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# Assessment of cancer risks due to environmental exposure to asbestos

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In a rural area widespread pollution of friable and non-friable waste products was present, used to harden dirt tracks, yards, and driveways during 1935–1974. Exposure to environmental asbestos was assessed by a site approach, based on number of polluted sites within postal code areas, and by a household approach, based on number of households in the close vicinity to polluted sites within postal code areas. Based on asbestos soil investigations, 293 sites were identified with asbestos waste material at the surface, of which 77% contained crocidolite fibres as well as chrysotile fibres. The 293 sites-at-risk varied from 5 $m^2$ to 2722 $m^2$ and were surrounded by 347 households within 100 m of these sites. Distance to the plant was associated with the number of sites ($r = 0.36$), and with the number of households ($r = 0.52$). However, categorization of postal code areas into low, intermediate or high likelihood of exposure to asbestos showed a modest agreement between the site and household approach. In the site approach a total of 2.3 million person-years at risk were estimated with an average exposure of 1674 fibres/$m^3$ and an expected 1.8 cases of malignant mesothelioma each year. The household approach resulted in estimates of 1.2 million person-years at risk, and 0.9 cases of malignant mesothelioma per year, respectively. This study illustrates that asbestos waste on the surface of roads and yards in an area with over 130,000 inhabitants may result in long-term exposure to asbestos that will cause several cases of malignant mesothelioma each year. Although distance to plant, number of polluted sites and number of exposed household were associated, the modest agreement among these measures of exposure indicate that the exposure assessment strategy chosen in a particular study may result in considerable misclassification. Without detailed information on individual behaviour within the polluted area, it is difficult to show that a more individually oriented approach will perform better than an ecological approach.
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## Introduction

The causal relationship between occupational asbestos exposure and malignancies in the lung, most notably lung cancer and malignant mesothelioma (MM), is well established (IARC, 1987; Cugell and Kamp, 2004). MM may also occur due to non-occupational exposure to asbestos, for example among household members of asbestos-exposed workers or citizens exposed to asbestos in the general environment (Miller, 2005; Maule et al., 2007). Owing to the widespread use of asbestos in society, there is a substantial overlap between occupational and non-occupational exposure to asbestos, which hampers the evaluation of the specific contribution of different exposure routes to the occurrence of MM (Magnani et al., 2001).

A few hallmark studies have shown an increased occurrence of MM because of environmental exposure to asbestos. In the Republic of South Africa several regions around asbestos mines have been heavily polluted and epidemiological studies have shown that regional MM mortality rates were twice as high in men as well as in women as compared with national mortality rates, whereby especially the high mortality among women was indicative for environmental exposure (Reid et al., 1990; Kielkowski et al., 2000; Braun and Kisting, 2006). Abratt et al. (2005) have estimated that in South Africa approximately 26% of all MM cases can be attributed to environmental asbestos exposure.

A meta-analysis on six studies reported an increased risk of MM among subjects with high levels of environmental exposure to asbestos. Typically, these studies have relied on proxy estimates of exposure (Bourdes et al., 2000). Studies in the cities of Casale Monferrato and Bari in Italy and Amagasaki City in Japan showed that the distance of place of residence to an asbestos plant was associated with an increased occurrence of MM (Magnani et al., 2000; Maule et al., 2007; Kurumatani and Kumagai, 2008; Musti et al., 2009). The Japanese study modelled a relative asbestos exposure index, based on distance to plant and meteorological conditions, and could show a linear exposure–response relationship between this index and the MM risk (Kurumatani and Kumagai, 2008). In the Casale Monferrato situation, environmental exposure was caused by an asbestos cement plant as a localized

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source as well as multiple soil pollutions diffusely spread throughout the city (Magnani et al., 1995). A case–control study in the same region showed that residents with environmental exposure had a relative risk for mesothelioma of 10.5, after adjustment for occupational and domestic exposure (Maule et al., 2007).

The majority of studies on MM because of environmental asbestos exposure have used distance to the source as a crude proxy of exposure, primarily in an ecological study design. This approach with simple spatial models based on proximity measures inevitably leads to exposure misclassification, which will attenuate or bias the exposure–response association (Armstrong, 1998). A recent study on the Wittenoom mine in Western Australia used asbestos exposure measurements in the township during the period 1973–1992 to estimate individual exposure measures in a matched case–control study. A clear exposure–response relationship was found between cumulative exposure (f/ml-years) and occurrence of MM. However, cases in Wittenoom were exposed to a combination of environmental, occupational and domestic exposure to asbestos and it was not possible to separate the effects of these different routes of exposure on the occurrence of MM (Reid et al., 2007; Reid et al., 2008a, b). An earlier study has shown that residents who had lived at least 5 years in the area and were not directly employed in the crocidolite industry had in increased relative risk of 6.7, which was attributed primarily to environmental exposure to asbestos (Hansen et al., 1998). Hence, it remains a question how sources of asbestos pollution in the general environment will influence exposure patterns among citizens living in that area and how well geographic exposure models are able to estimate the magnitude of asbestos exposure at an individual level.

In this study, a rural area has a widespread pollution of asbestos waste material originating from an asbestos cement factory. This has resulted in environmental asbestos exposure from multiple sources, which have been identified in soil investigations (Sinninghe Damsté et al., 2007). Therefore, the aims of this study were (i) to assess the environmental asbestos exposure for subjects living in these areas using an approach based on the number and size of polluted sites in postal code areas and an approach based on number of households with close proximity to contaminated locations in postal code areas, (ii) to evaluate the agreement between both exposure assessment approaches, and (iii) to evaluate the consequences of the estimated asbestos exposure patterns in both approaches for the expected occurrence of MM.

## Methods

### *Identification and Evaluation of Asbestos-Polluted Sites*

The municipality Hof van Twente is a rural area with villages (Goor as the largest village), local industries, farms and nature reserves. An asbestos cement plant was located in the main village. The plant distributed asbestos waste (friable and non-friable waste products containing asbestos types chrysotile, crocidolite and amosite) for free to local residents for private and public use to harden dirt tracks, yards and driveways during 1935–1974. Therefore, the soil in this area is polluted with friable and non-friable waste materials (Sinninghe Damsté et al., 2007).

From 1978 onwards several investigations have been carried out in the municipality and its surroundings to establish the nature and size of the asbestos contamination in the area. An independent committee of experts has estimated that at least between 360 and 4400 tons of asbestos fibres has contaminated the area. Although the practice of the plant to hand out waste products to local residents took place during 1935–1974, most asbestos-containing materials were brought into the environment in the period 1960–1970 (Biesheuvel et al., 2003). An inventory in 1983 showed that within a radius of 12 km at least 83 roads were contaminated with asbestos waste, covering approximately 33,500 $m^2$ (Biesheuvel et al., 2003). Several investigations have followed, resulting in the Clean Up Asbestos Act 2003 that required notification of privately owned roads, dirt tracks and driveways contaminated with asbestos and arranged for clean up or full containment of the asbestos waste (Ministry of Housing, Spatial Planning and the Environment, 2003). During the first 6 months of 2003, citizens could file their application and all sites were investigated under guidance of the provincial authority before a licence for clean up of the asbestos soil pollution was provided ($n = 374$). Separately, the municipality and province identified and investigated all contaminated roads and dirt tracks with public ownership ($n = 42$). All investigations and subsequent clean up activities took place during 2003–2007. All reports of investigation of each site were available for this study. From these reports information about polluted sites was collected and categorized: (i) condition of the surface (open or closed [= concrete, asphalt, pavement]), (ii) presence, type of material, and type of fibre at the surface (friable and non-friable waste, fibre content), (iii) use of the site (active [= track, yard] or inactive use [= talus, green area]), and (iv) surface ($m^2$) of the polluted site. It is expected that asbestos in the general environment will be transported into the home environments, because asbestos residues can be carried on shoes, boots, vehicles, bicycles and pets into the house (Boeft, 1987; Anderson et al., 2005).

The area was divided into postal code areas at the 4-digit level. Within each postal code area the fraction of polluted surface was calculated by dividing the total polluted surface by the surface used for daily activities (postal code area surface minus area used for agriculture, nature, and water).

### *Site Approach*

The site approach (SA) represents a surface model, whereby it is expected that the number and size of polluted sites in a

postal code area will reflect the proportion of the population exposed in this area. In this approach we made the following assumptions: (i) inhabitants of a postal code area live homogenously spread through this area, (ii) only sites with an open surface with asbestos waste can contribute to asbestos exposure and (iii) inhabitants have a certain mobility throughout their postal code area. It is also assumed that the likelihood of asbestos exposure in a certain postal code area depends on the number of polluted sites in that area. For each postal code area the number of polluted sites was counted. These counts were categorized into postal code areas with low (0–1 polluted site), intermediate (2–5 sites) and high likelihood of exposure to asbestos (six and more sites). We assumed that all persons (100%) living in postal code areas with high exposure (six and more sites) were exposed to asbestos. The proportions of exposed subjects in the low and intermediate exposure categories were calculated as the fraction of average number of polluted sites in both categories relative to the average number of polluted sites in the high exposure category.

*Household Approach*

The household approach (HA) represents a model, whereby it is expected that the number of exposed households in a postal code area is a good approximation of the population at risk in this area. This approach required a detailed assessment of all houses with asbestos sites within its proximity. For the HA, the same assumptions were made as for the SA about asbestos in open surfaces and the consequences of mobility of persons in their living environment. For each postal code area the number of households within a distance of 100 m to polluted sites with asbestos at the surfaces were counted. The postal code areas were categorized into low (0–1 households), intermediate (2–5 households) and high likelihood of asbestos exposure (six and more households) in the areas. Again, it was assumed that all inhabitants in a postal code area with high exposure (six or more households) were exposed to asbestos. The proportion of exposed subjects in the low and intermediate exposure categories were calculated relatively to the average number of exposed households in the high exposure category.

*Assessment of Exposure Magnitude*

Historical measurements on asbestos exposure near an asbestos-polluted road in the municipality under investigation provided information about the relationship between air concentrations of asbestos fibres (amosite, chrysotile, and crocidolite) and distance to the road.(22) This road had friable asbestos waste on its surface, used as hardening material, and was used for local traffic by car or bike or persons on foot. Repeated air samples ($n = 60$) with a duration of 7 days were taken (May till September 1986) on three sites downwind the road (5, 100, and 1000 m). The samples were analysed with transmission electron microscopy. At 5 m distance to the road significantly elevated concentrations (average 1674 fibres/$m^3$) were found compared with background concentrations (average 68 fibres/$m^3$) at 1000 m distance. Approximately, 23% of the asbestos fibres consisted of crocidolite. At 100 m distance the average concentrations was slightly elevated, but not statistically different from the background concentration at 1000 m. In this study, we assumed as worst-case scenario that all persons living within a distance of 100 m to an asbestos-polluted site were exposed to an average concentration of 1674 fibres/$m^3$.

*Agreement between Site and Household Approaches*

The Pearson's correlation coefficient was used for agreement between number of polluted sites and number of exposed households within each area. The agreement between area categorization in both approaches was calculated by Cohen's weighted kappa.

*Risk Assessment*

We used the risk assessment from the review of Hodgson and Darnton (2000) to estimate the expected occurrence of MM. This review presents different exposure–response relationships derived from occupational cohort studies, and the current analysis is based on an expected mortality of about 10 cases of MM per 100,000 persons (with highest estimate of 55 cases) exposed for approximately 5 years, starting at age 30 years, to a cumulative exposure of 5000 fibres/$m^3$ $year^{-1}$ containing a significant proportion of crocidolite fibres. We have not adjusted cumulative exposure for differences in expected duration of exposure between occupational cohorts (only working hours) and the general population (only hours spent in the place of residence) because of lack of information. The calculations of expected cases of mesothelioma are based on the mean and maximum estimates presented in the risk assessment. With population data of the municipality of Goor about overall mortality, births, migration, and settlement from 1960 to 2007, we calculated a dynamic cohort of persons who lived in the village of Goor and assumed a similar dynamic cohort within each postal code area. This cohort consists of 48 categories of exposure duration, varying from 1 year to 48 years, with number of persons who contributed to each of these 48 categories based on their residential years. The period 1960–2007 was taken as total exposure period, because the environmental asbestos exposure became only substantial after 1960 and most clean-ups were completed at the end of 2007. To calculate the extra mortality of MM due to environmental asbestos exposure, we adjusted the calculations for age at first exposure to asbestos, using the correction factors proposed by Hodgson and Darnton (2000).

## Results

In total, 416 sites with asbestos pollution were identified and involved in this study. Figure 1 shows the research area and the distribution of the soil pollutions (black squares). Areas with a high population density, such as villages and cities, are outlined. The asbestos factory is located south of Goor. Most soil pollutions are located south of Goor and between Goor and Borne.

Table 1 shows that in almost 30% ($n = 123$) of the polluted sites there was a negligible risk on asbestos exposure, because the waste material was covered by a closed surface or a substantial top layer of soil. In the remaining 293 sites with asbestos waste material at the surface, 67% of the sites were dirt roads or yards daily used by inhabitants. At approximately 60% of all relevant sites friable waste material was identified, 77% of the asbestos samples contained crocidolite fibres as well as chrysotile fibres, and in 22% only chrysotile fibres were reported. Amosite was identified in very few samples.

Table 2 presents information about inhabitants, distance to the plant, asbestos-polluted sites, and exposed households by postal code area. The 293 sites-at-risk varied from 5 $m^2$ to 2722 $m^2$ and were surrounded by 347 households within 100 m of these sites. There were 44 sites of general use, of which 24 were actively used dirt tracks. In total, 249 sites

**Table 1.** Characteristics of the local sites with asbestos pollution in the soil ($n = 416$) in the surroundings of an asbestos factory.

| | Sites ($n$) | Percentage of all sites |
|---|---|---|
| *Sites with asbestos pollution (n = 416)* | *416* | |
| Surface closed (asphalt, concrete, pavement) | 13 | 3.1 |
| No asbestos in surface layer | 110 | 26.4 |
| Sites with asbestos present at open surface ($n = 293$) | | |
| *Daily active use by inhabitants* | *195* | *66.6* |
| Friable waste products | 112 | 57.4 |
| Containing crocidolite and chrysotile | 150 | 76.9 |
| Containing only amosite | 0 | 0 |
| Containing only chrysotile | 42 | 21.5 |
| *No daily active use by inhabitants* | *98* | *33.4* |
| Friable waste products | 60 | 61.2 |
| Containing crocidolite and chrysotile | 75 | 76.5 |
| Containing only amosite | 0 | 0 |
| Containing only chrysotile | 22 | 22.5 |

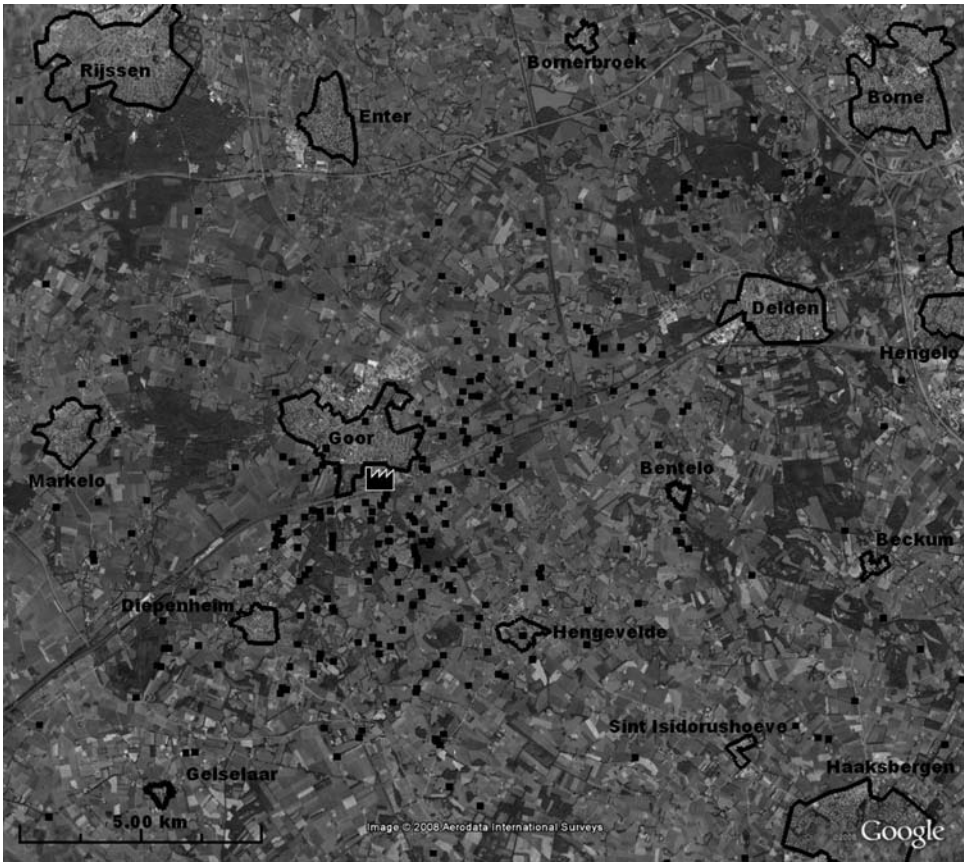


**Figure 1.** Research area with factory and locations of asbestos soil pollutions.

**Table 2.** Distribution of number of sites with asbestos pollution at the surface across postal code areas and number of adjacent households with potential environmental exposure to asbestos.

| Postal code area (villages) | Inhabitants (n) | Distance to plant (km) | Sites with asbestos waste at surface ($n = 293$) | Households with asbestos waste at surface within 100 m ($n = 347$) |
|---|---|---|---|---|
| 7472 (Goor) | 71 | 2.50 | 0 | 0 |
| 7471 (Goor) | 12,291 | 1.02 | 10 | 118 |
| 7478 (Diepenheim) | 2773 | 3.90 | 47 | 17 |
| 7496 (Hengevelde) | 2006 | 4.23 | 23 | 77 |
| 7497 (Bentelo) | 1434 | 6.10 | 8 | 2 |
| 7475 (Markelo) | 7234 | 6.68 | 74 | 75 |
| 7468 (Enter) | 7053 | 7.73 | 2 | 0 |
| 7275 (Gelselaar) | 715 | 7.75 | 1 | 4 |
| 7495 (Ambt Delden) | 2049 | 8.44 | 90 | 43 |
| 7491 (Delden) | 7186 | 9.11 | 1 | 1 |
| 7627 (Bornerbroek) | 1667 | 10.16 | 2 | 0 |
| 7554 (Twekkelo) | 1428 | 10.24 | 2 | 1 |
| 7161 (Neede) | 9918 | 10.50 | 6 | 1 |
| 7165 (Rietmolen) | 1125 | 10.57 | 2 | 0 |
| 7462 (Rijssen) | 15,150 | 10.57 | 1 | 2 |
| 7274 (Geesteren) | 1285 | 10.71 | 1 | 0 |
| 7482 (Haaksbergen) | 11,537 | 11.98 | 10 | 1 |
| 7555 (Woolde) | 9740 | 12.14 | 1 | 1 |
| 7451 (Holten) | 8838 | 12.80 | 1 | 0 |
| 7548 (Boekelo) | 2995 | 14.24 | 0 | 0 |
| 7151 (Hupsel-west) | 7496 | 14.28 | 0 | 0 |
| 7241 (Lochum) | 7821 | 14.36 | 0 | 2 |
| 7607 (Almelo) | 7315 | 15.14 | 1 | 0 |
| 7245 (Exel) | 3905 | 15.96 | 1 | 2 |
| 7481 (Haaksbergen) | 10,842 | 18.32 | 4 | 0 |
| 7688 (Daarle) | 1393 | 23.17 | 3 | 0 |
| 7215 (Joppe) | 412 | 24.79 | 1 | 0 |
| 7694 (Kloosterhaar) | 1579 | 30.45 | 1 | 0 |

were private property, of which 171 were actively used. About 31% of the households were exposed to multiple sites. The postal code areas with the highest number of polluted sites and exposed households were in the direct vicinity of the plant. Distance to the plant was associated with the number of sites ($r = 0.36$, $P$-value 0.06), and with the number of households ($r = 0.52$; $P$-value 0.005). The number of sites was associated with the number of households ($r = 0.53$; $P$-value 0.004). However, the population density was independent from these three proxy measures of environmental asbestos exposure.

Table 3 shows that the postal code areas with the highest exposure had on average 33.5 sites and 66 households per area, covering 0.55% (0.09–1.40%) of the total surface of the area available for daily human activities. The category with low exposure had postal code areas with at best one waste site and one exposed household, covering on average only 0.04% (0.00–0.20%) of the total area available for daily human activities. In the intermediate category 0.09% (0.01–0.34%) of the total surface available for daily activities was polluted with asbestos. The Cohen's weighted kappa between both approaches for categorization was 0.37.

Table 4 presents the person-years at risk and the estimated mortality of MM for the site and household approaches. For the high exposure category the SA estimated an additional 78 cases of MM, with a maximum of 431 cases of MM, during the 48-year period with asbestos contamination in the region. In the HA method these figures were 42 and 231, respectively. The categories with intermediate and low exposure to environmental asbestos contributed few cases to the overall expected extra mortality of MM in the research area.

## Discussion

There was a moderate agreement between the site and household exposure assessment approach (Pearson's correlation coefficient = 0.53; $\kappa = 0.37$). For the SA we calculated a total of 2.3 million person-years at risk with average exposure below 1674 fibres/m$^3$, resulting in an additional 1.8 cases of MM each year, with a maximum estimate of 9.6 cases of MM per year. For the HA approach these estimates were respectively 1.2 million person-years at risk,

**Table 3.** Stratification into three categories of likelihood of asbestos exposure with number of postal code areas, polluted sites, and exposed households within each stratum.

| Stratum | Sites with asbestos waste at surface | | Households with asbestos waste at surface within 100 m | |
|---|---|---|---|---|
| Likelihood of exposure | Postal code areas N | Number of sites Average | Postal code areas N | Number of households Average |
| Low exposure (0–1) | 13 | 0.88 | 17 | 0.65 |
| Intermediate exposure(2–5) | 6 | 2.50 | 5 | 2.40 |
| High exposure (6 and more) | 8 | 33.5 | 5 | 66.0 |

**Table 4.** Risk assessment for number of cases of pleural mesothelioma among inhabitants living in areas with local sites with asbestos pollution in the soil.

| Likelihood of exposure | Percentage of population exposed | Person-years at risk | Cases mean estimate | Cases maximum estimate |
|---|---|---|---|---|
| *Sites with asbestos waste at surface* | | | | |
| Low | 2.6 | 84,494 | 3.1 | 16.9 |
| Intermediate | 7.5 | 76,973 | 2.8 | 15.4 |
| High | 100.0 | 2,149,807 | 78.3 | 430.8 |
| *Households with asbestos waste at surface within 100 m* | | | | |
| Low | 1.0 | 40,082 | 1.5 | 8.0 |
| Intermediate | 3.6 | 45,618 | 1.7 | 9.1 |
| High | 100.0 | 1,150,519 | 41.9 | 230.5 |

and 0.9 and 5.2 cases of MM per year. Given the exposure period 1960–2007 and a latency period of at least 20 years, these additional cases of malignant mesothelioma will occur from 1980 onwards.

*Restrictions in Available Information*

In contrast to other studies we could not take into account the asbestos emission from the factory during its asbestos producing period (Magnani et al., 2000, 2001; Maule et al., 2007; Kurumatani and Kumagai, 2008; Musti et al., 2009). Although some information was available on the total amount of asbestos emitted by the pipe plant permitted by legal authorities in certain years, the information was too sparse. As measurements within 2 km of the plant could not show an increased air concentration due to plant emissions (Biesheuvel et al., 2003), we assumed that the polluted sites contributed most to the asbestos exposure of inhabitants.

The information on polluted sites was derived from soil investigations executed by an independent organization certified for asbestos measurements. All polluted sites that were identified for soil decontamination as part of the official clean up regulations enacted in 2003 were included in the study. The identification of privately owned polluted sites depended on voluntary notification by owners and, hence, it is unknown how many sites will have been missed. As the clean up activities were paid for by the national government, it is assumed that the coverage is high among larger sites and road and dirt tracks often used by local citizens. We also assume that additional, smaller polluted sites that will be identified in the immediate future will not greatly affect the overall distribution of polluted sites throughout the region. When more sites will be discovered, the risk assessment may present an underestimation of the risk on MM in the region.

For assessing the person-years at risk, the demographic development of the research area was investigated. Given the low number of inhabitants in some postal code areas, the age-specific distribution of inhabitants was not available for every postal code area. Therefore, we extrapolated the demographic composition of the village Goor (areas 7471 and 7472) to all postal code areas in the research area.

*Internal Validity*

The exposure assessment used was based on an investigation during the late 1980s on asbestos exposure emerging from an asbestos-polluted road. This investigation used robust week-long air samples during 5 months from late spring to early autumn (Boeft, 1987). During wintertime no samples were taken and, therefore, characteristic meteorological conditions for wintertime such as rainfall, frost, and storms could have affected the estimated annual air concentrations of asbestos. However, additional analyses based on the data presented in the original report does not show significant relationships of average rainfall, temperature, air humidity, evaporation, and wind direction with the weekly asbestos concentrations during the 5 months of air sampling. This may be explained by the large variation in weather conditions within a week.

Thus, these asbestos measurements were assumed to reflect exposure patterns during the year of investigation and also during the total period of environmental exposure. We have no information to determine whether the asbestos exposure ascertained in mid 1980s is a fair approximation of exposure conditions from 1960 onwards. The concentrations and fibre type were analysed with transmission electron microscopy. Significant elevated concentrations of fibres (chrysotile 78%; crocidolite 22%) were found within 5 m distance to the road. At 100 m distance to the road insignificant elevated concentrations were found, compared with the background concentration (1000 m distance). The pattern of the decrease in asbestos concentration between 5 and 100 m distance was unknown. Therefore, the concentration measured at 5 m was used as worse-case scenario for a maximum distance of 100 m to the road.

In the risk assessment a yearly average concentration of approximately 1700 fibres/$m^3$ was used. There are several reasons why this value may be challenged. First, as stated before it is expected that not all polluted sites have been identified for soil decontamination in 2003 and, hence, the exposure will be underestimated to some extent. On the other hand, some polluted sites will have been removed in previous years, for example because of paving local tracks. Second, the exposure assessment was based on week-long samples over a 5-month period along a local road. During this investigation few hour-long measurements were conducted in dust clouds because of car traffic on a dry day, showing levels varying between 6000 fibres/$m^3$ and 80,000 fibres/$m^3$ (Boeft, 1987). These findings indicate that local roads with higher traffic density will have resulted in higher long-term average concentrations. Third, environmental exposure along a road may differ from exposure of individuals who spend considerable time on polluted driveways and yards in the direct vicinity of their home. Such outdoor behaviour may increase exposure substantially. Fourth, asbestos waste on roads and yards may be transported into a house by clothes and footwear, similar to the well-known route whereby asbestos workers give rise to asbestos exposure of their next of kin through their work clothes (Magnani et al., 1993; Magnani et al., 1995; Ferrante et al., 2007; Rake et al., 2009; Reid et al., 2008a, b). An investigation in nine houses with erosion of asbestos roof plates on their yards showed indoor concentrations with a maximum of 27,000 fibres/$m^3$ that were substantially higher than outdoor concentrations well below 1000 fibres/$m^3$ (Tromp and Tempelman, 1994). It remains unclear how the estimated annual concentration of 1700 fibres/$m^3$ in the general environment translates into cumulative exposure of individuals. The reasons presented make it more likely that the average exposure of individual citizens in this area is underestimated rather than overestimated.

The study compared a site approach with a household approach, based on polluted sites *versus* exposed households. Although there was a moderate agreement between both approaches, the overall risk assessment presented similar results. It is assumed that the HA will be better able to discriminate between exposed and nonexposed persons in a postal code area than the SA, but the HA approach in this study was limited because of the fact that no information was available about patterns of movement in and around the house relative to exposure sources. For example, the HA does not take into account polluted sites at more than 100 m distance of a household, but household members may be regularly exposed to asbestos when using a particular road in the vicinity of their house. Without detailed information on movement patterns of citizens it will not be possible to verify whether the HA has less random misclassification than the SA.

The composition of the dynamic cohort was based on annual data (1960–2007) of the village of Goor about inhabitants, births, deaths, settlements, and migration. This information was considered representative for the whole research area, comprising of urban and rural areas. As the starting year we have chosen 1960, since most asbestos-containing materials were brought into the environment from 1960 onwards. Some information is available, indicating that migration in the rural area was lower than in the villages and, hence, these inhabitants may have been exposed to asbestos from an earlier age onwards and therefore could have had a higher risk on MM. In both SA and HA, the high exposure category contributes most to the person-years at risk and, thus, to MM cases. This is because of the assumption that the proportion of persons exposed to asbestos in these postal areas was assumed to be 100%, whereas in the categories with intermediate and low exposure less than 10% of the inhabitants were considered as exposed. The proportion-exposed persons within the exposure categories is a reflection of the distribution on number of sites or number of exposed households within postal code areas and, hence, seems to be a reasonable assumption.

*External Validity*

Exposure to asbestos in the environment is associated with an increased occurrence of MM, as shown in several studies. In general, these studies were not able to quantify the magnitude of environmental asbestos exposure, but used proxy measures such as distance to source. This study showed that the distance of the plant to the midpoint of a postal code area was associated with number of polluted sites and exposed households, but correlation coefficients between 0.36 and 0.52 indicate that spatial risk models based on proximity measures (Magnani et al., 2000; Magnani et al., 2001; Maule et al., 2007; Kurumatani and Kumagai, 2008; Musti et al., 2009) may give rise to considerable misclassification.

The estimated exposure to environmental asbestos, expressed by time-weighted exposure of 1 month, was below

2000 fibres/$m^3$ ($>5\,\mu m$) in this particular situation. These concentrations are within the reported environmental concentrations of 1000 to 4500 fibres/$m^3$ ($>5\,\mu m$) in Casale Monferrato in Italy, although the latter were short-term (4–8 h) measurements (Maule et al., 2007). Based on an available risk assessment (Hodgson and Darnton, 2000), it was estimated that these low concentrations will give rise to 1–2 cases of MM each year in a region with currently 130,000 inhabitants. It is important to emphasize that the presence of crocidolite in the majority of contaminated sites has strongly influenced this estimated burden of MM. It is expected that the historical burden of MM in this area is much higher because of domestic exposure among household members of asbestos workers and, of course, the occupational exposure among workers in the asbestos cement factory. As the factory abandoned the use of asbestos in 1993, it is expected that the proportion of environmental cases of MM will become more prominent in the near future.

In conclusion, this study illustrates that asbestos waste on the surface of roads and yards may result in long-term exposure to asbestos of approximately 2000 fibres/$m^3$ and that these concentrations will result in a couple of cases of MM each year. Although distance to plant, number of polluted sites, and number of exposed households were associated, the modest agreement among these measures of exposure indicate that the exposure assessment strategy chosen in a particular study may result in considerable misclassification. Without detailed information on individual behaviour within the polluted area, it is difficult to demonstrate that a more individually oriented approach will perform better than an ecological approach.

## Conflict of interest

The authors declare no conflict of interest.

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