National Institute for Public Health
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The case of shock-absorbing tiles

RIVM Report 2016-0184

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Colophon

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Synopsis

Assessment of the product limit for PAHs in rubber articles.
The case of shock-absorbing tiles

Polycyclic aromatic hydrocarbons (PAHs) are harmful substances that can be present in, among other things, rubber articles. For the safe use of rubber articles, like shock-absorbing tiles, producers have to comply with the European limit which has been set for PAHs in consumer products. Shock-absorbing rubber tiles are made of used car tyres and contain PAHs.

RIVM has received a request to investigate whether the current product limit for PAHs in rubber tiles provides an adequate level of protection against the development of cancer. At this moment, only an indication of the extra risk of developing cancer can be given, because there is a lack of reliable data regarding the exposure of children to PAHs from the tiles (via dermal contact and hand-to-mouth contact). These data, however, are necessary for a proper risk assessment to be made. Missing data, for example, include the skin-tile contact time and the rate that the PAHs migrate from the tiles. Further investigation into these data was not possible within the time limits of the study. To reduce the uncertainties in the present risk assessment, additional data on the exposure are required.

In addition, to derive the extra risk of adverse health effects from the results of animal studies, so-called safety factors are used. In this study, a standard safety factor for substances which cause cancer was used. At this moment, there is no agreement within Europe on the use of extra safety factors for cancer-causing substances. For this reason, RIVM recommends initiating a discussion at European level to obtain agreement on this subject.

In general, the risks of the exposure to substances which cause cancer is indicated as the *extra* number of people who get cancer per million of exposed people; the term 'extra' is used as people have the risk of developing cancer without this exposure. An extra risk of 1 in a million exposed people is regarded as negligible in the risk assessment of substances causing cancer. In this study, because of the quantified uncertainties, the extra cancer risk is presented as a range, meaning that the extra risk lies between two extreme values. If the PAH-concentration in the rubber tiles is equal to the limit for consumer products, this range lies around the negligible risk level of 1 per million. At the maximum value of the range the negligible risk level is slightly exceeded.

The results of this study can be used in the evaluation of the product limit for PAHs in consumer articles by the European Commission. In this evaluation it is important to also include the exposure to PAHs from other consumer articles.

Key words: PAHs, rubber tiles, polycyclic aromatic hydrocarbons, risk assessment, risk estimation, consumer product

Publiekssamenvatting

Evaluatie productnorm voor PAK's in rubberen artikelen.
Een casestudy naar valdempende tegels

Polycyclische aromatische koolwaterstoffen (PAK's) zijn schadelijke stoffen die onder andere in artikelen van rubber kunnen voorkomen. Voor een veilig gebruik van rubberen artikelen, zoals valdempende tegels, moeten producenten zich houden aan de Europese norm voor PAK's in consumentenproducten. Valdempende rubbertegels worden meestal gemaakt van afgedankte autobanden en bevatten PAK's.

Het RIVM is gevraagd te onderzoeken of de huidige productnorm voor PAK's in rubbertegels gebruikers voldoende beschermt tegen het ontstaan van kanker. Op dit moment kan alleen een indicatie van het risico op kanker worden gegeven. Dat komt doordat veel betrouwbare gegevens ontbreken over de mate waarin kinderen in contact komen met de PAK's uit de tegels (via contact van de huid en via hand-mond-contact). Het gaat onder andere om gegevens over de duur van het contact tussen de tegel en de huid en de mate waarin PAK's dan uit de tegels vrijkomen. Deze informatie is wel nodig om de onzekerheden in de huidige risicobeoordeling te verkleinen. Aanvullend onderzoek hiernaar kon binnen het tijdbestek van dit onderzoek niet worden uitgevoerd.

Daarnaast is er geen overeenstemming binnen Europa over de hoogte van zogeheten veiligheidsfactoren voor kankerverwekkende stoffen. Veiligheidsfactoren worden gebruikt om het risico op effecten op de gezondheid voor mensen te kunnen afleiden uit de resultaten van dierstudies. In de huidige studie is een standaard veiligheidsfactor voor kankerverwekkende stoffen gebruikt. Het RIVM beveelt daarom aan om op Europees niveau een discussie te initiëren om hierover overeenstemming te bereiken.

In het algemeen worden de risico's van de blootstelling aan kankerverwekkende stoffen uitgedrukt in het *extra* aantal mensen dat kanker krijgt per miljoen blootgestelden; de term 'extra' wordt gebruikt omdat mensen ook zonder blootstelling aan deze stoffen het risico lopen om kanker te krijgen. Een extra risico van 1 op de miljoen blootgestelde mensen wordt bij de risicobeoordeling van kankerverwekkende stoffen als verwaarloosbaar beschouwd. In dit onderzoek wordt, vanwege de doorgerekende onzekerheden, het extra risico op kanker weergegeven als een bandbreedte, wat betekent dat het extra risico tussen twee uiterste waarden ligt. Als de PAK-concentratie in rubbertegels gelijk is aan de norm voor consumentenproducten, ligt, bij de huidige kennis, deze bandbreedte rond het verwaarloosbare risiconiveau van 1 op de miljoen. Bij de hoogste uiterste waarde van de bandbreedte wordt het verwaarloosbare risiconiveau licht overschreden.

De resultaten van dit onderzoek kunnen worden gebruikt bij de evaluatie van de norm voor PAK's voor alle plastic en rubberen consumentenproducten door de Europese Commissie in 2017. Bij deze

evaluatie is het van belang ook rekening te houden met de blootstelling aan PAK's uit andere consumentenproducten.

Kernwoorden: PAKs, valdempingstegels, polycyclische aromatische koolwaterstoffen, risicobeoordeling, risicoschatting, consumentenproduct, rubberen tegels, rubbertegels

Contents

Summary

Currently, the limit value for PAHs in rubber tiles is set to 1 mg PAH per kg for each of eight marker PAHs, the so-called REACH PAH8. The Dutch Ministry of Public Health, Welfare and Sports (VWS) would like to evaluate whether this limit value provides an adequate level of protection or is overly conservative. With this aim, VWS requested an assessment of the extra risk of cancer resulting from exposure to PAHs present in rubber tiles used in playgrounds, in concentrations equal to the limit values.

Dermal and oral exposure are considered the main routes of PAH exposure from rubber tiles for children used in playgrounds. We selected one of three previous applied approaches as a preferred approach to estimate dermal exposure. In addition, we assessed the oral exposure due to hand-to-mouth contact. We aimed at a reasonable worst-case exposure assessment, using 75th percentiles of the parameter distributions rather than maximum reported values. Exposure from inhalation of PAHs that evaporated from the tiles, and oral and dermal exposure from small pieces of worn tiles were not considered in this study, leading to an expectedly small underestimation of the exposure. Nevertheless, we recommend investigating exposure from pieces of worn tiles to verify this assumption.

In the hazard assessment, we firstly assumed that the carcinogenicity of the EFSA PAH8 was similar to that of the REACH PAH8, regardless of the fact that the two groups have two differing PAHs of the eight[1]. The second assumption was that the composition of the REACH PAH8 in the rubber tiles is similar to the composition of the PAH mixture used in the carcinogenicity studies. These assumptions can only be partially verified, and may have a large influence on the outcome of the risk assessment, either by under- or overestimating the risk.

The final step of the risk assessment is the extrapolation of the cancer risk to a low dose. This is performed by two methods: linear extrapolation and model extrapolation. Whereas linear extrapolation is a worst-case method for high dose-low dose extrapolation, the model extrapolation method quantifies the uncertainty in the dose-response curve and yields a confidence interval for the calculated risk. In addition, to derive the extra risk on cancer from results of animal studies, so-called safety factors are used. In this study, a standard safety factor for substances causing cancer (allometric scaling factor of 7) was used. At this moment, there is no agreement within Europe on the use of additional safety factors for substances causing cancer. For this reason, RIVM recommends initiating a discussion at the European level to obtain agreement on this subject.

[1] EFSA PAH8 contain benzo[ghi]perylene and indeno[1,2,3-cd]pyrene, whereas these PAHs are not included in the REACH PAH8. Instead, the REACH PAH8 include benzo[e]pyrene and benzo[j]fluoranthene.

Based on the current knowledge, it is estimated that contact with rubber tiles with concentrations at the product limit of 1 mg PAH/kg tile for each of the eight REACH PAHs leads to an extra cancer risk between 1 per 590,000 and 1 per 7.7 million individuals who play(ed) regularly on rubber tiles. The upper level of this range is close to, but just higher than the negligible cancer risk level of 1 per 1 million. Nevertheless, the outcome of the risk assessment is uncertain, as the three parts (exposure, hazard and risk assessment) all contain significant uncertainties, in both directions (under- and overestimation of the risk). For this reason, it is recommended to refine the current risk assessment by collecting more information on a number of exposure parameters and by performing a full integrated probabilistic risk assessment (IPRA) for carcinogens.

The results of this study can be used in the evaluation of the product limit for PAHs in consumer articles by the European Commission. In this evaluation it is important to also include the exposure to PAHs from other consumer articles.

1 Introduction

The Dutch Ministry of Health Welfare and Sports (VWS) is reviewing the consequences of the REACH restriction on the polycyclic aromatic hydrocarbon (PAH) content of articles placed on the market (Annex XVII to REACH regulation) (EC, 2013), with regard to rubber tiles[2] used in playgrounds. According to the REACH restriction, the current product limit value in accessible plastic and rubber parts of consumer articles (with the exception of toys) is set to 1 mg PAH per kg product, for each of the eight PAHs mentioned in the REACH restriction (EC, 2013); the so-called REACH PAH8[3]. Within Annex XVII of REACH an evaluation of this specific REACH restriction by the European Commission is prescribed before 27 December 2017. As background information for the present review, VWS needs information on the consumer risks associated with exposure to different PAH-concentrations in rubber tiles. Consequently, VWS has asked for an estimation of the extra cancer risk following the exposure to the REACH PAH8 present in rubber tiles at playgrounds, using three different concentrations: concentrations at the limit value for rubber tiles, i.e. 8.0 mg REACH PAH8/kg, at 4.0 mg/kg and at 0.8 mg/kg.

In earlier studies, three different methods were applied to estimate dermal exposure to PAHs from rubber tiles. Two methods were described by the German Institute for Occupational Health BAuA in their proposal on the restriction of the use of PAHs in rubber products under REACH (BAuA, 2010), while the third is the diffusion approach used in an earlier study on PAHs in rubber tiles (RIVM 2013). The present report summarizes these methods before selecting one of them to estimate the dermal exposure (Chapter 2). Other pathways of exposure were also considered and some were included in the exposure assessment (Chapter 2). A literature search was performed to obtain information on the input parameters of the exposure assessment (Chapter 3). We aimed at a conservative but realistic exposure estimation ('reasonable worst-case'), using 75th percentiles from the distributions of the parameters, rather than maximum reported values, whenever information on distributions was available.

In Chapter 4, the carcinogenicity of PAHs is reviewed and in Chapter 5 we assess the extra cancer risk due to the exposure to PAHs from rubber tiles. For the extrapolation of the cancer incidence in animal studies to low incidences, in addition to the traditionally used linear extrapolation method, an extra approach was applied to visualize (a part of) the uncertainty in the cancer risk estimation. Furthermore in Chapter 5, we derived the PAH product concentrations leading to several pre-defined extra risk levels (such as $1{\cdot}10^{-5}$, $1{\cdot}10^{-6}$). This may facilitate the task for risk managers when establishing a founded product limit value at these pre-defined risk levels. In addition, the Margin of Exposures for

[2] including rubber mats, pavement, etc., or rubber granules which are compressed, coagulated, glued or otherwise fused into tiles, mats, pavement, etc.
[3] benzo[a]pyrene, benz[a]anthracene, benzo[b]fluoranthene, chrysene, benzo[k]fluoranthene, dibenz[a,h]anthracene, benzo[e]pyrene, benzo[j]fluoranthene.

the three different PAH-concentrations were derived. Finally, the discussion on the applied methodology and the findings can be found in Chapter 6, whereas Chapter 7 presents the conclusions and recommendations.

2 Method for exposure assessment

2.1 Dermal exposure

To derive the dermal exposure of children to PAHs from rubber tiles in playgrounds, BAuA (2010) describe two approaches, and the RIVM (2013) describe a third. The methods used by BAuA are the ECETOC and migration approach, whereas RIVM used the diffusion approach. The three approaches are briefly described in sections 2.1.1-2.1.3. In section 2.1.4, one of these methods is selected as the preferred approach for the current study.

2.1.1 ECETOC approach

The approach used by the European Centre for Ecotoxicology and Toxicology of Chemicals (ECETOC) (BAuA, 2010 page 120) provides an exposure estimate based on the recommendations of the ECHA consumer exposure guidance document (ECHA, 2010)[4] and the ECETOC TRA guidance documents (ECETOC, 2004, ECETOC, 2009). Note that the intention of using the ECHA and ECETOC TRA guidance documents is to provide a conservative estimate of the exposure (Oltmanns et al., 2015, Delmaar et al., 2013).

In the ECETOC approach, the external dermal exposure amount is derived as:

$$EA_{ext\ dermal} = C * CA * LT * PD \qquad \textit{eq. 1}$$

$EA_{ext\ dermal}$	external dermal exposure amount [g]
C	PAH concentration in the rubber product [g/kg]
CA	contact area of bare skin with the product [m^2]
LT	layer thickness [m]
PD	product density [kg/m^3]

The layer thickness (LT) represents a fictive layer of the product from which all PAHs present in that layer are released. The exposure can be expressed as dermal load (e.g. in g/m^2) or external exposure (e.g. in g/kg bw) by dividing the exposure amount by the contact area or body weight respectively. In the ECETOC approach, contact times are not considered, i.e. the exposure is assumed to occur over an unknown period.

2.1.2 Migration approach

In the BAuA migration approach (BAuA, 2010) the exposure calculation of the ECETOC approach is extended by taking the release rates of PAHs from the rubber product into consideration and the contact time with the product per playground visit:

$$EA_{ext\ dermal} = C * CA * LT * PD * RR * CT \qquad \textit{eq. 2}$$

[4] It is noted that the ECHA 2010 document is outdated. However, as this document was used by BAuA in 2010, reference to it is maintained in the current document.

$EA_{ext\ dermal}$	external dermal exposure amount [g]
C	PAH concentration in the rubber product [g/kg]
CA	contact area of bare skin with the product [m^2]
LT	layer thickness [m]
PD	product density [kg/m^3]
RR	release rate [h^{-1}]
CT	contact time [h]

In the migration approach, it is assumed that the child moves around on a large area covered with rubber tiles. To estimate the dermal exposure, BAuA estimates the mass of the product from which PAHs can migrate to the skin by multiplying the contact area with a specific layer and product density. Subsequently, the amount released from this layer is related to the time of contact (in contrast with the ECETOC approach where *all* PAHs present in the layer are assumed to migrate). Again, the exposure can be expressed as dermal load (e.g. in g/m^2) or external exposure (e.g. in g/kg bw) by dividing the exposure amount by the contact area or body weight respectively.

In addition, the results of the migration approach can be multiplied by the frequency of playground visits to derive the chronic external exposure. Subsequently, the chronic internal exposure can be derived by using a dermal absorption fraction.

$$E_{int\ dermal} = EA_{ext\ dermal} * DA * F/BW \qquad \textit{eq. 3}$$

$E_{int\ dermal}$	chronic internal exposure due to dermal exposure [g/kg bw/day]
$EA_{ext\ dermal}$	external dermal exposure amount [g]
DA	dermal absorption fraction [-]
F	frequency of playground visits [day^{-1}]
BW	body weight [kg]

2.1.3 Diffusion approach

RIVM (2013) used a mechanistic emission model based on the well-established theory of diffusion of substances in materials. This approach firstly describes the diffusion of PAHs through the product to the surface of the product. Secondly, at the surface of the tile, dermal exposure occurs when the substances are transferred to skin coming into contact with this surface. The dermal diffusion model in ConsExpo[5] estimates the amount of PAHs available for this transfer (Delmaar et al., 2005). Subsequently, the concentration in the product's surface layer is supplemented with substances diffusing from deeper within the product.

The diffusion model can be used if the diffusion coefficient of the compound in the product is known or can be estimated. The model requires the following parameters:

- Concentration (C [g/m^3]): the concentration of PAHs in the product.
- Diffusion coefficient (D [m^2/s]): a value indicating how fast a substance can diffuse through the product.

[5] ConsExpo Web, www.ConsExpo.nl

- Product thickness (PT [m]): the thickness of the product that is in contact with the skin.
- Contact time (CT [h/day]): the duration of skin contact.
- Contact area (CA [m^2]): bare skin area in contact with the product.

The concentration in the product is described by the diffusion equation:

$$\frac{\delta C(x,t)}{\delta t} = D\frac{\delta^2}{\delta x^2}C(x,t) \qquad \textit{eq. 4}$$

D	diffusion coefficient [m^2/s]
C(x,t)	concentration in the product at depth x and time t [g/m^3]
x	depth in the product [m]

The rate of transport of the substance in the material is determined by the diffusion coefficient D in the material. The equation above is integrated numerically assuming that at the product-air surface, the flux of compound is zero (the evaporation of compound from the surface is disregarded, which is a worst-case assumption for dermal exposure). This integration yields the rate at which the substance is released from the surface of the material to the skin, the diffusional flux Φ [g/m^2/s]:

$$\Phi = -D\frac{\delta C(x,t)}{\delta x}\Big|_{x=at\ surface} \qquad \textit{eq. 5}$$

D	diffusion coefficient [m^2/s]
C(x,t)	concentration in the product at depth x and time t [g/m^3]
x	depth in the product [m]

This leads to the equation for the diffusion of PAHs from the material to the skin (i.e. the flux through the contact area):

$$\frac{dEA_{ext\ dermal}}{dt} = CA * D\frac{\delta C}{\delta x}\Big|_{x=at\ surface} \qquad \textit{eq. 6}$$

$EA_{ext\ dermal}$	external dermal exposure amount [g]
CA	contact area [m^2]
D	diffusion coefficient [m^2/s]
C(x,t)	concentration in the product at depth x and time t [g/m^3]
x	position in the product [m]

When this function is integrated over the contact time, the external dermal exposure amount ($EA_{ext\ dermal}$ [g]) is obtained. Subsequently, similar to the migration approach, the (chronic) internal dermal exposure ($E_{int\ dermal}$ [g/kg bw/day]) is given by equation 3.

2.1.4 Comparison of the three approaches

To perform the exposure assessment, one of the three approaches described above needs to be selected as a preferred approach. The aim of the exposure assessment is to estimate a realistic exposure, therefore, the model giving the most realistic description of the processes involved in the exposure will be selected.

As already stated, the ECETOC approach provides a conservative estimate of exposure as it is designed for screening purposes to identify those cases requiring further attention in a risk assessment. The main drawbacks of this approach are the use of a hypothetical layer and that it disregards contact time. It is assumed that at maximum, on one day, all substance present in the defined layer is available for exposure. By default, the approach does not describe a specific exposure scenario, as it does not differentiate between short-lasting or prolonged contact. Clearly, an approach not including an exposure time cannot yield a reliable estimate of exposure. This is further elaborated by evaluating the use of a 'layer thickness', a hypothetical layer limiting the release of a substance from the product. The concept of this layer contradicts general physics principles, which state that there is a flux of (PAH) molecules from a region of higher concentration to one of lower concentration. As a consequence, it is physically possible that PAHs outside of the defined layer will (over time) contribute to the exposure. Furthermore, the definition of a layer is an arbitrary choice. In conclusion, because of the physical impossibility of a layer, no empirical evidence exists to determine the actual thickness of the layer, and default values are solely based on expert judgment (Delmaar et al., 2013, Oltmanns et al., 2015).

In the migration approach, the absence of the parameter time in the ECETOC approach is solved by adding a release rate (i.e. fraction of the remaining amount in the product that is released per unit contact time), and the contact time. However, as in the ECETOC approach, the migration approach assumes PAHs solely migrating out of a particular (thickness) layer. As stated above, there is no evidence for an exposure limited to the amount present in a defined layer of the product.

The diffusion approach does not have the two drawbacks described above: It employs an exposure time and does not apply a hypothetical layer thickness, but uses the thickness of the tile. Moreover, of the three available approaches, the diffusion approach most accurately describes the processes involved in the dermal contact with a contaminated product. Models describing the diffusion of substances in materials have been developed and successfully applied in the fields of exposure assessment of chemicals from building materials and flooring (Huang and Haghighat, 2002, Xu et al., 2009) and the modelling of migration of chemicals from food packaging (Brandsch et al., 2002, Begley et al., 2005).

Dermal exposure to a substance emitted from a source can conceptually be described as the result of a number of transport processes (Schneider et al., 1999). The substance is emitted from the source and subsequently transferred to the skin. The emission is driven by diffusion of the substance in the material to the article's surface. In case of PAHs, it is assumed that transfer to skin is by direct contact with the skin. At the skin, the substance will partition into a layer on the skin, such as sweat or skin lipids, or directly into the skin. A disadvantage of the diffusion model is that, in general, the processes of mass transfer from product to the skin are incompletely understood and poorly quantified. For example, it is not known whether a substance first dissolves in sweat before it enters the skin, or whether it directly adheres to lipids or

proteins in the skin. Nevertheless, as we assume that the emission of the substance is limited by the diffusion to the materials' surface, detailed information on the transfer of the product to the skin is not needed. Given that mass transfer processes from product to the skin are not well quantified, the approach to estimate exposure by considering the intrinsic emission potential of a product itself seems reasonable.

In conclusion, the diffusion approach is the preferred method because it is the most conceptually realistic model.

2.2 Other routes of exposure

The three approaches described above solely consider dermal exposure to PAHs from rubber tiles. Nevertheless, there may be other relevant pathways of exposure, such as inhalation and oral exposure. These exposure pathways are addressed below.

2.2.1 Inhalation exposure

Llompart et al. (2013) searched for evaporated EFSA PAH8 from playground tile samples at temperatures up to 60°C[6] (selected temperature based on (Mattina et al., 2007)), but could not detect any. On the other hand, Llompart et al. (2013) did measure evaporation of benz[a]anthracene and chrysene, the two most volatile PAHs included in EFSA PAH8, at 60°C from newly purchased tiles. This indicates that inhalation exposure of these two PAHs may occur and subsequently this will result in higher exposure than that currently derived based on dermal and oral exposure only. However, the information on the evaporation is too limited to quantify the possible underestimation of the exposure to the two relatively high volatile PAHs. Other investigators measured PAHs evaporating from rubber granulate and concluded that inhalation exposure at artificial turf athletic fields results in extra risks of 10^{-9} up to 10^{-6} in a lifetime (Menichini et al., 2011, Kim et al., 2012, Marsili et al., 2014). On the other hand, a Norwegian study demonstrated that indoor air concentrations of benzo[a]pyrene (BaP) evaporated from rubber granulate in sports halls were lower than the indoor air concentration limit for BaP (NIPH, 2006).

In summary, the information on the relevance of evaporation of PAHs from rubber is inconclusive. Moreover, evaporation from artificial-turf fields is not a good a proxy for rubber tiles, because the former have a higher surface area to weight ratio, which heavily influences evaporation. For these reasons, we excluded the inhalation route (including the inhalation of particle-bound PAHs) from the present assessment; this may result in an underestimation of the exposure. Nevertheless, this underestimation is expected to be limited, since the six low volatile PAHs have not been detected in air above new or old rubber tiles. Furthermore, it is assumed that outdoors, PAH concentrations in the air will quickly be diluted to negligible concentrations.

[6] A temperature of 60°C was selected because Mattina et al. (2007) measured temperatures of 55°C when crumb rubber was directly exposed to sunlight at an exterior air temperature of 31°C, concluding that 60°C belongs to the reasonable temperature range accessible under field conditions.

2.2.2 *Oral exposure*

Oral exposure to PAHs from rubber tiles may occur due to hand-to-mouth transfer of PAHs present on the skin of hands after dermal exposure, and is calculated by multiplying the dermal exposure by a hand-to-mouth transfer fraction (see eq. 7). This exposure is taken into account in the current study. The internal oral exposure, required for the summation of different exposure routes, is calculated using an oral absorption fraction (see eq. 7). Subsequently, the results can be multiplied by the frequency of playground visits to derive the chronic internal exposure:

$$E_{int\ oral} = \frac{EA_{ext\ dermal,hands} * HMT * OA * F}{BW} \qquad \text{eq. 7}$$

$E_{int\ oral}$	chronic internal exposure due to oral exposure [g/kg bw/day]
$EA_{ext\ dermal,hands}$	external dermal exposure amount on hands [g]
HMT	hand-to-mouth transfer fraction [-]
OA	oral absorption fraction [-]
F	frequency of playground visits [day^{-1}]
BW	body weight [kg]

2.2.3 *Exposure to small pieces or particles*

Oral, dermal and inhalation exposure to PAHs from the tiles may also occur due to ingestion, adhesion to skin and inhalation of small pieces of rubber tiles which may be present due to wear and abrasion of the tiles. We did not consider these routes of PAH exposure because information on wear and abrasion is not available, neither is information on ingestion, adhesion to skin, and inhalation processes of small pieces of rubber. Exclusion of the exposure to small pieces and particles results in an underestimation of the exposure in the present assessment. This underestimation is probably small as it is assumed that damaged tiles will be quickly replaced.

3 Exposure assessment

3.1 Scenario description

In the reasonable worst-case scenario used for the exposure assessment, a child is assumed to visit a playground with rubber tiles containing PAHs for a few hours per day, on a number of days per year, from the age of 2 up to and including 12. This age range was selected since children in this age range start walking, visit playgrounds, and go to a day care centre or elementary school where playground equipment accompanied by rubber tiles are likely to be present. During these visits, the child can contact the rubber tiles with his/her bare hands, feet or legs. Due to hand-to-mouth behaviour, oral exposure to the PAHs is assumed to take place for children younger than 6 years. Inhalation of, dermal contact with, and the oral ingestion of small pieces of worn tiles were not included in the exposure assessment.

The exposure assessment is performed for the eight PAHs as defined in the REACH restriction for PAHs in accessible rubber or plastic parts in articles or toys (REACH PAH8, see Chapter 4). For the assessment of the PAH exposure from rubber tiles, three product concentrations were used: 8 mg PAH8/kg tile (product limit), 4 mg/kg tile and 0.8 mg/kg tile.

As described in Chapter 2, the diffusion approach was used to derive the dermal exposure, and in addition, the oral exposure due to hand-to-mouth contact was estimated. Calculation of the dermal exposure using the diffusion approach requires information on the time of contact with the tiles, the diffusion coefficient of PAHs in the tiles, the contact area, and the thickness of the tiles. The PAH concentration in mg/kg tile was transformed into g/m^3 using the tile's weight per surface area and the tile thickness. To calculate the oral exposure, the hand-to-mouth transfer was used. For estimation of the long-term internal exposure, the dermal and oral absorption fractions, the playground visiting frequency, and body weight are essential. The values selected for each of these parameters are described below.

3.2 Parameter values

3.2.1 Selection of parameter values

Since the objective of the current study is to perform a reasonable worst-case exposure assessment, we aimed at using 75^{th} percentiles from the parameter distributions rather than using maximum values or means. It is expected that in general, when using the 75^{th} percentiles of multiple uncorrelated parameters as input for a multiplicative model, (approximately) the 99^{th} percentile of exposure will be obtained. Choosing higher percentiles from each of the input data, such as a 90^{th} percentile, quickly leads to an unrealistic overestimation, and the effect of this accumulation of worst-case assumptions increases with the increasing number of input parameters (te Biesebeek et al., 2014). For this reason, (an estimate of) the 75^{th} percentile of the parameter's distribution was used. However, for some parameters, insufficient information was available about the distribution of the parameter

values. In those cases, we applied the maximum value (see also Table 16 in the discussion).

3.2.2 *Frequency and duration of playground visit*

The scenario describes children playing in a playground for a specific duration.
Table 1 gives an overview of the literature available on the duration and frequency of playground visits. The lack of recent data hampers an accurate estimation of frequency and duration of a playground visit, as we expect that children currently spend more time indoors than they did a decade ago. Despite the uncertainty, the available data indicate that a considerable proportion of children visit a playground one or multiple times per week, and a considerable number of these visits are for one or more hours. Based on this, we consider a frequency of playground visits for all age groups, including visits to playground equipment at schools and day care centres, of 5 days per week, with a duration of 2 hours per day per visit.

Table 1: Summary of literature about duration and frequency of playground visit (OEHHA, 2007)

Study	Age	Time and/or frequency of playground visit	Country
(Bjorklid-Chu, 1977)	1-15 years	"practically every day": 56% "About once a week": 27% "About once a month": 7% "Never": 7% "Don't know": 3%	Sweden
(Air Recources Board, 1991)	Under 12 years	An average of 49 minutes per day for those surveyed	US California
(Gallup, 2003) (as cited by OEHHA, 2007)	3-12 years	1) Daily or several times a week for 29% of those surveyed. 2) At least 1-2 hours per visit for 52% of those surveyed.	US National

On playground visiting days, dermal contact may occur to various parts of the body. For the present assessment, it is assumed that direct dermal contact is relevant for hands, legs and feet. We assume that throughout the year, children have dermal contact with rubber tiles with both bare hands, with a frequency of 5 days per week, resulting in 261 days/year. In addition, it is assumed that on days with a maximum temperature above 20°C, children play outside with bare feet and wearing shorts. This implies that on these days, not only is dermal contact with both bare hands possible, but also with bare feet and/or legs.
Table 2 shows the number of days per month in the past four years when the maximum temperature was above 20°C, in De Bilt, the Netherlands (KNMI, 2016); on average there were 93 days in the period April-September. Given a frequency of 5 days per week, it is assumed that children will be exposed via feet and legs on 66 days per year.

Table 2: Number of days per month where the maximum temperature was higher than 20°C, in the past 4 years, in De Bilt, the Netherlands (KNMI, 2016).

	Number of days >20° C				
	2016	2015	2014	2013	Average per year
April	1	3	8	4	
May	13	4	10	5	
June	18	14	19	12	
July	26	23	28	26	
August	27	27	18	30	
September	27	3	21	6	
Total	112	74	104	83	93

3.2.3 *Tile contact time with hand, feet and legs*

There is a lack of data on ground contact time with hands, feet and legs during playground visits. Studies on play-behaviour show a maximum contact of hands with the ground of 12-16 min/hour (Beamer et al., 2012, Auyeung et al., 2006) (see Table 3). For the exposure assessment, the 75th percentile of the study of Auyeung et al. was taken for the hand-tile contact time (7.2 min/hour) for all age groups. On days with temperatures above 20°C, for the legs-tile contact time, the assumption was made that tile-contact with legs is the same as with hands (7.2 min/hour). For the feet-tile contact time a (maximum) value of 30 min/hour is assumed. This assumption is based on the fact that a child does not always stand on both 2 feet for example during walking/running or may not be standing/walking on the rubber tiles at all.

Table 3: Hands to floor hourly contact duration (minutes per hour).

(Auyeung et al., 2006) children aged 1-6 years	
Both hands	Min/hour
Range	0-16.4
Mean	4
5th percentile	0.2
25th percentile	0.5
50th percentile	2.4
75th percentile	7.2
95th percentile	12.2
99th percentile	15.2
(Beamer et al., 2012) children aged 7-12 years	
Range	0-12.2
Mean	1.9
Median	0.9

3.2.4 *Body weight and contact area of feet hands, legs and feet*

Table 4 shows the 25th percentiles for the body weight in kg and surface area of relevant parts of the body in m^2. As a lower body weight will give a higher exposure, the 25th and not the 75th percentile is used. For surface area, the 25th percentile is also applied because surface area is proportional to body weight. Only part of these body surfaces can be in contact with the ground at the same time. However, there is a little information on which parts of these body-parts are actually in contact with the object, in this case the rubber tiles. Therefore, in the exposure

calculations in this report, as a worst-case assumption, the surface areas of the body parts are divided by two to obtain the contact surface areas. The anthropometric data apply to both genders. The approach then assumes an effective contact between tile and the skin for the contact time, as described in section 3.2.3.

Table 4: Body weight and contact areas of hands, legs and feet (te Biesebeek et al., 2014, Tables 18 and 31).

Age (year)	**Body weight (kg)**	**Surface area of relevant parts of the body (m^2)[a]**		
		Hands	Legs	Feet
1	9.8	0.027	0.109	0.030
2	12.4	0.027	0.144	0.036
3-6	15.7	0.033	0.176	0.044
6-11	24.3	0.046	0.256	0.062
11-16	44.8	0.064	0.421	0.095

[a] note that in the exposure calculations the areas are divided by 2, because only a part of the body parts can be in contact with the ground at the same time.

3.2.5 Tile thickness

Recycled car tyres are used for the production of rubber tiles. The tiles mainly consist of styrene-butadiene rubber (SBR), a general-purpose synthetic rubber produced from a copolymer of styrene and butadiene (Ropema-europe, 2016).

Rubber tiles are produced in different sizes and thickness (Table 5). The tile thickness correlates with the fall height. At playgrounds with slides or swings, there is a minimum need for rubber tiles with an appropriate fall height of at least one meter, even if the slide or swing is less than one meter high. Tiles with a greater thickness are needed when the fall height is higher (EN1177, 2008). The rubber tile data in Table 5 shows that a rubber tile thickness of 40 mm corresponds with a fall height of 1.3m or 1.4m depending on the information provided by the manufacturer. Therefore we chose a tile thickness of 40 mm for this parameter, assuming most playgrounds contain slides or swings with a minimum fall height of at least one meter (EN1177, 2008).
Table 5 shows that a rubber tile of 40 mm thickness (Ropema-europe, 2016) corresponds with a weight of 23 kg per m^2 which was used in the exposure assessment.

Table 5: Specifications of rubber tiles.

Size (mm)	**Thickness (mm)**	**HIC[a]/m (EN1177)**	**Weight/m^2 (kg)**	**Source**
500 x 500	25	1.0m	16	(Ropema-europe, 2016)
500 x 500	40	1.4m	23	(Ropema-europe, 2016)
500 x 500	45	1.6m	27	(Ropema-europe, 2016)
500 x 500	75	2.5m	37	(Ropema-europe, 2016)
500 x 500	80	2.6m	50	(Ropema-europe, 2016)
500 x 500	100	3.0m	57	(Ropema-europe, 2016)
500 x 500	45	-	24	(Rubbermagazijn, 2016)
1000 x 1000	65	-	31.5	(Rubbermagazijn, 2016)
500 x 500	40	1.3m	-	(Boer, 2016)

Size (mm)	Thickness (mm)	HIC[a]/m (EN1177)	Weight/m^2 (kg)	Source
500 x 500	45	1.5m	-	(Boer, 2016)
500 x 500	65	2.0m	-	(Boer, 2016)
500 x 500	80	2.6m	-	(Boer, 2016)
500 x 500	25	0.9m	19.2	(Gamma, 2016)
500 x 500	30	0.9m	18	(Rubberen-tegel.nl, 2016)
500 x 500	40	1.3m	22	(Rubberen-tegel.nl, 2016)
500 x 500	25	0.9m	16	(Rubbertegelwinkel, 2016)
500 x 500	25	0.9m	-	(Technoah, 2016)
500 x 500	45	1.6m	-	(Technoah, 2016)
500 x 500	60	1.9m	-	(Technoah, 2016)

[a] HIC=Head injury criterion (EN1177, 2008)

3.2.6 *Diffusion coefficient of PAHs in tile*

As mentioned above, rubber tiles are made of recycled car tyres and consist mainly of styrene-butadiene rubber (SBR) (Ropema-europe, 2016). Schwope and Goydan (1990), presenting diffusion coefficients as a function of molecular weight, estimated that the diffusion coefficient for PAHs at 25°C ranges from values of $6 \cdot 10^{-14}$ - $6 \cdot 10^{-13}$ m^2/s (low density polyethylene, LDPE), $6 \cdot 10^{-13}$ - $1 \cdot 10^{-11}$ m^2/s (rubber including SBR) and $6 \cdot 10^{-11}$ m^2/s (silicone rubber). Measurements of diffusion coefficients of PAHs at 20°C by Rusina et al. (2010) confirm the values for LPDE (10^{-13} - 10^{-12} m^2/s) and silicon rubber (10^{-11} - 10^{-10} m^2/s). For this reason, the values for SBR estimated by Schwope and Goydan (1990) are also considered reliable. The upper estimate of their range, (10^{-11} m^2/s) is used in the exposure calculations.

The diffusion coefficient (estimated at 25°C, Schwope and Goydan, 1990) increases exponentially with temperature (Schwope and Goydan, 1990). Tiles can be heated by the sun; a temperature of 55°C has been measured in rubber crumbs exposed to direct sunlight (Mattina et al., 2007)), so a higher diffusion coefficient and therefore higher dermal exposure will occur on warm, sunny days. This will especially affect the exposure to the feet and legs, because we assume dermal contact with legs and feet on days with a temperature above 20°C. For the exposure of the hands throughout the year, the varying diffusion coefficients due to low and high temperatures may average out.
Although the temperature dependency of the diffusion coefficient affects the dermal exposure, we did not take temperature dependency into account due to a lack of quantitative information. This probably has led to an underestimation of the dermal exposure in legs and feet.

The migration rate of PAHs from rubber to skin has been investigated using lipophilic matrices, e.g. vaseline, massage oil and Tenax (Hofstra, 2007, Fraunhofer, 2016), human and pig skin (Bartsch et al., 2016). Nevertheless, as information on the initial concentration in the product and time-dependency of the migration is not given in these studies, a diffusion coefficient for PAHs in rubber could not be derived from these data.

3.2.7 *Dermal absorption fraction*

Studies investigating the dermal absorption fraction of PAHs in animals and humans have used soil or a solvent like acetone or ethanol as

vehicle. Ruby et al. (2016) and Spalt et al. (2009) reviewed earlier investigations of dermal absorption of BaP from a different matrix, namely soil. Figure 1 shows an overview of all available *in vitro* and *in vivo* dermal absorption data in both animals and humans with the vehicle soil or solvent (acetone or ethanol) (see Appendix 1 for detailed information on the data). Dermal uptake of BaP/PAHs from soil appears to be lower compared to the situation when acetone or ethanol was used as a vehicle (Figure 1). In general, animal studies report percentages between 7-100% or 0-65% in solvent and soil respectively. Human studies report percentages between 4-78% or 0-27% in solvent and soil respectively (Figure 1). In the current assessment, it is assumed that after diffusion to the skin, the PAHs are present on the skin in an unbound state, i.e. not bound to soil, rubber or any other particles. Implicitly, it follows that absorption of unbound PAHs is more efficient compared to absorption of PAHs from soil, which first need to partition from the soil before they can be absorbed. Hence, the required absorption fraction is probably larger than those empirically derived with soil as vehicle. On the other hand, it is assumed that applying PAHs in the presence of a solvent enhancing the absorption, overestimates the required absorption fraction. This is in agreement with BAuA (2010), who report that the use of these highly lipophilic solvents may result in an overestimation of PAH migration rates. For this reason, an estimate of 20% for dermal absorption was used in the present report, which is smaller than most empirical findings in humans using a solvent and larger than most findings using soil as a vehicle (Figure 1).

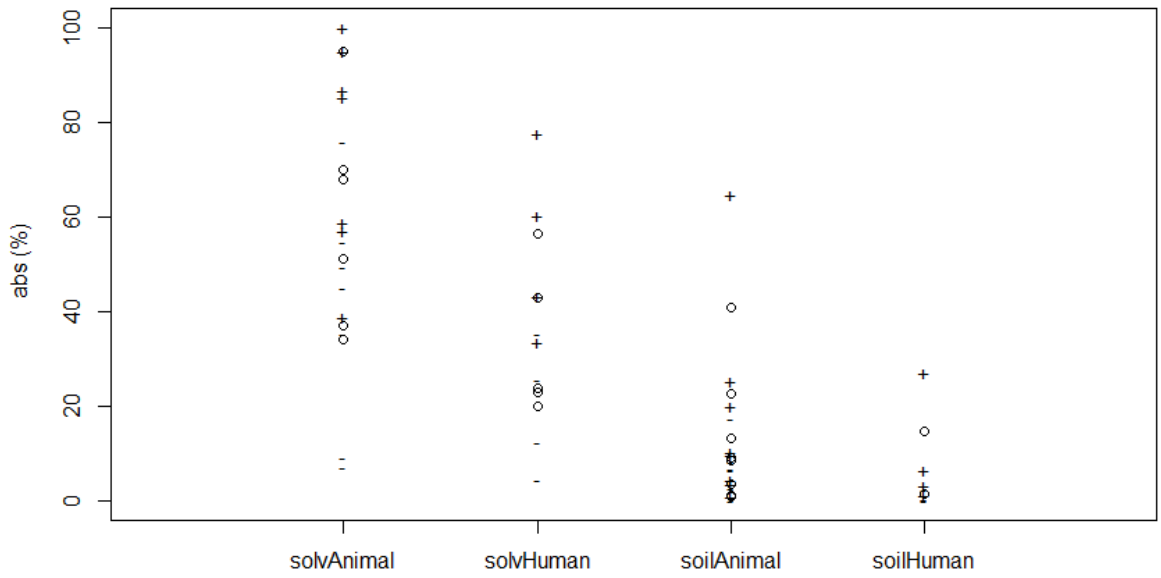

Figure 1: Dermal absorption data based on literature in vitro and in vivo data in soil or solvent (acetone / ethanol). Circles indicate mean, -/+ indicate reported minimum and maximum values or are an approximation of the range obtained by taking mean -/+ 2SD.

3.2.8 *Oral absorption fraction*

For experimental animals, the gastro-intestinal absorption of PAHs, especially BaP, is well documented. Absorption of (unbound) PAHs from the gastro-intestinal tract appears to vary per animal species. Table 6

provides an overview of studies on oral bioavailability of PAH in different species. Oral absorption of BaP was reported to be 35-99% in rats, 12% in goats and 30.5% in pigs. It is known that the use of rodent models for human exposure assessment is limited by the physiological differences between rodents and primates (Zhang et al., 2013). In fact, no single animal can mimic the gastro-intestinal tract characteristics of humans. However, pig and human colon morphology appears similar (Zhang et al., 2013, Kararli, 1995). Furthermore, in the pig study the PAHs were administered orally via milk, which is considered a relevant vehicle because it is likely that children playing outside are (semi-) fed rather than fasted. For these reasons, in this report an oral absorption fraction of 0.3 (30%) was assumed, based on the report by Cavret et al. (2003)

Table 6: Overview oral bioavailability studies.

PAH	Animal	Route of administration	Bioavailability %	Reference
BaP	Rat	Oral gavage	35 - 99%	(Ramesh et al., 2004) as cited by EFSA, 2008
Chrysene	Rat	Oral gavage	75 - 87%	(Ramesh et al., 2004)
BaP	Pig	Orally via milk	30.5%	(Cavret et al., 2003)
BaP	Goat	Oral gavage	12%	(Grova et al., 2002)
BaP	Rat	Intraduodenal infusion	30%	(Foth et al., 1988)
BaP	Rat	Oral gavage	10%	(Foth et al., 1988)
BaP	Rat	Oral gavage	40%	(Ramesh et al., 2001)

3.2.9 *Hand-to-mouth transfer*

Hand-to-mouth activity is an important contributor to child exposure to environmental contaminants (OEHHA, 2007). In our report, oral exposure via hand-to-mouth contact is assumed to be applicable for children up to and including 5 years old (Ter Burg et al., 2007).
Table 7 shows an overview of hand-to-mouth transfer efficiency studies, of which most were performed with adults. In the studies listed below, hand-to-mouth transfer ratios (i.e. the amount of –free, so not bound to particles- PAHs that is transferred from the hand to the mouth divided by the total amount, expressed as percentage) are between 10-56% for adults, and 3-53% for children.

A hand-to-mouth transfer efficiency value of 50% has been used by other agencies in Europe and the US. In Europe this value is used in the admittance of biocides (ECHA, 2016) as well as in the exposure assessment of these substances (Bremmer et al., 2006). The U.S. Consumer Product Safety Commission (CPSC, 1997) used a value of 50% when estimating oral exposure via hand-to-mouth contact to children's PVC products (Sahmel et al., 2015). The U.S. EPA Office of Pesticide Programs used 50% as a default value for estimating hand-to-mouth exposure to pesticides (US EPA, 2001). An exposure assessment of wood preservatives by the California Department of Health Services used 50% for arsenic, chromium and copper (Sahmel et al., 2015).

OEHHA (2007) provides a hand-to-mouth transfer of 50% based on the (US EPA, 2001) standard value of 50% for hand-to-mouth transfer efficiency. Since specific information on the hand-to-mouth transfer of PAHs is not available, considering the data above, the value of 50 % for hand-to-mouth transfer is used in the present study.

Table 7: Overview of hand-to-mouth transfer efficiency studies.

Test substance	Population	Hand-to-mouth transfer efficiency	Reference
Liquids (vinegar)	Adults	36.8% (± 31.9)	(Gorman Ng et al., 2014)
Powders (calcium acetate and magnesium carbonate)	Adults	56.6% (± 42.2)	(Gorman Ng et al., 2014)
Powders	Adults	10.1-21.9% (median 15.9%)	(Gorman Ng et al., 2014)
Biological substances	Adults	33.9-41.0% (median 34%)	(Gorman Ng et al., 2014)
Lead	Adults	24% (range 12-34%)	(Sahmel et al., 2015)
Soil	Adults	10.1% (range 8.7-11.8%) (thumb sucking) 15.9% (range 13.8-18.4%) (finger mouthing)	(Kissel et al., 1998)
Riboflavin	College-aged	34% (range 0.7-34%)	(Cohen Hubal et al., 2005)
Dust	Toddler Child Teen Adult Senior	53% ± 101 41% ± 82 2.8% ± 5.2 3.3% ± 5.8 3.4% ± 6.1	(Wilson et al., 2013)

3.3 Exposure assessment

3.3.1 Summary of selected parameters for exposure assessment

Tables 8 and 9 present a summary of all the parameters used in the dermal and oral exposure assessment of PAHs from rubber tiles (diffusion approach) in the present study.

Table 8: Input parameters for the dermal and oral exposure calculation.

Parameter	Abbreviation	Value	Unit	Reference
General/dermal exposure				
Concentration[a]	C	8; 4; 0.8	mg/kg	Current report[b]
Duration of playground visit		2	h/day	(BAuA, 2010)
Product mass per surface area		23	kg/m^2	(Ropema-europe, 2016)
Tile thickness		4	cm	(Ropema-europe, 2016)
Diffusion coefficient in product	D	1×10^{-11}	m^2/s	(Rusina et al., 2010)
Dermal absorption fraction	DA	0.2		Current report, based on Ruby et al. (2016) and Spalt et al. (2009)
Hands				
Frequency of playground visit with hand-ground contact	F	261 / 365	day^{-1}	Current report, based on (Gallup, 2003)
Hand-ground contact time	CT	7.2	min/h	(Beamer et al., 2012, Auyeung et al., 2006)
Legs				
Frequency of playground visit with leg-ground contact	F	66 / 365	day^{-1}	Current report
Leg-ground contact time	CT	7.2	min/h	Current report
Feet				
Frequency of playground visit with feet-ground contact	F	66 / 365	day^{-1}	Current report
Feet-ground contact time	CT	30	min/h	Current report
Oral exposure				
Oral absorption fraction	OA	0.3		(Cavret et al., 2003)
Hand-to-mouth transfer[c]	HMT	50	%	(OEHHA, 2007, US EPA, 2001)

[a] sum of eight REACH PAHs
[b] As described in the assignment by the Ministry of Public Health, Welfare and Sports
[c] Only used for the age group 2-6 years

Table 9: Body weight and contact areas of hands, legs and feet (te Biesebeek et al., 2014, Tables 18 and 31).

Age (year)	**Body weight (kg)**	**Contact area of relevant parts of the body (m^2)**		
		Hands	legs	Feet
2	12.4	0.014	0.072	0.018
3 to 6	15.7	0.017	0.088	0.022
6 to 11	24.3	0.023	0.128	0.031
11 to 13	44.8	0.032	0.211	0.048

3.3.2 Exposure assessment

Using the parameter values presented in Tables 8 and 9, the diffusion model was applied to calculate the external dermal exposure amount after contact with rubber tiles containing 0.8, 4 and 8 mg PAH8/kg tile. This calculation was performed for hands, legs and feet separately because the contact area and contact time vary with the body parts. Similarly, the calculations were performed for each age (group) separately because of the varying contact areas with age.
The internal dose due to dermal exposure was derived using equation 3. The internal dose due to oral exposure after hand-to-mouth contact was derived using equation 7, based on the parameters presented in Tables 8 and 9. The oral exposure for children aged 6 or more is zero, because for these ages, hand-to-mouth contact is assumed to be absent. The total chronic internal dose was derived by summing the internal doses from dermal hand, leg and feet and from oral exposure. Subsequently, the age-weighted mean internal daily dose for over the ages 2 to 13 was derived. All results are reported in Table 10. From this table it can be concluded that dermal exposure is more important (a factor of 5 higher) than oral exposure for those age groups where hand-to-mouth contact is assumed. Furthermore, within the dermal exposure pathway, the exposure of legs and feet is higher than that of hands (a factor of 3).

Table 10: Estimation of exposure using the diffusion approach.

Tile concen-tration (mg/kg)	**Age (year)**	**External dermal exposure amount ($EA_{ext\ dermal}$, mg)**			**Dermal chronic (internal) dose (mg/kg bw/day)**			**Oral chronic (internal) dose (mg/kg bw/day)**	**Total chronic (internal) dose (mg/kg bw/day)**
		Hands	Legs	Feet	hands	Legs	feet		
0.8	2	$5.6 \cdot 10^{-6}$	$2.9 \cdot 10^{-5}$	$3.1 \cdot 10^{-5}$	$6.5 \cdot 10^{-8}$	$8.4 \cdot 10^{-8}$	$8.9 \cdot 10^{-8}$	$4.8 \cdot 10^{-8}$	$2.9 \cdot 10^{-7}$
	3 to 6	$6.8 \cdot 10^{-6}$	$3.5 \cdot 10^{-5}$	$3.7 \cdot 10^{-5}$	$6.2 \cdot 10^{-8}$	$8.1 \cdot 10^{-8}$	$8.6 \cdot 10^{-8}$	$4.6 \cdot 10^{-8}$	$2.8 \cdot 10^{-7}$
	6 to 11	$9.2 \cdot 10^{-6}$	$5.1 \cdot 10^{-5}$	$5.3 \cdot 10^{-5}$	$5.4 \cdot 10^{-8}$	$7.6 \cdot 10^{-8}$	$7.8 \cdot 10^{-8}$	0	$2.1 \cdot 10^{-7}$
	11 to 13	$1.3 \cdot 10^{-5}$	$8.4 \cdot 10^{-5}$	$8.2 \cdot 10^{-5}$	$4.1 \cdot 10^{-8}$	$6.8 \cdot 10^{-8}$	$6.6 \cdot 10^{-8}$	0	$1.7 \cdot 10^{-7}$
	2 to 13								$2.3 \cdot 10^{-7}$ [a]
4	2	$2.8 \cdot 10^{-5}$	$1.4 \cdot 10^{-4}$	$1.5 \cdot 10^{-4}$	$3.2 \cdot 10^{-7}$	$4.2 \cdot 10^{-7}$	$4.4 \cdot 10^{-7}$	$2.4 \cdot 10^{-7}$	$1.4 \cdot 10^{-6}$
	3 to 6	$3.4 \cdot 10^{-5}$	$1.8 \cdot 10^{-4}$	$1.8 \cdot 10^{-4}$	$3.1 \cdot 10^{-7}$	$4.1 \cdot 10^{-7}$	$4.2 \cdot 10^{-7}$	$2.3 \cdot 10^{-7}$	$1.4 \cdot 10^{-6}$
	6 to 11	$4.6 \cdot 10^{-5}$	$2.6 \cdot 10^{-4}$	$2.6 \cdot 10^{-4}$	$2.7 \cdot 10^{-7}$	$3.8 \cdot 10^{-7}$	$3.8 \cdot 10^{-7}$	0	$1.0 \cdot 10^{-6}$
	11 to 13	$6.4 \cdot 10^{-5}$	$4.2 \cdot 10^{-4}$	$4.0 \cdot 10^{-4}$	$2.0 \cdot 10^{-7}$	$3.4 \cdot 10^{-7}$	$3.2 \cdot 10^{-7}$	0	$8.7 \cdot 10^{-7}$
	2 to 13								$1.1 \cdot 10^{-6}$ [a]
8	2	$5.6 \cdot 10^{-5}$	$2.9 \cdot 10^{-4}$	$3.1 \cdot 10^{-4}$	$6.5 \cdot 10^{-7}$	$8.4 \cdot 10^{-7}$	$8.9 \cdot 10^{-7}$	$4.8 \cdot 10^{-7}$	$2.9 \cdot 10^{-6}$
	3 to 6	$6.8 \cdot 10^{-5}$	$3.5 \cdot 10^{-4}$	$3.7 \cdot 10^{-4}$	$6.2 \cdot 10^{-7}$	$8.1 \cdot 10^{-7}$	$8.6 \cdot 10^{-7}$	$4.6 \cdot 10^{-7}$	$2.8 \cdot 10^{-6}$
	6 to 11	$9.2 \cdot 10^{-5}$	$5.1 \cdot 10^{-4}$	$5.3 \cdot 10^{-4}$	$5.4 \cdot 10^{-7}$	$7.6 \cdot 10^{-7}$	$7.8 \cdot 10^{-7}$	0	$2.1 \cdot 10^{-6}$
	11 to 13	$1.3 \cdot 10^{-4}$	$8.4 \cdot 10^{-4}$	$8.2 \cdot 10^{-4}$	$4.1 \cdot 10^{-7}$	$6.8 \cdot 10^{-7}$	$6.6 \cdot 10^{-7}$	0	$1.7 \cdot 10^{-6}$
	2 to 13								$2.3 \cdot 10^{-6}$ [a]

[a] age-weighted mean

4 Hazard assessment

4.1 PAH marker groups

PAHs constitute a large class of organic compounds composed of two or more fused aromatic rings. They are primarily formed by incomplete combustion, pyrolysis of organic matter, and during various industrial processes. PAHs generally occur in complex mixtures, which may consist of hundreds of compounds. PAHs are generally regarded as being potentially genotoxic and carcinogenic to humans (IARC, 2010, IPCS, 1998).

A main issue in the risk assessment of PAHs is the quantification of the carcinogenic potency of PAH mixtures. The composition of the mixtures encountered in food, consumer products and the environment varies, resulting in varying carcinogenic potencies. Benzo[a]pyrene (BaP) has been used as a marker of occurrence and effect of the carcinogenic PAHs in food, based on examinations of PAH profiles in food and on evaluation of a carcinogenicity study of two coal tar mixtures in mice (Culp et al., 1998). Based on the two experiments with coal tar by Culp et al., the total carcinogenic potency of PAH mixtures is related to their content of BaP (as marker). Nevertheless, in 2008, EFSA concluded that BaP is not a suitable indicator for the occurrence of PAHs in food and thus for exposure, and expanded the marker method from one (BaP) to four markers, the so-called EFSA PAH4 (Table 11), and to eight markers (EFSA PAH8). EFSA concluded that EFSA PAH4 and EFSA PAH8 are both suitable indicators of PAHs in food, with EFSA PAH8 not providing much added value compared to EFSA PAH4.

Table 11: Overview of PAH marker groups.

EFSA PAH4	EFSA PAH8	REACH PAH8	CAS
Benzo[a]pyrene	Benzo[a]pyrene	Benzo[a]pyrene	50-32-8
Benz[a]anthracene	Benz[a]anthracene	Benz[a]anthracene	56-55-3
Benzo[b]fluoranthene	Benzo[b]fluoranthene	Benzo[b]fluoranthene	205-99-2
Chrysene	Chrysene	Chrysene	218-01-9
	Benzo[k]fluoranthene	Benzo[k]fluoranthene	207-08-9
	Dibenz[a,h]anthracene	Dibenz[a,h]anthracene	53-70-3
	Benzo[ghi]perylene		191-24-2
	Indeno[1,2,3-cd]pyrene		193-39-5
		Benzo[e]pyrene	192-97-2
		Benzo[j]fluoranthene	205-82-3

In a similar fashion, BAuA argued that consumers will often be exposed to a mixture of many PAHs at different relative proportions, depending on the material used in the production of the respective consumer article. They assume that by setting limits for both BaP and the sum of all of the eight known carcinogenic congeners, a good part of the compositional variability is covered. The eight PAHs proposed by BAuA as PAH8 are classified carcinogens of category 1B (EC, 2008).

Benzo[a]pyrene and chrysene are also classified mutagens of category 1B and 2, respectively (EC, 2008).

The REACH PAH8 group was adopted the European Commission in the restriction of PAHs (EC, 2013), which regulates the maximum levels of PAHs permitted in consumer articles. Note that six of the eight PAHs from the groups from EFSA and REACH are the same, but two differ.

4.2 Carcinogenicity of REACH PAH8

Experimental data on the toxicity of the REACH PAH8 group of ECHA are not available, while the toxicity of the EFSA PAH8 group can be assessed using the data from Culp et al. (1998). Note that from Culp et al. (1998), only the total carcinogenicity of the PAH mixture is known at the applied concentrations of the PAHs. In the marker approach, this total carcinogenicity is assumed to correspond with a specific (e.g. PAH4 or PAH8) marker group. When applying the EFSA PAH8 group, it is possible to calculate the sum of the doses of the eight EFSA PAHs, and relate this summed dose to an effect. However, two of the eight PAHs from the REACH PAH8 group are not measured in the Culp et al.'s mixture (1998). Hence, to assess the carcinogenicity of the REACH PAH8 in the rubber tiles, we assumed that the concentrations of the deviating two PAHs benzo[e]pyrene and benzo[j]fluoranthene were present in the mixtures applied by Culp in similar concentrations to benzo[ghi]perylene and indeno[1,2,3-cd]pyrene[7]. In this case, the dose-response (and hence the $BMDL_{10}$) will be the same, regardless of the choice of PAH8 group. Subsequently, the toxicity of EFSA's PAH8 group can be applied in the current assessment to estimate the extra cancer risk of exposure to the REACH PAH8 from rubber tiles. As mentioned above, Culp et al. do not report benzo[e]pyrene and benzo[j]fluoranthene concentrations in the mixtures used. However, it is unclear if they attempted to identify these two PAHs at all. For this reason, a direct verification of the reliability of this assumption is not possible.

Information of the concentrations of PAHs in two other coal tar pitch mixtures is available (EU, 2008). This document shows that concentrations of REACH PAH benzo[e]pyrene in the coal tar pitch mixtures (11,891 and 8,976 mg/kg) are similar to concentrations of EFSA PAHs benzo[ghi]perylene (11,106 and 9,061 mg/kg) and indeno[1,2,3-cd]pyrene (9,945 and 8,664 mg/kg) in these mixtures. Unfortunately, the REACH PAH benzo[j]fluoranthene was not analysed. Nevertheless, the available information indicates that the assumption on similar concentrations of the deviating PAHs in the two PAH8 groups is defendable.

4.3 Carcinogenicity of PAHs in rubber tiles

With the assumed carcinogenic potency of the REACH PAH8 (see the section above) at the concentrations in the coal tar mixtures assessed by Culp et al. (1998), the next step in the hazard assessment is to consider the composition of the PAH mixture in the rubber tiles. The

[7] Note that it is not necessary to assume that these substances have a similar potency and contribution to the carcinogenic potency, because the potency of the entire mixture is considered. When marker PAHs are exchanged, the BMD(L) stays the same if the concentrations of marker PAHs are the same.

marker method can be applied to mixtures, provided that the composition of the mixture is similar to the composition of the coal tar applied in Culp's two animal experiments (1998). Available PAH4 concentrations measured in rubber tiles (RIVM, 2013) indicate that the mixture composition of PAHs in these tiles is indeed similar to the PAH4 and partial PAH8 mixtures applied in the animal experiments (as all PAH4 substances overlap with the PAH8 group by EFSA), based on the ratios to BaP content (Table 12). For this reason the EFSA PAH4 marker method was used in a previous RIVM report on PAHs in rubber tiles (2013) (see section 6.4.3 for more information). Nevertheless, as our study is on the REACH PAH8, because this is in line with the existing legislation on PAHs in consumer articles (EC, 2013), the PAH8 marker method will be used. For this, it is assumed that the remaining four PAHs are also present in the tile in the same ratio as noted in the toxicity study. Data from the Dutch Food and Consumer Product Safety Authority (NVWA, 2014) on five REACH PAHs in eight different rubber tiles, indicate that for benzo[k]fluoranthene, the ratio to BaP (n=1) is similar to that of Culp's study. For benzo[a]anthracene and benzo[b]fluoranthene the ratio is lower than in the mixtures of Culp, while for chrysene the ratio is dependent on the scenario assumed for the non-detects (Table 12). Consequently, the assumption that the composition of tiles is similar to that of the coal tar mixtures used by Culp can only be partially confirmed.

Based on the two animal studies with coal tar, EFSA derived an external oral $BMDL_{10}$ (the dose at which 10% of the study animals get a tumour) for the EFSA PAH8 of 0.49 mg/kg bw/day. As explained above, this is also the value we applied for the REACH PAH8 in the rubber tiles. Subsequently, we assumed the oral absorption in mice to be similar to that in rat, and selected a reasonable worst-case percentage of 30% (see Table 6). Applying these values, the internal $BMDL_{10}$ of the PAH8 in the rubber tiles is 0.15 mg/kg bw/day. The internal BMDL is required to enable comparison of the BMDL with the internal dose calculated from the dermal exposure using the diffusion approach, and the oral exposure resulting from hand-to-mouth contact.

Table 12: PAH mixture composition in rubber tile and toxicity study.

		PAH	Mixture in tile (RIVM, 2013)		Mixture in tile (NVWA, 2014)		Ratio to BaP in study of Culp (EFSA, 2008, Table 27)	
			Conc. (mg/kg)	Ratio to BaP	Mean conc.[a] (mg/kg)	Mean ratio to BaP[b]	Mixture 1	Mixture 2
EFSA PAH8		BaP	101	-	4.2	-	-	-
	EFSA PAH4	Chrysene	133	1.3	2.8	0.5/1.0[c]	1.3	1.1
	EFSA PAH4	Benzo[a]anthracene	121	1.2	2.2	0.6/0.6	1.3	1.2
	EFSA PAH4	Benzo[b]fluoranthene	144	1.4	2.7	0.6/0.7	1.1	1.0
	EFSA PAH4	Benzo[k]fluoranthene	ND		1.6	0.3/0.3[c]	0.38	0.37
		Benzo[ghi]perylene	ND				0.81	0.83
		Dibenz[a,h]anthracene	ND				0.74	0.72
		Indeno[1,2,3-cd]pyrene	ND				0.15	0.13

ND: not determined

[a] calculated assuming non-detects having a concentration equal to the limit of quantification.

[b] calculated leaving out the one tile without any positive concentrations, for two scenarios: assuming non-detects having a concentration equal to the limit of quantification and assuming non-detects having a concentration of 0 mg/kg.

[c] n=1 (the other measurements were non-detects).

5 Risk assessment

5.1 Estimating the extra cancer risk of PAH

At this point, we have estimated the total exposure (Chapter 3) and the dose of PAH8 at which 10% of the study animals develop a tumour ($BMDL_{10}$; Chapter 4), both expressed as an internal exposure. The next step, performed in this chapter, is the estimation of the extra cancer risk of PAH8 at the estimated internal exposure level. To achieve this, the $BMDL_{10}$ needs to be extrapolated to the risk following from the dose level calculated in Chapter 3. For this, two approaches were applied: linear extrapolation and model extrapolation. In both approaches, the extra cancer risk of exposure to PAH8 from rubber tiles during childhood are expressed as the extra risk during a lifetime.
As requested by VWS, the Margin of Exposure (MoE) was also calculated. The MoE is the ratio between the $BMDL_{10}$ and exposure, and should be at least 10,000 to be considered as "of low concern from a public health point of view" according to the EFSA guidance (EFSA, 2005). The MoE does not provide any information on the extra risk, but merely indicates whether the margin between exposure and hazard is sufficiently large. The results are presented in Appendix 2.

5.2 Linear extrapolation

In linear extrapolation, it is assumed that the ratio between the BMDL and the actual exposure equals the ratio between the extra risk corresponding to the BMDL and the extra risk corresponding to the actual exposure. When the BMDL, the extra risk corresponding to the BMDL and the exposure are known, then the extra risk of the exposure can be derived. The linear extrapolation is implemented according to:

$$\frac{BMDL}{expo} = \frac{ER_{BMDL}}{ER_{expo}} \qquad \textit{eq. 8}$$

where BMDL is the internal BMDL (of 0.15 mg/kg bw/day). ER_{BMDL} is the extra risk corresponding to the BMDL, i.e. 10%. ER_{expo} is the (to be calculated) extra risk corresponding to an internal exposure (expo). Route-to-route extrapolation (from oral-to dermal) is accounted for by using the internal BMDL and internal exposure. To correct for differences in metabolic rates between mice and humans, an allometric scaling factor (AS) of 7 is applied, following the REACH Guidance (ECHA, 2012). According to this guidance, no other adjustments are required to account for possible interspecies and intraspecies differences because these are sufficiently covered by the large high dose-low dose extrapolation (see section 6.4.1 for discussion). The less than lifetime exposure is accounted for by multiplying the exposure with the number of years with exposure (YE, 11 years) divided by the number of expected life years (YL, 70 years). Applying the factor YE/YL implies a linear relationship between risk and exposure duration, which is considered acceptable for carcinogens (Felter et al., 2011, Bos et al., 2004).

Equation 8 is amended as follows:

$$\frac{\frac{BMDL}{AS}}{\frac{YE}{YL}*expo} = \frac{ER_{BMDL}}{ER_{expo}} \qquad \textit{eq. 9}$$

This can be rewritten to derive the extra risk (eq. 10), or to derive the exposure corresponding to a fixed extra risk (eq. 11):

$$ER_{expo} = \frac{AS*YE*expo*ER_{BMDL}}{BMDL*YL} \qquad \textit{eq. 10}$$

$$expo = \frac{BMDL*YL*ER_{expo}}{AS*YE*ER_{BMDL}} \qquad \textit{eq. 11}$$

5.3 Model extrapolation

In the model extrapolation approach, a fitted dose response curve is used to estimate the extra risk at the exposure. In short, the human exposure is converted to an equivalent animal exposure. This animal exposure is put into the (animal) dose-response curve and the corresponding extra risk is derived. This approach is based on the model extrapolation approach used in the integrated probabilistic risk assessment (IPRA) of carcinogens (Slob et al., 2014, Slob et al., 2011). In a full probabilistic assessment, the variation in exposure and sensitivity between individuals and the uncertainties of all aspects of the risk assessment are quantified, resulting in detailed information on the population's risk and the uncertainties thereof. In the current assessment, only the uncertainty in the toxicological data is quantified and propagated, resulting in confidence intervals for the risk. The variation and uncertainties in the exposure are accounted for by using reasonable worst-case estimates.

The model extrapolation approach requires (1) the fitted dose response curve and (2) an animal equivalent of the human exposure as an input for the dose response curve. EFSA determined that the multistage (two-stage) model (eq. 12) resulted in the lowest BMDL of 0.49 mg/kg bw/day. This model:

$$y = a + (1-a)\left(1 - \left(e^{-\left(\frac{x}{b}\right)-c\left(\frac{x}{b}\right)^2}\right)\right) \qquad \textit{eq. 12}$$

where x is the (animal) dose and y is the fraction of tumour bearing animals, was refitted to the data (Table 13) using PROAST version 62.8 running in R version 3.2.0 (PROAST, 2016, R Core Team, 2016) to confirm that the correct data and model were used (see Figure 2 left panel). When fitting the model to the tumour incidence against the external dose, the same BMD and BMDL reported by EFSA (2008) of 0.97 and 0.49 mg/kg bw/day respectively, were obtained.

The dose response curve was also fitted on the internal dose (Figure 2 right panel), assuming a 30% oral absorption in mice (see section 4.3 for explanation). This resulted in a BMD and BMDL of 0.29 and 0.15 mg/kg bw/day, respectively. The internal dose response curve is needed because the diffusion approach produces an internal human exposure to compare with (section 2.1.3). The parameter values (*a*, *b* and *c*) of the

two-stage model were obtained. Omitting the background fraction (parameter *a*) gives the function describing the extra risk:

$$ER_{expo} = 1 - \left(e^{-\left(\frac{x}{b}\right)-c\left(\frac{x}{b}\right)^2}\right) \qquad \textit{eq. 13}$$

The dose response data were bootstrapped 10,000 times to obtain multiple sets of parameter values, enabling quantification of the uncertainty in the extra risk due to the uncertainty in the toxicity data. Similar to equation 11, the exposure corresponding to a fixed extra risk was calculated. This was done by optimizing x in equation 13 so that the fixed ER_{expo} was reached. Subsequently, the concentration of PAHs in tile corresponding to the obtained exposure (x) could be derived (see below).

The human (internal) exposures need to be adjusted to an equivalent animal dose, by multiplying (rather than dividing) the human exposure by the allometric scaling factor and a factor for the 'less than lifetime exposure' in humans. Then, the extra risk can be obtained from the animal dose response curve (eq. 13). Again, to correct for differences in metabolic rates between mice and humans, an allometric scaling factor of 7 was applied, following REACH Guidance (ECHA, 2012). The less than lifetime exposure is accounted for by multiplying the human exposure with the number years with exposure (YE), divided by the number of expected life years (YL).

Table 13: dose-response data of coal tar mixtures in mice from Culp (1998) as reported in Schneider (2002) and EFSA (2008).

External PAH8 dose (mg/kg bw/day)	Internal PAH8 dose (mg/kg bw/day)*	Number of tumour bearing animals	Sample size	Mixture number
0	0	5	48	1
0.181	0.0543	12	48	1
0.537	0.1611	14	48	1
1.81	0.543	12	48	1
5.37	1.611	40	48	1
0.771	0.2313	17	48	2
2.8	0.84	23	48	2
7.71	2.313	44	48	2

*derived by assuming a 30% oral absorption

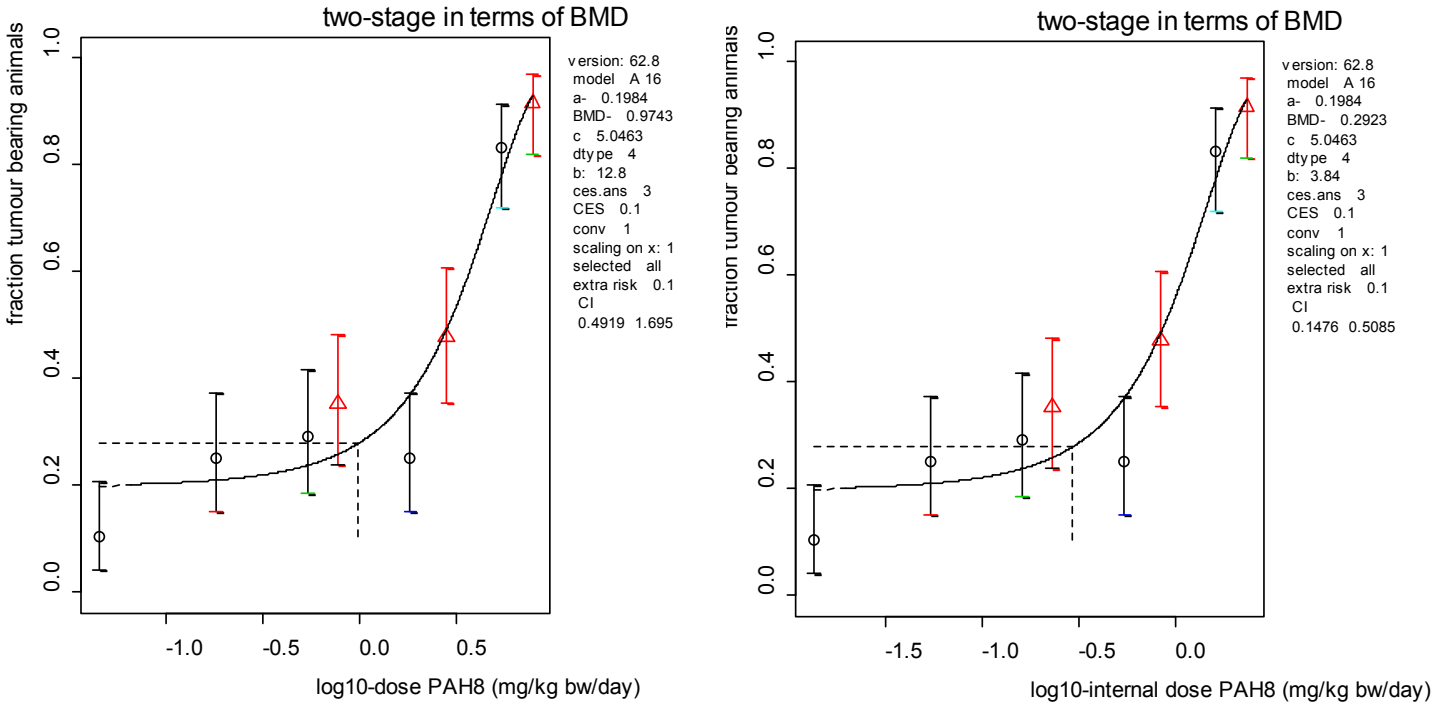

Figure 2: Fits of the two-stage model (eq. 12) to the fraction of tumour bearing animals against the (log_{10}) external dose (left panel) and the (log_{10}) internal dose (right panel). Whiskers indicate 95% confidence intervals. Black circles and red triangles are obtained with mixture 1 and 2, respectively. Horizontal dashed line indicates the 10% extra risk. The vertical dashed line indicates the corresponding BMD.

5.4 Calculation of extra risk

Table 14 presents the extra cancer risk of exposure to PAHs from rubber tiles in playgrounds. The inverse of the risk is given between brackets, i.e. the lifetime extra cancer incidence of one case per number of exposed individuals. The extra risks corresponding to three product concentrations are presented, namely the limit value of 1 mg PAH/kg rubber tile, the limit value for toys of 0.5 mg PAH/kg tile, and one arbitrarily chosen concentration of 0.1 mg PAH/kg for each of the eight PAHs. The exposure was calculated using the diffusion approach (Table 10).

The extra risks were calculated using the linear (eq. 10) and model (eq. 13) extrapolations. Linear extrapolation provides a worst-case estimate of the risk. The model extrapolation indicates the uncertainty due to the uncertainty of the risk at low doses. Therefore, the risk is reported as a confidence interval with its lower 5th and upper 95th confidence limits. Overall, the upper confidence limits of risk obtained with the model extrapolation are the same as the risk obtained using the linear extrapolation. This latter feature is a logical consequence of extrapolation to low risks (see Figure 3 and Slob et al., 2011, Slob et al., 2014).

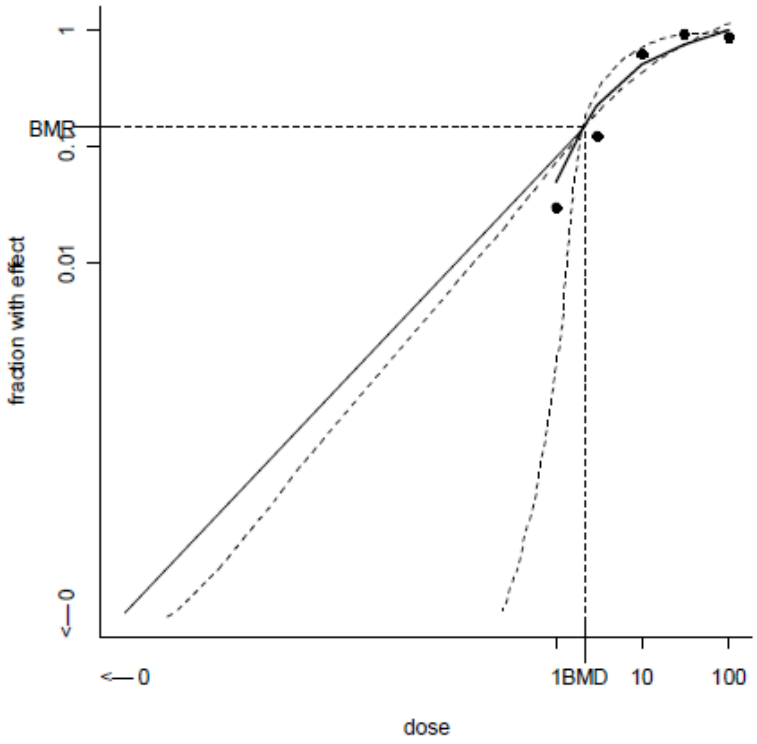

Figure 3: Illustration of the fact that the lower confidence limit for the dose at lower risks for the model extrapolation approach is similar to that for the linear extrapolation approach, while the upper bound for the model extrapolation approach may be much higher. The model extrapolation approach allows dose-response curves such as the two dashed curves, reflecting two possible dose-response relationships that are compatible with the observed responses, but differ widely at lower risks levels. However, they remain on the right side of the solid line that reflects linear extrapolation.

From Table 14, it can be read that a product concentration of 1 mg PAH/kg tile for the eight REACH PAHs results in a maximum extra risk of $1.7 \cdot 10^{-6}$ (linear extrapolation), and it is likely that the extra risk is between $1.3 \cdot 10^{-7}$ and $1.7 \cdot 10^{-6}$ (model extrapolation).
The current examples (0.8, 4 and 8 mg/kg tile) show a linear decrease in risk with decreasing exposure. This is to be expected from the linear extrapolation. The risk obtained using model extrapolation follows the (non-linear) dose-response curve (eq. 12 and Figure 2). Therefore, risks at other (higher) exposures cannot be simply derived by multiplication, instead equation 13 should always be applied when using model extrapolation.

Table 14: Rounded lifetime extra cancer risk of exposure to PAHs from rubber tiles in playgrounds. See text for further explanation.

	Extra risk (1 case per number of persons)		
Product concentration	0.8 mg/kg	4.0 mg/kg	8.0 mg/kg
Linear extrapolation	$1.7 \cdot 10^{-7}$ (5,800,000)	$8.2 \cdot 10^{-7}$ (1,200,000)	$1.7 \cdot 10^{-6}$ (580,000)
Model extrapolation			
LCL	$1.3 \cdot 10^{-8}$ (77,000,000)	$6.2 \cdot 10^{-8}$ (16,000,000)	$1.3 \cdot 10^{-7}$ (7,700,000)
UCL	$1.7 \cdot 10^{-7}$ (5,900,000)	$8.2 \cdot 10^{-7}$ (1.200,000)	$1.7 \cdot 10^{-6}$ (590,000)

LCL: 5% lower confidence limit
UCL: 95% upper confidence limit

By applying equation 11 and optimizing equation 13, the exposure corresponding to a particular extra risk can be derived. Assuming a fixed ratio between product concentration and exposure (from Table 10, for each approach and scenario), the product concentrations related to the specified extra risk were derived[8]. These concentrations are listed in Table 15. Again, using the model extrapolation provides the uncertainty of the product concentration leading to a particular extra risk. For example, from Table 15 it can be read that a product concentration of 4.7 mg PAH8/kg or higher results in an extra risk of one in a million (linear extrapolation). The model extrapolation approach indicates that it is likely that an extra risk of one in a million will be reached at a product concentration between 4.6 and 64 mg PAH8/kg (model extrapolation).

Table 15: Product concentrations corresponding to fixed lifetime extra risks.

Extra risk (1 per 10^{-x})	**Exposure (µg PAH8/kg bw/day)**		**Product (tile) concentration (mg PAH8/kg)[1]**	
Linear extrapolation				
10^{-4}	$1.3 \cdot 10^{-1}$		470	
10^{-5}	$1.3 \cdot 10^{-2}$		47	
10^{-6}	$1.3 \cdot 10^{-3}$		4.7	
10^{-7}	$1.3 \cdot 10^{-4}$		0.47	
Model extrapolation				
	LCL[2]	UCL[3]		
10^{-4}	$1.3 \cdot 10^{-1}$	1.7	460	6000
10^{-5}	$1.3 \cdot 10^{-2}$	$1.8 \cdot 10^{-1}$	46	640
10^{-6}	$1.3 \cdot 10^{-3}$	$1.8 \cdot 10^{-2}$	4.6	64
10^{-7}	$1.3 \cdot 10^{-4}$	$1.8 \cdot 10^{-3}$	0.46	6.4

[1]the total concentration of the 8 PAHs; for the limit per individual PAH this value needs to be divided by 8.
[2]LCL: 5% lower confidence limit
[3]UCL: 95% upper confidence limit

[8] All approaches to derive the exposure are linear, e.g. a ten-fold lower product concentration results in a ten-fold lower exposure.

6 Discussion

6.1 Introduction

For the assessment of the extra cancer risk of the REACH PAH8 from rubber tiles, we selected a method for dermal exposure assessment. For this method 'reasonable worst-case' parameter values were obtained from the literature and by expert judgement. In addition to dermal exposure, we accounted for oral exposure due to hand-to-mouth contact. For the hazard assessment, assumptions were made regarding the composition of the PAH mixture and relative concentrations of the PAHs in the tiles to be able to use the carcinogenicity information from the EFSA PAH8 for the REACH PAH8 present in the tiles. Subsequently, the risk was assessed for REACH PAH8 in rubber tiles at concentrations at the product limit (1 mg PAH/kg tile) and two lower concentrations, for a scenario in which children from the age of 2 up to and including 12, play in playgrounds five days a week, for two hours a day. We used the linear extrapolation method according to the REACH Guidance (ECHA, 2012) and an additional approach in which a part of the uncertainty of the risk could be quantified, namely the model extrapolation method. The calculated extra cancer risk lies between 1 per 590,000 and 1 per 7.7 million individuals who play(ed) on rubber tiles.

6.2 Exposure assessment

6.2.1 Selection of method

Of the three available approaches for dermal exposure assessment, the diffusion approach was selected, as this approach is considered to most accurately describe the processes involved in the availability of a substance for dermal exposure after dermal contact with a contaminated product. The other two approaches, the ECETOC approach and the migration approach, make use of a hypothetical layer in the tile from which the migration takes place. The ECETOC approach assumes that all PAHs present in the layer are available for skin contact, whereas the migration approach assumes that only the part of the PAHs released over the contact time can migrate from that layer. Although the definition of a layer in the tile is a convenient way of limiting the exposure, the actual thickness of a hypothetical layer cannot be verified. In addition, the two approaches mentioned above are based on the ECETOC TRA method, which aims at a conservative estimation, and therefore an overestimation, of the dermal exposure, while the diffusion approach aims at a realistic estimate of the exposure.

The diffusion approach is a conceptual model which considers the emission of the substance from the matrix as being driven by the diffusion to the surface.
Note that, also for the diffusion model, it is not known whether the model concept is valid, i.e. whether 1) the PAHs directly transfer from the surface of the tile to the skin and only to skin (no evaporation) when there is skin-tile contact and 2) subsequently a fraction of the PAHs is dermally absorbed (the remainder of the PAHs is removed or at least not available for absorption). Nevertheless, the diffusion model gives, to the

best of our knowledge, the best description of the physical processes occurring during exposure.

6.2.2 *Pathways of exposure*

From Table 10 it follows that the dermal pathway is more important than the oral one (factor of 5 higher). Other pathways were not considered in the current assessment: The inhalation of evaporated and particle-bound PAHs was not estimated and nor did we investigate the oral and dermal exposure to small pieces of rubber which may come off due to wear and abrasion of the tiles (see section 2.2). Ignoring these exposure pathways may have led to an underestimation of the exposure. However, the ingestion, inhalation of rubber pieces or particles, and dermal contact with rubber particles is expected to be unlikely, as tiles will be replaced when they are damaged. Nevertheless, we recommend that this expectation be confirmed.

Inhalation of evaporated PAHs from the tiles is assumed to be small (see section 2.2).
In conclusion, the underestimation of the risk due to excluding these pathways is likely to be small.

6.2.3 *Uncertainty in the exposure assessment*

Parameter values were selected to provide a reasonable worst-case rather than an overly conservative estimate of the exposure. Thus, when information was available on the range or the distribution of values, we estimated a realistic (75^{th} percentile) value for the parameter, whereas we used a maximum reported value if the available data were insufficient for estimating the actual distribution, which is common practice in risk assessment. When data were especially scarce, we made a reasonable worst-case estimate by expert judgement. For a few parameters (product information on the tile, hand-ground contact time, dermal and oral absorption fraction), we found sufficient data to derive a realistic estimate (see Table 16). For all other parameters, maximum reported values or conservative expert judgement (when possible in combination with data) was applied. Nevertheless, the exposure may have been underestimated for several reasons. The first is the fact that inhalation and wear/abrasion of tiles is ignored, leading to an expected small underestimation of the exposure (see 6.2.2). Secondly, we ignored the temperature dependency of the diffusion coefficient of PAHs in tiles. This may have led to a considerable underestimation of the dermal exposure to legs and feet (see section 3.2.6), whereas this route is the dominant exposure route (factor 3 higher than dermal exposure to hands, and a factor 4 higher than oral exposure). For this reason, we recommend refining the exposure estimation with a temperature dependent diffusion coefficient.

Due to time constraints, a sensitivity analysis of the parameters was not performed in the present study. Nevertheless, in our opinion, more detailed information on the behaviour of children (frequency and duration of playing in playground, feet/leg contact time with tile) and on the (temperature-dependent) diffusion coefficient of PAHs in the tiles, will lead to a more accurate exposure estimate.

Table 16: Type of estimation (estimate of 75th percentile/maximum reported value/conservative expert judgement) for input parameters for the exposure assessment.

Parameter	Value	Unit	Type of estimation	Comment
General/dermal exposure				
Concentration	8; 4; 0.8	mg/kg	-	Assigned by Ministry of Health Welfare and Sports (VWS)
Duration of playground visit	2	h/day	Combination of data and expert judgement	
Product mass per surface area	23	kg/m^2	Estimate of 75th percentile	
Tile thickness	4	cm	Estimate of 75th percentile	
Diffusion coefficient in product	1 x 10^{-11}	m^2/s	Max reported value	
Dermal absorption fraction	0.2		Combination of data and expert judgement	Absorption data in human skin; from soil (not from solvent)
Hand				
Frequency of playground visit with hand-ground contact	261 / 365	day^{-1}	Combination of data and expert judgement	
Hand-ground contact time	7.2	min/h	P75	
Contact area	0.014-0.032	m^2	Expert judgement	Based on 50% of total area
Leg				
Frequency of playground visit with leg-ground contact	66 / 365	day^{-1}	Combination of data and expert judgement	
Leg-ground contact time	7.2	min/h	Expert judgement	Based on hand-ground contact time
Contact area	0.072-0.211	m^2	Expert judgement	Based on 50% of total area
Feet				
Frequency of playground visit with feet-ground contact	66 / 365	day^{-1}	Expert judgement	
Feet-ground contact time	30	min/h	Expert judgement	
Contact area	0.018-0.048	m^2	Expert judgement	Based on 50% of total area
Oral exposure				
Oral absorption fraction	0.3		Realistic	Based on pig data
Hand-to-mouth transfer	50	%	Expert judgement	Common approach for children 0-3 years

6.3 Hazard assessment

6.3.1 REACH PAH8 and EFSA PAH8

In this study, we consider the REACH PAH8 to be in line with the existing restriction on PAH-concentrations in consumer articles (EC, 2013). The REACH PAH8 contains two PAHs that are not included in the EFSA PAH8 (and vice versa). Only for the latter group of PAHs the carcinogenicity has been studied. To be able to apply the carcinogenicity information of the EFSA PAH8 to the REACH PAH8, we assumed that the concentrations of the deviating two 'REACH PAHs' were present in the mixtures used in the toxicity study in similar concentrations to those of the two 'EFSA PAHs'. In this case, the toxicity of EFSA's PAH8 mixture can be applied to estimate the extra cancer risk of the REACH PAH8. The validity of this assumption cannot be verified, since the study by Culp et al. (1998) does not give information on the presence of the two 'REACH PAHs'. Although there is some information available on the composition of coal tar mixtures confirming the assumptions made, this is not sufficient to actually verify the assumption on the carcinogenicity of the REACH PAHs. Hence, this is an uncertainty of an unknown magnitude and direction, leading to an over- or an underestimation of the risk.

Rather than making the assumption on the carcinogenicity of the REACH PAHs described above, we would have used relative potency factors[9]. However, although it is known that the carcinogenic potency of different PAHs can have a large variation (IPCS, 1998), carcinogenicity studies with individual PAHs are scarce, except for BaP. Hence, relative potency factors for the individual PAHs have not been established (EFSA, 2008, WHO, 2006). Strictly speaking, the cancer risk of exposure to PAH mixtures with a different composition from the PAH mixtures found by Culp et al. (1998) cannot be assessed. It is remarkable that for a group of substances so ubiquitous and infamous for their carcinogenicity as PAHs are, the knowledge base on the dose-response on carcinogenicity is so small.

6.3.2 Composition of PAHs in tiles

The marker method (see section 4.1) can be applied to mixtures, provided that the composition of the mixture is similar to the composition of the coal tar applied in the two animal experiments by Culp et al. (1998). In the previous RIVM report (2013), the mixture composition of EFSA PAH4 in the rubber tiles was shown to be similar to the mixtures applied in the animal experiments. Data on eight tiles measured by the NVWA only partially confirm this similarity. For this reason the assumption that the eight PAHs are also in the tile in the same ratio as in the toxicity study is uncertain, and the actual carcinogenic potency form the PAH mixture in the tile could be either higher or lower.

[9] Relative potency factor: The ratio of the toxic potency of a given chemical to that of an index chemical in the group. Relative potency factors are used to convert exposures of all chemicals in the group into their exposure equivalents of the index chemical.

6.4 Risk assessment

6.4.1 Selection of assessment factors in linear extrapolation

For the linear extrapolation, the REACH Guidance (ECHA, 2012) was followed, stating that only one assessment factor is needed, namely for the possible difference in metabolic rate (allometric scaling) between mice and humans (factor of 7). The Guidance indicates that other interspecies and intraspecies differences are sufficiently covered by the large high dose-low dose extrapolation. Nevertheless, the latter can be disputed:

Firstly, in a standard carcinogenicity study, rodents are exposed starting from the age of 6-8 weeks[10], corresponding with a human age of around puberty. As a consequence, the study does not give information on the effect of the substance at a lower age. To take this uncertainty into account, the United States Environmental Protection Agency (US EPA, 2005) and the Office of Environmental Health Hazard Assessment (OEHHA, 2009) apply an age dependent adjustment factor (ADAF) in the estimation of the extra cancer risk with the linear extrapolation method. Values for the ADAF are preferably substance-specific. However, by default a factor of 10 is used for 0-2 year olds, a factor of 3 for the 2-16 year olds and a factor of 1 for individuals older than 16 years. Nevertheless, presently, in the Netherlands and in Europe there is no general agreement on the use of an ADAF for the early life stages. For this reason an ADAF was not applied in the current risk assessment. Furthermore, it can be argued that the high dose-low dose extrapolation only corrects for the level of the risk (from 10% to 1 per one million), and not for the possible intra- and interspecies differences. In this view, rather than solely applying an assessment factor of 7 for metabolic rate, an additional factor for (remaining) toxicokinetics and toxicodynamics (e.g. a factor of 3) and for intra-species differences (e.g. a value of 10) [11] should be used, in concordance with risk assessments for non-carcinogenic substances.

EFSA also has taken this issue into consideration in their opinion on the MoE (EFSA, 2005) and concludes that the usual default factor for inter- and intra-species differences of 10×10 for non-genotoxic substances would also be relevant for substances which are both genotoxic and carcinogenic. According to EFSA, these default factors could be reduced or increased when appropriate chemical specific data are available.

When additional assessment factors would be used in the present cancer risk assessment, consequently, the estimated risk would be higher than the currently derived risk. The application of additional assessment factors is also the approach used in the integrated probabilistic risk assessment (IPRA) of carcinogens (Slob et al. 2014), (see the next section for more information on IPRA).

[10] Note that at the start of the carcinogenicity study of Culp et al. (1998) the mice were five weeks old.
[11] This factor of 10 is assumed to also include possible age dependent differences.

Because of the known qualitative and quantitative differences in PAH metabolism between species and within humans, in addition to possible other inter-individual variation, e.g. in formation and repair of PAH-DNA adducts (see e.g. EFSA, 2008, WHO, 2006, WHO, 2010), we recommend to initiate a discussion in the Netherlands and Europe on introducing additional assessment factors into the cancer risk assessment of PAHs, with the aim to establish these values in legislation or guidance.

6.4.2 *Uncertainty in high dose-low dose extrapolation*

Previous work on carcinogenic substances in food shows that the estimation of extra risks at low doses is a major source of uncertainty (Slob et al., 2011, Slob et al., 2014). Because the traditional linear extrapolation method does not allow for a quantification of uncertainty, Slob et al. (2011; 2014) introduced the model extrapolation method in the integrated probabilistic risk assessment (IPRA) of carcinogens. In IPRA, variability and uncertainty in all parameters of the full risk assessment (i.e. exposure and hazard assessment) are taken into account. For the extrapolation of cancer risk to low doses, this means that the uncertainty in the extrapolation of the fitted dose response curve is propagated in the result of the assessment. We did not perform a full IPRA as the population variability in the exposure could not be quantified, but we conducted a small aspect of an IPRA (use of the model extrapolation with only one model), yielding a quantified uncertainty for the low dose extrapolation.

The linear extrapolation method provides a worst-case estimate of the extrapolated risk, while in the model extrapolation method, the uncertainty of one selected dose-response model (the multi- or two-stage model) is propagated, resulting in a range of the estimated extra risk. Note that the risks within this range are equally probable; the upper confidence level calculated with the model extrapolation method is similar to the worst-case estimate of the linear extrapolation method. The uncertainty in the extra risk, calculated as the ratio of the upper and lower confidence level of the results of the model extrapolation, equals an order of magnitude. Other models are available which also provide an adequate description of the dose-response (EFSA, 2008). Due to limited time, these models were not included in the current analysis. Based on the previous work with carcinogenic substances (Slob et al., 2011, Slob et al., 2014), it is expected that including multiple models would show that the uncertainty due to high dose-low dose extrapolation is even larger.

6.4.3 *Comparison with RIVM assessment in 2013*

In a previous report, RIVM (2013) assessed the extra cancer risk associated with the occurrence of EFSA PAH4 in rubber tiles at the product limit of 1 mg PAH/kg tile in playgrounds. The approach considered the diffusion of the PAHs in the rubber tile and the subsequent transfer to human skin in contact with the tile. The exposure estimate, based on worst-case assumptions for child behaviour and contact with rubber tiles using the linear extrapolation method, resulted in estimations of the extra cancer risk of 2.2 per million (RIVM, 2013).

It is difficult to compare the outcomes of the two studies because different scenarios and different parameter values have been used. For

example, in RIVM (2013), due to the short-term incidence, the exposure duration was much shorter (i.e. 6 months), while the contact area was assumed to be higher, resulting in a relatively high extra risk estimate.

A better option for comparison of the two methods applied in the two studies is to apply the exposure scenario of the current study described in Chapter 3 to EFSA PAH4 rather than to REACH PAH8. In this case, the extra risk of exposure to rubber tiles with a concentration of 1 mg PAH4/kg tile is 1.2 per million, which is approximately a factor of 2 lower than in the RIVM 2013 calculation. In conclusion, the exposure assessment of the current report is less conservative than that of RIVM 2013 despite the longer exposure duration in the current study.

6.4.4 Remaining issues

It should be noted that the product limit of 1 mg PAH/kg product is valid for *all* plastic and rubber articles (EC, 2013). Thus, because the contact with rubber tiles presently yields an estimated extra cancer risk in the order of the negligible risk level of 1 per million, it should be kept in mind that additional exposure to PAHs from other articles and exposure from other sources (baked or smoked food, inhalation of polluted air, etc.) will lead to an increased risk.

In addition, we would like to emphasize that the current study assesses whether the product limit of PAHs in rubber tiles provides an adequate level of protection. Hence, we did not investigate whether concentrations in rubber tiles currently present at playgrounds are within this limit. From the measurements on rubber tiles and other flooring for playgrounds by the NVWA (2014), it appears that rubber tiles do not always comply to this limit.

7 Conclusions and recommendations

The results of the risk assessment indicate that at the product limit of 1 mg PAH/kg tile, the extra cancer risk of exposure to the eight REACH PAHs from rubber tiles lies between 1 per 590,000 and 1 per 7.7 million individuals who regularly play(ed) on these tiles. The upper limit in this range is close to (but just exceeds) the generally accepted extra cancer risk of 1 per million during a lifetime. Nevertheless, the current risk assessment includes uncertainties, both in the direction of over-estimating and underestimating the risk. Uncertainties are present in all three parts of the assessment, i.e. exposure (Chapter 3), hazard (Chapter 4) and the methodology of risk assessment (Chapter 5).

Whereas the exposure scenario is considered as being conservative (with the exception of ignoring two different pathways of exposure), not taking into account of the temperature dependency of the diffusion coefficient is not. The latter may lead to a considerable underestimation of the exposure of the legs and feet. For the hazard assessment, as explained in section 6.3, the uncertainties are in both directions. Hence, the conclusion was drawn that the hazard could be over- or underestimated. The uncertainty in the risk assessment (Chapter 5) can be divided in the uncertainty in the application of assessment factors (see section 6.4.1) and in the high dose-low dose extrapolation (see section 6.4.2). The uncertainty in the latter was partly quantified, which is the only quantification of uncertainty in the present assessment. The final risk assessment does not include assessment factors for interspecies differences other than the difference in metabolic rates (by the allometric scaling factor) and neither does it include an assessment factor for intra-species differences. At this moment, there is no agreement within Europe on the use of additional safety factors for cancer-causing substances.

Since the presently calculated risk can be slightly higher than the negligible risk level and, moreover, the assessment contains uncertainties, we see the following possibilities to refine the current assessment. Firstly, this can be achieved by collecting more information on the exposure parameters. The collection of more information (especially on the playing behaviour of children –frequency, duration and contact time-, the transfer of PAHs from product surface to the skin and a temperature-dependent diffusion coefficient of PAHs in tiles, in combination with a sensitivity analysis to obtain information on those parameters having the largest influence on the exposure. In addition, the uncertainty can be reduced by investigating the exposure from pieces of worn tiles to verify the assumption that this exposure pathway is negligible. Refining the hazard of (individual) PAHs present in the tiles will also reduce the uncertainty, but would take much effort. Furthermore, we recommend initiating a discussion, preferably at the European level, on including assessment factors for interspecies differences (other than metabolic rate) and intra-species differences in the estimation of the extra cancer risk by PAHs. Lastly, we recommend performing a full IPRA, following the approach by Slob et al. 2014, to

obtain quantitative information on the uncertainty of the total risk assessment.

Finally, since the product limit is valid for all plastic and rubber articles, and the contact with rubber tiles yields an estimated extra cancer risk just above 1 per million, additional exposure to PAHs from other consumer articles will lead to a higher extra cancer risk.

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Appendix 1 Dermal absorption fractions

Animal	Vehicle	Value %	Reference
Human skin	Acetone	23.7% ± 9.7%	(Wester et al., 1990)
Rhesus monkey	Acetone	51%(± 22)	(Wester et al., 1990)
Hairless guinea pig	Acetone	37% (± 0.9)	(Ng et al., 1992)
Human Skin	Acetone	56.4%(±10.59)	(Moody et al., 2007)
Human Skin	Ethanol	20%	(Bartsch et al., 2016)
In vitro (rat, hairless guinea pig,human; Testskin;human **In vivo rat and guinea pig**	Acetone, according to abstract of ref #10	In Vitro: 95± 9.6% (rat), 51±3.0% (hairless guinea pig), 43 ± 8.7% (human; 50-year-old) 34±12.4% (Test skin); 23±5.3% (human; 32-year-old) In Vivo: 70±7.6% (rat) and 68±9.3% (guinea pig)	(Moody et al., 1995)
Human skin	Soil	1.4 % ± 0.9	(Wester et al., 1990)
Rhesus monkey	Soil	13.2% ± 3.4	(Wester et al., 1990)
Human Skin	Soil	14.8% ± 6.17	(Moody et al., 2007)
In vitro human skin	Soil	Between ~0.3% and ~1.1%	(Roy and Singh, 2001)
Pig skin	Sand or Clay, pure BaP	9.0 ± 0.4 – 22.7 ± 1.3	(Abdel-Rahman et al., 2002)
In vitro human skin	Soil. aged	0.14 - 1.1%	(Stroo et al., 2005)
In vitro pig skin	Pure, soil and aged soil	Pure: 76±3.2 Soil:8.5±0.9 Soil : 3.5±0.5 Aged soil: 3.7±0.5 Aged soil:1.8±0.2	(Turkall et al., 2010)
In vitro human skin	Soil	0.2-6.5%	(Roy et al., 1998)
In vitro and in vivo rat	1) BaP in crude petroleum 2) Soil fortified with BaP in crude petroleum	In Vitro, @24h 1) ~12% 2) ~1% In Vivo, @24h 1) 5.5% (se=1.4) 2) 1.1% (se=0.3)	(Yang et al., 1989)
Vitro human (n=14) and guinea pig (n=5) skin	Sediment	Guinea pig: Naphthalene: 59±15.5 Phenanthrene:62±6.5 BaP: 41±11.9 Human: Phenanthrene:14±6.6	(Moody et al., 1995)

Appendix 2 Margin of Exposure (MoE)

Calculation of MoE
The extra cancer risk of PAHs from rubber tiles was estimated with the linear and model extrapolations as presented Chapter 5. It has been argued that the extrapolation to low risks is scientifically unjustified (O'Brien et al., 2006, Barlow et al., 2006, EFSA, 2005). Therefore, the Margin of exposure (MoE) approach was introduced for genotoxic carcinogens. In this approach, the point of departure (e.g. $BMDL_{10}$) is divided by the human exposure, i.e. the margin is derived between the human exposure and the dose which causes a 10% extra risk in experimental animals. In contrast to the linear and model extrapolation, the MoE approach does not provide an explicit quantitative conclusion about the risk. EFSA (2005) suggests that the MoE should be at least a factor of 10,000 to be considered as "of low concern from a public health point of view".

In Table 2.1 the MoEs for the various tile concentrations are provided considering the internal $BMDL_{10}$ of 0.15 mg/kg bw/day.

Table 2.1: MoE for cancer risk of PAH8 from rubber tiles.

Tile concentration (mg/kg)	Total chronic (internal) dose (mg/kg bw/day) (weighted mean)	MoE
0.8	$2.3 \cdot 10^{-7}$	640,000
4	$1.1 \cdot 10^{-6}$	130,000
8	$2.3 \cdot 10^{-6}$	64,000

MoE vs. low dose extrapolation
The calculated MoEs are all above the value of 10,000 which is generally regarded by EFSA as 'of low concern'. Hence, from this point of view additional steps are not needed. The fact that the conclusion based on the MoE differs from that of the risk assessment using the extrapolation methods is, however, not contradictory, because the two methods are very different. Whereas the extrapolation method calculates the risk at low doses, the MoE approach considers that cancer risk cannot be calculated, since an extrapolation of about five orders of magnitude is needed. The MoE is just a measure of the distance between the point of departure (in this case $BMDL_{10}$) and the estimated human exposure, and does not give quantitative information on the cancer risk (Slob et al., 2014) .

Slob et al. (2014) showed that 1) the cancer risk estimate resulting from linear extrapolation in some cases cannot even be regarded as conservative and 2) ranking substances according to their point estimates for the MOE, ignoring the uncertainties in those estimates, may be misleading. These authors prefer the MoE method above the linear extrapolation method, but recommend performing an IPRA approach (applying the model extrapolation method), so that the uncertainty of the extra risk can be quantified. In line with this, in the

current risk assessment, we value the estimated extra cancer risk by the model extrapolation method above that of the MoE.

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