RIVM report 711701037/2004

Relevancy of human exposure via house dust to the contaminants lead and asbestos

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This investigation has been performed by order and for the account of the Ministry of Housing, Spatial Planning and the Environment (VROM), Directorate-General for Environmental Protection, Directorate of Soil, Water and Rural Area (BWL), within the framework of project 711701, 'Risks in relation to soil quality'.

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Abstract

The present report addresses the issues whether house dust is likely to contribute substantially to the exposure of humans, in particular for the contaminants lead and asbestos. House dust consists for 30-70% of soil material, indicating that contaminated soil can lead to contaminated house dust. It is concluded that exposure to lead via house dust should be included in risk assessment. The studies in which the amount of soil ingested by children are estimated use tracers in soil that are obviously both present in soil outdoors and in soil in house dust. Therefore, using this amount of soil ingested by children to cover both exposure of lead via soil outdoors and soil in house dust is reasonable. However, a correction factor of 2 is recommended to account for enrichment of lead in house dust in comparison with outdoor soil. This factor also accounts for lead in house dust from other lead sources than soil contamination. Significant contribution of asbestos to house dust from asbestos contaminated soil may occur at soil concentrations over 100 mg/kg. It is recommended to determine asbestos levels in house dust in houses adjacent to sites that are contaminated with asbestos above 1000 mg/kg for nonfriable asbestos, and above 100 mg/kg for friable asbestos. These recommendations are in agreement with a draft assessment protocol on site specific assessment of human risks to soil contamination with asbestos. For further recommendations concerning the risk assessment of asbestos in house dust, we refer to this protocol.

Key words: house dust; lead; asbestos; CSOIL; risk assessment; children

Rapport in het kort

In het rapport wordt bestudeerd of huisstof substantieel kan bijdragen aan de blootstelling van de mens aan contaminanten, met name voor de contaminanten lood en asbest. Huisstof bestaat voor 30-70% uit bodemmateriaal, wat betekent dat verontreinigde bodem kan leiden tot verontreinigd huisstof. Er is geconcludeerd dat blootstelling aan lood via huisstof zou moeten worden meegenomen in risicobeoordeling van bodemverontreiniging. De studies waarin de inname van bodem door kinderen worden geschat gebruiken merkstoffen in bodem die zowel in bodem buiten als in bodem in huisstof zitten. Het is daarom redelijk om deze hoeveelheid bodem te gebruiken om zowel blootstelling aan lood via bodem buiten en huisstof te omvatten. Echter, een correctiefactor van 2 wordt aanbevolen om voor verrijking van lood in huisstof te verdisconteren. Deze factor verdisconteert tevens voor andere loodbronnen dan lood afkomstig van gecontamineerde bodem. Een significante bijdrage van asbest in bodem aan asbest in huisstof kan gebeuren bij concentraties hoger dan 100 mg/kg. Daarom wordt aanbevolen de asbestconcentraties in huisstof te bepalen in huizen waarbij de bodem is verontreinigd met asbest boven de 1000 mg/kg voor hechtgebonden asbest, en boven de 100 mg/kg voor niet-hechtgebonden asbest. Deze aanbevelingen zijn in overeenstemming met een concept beoordelingsprotocol voor locatie-specifieke risicobeoordeling van asbest in bodem. Enkele aanmerkingen op dit protocol zijn beschreven. Voor verdere aanbevelingen met betrekking tot asbest in huisstof wordt verwezen naar dit protocol.

Trefwoorden: huisstof; lood; asbest; CSOIL; risicobeoordeling; kinderen

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Samenvatting

Blootstelling aan bodemverontreiniging voor de humane risicobeoordeling wordt momenteel meestal gebaseerd op orale inname van bodem en voedsel, inhalatie van lucht, en blootstelling via de huid. Blootstelling aan contaminanten via huisstof wordt in Nederland tot op heden niet expliciet meegenomen. In het huidige rapport wordt, op basis van literatuur, onderzocht of huisstof substantieel kan bijdragen aan humane blootstelling aan contaminanten, in het bijzonder voor de contaminanten lood en asbest. Om in te kunnen schatten of verontreiniging in huisstof is veroorzaakt door verontreinigde grond is tevens de bijdrage van bodem aan huisstof bestudeerd, en is bekeken of er een relatie bestaat tussen de lood- en asbestconcentratie in bodem buiten en in huisstof.

Huisstof algemeen. In het rapport is eerst algemene informatie over huisstof verzameld. In de meeste studies beschreven in de literatuur is minder dan 1 g stof/m^2 gevonden voor harde oppervlakken, wat in overeenstemming is met de huidige aanname in het blootstellingsmodel CSOIL van 0,56 g/m^2.
Verscheidene experimentele studies suggereren dat huisstof voor 30-70% bestaat uit bodemmateriaal. Deze range is lager dan de 80% die momenteel wordt gebruikt in het CSOIL model. Een verlaging van dit percentage wordt daarom geadviseerd. De concentratie zwevend stof in de lucht in huizen is over het algemeen 50 μg/m^3 of minder. Stofconcentraties in de lucht waren hoger vlakbij iemand (persoonlijke meting) of op lage hoogte (representatief voor kinderen). Persoonlijke metingen zijn waarschijnlijk representatiever voor humane blootstelling. Daarom worden persoonlijke metingen van 60 μg/m^3 voor matig drukke locaties en van 100 μg/m^3 voor drukke locaties zoals klaslokalen aangeraden voor toekomstig gebruik in CSOIL.

Lood. Vervolgens is de bijdrage van huisstof aan de blootstelling van lood bestudeerd. Op basis van de literatuur is geconcludeerd dat de gemiddelde loodconcentratie in huisstof meestal ligt in de range 300-700 mg/kg. De loodconcentratie in huisstof is over het algemeen, gemiddeld een factor 3, hoger dan in bodem buiten.
In sommige studies is een directe relatie gevonden tussen de loodconcentratie in huisstof en in bodem, terwijl andere studies niet een dergelijke relatie aantoonden. Desalniettemin suggereren veel experimentele en epidemiologische studies dat huisstof een belangrijke bron van blootstelling aan lood kan zijn. Hoewel weinig informatie over de hoeveelheid huisstof die ingeslikt wordt bekend is, geven berekeningen aan dat de blootstelling van kinderen aan lood via ingestie van bodem en huisstof waarschijnlijk van dezelfde ordegrootte is, terwijl blootstelling aan lood via inhalatie verwaarloosbaar is. Daarom zou blootstelling aan lood via huisstof expliciet moeten worden gemaakt in de risicobeoordeling. De studies waarin de hoeveelheid bodem wordt geschat die kinderen inslikken gebruiken merkstoffen die aanwezig zijn in zowel bodem buiten als in bodem in huisstof. Het is daarom redelijk om deze hoeveelheid bodem die kinderen inslikken te gebruiken voor bodemingestie zowel afkomstig van bodem buiten en bodem in huisstof (in totaal 100 mg/dag). Echter, een correctiefactor van 2 wordt aanbevolen om de verrijking van lood in huisstof ten opzichte van lood in bodem te verdisconteren. Deze factor verdisconteert ook de bijdrage van andere loodbronnen dan verontreinigde bodem. Op dit moment is het niet mogelijk de bijdrage van verrijking van lood in huisstof en de bijdrage van lood uit andere bronnen dan bodem te scheiden. Verder onderzoek naar de hoeveelheden huisstof en bodem die worden ingeslikt, en naar de hoeveelheid lood in bodemmateriaal in huisstof wordt aanbevolen.

Asbest. Tot slot is de bijdrage van huisstof aan de blootstelling van asbest bestudeerd. De achtergrondconcentratie van asbest in huisstof is ongeveer 1×10^3 vezels/cm^2. Deze waarde kan toenemen met een factor 1×10^4 in gecontamineerde huizen. Er is mogelijk een relatie tussen asbest in huisstof en asbest in de lucht. De achtergrondconcentratie van asbest in de binnenshuislucht is ongeveer 1×10^3 vezels/m^3.
Asbest gecontamineerde bodem draagt waarschijnlijk slechts in een enkel geval bij aan asbestverontreiniging binnenshuis. In deze gevallen is de bodem gecontamineerd met hoge asbestconcentraties en ligt de gecontamineerde bodem in de directe omgeving van het huis. Gebaseerd op een hypothetische berekening kan een significante bijdrage van bodem aan huisstof worden verwacht bij bodemconcentraties hoger dan 100 mg/kg. Daarom wordt aanbevolen om de asbestconcentratie in huisstof te meten als de asbestconcentratie in bodem hoger is dan 1000 mg/kg voor hechtgebonden asbest, en hoger dan 100 mg/kg voor niet-hechtgebonden asbest. Deze aanbevelingen zijn in overeenstemming met een concept beoordelingsprotocol voor locatie-specifieke humane risico's van bodemverontreiniging met asbest. In dit protocol, opgesteld door TNO-MEP, wordt een gedetailleerde procedure omschreven welke de beoordeling van asbest in huisstof omvat. Voor verdere aanbevelingen voor risicobeoordeling van asbest in huisstof wordt verwezen naar dit protocol. Enkele aanmerkingen op dit protocol zijn beschreven.

Beleidsrelevantie
Blootstelling aan contaminanten via huisstof zou, tenminste voor lood en andere immobiele contaminanten, bij de beoordeling van risico's van bodemverontreiniging moeten worden betrokken en op termijn moeten worden geïmplementeerd in het blootstellingsmodel CSOIL. Deze aanpassing van CSOIL vormt een onderdeel van een verbeterde methode voor de bepaling van locatiespecifieke humane risico's, waaraan op het RIVM momenteel onderzoek wordt verricht. Deze verbeterde methode kan onderdeel uit gaan maken van de uitwerking van het nieuwe bodembeleid, zoals geformuleerd in de Beleidsbrief bodem van December 2003. De verbeterde methode voor de bepaling van (locatiespecifieke) humane risico's zou onderdeel uit kunnen maken van het Saneringscriterium en/of van een procedure welke kan worden gebruikt om regionaal te bepalen Ambitieniveaus af te leiden. Afhankelijk van de formele positionering van de methode zal de rol van blootstelling aan contaminanten in huisstof in overleg met 'het beleid' in een volgende fase in een op het doel afgestemde vorm worden gegoten (bijvoorbeeld als onderdeel van een richtlijn voor beoordeling van humane risico's waar ook het blootstellingsmodel CSOIL binnen kan passen).

Summary

In current human risk assessment, exposure to contaminants is generally estimated on the basis of oral intake of soil, food and liquids, inhalation of air, and exposure via the skin. Exposure to contaminants via house dust has, up till now, not been considered in the Netherlands. The present report addresses the issues whether house dust is likely to contribute substantially to exposure of contaminants to humans, in particular, for the contaminants lead and asbestos. In addition, to investigate whether contamination of house dust is caused by soil material in dust, the contribution of exterior soil to house dust is investigated, and whether a relationship exists between lead or asbestos concentration in exterior soil and house dust.

General information house dust. First, general information on house dust was collected. Most studies were seen to have found less than 1 g dust/m^2 for hard surfaces, which is in agreement with the current assumption in the exposure model CSOIL of 0.56 g/m^2. Several experimental studies indicate that house dust consists for 30-70% of soil material. This range is lower than the 80% that currently assumed by the CSOIL model. A reduction is therefore advised.
The concentration of particulate matter in air inside homes was found, in general, to be 50 μg/m^3 or less. Particulate matter concentrations were higher when measured near the subject (personal samples) or at lower heights (representative for children), than in bulk indoor air. Personal samples are probably more representatives for human exposure. Therefore, personal samples of 60 μg/m^3 for moderately crowded places, and 100 μg/m^3 for crowded places such as classrooms are recommended in CSOIL for future use.

Lead. Subsequently, contribution of house dust to lead exposure was assessed. Based on studies described in literature, we found a mean concentration of lead in house dust generally occurred in the range 300-700 mg/kg. The lead concentration in house dust is generally averaged at a factor 3 higher than in exterior soil.
Some studies found a direct relationship between lead concentration in house dust and in soil, whereas other studies did not. Yet, many experimental and epidemiological studies suggest that house dust can be a major source of exposure to lead. Although very little information is available on amounts of dust ingestion, calculations suggest that exposure of children to lead via ingestion of soil and house dust is probably in the same order of magnitude, whereas exposure to lead via inhalation is negligible. Therefore, exposure to lead via house dust should be included in risk assessment.
The studies in which the amount of soil ingested by children are estimated, used tracers in soil that are obviously both present in soil outdoors and soil in house dust. Therefore, it is reasonable to use this amount of soil ingested by children to cover both soil outdoors and soil in house dust (100 mg/day). However, an extra correction factor of 2 is recommended to account for enrichment of lead in house dust in comparison with outdoor soil. This factor also accounts for sources of lead contamination in house dust other than contamination via soil. At present, it is not possible to separate the contribution of enrichment of lead in house dust and the contribution of lead from other sources than soil to the exposure of humans to lead. Further research about the amounts of dust and soil ingestion, and the contribution of house dust and soil to lead exposure is recommended.

Asbestos. Finally, the contribution of house dust to the exposure of asbestos was studied. Background levels of asbestos in house dust are about 1×10^3 fibres per cm^2. This value can increase up to a factor 1×10^4 for contaminated houses. There is possibly a relationship between asbestos in house dust and asbestos in indoor air. For background houses, asbestos levels in air are approximately 1×10^3 fibres per m^3.
Asbestos-contaminated soil contributes probably only in a few cases to asbestos levels inside houses. In these cases, the soil would have to be contaminated with high levels of asbestos and the contaminated site would have to be found in the vicinity of the house. On the basis of a hypothetical calculation, asbestos-contaminated soil may contribute significantly to house dust at soil concentrations greater than 100 mg/kg. Therefore, it is recommended to determine asbestos levels in house dust in houses adjacent to sites that are contaminated with asbestos above 1000 mg/kg for nonfriable asbestos, and above 100 mg/kg for friable asbestos. These recommendations are in agreement with a draft assessment protocol by TNO-MEP on site specific assessment of human risks to soil contamination with asbestos. Detailed procedures including assessment of asbestos in house dust, are described in the protocol. Therefore, for further recommendations for risk assessment of asbestos in house dust, we refer to this protocol. Several comments on the protocol are included in the report.

Usefulness for policy making
Exposure to contaminants via house dust should, at least for lead and other immobile contaminants, be included in the risk assessment of soil contamination and, in due time, be implemented in the exposure model CSOIL. This adaptation of CSOIL is part of an improved method for the assessment of human risks, which RIVM is currently working on. The improved method can be part of the development of the new soil policy, as formulated in the ‘policy letter on soil’ of December 2003. The improved method for the assessment of location specific human risks could be part of a remediation criterion and/or of the procedure that can be used to determine soil quality criteria for regional use. Depending on the status of the method, the role of contaminant exposure via house dust will, in consultation with policy makers, in the next phase be put in a for the purpose appropriate form (for example as part of the guideline on the assessment of human risks).

1. General introduction

In present human risk assessment, exposure to contaminants is estimated based on oral intake of soil, food and liquids, inhalation of air, and exposure via the skin. Exposure to contaminants via house dust has, up till now, not been explicitly considered in exposure assessment in the Netherlands (Lijzen et al., 2001). Because most individuals spent relatively little time outdoors and much time indoors, exposure to house dust may be an important exposure route. House dust is often resuspended due to the dry atmosphere and activity in the house, and contaminants may remain in the house for a long time. In addition, house dust consists of fine particles, which adhere well to skin and clothing (Duggan et al., 1985) and are easily ingested or respired. Contaminants in house dust can originate from many sources, such as interior sources, from soil that is brought inside, or via (particulates in) air.

The present report addresses the issues whether house dust is likely to contribute substantially to the exposure of contaminants, in particular for lead (Pb) and asbestos, and to what extent exterior soil contributes to house dust. We mainly focus on exposure to children because children ingest more soil and house dust than adults. Also from a toxicological point of view children are the group at risk. Pb already affects children at low doses. Children are also of main concern for asbestos exposure, since they have a long life expectancy and exposure to asbestos can cause (lung) cancer several decades after exposure. The reason for choosing to investigate Pb is that Pb is a contaminant of primary concern in the Netherlands, due to its occurrence and its effects on human health. The contribution of asbestos in house dust to human exposure was investigated as Swartjes et al. noticed that information about this issue was missing and recommended further research (Swartjes et al., 2003).
With the information gathered in this report, exposure of children to Pb and asbestos via ingestion of inhalation of house dust is calculated according to the exposure model CSOIL. If possible, values for characteristics of house dust obtained from studies described in the literature are compared to values of the same characteristics that were estimated by Hawley (Hawley, 1985). Estimates of Hawley are currently used as input for the exposure model CSOIL (Otte et al., 2001).

The present report is composed in the following manner. Chapter 2 is dedicated to some general characteristics of house dust. The origin of house dust, amounts of house dust on surfaces in the house, the fraction of soil in house dust, and amounts of house dust in air are addressed.
Chapter 3 deals with Pb. The levels of Pb in house dust, the relationship between the concentration of Pb in house dust and in exterior soil, and the concentration of Pb in air are addressed. Subsequently, exposure of Pb via house dust in exposure assessment in Canada and the USA is discussed, and the contribution of Pb in house dust, outdoor soil, and air to Pb exposure in children is addressed qualitatively and quantitatively when possible. Finally, recommendations for risk assessment are given. Main conclusions for Pb are summarised at the end of Chapter 3.
In Chapter 4 the same topics as for Pb are dealt with for asbestos. When available, concentrations of asbestos in house dust are summarised, information about the relationship between the asbestos in house dust and exterior soil, the concentration of asbestos in air, and the contribution of house dust to asbestos exposure is given.

Subsequently, recommendations for risk assessment on asbestos exposure via house dust are given.
Overall conclusions are summarised in Chapter 5.

It should be noted that an extensive literature search is performed on the issues addressed in this report. However, for some issues it was not feasible to study all related literature. Yet, the authors are convinced that the present report gives a representative picture of the different aspects on the contribution of house dust to Pb and asbestos exposure.

2. General characteristics house dust

The purpose of this chapter is to get notion of some general characteristics of house dust. First, the origin of house dust is addressed (§ 2.1). Subsequently, an overview is given of studies in which amounts of house dust are determined, to get a picture of the amounts of house dust that can be present on surfaces in the house under normal conditions (§ 2.2). The next section deals with the contribution of exterior soil in house dust (§ 2.3). Finally, the amounts of dust in air are addressed (§ 2.4), and conclusions are summarised (§ 2.5).

2.1 What is the origin of house dust

House dust is a heterogeneous mixture. A number of sources contribute to this mix, including tracked-in or resuspended soil particles, clothing, atmospheric deposition of particulates, hair, fibres (artificial and natural), molds, pollen, allergens, bacteria, viruses, arthropods, ash, soot, animal fur and dander, smoke, skin particles, cooking and heating residues, and building components among others ((Paustenbach et al., 1997), and references in Paustenbach).

2.2 Amounts of house dust

2.2.1 Sampling

Table 1 presents a list of the amounts of house dust that were determined in several studies. Various methods exist for sampling of house dust. The methodapplied can highly affect the outcome. In a study by Sterling et al. (Sterling et al., 1998), three vacuums and one wipe method were evaluated for the collection of house dust. The Pb loading, i.e. the amount of Pb per surface area, was determined with these methods; the amounts of house dust were not reported. The results showed that the Pb loading could differ to factors sometimes higher than 100 between sampling methods. Probably the dust loading determined by different sampling methods can vary to a similar extent. The two most reliable methods were the wipe and one vacuum method, showing differences up to a factor 28, with the wipe method giving lower collection efficiencies. The vacuum method was highly efficient in removing deeply embedded dust, whereas the wipe method collected surface dust, especially on carpets. Another vacuum method gave even lower efficiencies than the wipe method (Sterling et al., 1998), indicating that vacuum methods can show very large differences in sampling efficiencies.

2.2.2 Dust loading

As can be seen in Table 1, the geometric mean of the range of dust settled in cup or on plates based on experimental studies varies between 0.0022 and 0.0089 $g \times m^{-2} \times day^{-1}$, which is considerably lower than the geometric mean of the range of dust loading determined by wipes and vacuum, which ranges between 0.1 and 11.5 g/m^2. Amounts of house dust determined by wipes and vacuum are the results of several days. In addition, cleaning does not remove all dust, as can be seen in Table 1 from the study by Rhoads et al. (Rhoads et al., 1999). This study showed a reduction of a factor 1-2.8 in dust loading

after cleaning. Similarly, Rich et al. found a reduction of a factor 1.1-3.6 in dust loading after cleaning (Rich et al., 1999). Most vacuum cleaners do not trap small (<20 μm) particles and will simply re-enter them into the air (references in (Paustenbach et al., 1997)).
The amounts of dust determined on floors and window sills show large differences, with geometric mean dust loadings between 0.41 and 6.5 g/m^2 for floors, and between 0.76 and 11.5 g/m^2 for window sills. The amount of dust determined from carpets with vacuuming ranged from 7.4 to 8.4 g/m^2. The lower variation is probably because only one study is considered using the same sampling method.

2.2.3 Resuspension

Meyer et al. (Meyer et al., 1999) determined that the number of persons living in a residence was significantly associated with elevated amounts of dust sedimented per day, probably due to increased indoor activities such as vacuuming, sweeping, cleaning, and children playing. Thatcher and Layton have shown that the resuspension rate is particle size dependent (Thatcher et al., 1995). Particles with diameters of 5-25 μm are most readily resuspended and even light activity such as walking into and out of the room can have a significant impact on the concentration of airborne particles greater than 5 μm, whereas particles of 0.3 - 1 μm are not affected by either cleaning or walking (Thatcher et al., 1995).

2.2.4 Comparison to reference value

Estimates of Hawley have been used as input for the exposure model CSOIL. Hawley assumes an average dust covering of surfaces of 0.56 g/m^2 (Hawley, 1985). Most studies estimate a similar or lower value for dust covering of hard surfaces (Table 1), except for Rich et al. (Rich et al., 1999). For unknown reasons, they found dust loadings of a factor 12-21 higher.
Hawley assumes an average dust fall inside the house of 0.08 g×m^{-2}×day^{-1} (Hawley, 1985). Dust fall determined in studies by Meyer et al. (Meyer et al., 1999) and Edwards et al. (Edwards et al., 1998) was a factor 9-36 lower than the value assumed by Hawley. The estimate by Hawley was based on available data at that time and no reference or further information was given about the derivation of this figure. Therefore, the values of Meyer et al. and Edwards et al. are recommended, thus assuming that dust fall is approximately 0.002-0.009 g×m^{-2}×day^{-1}.

Table 1: Geometric mean, except where indicated differently, of dust loadings (g/m^2) and dust fall ($g \times m^{-2} \times day^{-1}$) measured in households.

Study	***Dust loading (g/m^2)***	***Dust fall ($g \times m^{-2} \times day^{-1}$)***	***Surface***	***N***	***Remarks***
Hawley 1985*	0.56	0.08	N/a	N/a	*
Rhoads 1999	0.41 and 0.42		Floor	42 and 42	Wipes (Pb intervention and control group)
Rhoads 1999	0.76 and 0.83		Sill	39 and 40	Wipes (Pb intervention and control group)
Rhoads 1999	8.4 and 7.4		Carpet	27 and 28	Vacuum (Pb intervention and control group)
Rhoads 1999	0.38		Floor	40	After thorough cleaning, wipes
Rhoads 1999	0.38		Sill	36	After thorough cleaning, wipes
Rhoads 1999	3.0		Carpet	22	After thorough cleaning, vacuum
Rich 1999	6.5		Floor (hard surfaces)	184	Wipes
Rich 1999	11.5		Window sill	78	Wipes
Meyer 1999		0.0089 0.0085-0.0094 (95% CI) 0.0013-0.049 (range)	N/a	454	House dust settled in a cup during a period of 1 year at a height of 1.7 m
Edwards 1998		0.0037 ± 0.0013 (arithmetic mean)	N/a	4	Dust settled on sample plates during a 30-day period in **summer** at a height of 0.3 and 1.5 m
Edwards 1998		0.0022 ± 0.0013 (arithmetic mean)	N/a	4	Dust settled on sample plates during a 30-day period in **winter** at a height of 0.3 and 1.5 m
Laxen 1988	Median: 0.14 10% 0.048; 90% 0.42		Floor	507	Commercial vacuum cleaner
Range all studies	***0.38-11.5***	***0.002-0.08***			

N/a: not available.

* Estimate for risk assessment based on available data at that time; no reference of further information was given about the derivation of this figure. The estimates of Hawley have been used as input for the exposure model CSOIL.

2.3 Contribution of soil to house dust

Various researches have examined the contribution of exterior soil to interior dusts, see Table 2.

Table 2: Estimates of the relative contribution of exterior soil to house dust. Estimates were based on research described in articles, except where indicated differently.

Study	***% soil in house dust***
Hawley (1985) *	>80
Thornton et al. (1985)**	20
Camann and Harding (1989) **	50
Fergusson and Kim (1991)	32-50
Calabrese and Stanek (1992)	31
Sterling (1998)	37 (based on particle volume weighting) 8 (based on particle concentration weighting)
EPA (1998)	70

* Estimate for risk assessment based on available data at that time; no reference or further information was given about the derivation of this figure.
** Value and reference adopted from Paustenbach et al. (Paustenbach et al., 1997).

Estimates of the contribution of soil to house dust range from 8 to >80%, depending on a wide variety of site-specific factors and methodological approaches. The value of 80% was mentioned by Hawley without any reference or further information given about the derivation of this figure (Hawley, 1985). Most studies suggest that approximately 30-50% of house dust originated from exterior soil. However, the US-EPA uses a default value of 70%, i.e. 0.70 g soil/g dust, in the IEUBK model (US-EPA, 1998). The IEUBK model stands for Integrated Exposure Uptake BioKinetic model, and is used to predict the risk of elevated blood lead levels in children that are exposed to environmental Pb from many sources. According to their own account, the default value of 70% in the IEUBK model reflects an analysis of empirical relationships between soil and dust lead concentrations measured in a variety of residential communities (US-EPA, 1998). Therefore, 30-70% of soil in house dust seems a good estimate for the exposure assessment of house dust.

2.4 Dust in air

An overview of the concentrations of suspended particulate matter in air, i.e. suspended dust, is presented in Table 3. The value for particulate matter in indoor air that is used in the exposure model CSOIL is 52.5 $\mu g/m^3$ (outdoor air 70 $\mu g/m^3$; Otte et al., 2001). This was based on Hawley, who assumes that the concentration of suspended dust in the air inside homes is 75% of that in the outside air (Hawley, 1985). By Whitby et al., a study cited by Hawley, the concentration of airborne particulate matter was determined to be 65 $\mu g/m^3$ indoors and 93 $\mu g/m^3$ outdoors (Hawley, 1985). It should be noted that this study was conducted in the 1950s.

2.4.1 Overview several recent studies on dust in air

More recently, Janssen et al. measured personal, indoor, and outdoor particulate matter levels among adults and children (10-12 years of age) in the Netherlands (Janssen et al., 1998a;

Janssen et al., 1997; Janssen, 1998b), see Table 3. PM_{10} refers to the Particulate Matter concentration with a cut off diameter of 10 μm. Similarly, $PM_{2.5}$ refers to the Particulate Matter concentration with a cut off diameter of 2.5 μm. Personal samples were measured by a device worn by the subject. In the adult study, outdoor concentrations (mean PM_{10} 41.5 $\mu g/m^3$) exceeded indoor concentrations (mean PM_{10} 35.0 $\mu g/m^3$), but underestimated personal adult exposure (mean PM_{10} 61.7 $\mu g/m^3$) (Janssen et al., 1998a). In contrast, for children, mean indoor PM_{10} levels in classrooms (81-157 $\mu g/m^3$) exceeded outdoor levels (39 $\mu g/m^3$), and were in the same range as personal samples (105 $\mu g/m^3$) (Janssen et al., 1997; Janssen, 1998b). It should be noted that the high PM_{10} values of Janssen et al. measured in classrooms (81-157 $\mu g/m^3$) were 7-8 hour average values for hours during school time, whereas 24 hour average PM_{10} concentrations were a factor two to five lower (46-74 $\mu g/m^3$). The large number of persons in the classrooms probably caused high resuspendation resulting in the high PM_{10} levels.
Janssen et al. found that personal PM_{10} concentrations of both adults and children were reasonably well correlated over time with outdoor PM_{10} concentrations, with an intercept of 11.5 ($\mu g/m^3$), a slope of 0.47, and a Pearson's correlation coefficient of 0.75 for the adult study (Janssen et al., 1998a; Janssen et al., 1997; Janssen, 1998b). Personal fine particles ($PM_{2.5}$) exposures were highly correlated with outdoor $PM_{2.5}$ concentrations, probably due to the smaller impact of (re)suspendation of coarse particles on $PM_{2.5}$ than on PM_{10}. After taking the influence of exposure to external tobacco smoke, physical activity and the high PM_{10} concentrations in classrooms into account, the major part of the difference between personal and outdoor PM_{10} concentrations in children was explained.
In another study, Sally Liu et al. determined that personal, indoor, and outdoor concentrations were similar (see Table 3). Hence, the conclusion of Hawley that the concentration of suspended dust in the air inside homes is 75% of that in the outside air was not confirmed by the thorough studies of Janssen et al. and Sally Liu et al. (Janssen et al., 1998a; Sally Liu et al., 2003; Janssen et al., 1997; Janssen, 1998b).

The studies also indicate that children are exposed to higher amounts of PM_{10} than the adults in the same room, due to the higher levels of particulate matter at lower height (Beamer et al., 2002). In addition, PM_{10} concentrations were related to the number of people and activity occurring in the room, which is likely to be higher in the presence of children.

2.4.2 Comparison to reference values

The values for particulate matter in indoor air for the more recent studies were between 13 and 35 $\mu g/m^3$ inside homes, between 41 and 58 $\mu g/m^3$ for daycare facilities (Beamer et al., 2002), and between 13 and 157 $\mu g/m^3$ including all buildings. The value used by CSOIL of 52.5 $\mu g/m^3$ and the value mentioned by Hawley of 65 $\mu g/m^3$ (Hawley, 1985) do not comprise the highest PM_{10} levels found in the recent studies. However, the high PM_{10} values of Janssen et al. (81-157 $\mu g/m^3$ in classrooms) were 7-8 hour average values for hours during school time, whereas 24 hour average PM_{10} concentrations were a factor two to five lower (46-74 $\mu g/m^3$). This indicates that the value assumed by Hawley is representative for the 24 hour average concentration including all types of building, i.e. including schools with a high resuspension of particulates for several hours per day. The value used by CSOIL (52.5 $\mu g/m^3$) and the value assumed by Hawley (65 $\mu g/m^3$) are probably also representative or somewhat high for the average PM_{10} concentrations in houses and other moderately crowded places, but these values do not represent PM_{10} concentrations in crowded locations such as full classrooms.

Several studies (Janssen et al., 1998a; Janssen et al., 1997; Janssen, 1998b) indicate that personal PM_{10} levels are generally higher than indoor and outdoor PM_{10} levels. As personal samples are probably better representatives for human exposure, these values should be used in risk assessment. In this light, the values assumed by Hawley (65 $\mu g/m^3$) and used in CSOIL (52.5 $\mu g/m^3$) are slightly underestimating the actual exposure situation. A value of 60 $\mu g/m^3$ is probably representative for moderately crowded places such as residents, whereas a value of 100 $\mu g/m^3$ should be used as personal exposure for crowded places such as classrooms.

Table 3: Overview of the concentrations of suspended particulate matter (μg/m³) in air.

		No. persons	No. Obser-vations	Mean (±SD) $PM_{2.5}$* conc (μg/m³)	Range $PM_{2.5}$* conc (μg/m³)	No. Observations	Mean (±SD) PM_{10}* conc (μg/m³)	Range PM_{10}* conc (μg/m³)
Whitby 1957**	Indoor						65	
Whitby 1957**	Outdoor						93	
Janssen 1998a[Δ]	Personal – adult	37				262	61.7 (± 18.3)	38.0-112.8
Janssen 1998a[Δ]	Outdoor – adult	37				285	41.5 (± 4.3)	31.9-50.2
Janssen 1998a[Δ]	Indoor – adult	36				247	35.0 (± 9.4)	18.6-65.3
Janssen 1997[Δ]	Personal – child	45				301	105.2 (± 28.7)[#]	56.9-195.4
Janssen 1997[Δ]	Ambient – child	45				301	38.5 (± 5.6)[#]	24.5-55.8
Janssen 1997[Δ]	Classroom – child					15 (school 1)[‡]	157.0 (± 38.8)	96.2-234.1
Janssen 1997[Δ]	Classroom – child					15 (school 2)[‡]	80.8 (± 18.7)	57.1-127.0
Janssen 1997[Δ]	Classroom – child					11 (school 3)[‡]	134.1 (± 42.1)	66.3-198.6
Janssen thesis 1998b[Δ]	Personal – child	13	77	28.3 ± 11.3	18.7-60.0			
Janssen thesis 1998b[Δ]	Ambient – child	13	77	17.1 ± 2.8	13.6-21.8			
Janssen thesis 1998b[Δ]	Classroom – child	12[‡]		19.9 ± 5.8	14.1-35.2			
Roorda-Knape 1998[Δ]	Outdoor-Delft		10	20.1 ± 4.5[◊]		10	32.2 ± 6.6[◊]	
Roorda-Knape 1998[Δ]	Outdoor-Overschie		8	20.8 ± 6.0[◊]		8	32.1 ± 6.1[◊]	
Sally Liu 2003	Personal	28	183	9.3 ± 8.4	0.8-96.2			
Sally Liu 2003	Indoor	28	193	7.4 ± 4.8	0.4-38.0	206	12.6 ± 10.6	0.6-62.2
Sally Liu 2003	Outdoor	28	194	9.0 ± 4.6	0.7-24.5	200	14.5 ± 7.0	2.9-54.9
Beamer 2002	Daycare, child					2	58.1	47.3-68.9
Beamer 2002	Daycare, adult					2	53.9	42.6-65.1
Stridh 2002	Daycare					9	41 ± 14	
Stridh 2002	Office					18	16 ± 5	
Stridh 2002	School					12	20 ± 10	
Mennen 2001[Δ]	Outside, background						30-50	
Range all studies				***7.4-28.3***	***0.4-96.2***		***12.6-157***	***0.6-234.1***

* $PM_{2.5}$ and PM_{10} refer to the particulate matter concentration with a cutoff diameter of 2.5 μm and 10 μm, respectively.

** Values and references adopted from Hawley et al. (Hawley, 1985). Hawley assumes that the concentration of suspended dust inside homes is 75% of that in the outside air (Hawley, 1985).

[#] Average of 4 schools

[‡] 7-8 hour average, the 24 hour average was a factor two to five lower than the 7-8 hour average.

[Δ] Dutch situation in 1990s.

[◊] Mean concentration at 15 m from the roadside of a major motorway, no gradient was found for PM2.5 and PM10 with distance.

2.5 Conclusions

In conclusion:
The amount of dust that settles per day is probably relatively constant, with values of 0.002 to 0.009 $g \times m^{-2} \times day^{-1}$ (= 2-9 $mg \times m^{-2} \times day^{-1}$). The amount of dust that is present on different surfaces in a household is highly variable and depends, among others, on the extent of cleaning. In addition, different sampling methods may lead to highly variable results. The highest dust loadings were 6.5 g/m^2 for floors (hard surfaces), 11.5 g/m^2 for window sills, and 8.4 g/m^2 for carpets. However, most other studies found lower values for dust loadings of < 1 g/m^2 for hard surfaces, which is in agreement with the value for dust covering of surfaces assumed by Hawley of 0.56 g/m^2 (Hawley, 1985).
Based on several studies it was concluded that soil contributes to 30-70% to house dust. The concentration of particulate matter in indoor air of 52.5 $\mu g/m^3$ that is used in the exposure model CSOIL is a reasonable value for inside houses and other moderately crowded places. However, data collected with personal samples, i.e. samples taken by a device worn by the subject, are probably more representative for human exposure. Particulate matter concentrations were higher when measured near the subject or on lower heights (children), than in bulk indoor air. Mean PM_{10} levels for personal samples are slightly higher (60 $\mu g/m^3$) for moderately crowded places, or considerably higher (100 $\mu g/m^3$) for very crowded places such as classrooms. These latter values are recommended in CSOIL for future use and for location specific risk assessment.

3. Lead

The purpose of this chapter is to study the contribution of house dust to Pb exposure in children. To that end, first an overview of the origin of Pb in house dust is given (§ 3.1), and of the Pb levels in house dust (§ 3.2). In § 3.3 the issue is addressed whether a relationship exists between Pb concentration in house dust and exterior soil. Levels of Pb in particulate matter in indoor air are presented in § 3.4. The approach on the exposure of Pb via house dust in risk assessment in Canada and the USA is addressed in § 3.5. In § 3.6 the contribution of Pb in house dust, outdoor soil, and air to Pb exposure in children is addressed qualitatively and quantitatively as well as possible. Recommendations for the contribution of house dust in risk assessment of Pb for the Netherlands are given in § 3.7. Finally, conclusions are summarised in § 3.8.

3.1 Origin of Pb in house dust

A number of sources contribute to Pb in house dust. Analysis of house dust by Hunt et al. suggests that paint, road dust, and garden soil may all be important Pb sources (Hunt et al., 1992). Pb may also originate from employees carrying home dust in their clothes. Enrichment factors ranging from 3 to 26 have been reported in the Pb concentration in the house dust of workers' homes compared with controls (Fergusson et al., 1991). In addition, the rates of dust metal loadings were significantly higher among the dwellings where someone smoked in the room compared to those dwellings where nobody smoked (Meyer et al., 1999), indicating that tobacco products can also be a Pb source. Fergusson and Kim mention in their review on sources and speciation of Pb in dust that factors affecting Pb concentrations in house dust are: the area of exposed soil, type of work, house decoration, distance from commercial garages, distance from roads, distance from smelters/mining, dustfall inside, dustiness, existence of a fireplace, house age, house material, old paint, road type, soil and street dust (Fergusson et al., 1991).

3.2 Pb levels in house dust

Two different methods of expressing the concentration of Pb in house dust are in use. Pb in house dust can be expressed as:
1) amount of Pb per amount of dust, i.e. *Pb concentration*, or
2) amount of Pb per surface area on which dust was collected, i.e. *Pb loading*.

An overview of studies in which the Pb concentration in house dust (mg/kg) was determined is presented in Table 4. Table 5 presents an overview of reported Pb loadings (mg/m^2). These tables do not give a complete overview of all studies that have been performed on Pb levels in house dust, but are intended to give a representative picture of the Pb concentration and Pb loading in house dust.

Most studies were conducted when the concentration of Pb in motor vehicle emissions was higher. It is therefore likely that current dust Pb levels are lower than those observed in many of the studies presented in Table 4 and 5.

*Table 4: Overview of the **Pb concentration** (mg/kg) in house dust determined in various studies.*

			Pb concentration in house dust (mg/kg)				
Study	***Location***	***N***	***Geometric mean***	***Arithmetic mean***	***Median***	***Range***	***Other information***
Tong 1998	USA, Cincinnati	37		377 ± 705	138		75%: 296
Thornton 1990	UK, London	683	1010			5-36900	
Thornton 1985	UK	3953	507			13-34530	
Rasmussen 2001	Canada, Ottawa	48	233	406	222	50-3226	90%: 969 95%: 1312
Meyer 1999	Germany, Smelter town	454	128		125	0.9-1947	95% CI: 119-138
Laxen 1987	UK, Edinburgh	495		503	308	48-13600	
Davies 1990	UK, Birmingham	97 94 92	311 (playroom) 424 (bedroom) 336 (householder's own vacuum cleaner)				5% 105; 95% 1030 5% 138; 95% 2093 5% 97; 95% 1440
Gulson 1995	Australia, Broken Hill	8		1540		40-4490	From vacuum cleaner dust
Gulson 1995	Australia, Sydney	9		1150		460-2950	From vacuum cleaner dust
Range all studies			***128-1010***	***377-1540***	***125-308***	***0.9-34530***	

*Table 5: Overview of **Pb loading** (mg/m^2) of surfaces determined in various studies.*

			Pb loading of surfaces (mg/m^2)			
Study	***N***	***Surface (sampling method)***	***Geometric mean***	***Arithmetic mean***	***Median***	***Information about distribution*****
Succop 2001	2561 377 2140	Floor (wipe) Window sill (wipe) Window throught (wipe)	0.151 1.1 10.8		0.15 0.94 8.6	95%: 0.76 95%: 10.2 95%: 313
Rhoads 1999*	42 and 42 39 and 40 27 and 28	Floor (wipe) Sill (wipe) Floor (vacuum)	0.24 and 0.28 0.81 and 0.66 5.1 and 5.0			In many houses flaking paint was observed. Many participating families had no vacuum cleaner.
Rich 1999	184 78	Floor (wipe method 1 and 2) Window sills (wipe method 1 and 2)	0.45 and 0.78 2.6 and 6.5			
Lanphear 1996	188 197 179 198 190	Overall surfaces (wipe) Noncarpeted floors (wipe) Carpeted floors (wipe) Window sill (wipe) Window throught (wipe)	1.1 0.17 0.12 1.8 30			± 2 SD: 0.11, 13 ± 2 SD: 0.022, 1.5 ± 2 SD: 0.022, 0.81 ± 2 SD: 0.13, 24 ± 2 SD: 0.31, 2.8×10^3
Sterling 1999	21	Floor living room (wipe and vacuum, see next column)	0.067 (wipe) 0.011 (MVM vacuum) 0.32 (GS80 vacuum) 0.95 (HVS3 vacuum)			SD 0.037 SD 0.066 SD 0.16 SD 0.054
Davies 1990	93	Floor living room and bedroom (vacuum)	0.06			5% 4 95% 486
Rabinowitz 1988	202	Floor living room, furniture top, and window sill (wipe)		0.083		
Laxen 1988	507	Floor (vacuum)			0.045	10% 0.010 90% 0.28 highest: 16152
Brunekreef 1985	43 62 11 50	Floor, Rotterdam inner city (vacuum) Floor, Rotterdam suburb (vacuum) Floor, The Hague inner city (vacuum) Floor, Zoetermeer suburb (vacuum)	0.081 0.030 0.058 0.032			Range: 0.005-0.740 Range: 0.001-0.410 Range: 0.022-0.166 Range: 0.003-0.201
Range all studies			***0.01-30***			

* Two values: one of the Pb intervention group and one control group. Pb intervention did not interact with dust loading.
** SD refers to standard deviation

3.2.1 Pb concentration

As can be seen from Table 4, the geometric mean of the Pb concentration in dust varies between 128 and 1010 mg/kg. The variation is considerable, but can easily be explained by factors such as the large variation in Pb sources, the Pb emission from the different Pb sources, the location of Pb sources, cleaning habits etc. This is also apparent from the variation in Pb levels between houses observed in the same study. For example, Laxen et al. measured in a particular house 13.7 g Pb per kg dust, whereas the median Pb concentration was 327 mg/kg (Laxen et al., 1988). Most extreme cases of Pb in house dust can be ascribed to Pb from Pb paint after redecoration of painted surfaces of the house (Laxen et al., 1988; Gulson et al., 1995). The effect of redecoration is short lived, levels returning to normal within 2 months of redecoration ending (Laxen et al., 1988).

A review by Fergusson lists Pb concentrations in house dust in the 1980s (Fergusson et al., 1991). In agreement with the values in Table 4, the Pb concentration in house dust showed a wide range of individual concentrations, but a reasonably constant mean or median concentration was between 300-700 mg/kg.

3.2.2 Pb loading

Table 5 shows that the Pb loading using a wipe method on the floor varies between 0.067 and 0.78 mg/m^2. The variation is similar to the variation determined in the Pb concentration in house dust (Table 4), approximately a factor 10.
Meyer et al. estimated that the geometric mean of the *surface loading rate* was 1.1 μg Pb×m^{-2}×day^{-1} (range 0.01-22.6 μg×m^{-2}×day^{-1}) (Meyer et al., 1999). The mean Pb loading rate in urban Sydney (Australia) houses in a background area was 2.7 μg×m^{-2}×day^{-1} (range 0.4-5.9 μg×m^{-2}×day^{-1}) in background houses, and 3.3 μg×m^{-2}×day^{-1} (range 1.3-7.7 μg×m^{-2}×day^{-1}) for contaminated houses, i.e. houses of children that were identified with elevated blood Pb (Gulson et al., 1995). Brunekreef found similar values for the surface loading rate of Pb in inner cities and suburbs in the Netherlands, with a geometric mean of 0.99-4.32 μg×m^{-2}×day^{-1}, see Table 6. The surface loading rate is a factor 16-788 lower than the geometric mean Pb loadings determined on floors, which can be explained by incomplete removal of dust and Pb during cleaning activities (Rhoads et al., 1999), and the accumulation of Pb in dust over several days. The effect of cleaning is demonstrated by Rhoads et al., who found a reduction of a factor 1.5-3.1 in Pb loading after cleaning (Rhoads et al., 1999).

According to Table 5, the Pb loading of window sills is considerably higher than the Pb loading of floors, varying between 0.66 and 6.5 mg/m^2 in the studies reported in Table 5, whereas the Pb loading of window throughts is even higher, between 10.8 and 30 mg/m^2. More dust accumulates on window sills and in window throught than on floors (see Table 1), leading to higher Pb loadings.
Using a vacuum cleaner on floors, Pb loadings between 0.011 and 5.1 mg/m^2 were determined. The large variation can be ascribed to the dust collection efficiencies of different vacuum cleaners (Sterling et al., 1999).

Because of the larger variation in Pb loading than in Pb concentration, probably caused by the large variation in dust collection efficiencies, Pb concentration will be used for risk assessment hereafter.

Table 6. Indoor lead surface loading rate ($\mu g \times m^{-2} \times day^{-1}$)

Study	Area	Geometric mean ($\mu g \times m^{-2} \times day^{-1}$)	range	N
Brunekreef 1985[▽]	Rotterdam the Netherlands, inner city	2.86	0.10-20.86	48
	Rotterdam the Netherlands, suburb	0.99	0.10-8.40	67
	The Hague the Netherlands, inner city	4.32	1.95-27.05	13
	Zoetermeer the Netherlands, suburb	1.51	0.48-4.40	49
Meyer 1999*	Hattstedt, Germany	1.1	0.01-22.6	454
Gulson 1995	Sydney, Australia, backround area	2.7	0.4-5.9	8
	Broken Hill, Australia, contaminated area	3.3	1.3-7.7	9

[▽] A major source of Pb in the study by Brunekreef was probably traffic. At present, the release of Pb from traffic is considerable less than in 1985.
* Mining and smelting area.

3.3 Is there a relationship between the concentration of Pb in house dust and Pb in exterior soil?

Table 7 shows the concentration of Pb in house dust and in soil determined at the same site. In general, the concentration of Pb appears to be higher in house dust relative to exterior soil. The dust/soil ratio for Pb ranges from 0.3 to 9.2 in studies conducted both in Europe and North America, see Table 7. Mining and smelting areas were more likely to have elevated concentrations of contaminants in soil compared with the concentration in dusts (Paustenbach et al., 1997). However, in mining and smelting areas, the concentrations of Pb found in house dust were usually much higher than the levels found in homes from non-source dominated areas.

Table 7: Survey of published values that present the ratio of the concentrations of Pb in house dust compared to the concentration in exterior soil.

Location	***Dust (mg/kg)***	***Soil (mg/kg)***	***Ratio***	***Source***
Australia (Broken Hill)	1540	1630	0.9	Gulson et al., 1995
UK	507	230	2.2	Thornton et al., 1985
UK	561	289	1.9	Thornton et al., 1990
UK	1263	486	2.6	Culbard et al., 1983*
UK (London)	1010	430	2.3	Thornton et al., 1990
UK (Derbyshire)	1870	4390	0.4	Thornton et al., 1990
UK (Shipham)	1185	3829	0.3	Thornton et al., 1990
UK (Birmingham)	311	313	1.0	Davies et al., 1990
Netherlands	957	240	3.9	Diemel et al., 1981*
Canada	713	99	7.2	Roberts et al., 1974*
Canada	1550	1715	0.9	Roberts et al., 1974*
Canada	233	42	5.5	Rasmussen et al., 2001
Spain	595	136	4.4	Cambra and Alonso, 1995*
USA (CT)	11000	1200	9.2	Lepow et al., 1975*
USA (IL)	1283	450	2.9	Kimbrough et al., 1995*
USA (MA)	1094	707	1.5	US EPA, 1995*
USA (MD)	1334	231	5.8	US EPA, 1995*
USA (MO)	608	599	1.0	MDH, 1995*
USA (IL)	540	388	1.4	Berny et al., 1994
Average (± SD)			***2.9 (± 2.5)***	

*Values and references adopted from Paustenbach et al. (Paustenbach et al., 1997).

Figure 1 graphically shows the Pb concentration in soil against the Pb concentration in house dust for studies listed in Table 7. Most data points are above the y=x line, indicating that in most cases the Pb concentration is higher in house dust than in soil.

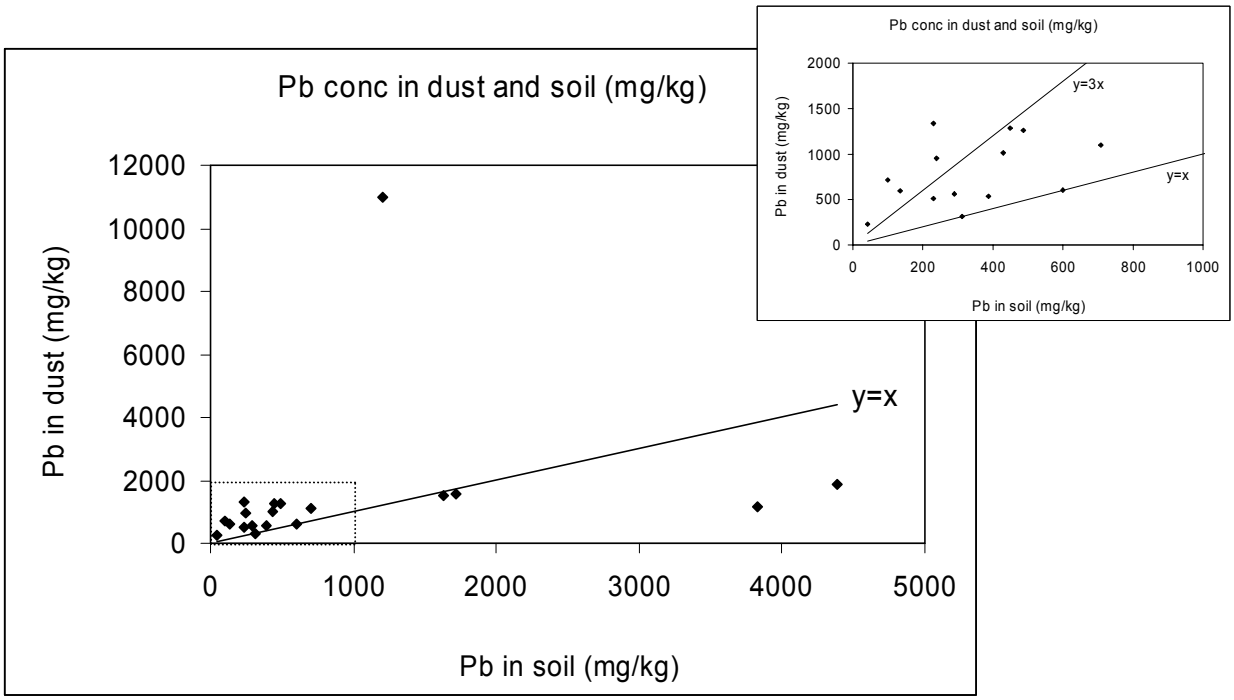

Figure 1. Pb concentration in soil versus Pb concentration in house dust. More data points are above the y=x line than below, indicating that in most cases the Pb concentration is higher in house dust than in soil.

Concentrations of Pb which are higher in house dust compared with the concentration in outside sources suggests that there is an internal source, but selective enrichment of finer particles may also occur (Fergusson et al., 1991). Up to 30% of particles in house dust can be < 5 µm, and most vacuums do not trap such dust, resulting in recontamination of the house (Gulson et al., 1995).

Several studies have explicitly examined whether there is a relationship between Pb concentration indoors and outdoors. In a study by Tong no relationship was found between the house dust Pb concentrations indoors and outdoor dust samples collected from paved hard surfaces on or around the doorsteps, porch, or patio (Tong, 1998). It should be noticed that in this study outdoor dust samples were collected, which does not necessarily give the same relationship as Pb in soil.
Rasmussen et al. compared Pb in house dust, garden soil and street dust on the 100-250-µm particle size fraction in Ottawa, Canada (Rasmussen et al., 2001). They found higher levels of Pb in house dust (geometric mean 233 mg/kg) than in garden soil (42 mg/kg) and street dust (33 mg/kg). Indoor dust concentrations could not be extrapolated based on exterior soil data. It was concluded that dust generated from sources within the house itself contributed significantly to exposure to certain elements including Pb (Rasmussen et al., 2001). This can be explained by the fact that Ottawa is a city with a low concentration of heavy industries, resulting in a relatively low contribution of Pb from soil to house dust compared to other possible sources.

In contrast to the studies by Tong and Rasmussen et al. (Rasmussen et al., 2001; Tong, 1998), Berny et al. found that soil and dust Pb concentrations were significantly related to one another, with a r^2-value of 0.52 and n=77 (Berny et al., 1994). Pb ranged from 100 to 25,000 mg/kg in dust, with a median value of 388 mg/kg. Pb in soil ranged from <100 to 4400 mg/kg, with a median value of 540 mg/kg. A secondary Pb smelter had been in operation in this town for almost 80 years and was shut down in 1982, about ten years before the research by Berny et al. (Berny et al., 1994). Hence, Pb in soil was a source that was relatively high compared to other sources, resulting in a positive relationship between soil and dust Pb concentration.

The studies mentioned above suggest that a relationship between soil and dust Pb concentration can exist. Such a relationship can be expected when considering that 30-70% of the house dust originates from soil. Obviously, a relationship between soil and dust Pb can only be found if Pb in soil dominates over the contribution of Pb from other sources. If other sources of Pb are dominant, the relationship between soil and dust Pb is obscured.
The arguments discussed above are in agreement with a study by Gulson et al. (Gulson et al., 1995). They characterised Pb in house dust in Broken Hill, Australia, which is the site of the world's largest lead-zinc-silver mine, and in Sydney, where Pb is mainly derived from gasoline and other point sources such as battery factories and secondary Pb industry. Sources of Pb were discriminated by isotope ratios ($^{206}Pb/^{204}Pb$). The major source of Pb in vacuum and surface dust in Broken Hill was from the orebody (in most cases considerable more than 70%). However, there were a few houses in which the Pb came from other sources, such as gasoline or leaded-paint. As a consequence, a strong correlation (r=0.95) was obtained between the isotopic composition of Pb in blood and dust-fall accumulation.
In contrast to the majority of houses in Broken Hill, the Pb isotope ratios for different fractions from inner Sydney vacuum dust exhibited considerable variations, meaning that the Pb in the dusts came from different sources.

3.4 Pb in air

Levels of Pb in particulate matter in indoor and outdoor air are gathered and listed in Table 8. Gallacher et al. conducted Pb air measurements in four areas in Wales with different degrees of environmental Pb (Gallacher et al., 1984). In two areas the source of the Pb was traffic and in one it was spoil from Pb mining in the past. The fourth area, which served as a control, was a village remote from heavy traffic, industry, and Pb mining. The geometric mean of the indoor air concentration in roadside dwellings was 0.18 $\mu g/m^3$, in cul-de-sac dwellings (NL: doodlopende straat) was 0.12 $\mu g/m^3$, and in dwellings in an old mining area 0.068 $\mu g/m^3$. In dwellings in the control village the geometric mean Pb air concentration was 0.039 $\mu g/m^3$. The highest indoor Pb air concentrations were found in the area with the most traffic.
Davies et al. conducted an extensive study on the various pathways of Pb intake (Davies et al., 1990). To that end, the indoor air concentration was measured. The geometric mean air Pb concentration in a child's bedroom was 0.26 $\mu g/m^3$, see Table 8. This value was very similar to that in the playroom of 0.27 $\mu g/m^3$. The mean external air Pb concentration was 0.43 $\mu g/m^3$, and was significantly higher than those found indoors ($p<0.001$). Results for the daily air Pb concentration in individual houses showed that the internal concentrations closely followed the external, but were usually lower, with a mean external:internal air Pb ratio of 1.6 (Davies et al., 1990).

Rabinowitz measured an indoor Pb air concentration of 0.13 μg/m^3 (Rabinowitz et al., 1980). Further details were not reported. Janssen found values ranging between 0.018 and 0.071 for indoor air in schools, including both measurements during school hours only and 24 hour average values (Janssen, 1998b). The 24 hour average value for outdoor air was 0.026 (Janssen, 1998b), indicating that the external:internal Pb air ratio of 1.6 of Davies et al. (Davies et al., 1990) did not apply to these data. It should be noted that indoor air levels of Pb in schools may be higher than in residents due to the high resuspension rate, see section 2.4.

Table 8. Overview of Pb air concentrations.

				Pb air concentration (μg/m^3)	
Study	***N***	***In/outdoor***	***Location***	***Geometric mean***	***Other information****
Gallacher et al. 1984	42 30 62 33	Indoor Indoor Indoor Indoor	Wales (UK), roadside Wales (UK), cul-de-sac Wales (UK), old mining area Wales (UK), control village	0.18 0.12 0.068 0.039	95% CI: 0.05-0.61 95% CI: 0.029-0.37 95% CI: 0.039-0.21 95% CI: 0.010-0.13
Rabinowitz et al., 1988	N/a	Indoor	Boston, USA		Arithmetic mean: 0.13
Davies et al., 1990	599 607 605	Indoor Indoor Outdoor	Birmingham (UK), bedroom Birmingham (UK), playroom Birmingham (UK)	0.26 0.27 0.43	5% 0.09; 95% 0.81 5% 0.08; 95% 0.88 5% 0.12; 95% 1.53
Gulson et al., 1995	N/a	Outdoor	Sydney, Australia		Highest monthly average usually less than 0.5 Usual range 0.1-1.0
Janssen thesis 1998b		School 1 School 1 School 1 School 2** School 2** School 2** Outdoor	The Netherlands 1995	0.043 0.031 0.018 0.071 0.061 0.049 0.026	School hours 24 hour average non-school hours School hours 24 hour average non-school hours 24 hour average
Janus et al., 1999	N/a	Outdoor Outdoor Outdoor Outdoor	The Netherlands 1985 The Netherlands 1989 The Netherlands 1992 The Netherlands 1995		Year average: 0.12 Year average: 0.08 Year average: 0.037 Year average: 0.024
Van Breugel et al., 2001	N/a	Outdoor	The Netherlands 1999		Year average 0.012

N/a: not available.
* CI refers to Confidence interval.
** School 2 had just been repainted. High lead levels were possibly caused by removal of old lead-based paint.

It should be noted that the study by Gallacher et al., Davies et al., and Rabinowitz et al. were performed in the 1980-ies (Gallacher et al., 1984; Davies et al., 1990; Rabinowitz et al., 1980). Present levels of Pb in air will be lower due to a decrease of Pb in emission by traffic. The decrease of Pb in air due to decreasing Pb emission by traffic is demonstrated by Janus et al. (Janus et al., 1999) and van Breugel et al. (Van Breugel et al., 2001), who provided an overview of the measured average Pb concentration in outdoor air in the Netherlands for several years. The Pb concentration decreased from 0.12 in 1985 to 0.024 μg/m^3 in 1995, and to 0.012 μg/m^3 in 1999, see Table 8 (Janus et al., 1999; Van Breugel et al., 2001). In absence of specific sources of Pb indoors, Janus et al. assume the Pb concentration in indoor air is a factor 2.5-3 lower than in outdoor air (Janus et al., 1999). Davies et al. also found a lower Pb

air level indoors than outdoors, but the external:internal air Pb ratio was lower (1.6) than the factor by Janus et al. (2.5-3) (Davies et al., 1990; Janus et al., 1999).
According to Gulson et al., the monthly Pb contents in Sydney total suspended particulates in outdoor air varied from 0.1 to 1.0 μg/m^3, which is not markedly higher than the average of between 0.1 and 0.3 μg Pb/m^3 in most US cities without Pb point sources (references in (Gulson et al., 1995)).

Although only few references were found on Pb in indoor air, it can be concluded that Pb concentration in indoor air *at present in the Netherlands*, in absence of high external Pb sources, is probably less than 0.1 μg/m^3. In most cases, outdoor Pb air levels exceeded indoor levels.

A comparison can be made between the values found for the Pb concentration in air (0.1 μg/m^3, and values in Table 8) versus a hypothetical calculation of the concentration of Pb in air, assuming that Pb in soil is the only Pb source to Pb in particulate matter in air. The calculation can be performed using assumptions for 1) the concentration of dust in air, 2) the contribution of soil to suspended dust, and 3) the Pb concentration in soil. In agreement with the recommendations of §2.4, a concentration of dust in air of 60 μg dust/m^3 is assumed. Assuming that 30-70% of the suspended dust in air originates from soil (this value is assumed for the contribution of soil in settled dust, see § 2.3), and that Pb in air originates from soil only, a value of 18-42 μg soil/m^3 in air can be calculated. Using a contamination level of Pb in soil of 85 mg/kg (Target Value), the concentration of Pb in air amounts 1.5-3.6 ng/m^3. This hypothetically calculated value is about a factor 28-67 smaller than the upper values measured in the field (100 ng Pb/m^3), but close to the year average of Pb in outdoor air in 1999 (12 ng/m^3), see Table 8. Hence, it can be concluded that the hypothetically calculated concentration of Pb in air is in the same order of magnitude as experimentally found levels. This agreement supports the assumptions that 1) 30-70% of suspended dust in air originates from soil, 2) the concentration of dust in air is approximately 60 μg/m^3.

3.5 Exposure to Pb via house dust in risk assessment in Canada and USA

3.5.1 Canada

Health and Welfare Canada (HWC, 1992) assumes an ingestion rate of 80 mg dust/day for a 2-year-old child (Rasmussen et al., 2001), and 30% absorption of Pb. According to Mielke and Reagan, hand-to-mouth behaviour results rarely in the ingestion of quantities of dust that exceeds 200 mg per day (Mielke et al., 1998). No references or argumentation was given for this value. Further information about the amounts of dust ingested by children was not found.

3.5.2 USA

In the USA, the Integrated Exposure Uptake Biokinetic (IEUBK) Model for lead in children is used to predict the risk of elevated blood lead (PbB) levels in children (under the age of seven) that are exposed to environmental Pb from many sources (US-EPA, 2002). Media that can act as sources of Pb for a child include air (both indoor and outdoor), which enters the body through the lungs, and water, soil, dust (indoor), diet, and other sources (e.g. lead paint),

which enter the body through the gastrointestinal tract. The default media intake rates used in the IEUBK model are presented in Table 9.

Table 9. Media intake rates of the US-IEUBK model for children between 0 and 7 years of age. The IEUBK model stands for Integrated Exposure Uptake Biokinetic model, and is used to predict the risk of elevated blood lead levels in children that are exposed to environmental Pb from many sources.

Age	Soil/dust (g/d)	Air (m^3/d)	Drinking water (L/d)	Diet (µg Pb/d)
0-1 yr	0.085	2	0.2	5.53
1-2 yrs	0.135	3	0.5	5.78
2-3 yrs	0.135	5	0.52	6.49
3-4 yrs	0.135	5	0.53	6.24
4-5 yrs	0.100	5	0.55	6.01
5-6 yrs	0.090	7	0.58	6.34
6-7 yrs	0.085	7	0.59	7.00

The US-EPA recommends default values for intake rates in the IEUBK model, unless adequate, site-specific monitoring data exist to define values that are higher or lower in magnitude (US-EPA, 2002). The default intake value for total soil and dust ingestion is a ratio of soil ingestion (45%) to dust ingestion (55%).
The soil/dust intake values are considered representative of average daily intake rates. The model assumes that 50% of the Pb intake from drinking water and food is absorbed in the bloodstream and that 30% of the Pb intake from soil and dust is absorbed.

3.6 Contribution of Pb in house dust, outdoor soil, and air to Pb exposure in children

3.6.1 Qualitative

Numerous studies have shown that dust Pb is an important source of exposure for young children (Sayre 1974, Bornscheid 1985, Davies 1985, Laxen 1987, Davies 1990, Thornton 1990, Al-Radady 1994, Lanphear 1996, 1998b). Several studies that support this statement are described below:

Studies

Rhoads et al. showed that after a year of thorough household cleaning house dust Pb levels decreased significantly and blood Pb by 17% for children between the ages of 6 and 36 months (Rhoads et al., 1999). Pb mainly originated from Pb paint. The blood Pb concentration was 12.4 µg/dl at start of study (n=46), versus 10.3 µg/dl after follow up. The blood Pb concentration in the control group stayed the same, 11.6 µg/dl at start and end of the experiment (n=53) (Rhoads et al., 1999).

Lanphear et al. pooled 12 epidemiological studies in multiple communities (1300 observations) for children between 6 and 36 months of age (Lanphear et al., 1998b). Analysis confirms that Pb-contaminated house dust is the major source of Pb exposure to children. There is a strong relationship between interior dust Pb loading and children's blood Pb levels. In addition, the analyses demonstrate that a child's age, race, mouthing behaviours,

and study-site specific factors influence the predicted blood Pb level at a given level of exposure. The r^2-value for this model was 0.53. The contribution of Pb-contaminated floor dust to children's blood Pb level was greater than the contribution from Pb-contaminated soil (Lanphear et al., 1998b).

In another study by Lanphear et al, an analysis was undertaken to estimate the relationship of environmental Pb exposures to Pb intake of urban children (Lanphear et al., 1998a). Environmental sources of Pb, including house dust, soil Pb, and water Pb, were independently associated with children's blood Pb levels. In contrast, paint Pb levels did not have a significant effect on blood Pb levels after adjusting for other environmental exposures. An increase in water Pb concentration from background levels to 0.015 mg/l, the EPA water Pb standard, was associated with an increase of 13.7% in the percentage of children estimated to have a blood Pb concentration exceeding 10 μg/dl; increasing soil Pb concentration from background to 400 μg/g was estimated to produce an increase of 11.6% in the percentage of children exceeding 10 μg/dl; and increasing dust Pb loading from background to 2.2 mg/m^2 is estimated to produce an increase of 23.3% in the percentage of children estimated to have a blood Pb level exceeding 10 μg/dl (Lanphear et al., 1998a). Pb blood levels exceeding 10 μg/dl have a negative impact on the mental development of children (Janus et al., 1999).

Laxen et al. showed a significant relationship between Pb in dust vacuumed from the floors of the children's homes and their blood Pb levels in a study of 495 children in Edinburgh, Scotland (Laxen et al., 1987). A multiple regression analysis incorporating drinking water and house dust estimates that a 1000 mg/kg increase in dust Pb concentration would increase blood Pb by 1.9 μg/dl, for a child with the median population blood Pb of 10.1 μg/dl (Laxen et al., 1987).

Gallicchio et al. studied 205 children of approximately 1 year of age and living in old, urban houses (Gallicchio et al., 2002). They found a statistically significant positive association between blood Pb and window sill dust Pb levels ($p<0.0001$) (Gallicchio et al., 2002).

Rabinowitz studied the exposure pathways of Pb to 202 children (Rabinowitz et al., 1988). Surface wipes (living room floor, furniture top, and window sill), indoor aerosol samples, paint, soil samples, and drinking water were collected. The mean level of dust Pb on surfaces was 83 μg/m^2, in tap water 5.0 μg/l, and 0.13 μg/m^3 in air. There were strong correlations between blood Pb levels and soil Pb and dust Pb levels, but not between blood Pb and air or water Pb levels. Soil and floor dust Pb levels were strongly correlated ($r=0.43$, $p<0.0001$). Those children judged to mouth the most had 2 to 3 times the Pb blood level of those who mouth the least, at given soil Pb level (Rabinowitz et al., 1988).

Gulson et al. found a strong correlation ($r=0.95$) between the isotopic composition of Pb in blood and dust-fall accumulation in Broken Hill (Gulson et al., 1995). This suggests that almost all Pb in blood originated from Pb in house dust.

In contrast, in a study by Berny et al. no significant relationship between soil or dust Pb and blood Pb concentration in humans was observed (Berny et al., 1994). However, the relationship was significant in pets. Blood Pb levels in humans varied between 0 and 13 μg/dl, whereas Pb levels in blood of pets ranged between 0 and 28 μg/dl (Berny et al., 1994).

Discussion
The studies addressed above indicate that there is ample evidence suggesting that house dust is a major source of exposure to Pb. Many studies suggest that exposure to house dust can be more important than exposure to contaminated exterior soils. Several reasons can be brought up for this. First, most people spend most of their time indoors. Second, house dust consists of finer particles than soil and these fine particles are more mobile, adhere better to the skin or clothing, and are more easily ingested or respired, and possibly more bioavailable. Third, contaminant levels may be higher in house dust than in the associated soils and may be higher in the finer, more available fraction of dust. Fourth, the indoor environment protects residues from the degradation, ageing, or dispersion that serves to reduce the levels or availability of contaminants outdoors. Finally, the normal activity that occurs in residents causes constant resuspension and settling of contaminants from dust traps, resulting in a higher frequency of contact with contaminated surfaces and a longer duration of exposure to dust-borne contaminants as a result.
However, the argumentation above does not imply that Pb in soil is not relevant. Direct soil ingestion should always be considered, but soil also contributes to a major part (about 30-70%) to house dust, leading to an indirect contribution of Pb in soil to exposure of children.

3.6.2 Quantitative assessment
The exposure to Pb via ingestion of house dust and soil, and via inhalation of Pb in air is calculated according to CSOIL (Otte et al., 2001) with the information available. These pathways of exposure are compared.

Ingestion
In CSOIL, uptake via ingestion is calculated according to equation 1. This equation can be used for house dust and soil.

$$DI = \frac{AID \times C_s \times F_a}{W} \qquad (1)$$

with:
- DI: uptake via ingestion ($mg_{contaminant} \times kg^{-1} \times d^{-1}$)
- AID: daily intake house dust/soil via ingestion ($kg_{dust} \times d^{-1}$)
- W: body weight (kg)
- F_a: relative absorption factor, set at 1 (-)
- C_s: initial dust content ($mg_{contaminant} \times kg_{dust}^{-1}$)

A reasonable mean Pb concentration in dust is 500 mg/kg (C_s=500 mg/kg), see Table 4. Using a daily intake of house dust via ingestion of 80 mg, as assumed by Health and Welfare Canada (see § 3.5; AID=80 mg), a relative absorption factor of 1 (F_a=1), the uptake for a child of 15 kg (W=15) via ingestion is estimated to be:

$$DI = \frac{80 \times 10^{-6} \times 500 \times 1}{15} = 2.7 \times 10^{-3}\, mg_{Pb} \times kg_{bodyweight}^{-1} \times d^{-1} \qquad (2)$$

For soil ingestion the following is assumed. The ingestion rate of soil is 100 mg soil per day (Lijzen et al., 2001)(AID=100 mg/kg). The Pb concentration in soil is assumed to be 530 mg/kg (C_s=530 mg/kg), which is the Intervention Value for Pb in standard soil (Lijzen et al., 2001). When using a relative absorption factor of 1 (F_a=1), the uptake for a child of 15 kg (W=15) via ingestion is estimated to be:

$$DI = \frac{100 \times 10^{-6} \times 530 \times 1}{15} = 3.5 \times 10^{-3}\ mg_{Pb} \times kg^{-1}_{body\ weight} \times d^{-1} \quad (3)$$

Hence, using a daily ingestion of 100 mg soil with a contamination level of 530 mg/kg soil the exposure to a child of 15 kg is estimated to be 3.5×10^{-3} mg×kg^{-1}×day^{-1}. Using a daily ingestion of 80 mg dust with a contamination level of 500 mg/kg dust, the exposure to the child is estimated to be 2.7×10^{-3} mg×kg^{-1}×day^{-1}. This suggests that exposure to Pb via soil and dust is of the same order of magnitude.
Using a Pb concentration in soil of 200 mg/kg, i.e. a factor 3 lower than the Pb concentration in dust (see § 3.3), the uptake via ingestion of soil would be still in the same order of magnitude as the uptake via house dust ingestion (1.3×10^{-3} mg×kg^{-1}×d^{-1}). Therefore, although based on little information, we assume that the contribution of Pb exposure to children via house dust and outdoor soil is roughly the same. Further experimental research on the amount of soil and dust ingestion, and on the contribution of soil and dust to Pb exposure is recommended.

Relative absorption factor F_a
In the present calculation the relative absorption factor (F_a) is set at 1. Oliver et al. determined the bioaccessibility of Pb from house dust (Oliver et al., 1999). The fraction of a contaminant that is mobilised from its matrix, in this case house dust, in the gastrointestinal tract is referred to as the bioaccessible fraction. The fraction contaminant still sorbed to house dust cannot be absorbed in the intestine and, consequently, cannot contribute to internal exposure and toxicity. Bioaccessibility is a prerequisite for bioavailability. A lower bioaccessibility of Pb from house dust than from the matrix used in the studies on which the MTR (Maximum Tolerable Risk) was based, indicates that the relative absorption factor F_a is smaller than 1.
The bioaccessibility of Pb from house dust determined by Oliver ranged from 26% to 46% in the stomach compartment (pH 1.3), and from 5-10% in the intestinal compartment (pH 7.0) (Oliver et al., 1999). The MTR of Pb was based on epidemiological studies in which people ingested Pb via food and water (Rompelberg et al., 2002). The bioaccessibility of Pb from food and water is probably considerable higher than 5-10%, suggesting that the relative absorption factor F_a for Pb in house dust is less than 1. Similar values for F_a can be found for Pb in soil. An exact figure of F_a cannot be given because the bioaccessibility of Pb from food and water has not been studied up till now.

Inhalation
In CSOIL, uptake via inhalation is calculated according to equation 4 and 5:

$$IP = \frac{ITPS \times C_s \times F_r \times F_a}{W} \quad (4)$$

$$ITPS = TPS \times AV \times t \times f_t \quad (5)$$

with:
- IP: Exposure to inhaled contaminant ($mg_{contaminant} \times kg_{body\ weight}^{-1} \times d^{-1}$)
- ITSP: Inhaled amount of dust particles ($kg_{dust} \times d^{-1}$)
- C_s: Concentration contaminant in dust particles ($mg_{contaminant} \times kg_{dust}^{-1}$)
- F_r: retention factor in lungs (-)
- F_a: relative absorption factor (-)
- W: body weight ($kg_{body\ weight}$)

TPS: amount of suspended dust particles in air ($mg_{dust} \times m_{air}^{-3}$)
AV: breathing volume ($m_{air}^{3} \times d^{-1}$)
t: duration of exposure (d)
f_t: time fraction of exposure (-)

In CSOIL it is assumed that the amount of suspended dust particles in indoor air (TPS) amounts 52.5 μg/m^3. According to Table 3 a slightly higher value (60 μg/m^3) is representative for moderately crowded places such as residents (TPS=60×10^{-3} mg×m^{-3}). The breathing volume of a child is 7.6 m^3/d, with a exposure duration of 16 indoors and 8 h outdoors. Hence:

$$\text{ITPS}=60 \times 10^{-3} \times 7.6 \times \frac{16}{24} = 0.304 \text{ mg/d} \qquad (6)$$

Assuming a Pb concentration in dust of 500 mg/kg (Cs), a retention factor in the lungs of 1 (F_r=1), a relative absorption factor of 1 (F_a=1), and a child of 15 kg (W), the exposure to inhaled Pb (IP) is:

$$IP = \frac{0.304 \times 10^{-6} \times 500 \times 1 \times 1}{15} = 1.0 \times 10^{-5} \, mg_{Pb} \times kg_{bodyweight}^{-1} \times d^{-1} \qquad (7)$$

Hence, it can be concluded that exposure to Pb via inhalation is negligible compared to exposure via ingestion of house dust and soil (about a factor 300 less). A low contribution of inhalation to Pb exposure is in accordance with Davies et al., who estimated that 97% of Pb exposure of 2-year-old children in Birmingham was from ingestion from dust, food, and water, and only 3% from inhalation (Davies et al., 1990).

3.7 Recommendations for handling the contribution of house dust in risk assessment in the Netherlands

The US-EPA assumes that children ingest 85-135 mg of soil, depending on the age of the child. This amount of ingested soil is the sum of direct soil ingestion and of ingestion of house dust that consist for a major part (assumption US-EPA: 70%) of soil. This is a simple, straightforward and workable approach. A drawback of the US-EPA model is that it does not account for enrichment of Pb house dust compared to Pb in soil. Also, the IEUBK model assumes that soil is the predominant source of Pb in house dust, which is not necessarily true. Yet, Table 7 and Figure 1 show that in most cases Pb concentration in house dust is higher than in soil. These assumptions may lead to an underestimation of the actual exposure of children to Pb.

A major gap in current exposure assessment is the lack of knowledge on the amount of dust that is ingested by children. The studies in which the amount of soil ingested by children are estimated use tracers in soil that are obviously both present in soil outdoors and soil in house dust. Therefore, using the amount of soil ingested by children to cover both soil outdoors and soil in house dust is reasonable. Hence, a soil ingestion rate of 100 mg/day should be used, which is the same ingestion rate as is used in present risk assessment in the Netherlands (Lijzen et al., 2001). However, in this manner, enrichment of Pb in house dust in comparison

with outdoor soil, and other Pb sources in house dust than contamination via soil are not accounted for.
In the assessment of *soil contamination* you want to incorporate the Pb in house dust caused by soil outdoors in the risk assessment, but not the Pb from other sources. Therefore, in the assessment of soil contamination, enrichment of Pb in house dust should be incorporated, but contribution of Pb from other sources than soil should not. Assuming that all Pb in house dust originates from soil particles in house dust, indicating that the higher Pb levels in house dust are caused by enrichment of Pb in house dust, and assuming approximately equal ingestion of soil and house dust, a correction factor of 2 is recommended, see *scenario 1* in text box below for the derivation of this factor.
However, alternatively, when assuming that Pb from outdoor soil is not enriched in house dust, and that higher Pb levels in house dust than in soil are caused by other sources than outdoor soil, a factor of 1 is recommended, see *scenario 2* in text box below for derivation. In this case, the exposure to Pb is the same as presently assumed in risk assessment of Pb contaminated soils.
The two scenarios describe the two extreme situations that are possible using different assumptions. The two extreme scenarios lead to very different recommendations, and the most likely scenario cannot be deduced based on the current knowledge. The truth probably lies in between both scenarios. A safe approach is to use the factor 2, or measure the Pb levels in soil outdoors and house dust. In the latter case, both experimentally determined concentrations can be averaged, i.e. assuming equal contribution of soil and house dust to the Pb exposure of a child, leading an overall Pb exposure level. This level should be compared to the current Intervention Value of Pb in soil, and if necessary, action should be taken. It should be noted that according to both approaches, both enrichment of Pb in house dust and contribution of Pb from other sources than soil are included. Hence, for the assessment of soil contamination this is a worst case situation. Further research on the contribution of Pb in house dust caused by soil is recommended.

Scenarios to account for exposure to Pb in house dust caused by Pb in outdoor soil

A problem in the assessment of *soil contamination* is that you want to incorporate the exposure to Pb in house dust caused by soil outdoors in the risk assessment, but not the exposure to Pb from other sources. Therefore, in the assessment of soil contamination, enrichment of Pb in house dust should be incorporated, but contribution of Pb from other sources than soil should not.
Below two possible scenarios are calculated to account for the exposure to Pb in house dust caused by Pb from soil.

Scenario 1:
When assuming that all Pb in house dust originates from soil particles in house dust, indicating that the higher Pb levels in house dust are caused by enrichment of Pb in house dust, the following calculation can be made. The average ratio Pb in house dust/Pb in soil is approximately 3, see Table 7. When x is the concentration of Pb in soil, the concentration of Pb in house dust can thus be approximated by 3x. Assuming a similar ingestion rate of soil and house dust, the total exposure of a child to Pb by ingestion is thus:
$0.5 \times (3x + x) = \mathbf{2}x$
Therefore, in this case, a correction factor (for enrichment) of 2 is recommended when assuming a soil ingestion rate of 100 mg/day, covering both direct soil ingestion and soil particles in house dust.

Scenario 2:
Alternatively, it can be assumed that Pb from outdoor soil is not enriched in house dust, and that higher Pb levels in house dust than in soil are caused by other sources than outdoor soil. In that case, the concentration of Pb from outdoor soil to Pb in house dust can be estimated by x, as house dust originating from soil has the same concentration as soil. Assuming again a similar ingestion rate of soil and house dust, the total exposure of a child to Pb by ingestion is: $0.5 \times (x + x) = x$
Therefore, using above mentioned assumptions, a factor of 1 should be used (no enrichment). indicating the same exposure to Pb as in present risk assessment of Pb-contaminated soils.

The two scenarios lead to different recommendations. At present, there is not enough knowledge to point out the true scenario, which is probably a scenario in between scenario 1 and 2, in which part of the lead originates from the dust from other sources than soil. A safe approach is therefore to use the factor 2, or to measure the Pb concentration in house dust (C_{dust}) and average this value with the concentration found in soil outdoors (C_{soil}):
$0.5 \times (C_{soil} + C_{dust})$
The outcome should be treated similar to the present risk assessment of Pb-contaminated soil. It should be noted that according to these approaches, both enrichment of Pb in house dust and contribution of Pb from other sources than soil are included, which is in the assessment of soil contamination a worst case situation.

3.8 Conclusions

In conclusion:
A reasonable mean or median concentration of Pb in house dust is 300-700 mg/kg. The data for Pb loading, i.e. the amount of Pb per surface area, were highly variable due to different sampling methods. The Pb concentration in house dust is generally higher than in exterior soil, with an average factor of approximately 3. Mining and smelting areas were more likely to have elevated Pb concentration in soil compared with the concentration in dust.
Some studies have found a direct relationship between Pb concentration in house dust and in soil, whereas other studies did not find such a relationship. A relationship between Pb in soil and house dust can exist when considering that 30-70% of house dust can originate from exterior soil. Obviously, a relationship between Pb in soil and house dust can only exist when the contribution of Pb from soil dominates over other Pb sources. Furthermore, many experimental and epidemiological studies suggest that house dust is a major source of exposure to Pb.
It is concluded that Pb exposure to children via ingestion of soil and house dust is probably in the same order of magnitude, whereas exposure to Pb via inhalation is negligible (approximately a factor 300 lower). Therefore, exposure to Pb via house dust should be included in risk assessment.
In the assessment of soil contamination, it is aimed for to include Pb in house dust caused by outdoor soil, but not Pb from other sources. For the Dutch situation it is recommended to use a soil ingestion rate of 100 mg/day, as is also presently used, to cover both Pb in soil and in house dust. In addition, a factor should be included to account for a different concentration of Pb in house dust caused by soil, compared to the concentration of Pb in outdoor soil. This factor may range between 1 and 2, depending on the assumptions made. Due to lack of knowledge on the contribution of Pb in house dust caused by soil outside, the factor cannot

be estimated more precise. A safe approach is therefore to use the factor 2, or measure the Pb concentration in house dust and average this value with the concentration found in soil outdoors. It should be noted that according to these approaches, both enrichment of Pb in house dust and contribution of Pb from other sources than soil are included, which is in the assessment of soil contamination a worst case situation. Further research on the contribution of Pb in house dust caused by soil is therefore recommended.

4. Asbestos

4.1 Origin and types of asbestos

Asbestos was used in the construction of public buildings such as schools, theatres, shopping centres, gymnasiums and swimming baths, primarily during the 1950s through early 1970s, because of its acoustical, fire-proofing, and decorative qualities (Landrigan et al., 1987; Slooff et al., 1989). Such dried materials can, over time, become friable. Friable asbestos often flakes off as a fine dust that settles on surfaces but is readily resuspended in air. Asbestos was also used in insulating materials for pipes, boilers, and structural beams in schools (Landrigan et al., 1987).
Since 1993, asbestos may practically not be used in the Netherlands (Brand et al., 1994). It is estimated that approximately 80% of all asbestos was used in construction.
In the Netherlands, emission of asbestos from natural sources, e.g. asbestos containing rock, does not occur. Emission of asbestos to the soil occurs primarily by dumping of asbestos containing waste, and during demolishing activities (Tromp et al., 1996).

Children are of particular concern for asbestos exposure, because, compared with adults, they are more active and they have a longer life expectancy (Landrigan et al., 1987). Children breath at higher rates and more often by mouth, they spend more time close to the floor, where sedimented dust and fibres accumulate. Furthermore, children are more likely to seek direct contact with deteriorating surfaces out of curiosity or mischief. These factors must be considered when potential childhood exposures are estimated.

Asbestos is classified into two groups: serpentine and amphibole. Chrysotile asbestos is the most commonly known type in the serpentine group and it was the most widely used commercially.
The Amphibole group has 5 types of asbestos: Amosite, Crocidolite, Tremolite, Actinolite, and Anthophyllite. The most common Amphibole asbestos types are Amosite (brown asbestos) and Crocidolite (blue asbestos).

The following year-average-allowable-risk-levels for asbestos concentrations in air are defined (Swartjes et al., 2003):

- Maximum Acceptable Risk Level (NL: Maximaal Toelaatbaar Risiconiveau, MTR): 100.000 fibre equivalents per m^3 air.
- Negligible Risk Level (NL: Verwaarloosbaar Risico-niveau: VR): 1.000 fibre equivalents per m^3 air.

The toxicity of different asbestos types is different. Therefore, equivalent factors are derived:

1 chrysotile fibre with a length > 5 μm:	equivalent factor 1
1 chrysotile fibre with a length < 5 μm:	equivalent factor 0.1
1 amphibole asbestos fibre with a length > 5 μm:	equivalent factor 10
1 amphibole asbestos fibre with a length < 5 μm:	equivalent factor 1

In most literature on asbestos in house dust no distinction is made between chyrosotile and amphibole asbestos. Therefore, most information is expressed in numbers of asbestos fibres. When available, also information about the asbestos type is provided.

4.2 Concentration of asbestos in house dust

Deposition of asbestos fibres takes place, depending on the aerodynamic diameter, by Brownian diffusion, interception, and impaction. The very fine fibres act approximately as molecules and move according to Brownian diffusion, causing them to coagulate with larger house particles before they sediment.

A problem of determination of asbestos in both house dust and air is that different sampling methods give different results (Crankshaw et al., 2000; Ewing, 2000). For example, wipe samples tend to more accurately reflect all accumulated asbestos, whereas a microvacuum method does not (Crankshaw et al., 2000).
Ultrasonication, which is used for sample preparation for some methods, may liberate asbestos from particles that break down into multiple fibres. As a result, the reported asbestos concentration overestimates the number of free respirable fibres in the dust, thereby negating any potential use as a surrogate for past or future airborne exposure (Lee et al., 1995). For example, an interlaboratory round robin of asbestos containing dust samples showed up to a 1000-fold differences in asbestos concentration between laboratories, probably caused for a large extent by the sonication process (Lee et al., 1996). This indicates that the figures of asbestos concentration in dust and air samples can be biased considerable due to sample handling (Lee et al., 1996).

Asbestos in dust can be regarded as an indicator of past airborne concentrations, as an indicator of future airborne exposure, or simply as another component in nuisance dust (Lee et al., 1995). If the measurement is to reflect past or potential future exposure, it should be performed in a manner that assesses total respirable fibres. Respirable fibres are fibres that can end up in the lungs, and are considered to have a diameter smaller than 3 μm and a length smaller than 200 μm. If it is of interest as a nuisance dust, a determination of mass fraction is necessary.

Little information on actual asbestos levels in house dust has been found. This is probably due to the use of qualitative methods that assess whether asbestos is present or not, and semi-qualitative methods that can identify different ranges of asbestos concentration in dust. In addition, most data have not been published in scientific journals.
Lee et al. and Millette and Mount reported information on the relationship between asbestos fibres in the air and in dust (Lee et al., 1995; Millette et al., 2000). The ratio between the concentration of fibres in the air and the reported concentration of fibres in dusts on surfaces is referred to as the K-factor (cm^{-1}). Typical K-factors for a variety of particulate materials including asbestos, lie in the range of 10^{-4}-10^{-7}, that is, one fibre is calculated to be airborne (fibres/cm^3) for every 10^4-10^7 fibres on surfaces (fibres/cm^2) (Lee et al., 1995; Millette et al., 2000). It should be noted that there is some discussion about the validity of the K-factor in literature.
It is suggested that the amount of asbestos fibre suspended into the air during a specific activity may be primarily dependent on the amount of asbestos on the surface initially and the energy of the activity (Millette et al., 2000).

Table 10 presents asbestos levels that were determined in house dust. Ewing analysed asbestos in dust samples in 66 buildings (Ewing, 2000). The geometric mean surface concentration was 3.7×10^6 asbestos fibres per cm^2 in areas with asbestos-containing fireproofing. Samples collected from areas having asbestos-containing acoustical plaster had

a geometric mean of 0.16×10^6 asbestos fibres per cm^2. Samples collected in six buildings without friable asbestos-containing surfacing materials indicated a geometric mean of 1×10^3 asbestos fibres per cm^2. Virtually all the asbestos fibres found were chrysotile (Ewing, 2000). Crackshaw determined asbestos in house dust using different sampling techniques (Crankshaw et al., 2000). The results of the wipe method, reflecting probably the most accurate measure of all accumulated asbestos, are presented in Table 10. The arithmetic mean for samples taken in the basement was 1.7×10^6, whereas asbestos levels in dust upstairs were lower with a mean of 4.0×10^3 fibres per cm^2 (Crankshaw et al., 2000).
Hence, asbestos levels in house dust averages probably in the order of 1×10^3 fibres per cm^2 in background houses, i.e. houses without friable asbestos containing materials or asbestos contamination from other sources such as contaminated soil in the direct surroundings. In houses with asbestos containing material, average levels can increase with a factor 1×10^4.

Table 10: Overview of several studies that determined asbestos levels in house dust.

		asbestos in house dust (fibres/cm^2)		
Study	Sampling method	Geometric mean	Range	Remarks
Ewing 2000	Vacuum	3.7×10^6	$7000\text{-}140\times10^6$	Areas with asbestos containing fireproofing
Ewing 2000	Vacuum	0.16×10^6	$<3500\text{-}74\times10^6$	Areas having asbestos containing acoustical plaster
Ewing 2000	Vacuum	1×10^3	$<240\text{-}0.21\times10^6$	Buildings without asbestos containing material
Crackshaw 2000	Wipe	1.7×10^6 *	$0.14\times10^6\text{-}7.0\times10^6$	Basement one residence (n=5)
Crackshaw 2000	Wipe	4.0×10^3 *	$1.8\times10^3\text{-}5.3\times10^3$	Upstairs one residence (n=3)

* Arithmetic mean.

4.3 Is there a relationship between the concentration of asbestos in house dust and asbestos in exterior soil?

No literature was found on a possible relationship between the concentration of asbestos in house dust and asbestos in soil outdoors.

The concentration of asbestos in house dust is highly variable and depends for the largest part on asbestos containing materials used in the building (personal communication P.C. Tromp, 2003). Asbestos contaminated soil in the vicinity, for example the garden, can result in a contribution to the asbestos in house dust. However, this was only found in a few cases in the surroundings of Goor and Harderwijk, the Netherlands, where asbestos pulp was used in asphalt on the road (personal communication P.C. Tromp, 2003). No asbestos was determined in indoor air in those houses. The asbestos concentration in house dust in Goor was determined by a semi-quantitative method and was between 10 and 100 fibres/cm^2 (personal communication P.C. Tromp, 2003). This suggests that the concentration of asbestos in air can vary between 1 and 10000 fibres/m^3 (calculated by using a K-value of $10^{-4}\text{-}10^{-7}$).

Some rubble roads in the province Twente, the Netherlands, were paved with pure asbestos cement, containing approximately 0.1-0.15 kg asbestos per kg cement (Tempelman, 1998). Nonfriable asbestos such as asbestos cement, will only release asbestos fibres after mechanical treatment. Driving on this road can result in asbestos concentrations in the air

between 1000 fibre equivalents/m^3 (Negligible Risk Level) and 100000 fibre equivalents/m^3 (Maximum Tolerable Risk Level). When assuming that the outdoor air concentration of asbestos approximates the indoor level, it can be anticipated that the rubble roads cause an increase in asbestos concentration in house dust.
Most other cases with rubble roads are less extreme, containing between 15 and 400 mg asbestos per kg rubble (Tempelman, 1998). This is a factor 250-10000 less asbestos than in the rubble roads in Twente, the Netherlands. Hence, the risk associated with these less extreme asbestos contamination roads is considered negligible, i.e. below the Negligible Risk Level of 1000 fibre equivalents/m^3.

Hypothetically, the concentration of asbestos in house dust can be calculated from the surface dust loading, the contribution of soil to house dust, and the concentration of asbestos in soil. Assuming that the dust loading of surfaces is 0.56 g dust/m^2 (see § 2.2), and 30-70% of house dust consists of soil material (see § 2.3) the soil loading in houses is 0.17-0.39 g/m^2. Using a contamination level of asbestos in soil of 100 mg/kg (proposed Intervention Value interim policy (Swartjes et al., 2003)), the asbestos concentration in house dust is 0.017-0.039 mg/m^2. This value can be converted into 0.85×10^3-2.0×10^3 fibres/cm^2, although it should be noted that this is only an indication because fibres of different sizes and weights exist (Slooff et al., 1989). This calculated value is similar to the background level of 1×10^3 fibres/cm^2, see § 4.1. Hence, a significant contribution of asbestos in exterior soil can only be expected for asbestos levels in exterior soil exceeding 100 mg/kg. Since little is known about the contribution of exterior soil to interior asbestos exposure, it is recommended to determine the asbestos level of house dust for cases where asbestos levels in soil adjacent to the house exceed 100-1000 mg/kg, depending on the risk expected based on the condition of the asbestos (friable versus nonfriable asbestos). This recommendation is in agreement with a draft assessment protocol drafted by TNO-MEP for site specific assessment of human risks to soil contamination with asbestos (TNO-MEP, 2003). The protocol recommends further research if the asbestos concentration is >1000 mg/kg dry weight for nonfriable asbestos, and >100 mg/kg dry weight for friable asbestos.

4.4 Asbestos in air

Table 11 gives an overview of levels of asbestos measured in air in several studies. It is generally assumed that the concentration of asbestos inside dwellings is roughly similar to the concentration in the outdoor air, the latter being on average < 0.1-5 ng/m^3 (~10^2-10^4 fibres/m^3) in the Netherlands in the 1980s (Slooff et al., 1989). The higher values mostly occur in urban and industrial areas (Slooff et al., 1989). Higher levels than 10^4 fibres/m^3 have been found in road tunnels, in the surroundings of asbestos processing industry, and sometimes near dumping sites. After the Enschede explosion in 2000 (see Table 11 for further explanation), asbestos levels in outdoor air were in most cases 250-580 fibre equivalents/m^3, whereas in a few cases asbestos levels of 10900 fibre equivalents/m^3 were detected.
Similar to Slooff and Blokzijl (Slooff et al., 1989), and in agreement with Mennen et al. (Mennen et al., 2001), Besson et al. estimated that asbestos levels in indoor air are usually around 10^3 fibres/m^3, which was determined for a case of pollution by asbestos fibres at low levels (Besson et al., 1999).
The concentration measured in the Netherlands in buildings in which sprayed asbestos layers were present varied between < 1 and 6000 ng/m^3, whereas concentrations of up to 170 ng/m^3

were found caused by a chrysotile containing floor covering (Slooff et al., 1989). Landrigan et al. reported that levels of airborne asbestos inside school buildings with asbestos-containing ceiling materials may exceed outdoor ambient levels by a factor of approximately 100 (Landrigan et al., 1987).

In the following calculation, the link between the asbestos concentration in air and in house dust via the K-factor can be verified for the present data. Using the background level of asbestos in indoor air of 10^3 fibres/m^3 (= 1×10^{-3} fibres/cm^3) (Besson et al., 1999; Brand et al., 1994), and a K-factor of 10^4 (see §4.1) (Lee et al., 1995; Millette et al., 2000) yields an estimated asbestos concentration in house dust of 10 fibres/cm^2. Using a K-factor of 10^7 (Lee et al., 1995; Millette et al., 2000) yields an asbestos concentration in house dust of 1×10^4 fibres/cm^2. The concentration of asbestos in house dust was estimated in several experimental studies to range between 1×10^3 and 1×10^7 fibres per cm^2, with the lower values for the background situation, see §4.2. Hence, although there is some overlap, the calculated levels of asbestos in house dust (fibres/cm^2) are low in comparison with experimentally determined levels. The most likely explanation is that the K-factor is too low (a factor of 1×10^6-1×10^9 would do better), or because a K-factor is not applicable.

Table 11. Overview of studies reporting asbestos levels in air.

Source	Location	Concentration asbestos in air$^{\Delta}$	Remarks
Slooff 1989	Outdoor	<0.1-5 ng/m^3 (~10^2-10^4 fibres/m^3)	Assumption: concentration inside houses roughly similar to concentration in outdoor air in 1980s.
Slooff 1989	Indoor	<1 – 6000 ng/m^3	Measured in building in which sprayed asbestos layers were present.
Slooff 1989	Indoor	Up to 170 ng/m^3	Caused by chrysotile containing floor covering
Brand 1994	Indoor	Asbestos levels in indoor air usually around 1000 fibre equivalents/m^3	Year average
Besson 1999	Indoor	3×10^3-8×10^4 fibres/m^3	Building with low levels of asbestos pollution; extrapolated from measurement of fibres greater than 3 μm in length.
Mennen 2001	Outdoor	250-580 fibre equivalents/m^3 highest value 10900 fibre equivalents/m^3	In most cases in days following the Enschede explosion*. Highest asbestos levels found in a few samples in days following the Enschede explosion*.
Swartjes 2003	Outdoor	10-100 fibers/m^3	Reference: ATSDR

$^{\Delta}$ Note that the asbestos concentration in air is expressed in ng/m^3 and in fibre (equivalents)/m^3. Asbestos concentrations expressed in weight/m^3 cannot be converted straightforward to fibres/m^3, due to difference sizes and weights of fibres.

* In May 2000 the city of Enschede, the Netherlands, was hit by a series of explosions in a fireworks storage depot in the middle of a residential city district. The explosion resulted in a huge fire, in which more than 200 houses and dozens of commercial buildings were damaged.

4.5 Contribution of asbestos in house dust and air to asbestos exposure

4.5.1 Qualitative

House dust can probably contribute substantially to asbestos exposure. There possibly is a relationship between asbestos in house dust and asbestos levels in indoor air (Lee et al., 1995; Millette et al., 2000), although there is no complete agreement on this issue in the literature. Asbestos in house dust can be resuspended into air, resulting in exposure of children and adults to asbestos via inhalation. Resuspension is much larger in the indoor environment than outdoors, as most surfaces are dry. Resuspension is increased with activity in a room, for example the presence of people (Besson et al., 1999). Most fibres can be removed by wet cleaning. Experiments by TNO show that high asbestos levels are mainly found in houses with deep-pile carpet, because other surfaces are often cleaned wet.

In most cases, asbestos in house dust and indoor air is not related to asbestos contamination in soil. Asbestos in building material is the most prominent source of asbestos in the indoor environment (personal communication Peter Tromp, 2003).

Asbestos from asbestos contaminated soil can be resuspended into the air by wind or mechanical activity. The wind conditions together with relative humidity and the mechanical activity determine how many fibres can be transported into the air. Probably due to the relative humid average weather conditions in the Netherlands and the large air volume in which fibres are diluted, asbestos from contaminated soil contributes only in extreme cases substantially to asbestos levels in house dust and indoor air.

4.5.2 Quantitative assessment

To have an impression of the relative importance of the exposure to asbestos via ingestion and inhalation of house dust a calculation according to CSOIL (Otte et al., 2001) is presented. These pathways of exposure are compared and discussed.
The calculations are performed to simulate exposure of children to asbestos, as they are the group at risk due to their high house dust intake and due to their long life expectancy.

Ingestion
In CSOIL, uptake via ingestion is calculated according to equation 8.

$$DI = \frac{AID \times C_s \times F_a}{W} \qquad (8)$$

with:
- DI: uptake via ingestion ($mg_{contaminant} \times kg_{dust}^{-1} \times d^{-1}$)
- AID: daily intake house dust via ingestion ($kg_{dust} \times d^{-1}$)
- W: body weight (kg)
- F_a: relative absorption factor, set at 1 (-)
- C_s: initial dust content ($mg_{contaminant} \times kg_{dust}^{-1}$)

Asbestos levels in house dust range between 1×10^3 and 1×10^7 fibres/cm^2, with the lower value representing the background situation. Here, exposure via ingestion is calculated for a

contaminated house, i.e. assuming an asbestos level in house dust of 1×10^5 fibres/cm^2. A problem is converting this asbestos level expressed in fibres/cm^2 to an asbestos concentration expressed in mg asbestos per kg dust. To that end, the following calculation is performed. The number of fibres is converted to weight, assuming that 1×10^3 fibres/cm^2 approximately equals 2 ng fibres/cm^2 (Slooff et al., 1989). It should be noted that this is a crude assumption because fibres of different sizes and weights exist.

$$1\times10^5 \ \frac{\text{fibres}}{\text{cm}^2} = 200 \ \frac{\text{ng}}{\text{cm}^2} \qquad (9)$$

Subsequently, this is converted to a concentration of asbestos by assuming that a surface is covered by 0.56 g of dust per m^2, see § 2.2 (Hawley, 1985):

$$\frac{200\times10^{-6} \ \frac{\text{mg asbestos}}{\text{cm}^2}}{5.6\times10^{-8} \ \frac{\text{kg dust}}{\text{cm}^2}} = 3.6\times10^3 \ \frac{\text{mg asbestos}}{\text{kg dust}} \qquad (10)$$

Using a daily intake of house dust via ingestion of 80 mg (AID=8×10^{-5} kg/day), see § 3.5, and a relative absorption factor of 1 (F_a=1), the uptake for a child of 15 kg (W=15 kg) via ingestion is estimated to be:

$$\text{DI}=\frac{8\times10^{-5}\times3.6\times10^3\times1}{15} = 0.019\ \text{mg}\times\text{kg}^{-1}\times\text{d}^{-1} = 19\ \mu\text{g}_{\text{asbestos}}\times\text{kg}^{-1}_{\text{body weight}}\times\text{d}^{-1} \qquad (11)$$

Inhalation

In CSOIL, uptake via inhalation is calculated according to equation 12 and 13:

$$\text{IP}=\frac{\text{TPS}\times\text{AV}\times\text{t}\times\text{f}_\text{t}\times\text{C}_\text{s}\times\text{F}_\text{r}\times\text{F}_\text{a}}{\text{W}} \qquad (12)$$

with:

IP:	Exposure to inhaled contaminant	($\text{mg}_{\text{contaminant}}\times\text{kg}_{\text{body weight}}^{-1}\times\text{d}^{-1}$)
C_s:	Concentration contaminant in dust particles	($\text{mg}_{\text{contaminant}}\times\text{kg}_{\text{dust}}^{-1}$)
F_r:	retention factor in lungs	(-)
F_a:	relative absorption factor	(-)
W:	body weight	($\text{kg}_{\text{body weight}}$)
TPS:	amount of suspended dust particles in air	($\text{mg}_{\text{dust}}\times\text{m}_{\text{air}}^{-3}$)
AV:	breathing volume	($\text{m}_{\text{air}}^3\times\text{d}^{-1}$)
t:	duration of exposure	(d)
f_t:	time fraction of exposure	(-)

Where TPS ($\text{kg}_{\text{dust}}/\text{m}^3$) × C_s ($\text{mg}_{\text{asbestos}}/\text{kg}_{\text{dust}}$) is the amount of asbestos per m^3 air. The value for the amount of asbestos per m^3 air is obtained from Table 11, and is 1-6000 ng/m^3, with a reasonable average for a contaminated house of 1000 ng/m^3 = 1×10^{-3} mg/m^3.
Furthermore, it is assumed that the retention factor in the lungs is 1 (F_r=1), the relative absorption factor is 1 (F_a=1), and the daily exposure duration is 16 h indoors and 8h outdoors ($t\times f_t$=16/24). A breathing volume of a child of 7.6 m^3/d is assumed:

$$IP = \frac{1\times10^{-3}\times7.6\times\frac{16}{24}}{15} = 3.4\times10^{-4}\ \mathrm{mg\times kg^{-1}\times day^{-1}} = 0.34\,\mu g_{asbestos}\times kg^{-1}_{body\ weight}\times d^{-1} \quad (13)$$

The present calculation for exposure to asbestos via ingestion and inhalation is very crude due to the uncertainty in the values assumed for asbestos in dust and air, and due to the conversion of asbestos levels to weight/weight units. However, it can be concluded that absolute exposure of asbestos via ingestion of house dust is probably higher than exposure via inhalation. However, toxicity of inhaled asbestos is far greater than toxicity of ingested asbestos (Slooff et al., 1989; Montizaan et al., 1987). Exposure to asbestos via inhalation is thus the exposure pathway of interest. House dust can contribute to the amount of asbestos in the air via resuspension, and thus to exposure via inhalation.

4.6 Recommendations for the contribution of house dust in risk assessment of asbestos in the Netherlands

Recently, a draft assessment protocol has been formulated by TNO-MEP (TNO-MEP, 2003), based on a RIVM/TNO/Grontmij report 711701034 (Swartjes et al., 2003). The draft assessment protocol describes a detailed procedure for the assessment of human risks to soil contamination with asbestos (TNO-MEP, 2003). The protocol consists of a step by step approach, i.e. a TIERed approach. The first step consists of a simple test, in which asbestos is determined in the upper soil layer. If the concentration of asbestos exceeds 1000 mg/kg dry weight for nonfriable asbestos or 100 mg/kg dry weight for friable asbestos, and depending on the land use, the second step should be taken. In the second step the respirable asbestos fibres in the soil outside and/or in house dust inside are determined according to the draft NEN guideline 2991 (Draft NEN 2991, 2004). When the concentration of sedimented asbestos fibres in house dust exceeds 100 fibres/cm^2 or the respirable asbestos fibres in soil exceeds 10 mg/kg dry weight, the third step is necessary. Finally, in the third step, asbestos levels in the air indoors or outdoors are measured. If the air concentration exceeds 1000 fibre equivalents/m^3 (Negligible Risk Level), there is a location specific risk.
As this draft assessment protocol has been formulated and well-considered, for recommendations for risk assessment on exposure to asbestos via house dust, the present report refers to this protocol. It should be noted that the concentration of asbestos in the upper soil layer above which further research is required, i.e. step 1, >1000 mg/kg for nonfriable asbestos and >100 mg/kg for friable asbestos, are in agreement with the present report (see § 4.3). However, the concentration of asbestos mentioned in step 2 and 3 are *not in agreement* with levels of asbestos presented in this report and found in the literature. In step 2 of the draft assessment protocol further research is required if the asbestos concentration in house dust exceeds 100 fibres/cm^2. In the present report, the background concentration of asbestos in house dust is considered to be 1000 fibres/cm^2 (Table 10). The method of measurements may have lead to this difference. Similarly, asbestos levels in air found in the literature in the present study indicated background levels of 1000 fibres/m^3 (Table 11), whereas levels higher than 1000 fibres/m^3 are considered to be linked to the presence of a location specific risk in the draft assessment protocol, in step 3. The draft assessment protocol or the NEN do not give a derivation of the critical asbestos levels (TNO-MEP, 2003; Draft NEN 2991, 2004). The cause of the difference found in the present report and these critical

asbestos levels in the draft assessment protocol should be investigated and a rationale for these critical asbestos levels should be given in the assessment protocol.

4.7 Conclusions

In conclusion:
There possibly is a relationship between asbestos in house dust and asbestos in indoor air, which is expressed by a K-factor. According to the literature, this K-factor amounts 10^{-4}-10^{-7}, indicating that one fibre is airborne (fibres/cm^3) for every 10^4-10^7 fibres on surfaces (fibres/cm^2).
Asbestos contaminated soil contributes probably only in a few, extreme cases to asbestos levels inside houses. In those cases, the soil should be contaminated with high levels of asbestos and the contaminated site should be in the direct surroundings of the house. However, little information is available on this issue. Based on a hypothetical calculation, a significant contribution of asbestos to house dust from asbestos contaminated soil may occur at soil concentrations greater than 100 mg/kg. To gain more insight into the contribution of exterior soil contamination to interior house dust contamination with asbestos, it is recommended to determine asbestos levels in house dust in houses adjacent to sites that are contaminated with asbestos above 1000 mg/kg for nonfriable asbestos, and above 100 mg/kg for friable asbestos. In most cases however, asbestos containing construction material is probably the main cause of asbestos contamination in houses.
Using several assumptions for reasonable amounts of asbestos in contaminated houses, and by calculating the concentration of asbestos in house dust in weight/weight units, the exposure to humans via ingestion of asbestos containing house dust is much greater than exposure via inhalation of air. However, orally ingested asbestos is less toxic than asbestos in the lungs. Asbestos in house dust can contribute to the exposure of asbestos via inhalation after resuspension. Therefore, if dust concentrations are elevated, indoor air measurements should be carried out.
The recommendations for further research are in agreement with a draft assessment protocol on site specific assessment of human risks to soil contamination with asbestos. In this protocol a detailed procedure is described including assessment of asbestos in house dust. Therefore, for recommendations for risk assessment of asbestos in house dust, we refer to this protocol. It should be noted that the levels of asbestos in house dust and air found in the literature in the present report are not in agreement with critical levels of asbestos mentioned in the draft assessment protocol. The cause for this difference should be investigated before setting these levels.

5. Overall conclusions and recommendations

General characteristics house dust

Conclusions

- The amount of dust that settles per day is probably relatively constant, with values of 0.002 to 0.009 $g \times m^{-2} \times day^{-1}$ (= 2-9 $mg \times m^{-2} \times day^{-1}$).
- Most studies found values for dust loadings of < 1 g/m^2 for hard surfaces, which is in agreement with the value for dust covering of surfaces assumed by Hawley of 0.56 g/m^2
- Based on several studies it was concluded that soil contributes to 30-70% to house dust.

Recommendations

- The concentration of particulate matter in indoor air of 52.5 $\mu g/m^3$ that is used in the exposure model CSOIL is a reasonable value for inside houses and other moderately crowded places. However, personal samples are probably better representatives for human exposure. Particulate matter concentrations were higher when measured near the subject or at lower heights (children), than in bulk indoor air. Mean particulate matter levels for personal samples are slightly higher (60 $\mu g/m^3$) for moderately crowded places, or considerably higher (100 $\mu g/m^3$) for very crowded places such as classrooms. These latter values are recommended in CSOIL for future use.

Lead

Conclusions

- A reasonable mean concentration of Pb in house dust lies generally in the range 300-700 mg/kg.
- The Pb concentration in house dust is generally higher than in exterior soil, with an average factor of approximately 3.
- Some studies have found a direct relationship between Pb concentration in house dust and in soil, whereas other studies did not find such a relationship.
- Many experimental and epidemiological studies suggest that house dust is a major source of exposure to Pb.
- Exposure to Pb to children via ingestion of soil and house dust is probably in the same order of magnitude, whereas exposure to Pb via inhalation is negligible (approximately a factor 300 lower). Therefore, exposure to Pb via house dust should be included in risk assessment.

Recommendations

- In the assessment of soil contamination, it is aimed for to include Pb in house dust caused by outdoor soil, but not Pb from other sources. For the Dutch situation it is recommended to use a soil ingestion rate of 100 mg/day, as is also presently used, to cover both Pb in soil and in house dust. In addition, a factor should be included to account for a different concentration of Pb in house dust caused by soil, compared to the concentration of Pb in outdoor soil. This factor may range between 1 and 2, depending on the assumptions made. A safe approach is to use the factor 2, or measure the Pb concentration in house dust and average this value with the concentration found in soil outdoors. It should be noted that according to these approaches, both enrichment of Pb in house dust and contribution of Pb from other sources than soil are included. Further research on the contribution of Pb in house dust caused by soil is recommended.

Asbestos

Conclusions

- There is possibly a relationship between asbestos in house dust and asbestos in indoor air, which is expressed by a K-factor. According to the literature, this K-factor amounts 10^{-4}-10^{-7}, indicating that one fibre is airborne (fibres/cm^3) for every 10^4-10^7 fibres on surfaces (fibres/cm^2).

Recommendations

- Asbestos contaminated soil contributes probably only in a few cases to asbestos levels inside houses. In those cases, the soil should be contaminated with high levels of asbestos and the contaminated site should be in the direct surroundings of the house. However, little information is available on this issue. Based on a hypothetical calculation, significant contribution of asbestos to house dust from asbestos contaminated soil may occur at soil concentrations greater than 100 mg/kg. To gain more insight into the contribution of exterior soil contamination to interior house dust contamination with asbestos, it is recommended to determine asbestos levels in house dust in houses adjacent to sites that are contaminated with asbestos above 1000 mg/kg for nonfriable asbestos, and above 100 mg/kg for friable asbestos.
- Exposure to humans via ingestion of asbestos containing house dust is greater than exposure via inhalation of air. However, orally ingested asbestos is less toxic than asbestos in the lungs. Asbestos in house dust can contribute to the exposure of asbestos via inhalation after resuspension. Therefore, if dust concentrations are elevated, indoor air measurements should be carried out.
- The recommendations for further research are in agreement with a draft assessment protocol written by TNO-MEP on site specific assessment of human risks to soil contamination with asbestos (TNO-MEP, 2003). In this protocol a detailed procedure is described including assessment of asbestos in house dust. Therefore, for recommendations for risk assessment of asbestos in house dust, we refer to this protocol. It should be noted that the levels of asbestos in house dust and air found in the literature in the present report are not in agreement with the respective levels of asbestos mentioned in the draft assessment protocol. The cause for this difference should be investigated.

Acknowledgement

Peter Tromp (TNO-MEP) and Frank Swartjes (RIVM) are acknowledged for their input on the issue of human exposure to asbestos via house dust. We also owe a debt of gratitude for the information, advice and remarks provided by the ‘expert group on human-toxicological risk assessment’ (J. Vegter, TCB-Secretariaat; C.J.M. van de Bogaard, VROM-Inspectie; T. Crommentuijn, DGM-BWL; S. Dogger, Gezondheidsraad; R. van Doorn, GGD Rotterdam; T. Fast; J.E. Groenenberg, Alterra; K.H. den Haan, VNO/NCW-BMRO; J. Tuinstra, Royal Haskoning; D.H.J. van de Weerdt, Hulpverleningsdienst Gelderland Midden; J. Wezenbeek, Grontmij; J.A. van Zorge, DGM-SAS; Th. Vermeire, RIVM-SEC; J. Lijzen, RIVM-LER).

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