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AD Jones, JW Cherrie, H Cowie and A Soutar

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The presence of asbestos in the filling material in farm tracks and rights of way (ROW) in South Cambridgeshire leads to the possibility that residents and other users may be exposed to airborne asbestos fibres. Measurements have shown that concentrations of airborne asbestos fibres are low, but there is no known threshold of exposure below which there is absolutely no risk. Therefore, this study was undertaken to provide a quantitative estimate of the risk of lung cancer or mesothelioma being caused by exposure to airborne asbestos fibres from the ROW. The study was commissioned by the Cambridgeshire County Council and South Cambridgeshire District Council.

From measurements of concentrations of airborne asbestos, we constructed estimates of the annual average concentrations for six ROW. These ROW had been selected in consultation with the Councils as being informative about the issues involved in dealing with the asbestos on ROW in South Cambridgeshire. Previous studies informed the choice and the chosen tracks included those previously identified as posing relatively higher risk.

Airborne asbestos creates a risk to health if it is inhaled. Estimates of the amount of time that residents could or would spend on the ROW, combined with the likely concentrations of asbestos in air, gave estimates of the annual cumulative exposures. Worst case scenarios provided estimates of risk that are possible but unlikely. A realistic scenario indicated the more likely level of risk from long term residence and use of the ROW.

The worst case estimates indicate the asbestos exposure on the ROW is unlikely to cause any deaths of the residents. However, for some of the ROW, the worst case predictions exceeded the upper boundary (of 10 in 100,000) of consensus values for the level of lifetime excess risk that could be deemed acceptable.

Under the realistic exposure estimation for regular use of the ROW by residents of houses accessed from the ROW, all predicted lifetime excess risks were at or below that boundary level of acceptability and it is very unlikely that the health of any resident will be affected.

The predicted risks have been compared to other more familiar risks. For example, the risks from smoking are much higher than those from the worst case scenario for exposure to airborne asbestos from the ROW.

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SUMMARY

Introduction

The presence of asbestos-containing materials as road filling in farm tracks and rights of way (jointly referred to as ROW in this report) gives rise to the possibility that people may be exposed to airborne asbestos fibres and that has led to a concern over the possible consequent risks to health. Evidence from previous sampling exercises conducted on the ROW showed low concentrations of airborne asbestos fibres.

There are well established relationships between exposure to asbestos and incidence of asbestos-related disease, derived from studies on workers exposed for decades at concentrations much higher than those on the ROW (HEI, 1991). The risks diminish as the amount of exposure decreases, but there is no known threshold of exposure to asbestos below which there is absolutely no risk. Therefore the current study was undertaken to provide a quantitative estimate of the risk that could arise to residents or other persons regularly using the ROW.

The aims were to develop estimates of annual average concentration (fibres/ml) of airborne asbestos fibre concentrations, then to combine those concentrations with information and suppositions on the amount of time that people could or would spend on the ROW to estimate annual average exposure (fibre.hours/ml). Worst case and realistic scenarios were considered. From the exposure levels, we predicted lifetime excess risk of lung cancer or mesothelioma due to exposure to airborne asbestos fibres on the ROW. This calculation used accepted mathematical models of the relationship between exposure to airborne asbestos fibres and risk of disease (HEI, 1991).

The study produces estimates for six ROW, Moor End Lane, Shepreth; Newling Non-lets, Melbourn; London Way, Melbourn; Shedbury Lane, Bassingbourn; Hill Top Farm, Croydon; Whaddon Estate (Fountain Farm).

Methods and materials

Airborne asbestos fibre concentrations were available from samples taken during September 2004. These were average concentrations over week-long periods, for four weeks, on the six ROW. Ninety air samples were evaluated by scanning electron microscopy to count asbestos fibres.

At one ROW (Newling Non-lets), the asbestos fibres were identified as mainly amosite (brown asbestos), whereas the fibres on the other five ROW were a mixture of chrysotile (white asbestos) and amosite. There were also measurements of airborne asbestos fibre concentrations from June 2001 (on London Way) and July 2004 (on Moor End Lane).

Data on weather conditions (daily rainfall, windspeed and direction, hours of sunshine etc) were obtained from a weather station (Iceni, Royston) within a few kilometres of the ROW.

A simple but adequate model of the influence of weather was developed by expressing a logical dependence as a mathematical relationship and adjusting two coefficients in the model to predict the observed changes in airborne fibre levels from week to week during the four weeks of sampling in September 2004. The validity of the model was checked by demonstrating consistency with the fibre concentrations measured in July 2004 and June

2001. The model was then used to calculate the annual average concentration of airborne asbestos fibres for each of the ROW.

Scenarios of possible or realistic exposure were based on estimates of the amount of time that residents might spend on the ROW. These estimates of time spent on the ROW were based on observations during visits in the sampling exercise, interview information gathered by Cambridgeshire County Council, reasonable suppositions (which overestimated rather than underestimated true exposure), and the limitations of what would be physically possible.

We predicted the risk of lung cancer and mesothelioma, separately and in total, for a range of scenarios, and for exposure from ages of 5, 20, 40 and 60. The risks were predicted for the individual, under three variations of the worst case scenario (i.e. exposure continuing for 5 years, 20 years or a lifetime from initial ages of 5, 20, 40 and 60). The probability of any resident developing a cancer from the estimated exposure was calculated, based on the approximate number of residents and their approximate age distribution.

For mesothelioma, the predicted risk increases with both amount of exposure and with time from start of exposure. Because of the dependence on time, the age at commencement of exposure affects the level of risk; predicted risk is lower if exposure starts at a later age. The predicted risk is higher for amosite than for the same concentration of amosite and chrysotile.

Lung cancer is caused mainly by smoking, and it is widely recognised that smokers have much higher risk of lung cancer than non-smokers. Exposure to asbestos produces an increase in that existing risk and the relative increase is proportional to the cumulative asbestos exposure. The relative increase in existing risk is approximately the same for smokers and non-smokers, but for non-smokers, a small increase on a low existing rate gives a very small absolute increase in risk. For smokers, a small increase in a high existing rate means that their risk of lung cancer is still mainly due to smoking but the increase affects the total excess risk from asbestos on the ROW.

Lung cancer risk also depends on asbestos type, with higher risk for amosite asbestos than for chrysotile asbestos for given cumulative exposure (fibre.hours/ml).

Influence of weather and annual average concentrations

The 24-hour average airborne asbestos concentration appears to vary with rainfall, dilution in proportion to mean windspeed, and hours of sunshine. We developed a relatively simple model allowing for these factors which was sufficient to extrapolate from measured weekly average concentrations to annual average concentrations. As would be expected, concentrations are highest on dry, calm, sunny days.

The estimated annual average concentrations of airborne asbestos fibres for the ROW range from

- 0.0002 fibres/ml (Hill Top Farm, and Whaddon Estate), through
- 0.0005 fibres/ml (Shedbury Lane, London Way, and Newling Non-lets), to
- 0.002 fibres/ml (Moor End Lane).

These concentrations may be compared with those summarised by HEI for levels in other circumstances:

- background asbestos fibres in outdoor rural air 0.00001 fibres/ml;
- outdoor urban air, 0.0001 asbestos fibres/ml;
- in well maintained buildings with asbestos in good undamaged condition, 0.00002 fibres/ml.

This comparison shows that, for example, the annual average concentrations on Hill Top Farm and Whaddon Estate are slightly higher than a concentration reported for urban air.

On the ROW, the airborne asbestos fibre concentration may vary during the day and is likely to be highest when there is dust disturbance by traffic, as shown by previous sampling. We used the estimation of the difference between day time levels and night time levels, with some suppositions about the possible peak flows of traffic at certain times of day, to account for these possible influences of time of day on exposures.

Estimated exposures

The measured concentrations at the side of the track were based on sampling for 24-hours of the day thus producing an estimate of a *potential cumulative exposure*. Since people cannot possibly spend all 24-hours at the trackside, we took the physically possible worst case as being exposure equal to half that of the 24-hour potential cumulative exposure. With only limited information about either the amount of time spent on the track by most residents or the pattern of traffic flow, the worst case scenario is a useful starting point. Illustration of more realistic conditions suggested that actual exposures might be a third or a twelfth of this level, or less.

Predicted Risks

The predicted excess lifetime risks of mesothelioma, lung cancer or either from the worst case scenario of exposure are presented, for each ROW, for males and females, and for exposures commencing at ages 5, 20, 40 or 60. Under the scenario of the worst case exposure continuing for a lifetime, the predicted lifetime excess risks range from 0.1 in 100,000 to 90 in 100,000. The lifetime risks are dependent on amount of exposure and on age at first exposure, with exposure starting at a younger age leading to higher risk.

The predicted risks demonstrate the significance of the differences in concentrations of airborne asbestos between the six ROW, and also the difference expected for type of asbestos (with amosite at Newling Non-lets).

The predictions for three durations of exposure demonstrate the different influences of exposure duration and age at which exposure commences. For given exposure, the exposure from youngest age gave, as expected, the highest risks.

The predicted excess lifetime risks from the scenario which is believed to be more akin to real exposures indicates risk between 0.01 in 100,000 to 10 in 100,000. The level of risk is affected by the persons age at the start of exposure, and the higher values are for someone exposed from age 5.

Based on age profiles from census data for the adjacent villages, we estimated the likely age profile for residents on the ROW. From the number of houses accessed from the ROW, we estimated the population that, as residents, might have exposure as frequent as that in the realistic scenario. With these assumptions, the chance that any of their deaths would be

attributable to a mesothelioma or lung cancer caused by exposure to airborne asbestos fibres from the ROW was estimated as 0.001.

Where people spend less time on the ROW, then their exposure will be correspondingly less and the consequent risks proportionately lower.

Criteria for designation of contaminated land

The guidance from the Department for Environment, Food and Rural Affairs (DEFRA) is summarised. Essentially, the advice is that where a contaminant could cause significant harm (such as cancer) then there is a need to make a quantitative risk assessment (QRA) to judge whether there is a “significant possibility” of that harm being caused.

The advice also recognises that in applying QRA, there is a need to take a view on the “acceptable” level of numerical estimate. Various authorities have offered guidance on levels of risk that might be regarded as “acceptable”, and DEFRA see 10 in 100,000 as the upper boundary for “acceptable” lifetime risk.

“The estimate of risk from a source such as a contaminated site, and an understanding of the uncertainties inherent in the estimate, can only help to guide decisions on action, which inevitably depend also on factors such as practicality, cost and competing priorities.”

Discussion

The reliability of the estimates of concentration is considered to be good. The derivation of annual average relies on a simple but adequate model of the influence of weather. Arguably, the model might overestimate the real concentrations for cold winter days.

Our expectation is that the main pathway for exposure would be direct exposure to airborne fibres on the ROW. We would expect that ingress of airborne fibres, or transfer on feet of residents, would be an insignificant pathway for eventual exposure to airborne fibres. There is however, only limited direct data to confirm that expectation.

The estimates of amount of time spent on the tracks is likely to overestimate rather than underestimate exposure.

Conclusions

There are some clear differences among the six ROW in the level of predicted total excess risk of cancer. Under the worst case scenario, the duration of exposure needed to produce predicted risks of 10 in 100,000 differed between the ROW.

- With the worst case scenario continuing for a lifetime, the predicted risks at two of the ROW (Whaddon Estate and Hill Top Farm) were of the order that has been described as generally “acceptable” i.e. below 10 in 100,000.
- With the worst case scenario continuing for 20 years, the predicted risks reach the 10 in 100,000 level for London Way and Shedbury Lane;
- With the worst case level of exposure continuing for 5 years, predicted risks reach approximately the 10 in 100,000 level for Moor End Lane and Newling Non-lets.

A more realistic estimate of the likely risk was based on interview information about the pattern of activity of the farmers using two of the ROW. This amount of exposure may also be likely to be more realistic for many of the residents. With this estimate of likely exposure, the predicted lifetime total excess risks were at or below 10 in 100,000 for all the six ROW. The risks of mesothelioma were all at or below 4 in 100,000.

From the risks predicted for a realistic estimate of exposure and with the approximation that there could be 100 residents living in houses accessed from the six ROW, we estimated the chance that any of their eventual deaths would arise from the asbestos exposure as being about 0.001. That value compares with the expectation that, there would be 2 or 3 smoking-related lung cancers deaths among a typical group of 100 people without asbestos exposure (i.e. assuming that the proportion of smokers is the same as the UK average).

The risks predicted for the ROW are generally low enough that they may be regarded as low compared to many other more commonplace risks. However, under assumptions of “worst case” exposure continuing for many years, predicted risks can reach the boundary level for acceptable risk for some of these ROW.

In summary, the current levels of airborne asbestos fibre concentrations would not lead to risks above about 1 in 100,000 unless a person spends a very high proportion of his/her time on the ROW during the dry (dusty) days of the year and does so for several years.

Main recommendations

The estimated asbestos exposures are low enough that they are unlikely to result in any deaths. However, decisions on whether to designate or treat as contaminated land have to take the level of risk to individuals into account in judging practicality, cost and competing priorities.

The predictions of risk are based upon the presumption that traffic activity continues to be at about the same level. It would be useful to have a record of current traffic levels so that future changes can be recognised. If traffic flows increase substantially in the future, then it would become appropriate to re-assess the likely risks.

1 INTRODUCTION

The presence of asbestos containing material as a filling material in farm tracks and rights of way (jointly called ROW in this report) has led to concern about the possible risks to health from exposure to airborne asbestos. The Cambridgeshire County Council and South Cambridgeshire District Council commissioned the Institute of Occupational Medicine (IOM) to undertake a study to predict the future risks to health due to exposure to airborne asbestos from the asbestos on the tracks.

In this report, we describe the available data on airborne concentrations of asbestos fibres, and the development of a simple model of the effects of weather on the relative levels of these concentrations to enable extrapolation to predict the annual average concentrations of airborne asbestos. Annual average concentrations are the necessary information needed to produce good estimates of the risks to health from asbestos exposure for residents living adjacent to the tracks, or for regular users of the tracks.

Data on the airborne asbestos concentrations had been gathered during two sampling studies conducted in 2004. The first, in July, was a pilot study to establish the techniques and procedures and it was conducted at two sites (Moor End Lane, Shepreth; and Hill Farm, Littleington). The second study, in September, obtained measurements of airborne asbestos fibre concentrations at six of the ROW in South Cambridgeshire:

- Shedbury Lane, Basingbourn cum Kneesworth;
- Moor End Lane, Shepreth;
- Newling Non-lets (including Solway Farm);
- London Way, Melbourn;
- Hill Top Farm, Croydon;
- Whaddon Estate Farm (Fountain Farm).

The sampling on these six ROW measured concentrations over four 7-day periods, commencing 6th September 2004 when it was anticipated that there would be vehicular activity associated with harvesting on some tracks which had been delayed due to wet weather earlier that summer.

As part of the second study, samples of dust from the track surface were analysed for the presence and type of asbestos. These data supported the measurements of type of asbestos in the air samples.

Data on weather conditions were obtained from a local weather station (Iceni, in Royston which is only a few km from these ROW).

The annual average concentrations together with estimates of the amount of time that residents are likely to spend on or near the tracks enable exposure scenarios to be constructed. The report describes the prediction of risk for these exposure scenarios. The relationship between exposure and risk to health originates from studies on occupationally exposed groups whose exposure lasted for decades at concentrations much higher (several orders of magnitude higher) than those measured on the ROW. The use of such models to predict

health risks from low levels of exposure is well established. For example, the prestigious Health Effects Institute (HEI) in the USA used these models to predict the risk to members of the public in public and commercial buildings with asbestos-containing materials (HEI, 1991).

Inhalation of asbestos can, in certain circumstances, cause asbestosis, lung cancer or mesothelioma of the pleura / peritoneum. Asbestosis is a disease that is associated with heavy exposure to asbestos, and is not relevant to the levels of exposure that could arise from asbestos contamination on the ROW. The risk of incidence of the cancers (lung cancer or mesothelioma) increases with increasing exposure to asbestos. Lower exposure causes lower risk, but there is no known threshold below which there is no risk.

The report:

- describes and explains the results of the calculations of exposure and risk;
- discusses the findings, including
 - the reliability of the results,
 - the consistency of the findings with other information;
 - comparisons with other risks and guidelines on acceptability of risks;
- draws conclusions and makes recommendations.

2 AIMS

The overall aim of the study was to assess the likely exposure to airborne asbestos fibres, arising from asbestos containing material on the farm tracks and rights of way (ROW) in South Cambridgeshire, and hence estimate the consequent risks to health.

The specific objectives were to:

- use the measurements of airborne asbestos fibre concentrations made on six ROW in September 2004, combined with annual weather data from a weather station located near the ROW, to estimate average concentrations over the course of a year;
- develop, in consultation with council staff, good descriptions of the typical time spent on or adjacent to the ROW by residents and/or users of the track, and use these to define typical and representative exposure scenarios;
- combine estimated airborne asbestos concentrations (for the ROW) with the scenarios (of time spent on or near the ROW) to estimate annual average exposure concentrations for each exposure scenario;
- make suitable allowance for effect of differences in weather conditions from year to year on the annual average asbestos fibre concentrations, and hence on the predicted future exposure on the ROW ;
- predict estimated future lifetime risk of disease being caused by the exposure in the scenarios, on each of the six ROW.

The predicted risks were to be expressed in two forms: the risk to individuals and the risk to the population of residents as a whole. Estimates of the risk to an individual living or working on the ROW in a selected scenario may be the most informative for individuals. The risk to the population examines the risk that incidence of disease, due to the asbestos on the ROW, will be seen in any of the exposed population.

3 METHODS AND MATERIALS

3.1 INTRODUCTION

In this chapter we outline briefly the process of producing estimates of exposure and consequent risk.

3.2 DATA

3.2.1 Airborne fibre concentration

Airborne fibre concentrations have been measured previously. These include measurements by IOM at the trackside of six ROW (September 2004), IOM pilot trial measurements on two ROW (July 2004), and measurements by the Health and Safety Laboratory (HSL) on London Way, Melbourn (in June 2001, (Chisholm, 2001)). The former produced concentrations over week long periods in September 2004, and the main results are summarised in Chapter 4.

3.2.2 Weather data

Weather data for the periods of sampling and for selected years were obtained from a local weather station, Icení at Royston, which is only a few kilometres from these ROW.

The data for the local weather and airborne asbestos concentrations on the ROW in September 2004 were used to develop a model of the dependence of airborne asbestos concentration on weather conditions (Chapter 5). Then the validity of the model was checked against airborne concentration measurements obtained by HSL in 2001, and by IOM in pilot trials in July 2004.

3.2.3 Population age profile

Census data on the age profile of the populations in the general area were provided by the South Cambridgeshire District Council. These data and their application in this study are described in Chapter 6.

3.3 DEVELOPMENT OF A MODEL OF WEATHER DEPENDENCE

The development of a model of weather dependence involved setting out a logically plausible model of the influence of selected parameters that are indicative of the average weather over the course of a day (rainfall, hours of sunshine, and mean wind speed). Comparison of model predictions of the relative levels of airborne fibre concentration provided a basis for selecting suitable values for the coefficients in the model, as described in Chapter 5. The model was used to predict average 24-hour concentrations (over the course of a year's weather) based on annual weather data for three 12-month periods also from the same local weather station (Icení at Royston).

3.4 ESTIMATION OF EXPOSURE

3.4.1 Estimation of diurnal variation of concentration

We use information from the HSL study which demonstrated that vehicles were the main cause of release of airborne fibres, and provided an estimation of the relative difference in concentration between the day (when there may be traffic) and the night.

Where there was an absence of more detailed information on patterns of vehicular traffic flow, we put forward suppositions that would give exposure estimations that are more likely to overestimate than underestimate the true exposure.

3.4.2 Activity and exposure

We describe the information available about the normal patterns of activity on the ROW. These are the basis for setting up hypothetical exposure scenario that should serve to calculate representative examples of the consequent risk.

3.5 PREDICTION OF RISKS

3.5.1 Basis for relating risks to exposure

The incidence of asbestos related disease has been related to exposure in several studies of heavily exposed workers in the industries that produced asbestos or used asbestos extensively (HEI, 1991). In those historical conditions, the workers were exposed at concentrations that were of the order of tens of fibres/ml, for decades. The various studies showed that the risks of disease increased with the amount of exposure. The same models derived from such studies have been used to predict the risks of the incidence of asbestos related disease at much lower concentrations by the highly reputed Health Effects Institute of the USA (HEI, 1991). They have reported their predictions of risks estimated for exposure to asbestos (at very low concentrations) in public and commercial buildings where there are asbestos-containing materials. They also calculated lifetime risks expected to arise from exposure to asbestos at the background concentrations that have been reported for rural air; for exposure to that level from birth, they estimated a lifetime risk of cancer of 4 in a million.

Heavy industrial exposure can cause asbestosis, but it is not relevant to the low exposures such as considered here.

There are two models for the relationship between exposure and disease that are relevant.

One is for the risk of mesothelioma, a cancer of the lining of the chest (pleural mesothelioma) or abdomen cavity (peritoneal mesothelioma). Mesothelioma is rare (about 2000 cases annually in the UK) and almost all cases have histories of significant exposure to asbestos.

The other model is for the risk of asbestos-related bronchial lung cancer. It is widely recognised that lung cancer is very common among smokers and much less frequent among non-smokers.

3.5.2 Model for mesothelioma

The model for prediction of mesothelioma reflects the key characteristics of the dependence on the pattern of exposure. The predicted risk:

- increases linearly with the annual average exposure concentration;
- reflects a long latency, of at least a decade from first exposure before any incidence of the disease;
- increases strongly with time from first exposure, after the estimated latency period of about 10 years.

The risk is dependent on the type of asbestos, with the risk being higher for crocidolite (blue) asbestos than that for amosite (brown asbestos) which is also higher than that for chrysotile (white asbestos), and this is predicted by the values of coefficients in the equation for the model (see Appendix 1).

The model equation gives the risk (of incidence of disease) in each year after exposure. Then life-tables are used to allow for the competing other causes of death, and the predicted risk over every year is summed to calculate a lifetime risk.

The risks of mesothelioma incidence are the same as those for mesothelioma mortality as the disease is incurable and usually fatal within months of diagnosis.

3.5.3 Model for lung cancer

Smoking is the main cause of lung cancer in the UK. Exposure to asbestos increases the risk of lung cancer, and the relative increase in existing risk is estimated to be approximately the same for smoker and for non-smoker, but the existing risk is much greater for the smoker than for the non-smoker. For the existing risk, we have used the lifetime risks of death from lung cancer as reported by Peto *et al* (2000) who calculated that this lifetime risk (by age 75) was 440 per 100,000 male lifelong non-smokers, and 16,000 in 100,000 for male current smokers. So, for example, if an asbestos exposure produced a relative risk of 1%, then the absolute risk for the current male smokers would rise to 16160 in 100,000 and to 444 in 100,000 for the lifelong non-smokers. The same relative increase in risk (e.g. 1%) thus produces a much larger absolute increase in risk for smokers (e.g. 160 extra cancers in 100,000 for the smokers compared to 4 extra cancers in 100,000 for the non smokers).

Most lung cancers occur after the age of 50. It is likely that the increase in risk follows the same age dependency, i.e. that any extra bronchial lung cancers would also occur after 50. The model predicts the same risk, for given level and duration of exposure, for exposure commencing at any age below 50.

The model equations for lung cancer are also shown in Appendix 1.

3.5.4 Overall risk

The overall additional risk of a cancer due to the estimated exposure is the sum of the two risks.

4 SUMMARY OF CONCENTRATION DATA

4.1 THE DATA AVAILABLE

The data on airborne concentrations comprise:

- measurements on six ROW, for weekly average concentrations, for the four weeks commencing 6th September 2004;
- weekly average concentrations from two ROW, from the pilot study in July 2004;
- concentrations measured by HSL on London Way in June 2001.

We used the first of these to develop a model of weather dependence. The latter two sets of data served to check the model. Then the model was used to predict annual average concentrations for each of the six ROW. The Pilot Study data also demonstrated that concentrations are low in wet weather. The HSL measurements provided information about the source of dust disturbance.

The samples in the 2004 sampling studies were all analysed by Scanning Electron Microscopy, which enabled asbestos fibres to be identified and distinguished from non-asbestos fibres.

In the sampling by HSL in 2001, a sample was analysed by Transmission Electron Microscopy (TEM) to estimate the relative proportions asbestos and non-asbestos fibres, and the other samples were counted by optical microscopy (which includes all fibres not just asbestos fibres).

4.2 DETECTION LIMITS AND AVERAGE CONCENTRATIONS

4.2.1 Detection limits and averages from combined samples

Where zero fibres are counted in examining a random selection of fields of view on the filter, then there is 95% confidence that the true mean count would be less than 3 fibres (from the Poisson statistics that describe the counts obtained on a subset of all possible fields of view). So where counts of asbestos fibres were 0, 1 or 2 fibres, they have been reported as being less than a detection limit corresponding to 3 fibres counted in the examined area of 1 mm² of the filter.

Where several samples were taken from a particular location, and it is reasonable to treat them as representing an average concentration for that place, then the data were combined as if they were for a single sample with the total volume of all the samples. This gives a lower detection limit. We have used this approach with the samples for each of the ROW. This calculation can lead to a measured concentration from the combined data even though the individual samples were all below the detection limit; this happens where the individual samples have 0, 1 or 2 fibres counted, but the total number of fibres for the set is 3 or more.

An average concentration calculated from the combined data for all the samples taken on a particular ROW is the best estimate of the average concentration that would be experienced if someone continuously walked up and down the ROW throughout the sampling period.

4.2.2 Average concentrations on the six ROW

In the September 2004 study, samples were collected with a total of 25 samplers located on the six ROW. There were 10 samplers on Shedbury Lane, 4 at Hill Top Farm, 4 at Newling Non-lets, 3 at London Way, 2 at Moor End lane, and 2 at Whaddon Estate Farm. As technical failures with some pumps caused a few samples to be lost from the collection programme, a total of 90 samples were evaluated.

When the data from all the individual samples on each ROW were combined, the average concentrations shown in Table 4.1 were obtained. The detection limits depend on the volume of air sampled in total and hence on the number of samples taken. As more samples were taken on some ROW than others, the detection limits were substantially different between ROW. Nine out of 24 average concentrations were below detection limits. Six of these were on two of the ROW (Hill Top Farm, and Whaddon Estate Farm). A measured average concentration (above detection limits) was obtained for all six ROW in Week 1. The number of average concentrations below detection limits increased progressively from week to week.

Table 4.1 Mean concentrations of airborne asbestos fibres from all the samples taken on the ROW during that week. Note that 9 out of 24 average concentrations were below detection limits.

Location	Mean Asbestos Fibre concentration for each week (fibres/ml)			
	Week 1	Week 2	Week 3	Week 4
Moor End Lane, Shepreth	0.00070	0.00013	<0.00002	<0.00002
Shedbury Lane	0.00022	0.00014	0.000033	0.000045
Newling Non-lets	0.00019	0.000021	0.000062	<0.000011
London Way	0.00017	0.000045	0.000045	0.000042
Hill Top Farm	0.000069	<0.000012	<0.00001	<0.00001
Whaddon Estate Farm	0.000064	<0.000019	<0.00002	<0.00004

In the Pilot study, each individual sample of airborne fibres indicated asbestos fibre concentrations below the detection limits. For those samples, the individual samples' detection limits ranged between 0.00003 and 0.0002 fibres/ml. When all data from the pilot trial were combined to give an overall estimate of fibre concentration, the value obtained (0.00001 fibres/ml) happened to be the same as that reported by the Health Effects Institute (HEI, 1991) as being the background rural level of airborne asbestos. The weather during the week of sampling in the Pilot trial was mostly damp with occasional light rain, and therefore may have been not conducive to release of airborne fibres from dust on ROW.

4.2.3 Asbestos type

Evidence on asbestos type was obtained partly by IOM's SEM measurements of the fibres sampled from the air, which distinguished chrysotile from amphibole. The type of amphibole was identified as amosite asbestos by polarising light microscope analysis of fibres in samples of loose dust from the track surface.

In the air samples:

- at Shedbury Lane, the asbestos fibres were mostly chrysotile;
- at Newling Non-Lets mostly amphibole,
- at Moor End Lane and London Way, about equally split between amphibole and chrysotile.
- At the other two ROW (Hill Top Farm and Whaddon Estate Farm), there were few asbestos fibres detected in the air samples – so there was limited data on the asbestos type - but chrysotile was more frequent than amphibole.

Our finding that the asbestos on London Way was about 50% chrysotile and about 50% amosite was supported by HSL's analysis of fibres on a sample they collected in 2001 (Chisholm, 2001). Their analysis by transmission electron microscopy (TEM), which was able to distinguish chrysotile, amosite, or crocidolite. showed that the fibres were mainly chrysotile or amosite.

The September 2004 sampling by IOM included not only air samples but also 48 samples of the dust from the track surface adjacent to air sampling positions on the ROW. These track surface dust samples were analysed by polarising light microscopy which distinguished the type of asbestos, chrysotile and type of amphibole. Chrysotile asbestos was detected in 46 samples. Amosite asbestos was detected in samples from Newling Non-lets and Moor End Lane. No crocidolite was detected in any of the surface dust samples. Therefore the amphibole asbestos detected by SEM on air samples was assumed to be amosite.

5 INFLUENCE OF WEATHER ON AIRBORNE FIBRE CONCENTRATION

5.1 INTRODUCTION

We used airborne fibre concentrations measured on six ROW in September 2004 and weather data from a local weather station (Iceni, at Royston) to estimate the dependence of airborne fibre concentration on weather conditions, and hence extrapolate to predict average fibre concentrations over the course of a year. The fibre concentration measurements comprised the average concentrations over the course of 7 day periods, for the weeks commencing September 6th, 13th, 20th, 27th in 2004.

These data from only four weeks are of course a limited amount of information about dependence on weather. Nevertheless, the four sampling weeks in 2004 provided some contrasting weather conditions. At the start of the month, there was dry sunny weather, and by the end of the month there were wet days.

We sought a model that would predict the variation in the relative level of airborne dust and fibres from day to day, due to weather factors. The predicted variation in daily average concentration would contribute to the predicted average over each week. The agreement between observed weekly averages and the relative levels predicted by the model would be the basis for confirming values assigned to parameters in the model.

The process of developing the model involved hypothesising a sensible dependence of relative level of airborne fibre concentration on each weather parameter, and then examining whether it produced predictions acceptably close to the observed data. Essentially, we set out assumptions about how we would expect weather to influence the relative airborne fibre concentrations. Then we expressed these in terms of a simple mathematical relationship which contained a few coefficients with values to be chosen (from a limited range of alternative possibilities). With limited data on relative dustiness of different weeks (i.e. fibre levels in Weeks 2, 3 and 4 compared to Week 1), the choice of values for these coefficients was based on empirical testing of trial models with the model outputs compared to this data.

Once the model had been selected, its validity was cross checked by comparing predictions with the data on fibre concentrations obtained separately in our pilot study in June 2004 and in June 2001 by HSL (Chisholm, 2001).

5.2 ASSUMPTIONS

The dust disturbed from the surface of the ROW could be dispersed by vehicular activity, pedestrians or by wind. In the measurements of concentrations of airborne dust and fibres on one of the ROW (London Way) conducted by HSL (Chisholm, 2001), vehicular movement was identified as the major cause of dust disturbance. Wind is a recognised mechanism of soil erosion, although there appears to be relatively little basis for predicting how much dispersion is produced. A recent paper (Lu, 2001) describes development towards a model of the wind erosion effect.

Our assumptions relating to the effect of weather are that:

- the source of dust, the track surface, would be affected by moisture content. A laboratory-based study on release of fibres from prepared and homogenised soil mixtures containing known amounts of asbestos showed that release of asbestos was greatly reduced by even a small percentage of moisture (Addison, 1988). Therefore:
 - days with rainfall less than 0.2 mm would be regarded as “dry days”;
 - the fibre concentration on wet days would be small, probably negligible, compared to that on dry days.
 - a wet day is likely to affect the condition of the track surface on the following day. Therefore, a dry day preceded by a wet day would have lower concentration, on average, than a dry day preceded by a dry day.
- the hours of sunshine (in a day) would contribute to drying the track surface, and therefore release of airborne fibre would tend to be higher on days with a lot of sunshine.
- on most days, the wind will carry dust away from the track and therefore wind primarily dilutes the trackside concentrations. (There will be some days when wind dispersion contributes to trackside concentrations, for example if the wind is blowing along rather than at an angle to the track; however, we assume that those days will not have a large effect on the annual average trackside concentration when there are other greater and more regular sources of dust disturbance.)

The results obtained by HSL (Chisholm, 2001) on London Way indicated that the main source of disturbance of airborne dust was the passage of vehicles, during the period when they were sampling. We assume that this is generally the case throughout the year on this and other ROW where there is vehicular access (which includes the ROW in Table 4.1).

Weather is clearly the common factor that affected the change in relative level of airborne fibre concentration from week to week on the six ROW. There may have been other unrecorded factors (such as changes in traffic flow) which may have contributed, but they appear to have been negligible compared to the weather as the pattern of change was so broadly consistent among the ROW.

5.3 POSSIBLE WEATHER FACTORS

5.3.1 Weather parameters

The weather parameters that we assessed as potentially useful for describing the influence of weather on the day to day variation in average concentration during the measurement period included:

- mean wind speed for the day (in mph);
- hours of sunshine;
- rainfall (collected from 09:00 on that day to 09:00 on the following day; below 0.2 mm to define a dry day).

These three parameters varied substantially within the measurement period (6th September to 4th October 2004) from day to day and from week to week.

Further weather parameters (such as relative humidity, amount of rainfall, average temperature or maximum temperature) could be influential but several of these changed relatively little over the period. Furthermore, inclusion of more parameters would probably have been excessive in accounting for variation due to changes in weather between just four time periods (i.e. between Week 1, Week 2, Week 3 and Week 4). The model was therefore adequate for the period for which data were obtained, but did not take into account the possibility that lower temperatures over winter might lead to more moisture remaining in the track surface. So the model would probably overestimate annual average concentrations.

5.4 ESTIMATED DEPENDENCE

5.4.1 Form of dependence

We tested a dependence of the relative level of airborne fibre (R) which expressed the idea that relative concentration would:

- be lower on a dry day preceded by a wet day compared to a dry day preceded by a dry day;
- be negligible on wet days compared to dry days;
- increase with sunshine drying the track surface, but by less than linear proportion to hours of sunshine;
- be inversely dependent on the average wind speed, due to dilution.

This was expressed as a relative level R , defined as:

$$R = \frac{A(\text{rain}) \cdot B(\text{rain_previous_day})}{(v_{\text{wind}} + 0.1)} \sqrt[n]{H}$$

where $A(\text{rain}) = 1$ if that day's rainfall is less than 0.2 mm, or otherwise = 0;

$B(\text{rain_previous_day}) = 1$ if the previous day's rainfall is less than 0.2 mm, or otherwise is $\frac{1}{3}$;

v_{wind} = average wind speed for the day (in miles per hour – mean wind speeds were provided to 1 decimal place i.e. to 0.1 mph);

H = hours of sunshine for the day; and

n = an integer coefficient, which sets dustiness as proportional to either square root ($n=2$), cubic root ($n=3$), or quadratic root ($n=4$) of hours of sunshine (H).

Choosing values of 2, 3 or 4 for n means that the relative level of airborne dust would double for, respectively, four fold, eight fold or sixteen fold increase in hours of sunshine in a day (if other factors remain constant). Thus a choice of $n=4$ gives a model with a relatively slow dependence on hours of sunshine; and that appeared to be consistent with the limited data available in this study.

The addition of 0.1 to the average wind speed prevents the possibility of division by zero.

Table 5.1 summarises the values of these selected weather parameters for the four sampling weeks, and the calculated values of *R*. It is noteworthy that Week 1 had more dry days, with longer hours of sunshine and two days with lower mean wind speeds than the other weeks.

The weather at these ROW may not match up exactly with the local weather station. For example, some of these ROW are more sheltered than others, so the change in mean wind speed as measured at Royston may over or underestimate the changes at the particular ROW.

Table 5.1 The selected weather parameters for the dry days during the sampling weeks and the relative dust-weather factor calculated from these values.

Sampling week	Date [†]		Sunshine (hours)	Mean wind speed (mph)	<i>R</i> Relative dust-weather factor, average for week
	Dry day preceded by dry day	Dry day preceded by wet day			
Week 1	6 th Sept		7	3.7	0.602
	7 th Sept		10.3	4.1	
	8 th Sept		10.9	1.1	
	9 th Sept		10.2	0.9	
		11 th Sept	6.8	9.4	
Week 2	16 th Sept		10.8	3.9	0.097
	17 th Sept		0.2	7.8	
		15 th Sept	7.3	3.7	
Week 3	27 th Sept		0.9	2.9	0.119
		21 st Sept	5.4	9.6	
		24 th Sept	4.6	1.3	
		26 th Sept	2.6	3.8	
Week 4		27 th Sept	0.9	2.9	0.040
		2 nd Oct	7.1	6.6	
		4 th Oct	6.2	6	

[†] Note that dates not in this table (i.e. 10th, 12th, 13th, 14th, 18th, 19th, 20th, 22nd, 23rd, 25th, 28th, 29th, 30th, of September and 1st, and 3rd of October) are the days that have rainfall ≥ 0.2 mm, and hence $R=0$.

5.4.2 Comparison of model with measured airborne asbestos concentrations

For two of the ROW, only the first week's average airborne asbestos concentrations were above detection limits. The other four ROW had 13 out of 16 average concentrations above the detection limit. These four ROW provide the best indication of change in airborne asbestos concentration from week to week.

For the concentrations that were below detection limits, the real value is known only to be between the detection limit and zero. Conventionally, a value equal to half or two thirds of the detection limit may be taken as an estimate of the concentration on the basis that it is likely to be closer to the real value than an assumption near the extremes of either 0 or the detection limit. When these data were used to plot Figure 5.1 (see next paragraph), the choice

of values between zero and the detection limits made very little difference to either the position of these 3 data points on the plot or the value of the average (across the four ROW) of the relative level of fibre concentration in Weeks 3 and 4. So we took a value of half the detection limit as the estimate.

Figure 5.1 shows the data for these four ROW. The concentrations are all normalised to give a value of unity for Week 1. The simple model of the weather-dependence gave the predicted levels in Figure 5.1. (The model predicts the concentrations for Weeks 2, 3 and 4 relative to Week 1.) The predicted relative levels show a pattern of change from week to week that resembles that of the measured concentrations from the ROW. These predictions lie within the spread of the data and fall reasonably close to the average for the four ROW. This indicates that a simple, logical model of the influence of the effect of weather describes the observed trend. The agreement is close enough that slight improvements in the consistency (with the limited data in Figure 5.1) is unlikely to create a truer model of the real effect of weather. We can conclude that this appears to be a good enough model, and the next check on the model is to compare its predictions with the independent data from the other sampling periods in June 2004 and July 2001.

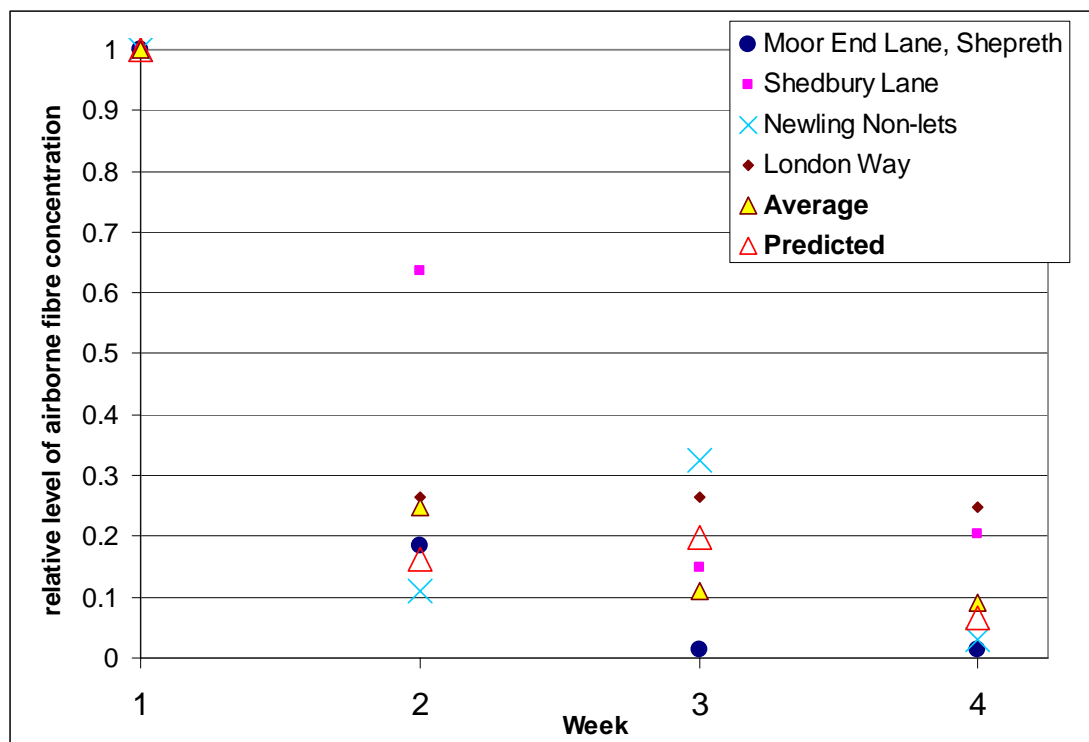


Figure 5.1. Airborne fibre concentrations at each ROW, normalised to the concentration in Week 1, measurements compared to the predicted relative level. Average and individual levels for the four ROW are shown.

The fact that Figure 5.1 shows that the logical expectation (from the model) appears generally consistent with the data suggests that the general pattern of the data is mainly due to the influence of weather.

Variation in the concentration data is expected, and the relative levels in Figure 5.1 are based on dividing one estimate of average concentration by another. The inherent variation in the data may be the main cause of the apparent differences between the individual ROW as regards the change in level from week to week.

There are some differences (among individual ROW), for example the highest concentration of all was measured at Moor End Lane, in Week 1, and very low concentrations in Weeks 3 and 4. Differences between individual ROW could arise from other factors such as changes in the traffic on particular ROW in a given week. However, most of the data are fairly tightly grouped, and therefore it is reasonable to assume that the common pattern reflects the influence of weather.

In summary, the model prediction of relative level of airborne fibre concentration, at Weeks 2, 3 and 4 follows the general trend and its distance from the average measured relative level is less than the likely uncertainty in that average relative level.

Alternative simpler models, such as an assumption that concentration would be the same on all dry days (and negligible on wet days) did not explain the difference between weeks. So the selected model was sufficient and necessary to explain the observed pattern.

The model sufficed to explain the data obtained during September without taking explicit account of temperature, or relative humidity. Temperature varied only slightly between sampling weeks in that period. Relative humidity may be inversely related to hours of sunshine, so relative humidity may have been implicitly involved through the inclusion of average hours of sunshine. Exclusion of these parameters is, we think, unlikely to lead to underestimation of average concentrations over the course of a year.

5.4.3 Cross check with data from other sampling

A cross check on the model was obtained by comparison with the data from the Pilot trial measurements at Moor End Lane (12 to 19th July 2004) and HSL's measurement of four-day average concentration at London Way, Melbourn (21st to 25th June 2001). HSL's measurement was a concentration of all fibres, but they also estimated that about 75-80% of those fibres were likely to be asbestos. So their reported concentration of 0.0004 all fibres/ml corresponds to about 0.0003 asbestos fibres/ml.

The weather was damp or rainy during the pilot trial. The measurements by HSL were obtained during a period of calm dry hot weather. The comparisons in Table 5.2 suggest that the model may predict an asbestos fibre concentration that is too high for damp periods, and is within the variation expected for repeat measurements for the one measurement for the dry period in June 2001. Thus, the two comparisons are based on substantially different weather conditions, and the measurements on London Way reflect probably the driest and dustiest of conditions. **Although these checks are based on only a limited amount of independent data, they suggest that the model is unlikely to give a significant underestimate of the airborne concentration.** Indeed the model may overestimate because annual average concentration because it does not take account of any effect of lower temperatures during the winter months.

Table 5.2 Comparison of weather model predictions of airborne asbestos concentrations for periods of damp and dry weather

ROW and measurement study	Weather factor for level of concentration relative to Week 1 of Sept 2004	Predicted asbestos fibre concentration	Measured asbestos fibre concentration
Moor End Lane, Pilot Study (two samples)	0.19	0.0013	<0.00007 <0.00004
London Way, HSL's measurements	1.44	0.00025	0.0003 [†]

[†]based on about 75% of fibres being asbestos in the sample evaluated by TEM by HSL.

5.5 ANNUAL WEATHER PATTERNS

5.5.1 Data provided by local weather station Icen, Royston

The data provided by the local weather station for three years was examined as a basis for extrapolating to annual average concentrations. Three years were chosen, in discussion with the Cambridgeshire County Council and South Cambridgeshire District Council. The chosen years were 2001 and 2004 because they were when measurements had been made of airborne fibre concentrations on the ROW, and 1995 because that summer was particularly dry.

For 2004 and 2001, the weather data from Icen included mean wind speeds, daily rainfall, and hours of sunshine as well as other data (such as relative humidity and temperatures). The data available for 1995 included the daily rainfall except for a short period when the rainfall was the summation rather than daily. The 1995 data did not include hours of sunshine nor mean windspeeds. Therefore we can use the model (from Chapter 4) to estimate the annual average concentrations for the more recent years, but only a simpler estimate of the relative level of fibre concentrations can be made for 1995 based on the weather data available for the three years.

The numbers of dry days (for the three selected years) are summarised in Table 5.3. In section 5.4.2, we found that numbers of dry days are not on their own sufficient to predict relative airborne fibre concentrations. Nevertheless, the model does include (among other dependencies) the estimate that airborne asbestos fibre levels are proportional to the number of dry days. Table 5.3 shows for each year the number of dry-days-preceded-by-a-dry-day, dry-days-preceded-by-a-wet-day, total of all dry days, and a weighted total with a lower weighting (as in the model of section 5.4.2) for dry days preceded by wet days. The columns for overall totals of days (unweighted or weighted) indicate that 1995 had about 20% more dry (hence dusty) days. The year 1995 was included in the weather data for this study because it was considered to be one of the driest years in the past decade. Given this limited data on 1995, we estimate that 1995 probably had annual average concentrations about 20% higher than the annual averages estimated for 2001 or 2004. So the lack of more detailed data for 1995 does not seriously weaken the estimation of annual average concentration for future years (based on the more detailed data for the more recent years).

Our best estimate of relative levels of airborne fibre for these periods is obtained from the model which includes the daily sunshine and daily mean wind speed, and that is calculated in the next section.

Table 5.3 Numbers of dry days in four 12 month periods.

Year	Number of dry days			
	Preceded by dry	Preceded by wet	total	weighted total
April 2004-March 2005	123	75	198	148
2004 calendar year	122	72	194	146
2001 calendar year	121	64	185	142
1995 calendar year	153	61	214	173

5.6 ESTIMATED ANNUAL AVERAGE CONCENTRATIONS

5.6.1 Best estimates of annual average

From the model of the dependence of airborne asbestos concentration on the selected weather parameters, we estimated the *relative level R* of the annual concentration for each of the three 12 month periods (April 2004 to March 2005; 2004 calendar year, and 2001 calendar year). Then these factors were used to extrapolate from the measured concentration in the first week of sampling in September 2004 to the annual average concentration for the six ROW. The data for Week 1 are chosen because that was the one week when all the six ROW had average concentrations measurements that were above the detection limit.

These predicted annual average concentrations are shown in Table 5.4.

Table 5.4a Predicted relative level of concentration for each 12 month period, compared to Week 1 of the sampling based on the model of weather dependence

March 2004-April 2005	$R_{\text{year}}/R_{\text{week 1 of sampling}}$	
	2004 calendar year	2001 calendar year
0.29	0.28	0.24

Table 5.4b Predicted annual average asbestos concentrations, using relative levels from Table 5.4a and measurement in Week 1.

ROW	Airborne asbestos concentration (fibres/ml)			
	<i>Measured in Week 1</i>	Predictions for each 12 month period		
		March 2004-April 2005	2004 calendar year	2001 calendar year
Moor End Lane	0.007	0.00205	0.0019	0.0017
Shedbury Lane	0.0022	0.00065	0.00061	0.00053
Newling Non-lets	0.0019	0.00056	0.00053	0.00046
London Way	0.0017	0.00050	0.00047	0.00041
Hill Top Farm	0.00069	0.00020	0.00019	0.00017
Whaddon Estate (Fountain) Farm	0.00064	0.00019	0.00018	0.00015

The relative level of airborne asbestos concentration predicted for each annual average is based on the weather dependence model of equation 5.1.

For the purpose of calculating future risks from exposure at the predicted annual average levels, Table 5.4 shows essentially three groupings by level of predicted likely annual average concentration:

- one ROW at about 0.002 fibres/ml;
- three ROW at about 0.0005 fibres/ml, and
- two ROW at 0.0002 fibres/ml.

These concentrations may be compared with those summarised by HEI for levels in other circumstances:

- background asbestos fibres in outdoor rural air 0.00001 fibres/ml;
- outdoor urban air, 0.0001 asbestos fibres/ml;

- in well maintained buildings with asbestos in good undamaged condition, 0.00002 fibres/ml.

This comparison shows that, for example, the annual average concentrations on Hill Top Farm and Whaddon Estate are slightly higher than a concentration reported for urban air.

6 RESIDENTIAL POPULATION

6.1 INTRODUCTION

The risk models (described later) predict risk of mesothelioma to increase with level of exposure and also with time from start of exposure. The dependence on time from start of exposure means that the age profile of an exposed population is part of the information needed to produce a reliable estimate of the likely lifetime incidence of disease in the group.

6.2 POPULATION AGE PROFILE

6.2.1 Age profile based on census data

South Cambridgeshire District Council provided census data for the age profile of the populations living in the community (village or parishes) nearest to each of the ROW. These data included many more dwellings than are directly adjacent to the ROW. For example, on most of these ROW, there are not more than about 10 dwellings immediately adjoining the ROW. For calculation of risk to the population of residents living in properties adjoining the ROW, the ideal information would be the age profile information for that small group *if* the group is stable. However, profiles of small groups could change rapidly if there are changes in the members of the group (e.g. if a few families move house). Therefore, the age profile for the larger adjacent community may be as good a guide, perhaps a better guide, to the average profile for the smaller group over the course of future years.

Table 6.1 Age profiles indicative of the likely average profiles for residents on the ROW. The data shown are for the larger community that live in the nearest village.

Age interval	Moor End Lane	Newling Non-lets	London Way	Shedbury Lane	Hill Top Farm	Whaddon Estate (Fountain) Farm
	Shepreth	Melbourn	Melbourn	Bassingbourn	Croydon	Croydon
0 -4	9	12	22	7	9	15
5-14	64	36	47	21	44	37
15-24	38	22	29	15	6	24
25-34	28	6	20	20	24	22
35-44	69	48	63	35	54	54
45-54	59	50	60	29	27	62
55-64	49	41	46	23	26	54
65-74	33	38	23	21	17	37
75+	13	44	10	16	14	18
Total	362	297	320	187	221	323

The distribution of ages is relatively similar for each of these communities, as shown in Figure 6.1. The total number of people in each community ranges from 187 to 362. However, as stated above, most of them do not live immediately adjacent to the ROW. For example, in the case of Hill Top Farm, the majority of the residents are a very considerable distance from the ROW. At Shedbury Lane (Bassingbourn) there are about 10 dwellings adjacent to the track. That appears to be the ROW with the most residential dwellings which are immediately adjoining the track. If between two and four residents live in each dwelling, then that would amount to about 30 residents living next to the track at that ROW. That is a sixth of the population (listed in Table 6.1) for the community of Bassingbourn.

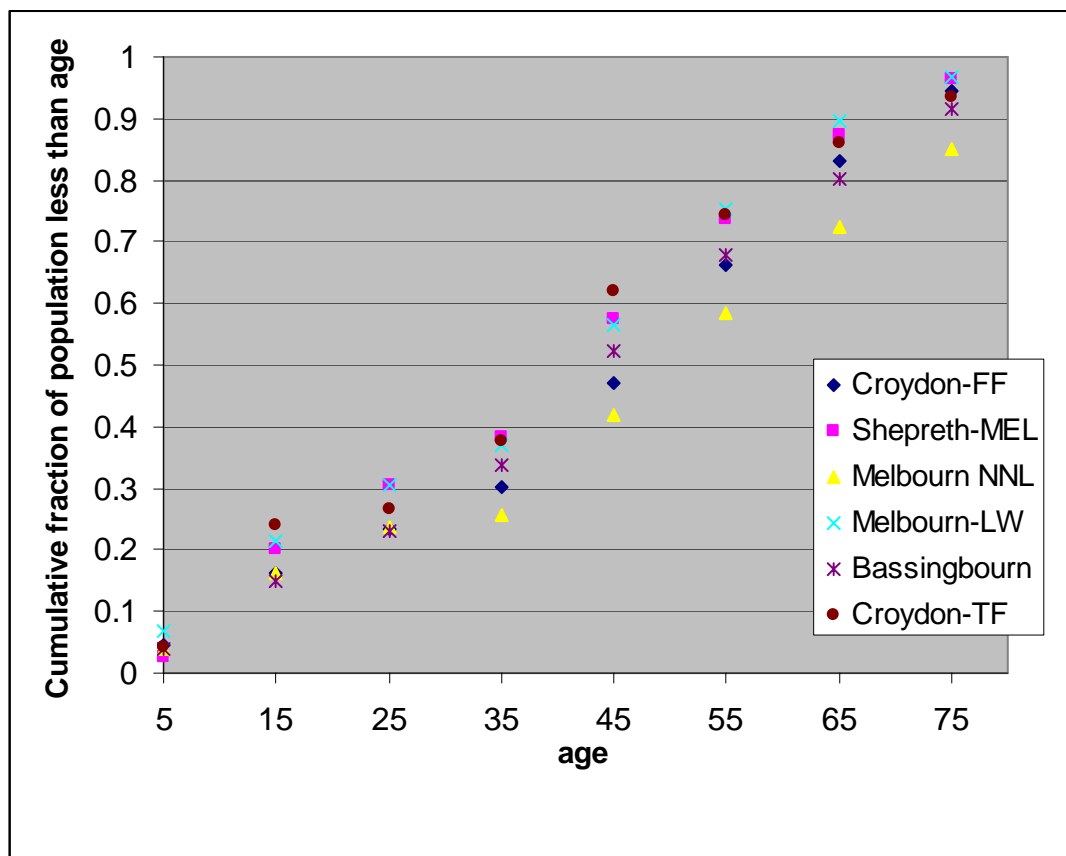


Figure 6.1 Cumulative age distribution of population in the communities adjacent to each ROW

We took a simplified summary of the age profile for calculating how frequent or infrequent an incidence of exposure-related disease is likely to be. From the simplified profiles in Table 6.2, we used the age profile which has the highest percentage in the youngest age group (in bold in the table), which happens to be for the village nearest to Hill Top Farm. This age profile was chosen because it is realistic and fully represents the possible exposure of younger persons.

Table 6.2 Simplified representative age profile for calculations of predicted incidence for the residents of the ROW.

Age interval	Estimate of percentage of population in age interval						
	Moor End Lane	Newling Non-lets	London Way	Shedbury Lane	Hill Top Farm	Whaddon Estate	All six ROW
<15	20.2	16.2	21.6	15.0	24.0	16.1	18.9
15-34	18.2	9.4	15.3	18.7	13.6	14.2	14.9
35-54	35.4	33.0	38.4	34.2	36.7	35.9	35.7
55+	26.2	41.4	24.7	32.1	25.8	33.7	30.6
Total	100	100	100	100	100	100	100

Based on the census data supplied, we assumed that 51% of the population was female and 49% was male. Furthermore, we applied general population smoking rates to estimate that 28% of men and 24% of women would be smokers. Table 6.3 shows the estimated proportional breakdown of the population by age, gender and smoking habit (for children it is assumed that they will become smokers later in life).

Table 6.3: Assumed distribution of population

Estimated distribution of population (each cell contains proportion of whole population)					
Age interval	Males		Females		Total
	Smokers	Non-smokers	Smokers	Non-smokers	
<15	0.033	0.085	0.029	0.093	0.240
15-34	0.019	0.048	0.017	0.053	0.136
35-54	0.050	0.129	0.045	0.142	0.367
55+	0.035	0.091	0.032	0.100	0.258
All	0.137	0.353	0.123	0.388	1.000

The estimated numbers of individuals in each age, gender and smoking group for each ROW were calculated by applying the proportions shown in Table 6.3 to the estimated total population leaving near each ROW. These were assumed to be as follows:

Moor End Lane	10
Newling Non-lets	10
London Way	40
Shedbury Lane	20
Hill Top Farm	10
Whadden Estate	10
Total	100

Later in this report, the individual risks are calculated for exposure starting at age 5, 20, 40 or 60. The risk calculated for these four ages were then taken as an estimate of the individual risk for people in each of these four groups. These risks are expressed separately for men and women and for smokers and non-smokers. The risk of any death for the population is calculated by applying the risks for each subgroup to the numbers in the population calculated by applying the proportions shown in Table 6.3 to the population numbers at each ROW and then summing the individual risks. For example, in a simplified situation if there were 100 people each with individual lifetime risk of 1 in 100,000 (i.e. risk = 0.00001) of disease caused by the estimated exposure, then the chance of any eventual death in that group being due to that exposure is 0.001.

7 EXPOSURE SCENARIO

7.1 INTRODUCTION

The information needed to predict the risks to health from the asbestos exposure are the *duration of exposure*, the *annual cumulative exposure* (E in fibre.hours/ml) for each year of exposure, the *type of asbestos*, and the *age* of the person when first exposed. The *cumulative exposure* E is the airborne asbestos fibre concentration multiplied by the duration of the exposure. Where an individual has exposure at different concentrations over various periods, then a cumulative exposure is calculated for each period and then summation over such periods gives the cumulative exposure for the year.

Since *annual cumulative exposure* is the information needed, different patterns of exposure that amount to the same annual cumulative exposure are equivalent so far as predicted risk is concerned. For example, a cumulative exposure of 6 fibre.hours/ml could arise from exposure to 1 fibre/ml for 6 hours, or from exposure to 6 fibres/ml for 1 hour, or from 2 fibres/ml for 3 hours.

The information that we have about the concentrations on each of the six ROW comprises:

- average concentrations over night and day for 7-day periods, with data from four weeks in September 2004;
- a modelled dependence of airborne asbestos fibre concentration on weather;
- a predicted annual average concentration, also for the same sampling period (day and night).

To construct realistic estimates of the exposures, we also need to take account of:

- how concentration is likely to vary with activity and time of day and how that may be linked to the periods when people are on or near the ROW;
- the amount of time that people spend or are likely to spend on or near the ROW;
- how concentration varies with distance from the ROW.

The concentration measurements (of Table 4.1) are based on samples taken on filters mounted at about 1.5 m height above ground level and with the sampler placed as close to the trackside as safely possible, i.e. generally less than 1 m from the edge of the track. So they directly reflect the exposure of someone actually on or immediately beside the track. In theory, concentration diminishes approximately linearly with distance from a line source such as a long track. So concentrations more than 5 m from the track might well be less than a tenth of that measured within about 0.5 m of the track. Therefore, we would expect concentrations about 5 to 10 m away from the track to be (on average) less than a tenth of that at the trackside. Chisholm (2001) commented that a distance of 1 m from a source of dust release had given a tenfold difference in concentration.

We consider exposure scenario that include:

- residents regularly using the track, as their only access to and from their homes;

- residents using gardens within 5m of the trackside;
- residents in rooms with windows opening onto the ROW.

To facilitate comparisons, we set out the same exposure scenarios for the calculations for each ROW. For the farmers, we have some interview information about their patterns of activity. For the residents who live near a ROW, we have notes from observations during site visits (e.g. during the sampling exercise), but no direct interview information.

7.2 CONCENTRATION DIURNAL VARIATION

The concentrations are expected to vary with time of day. During day time, there is more vehicular activity and HSL (Chisholm, 2001) during their measurements on London Way demonstrated that a moving vehicle was the main cause of asbestos fibres becoming airborne. From his measurements, Chisholm estimated that concentrations overnight would be relatively low and therefore the concentration during daylight hours might account for almost all of the average asbestos concentration. On that assumption, he estimated that the daytime concentrations (during an assumed 10 hours of daylight) would be approximately 2.5 times higher than the average over 24 hours. This is equivalent to stating that the cumulative exposure from 24 hours at one concentration (e.g. 1 fibre/ml for 24 hours = 24 fibre.hrs/ml) would be equivalent to 2.4 fold higher concentration for 10 hours (2.4 fibres/ml for 10 hours = 24 fibre.hrs/ml). The same calculation can be applied to the much lower concentration (Table 4.1) on the ROW. For example, if the average concentration over 24 hours is C fibres/ml, then the cumulative exposure for that period is $24C$ fibre.hours/ml.

During the day of measurements by HSL, there was relatively little traffic on that ROW (e.g. one to two vehicles per hour). In our own sampling on the six ROW, the regular but brief daily site visits recorded similarly low levels of traffic.

The pattern of traffic activity during the day could be important in determining risk, for example, it would affect the exposure of someone who regularly walks the ROW when it is relatively busy with traffic.

We set out some suppositions about the likely variation in traffic during the day in order to make a best estimate of the possible exposure during relatively busy times of day. We suppose that these relatively busy periods last an hour per day, and we also suppose that each accounts for $1/3^{\text{rd}}$ of the potential exposure for the day. It seems unlikely that it would account for more than a third. Then, continuing the simplified numerical example used above, 10 hours with a potential cumulative exposure of $24C$ fibre.hrs/ml would consist of three periods each with $8C$ cumulative exposure:

- 1 hour at $8C$ fibre/ml ($8C$ fibre.hrs/ml) in the morning hour of work-bound traffic, and
- the equivalent hour for homeward bound evening traffic (also $8C$ fibre.hrs/ml), and
- 8 hours at $1C$ fibres/ml ($8C$ fibre/hrs/ml).

The total exposure for the 10-hours of the day is still $24C$ fibre.hours/ml.

We use this supposition to estimate possible exposure for people who might regularly be on the track when it is at its busiest (in Section 7.5). They are only suppositions as to possible

traffic flow, but we think that they ensure that exposures are overestimated rather than underestimated.

7.3 INFORMATION ON ACTIVITIES

7.3.1 Farmers

Cambridgeshire County Council staff interviewed farmers (at Newling Non-lets and Whaddon Estate Farm) to assess how much time they and other people spent in the vicinity of the farm track / farm yard with asbestos-containing materials.

At Newling Non-lets (Solway and Newtons) Farm, one of the farmers usually spends about 5 or 6 hours per week within about 5 m of the areas where asbestos is evident. Other family members spend much less time in the vicinity, estimated at only about 15 minutes per week in crossing the yard to take dogs for a walk. There are a few customers (purchasing fire wood in winter) who come in vehicles and pass close to the area (in the vehicles).

At Fountains Farm (Whaddon Estate), the farmer usually spends a few minutes a day within 5 m of the asbestos affected track. This was estimated at about 5 minutes per day in winter, increasing to about 7 minutes per day in spring, and to about 10 minutes per day in summer.

7.3.2 Residents

The amount of time spent by residents on the tracks may vary considerably. Some of the tracks (such as London Way) are about a mile long. Most of the other tracks are shorter and time spent daily on them, would be correspondingly shorter. Shedbury Lane and London Way are access routes to homes, and some residents would have a walk of about half a mile along the track. The track at Top Farm is a leisure walking route rather than the main access route.

Given these differences between the ROW, the amount of time that residents spend on the tracks probably varies among the ROW. However, if we take an hour per day spent on the track as an illustrative scenario, then it probably exceeds the time spent on the track by most residents.

From observation of the tracks, we suppose that most residents use vehicles for access. They therefore probably spend only a few minutes daily in travelling the ROW. Most of the dwellings on the tracks in this study, are at least 5 m from the track. We would therefore expect that average concentrations at that distance from the track would be less than a tenth of that at the trackside. Concentrations within the building are likely to be slightly lower again, as the building is also ventilated from the sides further from the ROW. An assumption that concentrations in buildings that are approximately 5 m from the track are at 10% of that measured at the trackside is unlikely to underestimate residents' indoor exposure to asbestos from the tracks. The concentration may well be much less than that, especially if the house is mainly ventilated by windows facing away from the track.

Pre-school children might spend a high proportion of time at the house adjacent to a track, and some time playing in a garden next to the track or on the track. However, they probably spend much less time on the ROW itself than in the garden or house. Their exposure is probably mainly at the concentration in or around the house which is much less than at the trackside.

7.3.3 Leisure users

The tracks are public rights of way which are used by people for leisure activities. We are informed that there is only limited choice of alternative routes for exercising dogs locally, so that regular use of the same ROW for that purpose is likely. Dog walkers were recorded in the site visit observations on at least one of the ROW.

Leisure use includes children cycling on the track. In the absence of more detailed information, we suggest that an assumption of an average (over the year) of half an hour per day probably covers the reality.

7.4 CONCENTRATIONS IN SUMMER

7.4.1 Prediction of concentrations and exposure for hot dry days

If there are activities which involve spending time on the track in the hot dry periods of summer, for example harvesting activity, then the weather model and the concentrations measured in Week 1 would be a good model for predicting those exposure concentrations. In Section 5.4.3, the model predictions for asbestos fibre concentrations during hot dry weather in June 2001 were shown to be consistent with measurements made by HSL.

7.5 EXPOSURE SCENARIO FOR RISK PREDICTIONS

7.5.1 Basis of exposure estimations

We have used the available information about how much time farmers, residents and other users spend on the tracks, to suggest some hypothetical exposure scenario that are likely to encompass the real exposure situations.

Conventionally, cumulative exposure is used to describe the exposure of persons. The measurements of trackside concentration were obtained with pumps continuously at the side of the track 24 hours per day. We call the fibre.hours/ml derived from those concentrations “*potential cumulative exposure*”. It is “*potential*” in that it is a theoretically possible exposure but only if someone spent all 24 hours, or perhaps the 10 daytime hours, walking up and down the ROW. Therefore it is not realistically possible for anyone to have the full potential exposure as people spend time sleeping, eating, working, etc. If 24 hours at the trackside gives the theoretical *potential cumulative exposure* (24C fibre.hours/ml), a practical worst case scenario could be taken as 50% of that theoretical potential (i.e. 12C fibre.hours/ml). Given the various other necessary activities of everyday life, this case is likely to be worse than any real exposure situation.

7.5.2 Suppositions and scenarios

As the passage of vehicles is considered to be the main cause of fibre release, then any peaks in traffic flow might cause concentrations to change systematically over the course of a day. In the absence of data on traffic flow, we have made suppositions as to how traffic patterns could affect exposure scenarios (Section 7.2). We choose suppositions that are, we think, likely to lead to overestimation rather than underestimation of exposure.

The theoretical potential exposure is shown as the first line of Table 7.1. However, it is implausible that anyone could spend more than about half the day every day at the trackside. So the first scenario is the hypothetical worst case of someone who spends half of every day

at the trackside. This is also very unlikely to be representative of real situations, but is a useful starting point.

On the track, for access and leisure

For the track user who regularly uses the track for about an hour a day on average (Scenario 2 in Table 7.1). Scenario 2A is for the person who is regularly on the track (e.g. walking for access, dog-walking, cycling,) when there is some peak activity, and we think that this hypothetical scenario, with the supposition of a substantial peak of traffic activity, is likely to more than cover his/her possible exposure.

Scenario 2B represents the person who uses the track at random times of day (when it may be quiet or busy), and it would also cover the general user if there are not regular peak periods. The hypothetical scenario based on the average concentration during daytime is a good estimate of exposure.

Scenario 2C is the converse of Scenario 2A; if there are peaks there will also be periods of relatively little traffic and lower concentrations as illustrated by this scenario. This is included mainly to illustrate that if there are peaks, then there are also troughs and that would be relevant to the overall risk for the residents as a group.

In dwellings on the ROW

Many of these residents live in buildings where the indoor concentrations are probably more than two orders of magnitude less than the trackside concentrations. The additional exposure from being in the building will be a minor additional component.

However, an estimate of indoor concentration at a tenth of the trackside annual 24 hour average concentration is relevant to the properties nearest to the ROW (e.g. within about 5 m of the trackside). As a house may have air entering from four sides, so its average indoor concentration will be affected by the outdoor concentration on the four sides. Concentration in the outdoor air will reduce with distance from the track due to dispersion and dilution. So much of the air entering a house will have lower concentration than that at the trackside. An estimate of an average indoor concentration at a tenth of that at the trackside is, we believe, a reasonable estimate given that we do not have direct measurements of indoor concentrations for these houses.

Scenario 3A (50% of time at home) is for those who work at other premises; Scenario 3B (100% of time at home) is for those who spend the majority of their time at home (e.g. home workers, housewives).

Activities described in interview information

Scenario 4 (Table 7.1) illustrates the likely exposure for the farmers with activity patterns as described in the interviews. Scenarios 4A and 4B cover the possibility that the farmer's presence on the ROW may be linked to traffic activity (i.e. to peak periods of concentration). These are, we think, likely to be overestimates rather than underestimates of exposure.

Combination of scenarios

Some real situations would involve combinations of the above scenarios, and the overall cumulative exposure would be the sum from those scenarios. For example, scenarios 2 (A or B or C) and 3 (A or B) will be relevant to some residents. These overall cumulative

exposures do not exceed the worst case scenario (Scenario 1). For example, the highest of these (2A + 3A) gives a daily cumulative exposure of 10.4C fibre.hours/ml.

Other residents (e.g. car users) may well spend only a few minutes per day on average at the trackside. This might be a combination of scenarios 3B with 4B or 3B with 4C, giving total daily cumulative exposures of respectively 2.5C or 1.6C.

If the farmer lives on a house by the ROW, the relevant combination of scenarios would be 3B with Scenario 4 (A, B or C).

Table 7.1 Scenarios with their estimated average daily cumulative exposure for ROW where 24 hour average concentration is C.

	Scenario	Assumption	Daily cumulative exposure Fibre.hours/ml	Comment
	Theoretical potential exposure for 24 hours at the trackside	As measured by trackside sample	24C	Potential only; impossible for a person to spend whole time at the trackside.
1	Worst case (half the day at the trackside)		12C	Very unlikely to occur, but physically possible
2	A Average of one hour per day at supposed busy time on ROW	a “peak period for traffic”	8C	Relevant to the person who walks to work, or dog walker, at these times.
	B Average of one hour per day on ROW at random times of day	Random times averages out traffic fluctuation effects.	2.4C	Better estimate if the peak traffic flows is not a regular pattern.
	C One hour per day on ROW when it is quiet (The converse of the “peak periods”)		0.25C	Relevant to someone who walks at quiet time of day (after supposed morning work-bound traffic)
3	A Supposed indoor exposure, if at home for 24 hours per day, in a house very close to track	Assuming indoor concentration to be a tenth of trackside concentration	2.4C	May be overestimate of indoor concentration for most residents.
	B Supposed indoor exposure, if at home for 12 hours per day		1.2C	As above
4	A Farmer within 5 m of the ROW for an average of 1 hour per day, assuming that it includes busiest periods	Average peak concentration reduced by 50% due to distance	4C	Based on the interview information from farmers as to amount of time. Our supposition is that the presence of the farmer on or near the ROW is linked to periods of higher traffic activity and associated dust disturbance.
	B Farmer, 10 minutes per day on ROW	At peak-traffic times	1.3C	
	C As above	At random times	0.4C	

7.5.3 Summary

The exposure scenarios provide a conservative estimate of the exposure for those who live on or use the ROW regularly. That is, we believe the estimates are more likely to be overestimates than underestimates.

The information used to derive the exposure estimates included:

- measurements of 24 hours concentrations of asbestos fibres at the trackside;
- a model of the influence of weather data to predict annual average concentrations at the trackside;
- observations of activity on the ROW;
- logical supposition that fill gaps in data (e.g. over traffic flow, and indoor concentration for houses within about 5m of the tracks) that give conservative estimates of exposure.

The predictions of risk in the next chapter use these exposure scenarios and the population age profiles to predict risks, for the individual and for the residents as a group.

8 PREDICTED HEALTH RISKS

8.1 INTRODUCTION

8.1.1 What the scenarios represent

This chapter reports the predicted health risks that arise if someone is exposed under some of the exposure scenarios proposed in Chapter 7.

A brief summary of the main features of the exposure scenario helps explain the significance of the predicted risks. The scenarios involve time and concentration for the exposure.

The concentrations are based on measurements of concentrations, mainly 24-hour concentrations, taken during a period when there would have been harvesting traffic on some of the ROW (as described in Chapter 4). This represents the heaviest form of traffic on the ROW. For assessing risks from repeated exposures that continue for years, we estimated the annual average concentration based on a simple model of the influence of weather (as described in Chapter 5).

We present the scenarios in terms of average time spent on the ROW per day as that ties in with the use of an overall annual average concentration. However, the concentrations will be higher on dry days than wet days, so the cumulative exposure is dependent more on amount of time spent on the ROW during dry weather than during wet weather. There are about 190 dry days in a typical year, as recorded at the local weather station (for the three years of data used in chapter 7). So if someone spends about 19 dry days per year on the ROW in activity similar to one of the scenarios, then their exposure would be estimated as about a tenth of that in the scenario.

The sampling pumps, stationed at the trackside for 24-hours per day measured a *potential cumulative exposure*; no persons are actually exposed at the trackside for that daily duration.

Scenario 1 is the hypothetical “worst case” extreme of someone receiving half the average daily *potential cumulative exposure* every day. Risks based on Scenario 1 (“worst case” exposure) being repeated year after year are an upper boundary on the possible risk.

Scenario 2 may be a realistic amount of time for someone who walks along the ROW daily for the longer ROW such as London Way or Shedbury Lane. Scenario 2A illustrates the daily cumulative exposure that would be expected if there are distinct peak periods of traffic on the ROW. It may well overestimate real exposure. Scenario 2B uses HSL’s estimate that most traffic would be within the daylight hours (of 10 hours per day), and takes the average concentration for the supposed 10 hours when most of the dust disturbance takes place.

Scenario 3 recognises that for dwellings that are very close to the ROW, e.g. within 2 or 3 m, there may be some exposure within the home. The assumption is intended to be precautionary, i.e. it should ensure that exposure is not underestimated.

Scenario 4 is based on interview information supplied by Cambridgeshire County Council on the pattern of activity reported by farmers. For Scenario 4A (a farmer who spends an hour a day within 5 m of the ROW), we assume a very modest reduction in exposure concentration due to distance from the ROW. Scenarios 4B and 4C are estimates for the farmer who spends 10 minutes a day on the ROW; that duration may also be typical of many residents who live

on a ROW, and therefore scenarios 4B or 4C might be the closest estimate of exposure for them.

8.1.2 Duration of exposure

The predictions of risk are based on the exposure scenarios continuing for several years: 5 years, 20 years or a lifetime.

8.1.3 Asbestos type

The risks of lung cancer or mesothelioma are greater for amosite asbestos than for chrysotile asbestos. The prediction models (see Appendix 1) treat amosite as being equivalent to twice the concentration of mixed chrysotile and amosite for prediction of mesothelioma risk. For lung cancer risk, we treat amosite as being equivalent to 4 times the concentration of chrysotile and amosite (as described in more detail in the appendix Section A1.4).

On one ROW (Newling Non-lets), the asbestos appeared to be predominately amosite asbestos. The measurements for the other five ROW showed that approximately 50% or more of the fibres were chrysotile asbestos.

8.1.4 Gender dependence

The effect of asbestos is believed to be independent of gender of those exposed. However, women tend to live longer than men and the part of the calculation that allows for competition of other causes of death is gender dependent. This affects the prediction of risk of mesothelioma for males or females.

The model of excess lung cancer risk due to asbestos exposure, as a fraction of the existing risk for smoker or non-smoker, is believed to be independent of gender. However, according to recent UK national statistics, there are differences in the existing risks of lung cancer for males and females (see next section).

8.1.5 Smoking dependence

Asbestos exposure is believed to increase the existing risk of lung cancer by approximately the same fraction for smokers and non-smokers, but of course the existing risk of lung cancer is much higher for smokers than for non-smokers. (So the absolute increase in risk due to asbestos exposure is much larger for the smoker than for the non-smoker.) The predicted risks are based on national background rates of lung cancer for smokers and non-smokers. The prediction of the risk from exposure to asbestos from an early age assumes only that the national background rates apply if he or she becomes a smoker.

We base our estimates of the existing lifetime risks of mortality from lung cancer on values reported by Peto *et al* (2000). They calculated that, in 1990 in the UK, the cumulative risk of death from lung cancer by age 75 was 16% in male current smokers and 0.44% in male lifelong non-smokers. For female smokers, the corresponding risks are 10% and 0.42%.

8.1.6 How the risks are expressed

The predictions of lifetime risks are expressed as incidence of disease per 100,000 people. They represent the number of extra mesotheliomas or lung cancers that would be expected to occur as a result of the given exposure scenario if 100,000 people were exposed.

The numbers of people actually residing in houses accessed from these six ROW is of the order of 100.

8.2 WORST CASE – HIGHLY UNLIKELY TO OCCUR

8.2.1 Worst case scenario over a lifetime

The “worst case” scenario is the level of exposure which is highly unlikely to be reached but is not physically impossible; (i.e. 12 hours per day on the trackside daily throughout the year, and thus 12 hours exposure on all the dry days of the year).

Table 8.1 shows the risks predicted for the “worst case” scenario (scenario 1 of Table 7.1) in the situation where the persons continued to be exposed for their lifetimes, from an initial age of 5, 20, 40 or 60. The risks are shown for mesothelioma, for lung cancer, and in total (for succumbing to one or the other disease).

The younger the age at commencement of exposure, the more years the individual will be exposed under this scenario. This leads to higher risk for younger age at commencement. For example, the predicted risk of lung cancer for exposure from age 5 is approximately thrice that for exposure from age 60.

Over and above the effect of duration of exposure, the risk of mesothelioma increases with time from start of exposure. So the predicted risks of mesothelioma for exposure from age 5 are more than ten times that for exposure from age 40, and more than 100 times that from age 60.

The left hand side of the table is for males; the right hand side (in italics) is for females. Because females have a longer life expectancy, they have more years at risk of succumbing to mesothelioma and therefore correspondingly higher predicted risk of mesothelioma for the given exposure. For lung cancer, males have higher background risks of lung cancer (as calculated by Peto *et al*, 2000) than females. This difference between males and females leads to higher predicted excess risk due to asbestos exposure for males.

For the non-smoker, the predicted risk of mesothelioma dominates the total risk unless exposure started late in life (e.g. at 60). For the smoker, the excess risk of lung cancer is generally larger than the excess risk of mesothelioma. However, the smoker’s excess risk of lung cancer (due to the asbestos exposure scenario) is much less than his or her background risk from smoking.

The absolute risks (i.e. incidence per 100,000) are smaller for asbestos exposure commencing later in life for both males and females. The relative difference between male and female in predicted mesothelioma risks is smallest when the absolute risks are largest (for example, about 25% relative difference between genders for exposure commencing at age 5). For exposure commencing later in life, the relative difference between genders is larger (about 30% for exposure from age 40, and about 100% for exposure from age 60), but the absolute difference is smaller (i.e. risks differ between genders by less than 1 in 100,000 for exposure from age 60 in Table 8.1).

8.2.2 Worst case scenario over 20 or 5 years

Predictions

Scenario 1 represents a person being on the trackside for about half a day for all the dry days, and about 190 dry days are expected annually in this area. Scenario 1 is likely to exceed the exposure that will occur for any resident or user. In the previous section we predicted the risk that would arise if Scenario 1 exposure continued throughout a lifetime, from given ages. As the amount of time that someone spends on the ROW is likely to change over the years, we produced predictions for the worst case scenario exposure occurring for a part of a lifetime (20 years or 5 years duration). These predicted risks are shown in Tables 8.2 and 8.3, with exposure again starting from the same ages (5, 20, 40 and 60).

Lung cancer

The predicted risk of lung cancer is directly proportional to the cumulative exposure (concentration \times time). So a reduction of exposure *duration* produces a proportional reduction in excess risk of lung cancer. For lung cancer, the model (see Appendix 1 for details) predicts lifetime incidence of lung cancer based on total cumulative exposure. So the twenty years of exposure produces predicted excess risks of lung cancer (in Table 8.2) that are 4 times those for five years of exposure (Table 8.3).

The proportionality with lung cancer risks in Table 8.1 varies depending on the age at which lifetime exposure started. For lifetime exposure, we took average exposure duration (in the calculation of lung cancer risk) as continuing until only 20% natural survival is expected from UK national life tables (that is to age 85 for males, age 90 for females). For example, the lifetime exposure duration in calculation of lung cancer risk for males exposed from age 5 is 80 years. So exposure durations (from age 5) decrease from 80 years (in Table 8.1) to 20 (in Table 8.2) to 5 years (Table 8.3) and the excess lifetime risks of lung cancer change proportionately.

Mesothelioma

The lifetime risks for mesothelioma are very strongly dependent on time since start of exposure. (In Appendix 1, the time since commencement of exposure appears in the calculation as a cubic function. The dependence on annual average concentration is linear.) Consequently, the mesothelioma risks decrease (from Table 8.1 to Table 8.2 to Table 8.3) but not in linear proportion to the reduced duration.

Total risk from 5 years of worst case

In Table 8.3, the predicted risks from a “worst case” exposure scenario continuing for 5 years, show predicted risks which can be as high as 10 in 100,000 for two of the ROW (Moor End Lane and Newling Non-lets, and about 1 in 100,000 for the other four ROW.

Table 8.1 Predicted excess lifetime risk due to “**worst case**” scenario (Scenario 1) continuing **every year from given initial age** throughout life. This represents exposure for half the day at the trackside every day from age (5, 20, 40 or 60) until natural survival (up to age 105).

Predicted increase in lifetime risk, as incidence expected if 100,000 males or females had that exposure										
Initial Age	Males				Females					
	Mesothelioma	Lung cancer		Total [†]		Mesothelioma	Lung cancer		Total	
		Non-smoker	Smoker	Non-smoker	Smoker		Non-smoker	Smoker	Non-smoker	Smoker
Moor End Lane – mixed 0.002 fibres/ml										
5	29	2	64	31	93	39	2	42	41	82
20	11	1	52	12	62	15	1	35	17	50
40	2	1	36	3	38	3	1	25	4	28
60	0.08	0.55	20	0.6	20	0.2	1	15	1	15
Newling Non-lets - amosite fibres 0.0005 fibres/ml										
5	15	2	64	16	78	20	2	42	21	62
20	5	1	52	7	57	8	1.5	35	9	42
40	0.82	1	36	2	37	1	1.0	25	2	26
60	0.04	0.5	20	0.59	20	0.1	0.6	15	0.7	15
Shedbury Lane, London Way - mixed 0.0005 fibres/ml										
5	7	0.4	16	8	23	10	0.4	11	10	20
20	3	0.4	13	3	16	4	0.4	9	4	13
40	0.4	0.2	9	0.7	9	0.7	0.3	6	1	7
60	0.02	0.1	5	0.2	5	0.05	0.2	3.7	0.2	4
Hill Top Farm, Whaddon Estate - mixed 0.0002 fibres/ml										
5	3	0.2	6	3	9	4	0.2	4	4	8
20	1	0.1	5	1	6	2	0.1	3	2	5
40	0.2	0.1	4	0	4	0.3	0.1	2	0.4	3
60	0.01	0.1	2	0	2	0.02	0.1	1	0.1	2

[†] Values in Tables 8.1 and 8.2 are rounded to one or two significant figures. Totals calculated before rounding sometimes differ slightly from summed rounded values.

Table 8.2 Predicted excess lifetime risk due to “**worst case**” scenario (Scenario 1) for **20 years** from given initial age. This represents exposure for half the day at the trackside every day over 20 years from the given age.

Predicted increase in lifetime risk, as incidence expected if 100,000 males or females had that exposure										
Initial Age	Males					Mesothelioma	Females			
	Mesothelioma	Lung cancer		Total [†]			Mesothelioma	Lung cancer		Total
		Non-smoker	Smoker	Non-smoker	Smoker		Non-smoker	Smoker	Non-smoker	Smoker
Moor End Lane –mixed amosite and chrysotile fibres 0.002 fibres/ml										
5	22	0.4	16	23	38	29	0.4	10	29	39
20	9	0.4	16	9	25	12	0.4	10	13	22
40	1.6	0.4	16	2.0	18	3	0.4	10	3	13
60	0.08	0.44	16	0.52	16	0	0.4	10	1	10
Newling Non-lets - amosite fibres 0.0005 fibres/ml										
5	11	0.4	16	12	27	14	0.4	10	15	24
20	4	0.4	16	5	20	6.2	0.4	10	7	16
40	0.79	0.4	16	1.2	17	1.3	0.4	10	2	11
60	0.04	0.4	16	0.48	16	0.1	0.4	10	0.5	10
Shedbury Lane, London Way - mixed amosite and chrysotile 0.0005 fibres/ml										
5	6	0.1	4	6	10	7	0.1	2	7	10
20	2	0.1	4	2	6	3.1	0.1	2	3	6
40	0.4	0.1	4	0.5	4	0.6	0.1	2	1	3
60	0.02	0.1	4	0.1	4	0.05	0.1	2.5	0.2	3
Hill Top Farm, Whaddon Estate - mixed amosite and chrysotile 0.0002 fibres/ml										
5	2	0.04	2	2	4	3	0.04	1	3	4
20	1	0.04	2	1	2	1	0.04	1	1	2
40	0.16	0.04	2	0.2	2	0.3	0.04	1	0.3	1
60	0.01	0.04	2	0.1	2	0.02	0.04	1	0.1	1

Table 8.3 Predicted excess lifetime risk due to “**worst case**” scenario (Scenario 1) for **5 years** from given initial age. This represents exposure for half the day at the trackside every day over 5 years from the given age.

Predicted increase in lifetime risk, as incidence expected if 100,000 males or females had that exposure										
Initial Age	Males					Females				
	Mesothelioma	Lung cancer		Total [†]		Mesothelioma	Lung cancer		Total	
		Non-smoker	Smoker	Non-smoker	Smoker		Non-smoker	Smoker	Non-smoker	Smoker
Moor End Lane – mixed amosite and chrysotile fibres 0.002 fibres/ml										
5	8	0.1	4	8	12	10	0.1	2	10	13
20	4	0.1	4	4	7	5	0.1	2	5	7
40	0.8	0.1	4	0.9	5	1	0.1	2	1	4
60	0.06	0.11	4	0.17	4	0	0.1	2	0	3
Newling Non-lets - amosite fibres 0.0005 fibres/ml										
5	4	0.1	4	4	8	5	0.1	2	5	8
20	2	0.1	4	2	6	2.4	0.1	2	2	5
40	0.4	0.1	4	0.5	4	0.6	0.1	2	1	3
60	0.03	0.1	4	0.14	4	0.1	0.1	2	0.2	3
Shedbury Lane, London Way - mixed amosite and chrysotile 0.0005 fibres/ml										
5	2	0.03	1	2	3	3	0.03	1	3	3
20	1	0.03	1	1	2	1.2	0.03	1	1	2
40	0.2	0.03	1	0.2	1	0.3	0.03	1	0.3	1
60	0.01	0.03	1	0.02	1	0.03	0.03	0.6	0.1	1
Hill Top Farm, Whaddon Estate - mixed amosite and chrysotile 0.0002 fibres/ml										
5	1	0.01	0.4	1	1	1	0.01	0.2	1	1
20	0.4	0.01	0.4	0.4	1	0.5	0.01	0.2	0.5	1
40	0.08	0.01	0.4	0.1	0.5	0.1	0.01	0.2	0.1	0.4
60	0.01	0.01	0.4	0.007	0.4	0.01	0.01	0.2	0.01	0.3

8.3 RISKS FOR REALISTIC EXPOSURE ESTIMATES

8.3.1 Prediction for a scenario based on interview information

Scenario 4 was based on interview information, and therefore a risk prediction for this scenario is perhaps the best estimate of the likely risk for persons who are residents of houses accessed from the ROW for their lifetimes. These individual risks are shown in Table 8.4 for scenario 4B.

The predicted total excess risks, for those exposed from an early age, are of the order of 10 in 100,000 for two of the ROW (Moor End Lane and Newling Non-lets), about 2 in 100,000 for Shedbury Lane and London Way, and about 1 in 100,000 for Hill Top Farm and Whaddon Estate.

If the reality involves exposure under a combination of exposure scenarios, e.g. 4B and 3B, then the total predicted risk would increase proportionately with the cumulative exposure (where exposure follows the same time course). The combination of Scenarios 4B and 3B would approximately double the predicted risks from those in Table 8.4.

For most residents of the ROW, the predictions in Table 8.4 are probably the best guide to the likely risks from exposure to asbestos from the ROW.

Table 8.4 Predicted excess lifetime risk due to the realistic scenario 4B for a lifetime from given initial age. This represents exposure at the trackside during assumed periods of peak activity, for ten minutes every dry day of each year.

Predicted increase in lifetime risk, as incidence expected if 100,000 males or females had that exposure										
Initial Age	Males					Females				
	Mesothelioma	Lung cancer		Total [†]		Mesothelioma	Lung cancer		Total	
		Non-smoker	Smoker	Non-smoker	Smoker		Non-smoker	Smoker	Non-smoker	Smoker
Moor End Lane – mixed 0.002 fibres/ml										
5	3	0.2	7	3	10	4	0.2	5	4	9
20	1	0.2	6	1	7	2	0.2	4	2	5
40	0.2	0.1	3.9	0.3	4	0	0.1	3	0.4	3
60	0.01	0.06	2.16	0.07	2	0	0.1	2	0.1	2
Newling Non-lets - amosite fibres 0.0005 fibres/ml										
5	2	0.2	7	2	8	2	0.2	5	2	7
20	1	0.2	6	1	6	0.8	0.2	4	1	5
40	0.09	0.1	4	0.2	4	0.2	0.1	3	0.3	3
60	0.004	0.1	2	0.06	2	0.01	0.1	2	0.1	2
Shedbury Lane, London Way - mixed 0.0005 fibres/ml										
5	1	0.05	2	1	3	1	0.05	1	1	2
20	0.3	0.04	1	0.3	2	0.4	0.04	1	0.5	1
40	0.04	0.03	1	0.1	1	0.1	0.03	1	0.1	1
60	0.002	0.01	1	0.02	1	0.01	0.02	0.4	0.02	0.4
Hill Top Farm, Whaddon Estate - mixed 0.0002 fibres/ml										
5	0.3	0.02	1	0.3	1	0.43	0.02	0.5	0.4	1
20	0.1	0.02	1	0.1	1	0.16	0.02	0.4	0.2	1
40	0.02	0.01	0.4	0.03	0.41	0.03	0.01	0.3	0.04	0.3
60	0.001	0.01	0.2	0.007	0.217	0.002	0.01	0.2	0.01	0.2

8.4 RISKS FOR THE POPULATION OF RESIDENTS

8.4.1 An estimate of the incidence in the population

The risks to individuals (in Table 8.4) were used as the basis for calculating the likely incidence in the community. For this calculation, we assumed:

- the proportions of male and female in the local population were the same as the average in the UK (49% and 51% respectively);
- that the percentages of smokers were the same as in UK national statistics for the year 2003-2004 (<http://www.statistics.gov.uk/cci/nugget.asp?id=866>) (i.e. 28% of men and 24% of women are smokers);
- that the age profile followed that highlighted in bold in Table 6.2 (i.e. the profile with highest proportion of children).

Table 8.5 shows (in the column in italics) the predicted incidence (of asbestos-exposure related lung cancer or mesothelioma) per 100 residents. A predicted incidence of 0.002 in 100 residents is the same as 1 in 50,000 so there would have to be 50,000 residents (exposed for a lifetime) to expect that exposure to cause one death. So the realistic estimate of lifetime exposure (Scenario 4B) is not expected to cause any deaths.

Even under the worst case exposure scenario, where individual risks were ten fold higher, the likely outcome would be no deaths from the asbestos exposure arising from the ROW.

With an estimate of the actual number of residents living in the houses accessed from the ROW, we can estimate the chance of any death being caused by the exposure. Based on the approximate numbers of houses accessed from the ROW, the population for each ROW may be approximately that estimated in column 3 of Table 8.5. We have taken a minimum population of 10 even where there appears to be only one house near the ROW.

For example, if there are 10 people living at Moor End Lane, and assuming that they have exposure as estimated in Scenario 4 for their lifetime, then the expectation is that there is a 0.0002 (i.e. 20 in 100,000) risk that one of their deaths will be due to a disease (lung cancer or mesothelioma) caused by exposure to the asbestos from the ROW.

In Table 8.5, we have assumed a total of 100 people resident on the six ROW. The figure in the bottom right hand corner of the table is the predicted chance (0.0008) of one of their deaths being caused by the asbestos exposure. So if the total population on these six ROW is about 100, then the risk of one of their deaths being due to an asbestos related disease caused by exposure on the ROW is probably about 1 in 1,000.

Table 8.5 shows the total risk of an asbestos-related lung cancer or mesothelioma. The predicted risk of a mesothelioma is approximately half the predicted total risk.

In the UK population, lung cancer is caused primarily by smoking. In a population of 100 with the same assumed percentage of smokers, but *no exposure to asbestos*, we estimate that 3 or 4 deaths due to smoking-related lung cancer would be expected. This is based on the risks of mortality from lung cancer (by age 75), for smokers and non-smokers, calculated by Peto et al (2000): 16 % for male smokers, 0.44 for male lifelong non-smokers, and correspondingly 10% and 0.42% for females.

Table 8.5 Predicted incidence of asbestos-related lung cancer or mesothelioma for lifetime exposure as estimated under a realistic exposure scenario for the residents of the ROW.

	<i>Incidence per 100 residents</i>	Estimated number of residents in houses accessed from the ROW	Number of cases predicted for estimated population
Moor End Lane Mixed 0.002 fibres/ml	<i>0.0022</i>	10	0.0002
Newling Non-lets – amosite fibres 0.0005 fibres/ml	<i>0.0017</i>	10	0.0002
Shedbury Lane, mixed 0.0005 fibres/ml	<i>0.0006</i>	40	0.0002
London Way – mixed 0.0005 fibres/ml	<i>0.0006</i>	20	0.0001
Whaddon Estate – mixed 0.0002 fibres/ml	<i>0.0002</i>	10	0.00002
Hill Top Farm – mixed 0.0002 fibres/ml	<i>0.0002</i>	10	0.00002
<i>average</i>	<i>0.0009</i>	Total 100	0.0008

9 CRITERIA FOR DESIGNATING CONTAMINATED LAND

9.1 INTRODUCTION

The Department for Environment, Food and Rural Affairs (DEFRA) provides guidance on the definition of contaminated land, and the statutory duties that arise under the environmental Protection Act 1990 (as amended). Under section 78(2) of the Act, contaminated land is defined as: "*any land which appears to the local authority in whose area it is situated to be in such a condition, by reason of substances in, on or under the land, that -(a) significant harm is being caused or there is a significant possibility of such harm being caused; or (b) pollution of controlled waters is being, or is likely to be caused.*"

The presence of asbestos on the ROW gives rise to a possibility of significant harm (lung cancer or mesothelioma) being caused to humans. The question is whether the possibility is "significant".

DEFRA advises that *quantitative risk assessment* (QRA) is the appropriate approach to assess whether there is "*significant possibility*" of "*significant harm*" being caused. In paragraph A (of Annex 2 of Contaminated Land DETR Circular 2000 <http://www.defra.gov.uk/environment/land/contaminated/circ2-2000/3.htm#4>), the elements that contribute to "risk" are defined as:

*"(a) the probability, or frequency, of occurrence of a defined hazard (for example, exposure to a property of a substance with the potential to cause harm); and
(b) the magnitude (including the seriousness) of the consequences."*

The designation of land as contaminated land requires that a significant pollutant linkage (source-pathway-target) be identified. In the case of the ROW, a contaminant has been identified on the tracks (asbestos), there is a recognized pathway (inhalation of fibres that become airborne), and the people residing on or using the tracks are potential receptors who might be harmed by the inhalation of the contaminant. The question is whether the amount of exposure is enough to give rise to a *significant possibility* of significant harm

DEFRA ((2002a), report CLR7, paragraph 2.10) also states that "*The question of whether an intake or exposure is unacceptable is independent of the number of people who might experience or be affected by that intake or exposure*". The quantitative risk assessments that are relevant to a decision on whether the ROW should be designated as contaminated land are therefore those for the individual risk (Sections 8.2 and/or 8.3).

9.2 GUIDELINES

9.2.1 Approaches

DEFRA's publication CLR9 (DEFRA 2002b), on contaminants in soils, outlines current approaches to assessing non-threshold effects, which would include lung cancer and mesothelioma caused by asbestos exposure.

"*Index doses*" are used for substances for which a threshold for adverse effects cannot be presumed. These substances carry some risk at any level of exposure, although it may be very low at low levels of exposure. Index dose values have been defined for a few substances (arsenic, benzene, benzo[a]pyrene). Index doses have not been developed for asbestos. Indeed as index doses appear to be intended only for application for direct intake from the

soil, they would not be relevant to inhalation of asbestos fibres. The adverse health effects associated with asbestos are related to inhaled asbestos fibres deposited in the lungs.

Appendix A of DEFRA's CLR9 deals with approaches used for assessing non-threshold effects. Paragraph A7 advocates the greater reliability of dose-effect relationships derived from epidemiological studies (as opposed to animal studies). Paragraph A8 notes that there are large amounts of human data from which dose-response can be estimated for asbestos.

9.2.2 Advice on "acceptable" levels of risk

Paragraph A9 recognizes that *"To apply the concept of QRA to derive an acceptable numerical level of risk for non-threshold chemicals, it is necessary to take a view about the "acceptability" of levels of additional risk."*

The same paragraph also recognises that *"The acceptable lifetime excess risk (of death), for an individual member of the public, can range over orders of magnitude (for example 1 in 100 to 1 in 1,000,000) between different organisations. This is largely attributable to differences in the meanings of the various terms used (for example, maximum tolerable risk, acceptable risk or negligible risk) and to differences in application."*

Paragraph A.10 Offers advice on the excess risk that may be considered "acceptable" in respect of environmental contaminants. *"Despite the wide-ranging values reported by different authoritative organisations, there is some consensus for selecting a figure of 10 in 100,000 as the upper bound of "acceptable" additional lifetime risk from exposure to environmental contamination from any one source (such as, for example, a contaminated site). This corresponds to an annual excess risk of cancer of about 10^{-6} (one in a million per year)."*

Paragraph A10 concludes by offering advice on the role of the quantitative risk assessment. *"However, making a decision as to what is an acceptable level of risk to individual members of the public from exposure to ambient levels of an environmental pollutant is a value judgement. Although knowledge of some background scientific and technical information is necessary, the essential decision is one that involves socio-political judgements. The estimate of risk from a source such as a contaminated site, and an understanding of the uncertainties inherent in the estimate, can only help to guide decisions on action, which inevitably depend also on factors such as practicality, cost and competing priorities."*

The approach that DEFRA advocates for the quantitative risk assessment is to assess the risk to the most susceptible member of the population, which is usually taken to be a 6-year old female. The predictions for a five-year old female are therefore a good estimate of risk to the most susceptible member of the population.

9.3 OUTCOMES OF THE ASSESSMENT

DEFRA has used the Index Dose to express contamination levels which carry minimal risk for non-threshold contaminants. However, exposure to non-threshold contaminants should always be kept *"as low as reasonably practicable"* (ALARP). The quantitative risk assessment provides an essential part of the information which is needed to assess the practicality, cost, and competing priorities.

10 DISCUSSION

10.1 INTRODUCTION

The lifetime excess risks that may arise from the possible or likely exposure to asbestos from the ROW have been predicted using various pieces of information, assumptions and methods. This discussion examines the reliability of the inputs and methods. This provides an indication of the reliability of, and uncertainty in, the predicted risks.

Predicted risks may be more useful when considered in comparison with other more familiar risks. Comparison with other risks may provide a basis for assessing the significance of the risks for the individuals affected.

There have been attempts to provide guidelines on the levels of risk that might be considered low enough to be “acceptable” or “tolerable”. We discuss how these guidelines compare with the risks reported in Chapter 1.

Where there are limitations in the information that was used to produce the predictions, we discuss the possible implications and ways that the information gaps could usefully be addressed.

10.2 RELIABILITY OF PREDICTIONS

10.2.1 Concentration estimates

The estimates of airborne asbestos concentration are based on a substantial body of measurements. For example, a total of 90 samples of airborne fibres were evaluated by Scanning Electron Microscopy. Analyses of samples of dust from the track surface confirmed the presence of the asbestos, and type of asbestos, in the adjacent track surface for each air sampling position. The concentrations of asbestos in air (fibres/ml) showed a correlation with the asbestos concentration in the corresponding track surface dust samples (fibres/mg). So the airborne fibre concentrations, measured during the period of the sampling exercise, are believed to be reliable.

The estimate of annual average concentration depended on a model of the influence of weather conditions on concentrations and annual weather data. This model is probably adequate for the purpose. It tied in with the available data for two other periods when air concentrations had been sampled. However, it was based on data solely for the warmer part of the year and did not take into account the possible effect of temperature. It might be that the track surface would be less dusty on very cold dry days than on hot dry days. So it is possible that the concentrations on dry, sunny calm winter days would be less than predicted from the model of weather dependence.

In summary we believe that the estimates of the average airborne asbestos fibre concentrations used in this assessment are reliable. Where we have had to make assumptions to achieve estimates of annual average, then we have chosen assumptions that are more likely to overestimate rather than underestimate the levels.

10.2.2 Exposure

The estimates of amount of time that someone could spend on the trackside have been used to estimate a worst case exposure. We also used interview information (provided by Cambridgeshire County Council) to estimate a likely amount of time that residents spend on the ROW.

We have made a plausible supposition about how the pattern of traffic flow might vary during the day, and the chosen supposition was likely to ensure that exposure is overestimated rather than underestimated.

The exposure estimates are based on the current level of traffic activity, or more precisely the traffic activity during September 2004. If the level of traffic increases in the future, then the amount of asbestos disturbed might also increase. Therefore it would be important to review the risk assessment if there is any significant change in the number of residents or other traffic activity.

10.2.3 Risk prediction

The risk prediction models are based on human data from industrial workplace where exposure were of the order of tens of fibres/ml, which is about 10,000 times higher than the concentrations measured here. Conversely, some of the occupational exposures lasted decades, but the environmental exposures could last for a lifetime (for long term residents of the ROW). The models are therefore being extrapolated from high exposure concentrations to low concentrations, from adults to include children, and to cover lifetime exposure. They are believed to be the best basis for making predictions, and – for example – have been used by the highly regarded Health Effects Institute of the USA to estimate the likely consequences of low level prolonged asbestos exposure in public and commercial buildings.

In summary, the models are probably the best and most accepted approach available, they are based on extensive human data and should be a good estimate, but they are being extrapolated. The predictions are probably about right but should not be regarded as precise values.

10.3 COMPARISON WITH OTHER RISKS

10.3.1 Factors which affect perception of risks

There are many factors which may affect the way that risks that are perceived. For example, familiar risks are more readily accepted than unfamiliar risks. Studies on perception of risk have shown that unfamiliar risks are often perceived as being larger than familiar risks. Therefore, it can be useful to compare the magnitude of the predicted risks with risks from a few more familiar hazards.

Risks that are voluntary may be regarded as more acceptable than risks that are seen as imposed involuntarily. Therefore comparison of risks arising from voluntary activities such as smoking does not imply that the risks are equally acceptable. Nevertheless, Table 10.1 includes the risks from lung cancer that arise from smoking because smoking is the major cause of lung cancer in the UK.

Table 10.1 Estimated risk of death for various activities or circumstances.
Predictions for the exposure Scenarios included in italics

Activity	Risk per 100,000
Smoking 10 cigarettes per day for 40 years, lung cancer (Calman, 1996)	20,000
Road accident, during the next 40 years (Calman, 1996)	200
<i>Smoker, asbestos exposure as in lifetime worst case exposure from Table 8.1, total risk of asbestos-related cancer, the ROW with the highest concentration and exposure from age 5</i>	93
<i>Non-smoker, asbestos exposure as above</i>	31
Homicide (sometime during the next 40 years) (Calman, 1996)	60
Mesothelioma from exposure to amosite asbestos at the clearance indicator concentration (0.01 fibres/ml) for 40 years starting at age 20, from equation in Appendix 1.	30
<i>Smoker, total risk of asbestos-related disease from realistic estimate of exposure on ROW with highest concentration (Table 8.4) exposure from age 5</i>	10
<i>Smoker, asbestos exposure as in lifetime worst case exposure from Table 8.1, total risk of asbestos-related cancer, the ROW with the lower concentrations Hill Top Farm, Whaddon Estate (exposure from age 5)</i>	9
Burn or scald at home (in the next 40 years), (HSE, 2001; Table 4)	6
Lifetime cancer risks due to exposure to outdoor rural airborne asbestos concentration (0.00001 fibres/ml) from birth, as reported by HEI (HEI, 1991; their Table 8.3)	0.4
Struck by lightning (in the next 40 years), (HSE, 2001; Table 2)	0.2

The comparisons of magnitudes in Table 10.1 give a context to the predictions. In particular, the additional risk for lung cancer may be compared with the existing risk from smoking.

The level that the HSE has described as “acceptable” is an annual risk of 1 in a million. The risks mentioned elsewhere in this report are all lifetime risk. An annual risk of 1 in 1,000,000 if repeated over 40 years (e.g. from age 20 to 60), would amount to a lifetime risk of 4 in 100,000. If repeated over 70 years, from age 5 to age 75, it would amount to 7 in 100,000. If repeated over 100 years, it would amount to 10 in 100,000 which is the value described in DEFRA’s CLR9 as a general consensus on an upper bound for an acceptable risk (Section 9.2).

10.4 LIMITATIONS

We have considered that the main pathway for the contaminant to reach the target is by inhalation of fibres dispersed into the air from the surface of the track. It is also important to consider if there are any other pathways for exposure.

Ingestion is not considered to be a significant pathway for asbestos (e.g. as discussed by Doll and Peto, 1985).

If there is significant transfer of asbestos into the houses adjacent to the ROW (e.g. on the feet of the residents), then there could be potential secondary exposure when and if those fibres become re-dispersed (e.g. during sweeping or cleaning). We note that HSL took a wipe

sample from a house by London Way and did not find any asbestos fibres (Chisholm, 2001). That is an indication that this pathway is probably not significant.

We have assumed that ingress of airborne fibres into houses close to the ROW would not contribute much to the overall exposure.

There is obviously less data to support these suppositions (that transfer or ingress) are probably not significant pathways. These suppositions could be investigated further if required.

11 CONCLUSIONS AND RECOMMENDATIONS

11.1 CONCLUSIONS

11.1.1 Exposure conditions

The predictions of risk have been produced for people who are frequently and regularly exposed for a minimum of 5 years, and for up to a lifetime. The same durations of exposure were assumed to be applicable to each of the ROW.

The lifetime excess risk of an asbestos related disease increases with the amount of exposure. Therefore, any other people who have spent less time on the ROW and have had less exposure will have lower predicted risks.

The predicted risks of mesothelioma also increase strongly with time from first exposure. This leads to higher predicted lifetime risks for those exposed at a young age. In this report we predict risks for persons exposed from age 5, 20, 40 and 60, and so the highest predictions appear for someone exposed from age 5.

The worst case scenario illustrates cumulative exposure that would only arise if someone spends a very high proportion of their time on the trackside. It is not impossible for someone to have that exposure, but it is very unlikely. Risks were estimated under the supposition that the worst case exposure scenario continued for a lifetime, for 20 years, or for 5 years. Longer duration produced higher predicted risks for each exposure scenario. Real exposures may occur for these durations but the daily cumulative exposure would probably be substantially less than the worst case.

11.1.2 The condition of the ROW

There are some clear differences among the six ROW in the level of predicted total excess risk of cancer. Under the worst case scenario, the duration of exposure needed to produce predicted risks of 10 in 100,000 differed between the ROW.

- With the worst case scenario continuing for a lifetime, the predicted risks at two of the ROW (Whaddon Estate and Hill Top Farm) were of the order that has been described as generally “acceptable” i.e. below 10 in 100,000.
- With the worst case scenario continuing for 20 years, the predicted risks reach the 10 in 100,000 level for London Way and Shedbury Lane;
- With the worst case level of exposure continuing for 5 years, predicted risks reach approximately the 10 in 100,000 level for Moor End Lane and Newling Non-lets.

A more realistic estimate of the likely risk was based on interview information about the pattern of activity of the farmers using two of the ROW. This amount of exposure may also be likely to be more realistic for many of the residents. With this estimate of likely exposure, the predicted lifetime total excess risks were at or below 10 in 100,000 for all the six ROW. The risks of mesothelioma were all at or below 4 in 100,000.

From the risks predicted for a realistic estimate of exposure and with the assumption that there are about 100 residents living in houses accessed from the six ROW, we estimated the chance that any of their eventual deaths would arise from the asbestos exposure as being

about 0.001. That value compares with the expectation that, there would be 2 or 3 smoking-related lung cancers deaths among a typical group of 100 people without asbestos exposure (i.e. assuming that the proportion of smokers is the same as the UK average).

The risks predicted for the ROW are generally low enough that they may be regarded as low compared to many other more commonplace risks. However, under assumptions of “worst case” exposure continuing for many years, predicted risks can reach the boundary level for acceptable risk for some of these ROW.

In summary, the current levels of airborne asbestos fibre concentrations would not lead to risks above about 1 in 100,000 unless a person spends a very high proportion of his/her time on the ROW during the dry (dusty) days of the year and does so for several years. Taking the index of relative dustiness calculated from the weather conditions (daily hours of sunshine, rain, and mean windspeed), approximately 140 days in 2001 had an index above 50% of the value calculated for the dry sunny day (21st June) on which the airborne fibre concentrations were measured by HSL. On 222 days, the weather conditions indicated that fibre concentrations would be negligible.

DEFRA’s guidance on the role of the quantitative risk assessment (as described in Chapter 9, section 9.3) are that *“The estimate of risk from a source such as a contaminated site, and an understanding of the uncertainties inherent in the estimate, can only help to guide decisions on action, which inevitably depend also on factors such as practicality, cost and competing priorities.”*

11.2 RECOMMENDATIONS

The estimated asbestos exposures are low enough that they are unlikely to result in any deaths. Exposure to airborne fibres while on or by the track is almost certainly the main route of exposure. However, our supposition that other pathways (such as cross contamination on the feet of residents, or ingress of airborne fibres into houses) would be minor or insignificant are supported by much less data than was available for the airborne concentrations. Additional work could be undertaken to strengthen the basis for that view.

The model of weather dependence is based on sampling undertaken during warm weather, and consequently does not take account of the possibility that release of dust and asbestos fibres from the track surface might be substantially less on dry winter days than on dry summer days. If that information were obtained it would very probably lead to somewhat lower estimates of annual average concentrations. However, the present estimation of annual average concentrations may be sufficient.

The predictions of risk are based upon the presumption that traffic activity continues to be at about the same level. It would be useful to have a record of current traffic levels so that future changes can be recognised. If traffic flows increase substantially in the future, then it would become appropriate to re-assess the likely risks.

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APPENDIX 1 CALCULATION OF PREDICTED RISKS

A1.1 INTRODUCTION

The risk of excess of cancer (lung cancer or mesothelioma) is predicted from a mathematical model which describes the relationship estimated between the incidence observed in occupationally exposed cohorts and their measured exposure concentration, exposure duration and time since first exposure. The extension of the model to exposures that arose here involves extrapolating the relationship to levels that are 1000 or 10,000 times lower than those upon which the model was developed, and therefore there is obviously some uncertainty over the predicted risks.

Quantitative estimates of risk are however essential to be able to put the significance of the estimated exposure into some context with the many other risks that are faced in normal life. As the risks in this case are for the possible incidence of cancer that would occur many years in the future, a risk prediction model is essential. We have considered the uncertainties involved in the calculations when interpreting the results.

A1.2 CHARACTERISTICS OF MODELS

In principle, models could differ in the weighting given to exposure concentration, exposure time and time elapsed since exposure.

In the risk model that we use for lung cancer, the estimate of risk of excess lifetime incidence of cancer increases linearly with cumulative exposure (i.e. exposure concentration \times exposure duration). The weighting of concentration and exposure time is equal, which is sensible as the risk is for a long term effect associated with asbestos fibres that can remain in the lung for a very long time.

The model we use to predict risk for mesothelioma includes a strong dependence on time since first exposure, expressing the general experience of a long lag period between exposure and mesothelioma. Whereas, the model for lung cancer risk expresses dependence on solely exposure concentration and exposure duration without any influence of time elapsed since exposure.

A1.3 METHODS USED TO ESTIMATE THE RISK OF MESOTHELIOMA

Research into the risks of mesothelioma associated with exposure to asbestos has demonstrated that there is a positive exposure-response curve for mesothelioma (Hillerdahl, 1999) with the risk of mesothelioma increasing as exposure to asbestos increases. The risks of mesothelioma incidence are the same as those for mesothelioma mortality as the disease is incurable and usually fatal within months of diagnosis.

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 same dependence was noted by others including Schneiderman *et al* (1981). Incidence of mesothelioma was found to be proportional to a power of time since first exposure, and for continuous exposure this takes the form:

$$I_M(t, f) = k_m \cdot f \cdot (t - t_1)^n \quad (\text{A1.1})$$

where:

$I_M(t,f)$ = mesothelioma incidence at time “ t ” and concentration “ f ”
 k_m = parameter expressing the mesothelioma risk per unit of exposure, and having dimensions $1/(\text{fibres/ml} \times \text{time}^n)$
 f = asbestos exposure concentration (fibres/ml)
 $(t-t_1)$ = time since first exposure (in years)
 n = parameter expressing the relative importance of elapsed time since start of exposure

The values estimated for the two parameters in equation (A1.1), “ k_m ” and “ n ”, vary between epidemiological studies and between exposures to asbestos of different types (see below).

Whilst the above equation is a reasonable expression of mesothelioma incidence occurring at any stage of a continuous exposure to asbestos at a constant concentration f , a modified form is needed to estimate incidence after exposure has ceased:

$$I_M(t, f) = k_m \cdot f \left[(t - t_1)^n - (t - t_2)^n \right] \quad (\text{A1.2})$$

where $(t-t_2)$ = time since exposure ceased.

It has also been suggested that the equation should be modified to include a lag effect of about 10 years to allow for the latency of disease development after exposure. This gives a further revision to the equation:

$$I_M = 0, \quad \text{for } (t-t_1) < 10 \text{ and}$$

$$I_M(t, f) = k_m \cdot f \left[(t - t_1 - 10)^n - (t - t_2 - 10)^n \right] \quad \text{for } (t-t_1) > 10 \quad (\text{A1.3})$$

This formula gives the predicted incidence of mesothelioma during the year $t-t_1$ after the start of exposure. To compute the risk to an individual of a mesothelioma occurring during a normal lifetime, account has to be taken of the competition from other causes of death. So life tables were used to take account of the diminishing probability of still being alive. The cumulative lifetime probability of incidence of mesothelioma was then computed as the sum of annual risk of incidence over the period taken as a normal lifetime. The calculation was run for a lifetime of 105 years. The longer the assumed lifetime, the higher the predicted incidence of mesothelioma becomes, although in the final years the competition from other causes of death naturally becomes a major factor. So the cumulative risk increases significantly from age 75 to 85 but only marginally from age 85 to 105.

As noted above, the values estimated for the two constants, “ k_m ” and “ n ” vary between epidemiological studies. A report from the Health Effects Institute (HEI, 1991) summarised the results from four studies:

- three similar values of k_m ranging from 1.0×10^{-8} to 3×10^{-8} ;
- one study giving a much higher value of 12×10^{-8} ;

However, the latter study was criticised as having exposure estimates that were dubious, showed an inconsistent exposure response relationship (for lung cancer), and it was considered by several authors (HEI report page 6-13) too unreliable to be used for predicting risk. The three studies with similar values included a textile factory with mainly chrysotile but also some crocidolite exposure ($k_m = 1.0 \times 10^{-8}$), insulation workers with mixed asbestos exposure ($k_m = 1.5 \times 10^{-8}$) and cement factory workers with amosite exposure ($k_m = 3.0 \times 10^{-8}$).

For our risk predictions, we have chosen to use $k_m = 3 \times 10^{-8}$ for amosite asbestos, 1.5×10^{-8} for chrysotile with amosite asbestos.

Values of “ n ” fall typically between 2 and 4, although they are generally between 2 and 3 where the models include an adjustment for the latency of mesotheliomas. Many studies of mesothelioma risk have used values of 3.0 or 3.2 for “ n ”, although Peto (1982) recommended using a value of 3.5 for risk assessment purposes. Because we included an allowance for latency (in equation A1.3), we chose to use $n=3.0$. This is also similar to the value (of $n=3.2$) used by Chang *et al* (1999) for their environmental epidemiological study.

A1.4 METHODS USED TO ESTIMATE LUNG CANCER RISK

The situation for lung cancers contrasts with that for mesothelioma. The increase in lung cancer deaths is just as important as mesothelioma in terms of numbers of deaths, but the excess lung cancers appear against a background of a relative high incidence of lung cancers that would occur from other causes. This hinders the epidemiology, and makes risk prediction less precise for lung cancers than for mesotheliomas.

Estimations of the risk of lung cancer are less precise than for mesothelioma because the risk models that have been developed generally express the excess lung cancer risk relative to that in a similar population without the asbestos exposure. Ideally then, we would calculate the increase in risk by taking data on lifetime risk of lung cancer for a population with similar lifestyle and smoking habits as for the people involved.

For lung cancer, the model is dependent on the cumulative inhalation exposure to asbestos, i.e. the average concentration of asbestos fibres inhaled multiplied by the duration of exposure. The model predicts a percentage increase on the existing risk, where the existing risk is much larger for a smoker than for a non-smoker.

This equation implies an assumption that the relative risk for lung cancer among individuals exposed to asbestos is independent of age at first exposure and gender. Most cases of lung cancer occur after the age of 55.

It is well-known that the lifetime risks of developing lung cancer depend hugely on smoking habits. In the current study, we have estimated I_U separately for smokers and non-smokers by using the lifetime risks of mortality from lung cancer reported by Peto *et al* (2000). They calculated that, in 1990 in the UK, the cumulative risk of death from lung cancer by age 75 was 16% in male current smokers and 0.44% in male lifelong non-smokers. For female smokers, the corresponding risks are 10% and 0.42%.

It is normally assumed that the lifetime risk of lung cancer for an asbestos exposed population increases in linear proportion to the cumulative exposure to asbestos (which may be calculated as average exposure concentration multiplied by duration of exposure), using an equation of the form:

$$I_{Add} = (K_L \cdot f \cdot d) \cdot I_U \quad (A1.4)$$

where: I_{Add} = mortality risk in asbestos exposed population;

- I_U = mortality risk in non-exposed population
- K_L = parameter expressing risk per fibre.year/ml
- f = average concentration (fibres/ml)
- d = duration of exposure (years).

The coefficient K_L representing the risk per unit exposure has been estimated at values ranging from 0.0005 to 0.05 (fibre.years/ml), with several estimates close to 0.01 (<http://www.EPA.gov/iris/asbestos> - US EPA), including from studies with mixed types of asbestos (amosite, crocidolite, and chrysotile) and for cement product workers. For mixed exposure to amosite and chrysotile asbestos, a value of 0.01 appears appropriate; and it was used for the current risk estimation for lung cancer. For exposure to predominately amosite exposure, a value of 0.04 appears to be the appropriate value, and that was used for a ROW where the majority of the fibres were amosite asbestos.

Substituting the value of 0.01 for K_L in equation A1.4, it is readily apparent what risk would be produced by a given period of exposure at a given mean concentration. For example, 10 years at 0.1 fibres/ml (for chrysotile / amosite) would give:

$$I_{Add} = 0.01 \cdot I_U$$

that is a 1% increase in risk of lung cancer. For amosite asbestos, the same concentration and duration would give 4%.

The prediction model (HEI, 1991) is based on lung cancer up to the age of 75. In applying the model to lifetime exposure, we take lifetime exposure as continuing until only 20% of the population are surviving. That is up to age 85 for males, age 90 for females. For example, for males with lifetime exposure from age 5, the exposure duration d is taken as 80 years. That will slightly over estimate the exposure duration for 80% of the population, but underestimate it for 20%. The net result is still likely to be a slight overestimate of lifetime risk from lifetime exposure.

Equation A1.4 treats the relative risk (of lung cancer in individuals exposed to asbestos) as being independent of age at first exposure, gender and smoking habit. This assumption of independence from smoking may not be completely valid, as recent studies suggest that the relative risk may not be exactly the same for non-smokers as for smokers. However, because of the very few lung cancers expected for non-smokers, the differences in prediction would be small (HEI, 1991). Therefore the calculations below continue with the assumption of relative risk being independent from smoking habit. The absolute risk for lung cancer, being a multiplicative function of an asbestos related relative risk and a smoking dependent absolute risk, reflects a synergism between the effects of smoking and asbestos exposure.

A1.5 ASBESTOS TYPE

In the model expressed by equation A1.3, we used the value of " k_m " to express the risk that expected from a mixed asbestos with a substantial proportion of chrysotile ($k_m=1.5$) as discussed in Section 0 above.

The recent review by the HSE epidemiologists (Hodgson and Darnton, 2000) concluded that the relative risks of mortality for equal fibre number exposure to chrysotile, amosite and crocidolite are in the proportions 1: 100: 500. So where the fibres may be a mixture of amosite and chrysotile, the risk would be largely determined by the presence of amosite given its 100-fold higher contribution to risk. Given their estimation of the influence of asbestos type, the risks estimated here (for chrysotile exposure) are unlikely to be an underestimate.

A1.6 SUMMARY

The risks of mesothelioma were calculated from equation A1.3, and were obtained directly as excess risk arising from the exposure to asbestos. The lifetime risks were then computed by summing additional risks year by year over the life time.

The risks of lung cancer due to the asbestos exposure were calculated relative to the lifetime risks in the unexposed population from equation A1.4. Since lung cancer occurs only very rarely in non-smokers, the predicted increased risk of lung cancers affects mainly smokers.

The overall increase in risk of a cancer was obtained by summing the predicted risks of mesothelioma and lung cancer.

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