical - pericardial involvement in mesothelioma
Panetta A, Geminiani ML (2003). Mesothelioma following exposure to asbestos used in sugar refineries: report of two cases and review of the literature. Tumori, 89(5), p573-4.	Y			N		Report of two cases of mesothelioma following exposure to asbestos in a sugar refinery – worker and his wife.
Paustenbach DJ, Finley BL, Lu ET, Brorby GP, Sheehan PJ (2004). Environmental and occupational health hazards associated with the presence of asbestos in brake linings and pads (1900 to present): a "state-of-the-art" review. Journal of Toxicology and Environmental Health Part B: Critical Reviews. 7(1), p25-80.	N					Predominantly occupational (states brakes don't contribute significantly to asbestos in air)
Petrini MF (1998). Cigarette smoking, asbestos exposure, lung cancer, and sample size. American Journal of Respiratory and Critical Care Medicine, 158(5 Pt 1), p1688.	N					Comment on medical paper on tumour location and histology
Piirila P, Lehtola H, Zitting A, Kivisaari L, Koskinen H, Luukkonen R, Salo SP, Vehmas T, Nordman H, Sovijarvi AR (2000). Lung sounds in asbestos induced pulmonary disorders. European Respiratory Journal, 16(5), p901-8.	N					Medical – lung sounds in patients
Powers A, Carbone M (2002). The role of environmental carcinogens, viruses and genetic predisposition in the pathogenesis of mesothelioma. Cancer Biology and Therapy, 1(4), p348-53.	N					Medical – review of role of various factors in the development of mesothelioma
Qureshi RA, Gazney JA, Soorae AS (2002). Rare pigmated tumor of the lung. JPMA - Journal of the Pakistan Medical Association, 52(4), p183-5.	N					Medical – single case

Ramanathan AL, Subramanian V (2001). Present status of asbestos mining and related health problems in India--a survey. Industrial Health, 39(4), p309-15.	N					Occupational exposures to asbestos
Ramazzini C (1999). Call for an international ban on asbestos. Journal of Occupational and Environmental Medicine, 41(10), p830-2.	N					Call for asbestos ban
Rees D, Goodman K, Fourie E, Chapman R, Blignaut C, Bachmann MO, Myers J (1999). Asbestos exposure and mesothelioma in South Africa. South African Medical Journal. Suid-Afrikaanse Tydskrif Vir Geneeskunde, 89(6), p627-34.	Y			N		Study of the asbestos exposures of patients diagnosed with mesothelioma, no useful quantitative information on risks.
Rees D, Myers JE, Goodman K, Fourie E, Blignaut C, Chapman R, Bachmann MO (1999). Case-control study of mesothelioma in South Africa. American Journal of Industrial Medicine, 35(3), p213-22.	Y		Y			
Renner R (2003). Asbestos investigation under way. Environmental Science and Technology, 37(23), p426A-428A.	Y			N		Brief comments on asbestos exposure investigations underway in Libby, Montana and El Dorado, California
Richter ED, Chlamtac N, Berman T, Laster R (2001). A review of environmental and occupational exposure to asbestos in Israel. Public Health Reviews, 29(2-4), p247-64.	N					Review of history of asbestos use in Israel and the management of exposures, no epidemiology
Robinson BW, Creaney J, Lake R, Nowak A, Musk AW, de Klerk N, Winzell P, Hellstrom KE, Hellstrom I (2003). Mesothelin-family proteins and diagnosis of mesothelioma. Lancet, 362(9396), p1612-6.	N					Medical – identification of tumour markers
Rogan WJ, Ragan NB, Dinse GE (2000). X-ray evidence of increased asbestos exposure in the US population from NHANES I and NHANES II, 1973-1978. National Health Examination Survey. Cancer Causes and Control, 11(5), p441-9.	N					Medical – X-rays of pleural thickening linked to asbestos
Rom WN, Hammar SP, Rusch V, Dodson R, Hoffman S (2001). Malignant mesothelioma from neighborhood exposure to anthophyllite asbestos. American Journal of Industrial Medicine, 40(2), p211-4.	Y			N		Report of an individual with pleural mesothelioma who lived and worked near an asbestos facility.
Rosenthal R, Langer I, Dalquen P, Marti WR, Oertli D (2003). Peritoneal mesothelioma after environmental asbestos exposure. Swiss Surgery, 9(6), p311-4.	Y			N		Report of a Turkish patient with peritoneal mesothelioma
Sakellariou K, Malamou-Mitsi V, Haritou A, Koumpaniou C, Stachouli C, Dimoliatis ID, Constantopoulos SH (1996). Malignant pleural mesothelioma from nonoccupational asbestos exposure in Metsovo (north-west Greece): slow end of an epidemic? European Respiratory Journal, 9(6), p1206-10.	Y			N		Considers changes in mesothelioma incidence with time following the decline in the use of naturally occurring asbestos-containing materials “luto”

Saric M, Curin K (1996). Malignant tumours of the gastrointestinal tract in an area with an asbestos-cement plant. Cancer Letters, 103(2), p191-9.	Y			N		GI tract tumours
Schneider J, Rodelsperger K, Bruckel B, Kayser K, Woitowitz HJ (1998). Environmental exposure to tremolite asbestos: pleural mesothelioma in two Turkish workers in Germany. Reviews on Environmental Health, 13(4), p213-20.	N					Two cases, no exposure or risk assessment
Schneider J, Straif K, Woitowitz HJ (1996). Pleural mesothelioma and household asbestos exposure. Reviews on Environmental Health, 11(1-2), p65-70.	N			N		Paraoccupational exposures
Seniori Costantini A, Chellini E (1997). The experience of the Mesothelioma Registry of Tuscany in assessing health hazard associated with asbestos exposure. Medicina del Lavoro, 88(4), p310-5.	N					Occupational exposures
Senyigit A, Babayigit C, Gokirmak M, Topcu F, Asan E, Coskunsel M, Isik R, Ertem M (2000). Incidence of malignant pleural mesothelioma due to environmental asbestos fiber exposure in the southeast of Turkey. Respiration. 67(6), p610-4, 2000.	Y			N		No useful quantitative risk information
Senyigit A, Bayram H, Babayigit C, Topcu F, Balci AE, Satici O (2000). Comparison of the effectiveness of some pleural sclerosing agents used for control of effusions in malignant pleural mesothelioma: a review of 117 cases. Respiration, 67(6), p623-9.	N					Medical – mesothelioma treatments
Senyigit A, Bayram H, Babayigit C, Topcu F, Nazaroglu H, Bilici A, Leblebici IH (2000). Malignant pleural mesothelioma caused by environmental exposure to asbestos in the Southeast of Turkey: CT findings in 117 patients. Respiration, 67(6), p615-22.	N					Medical – diagnostic technique
Sokas RK (1998). Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. New England Journal of Medicine, 339(14), p1000.	Y					Comment on Camus et al (1998)
Stern F, Lehman E, Ruder A (2001). Mortality among unionized construction plasterers and cement masons. American Journal of Industrial Medicine, 39(4), p373-88.	N					Occupational exposures
Stewart DJ, Edwards JG, Smythe WR, Waller DA, O'Byrne KJ (2004). Malignant pleural mesothelioma--an update. International Journal of Occupational and Environmental Health, 10(1), p26-39.	N					Medical diagnosis and treatment
Szeszenia-Dabrowska N, Wilczynska U, Szymczak W, Laskowicz K (1998). Environmental exposure to asbestos in asbestos cement workers: a case of additional exposure from indiscriminate	N					Occupational exposures

use of industrial wastes. International Journal of Occupational Medicine and Environmental Health, 11(2), p171-7.						
Trent T (2004). Asbestos problems in Montana and California Environmental Science and Technology, 38(3), p46A.	Y			N		Brief comments on the situation regarding the clean-up of asbestos in Libby, Montana and El Dorado, California
Trosic I (2001). Fate of the miraculous mineral--ban asbestos worldwide campaign. Collegium Antropologicum, 25(2), p713-8.	N					General review of use and dangers of asbestos
Vacek PM (1997). Assessing the effect of intensity when exposure varies over time. Statistics in Medicine, 16(5), p505-13.	N					Analysis of asbestos exposure response models, occupational example
Valic F (2002). The asbestos dilemma: I. Assessment of risk. Arhiv Za Higijenu Rada i Toksikologiju, 53(2), p153-67.	Y			N		Review, mainly concerned with occupational exposures
Wang LI, Neuberg D, Christiani DC (2004). Asbestos exposure, manganese superoxide dismutase (MnSOD) genotype, and lung cancer risk. Journal of Occupational and Environmental Medicine. 46(6), p556-64.	N					Genetic susceptibility to asbestos induced lung cancer
Wang X, Christiani DC, Mark EJ, Nelson H, Wiencke JK, Gunn L, Wain JC, Kelsey KT (1999). Carcinogen exposure, p53 alteration, and K-ras mutation in synchronous multiple primary lung carcinoma. Cancer, 85(8), p1734-9.	N					Medical – lung cancer no mention of asbestos
Wang XD, Liu C, Bronson RT, Smith DE, Krinsky NI, Russell M (1999). Retinoid signaling and activator protein-1 expression in ferrets given beta-carotene supplements and exposed to tobacco smoke. Journal of the National Cancer Institute, 91(1), p60-6.	N					Not asbestos
Weir NA, Gerstenhaber B (2001). A case of pleural mesothelioma with effusive-constrictive pericarditis. Yale Journal of Biology and Medicine, 74(3), p159-63.	N					Medical – case report
Xu X, Kelsey KT, Wiencke JK, Wain JC, Christiani DC (1996). Cytochrome P450 CYP1A1 MspI polymorphism and lung cancer susceptibility. Cancer Epidemiology, Biomarkers and Prevention, 5(9), p687-92.	N					Medical – genetics
Xu Z, Pan GW, Liu LM, Brown LM, Guan DX, Xiu Q, Sheng JH, Stone BJ, Dosemeci M, Fraumeni JF Jr, Blot WJ (1996). Cancer risks among iron and steel workers in Anshan, China, Part I: Proportional mortality ratio analysis. American Journal of Industrial Medicine, 30(1), p1-6.	N					Occupational exposure
Yates DH, Corrin B, Stidolph PN, Browne K (1997). Malignant mesothelioma in south east England: clinicopathological experience of 272 cases. Thorax, 52(6), p507-12.	N					Medical – clinical features of mesothelioma
Yeung P, Rogers A, Johnson A (1999). Distribution of	N					Occupational exposure

mesothelioma cases in different occupational groups and industries in Australia, 1979-1995. Applied Occupational and Environmental Hygiene 14(11), p759-67.						
Zellos L, Christiani DC (2004). Epidemiology, biologic behaviour, and natural history of mesothelioma. Thoracic Surgery Clinics, 14(4), p469-77.	N					Medical - biological behaviour and prognosis
Zeren EH, Gumurdulu D, Roggli VL, Zorludemir S, Erkisi M, Tuncer I (2000). Environmental malignant mesothelioma in southern Anatolia: a study of fifty cases. Environmental Health Perspectives, 108(11), p1047-50.	N					Medical – clinical features of mesothelioma

Notes:

* References in bold are those for which detailed reviews were undertaken.

+ Inclusion and exclusion criteria -

Stage 1: Review of abstracts. Papers excluded from further consideration if were not on asbestos, or related solely to occupational exposure or 'medical' ie describing diagnosis, characteristics and treatment.

Stage 2: Papers were obtained for all abstracts included after Stage 1. The 65 papers were then reviewed in turn to establish their relevance to the objective of summarising quantitative risk factors for environmental exposures to asbestos. To this end each paper was examined and put into 1 of the following 3 categories:

Category 1 - Epidemiological study relating to environmental asbestos exposures with clear quantitative asbestos exposure levels (eg in terms of f/ml). There were only a few papers with any quantitative exposure data.

Category 2 - Epidemiological study relating to environmental asbestos exposures with no clear quantitative asbestos exposure levels (eg exposure may be related to a surrogate, such as distance from an asbestos plant).

Category 3 - Non epidemiological study (eg case series), or epidemiological study on purely occupational or paraoccupational exposures, or epidemiological study on environmental asbestos exposures with insufficient quantitative data (ie no risks provided).

The 49 papers in category 3 were excluded from further consideration

Stage 3: Papers excluded at this stage if they were review papers or preliminary results of a study addressed in one of the other papers.