ANNEX XV RESTRICTION REPORT

PROPOSAL FOR A RESTRICTION

BENZO[A]PYRENE
CAS No: 50-32-8
EINECS No: 200-028-5

BENZO[E]PYRENE
CAS No: 192-97-2
EINECS No: 205-892-7

BENZO[A]ANTHRACENE
CAS No: 56-55-3
EINECS No: 200-280-6

DIBENZO[A,H]ANTHRACENE
CAS No: 53-70-3
EINECS No: 200-181-8

BENZO[B]FLUORANTHENE
CAS No: 205-99-2
EINECS No: 205-911-9

BENZO[J]FLUORANTHENE
CAS No: 205-82-3
EINECS No: 205-910-3

BENZO[K]FLUORANTHENE
CAS No: 207-08-9
EINECS No: 205-916-6

CHRYSENE
CAS No: 218-01-9
EINECS No: 205-923-4

CONTACT DETAILS OF THE DOSSIER SUBMITTER:

Bundesanstalt für Arbeitsschutz und Arbeitsmedizin (BAuA)
Anmeldestelle Chemikalien / Zulassungsstelle Biozide
(Federal Institute for Occupational Safety and Health
Division for Chemicals and Biocides Regulation)
Friedrich-Henkel-Weg 1-25
4149 Dortmund (Germany)

fax: +49(231)9071-2679
e-mail: chemg@baua.bund.de

VERSION NUMBER: 1 DATE: [31/05/2010]

TABLE OF CONTENTS

List of Tables

List of Figures

PROPOSAL FOR A RESTRICTION

A. Proposal

A.1 Proposed restriction(s)

A.1.1 The identity of the substances

Chemical Name: Benzo[a]pyrene
EC Number: 200-028-5
CAS Number: 50-32-8

Chemical Name: Benzo[e]pyrene
EC Number: 205-892-7
CAS Number: 192-97-2

Chemical Name: Benzo[a]anthracene
EC Number: 200-280-6
CAS Number: 56-55-3

Chemical Name: Dibenzo[a,h]anthracene
EC Number: 200-181-8
CAS Number: 53-70-3

Chemical Name: Benzo[b]fluoranthene
EC Number: 205-911-9
CAS Number: 205-99-2

Chemical Name: Benzo[j]fluoranthene
EC Number: 205-910-3
CAS Number: 205-82-3

Chemical Name: Benzo[k]fluoranthene
EC Number: 205-916-6
CAS Number: 207-08-9

Chemical Name: Chrysene
EC Number: 205-923-4
CAS Number: 218-01-9

A.1.2 Scope and conditions of restriction(s)

It is suggested to add the following text to entry No. 50 of Annex XVII of the REACH regulation[1]:

50. Polycyclic aromatic hydrocarbons (PAH) 1. Benzo[a]pyrene (BaP) CAS No 50-32-8 2. Benzo[e]pyrene (BeP) CAS No 192-97-2 3. Benzo[a]anthracene (BaA) CAS No 56-55-3 4. Chrysene (CHR) CAS No 218-01-9 5. Benzo[b]fluoranthene (BbFA) CAS No 205-99-2 6. Benzo[j]fluoranthene (BjFA) CAS No 205-82-3 7. Benzo[k]fluoranthene (BkFA) CAS No 207-08-9 8. Dibenzo[a,h]anthracene (DBAhA) CAS No 53-70-3	5. Articles which could be used by consumers (including articles in contact with the oral mucosa, toys, and childcare articles) shall not be placed on the market, if they contain any of the PAHs listed in column 1 at levels above the limit of quantitation (LOQ). Currently this LOQ is located at 0.2 mg/kg for any of the listed PAHs when measured with the method ZEK 01-08 as issued by the German 'Zentralstelle der Länder für Sicherheitstechnik' on December 3, 2008. If such articles consist of more than one component, the above limit applies to any particular component of an article containing PAHs for which direct contact with skin and/or mucous membranes is foreseeable. The latter also includes exposure by inhalation or the oral route. The Commission shall re-evaluate at five-year intervals the state of the art in chemical analysis methodology routinely available to monitoring authorities in the European Union. The above LOQ shall then be adapted to the technical progress, if necessary. 6. Paragraph 5 shall apply beginning from… *(DD/MM/YYYY)*

[1] It is noted that the current entry already consists of four sub-sections. It is therefore suggested to add the proposed restriction text starting with point no. 5. Please note also that the text describing the proposed method of chemical analysis is reproduced in Appendix 7 (German version). A detailed summary in English will be made available in the accompanying IUCLID dossier.

A.2 Summary of the justification

A.2.1 Identified hazard and risk

A.2.1.1 Description of and justification for targeting of the information on hazard and exposure

This restriction proposal is limited to consumer articles, i. e. articles 'that could be used by consumers'[1] (Art. 68 (2) of the REACH regulation), also referred to as 'consumer products' in this dossier. Single substances or PAH-containing mixtures that could be used by consumers are not addressed by this proposal.

A.2.1.2 Description of the risk to be addressed by the proposed restriction

A.2.1.2.1 Information on hazard(s)

General information on hazard with regard to human health

Consumer products containing one or more polycyclic aromatic hydrocarbons (PAHs) as listed in entry 50 of Annex XVII of the REACH regulation (Reg. (EC) 1907/2006) are considered severely hazardous based on their carcinogenic and mutagenic properties, as well as their potential for being toxic to reproduction.

All eight PAHs discussed in this dossier are classified carcinogens of category 2 (DSD) or 1B (CLP), respectively. Benzo[a]pyrene (DSD Cat. 2) and chrysene (DSD Cat. 3) also are legally classified mutagens. Lack of classification for the other congeners does not necessarily reflect absence of genotoxicity, but may rather be attributed to the comparatively limited database available for these compounds.

The focus of this dossier is placed on the carcinogenicity of BaP and the other PAHs under question. The observed mutagenic properties by themselves do not allow for a quantitative risk characterisation but are considered in a qualitative way, i. e. in establishing the mechanism behind PAH carcinogenicity.

The potential of at least BaP to cause toxicity to reproduction is noted. However, for this type of toxicity usually threshold dose levels can be assumed, below which no substance-related adverse effects are expected. Consequently, it was considered unlikely that the resulting DNELs for reproduction toxicity would fall below the DMELs proposed in this dossier on the basis of non-threshold carcinogenicity. Therefore reproduction toxicity was not evaluated further in this dossier.

It is noted that with respect to carcinogenicity, children have to be considered a particularly sensitive sub-population, both in terms of an inherent greater sensitivity and their longer remaining life-span (increasing the statistical risk for the development of cancer following exposure towards PAHs).

[1] Substances for which restriction is proposed may occur in consumer products defined as ‘article’ in REACH Art. 3(3). The use of CMR substances as such or in preparations (according to REACH Art. 3(2)) placed on the market for sale to the general public is restricted by entry 28 to 30, Annex XVII, REACH regulation.

A number of comprehensive toxicological evaluations of PAHs is available in the published literature. Therefore many other aspects of PAH toxicity have not been treated in-depth or even were omitted in this dossier, if they were not considered relevant for its purpose.

Evaluation of the database of animal tests regarding the dermal absorption of BaP resulted in the establishment of rough working estimates of 50 and 20 % from acetone or aqueous media (such as sweat), respectively. Likewise, coarse grain estimates of 50 % each were used to cover absorption along the oral route and by inhalation.

Derivation of DMELs for carcinogenicity

From a set of available animal studies selected by specified criteria, DMELs were calculated following the scheme as provided in the REACH IR/CSA guidance on dose-response assessment (R.8). For each of the selected studies (where appropriate) T25, BMD_{10}, and $BMDL_{10}$ estimates were used as dose descriptors. For all of these descriptors (thus, for each of the studies) DMELs were calculated applying both the 'Large Assessment Factor' and the 'Linearised' approach (the latter at both the 10^{-5} and 10^{-6} risk levels and using the 'Probit' as well as the 'Multistage Cancer' algorithms for curve fitting).

Instead of taking the most sensitive DMEL from a single study forward to quantitative risk characterisation, the whole range of DMELs using different approaches were accounted for.

The following DMEL results ranges were obtained:

Large Assessment Factor approach: 0.1 – 30 ng/kg bw/d

Linearised approach, 10^{-5} risk level: 0.03 – 10 ng/kg bw/d

Linearised approach, 10^{-6} risk level: 0.004 – 1 ng/kg bw/d

The boundaries of these ranges roughly represent the results from the most (lower boundaries) and least (upper boundaries) sensitive studies, respectively.

A.2.1.2.2 Information on levels and exposure of PAHs from consumer goods

A.2.1.2.2.1 PAH levels in consumer goods

The present dossier is based on the evaluation of more than 5300 samples from consumer articles analysed for their PAH content. The samples analysed covered a multitude of different consumer products, which – based on similar exposure patterns - were subdivided into the following eight broader categories:

1. Electrical devices
2. Grips/handles
3. Skin contact areas of sports equipment or other consumer products
4. Toys
5. Materials with close contact to the body
6. Other products with skin contact
7. Tyres and rolls, and
8. Other products.

Calculated over all product groups, in 91.9 % of the cases BaP was not detectable and in 95 % of all samples the concentration was below 1 mg/kg. The corresponding values were 83.9 % not detectable, 90.7 % of the values below 1 mg/kg for the sum of the 6 EU-PAHs contained in the EPA-PAH list ('PAH-6'). However, detected levels vary considerably between different product groups.

In the remaining samples, the highest PAH levels found were 1200 mg/kg for BaP, 25400 mg/kg for the sum of all EPA-PAHs and 6930 mg/kg for the sum of PAH-6.

Taking all data together, the results clearly show that consumer products may contain high amounts of polycyclic hydrocarbons. On the other hand, these data demonstrate that levels of BaP and of PAH-6 above LOQ in consumer products are technically avoidable. In the analytical method the LOQ is reported to be below 0.2 mg/kg. This level therefore reflects the ALARA ('As Low As Reasonably Achievable') principle.

A.2.1.2.2.2 Migration rates

For the exposure estimation of consumers in this dossier, migration rates quantified under dynamic conditions were used. These are strongly influenced by the material and the contact conditions such as mechanical friction and intensity of contact (pressure) between skin and product.

Three different migration rates have been estimated. In order to cover the different materials on the market and the influence of the dynamic conditions on the realistic expectancy range of dermal exposure, all of the following three migration rates for BaP were used for the calculations:

- 10 % as the worst case of dynamic migration with friction,
- 1.5 % as the approximate geometric mean of dynamic migration with friction,
- 0.2 % as the mean of dynamic migration for materials with low migration, when friction is not considered.

The knowledge about release of these compounds needs to be improved. Standardised migration methods that cover the typical uses of the consumer articles investigated should be developed by further projects.

A.2.1.2.2.3 Exposure assessment

<u>Exposure of children to rubber granules</u>

The assessment of dermal exposure of children to rubber granules from synthetic turf was performed using a) the ECETOC TRA approach b) a similar approach, but considering migration. The results clearly show that rubber products may contain high amounts of PAHs. The knowledge about release of these compounds needs to be improved, but there is concern that considerable amounts can be released.

External exposure to other consumer articles

The bulk of exposure assessment was carried out on the articles listed in section B.9.3.2.1 above. The focus of this evaluation was on the dermal route. The external dermal exposure by consumer products was estimated in two different ways:

1. The results of the dynamic migration tests described in chapter B.9.3.2.2.4 were used for estimating the external exposure to BaP by the products included in these tests.

2. The external dermal exposure was estimated based on the estimated migration rates and on the available data on BaP concentrations in consumer products.

By approach 1, external dermal exposure levels from a total of 7 products (grips, handle of a torch, a hooter, a steering wheel cover, and a hammer grip) were estimated to range from ca. 2 ng/kg bw/d up to 6.7 µg/kg bw/d.

Approach 2 was applied to > 100 articles assumed to be used by adults and ca. 100 articles assumed to be used by children. In each case, exposure estimates were calculated using all three of the above migration rates. The assumed exposure parameters contact time and contact frequency reflect user experience. These exposure scenarios therefore do not correspond to the worst case. Using the highest (most conservative) assumption of 10 % migration/h, exposure estimates of 0.3-68613 ng/kg bw/d for adults and 3-66780 ng/kg bw/d for children were obtained. Estimates using 1.5 or 0.2 % migration/h ranged lower by a factor of 6.67 or 50, respectively.

Summarising the results, there is clear evidence that under normal use conditions considerable amounts of BaP can be released even when a comparatively low migration rate is assumed.

Simulation of the proposed restriction conditions

In order to simulate the effect of the proposed restriction, another set of calculations was carried out, this time assuming that the same products used with approach 2 before would have contained 1 or only 0.2 mg BaP/kg product (but otherwise making the same assumptions regarding exposure scenarios and migration rates).

For the 1 mg BaP/kg article simulation, this resulted in a dermal exposure range of 133.3 – 750 ng/kg bw/d for adults assuming 10 % migration/h (children: 8.5 – 1600 ng/kg bw/d). Again, estimates using 1.5 or 0.2 % migration/h can be obtained by dividing by a factor of 6.67 or 50, respectively.

For the 0.2 mg BaP/kg article simulation, corresponding dermal exposure values could be obtained by dividing the results from the 1 mg/kg simulation by a further factor of 5, i. e. a range of 26.67 – 150 ng/kg bw/d for adults assuming 10 % migration/h was calculated (children: 1.7 – 320 ng/kg bw/d). Estimates using 1.5 or 0.2 % migration/h again would be lower by factors of 6.67 or 50, respectively.

The simulation of the proposed restriction conditions clearly demonstrates, that the expected dermal exposure is reduced. But, even in the case of 0.2 mg BaP/kg article the external dermal exposure is in some cases still considerable, when materials with high migration are present in consumer product. Consequently the proposed limit value should be as low as reasonable achievable (ALARA principle).

Other exposure scenarios

Exposure at the workplace or indirect exposure via the environment via food or smoking have not been considered due to lack of relevance for the problem addressed by this restriction dossier.

Combined/aggregate exposure assessment

Owing to lack of suitable data, a combined/aggregate human exposure assessment has not been performed in this dossier. From the analytical data presented in this dossier as well as from everyday experience it is clear that consumers are exposed to a multitude of potentially PAH-containing articles via several routes and will most likely come in contact with more than just one of these products on a daily basis.

Significantly higher risk characterisation ratios (RCR) than those calculated in this report could be expected for combined exposure. However, in this dossier, concern has already been demonstrated for several exemplary exposure scenarios involving just one PAH source. For this reason, a combined exposure assessment did not appear necessary.

A.2.1.2.3 Characterisation of risk(s)

Under the restriction procedure as laid out in Art. 68 (2) of the REACH regulation, consumer exposure to category 1 or 2 (within the classificatio scheme of Dir. 67/548/EEC) CMR substances via consumer articles is by itself seen as a sufficient justification for restriction. Nevertheless, in this dossier a section has been included in which potential PAH exposure from consumer products was compared against derived DMELs. Risk characterisation (RC) was only performed for the scenarios directly relevant for this dossier, i. e. use of PAH-contaminated consumer articles. Furthermore, only the dermal route has been taken forward to quantitative risk characterisation (cf. section B.9.3). A significant risk was demonstrated.

A.2.1.2.3.1 Risk characterisation for contaminated articles

DMELs (or rather, DMEL ranges) were contrasted with the exposure estimates given in Table 47 and Table 48 for adults and children, respectively, for those consumer articles which had been tested positive for PAHs. For these products, the following conclusions with respect to the current risk situation (without restriction) were drawn when comparing DMELs and exposure estimates:

- when using the most conservative estimate of a migration rate of 10 %/h, estimated external dermal exposure of *adults* may amount to as much as 70 µg/kg bw/d. The corresponding RCR would thus range as high as > 2000-fold above the highest (i. e. least conservative) DMEL values given in Table 32.

- still, when using the least conservative migration assumption of only 0.2 %/h, adult exposure could be assumed to reach up to nearly 1.5 µg/bw/d with the most highly contaminated articles.

- for children, comparably high exposure estimates up to ca. 70 µg/kg bw/d (assuming 10 % migration/h) and 1.3 µg/kg bw/d (0.2 %/h) were obtained (corresponding to RCRs of ca. 2300 and 43, respectively).

A.2.1.2.3.2 Risk characterisation for the effect of simulated restrictions

Table 47 and Table 48 in section B.9.3.2.3.4 give esposure estimates for the hypothetical situation that BaP would be restricted to a *limit of 1 mg/kg article* or down to the LOQ of 0.2 mg/kg bw/d as achieved by the analytical method:

- a limit of 1 mg BaP/kg product (component) would – depending on the exposure scenario - reduce exposure of adults to levels of up to 750 ng/kg bw/d (10 % migration/h), or 15 ng/kg bw/d (0.02 %/h). In light of the uncertainties discussed, the latter value might seem acceptable, however, it is underlined that this would only be true if the least conservative assumptions were made.

- for children, nevertheless, on top of their postulated higher vulnerability, a clearly higher exposure of up to 1600 ng/kg bw/d would be expected at a BaP level of 1 mg/kg product (10 % migration). Assuming a low migration rate of 0.2 %/h, exposure could be clearly reduced, the highest estimate still amounting to as much as 32 ng/kg bw/d, i. e. in the order of the least conservative DMEL derived, but clearly above those DMEL ranges obtained by the calculation methods recommended in the REACH guidance.

Finally, a hypothetical *limit set at the LOQ of 0.2 mg/kg* can be shown to bring exposure down to a level that could be considered as tolerable for the bulk of products/uses examined, although for some extremely contaminated articles still an exposure up to one order of magnitude above the least conservative DMELs is found when assuming the most conservative migration rate:

- according to the calculations performed, adults would be at most exposed to levels of 150 ng/kg bw/d when assuming 10 % migration/h, or 3 ng/kg bw/d, when using 0.2 %/h.

- for children, maximum exposure levels of 320 or 6.4 ng/kg bw/d are calculated, when migration rates are set to 10 or 0.2 %, respectively.

As 0.2 mg BaP/kg article (component) constitute the official LOQ of the currently available analytical method, there is at present no sense in postulating a restriction below this level. However, at least for articles accessible to or used by children, future progress in analytical methodology should be frequently monitored and the technological feasibility of a further reduction considered.

Exposure is still modeled to be somewhat above even the least conservative DMELs. The results presented in this section therefore suggest that the ALARA principle (in the form of setting the limit value to the analytical LOQ) should also be applied equally to the other known carcinogenic PAHs addressed in this dossier. Moreover, it should be kept in mind, that these substances are regulated as surrogates/placeholders for a group of hundreds of congeners, some of which are known to (and many more might) be even more carcinogenic than BaP.

A.2.1.3 Evidence that the risk management measures and operational condition implemented and recommended by the manufacturers and/or importers are not sufficient

No particular RMMs are known which specifically aim at reducing exposure of consumers to PAHs via contact with consumer products.

Under the Operational Conditions known to the authors (and which have been taken into account with the exposure scenarios considered in section B.9), no specific protection against PAH exposure from contaminated articles is foreseen.

High concentrations of PAHs in numerous consumer products also indicate that no adequate risk management measure is in place. Moreover, some manufacturers and importers appear not to be aware of the intrinsic risks by placing products on the market which contain high concentrations of PAHs.

A.2.1.4 Evidence that the existing regulatory risk management instruments are not sufficient

At present, restrictions were already installed for the same eight PAHs with respect to their content in extender oils for the specific use in tyres (entry No. 50 in Annex XVII, REACH regulation). This restriction has no impact on other products including those that might be used by consumers.

In toys, Directive 2009/48/EC – while stating that, in principle, no CMR substances of DSD categories 1 and 2 should be present – nevertheless allows BaP contents up to the specific concentration limit as specified in the CLP regulation, i. e. 0.01 % or 100 mg BaP/kg article. The authors of this dossier considered this value unacceptable from the perspective of risk assessment (cf. sections 0 and B.10), in particular, as children are seen as particularly vulnerable to carcinogenic agents (cf. section B.5.8.5).

The said specific concentration limits apply also to mixtures in general. For consumer *articles*, however, currently no EU legislation limiting their PAH content is in place.

Finally, the results of analytical examinations of product samples, which are evaluated extensively in this dossier, empirically confirm the presence of a considerable number of highly PAH-contaminated products on the market.

A.2.2 Justification that action is required on a Community-wide basis

Production, import, and marketing of consumer products containing PAHs is not constricted to any national market within the EU.

Several Member States (MS) have indicated that consumer products containing high concentrations of PAHs are on their market. These MS expressed concern with respect to the high human exposure towards PAHs. A need for limiting risks for consumers from exposure to migrating PAHs is also seen and the present proposal for restriction of PAHs in consumer products at the earliest possible date has been supported.

Aside from the pressing need for restriction of PAHs in toys and childcare products, an urgent need is also seen for other consumer products. PAH concentrations should be restricted to levels which do not pose a significant risk to consumers.

It is proposed to amend the current restriction No. 50 applying the provisions in Art. 68 (2) REACH regulation in order to reduce unacceptable risks through exposure to consumer products containing PAHs with CMR properties of category 1 or 2. The procedure as laid out in Art. 68(2) particularly addresses health risks from consumer products and has been chosen to reach inclusion into Annex XVII as soon as possible.

The effectiveness is also shown in this dossier by comparing BaP estimated uptake from contaminated consumer products with uptake from products fulfilling the limit values proposed by this dossier (cf. Table 47 and Table 48 in section B.9.3.2.3.4).

A.2.3 Justification that the proposed restriction is the most appropriate Community-wide measure

A.2.3.1 Effectiveness in reducing the identified risks

The PAHs listed in this proposal are legally classified as carcinogenic (Carc. Cat. 2/Carc. 1B) , furthermore BaP and CHR are classified for their mutagenic effects (Muta. Cat. 2 / Muta. 1B) and BaP for toxicity to reproduction (Repr. Cat. 2/Cat. 1B) acc. to Dir. 67/548/EEC (DSD)/Regulation (EC) No 1272/2008 (CLP, cf. Table 2). They were found in high concentration in articles used by consumers and in toys. The risks resulting from consumer exposure via these articles can be effectively reduced by a restriction procedure according to Article 68(2) of REACH, because it is focused and limited to CMR substances in products including articles, which can be used by consumers. It is furthermore effective, because the risk resulting from imported articles is also fully covered. Article 68(2) is more specific with respect to the identified risk compared to article 68(1) being not limited to consumer products, it is more effective as the risk can be reduced in the fraction of the time necessary for article 68(1).

A.2.3.2 Proportionality to the risks

Since the listed PAHs are carcinogens, for which no threshold can be assumed, the restriction for articles used by consumers containing more than 0.2 mg/kg BaP is proportionate. In the light of a specific vulnerability of children, it is considered reasonable and proportionate to follow the ALARA principle (as low as reasonably achievable) in order to minimise to the greatest extent possible the exposure of children from toys and childcare products to any single one or a mixture of the listed PAHs.

Many products analysed positive for high PAH contents were imported and not specially designed for the German market. Consequently a national measure would not be appropriate.

A.2.3.3 Practicality, including enforceability

PAHs occur in oil, coal, and tar deposits, are produced as byproducts of fuel burning (whether fossil fuel or biomass), and are not intentionally produced substances but impurities. As PAH-free alternatives are available, articles can be produced from materials without PAH.. Analytical methods exist to monitor PAH free production or import of articles by producers/importers. The same analytical methods can be used by enforcement agencies. In addition to this, enforcement agencies already have some experiences from the enforcement for PAH restriction in tyres. Targeting PAHs via Article 68(2) of REACH would assure a rapid implementation and enforcement of a possible restriction.

A.2.3.4 Monitorability

It is assumed that most MS have regulatory agencies to monitor the market for consumer products. Non-Governmental Organisations (NGOs) are also active in this field in some member states. Furthermore an EU wide alert system for dangerous consumer products exists under the name RAPEX (Rapid Exchange of Information System) and is administrated by the authority of the

Directorate-General for Health and Consumers of the European Commission. It allows the rapid exchange of information between Member States (via central contact points) and the Commission about measures taken to prevent or restrict the marketing or use of products posing a serious risk to the health and safety of consumers. Both measures ordered by national authorities and measures taken voluntarily by producers and distributors are covered by RAPEX. Every Friday, the Commission publishes a weekly overview of the dangerous products reported by the national authorities (the RAPEX notifications).

B. Information on hazard and risk

B.1 Identity of the substances and physical and chemical properties

B.1.1 Name and other identifiers of the substances

Chemical Name: Benzo[a]pyrene
EC Number: 200-028-5
CAS Number: 50-32-8
IUPAC Name: Benzo[d,e,f]chrysene

Chemical Name: Benzo[e]pyrene
EC Number: 205-892-7
CAS Number: 192-97-2
IUPAC Name: 1,2-Benzopyrene

Chemical Name: Benzo[a]anthracene
EC Number: 200-280-6
CAS Number: 56-55-3
IUPAC Name: 1,2-Benzanthracene

Chemical Name: Dibenzo[a,h]anthracene
EC Number: 200-181-8
CAS Number: 53-70-3
IUPAC Name: 1,2:5,6-Dibenzanthracene

Chemical Name: Benzo[b]fluoranthene
EC Number: 205-911-9
CAS Number: 205-99-2
IUPAC Name: 2,3-Benzfluoranthene

Chemical Name: Benzo[j]fluoranthene
EC Number: 205-910-3
CAS Number: 205-82-3
IUPAC Name: 10,11-Benzofluoranthene

Chemical Name: Benzo[k]fluoranthene
EC Number: 205-916-6
CAS Number: 207-08-9
IUPAC Name: 11,12-Benzofluoranthene

Chemical Name: Chrysene
EC Number: 205-923-4
CAS Number: 218-01-9
IUPAC Name: 1,2-Benzophenanthrene

B.1.2 Composition of the substances

Chemical Name: Benzo[a]pyrene

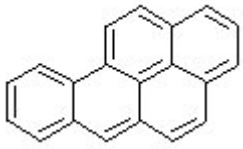
EC Number: 200-028-5

CAS Number: 50-32-8

IUPAC Name: Benzo[def]chrysene

Molecular Formula: $C_{20}H_{12}$

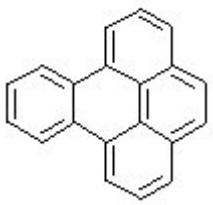
Structural Formula:

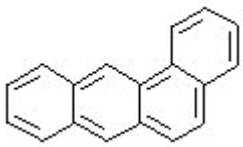


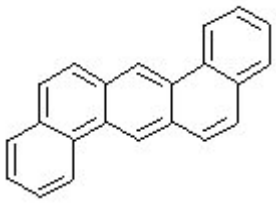
Molecular Weight: 252.3 g/mol

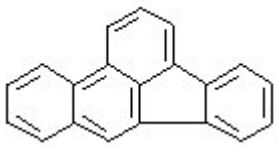
Typical concentration (% w/w): 99.9 %

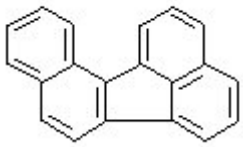
Concentration range (% w/w): Not available

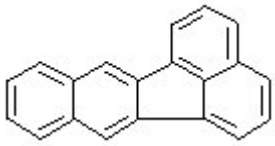
Chemical Name:	Benzo[e]pyrene
EC Number:	205-892-7
CAS Number:	192-97-2
IUPAC Name:	1,2-Benzopyrene
Molecular Formula:	$C_{20}H_{12}$
Structural Formula:	
Molecular Weight:	252.3 g/mol
Typical concentration (% w/w):	99 %
Concentration range (% w/w):	Not available

Chemical Name:	Benzo[a]anthracene
EC Number:	200-280-6
CAS Number:	56-55-3
IUPAC Name:	1,2-Benzanthracene
Molecular Formula:	$C_{18}H_{12}$
Structural Formula:	
Molecular Weight:	228.3 g/mol
Typical concentration (% w/w):	99 %
Concentration range (% w/w):	Not available

Chemical Name:	Dibenzo[a,h]anthracene
EC Number:	200-181-8
CAS Number:	53-70-3
IUPAC Name:	1,2:5,6-Dibenzanthracene
Molecular Formula:	$C_{22}H_{14}$
Structural Formula:	
Molecular Weight:	278.3 g/mol
Typical concentration (% w/w):	97 %
Concentration range (% w/w):	Not available

Chemical Name:	Benzo[b]fluoranthene
EC Number:	205-911-9
CAS Number:	205-99-2
IUPAC Name:	2,3-Benzfluoranthene
Molecular Formula:	$C_{20}H_{12}$
Structural Formula:	
Molecular Weight:	252.3 g/mol
Typical concentration (% w/w):	98 %
Concentration range (% w/w):	Not available

Chemical Name:	Benzo[j]fluoranthene
EC Number:	205-910-3
CAS Number:	205-82-3
IUPAC Name:	10,11-Benzofluoranthene
Molecular Formula:	$C_{20}H_{12}$
Structural Formula:	
Molecular Weight:	252.3 g/mol
Typical concentration (% w/w):	99.9 %
Concentration range (% w/w):	Not available

Chemical Name:	Benzo[k]fluoranthene
EC Number:	205-916-6
CAS Number:	207-08-9
IUPAC Name:	11,12-Benzofluoranthene
Molecular Formula:	$C_{20}H_{12}$
Structural Formula:	
Molecular Weight:	252.3 g/mol
Typical concentration (% w/w):	98 %
Concentration range (% w/w):	Not available

Chemical Name: Chrysene

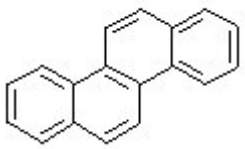
EC Number: 205-923-4

CAS Number: 218-01-9

IUPAC Name: 1,2-Benzophenanthrene

Molecular Formula: $C_{18}H_{12}$

Structural Formula:



Molecular Weight: 228.3 g/mol

Typical concentration (% w/w): 98 %

Concentration range (% w/w): Not available

B.1.3 Physicochemical properties

Table 1: Physicochemical properties of the PAHs affected by this dossier

Property	IUCLID section	Substance	Value	Reference
Physical State	4.1	Benzo[a]pyrene	Yellowish	WHO, 1998
		Benzo[e]pyrene	Pale yellow	WHO, 1998
		Benzo[a]anthracene	colourless	WHO, 1998
		Dibenzo[a,h]anthracene	colourless	WHO, 1998
		Benzo[b]fluoranthene	colourless	WHO, 1998
		Benzo[j]fluoranthene	Yellow	WHO, 1998
		Benzo[k]fluoranthene	Pale yellow	WHO, 1998
		Chrysene	Colourless	WHO, 1998
Melting point	4.2	Benzo[a]pyrene	178.1 °C	WHO, 1998
		Benzo[e]pyrene	178.7 °C	WHO, 1998
		Benzo[a]anthracene	160.7 °C	WHO, 1998
		Dibenzo[a,h]anthracene	266.6 °C	WHO, 1998
		Benzo[b]fluoranthene	168.3 °C	WHO, 1998
		Benzo[j]fluoranthene	165.4 °C	WHO, 1998
		Benzo[k]fluoranthene	215.7 °C	WHO, 1998
		Chrysene	253.8 °C	WHO, 1998

Property	IUCLID section	Substance	Value	Reference
Boiling point	4.3	Benzo[a]pyrene	496 °C	WHO, 1998
		Benzo[e]pyrene	493 °C	WHO, 1998
		Benzo[a]anthracene	400 °C	WHO, 1998
		Dibenzo[a,h]anthracene	524 °C	WHO, 1998
		Benzo[b]fluoranthene	481 °C	WHO, 1998
		Benzo[j]fluoranthene	480 °C	WHO, 1998
		Benzo[k]fluoranthene	480 °C	WHO, 1998
		Chrysene	448 °C	WHO, 1998
Relative density	4.4	Benzo[a]pyrene	1.351	WHO, 1998
		Benzo[e]pyrene	Not available	
		Benzo[a]anthracene	1.226	WHO, 1998
		Dibenzo[a,h]anthracene	1.282	WHO, 1998
		Benzo[b]fluoranthene	Not available	
		Benzo[j]fluoranthene	Not available	
		Benzo[k]fluoranthene	Not available	
		Chrysene	1.274	WHO, 1998
Vapour pressure	4.6	Benzo[a]pyrene	7.3 E-7 Pa at 25 °C	WHO, 1998
		Benzo[e]pyrene	7.4 E-7 Pa at 25 °C	WHO, 1998
		Benzo[a]anthracene	2.8 E-5 Pa at 25 °C	WHO, 1998
		Dibenzo[a,h]anthracene	1.3 E-8 Pa at 20 °C	WHO, 1998
		Benzo[b]fluoranthene	6.7 E-5 Pa at 20 °C	WHO, 1998
		Benzo[j]fluoranthene	2.0 E-6 Pa at 25 °C	WHO, 1998
		Benzo[k]fluoranthene	1.3 E-8 Pa at 20 °C	WHO, 1998
		Chrysene	8.4 E-5 Pa at 20 °C	
Water solubility	4.8	Benzo[a]pyrene	0.0038 mg/L at 25 °C	WHO, 1998
		Benzo[e]pyrene	0.0051 mg/L at 23 °C	WHO, 1998
		Benzo[a]anthracene	0.014 mg/L at 25 °C	WHO, 1998
		Dibenzo[a,h]anthracene	0.0005 mg/L at 27 °C	WHO, 1998
		Benzo[b]fluoranthene	0.0012 mg/L at 20 °C	WHO, 1998
		Benzo[j]fluoranthene	0.0025 mg/L at 25 °C	WHO, 1998
		Benzo[k]fluoranthene	0.00076 mg/L at 25 °C	WHO, 1998
		Chrysene	0.0020 mg/L at 25 °C	WHO, 1998

Property	IUCLID section	Substance	Value	Reference
Partition coefficient n-octanol/water (log value)	4.7	Benzo[a]pyrene	6.5	WHO, 1998
		Benzo[e]pyrene	6.44	WHO, 1998
		Benzo[a]anthracene	5.61	WHO, 1998
		Dibenzo[a,h]anthracene	6.5	WHO, 1998
		Benzo[b]fluoranthene	6.12	WHO, 1998
		Benzo[j]fluoranthene	6.12	WHO, 1998
		Benzo[k]fluoranthene	6.84	WHO, 1998
		Chrysene	5.91	WHO, 1998

B.1.4 Justification for grouping

Based on the knowledge about the sources of contamination of consumer products with PAHs, consumers exposed to PAH-containing articles will inevitably be exposed to complex, UVCB-type mixtures of probably up to several hundred PAH congeners. The eight PAHs addressed by this dossier are the ones explicitly known and legally classified as genotoxic carcinogens in Annex VI to Reg. No. 1272/2008 (CLP regulation). Furthermore, BaP and CHR are classified for mutagenicity and BaP also for toxicity to reproduction. Consequently, from the perspective of consumer protection, highest priority should be given to the regulation of these eight substances in one group.

In the absence of definite proof of the opposite, it can be assumed that these compounds exert their carcinogenic action by the same or (a) similar mechanism(s), albeit at different potencies. Their action has therefore to be considered as being additive.

In addition to those addressed in this dossier, clearly many more of the PAHs possibly contained in consumer articles may be genotoxic carcinogens (while others may not) and the reason for them not being listed in Annex VI to the CLP regulation may simply be that they have up to now not been evaluated for their carcinogenicity by regulatory bodies.

Consumers will often be exposed to many or even all of these substances (at different relative proportions depending on the material used in the production of the respective consumer article). However, it is assumed that by setting limits for both BaP and the sum of all of the eight known carcinogenic congeners in a 'surrogate approach', a good part of the compositional variability is covered.

B.2 Manufacture and uses

PAHs can be found ubiquitously in the environment. They form a fraction of fossil fuels such as crude oil or coal and are generated by (incomplete) combustion of these fuels or other organic materials. In addition they also originate from a number of technical processes (e.g. aluminium, iron, and steel production, oil refining). In the narrow sense of the word, PAHs themselves are not directly 'used' but become relevant with the use of oils or carbon black in which PAHs occur 'naturally' and which are added during the production process to provide the materials with different mechanical and process-related properties as needed.

PAHs in consumer products (including toys) may originate from the following various sources:

- use of mineral oil- or coal-based extender/plasticiser oils in the production of rubber and plastics; oils may (unintentionally, but uncontrolled) contain different concentrations of PAHs and are added to materials to achieve the desired material properties,

- carbon black (soot), which is intentionally added to elastomers to achieve the required properties of the material (e.g. flexibility, damping, solubility in the polymer matrix),

- recycled tyres.

Typically, PAHs are contained in certain elastomer/rubber materials, but potentially also in plastic materials, lacquers/varnishes, or coatings that may be encountered in or as part of consumer products, including toys. Numerous examples of such products include e. g. tool handles, bicycle handlebars, slippers, flip-flops, beach sandals, diver equipment, toy car tyres, or clay pigeons used in skeet shooting. PAHs may also be contained in synthetic turf or in materials used for construction work, e.g. flooring material. For the discussion of consumer exposure (cf. section B.9) in this dossier, products were assigned to the following product categories: electrical articles, handles, sports equipment, toys, body contact materials, other products with skin contact, tyres/rolls and others.

Recycled tyres that were placed on the market after 1 January 2010 should not contain extender oil exceeding the limits of more than 1 mg/kg BaP or more than 10 mg/kg of the sum of all PAHs listed in the current entry 50 of Annex XVII of the REACH regulation.

However, conversely, consumer products produced from recycled tyres before 1 January 2010 should therefore be expected to exceed the limit values proposed in this dossier. In fact, considering that a tyre might be used several years before being exchanged and subsequently recycled, it can be assumed that this situation will only gradually improve over the next 5-10 years. In addition, it should be noted that PAHs in tyres not only stem from extender oils but might also originate from carbon black as a further additive in tyre production.

B.2.1 Description of targeting

Within the group of consumer articles, no further targeting is performed.

B.3 Classification and labelling

B.3.1 Classification and labelling in Annex VI of Regulation (EC) No 1272/2008 (CLP Regulation)

Table 2: Overview of legal classification and labelling of the PAHs addressed in this dossier

Name	CAS	Acc. to Dir. 67/548/EEC (DSD)	Acc. to Dir. 1272/2008 (CLP)
Benzo[a]anthracene	56-55-3	Carc. Cat. 2; R45 N; R50-53 T; N; R: 45-50/53 S: 53-45-60-61	Carc. 1B Aquatic Acute 1; Aquatic Chronic 1 H350; H410

Name	CAS	Acc. to Dir. 67/548/EEC (DSD)	Acc. to Dir. 1272/2008 (CLP)
Chrysene	218-01-9	Carc. Cat. 2; R45 - Muta. Cat. 3; R68 N; R50-53 T; N R: 45-68-50/53 S: 53-45-60-61	Carc. 1B; Muta. 2 Aquatic Acute 1; Aquatic Chronic 1 H350; H341; H410
Benzo[b]fluoranthene	205-99-2	Carc. Cat. 2; R45 T; N N; R50-53 R: 45-50/53 S: 53-45-60-61	Carc. 1B Aquatic Acute 1 Aquatic Chronic 1 H350; H410
Benzo[k]fluoranthene	207-08-9	Carc. Cat. 2; R45 N; R50-53 T; N R: 45-50/53 S: 53-45-60-61	Carc. 1B Aquatic Acute 1 Aquatic Chronic 1 H350; H410
Benzo[a]pyrene	50-32-8	Carc. Cat. 2; R45 - Muta. Cat. 2; R46 - Repr. Cat. 2; R60-61 - R43 N; R50-53 T; N R: 45-46-60-61-43-50/53 S: 53-45-60-61	Carc. 1B ; Muta. 1B Repr. 1B ; Skin Sens. 1 Aquatic Acute 1 Aquatic Chronic 1 H350; H340; H360FD; H317 H410
Dibenzo[a,h]anthracene	53-70-3	Carc. Cat. 2; R45 N; R50-53 T; N R: 45-50/53 S: 53-45-60-61	Carc. 1B Aquatic Acute 1 Aquatic Chronic 1 H350; H410
Benzo[e]pyrene	192-97-2	Carc. Cat. 2; R45 N; R50-53 T; N R: 45-50/53 S: 53-45-60-61	Carc. 1B Aquatic Acute 1 Aquatic Chronic 1 H350; H410
Benzo[j]fluoranthene	205-82-3	Carc. Cat. 2; R45 N; R50-53 T; N R: 45-50/53 S: 53-45-60-61	Carc. 1B Aquatic Acute 1 Aquatic Chronic 1 H350; H410

B.3.2 Classification and labelling in classification and labelling inventory/Industry's self classification(s) and labelling

No data available

B.4 Environmental fate properties

B.4.1 Degradation

B.4.1.1 Stability

B.4.1.1.1 Phototransformation

Photolysis of PAHs in the troposphere results in the formation of reactive hydroxyl (OH) and nitrate (NO_3) radicals and ozone (O_3), which react as oxidising agents with organic compounds like PAHs. Reactions with these radicals and ozone comprise the main degradation path of gas-phase PAHs (Calvert et al., 2002).

Under environmental conditions in the troposphere, PAHs of higher molecular mass (i.e. those with five or more rings) are almost completely adsorbed onto fine particles, which reduce the photolytic degradation rate significantly. In a study of the degradation rate of 18 PAHs on 15 types of fly ash, carbon black, silica gel, and alumina, the PAHs were stabilised. It was found that there is a correlation between carbon content and PAH stability: the higher the carbon content, the more stable the PAHs (Behymer and Hites, 1988). Table 3 and Table 4 show examples for the representative lifetimes of six of the eight PAHs in question in this dossier with respect to photolysis when adsorbed on different particles (European Commission, 2007).

Table 3: Representative lifetimes of some 4- and 5-ring PAHs with respect to thermal reaction with nitrogen dioxide (NO2), ozone (O3) and dinitrogen pentoxide (N2O5) on "wood soot" particles

PAH (number of rings)	Representative lifetime with respect to reaction with		
	O_3[(a)]	NO_2[(b)]	N_2O_5[(c)]
Benzo[a]pyrene (5)	1.6 days[(d)] 1.8 days[(e)]	19 days[(e)]	5.1 years[(e)]
Benz[a]anthracene (4)	1.2 days[(d)]		
Chrysene (4)	2.2 days[(d)]		
Benzo[b]fluoranthene (5)			19 years[(e)]
Benzo[k]fluoranthene (5)	1.2 days[(d)]		14 years[(e)]

(a) Typical UK background O3 concentration of 30 ppbv assumed (PORG, 1997); (b) Average UK urban NO2 concentration taken to be 50 ppbv, on the basis of data presented by PORG (1997); (c) A typical 24-hour averaged N2O5 concentration estimated to be 50 pptv, on the basis of observed concentrations of NO3 (Carslaw et al., 1997) and NO2 (PORG, 1997), and the equilibrium constant for the reaction NO2+NO3 → N2O5 (Wayne et al., 1991); (d) Based on environmental chamber data (Kamens et al., 1985); (e) Based on environmental chamber data (Kamens et al., 1990).

Table 4: Representative lifetimes of some surface-adsorbed PAHs with respect to photolysis under conditions representative of a cloudless sky over the southern UK. The group classifications refer to fly ash of different compositions, as defined by Behymer and Hites (1988)

PAH (number of rings)	Classification of ash[(a)]							
	"White group"		"Red group"		"Grey group"		"Black group"	
	Summer	Winter	Summer	Winter	Summer	Winter	Summer	Winter
Benzo[a]pyrene (5)	15 min	45 min	8.0 hr	1.0 day	18 hr	2.3 day	20 hr	2.5 day

PAH (number of rings)	Classification of ash[(a)]							
	"White group"		"Red group"		"Grey group"		"Black group"	
	Summer	Winter	Summer	Winter	Summer	Winter	Summer	Winter
Benzo[e]pyrene (5)	9.1 hr	1.1 day	2.4 day	7.2 day	2.5 day	7.5 day	1.1 day	3.3 day
Benz[a]anthracene (4)	15 min	45 min	6.6 hr	20 hr	17 hr	2.1 day	1.1 day	3.3 day
Chrysene (4)	10 hr	1.3 day	2.6 day	7.8 day	2.3 day	6.9 day	1.0 day	3.0 day

(a) The classification of the ash into four groups depends on the relative contents of 10 elements, which influences the colour of the substrate (Behymer and Hites, 1988). The photolysis lifetimes measured in that study have been scaled to provide values representative of 24-hour averaged conditions in the boundary layer over the southern UK.

B.4.1.1.2 Hydrolysis

Hydrolysis as a way of abiotic degradation can be considered as not relevant for the PAHs because the chemical structure of PAHs lack functional groups susceptible to hydrolysis.

B.4.1.2 Biodegradation

B.4.1.2.1 Biodegradation estimation

Mackay et al. ranked the 16 so-called EPA-PAHs (acenapthene, ancenaphtylene, anthracene, benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, benzo[a]-pyrene, chrysene, dibenzo[a,h]anthracene, fluoranthene, fluorene, indeno[1,2,3-c,d]pyrene, naphtaline, phenanthrene and pyrene) according to their persistence in water, soil and sediment in different classes which correspond to a specific half-life in these compartments (Mackay et al., 2000). The basis for this ranking were model calculations. Table 5 shows the ranking half-life times associated to the rankings in the different compartments for the six EPA-PAHs relevant in this dossier.

Table 5: Ranking of PAHs in different half-life classes (Mackay et al., 2000)

PAH (number of rings)	Ranking					
	Water		Soil		Sediment	
	Class	Calculated Half-life [d]	Class	Calculated Half-life [d]	Class	Calculated Half-life [d]
Benzo[a]pyrene (5)	5	42 – 125	7	420 – 1250	8	> 1250
Benzo[a]anthracene(4)	5	42 – 125	7	420 – 1250	8	> 1250
Chrysene (4)	5	42 – 125	7	420 – 1250	8	> 1250
Benzo[b]fluoranthene (5)	5	42 – 125	7	420 – 1250	8	> 1250
Benzo[k]fluoranthene (5)	5	42 – 125	7	420 – 1250	8	> 1250
Dibenzo[a,h]anthracene (5)	5	42 – 125	7	420 – 1250	8	> 1250

B.4.1.2.2 Screening Tests

Coover and Sims tested the persistence of PAHs in an unacclimated agricultural sandy loam soil in dependence on the temperature (Coover and Sims, 1987). Due to the method used for extraction and analysis, it remains unclear to which extent evaporation, adsorption and biodegradation may have

contributed to the elimination process. The soil was spiked with a standard solution of 16 EPA-PAHs and incubated for 240 days. The results for six PAHs relevant in this dossier are shown in Table 6.

Table 6: Persistence of six PAHs in an unacclimated agricultural sandy loam soil after 240 days (Coover and Sims, 1987)

PAH (number of rings)	Percent of PAH remaining at		
	10 °C	20 °C	30 °C
Benzo[a]pyrene (5)	73	54	53
Benzo[a]anthracene(4)	82	71	50
Chrysene (4)	85	88	86
Benzo[b]fluoranthene (5)	77	75	62
Benzo[k]fluoranthene (5)	93	95	89
Dibenzo[a,h]anthracene (5)	88	87	83

B.4.1.2.3 Simulation Tests

B.4.1.2.3.1 Biodegradation in soil

Biodegradation rates of PAHs in soil depend on several factors like soil type, pH, moisture content, oxygen and nutrient content and soil microbial population. In addition, vegetation has been observed to enhance microbial biodegradation in the rhizosphere. Some of these factors may also explain why the half-lives observed under laboratory conditions are much shorter than those obtained from long-term field-based experiments (European Commission, 2007). The results of Wild et al. (1991) and Wild and Jones (1993) demonstrate the difference of tests conducted for several PAHs in field conditions compared to laboratory tests. Wild et al. (1991) observed elimination half-lives (in form of dissipation times) starting from 8.1 years for benzo[a]anthracene and chrysene up to 9 years for benzo[b]fluoranthene. In this field experiment soils were enriched with PAH-contaminated sludge (Wild et al., 1991).
In another study Wild and Jones derived different half-lives in a microcosm study with four soil types (Wild and Jones, 1993). The elimination half-lives for the tested PAHs are much shorter than in the field study spanning a range starting from 106-313 days for benzo[a]anthracene and chrysene up to 143-359 days for benzo[k]fluoranthene. It has to be noted that the latter results were derived from a greenhouse study and should therefore not be used for the assessment of persistence. Various studies on PAH-contaminated soils have revealed that the number of PAH-degrading microorganisms and the degrading capacity are much higher in PAH-contaminated soils than in pristine soils indicating that adaptation may occur (European Commission, 2007).

The fate of several PAHs in two different soils was tested by Park et al. (1990). For this dossier the results for benzo[a]pyrene, benzo[a]anthracene, chrysene and benzo[b]fluoranthene are important. The calculated half-live times for the aforementioned PAHs are all in the range of up to several hundred days.

In a 1280 days laboratory simulation of the “landfarming” process an oily sludge of a petrochemical plant was seven times applied to sandy loam samples during a 920-day active disposal period followed by a 360-day inactive 'closure' period. The decreases in the concentrations of several

PAHs including benzo[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene and benzo[e]pyrene in soil were determined. While the four-ring substances (benzo[a]anthracene, chrysene) had been partly degraded, the five-ring compounds remained at fairly high concentrations (Bossert et al., 1984). Since the duration of this study is too long for the inocculum to remain stable this study as such is regarded not to be reliable. Nevertheless the trend that shows in the other studies is supported by this work.

Table 7: Elimination half-lives for PAHs in soil

Substance	Result	Reference
Benzo[a]pyrene	$DisDT_{50}$ =8.2 years (field study)	(Wild et al., 1991)
	$DisDT_{50}$ =120 – 270 d (microcosm study)	(Wild and Jones, 1993)
	Elimination half- life in two different soils: $DisDT_{50}$ = 178 – 315 d $DisDT_{50}$ = 239 – 462 d	(Park et al., 1990)
	55.6 % remaining after 1280 days (laboratory simulation of landfarming process)	(Bossert et al., 1984)
Benzo[e]pyrene	87.0 % remaining after 1280 days (laboratory simulation of landfarming process)	(Bossert et al., 1984)
Benzo[a]anthracene	$DisDT_{50}$ = 8.1 years (field study)	(Wild et al., 1991)
	$DisDT_{50}$ = 106 – 313 d (microcosm study)	(Wild and Jones, 1993)
	Elimination half- life in two different soils: $DisDT_{50}$ = 131 – 217 d $DisDT_{50}$ = 210 – 347 d	(Park et al., 1990)
	1.5 % remaining after 1280 days (laboratory simulation of landfarming process)	(Bossert et al., 1984)
Chrysene	$DisDT_{50}$ =8.1 years (field study)	(Wild et al., 1991)
	$DisDT_{50}$ =106 – 313 d (microcosm study)	(Wild and Jones, 1993)
	Elimination half- life in two different soils: $DisDT_{50}$ = 257 – 866 d $DisDT_{50}$ = 289 – 533 d	(Park et al., 1990)
	3.1% remaining after 1280 days (laboratory simulation of landfarming process)	(Bossert et al., 1984)
Benzo[b]fluoranthene	$DisDT_{50}$ =9 years (field study)	(Wild et al., 1991)
	$DisDT_{50}$ =113 – 282 d (microcosm study)	(Wild and Jones, 1993)
	Elimination half- life in two different soils: $DisDT_{50}$ = 169 – 277 d $DisDT_{50}$ = 231 – 385 d	(Park et al., 1990)
	79.4% remaining after 1280 days (laboratory simulation of landfarming process)	(Bossert et al., 1984)
Benzo[j]fluoranthene	79.4% remaining after 1280 days (laboratory simulation of landfarming process)	(Bossert et al., 1984)

Substance	Result	Reference
Benzo[k]fluoranthene	$DisDT_{50}$ =8.7 years (field study)	(Wild et al., 1991)
	$DisDT_{50}$ =143 – 359 d (microcosm study)	(Wild and Jones, 1993)
	29.9% remaining after 1280 days (laboratory simulation of landfarming process)	(Bossert et al., 1984)
Dibenzo[a,h]anthracene	Elimination half- life in two different soils: $DisDT_{50}$ = 267 – 990 d $DisDT_{50}$ = 267 – 533 d	(Park et al., 1990)

B.4.1.3 Summary

The presented data show that the PAHs relevant for this dossier degrade in the environment very slowly:

The model calculations by Mackay et al. (1992) indicate that benzo[a]pyrene, benzo[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene and dibenzo[a,h]anthracene persist in sediments with half-lives of more than 180 days. Screening studies (OECD TG 301C) for these substances demonstrate that they are not readily biodegradable (MITI-List, 2002). When finally considering biodegradation studies in soil, it becomes clear that for the six substances the dissipation half-lives are clearly longer than the 180-days-criteria of REACH article 57 e), especially when also considering the field studies of Wild et al., (1991). Therefore benzo[a]pyrene, benzo[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene and dibenzo[a,h]-anthracene fulfil the P and the vP criteria according to article 57 d) and e) of the REACH regulation.

For the two remaining PAH, benzo[e]pyren and benzo[j]fluoranthene there is much less data available, probably due to the fact that these two substances do not belong to the pool of the 16 PAHs defined by EPA. Since no degradation studies with half-life times were published for these two PAHs it cannot definitely be shown whether the P and/or vP criteria are met. Nevertheless, the substances are structurally closely related to the other six PAHs and also the study of Bossert et al. (1984) suggests similar dissipation behaviour in soil. Therefore it can be assumed that they, too, are persistent in the environment.

B.4.2 Environmental distribution

B.4.2.1 Adsorption/desorption

The distribution of a substance between air and particulate matter under normal atmospheric conditions depends on the lipophilicity, vapor pressure, and aqueous solubility of the substance. Generally, PAHs with few (two to four) aromatic rings occur mainly in the vapor phase and are adsorbed, whereas PAHs consisting of more aromatic rings exist mainly in the adsorbed state (Hoff and Chan, 1987; McVeety and Hites, 1988; Baker and Eisenreich, 1990).

The organic carbon partitioning coefficient log K_{OC} is a measure for the tendency of a substance to adsorb on organic carbon. An overview for the PAHs in question is shown below in Table 8.

Table 8: Distribution relevant date: Henry`s constants, log K_{OW} and log K_{OC} data of the PAHs in this dossier.

Substance	Henry`s constant (Pa/m^3mol at 25 °C)	log K_{OW}[(a)]	log K_{OC}[(a)]	Comments reg. log K_{OC}[(a)]
Benzo[a]pyrene	0.034[(b)]	6.50	8.3	Specified particulate
			6.66	LSC
			6.26	Average on sediments
			4.0	Predicted to be dissolved
Benzo[e]pyrene	0.020[(c)]	6.44	7.20	Specified particulate
			4.0	Predicted to be dissolved
Benzo[a]anthracene	0.81[(b)]	5.61	7.30	Specified particulate
			6.30	Average on Sediments
			4.52	Suspended particles
Chrysene	0.079[(b)]	5.91	6.90	Specified particulate
			6.27	Average on Sediments
			4.0	Predicted to be dissolved
Benzo[b]fluoranthene	0.051[(b)]	6.12	5.06[(d)]	Calculated[(d)]
Benzo[j]fluoranthene	0.021[(e)]	6.12	5.06[(d)]	Calculated[(d)]
Benzo[k]fluoranthene	0.043[(b)]	6.84	7.00	Specified particulate
			5.99	Average on Sediments
			4.00	Predicted to be dissolved
Dibenzo[a,h]anthracene	$1.3\ 10^{-4}$ [(b)]	6.50	6.31	Average of 14 soil or sediment samples, shake flask, LSC

(a) Unless stated otherwise values were taken from the WHO-report on PAHs (WHO, 1998); (b) Values were taken from Annex XV transitional report – CTPHT (European Commission, 2007); (c) Values were taken from Mackay *et. al.*, 1992; (d) Values were calculated according to the Endpoint Specific Guidance of ECHA (European Chemicals Agency, 2008); (e) Values were taken from a study of Meylan and Howard (1991)

It can be concluded that PAHs with four or five rings have a very high potential to adsorb to organic matter and will be distributed to sediments and soil. Since they are not mobile in soil and sediment, they can be expected to persist in these compartments.

B.4.2.2 Volatilisation

The process of volatilisation depends on temperature, wind, water movement and molecular size of the substance in question. Southworth showed that benzo[a]pyrene (five ring PAH) and benzo[a]anthracene (four ring PAH) are persistent with respect to volatilisation due to their low Henry`s Law constant (Southworth, 1979). Since the Henry`s Law constants for the other six PAHs in this dossier (all four and five ring PAHs) are all in the same range or even lower as for benzo[a]pyrene and benzo[a]anthracene (see Table 8) low volatilisation is expected to be a negligible dispersion process for these substances.

B.4.2.3 Distribution modelling

The behaviour of the eight PAHs in this dossier in a municipal wastewater treatment plant was calculated using the Simple Treat Model under the assumption that no biodegradation occured (k=0/h). The results are shown in Table 9.

Table 9: Distribution of considered PAHs in municipal waste water treatment plants (STP)

Substance	log K_{OC}	Distribution of PAH in STP[(b)]			
		% to air	% to water	% to sludge	% degraded
Benzo[a]pyrene	8.3	0.0	8.0	92.0	0.0
	6.66	0.0	8.2	91.8	0.0
	6.26	0.0	8.4	91.6	0.0
	4.0	0.0	46.9	53.1	0.0
Benzo[e]pyrene	7.20	0.0	8.0	92.0	0.0
	4.0	0.0	46.9	53.1	0.0
Benzo[a]anthracene	7.30	0.0	8.0	92.0	0.0
	6.30	0.0	8.3	91.7	0.0
	4.52	0.0	24.4	75.6	0.0
Chrysene	6.90	0.0	8.1	91.9	0.0
	6.27	0.0	8.4	91.6	0.0
	4.0	0.1	46.8	53.1	0.0
Benzo[b]fluoranthene	5.06	0.0	13.5	86.5	0.0
Benzo[j]fluoranthene	5.06	0.0	13.5	86.5	0.0
Benzo[k]fluoranthene	7.00	0.0	8.1	91.9	0.0
	5.99	0.0	8.7	91.3	0.0
	4.00	0.0	46.8	53.2	0.0
Dibenzo[a,h]anthracene	6.31	0.0	8.3	91.7	0.0

(b) Values for distribution in STP calculated with SimpleTreat 3.0 (debugged version, 7 Feb 97)

Due to the partitioning to solids, low concentrations of these PAHs in aqueous solutions are expected.

B.4.3 Bioaccumulation

B.4.3.1 Aquatic Bioaccumulation

Bioaccumulation of PAHs from water is strongly dependent on the physicochemical properties of the compound and the species exposed: the rates of accumulation and elimination generally decrease with increasing molecular weight (corresponding to a lower solubility). Different factors linked to animal behaviour and characteristics influence uptake and accumulation of PAHs, such as biotransformation, size of the organism, avoidance of highly contaminated sites, burrowing behaviour, density of the organism population and bioturbation. Metabolism may be very important

in explaining PAH accumulation patterns. It is suspected that high molecular weight PAHs are more rapidly metabolized than low molecular weight PAHs due to differences in enzyme affinity (Schnell et al., 1980).

Molluscs have a very limited ability to metabolize PAHs, while in algae and oligochaete worms no evidence of PAH metabolism has been found.

B.4.3.1.1 Bioaccumulation estimation

Based on the substance's log K_{OW} range from 5.91 - 6.5 the PAHs considered are expected to bioaccumulate.

B.4.3.1.2 Measured Bioaccumulation data

Bioaccumulation of many PAHs has been measured in various species. Most of the studies mentioned below have already been discussed in detail in the Annex XV transitional dossier for coal tar pitch high temperature (European Commission, 2007), in the Annex XV Support document for the identification of coal tar pitch high temperature as a SVHC-substance (European Chemicals Agency, 2009) and in a report by RIVM on the Bioaccumulation potential of PAHs (Bleeker and Verbruggen, 2009). Experimental data from studies on other aquatic organisms, i.e. crustaceans, were considered for the PAHs for which no studies were available with fish or molluscs.

The most relevant studies and results are summarised in Table 10.

Table 10: Overview of the bioaccumulation studies for the eight considered PAHs.

Substance	Species	BCF	R[a)]	Test system[b)]	Type[c)]	References
Benzo[a]pyrene	Lepomis macrochirus	367 - 608 [h)]	2	F	k_1/k_2 (total); fed	(Jimenez et al., 1987)
	Lepomis macrochirus	30	2	F	k_1/k_2	(McCarthy et al., 1985)
	Dreissena polymorpha	84000[i)]	2	S	k_1/k_2 (total=parent)	(Bruner et al., 1994)
	Dreissena polymorpha	41000[j)]	2	S	k_1/k_2 (total=parent)	(Bruner et al., 1994)
	Dreissena polymorpha	77000[k)]	2	S	k_1/k_2 (total=parent)	(Bruner et al., 1994)
	Dreissena polymorpha	133000[n)]	2	S	k_1/k_2 (total=parent)	(Gossiaux et al., 1996)
	Dreissena polymorpha	142000[o)]	2	S	k_1/k_2 (total=parent)	(Gossiaux et al., 1996)
	Perna viridis	8500 [l)]	2	SR	Equilibrium	(Richardson et al., 2005)
	Daphnia magna	12761	2[f)]	S	Equilibrium	(Newsted and Giesy, 1987)
	Daphnia magna	2837	2[f)]	S	Equilibrium	(Leversee et al., 1981)
	Eurytemora affinis	1750 [g)]	2	F	Equilibrium	(Cailleaud et al., 2009)
	Mysis relicta	8496	2	F	k1/k2	(Evans and Landrum, 1989)
	Pontoporeia hoyi	73000	1	F	k1/k2	(Landrum, 1988)
	Pontoporeia hoyi	48582	2	F	k1/k2	(Evans and Landrum, 1989)
	Chironomus riparius (4th instar larvae)	650	2	S	Equilibrium	(Leversee et al., 1982)
	Chironomus riparius (4th instar larvae)	166	2	S	Equilibrium	(Leversee et al., 1981)
	Hexagenia limbata	2725 – 11167 [m)]	2	F	k1/k2	(Landrum and Poore, 1988)
	Stylodrilus heringianus	7317	2	F	k1/k2	(Frank et al., 1986)
	Lemna gibba	7 - 910	2	F	k1/k2	(Duxbury et al., 1997)
	Capitella capitata	0.7[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
	Polychaete sp.	13.8[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)

Substance	Species	BCF	R[a)]	Test system[b)]	Type[c)]	References
Benzo[e]pyrene	Daphnia magna	25200	2[e)]	S	NS	(Newsted and Giesy, 1987)
	Capitella capitata	1.5[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
	Polychaete sp.	11.6[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
Benzo[a]anthracene	Pimephales promelas	260	2	F	Equilibrium	(de Maagd et al., 1998)
	Pontoporeia hoyi	63000	1	F	k_1/k_2	(Landrum, 1988)
	Daphnia pulex	10109	2 [f)]	S	Equilibrium	(Southworth et al., 1978)
	Daphnia magna	10226	2 [f)]	S	Equilibrium	(Newsted and Giesy, 1987)
	Capitella capitata	3.6[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
	Polychaete sp.	9.4[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
Chrysene	Daphnia magna	6088	2 [f)]	SR	Equilibrium	(Newsted and Giesy, 1987)
	Eurytemora affinis	950 [g)]	2		Equilibrium	(Cailleaud et al., 2009)
	Capitella capitata	6.2[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
	Polychaete sp.	14. [d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
Benzo[b]fluoranthene	Capitella capitata	1.7[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
	Polychaete sp.	9.1[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
Benzo[j]fluoranthene	Capitella capitata	8.2[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
	Polychaete sp.	0.6[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
Benzo[k]fluoranthene	Daphnia magna	13225	2 [f)]	SR	Equilibrium	(Newsted and Giesy, 1987)
	Capitella capitata	1.8[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
	Polychaete sp.	14.1[d)]	3[e)]	FD	Equilibrium	(Bayona et al., 1991)
Dibenzo[a,h]anthracene	Daphnia magna	50119	2 [f)]	S	Equilibrium	(Newsted and Giesy, 1987)

a) Reliability score: 1-reliable without restrictions, 2-reliable with restrictions, 3-unreliable, 4-not assignable; b) F: flow-through system, S: static exposure system, SR: static renewal, FD: organisms collected from the field; c) k1/k2: kinetik: uptake rate/depuration rate, total: total compound concentration (including transformation products), parent: parent compound concentration, NS: not steady state; d) "Apparent" bioconcentration factors (ratio between tissue and sediment concentrations); e) Reliabilty scores according to (Bleeker and Verbruggen, 2009); f) Reliability scores according to draft rivm report (2009); g) BCFs are based on dry weight

B.4.3.2 Summary

Benzo[a]anthracene accumulates in fish (Pimephales promelas) but fails to reach the Bioaccumulation trigger value (i.e. BCF >2000). In crustaceans BCF values are very high (>10000), meeting the trigger values for the B and vB-characteristic. Benzo[a]pyrene shows similar patterns with BCF fish clearly not reaching the trigger value and invertebrates exceeding it by far, with the exception of Chironomus riparius that is capable of metabolizing benzo[a]pyrene. For benzo[e]pyrene, chrysene, benzo[k]fluoranthene, and dibenzo[a,h]anthracene valid data are only

available for Daphnia magna showing very high BCF values for all compounds. For benzo[b]fluoranthene and benzo[j]fluoranthene no reliable data are available at all, although benzo[b]fluoranthene appears to accumulate in molluscs in the field (Takeuchi et al., 2009). From a weight of evidence approach on the basis of similarities of the log K_{OW}-values and molecular sizes it can be assumed that these PAHs will fulfill the B criterion (i.e. BCF >2000) as well.

B.4.4 Secondary poisoning

Food web transfer of PAHs and accumulation of PAH metabolites by predators may theoretically occur in both aquatic and terrestrial environments. However, several studies have shown that biomagnification of PAHs does not occur. This effect can partly be explained due to the relatively high rates of metabolism and also due to the excretion of PAHs in vertebrates and some invertebrates (The Netherlands - Bureau REACH, 2009).

B.5 Human health hazard assessment

Hazards and risks of PAHs and PAH-containing materials were reviewed in various reports (WHO, 1995 (JECFA), 1998 (IPCS), and 2003; ATSDR 1995; EFSA 2008). These reports have assessed the toxicological data on PAHs in detail and it is not the goal of this dossier to reiterate their content.

Numerous PAHs are known to be carcinogenic, mutagenic, and/or toxic to reproduction. The human health endpoint of utmost concern is their potential for genotoxic carcinogenicity. Carcinogenicity of PAHs will presumably be exerted in humans and is being regarded as the critical effect for the purpose of this restriction proposal.

In addition to the above-mentioned (and some additional) review reports, which covered the published literature up to and including the year 2002, a literature research was performed for this dossier in order to account for the recent literature on BaP and PAH toxicity and risk assessment from 2003 until today. The following databases were searched: DIMDI, Scopus, HighWire, ISI Web of Knowledge, and TOXNET. The databases were queried for the terms 'PAH' and/or the CAS numbers of the substances evaluated in this dossier in combination with the terms 'cancer', 'carcinogenicity', 'tumor', or 'tumour'. A similar search was performed using the terms 'metabolite', 'metabolism', 'toxicokinetics', 'absorption', or 'ADME'.

In contact with consumer articles, consumers are exposed to a multitude of PAH mixtures of different composition. Each of the (sometimes up to several hundred) different PAH mixture components possesses its own toxicity profile, absorption behaviour, and may potentially be carcinogenic. Basically three approaches have been chosen by risk assessors to address this issue (cf. WHO 1998): the Toxicity Equivalence Factor (TEF) approach, the Relative Potency approach, or the 'surrogate' approach (i. e. taking BaP as a representative surrogate of the toxicity of the whole PAH mixture).

The surrogate approach, which has been justified by a number of authors, has also been pursued in the creation of this dossier, as the authors agree with the conclusion given by EFSA (2008):

> *'[…] A toxic equivalency factor (TEF) approach to the risk characterisation was not considered to be scientifically valid because of the lack of data from oral carcinogenicity studies for different PAHs, their different modes of action and the evidence of poor predictivity of the carcinogenic potency of PAH mixtures based on the currently proposed TEF values.[…]'*

B.5.1 Toxicokinetics (absorption, metabolism, distribution and elimination)

B.5.1.1 Absorption

B.5.1.1.1 Oral

Extensive descriptions are available in the standard reviews, e.g. Mumtaz and George (ATSDR) 1995, WHO (1998) and EFSA (2008). The following short summary is given in WHO (2003):

> *'[...] PAHs are absorbed in experimental animals and humans through the pulmonary tract, the gastrointestinal tract, and the skin. Absorption of BaP, DBahA*

[dibenzo[a,h,]anthracene], and PY [pyrene] was high (30–90 %) following low and high oral doses in rats (Chang, 1943; Foth et al., 1988; Withey et al., 1991). Absorption from the gastrointestinal tract occurs rapidly. Oral administration of FA [fluoranthene], PY, and BaA [benzo[a]anthracene] to rats caused peak concentrations of these compounds in the blood after 1–2 hours (Lipniak & Brandys, 1993). The intestinal absorption of the individual PAHs is highly dependent on their solubility, their lipidity, the presence of bile (Rahman et al., 1986), and the lipidity of the various PAH-containing foods ingested. Whereas oils enhanced the absorption of BaP, water and solid food suppressed the absorption (Kawamura et al., 1988). [...]'

Mainly due to the use of non-standardised test methodology instead of following established test guidelines, the results described in the literature are sometimes confusing and contradictory, as is well demonstrated at the example of a summary given by Bulder (2006):

'[…] Absorption of PAHs from the gastro-intestinal tract appears to vary per animal species. BaP absorption reached 89-99 % after oral (food) exposure of rats (Rabache et al., cited by Kan et al, 2003). Another study in rats showed that first a direct absorption occurs 1-2 hours after feeding. After 3-4- hours a second increase in serum concentration occurs due to entero-hepatic circulation (Van Schooten et al., 1997, cited by Kan, 2005). In contrast, a study from Grova et al. (2002) showed that activity from radio-labeled BaP was not traced in blood and milk from orally exposed lactating goats. Hoogenboom et al. (2005) concluded from this study that the heavier PAHs are apparently not absorbed from the gastro-intestinal tract (and transferred to milk). Since the rat seems more relevant as a model for human uptake, it is considered that in humans PAHs may be readily absorbed from the gastro-intestinal tract. […]'

The study by Foth et al. (1998) arrived at an oral bioavailability of BaP of 40 %, but the conditions used were not fully transferable to the *in vivo* situation in humans (the test substance was not administered via the diet, but by intraduodenal infusion instead).

Overall, in order to account for the uncertainties in the oral absorption database studies, an oral absorption rate of 50 % was assumed. It should be noted, that the absorption rate for some PAHs from cigarette smoke is considerably higher (cf. section B.9.3.3.1).

B.5.1.1.2 Inhalation

Literature on absorption of BaP/PAHs via inhalation is somewhat vague when it comes to concluding on concrete bioavailability figures following exposure along this route. Even in the standard reviews (WHO 1998, Mumtaz and George (ATSDR) 1995) no precise absorption rates are established. However, there appears to be some degree of conviction that a significant amount of PAHs might be systemically available after inhalation.

In order to account for the uncertainties in the assessment of bioavailability following inhalation, an absorption rate of 50 % was assumed.

B.5.1.1.3 Dermal

A variety of reports of experiments on dermal absorption performed with different PAHs both *in vivo* and *in vitro* are available. Given the 'one-for-all'/substitute approach followed in this dossier, the focus is placed on studies performed with BaP.

The following studies were evaluated: Sanders et al. (1986), Payan et al. (2009), Kao et al. 1985, Fasano et al. (2007 and 2007b), Ng et al. (1992), Wester et al. (1990). The majority of these studies showed – to a varying degree - deficits in experimental setup and/or reporting, and/or they were often not readily transferable to the problems discussed in this report. Kao and co-workers (1985) demonstrated the importance of using viable skin as they found that active skin metabolism significantly enhanced the uptake of radioactivity in absorption studies with BaP.

The two studies found most suitable for the purpose of this dossier were the ones by Wester et al. (1990) and Ng et al. (1992).

Overall, notwithstanding the fact that even higher dermal absorption rates were observed in animal studies with BaP dissolved in acetone both *in vivo* and *in vitro*, a value of 50 % was established as a realistic scenario in animals. It is noted that the question of whether acetone constitutes a 'realistic' vehicle in terms of human exposure is not relevant in this context. The majority of dermal carcinogenicity studies in animals were also performed using acetone.

However, for risk characterisation (comparison of human exposure against DMELs), an estimate of 20 % was used, as – based on assessment of the whole available database - dermal uptake of BaP/PAHs from sweat was considered likely to be clearly lower compared to the situation when acetone was used as a vehicle. It must be stressed that this rate only refers to dermal uptake of BaP/PAHs following migration of these substances from the contaminated article into the sweat. The migration rated is accounted for separately (cf. section B.10).

In summary, the database in animals demonstrated considerable absorption across all possible routes of exposure. For DMEL derivation, the following assumptions were made:

- In the absence of any substantial evidence to the contrary, absorption rates were seen as being basically comparable for all routes in these experiments i. e. all were estimated to be in the range of about 50 %.

- While this holds true for dermal absorption of BaP out of an acetone matrix, absorption from sweat was assumed to be lower by a factor of 2.5, i. e. in the order of 20 %.

B.5.1.2 Distribution

Extensive summaries of the available data on distribution have been provided a. o. by ATSDR (1995), WHO (1998 or 2003), or EFSA 2008. Apart from the fact that a different time-course of distribution along different routes of application might be part of the explanation, why in the carcinogenicity studies summarised below tumours were often noted at the site of first contact, i. e. before systemic distribution, this endpoint does not play a central role for the argumentation in this dossier and is therefore not considered any further.

With regard to bioaccumulation, specific data in mammals were not available for this dossier. Based on observed metabolisation rates and on the recovery of radioactivity in oral or dermal absorption experiments, it is considered unlikely that the PAHs covered by this dossier or their metabolites possess an excessive potential to accumulate in the human body.

B.5.1.3 Metabolism

A short summary is provided in WHO (2003):

'[...] The metabolism of PAHs is complex. Generally, the process involves epoxidation of double bonds, a reaction catalysed by the cytochrome P-450-dependent monooxygenase, the rearrangement or hydration of such epoxides to yield phenols or diols, respectively, and the conjugation of the hydroxylated derivatives. Reaction rates vary widely, and interindividual variations of up to 75-fold have been observed, for example, with human macrophages, mammary epithelial cells, and bronchial explants from different donors. Most metabolism results in detoxification, but some PAHs in some situations become activated to DNA-binding species, principally diol-epoxides, that can initiate tumours (WHO, 1997). Although the PAHs are similar, they have structural differences that are the basis for differences in metabolism and relative carcinogenicity. The metabolism of the more carcinogenic, alternant (equally distributed electron density) PAHs, such as BaP, BaA, and DBahA, seems to differ in some ways from that of non-alternant (uneven electron density distribution) PAHs, such as FA, BbFA, BkFA [benzo[k]fluoranthene], BjFA [benzo[j]fluoranthene], IP [indenopyrene], BghiP [benzo(g,h,i)perylene], and PY (Phillips & Grover, 1994; ATSDR, 1995). In general, little is known about the metabolism of most PAHs, particularly in non-rodent species. It should be noted that there appear to be species differences in the enzymes that activate PAHs (Michel et al., 1995) and in the formation of DNA adducts (Kulkarni et al., 1986).[...]'

Metabolic activation is seen as a prerequisite for the carcinogenic potential of the PAHs covered by this dossier but has been extensively discussed in other reviews of PAH toxicity (IARC 2006, cf. also WHO 1998, Table 74: Putative ultimate carcinogens). Cf. also section B.5.7 on mutagenicity below.

As the focus of this dossier is on carcinogenicity, a discussion of metabolism by itself is not considered relevant and this endpoint therefore was therefore not elaborated any further.

B.5.1.4 Excretion

Not relevant for this dossier

B.5.2 Acute toxicity

Not relevant for this dossier

B.5.3 Irritation

Not relevant for this dossier

B.5.4 Corrosivity

Not relevant for this dossier

B.5.5 Sensitisation

Not relevant for this dossier. Of the eight compounds treated in this dossier, BaP is the only one legally classified/labelled as sensitising via skin in the EU (cf. section B.3).

B.5.6 Repeated dosed toxicity

Non-neoplastic effects have not been considered for this dossier which is focussed on potential carcinogenic risks arising from PAH-contaminated consumer products. It is assumed that thresholds for non-neoplastic effects will be clearly higher than the range of DMELs which is deduced below (section B.10).

B.5.7 Mutagenicity

PAHs with relatively planar, highly conjugated aromatic structures such as the considered PAHs have a genotoxic potential which is characterised as indirect because metabolic activation is required for the induction of mutagenic effects. Mutagenic effects are induced after biotransformation of these PAHs to reactive electrophilic metabolites, which can bind covalently and form adducts with intracellular macromolecules such as DNA. DNA adducts, which may result in gene mutations, DNA strand breaks or chromosomal aberrations, are the precursor lesions for mutations, which arise through replication of errors in the DNA during DNA synthesis.

In a summary report of Xue and Warshawsky (2005) the following pathways for metabolic activation of PAHs are discussed:

- dihydrodiol epoxide pathway
- one-electron oxidation pathway
- orthoquinone pathway and
- sulphuric acid ester pathway.

The *dihydrodiol epoxide* pathway is catalysed by several enzymes such as CYPs and epoxide hydroxylases. Metabolic activation finally leads to the formation of electrophilic diolepoxides, which belong to the most potent chemical mutagens reported so far. BaP, chrysene and benzo[a]anthracene are metabolised through this pathway. BaP, whose metabolism is well examined, is rapidly metabolised to its main metabolite, 7,8-dihydrodiol-9,10-epoxide, which then can form DNA adducts.

The *one-electron oxidation* pathway seems to play a relevant role for certain PAHs with lower ionisation potential. It is known that a PAH radical cation is formed by removal of one electron through oxidation. Electrophilic radical cations are capable of interacting with nucleophilic regions in cellular macromolecules.

The oxidation of PAH dihydrodiols by dihydrodiol dehydrogenases to PAH-derived *orthoquinones* is described as a possible metabolic activation pathway for PAHs. Also PAH-orthoquinones have the potential to form stable DNA-adducts.

Formation of *sulphuric acid esters* has been observed with several primary or secondary hydroxymethyl PAHs and is catalysed by the enzyme sulfotransferase. Apparently, by this mechanism, an electrophilic benzylic carbonium ion is generated which can then bind to DNA.

Contrasting some of the earlier views on the mechanisms involved in PAH mutagenicity, most likely no single mechanism can be found to dominate metabolic activation of PAHs as a whole. Rather the relative significance of each mechanism apparently will depend on variable factors such as the chemical/biological characteristics of each individual compound or the level of expression of activation enzymes involved.

An overview of statements on the mutagenicity of PAHs considered in this restriction proposal is given in Table 11.

Table 11: Genotoxicity/carcinogenicity of polycyclic aromatic hydrocarbons: overall evaluation

Substance (CAS no.)	**Mutagenicity**				**Carcinogenicity**	
	Classification/ labelling	**WHO/ IPCS (1998)**	**EC (2002)**	**FAO/WHO (2006)**	**Classification/ labelling**	**Mutagenic mode of action (US EPA, 2007)**
Benzo[a]anthracene (56-55-3)	No	Genotoxic	Genotoxic (positive results in vitro and in vivo for multiple end-points; positive also at germ cell level)	Genotoxic, both *in vitro* and *in vivo*	Carc. Cat. 2, R45[1] Carc. 1B, H350[2]	x
Benzo[a]pyrene (50-32-8)	Mut. Cat. 2, R46[1] Muta. 1B, H340[2]	Genotoxic	Genotoxic (positive results in vitro and in vivo for multiple end-points; positive also at germ cell level)	Genotoxic, both *in vitro* and *in vivo*	Carc. Cat. 2, R45[1] Carc. 1B, H350[2]	x
Benzo[b]fluoranthene (205-99-2)	No	Genotoxic	Genotoxic (positive results in vitro and in vivo for multiple end-points)	Genotoxic, both *in vitro* and *in vivo*	Carc. Cat. 2, R45[1] Carc. 1B, H350[2]	x
Benzo[e]pyrene (192-97-2)	No	Genotoxic	Equivocal results (mixed results in vitro; inconsistent results in vivo)	-	Carc. Cat. 2, R45[1] Carc. 1B, H350[2]	-
Benzo[j]fluoranthene (205-82-3)	No	Genotoxic	Genotoxic (positive results *in vitro* and for DNA binding *in vivo*)	Genotoxic, both *in vitro* and *in vivo*	Carc. Cat. 2, R45[1] Carc. 1B, H350[2]	-

Substance (CAS no.)	Mutagenicity				Carcinogenicity	
	Classification/ labelling	WHO/ IPCS (1998)	EC (2002)	FAO/WHO (2006)	Classification/ labelling	Mutagenic mode of action (US EPA, 2007)
Benzo[k]fluoranthene (207-08-9)	No	Genotoxic	Genotoxic (positive results *in vitro* and for DNA binding *in vivo*)	Genotoxic, both *in vitro* and *in vivo*	Carc. Cat. 2, R45[1] Carc. 1B, H350[2]	x
Chrysene (218-01-9)	Mut. Cat. 3, R68[1] Muta. Cat. 2, H341[2]	Genotoxic	Genotoxic (positive results *in vitro* and *in vivo* for multiple end-points; positive also at germ cell level)	Genotoxic, both *in vitro* and *in vivo*	Carc. Cat. 2, R45[1] Carc. 1B, H350[2]	x
Dibenzo[a,h]anthracene (53-70-3)	No	Genotoxic	Genotoxic (positive results *in vitro* and *in vivo* for multiple end-points)	Genotoxic, both *in vitro* and *in vivo*	Carc. Cat. 2, R45[1] Carc. 1B, H350[2]	x

[1] Classification under Directive 67/548/EEC; [2] Classification and hazard statements under Regulation (EC) No 1272/2008

BaP and CHR are classified as mutagenic (Directive 67/548/EEC; Regulation (EC) No 1272/2008). Harmonised (= legally binding) classification and labelling of the other PAHs for this endpoint has not been proposed and discussed in the EU committees for classification and labelling up to now.

PAHs were evaluated as genotoxic in an overview report by the IPCS (International Programme on Chemical Safety, 1998).

On the basis of available data EC (European Commission, 2002) and FAO/WHO (Food and Agriculture Organization of the United Nations/World Health Organization, 2006) characterised 7/8 of the considered PAHs as genotoxic. In contrast to the IPCS (1998), the EU (2002) has evaluated the same mutagenicity data for benzo[e]pyrene as equivocal. For the assessment of the mutagenic effect from benzo[e]pyrene no additional *in vivo* data of standard mutagenicity tests are available.
Within the group of PAHs evidence of genotoxicity overlaps with that for carcinogenicity (Table 11 above). All considered PAHs have been classified with regard to their carcinogenicity. For a mutagenic mode of action, a carcinogen or one of its metabolites has to be DNA-reactive and/or has to be able to bind to DNA. EC (2002) concluded in agreement with the mechanistic link between DNA adduct formation, mutations, and carcinogenesis that the considered PAHs with exception of benzo[e]pyrene (because of its equivocal mutagenicity data) have a mutagenic mode of action. The US EPA (2007) has identified six out of the eight PAHs in this dossier as possible carcinogens via a mutagenic mode of action.

BaP, the most studied PAH, has been accepted as reference compound for PAHs. In a large number of publications it has been shown that BaP induces DNA adducts as well as mutagenic effects such as gene mutations or chromosomal aberrations *in vitro* and *in vivo* in rodents. Therefore, only some publications, which were published after the overview report of WHO/IPCS (1998) were referred exemplarily. In mutagenicity tests with rodents it was shown that BaP induces mutagenic effects at the site of application in the skin after dermal exposure (Dean et al., 1998; Nishikawa et al., 2002)

as well as in the lung after intratracheal exposure (Garry et al., 2003, Hashimoto et al., 2005) and in the viscera after oral exposure (Hakura et al., 1998).

In the last years, further studies were carried out to clarify details of chemical mechanisms of DNA adduct formation (Hashimoto et al., 2005; Sagredo et al., 2006; Wu et al., 2008) as well as the role of PAH-DNA adducts in the context of carcinogenicity (Baird et al., 2005). Some studies were carried out in order to compare the *in vivo* mutagenicity and carcinogenicity induced by BaP in different organs. Thus Hakura et al. (1998) investigated the induction of mutagenic effects and carcinogenicity by BaP under the same experimental conditions in different organs after oral administration of 125 mg/kg bw BaP for a period of 5 consecutive days using the mutagenicity test system lacZ transgenic mice (Muta^{TM}Mouse). The highest mutant frequency was observed in the colon, followed in descending order by the ileum > forestomach > bone marrow, spleen > glandular stomach > liver, lung > kidney and heart. No significantly increased rate was found in the brain. BaP induced squamous cell carcinoma, papilloma, and hyperplasia in the forestomach and malignant lymphoma primarily in the lymphatic organs including the spleen. Furthermore, bronchio-alveolar hyperplasia was induced in the lung. No tumours were observed in the other organs. These results indicate that the magnitude of *in vivo* lacZ mutant frequencies induced by BaP in different organs did not fully correlate with the target organs for carcinogenicity.

Hakura et al. (1999) confirmed this result in a further study in which they examined frequencies of lacZ gene mutations in target organs (forestomach, spleen, lung) and non-target organs (colon, glandular stomach, liver) of BaP carcinogensis in Muta^{TM}Mice treated with an oral dose of 125 mg/kg bw BaP for five consecutive days. The *in vivo* mutation analysis indicated that mutation frequencies were markedly increased above spontaneous frequencies in all organs. The highest mutation frequency was observed in the colon followed in descending order by forestomach > spleen > glandular stomach, liver and lung.

The observations made in both studies have clearly shown that the generation of tumours requires the induction of mutations as well as other factors which are specific for the target organ.

A corresponding conclusion was given by Kroese et al. (2001) who determined DNA-adducts by ^{32}P-postlabelling after oral administration of 0.1 mg/kg bw BaP in different tissues of rats parallel to a carcinogenicity study. The results indicated that DNA-adducts were present in tissues in which tumours developed (forestomach and liver) as well as in tissues in which no tumour development was observed. The authors concluded that the generation of tumours requires the induction of mutations as well as factors specific to the target organs and that local cell proliferation might be such a critical factor.

In summary it can be stated that the formation of DNA-adducts by reactive intermediates of PAHs may be an indicator of the earliest step in the carcinogenesis. Due to the ability to induce genotoxic effects the existence of a threshold value below which no health risks exist can be excluded for mutagenic PAHs.

B.5.8 Carcinogenicity

The PAHs covered by this restriction proposal (benzo[a]pyrene (BaP), benzo[e]pyrene (BeP), benzo[a]anthracene (BaA), chrysene (CHR), benzo[b]fluoranthene (BbFA), benzo[j]fluoranthene (BjFA), benzo[k]fluoranthene (BkFA), and dibenzo[a,h]anthracene (DBAhA)) are identified as

carcinogenic substances according Regulation (EC) No 1272/2008 and Directive 67/548/EEC. Furthermore BaP and CHR are classified for their mutagenic effects (cf. Table 2 above).

As regards the eight PAHs evaluated in this dossier, it is considered worthwile to point to the conclusion of the International Agency for Research of Cancer (IARC 2006):

> *'[…] There is sufficient evidence in experimental animals for the carcinogenicity of benz[a]-anthracene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, benzo[a]-pyrene, chrysene, […], dibenz[a,h]anthracene […]. There is inadequate evidence in experimental animals for the carcinogenicity of […] benzo[e]pyrene […] Benzo[a]pyrene is carcinogenic to humans (Group 1).[...] dibenz[a,h]anthracene and [...]… are probably carcinogenic to humans (Group 2A). benz[a]anthracene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, chrysene, dibenzo[a,h]pyrene, are possibly carcinogenic to humans (Group 2B). Benzo[e]pyrene is not classifiable as to its carcinogenicity to humans (Group 3)[...]'*

The Scientific Committee on Toxicity, Ecotoxicity and the Environment of the European Commission (SCTEE) concluded in 2003:

> *'[…] The **carcinogens beyond reasonable doubt** are benz[a]anthracene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene and have been classified by IARC.[…] Thus, the SCTEE considers PAHs as likely carcinogens for man [...]'*

Reviews of PAH toxicity, in particular on possible carcinogenic effects due to exposure to PAHs conducted since 1990 by Health Canada/Environment Canada (EC/HC, 1994), the US Agency for Toxic Substances and Disease Registry (ATSDR, 1995), the European Food Safety Authority (EFSA, 2008), the US Environmental Protection Agency (US EPA, 2002, 2003), the Dutch Expert Committee on Occupational Standards, a committee of the Health Council of the Netherlands (HCN, 2006), the International Agency for Research on Cancer (IARC, 1998, 2006), the German Senate Commission for the Testing of Harmful Working Materials (MAK Commission, 2008), the World Health Organization WHO, 1998, 2003) were searched to obtain citations of adequate animal and human cancer data. In addition, several current databases of primary literature published since 2003 were searched for cancer bioassays or life-time studies with PAHs and/or benzo[a]pyrene (cf. above, section B.5). Long-term animal studies by the oral, inhalation, or dermal route of exposure identified within these review documents were collected and evaluated for acceptability (i.e., use of control groups, adequate dose spacing, clear identification of dose levels, presence of a dose response relationship, statistically significant differences compared to controls). Those which met these criteria were selected for evaluation of their suitability for further estimations.

The above-mentioned database was also evaluated for evidence of a relationship between occupational PAH exposure and cancer in humans. More recent data were interpreted in particular by Armstrong et al. (1994, 2003, 2004, 2009), Constantino et al. (1995), Mastrangelo et al. (1996), Boffetta et al. (1997), Moolgavkar et al. (1998), and Bosetti et al. (2007). These included cohort and case-control studies with exposure to various PAH-rich sources (e. g. evaporation of carbon electrode materials, coal tar distillation and purification, coke production, thermal decomposition of organic materials) in various industries (e.g. aluminium production, coal gasification, and coke production, iron and steel foundry). Most of the available human data come from inhalation and dermal exposure to PAHs in a large range of occupational settings (Greim, 2008). They vary with

respect to type of industry and workplace. However, in more than half of the cases, no information with regard to the exposure profile and/or the air concentrations of the different PAHs is presented.

In these occupations, often PAHs were not the only compounds to which workers were exposed. There was also exposure to other chemicals, including organic solvents, nitro-PAHs, aromatic amines and metals, but also dust particles and in some cases asbestos. Some of these chemicals are themselves known carcinogens (HCN, 2006). In addition, many studies were confounded by smoking. These epidemiological studies confirm that high occupational exposure levels to mixtures of PAHs entail a substantial risk of lung, skin, or bladder cancer. The lung seems to be the major target organ of PAH carcinogenicity and an increased risk was observed for most of the industries investigated. An increased risk of skin cancer follows high dermal exposure, an increase in bladder cancer risk is found mainly in industries with high exposure to PAHs from coal tars and pitches. However, no studies could be identified which were able to demonstrate cancer in humans as a consequence of exposure to BaP or other single PAH compounds alone, i. e. without any confounding factors(s).

Numerous animal studies have been published on the carcinogenic effects of PAHs, as single compounds or as mixtures, by various routes of exposure. A number of individual PAHs are carcinogenic to experimental animals indicating that they are potentially carcinogenic to humans (HCN, 2006).

BaP is the best-studied PAH. It has been tested in multiple animal species and is the only carcinogenic PAH for which carcinogenicity studies along all routes of exposure (i. e. oral, inhalation, and dermal) are available. It has been shown to be carcinogenic by all routes tested in a number of animal species. Thus, for the purpose of this dossier it was considered the most adequate approach to use BaP as the lead compound for assessment of all the PAHs addressed by this restriction proposal.

Within the purpose of this restriction dossier it is not intended to re-evaluate the carcinogenic potential of the already classified eight PAHs. In fact, carcinogenicity studies were assessed with the main purpose of identifying the most suitable starting point(s) for DMEL calculation in order to support quantitative risk characterisation.

B.5.8.1 Carcinogenicity: oral

Three long-term studies of recent date were selected, a study in rats treated by gavage with BaP and two feeding studies in mice, one comparing pure BaP with two coal tar mixtures and a similar experiment with BaP and a PAH-rich manufactured gas plant residue. These studies are considered as the most adequate studies for the derivation of DMEL(s) for the oral route of exposure.

Following oral administration of pure BaP or PAH mixtures, induction of local and systemic tumours has been noted in rats and mice. In rats, treatment-related tumours were found in the gastrointestinal tract and liver, and mice studies revealed tumours in the gastrointestinal tract, liver and respiratory tract.

B.5.8.1.1 Rat

B.5.8.1.1.1 Lifetime gavage study in rats (Kroese et al., 2001)

A combined chronic and carcinogenicity study in Wistar rats clearly showed BaP to be a potent carcinogen upon chronic oral administration. Administration of oral doses of 0, 3, 10, or 30 mg BaP/kg bw/d by gavage to groups of male and female Wistar rats (n = 52/group) on 5 days per week for 104 weeks resulted in a dose-dependent increase in tumour incidences in a large variety of organs, i. e., liver, forestomach, auditory canal, oral cavity, skin, intestines, (in males only) kidney, and the mammary and oesophagus in females. The most potent carcinogenic effects of BaP under these testing conditions were observed in the liver and forestomach, while for both organs a low spontaneous incidence was noted in this rat strain. Papillomas and carcinomas were observed in the forestomach, and adenomas and carcinomas in the liver of both female and male rats. Tumours were found at the lowest dose tested (3 mg/kg bw/d), though at a (borderline) non-significant incidence. Statistically significant incidences were observed at 10 mg/kg bw/d and above.

Liver tumours were also responsible for morbidity and the high mortality rate at the highest dose level in both sexes (100 % after about 70 weeks). Mortality was mainly due to sacrifice for humane reasons when rats became emaciated, often with distended abdomen in which frequently one ore more palpable masses were present in the cranial area (liver). In control animals, survival after 104 weeks was about 65 % and 50 % in males and females, respectively. The main cause of death was tumour development in the pituitary, which was consistent with earlier findings in historical controls of this laboratory (Kroese et al., 2001).

Table 12: Incidence of tumours in liver and forestomach in male and female Wistar rats following treatment with pure BaP

Dose (mg/kg bw/d)	**0**		**3**		**10**		**30**	
Sex Tumour site	**M**	**F**	**M**	**F**	**M**	**F**	**M**	**F**
Liver	0/52	0/52	4/52	2/51	**38/52**	**39/51**	**49/52**	**51/52**
Forestomach	0/52	1/52	8/52	6/51	**43/52**	**39/51**	**52/52**	**50/52**
Auditory Canal	0/52	0/52	0/52	0/51	2/52	0/51	**23/52**	**14/52**

Note: bold print of dose values means $p < 0.00001$, Fisher's exact test

The lowest dose level associated with a significantly increased tumour response in male and female Wistar rats was identified at 10 mg/kg bw/d. At this dose level significantly increased hepatocellular adenomas and carcinomas in the liver as well as squamous cell papillomas and carcinomas in the forestomach were induced in animals of both sexes:

Liver tumours: 10 mg BaP/kg bw/d
Forestomach tumours: 10 mg BaP/kg bw/d

B.5.8.1.2 Mouse

B.5.8.1.2.1 Lifetime feeding study in mice (Culp et al., 1998/Schneider et al., 2002)

In a 2-year carcinogenicity study, female B6C3F1 mice (n= 48/group) were fed pure BaP or two different coal tar mixtures containing high amounts of several PAHs. Two additional groups of 48 mice each served as controls, one group was fed the standard diet, while the other was fed the standard diet treated with acetone in a manner identical to the BaP diets (Culp 1998; Schneider et al., 2002).

BaP

BaP was fed at concentrations of 5, 25, or 100 ppm in the diet (equivalent to doses of 20.5, 104 or 430 µg/d). Papillomas and carcinomas were observed in the forestomach, oesophagus, and tongue. All of the mice fed 100 ppm BaP were removed by 80 weeks because of morbidity or death. A significant ($p = 0.0009$) number of mice treated with 25 ppm also died early. The percentage survival of mice fed 5 ppm (56 %) was similar to the control group. The increase in incidence of neoplasms was related to dose, with high statistical significance in the 25 and 100 ppm groups.

Table 13: Incidence of neoplasms in female B6C3F1 mice fed pure BaP

Conc. in food (ppm)	**0**	**5**	**25**	**100**
BaP (mg/kg bw/d) [1] Tumour site	**0**	**0.65**	**3.25**	**13.0**
Forestomach	1/48 (2 %)	3/46 (6 %)	36/46 (78 %)	46/47 (98 %)
Oesophagus	0/48 (0 %)	0/48 (0 %)	2/45 (4 %)	27/46 (59 %)
Tongue	0/48 (0 %)	0/48 (0 %)	2/46 (4 %)	23/48 (48 %)

[1] The values were calculated according to REACH Guidance (Appendix R.8-2, Table R.8-17) for female mice: 25 g bw, daily food consumption: 130 g food/kg bw/d; Note: bold print means significantly different ($p < 0.05$) from control.

Coal tar mixtures

Coal tar mixture 1 (CTM 1, CAS No 8007-45-2) was a standardised composite from seven manufactured gas plant waste sites and coal tar mixture 2 (CTM 2) was a composite from two of the seven waste sites plus a third site having a very high BaP content. The composition of CTM 1 and CTM 2, respectively, is given in Table 14.

Table 14: PAH composition of CTM 1 and CTM 2

Compound	**CTM 1 (mg/kg)**	**CTM 2 (mg/kg)***
Acenaphthene	2049	1270
Acenaphthylene	3190	5710
Anthracene	2524	2900
Benz[a]anthracene	2374	3340
Benzo[b]fluoranthene	2097	2890
Benzo[k]fluoranthene	699	1010
Benzo[g,h,i]perylene	1493	2290

Compound	**CTM 1 (mg/kg)**	**CTM 2 (mg/kg)***
Benzo[a]pyrene	1837	2760
Chrysene	2379	2960
Dibenz[a,h]anthracene	267	370
Dibenzofuran	1504	1810
Fluoranthene	4965	6370
Fluorene	3692	4770
Indan	1133	490
Indeno[1,2,3-cd]pyrene	1353	1990
1-Methylnaphthalene	6550	5660
2-Methylnaphthalene	11289	10700
Naphthalene	22203	32300
Phenanthrene	7640	10100
Pyrene	5092	7220

* This material is identical to the one used by Weyand et al. (1995) under the name of 'MGP'

CTM 1 was given at concentrations of 0.01, 0.03, 0.1, 0.3, 0.6, and 1.0 % in the diet, which contained BaP at a concentration of 1837 mg/kg (equivalent to dose levels of 0.029, 0.086, 0.286, 0.858, 1.742, and 2.86 mg/kg bw/d). None of the mice fed 1.0 % survived the treatment period. The mortality rate for the mice fed 0.6 % was also 100 % already at an early stage. Only 10 mice (21 %) in the 0.3 % group survived to the end of the 2-year treatment, a difference that was significant ($p = 0.00006$) from the control group. The survival for the mice in the control group was 65 %. In the 0.01, 0.03, and 0.1 % CTM 1 dose groups the survival rate was 71, 69 and 63 %, respectively.

CTM 2, which contained BaP at a concentration of 2760 mg/kg, was given at concentrations of 0.03, 0.1, and 0.3 % in the diet (equivalent to dose levels of 0.143, 0.481, and 1.443 mg/kg bw/d). Survival was significantly ($p = 0.00003$) lower in the 0.3 % dose group (15 %) as compared to the control group (65 %). The survival in the remaining two dose groups was similar to the control group.

Both coal tar mixtures induced a dose-dependent increase in tumours at various locations, i. e. in the liver: hepatocellular adenomas and carcinomas, in the lung: alveolar/bronchiolar adenomas and carcinomas, in the forestomach: squamous epithelial papillomas and carcinomas, in the small intestine: adenocarcinomas, histiocytic sarcomas, and, furthermore, haemangiosarcomas in multiple organs, and sarcomas. The incidence of CTM1-induced tumours in forestomach, lung, and liver in female B6C3F1 mice is given in Table 15.

Table 15: Incidence of forestomach, lung and liver tumours in female B6C3F1 mice fed CTM 1

Conc. in food (%)	**0**	**0.01**	**0.03**	**0.1**	**0.3**	**0.6**	**1.0**
BaP (mg/kg bw/d)[1] Tumour site	**0**	**0.029**	**0.086**	**0.286**	**0.858**	**1.742**	**2.86**
Forestomach	0/47 (0 %)	2/47 (4 %)	6/45 (13 %)	3/47 (6 %)	**14/46 (30 %)**	**15/45 (33 %)**	**6/41 (15 %)**
Lung	2/47 (4 %)	3/48 (6 %)	4/48 (8 %)	4/48 (8 %)	**27/47 (57 %)**	**25/47 (53 %)**	**21/45 (47 %)**
Liver	0/47 (0 %)	4/48 (8 %)	2/46 (4 %)	3/48 (6 %)	**13/45 (31 %)**	1/42 (2 %)	**5/43 (12 %)**

[1] The values were calculated according to REACH Guidance (Appendix R.8-2, Table R.8-17) for female mice: 25 g bw, daily food consumption: 130 g food/kg bw/d. Note: bold print means significantly different ($p<0.05$) from control

A statistically significantly increased incidence of tumours was noted at ≥ 0.3 % (equivalent to ≥ 0.858 mg BaP/kg bw/d) in:

Lung: 27/47 (57 %)
Liver: 14/45 (31 %)
Forestomach: 14/46 (30 %).

Schneider et al. (2002) used the original, unpublished raw data from Culp and co-workers (1998) in order to establish the total number of tumour-bearing animals at each dose level. The results for CTM 1 can be found in Table 16.

Table 16: Number of tumour-bearing female B6C3F1 mice fed CTM 1

Conc. in food (%)	**0**	**0.01**	**0.03**	**0.1**	**0.3**	**0.6**	**1.0**
BaP (mg/kg bw/d)[1]	**0**	**0.029**	**0.086**	**0.286**	**0.858**	**1.742**	**2.86**
No. of tumour-bearing animals	5/47 (11 %)	**12/48 (25 %)**	**14/48 (29 %)**	**12/48 (25 %)**	**40/48 (83 %)**	**42/48 (88 %)**	**43/48 (90 %)**

[1] The values were calculated according to REACH Guidance (Appendix R.8-2, Table R.8-17) for female mice: 25 g bw, daily food consumption: 130 g food/kg bw/d.; Note: bold print means significantly different ($p<0.05$) from control

As can be seen, the number of tumour-bearing mice was statistically significantly increased already at the lowest dose level of 0.01 %/0.029 mg BaP/kg bw/d. However, the dose-response relationship was not monotonous in the lower dose range, which can be explained by the fact that by the parameter chosen (no. of tumour-bearing animals) both substance-related and spontaneous neoplasms were counted side-by-side.

The incidence of tumours in forestomach, lung, and liver observed in female B6C3F1 mice treated with CTM2 are given in Table 17.

Table 17: Incidence of forestomach, lung, and liver tumours in female B6C3F1 mice fed CTM 2

Conc. in food (%)	**0**	**0.03**	**0.1**	**0.3**
BaP (mg/kg bw/d)[1] Tumour site	**0**	**0.143**	**0.481**	**1.443**
Forestomach	0/47 (0 %)	3/47 (6 %)	2/47 (4 %)	**13/44 (30 %)**
Lung	2/47 (4 %)	4/48 (8 %)	**10/48 (21 %)**	**23/47 (49 %)**
Liver	0/47 (0 %)	7/47 (15 %)	4/47 (9 %)	**10/45 (22 %)**

[1] The values were calculated according to REACH Guidance (Appendix R.8-2, Table R.8-17) for female mice: 25 g bw, daily food consumption: 130 g food/kg bw/d.; Note: bold print means significantly different ($p<0.05$) from control

Statistically significantly increased incidences of tumours were noted for

Lung: ≥ 0.1 %: 10/48 (21 %)
Liver: 0.3 %: 10/45 (22 %)
Forestomach: 0.3 %: 13/44 (30 %).

Schneider et al. (2002) used the original, unpublished raw data from Culp and co-workers in order to establish the total number of tumour-bearing animals at each dose level. The results for CTM 2 can be found in Table 18.

Table 18: Number of tumour-bearing female B6C3F1 mice fed CTM2

Conc. in food (%)	**0**	**0.03**	**0.1**	**0.3**
BaP (mg/kg bw/d)[1] Test item	**0**	**0.143**	**0.481**	**1.443**
No. of tumour-bearing animals/no. of all animals	5/48 (10 %)	**17/48 (35 %)**	**23/48 (48 %)**	**44/48 (92 %)**

[1] The values were calculated according to REACH Guidance (Appendix R.8-2, Table R.8-17) for female mice: 25 g bw, daily food consumption: 130 g food/kg bw/d.; Note: bold print means significantly different ($p<0.05$) from control

As can be seen, the number of tumour-bearing mice was statistically significantly increased already at the lowest dose level of 0.03 %/0.143 mg BaP/kg bw/d.

In summary, this study with female B6C3F1 mice, feeding of pure BaP resulted in an increased incidence of papillomas and/or carcinomas of the forestomach, oesophagus, and tongue. The coal tar mixture diets induced a dose-related increase in hepatocellular adenomas and/or carcinomas; alveolar/bronchiolar adenomas and or carcinomas as well as papillomas and/or carcinomas of the forestomach, adenocarcinomas in the small intestine, histiocytic sarcomas, and haemangiosarcomas in various organs.

A comparison of the results of the different feeding experiments in female B6C3F1 mice indicated that pure BaP induced tumours of the alimentary tract at ≥ 5 ppm (equivalent to doses of ≥ 0.65 mg/kg bw/d), significantly at ≥ 25 ppm (equivalent to doses of ≥ 3.25 mg/kg bw/d), whereas the coal tar mixtures - in addition to forestomach tumours - also induced a significant dose-

dependent increase in lung tumours (≥ 0.481 mg BaP/kg bw/d) and liver tumours (≥ 0.858 mg BaP/kg bw/d).

The lowest dose levels associated with a significantly increased tumour response in female B6C3F1 mice were identified at:

Lung: 0.481 mg/kg bw/d BaP
Liver: 0.858 mg/kg bw/d BaP
Forestomach: 0.858 mg/kg bw/d BaP

B.5.8.1.2.2 Lifetime feeding study in mice (Weyand et al., 1995/Schneider et al., 2002)

In another study, groups of female A/J mice (n=30/group) were used for a similar feeding experiment with pure BaP and a PAH-rich manufactured gas plant residue. This mouse strain was chosen because of its sensitivity to chemical induction of pulmonary adenomas. A negative control group was fed the basal gel diet. In addition, a non-treated group of mice and a group dosed with vehicle only were fed with a NIH-07 pellet diet and used as negative controls. A further group served as positive control and was administered pure BaP (100 mg/kg) by i.p. injection in 0.25 mL of tricaprylin. After the last exposure day, the animals were sacrificed and their lungs and stomach removed for histology (Weyand et al., 1995; Schneider et al., 2002).

In this study, the test item was denominated as 'Manufactured Gas Plant Residue' (MGP). MGPs, commonly also referred to as coal tar, are waste by-products formed in large quantities during coal gasification. It is noted that the test material used in this study was the same as the one designated 'CTM 2' by Culp and co-workers (1998, cf. above) and the levels of the individual PAH congeners can be found in the corresponding table above.

<u>BaP</u>

BaP was fed at concentrations of 16 or 98 ppm (40.6 or 256.6 µg BaP/day/mouse, equivalent to doses of 1.624 or 10.264 mg BaP/kg bw/d, respectively) in the diet. The survival rate for both treatment groups was 25/30 and 27/30, respectively. In the control group 21/30 mice survived to the end of the study. Increased numbers of tumours in the forestomach and the lung were induced after treatment with pure BaP in feed for 260 days. In Table 19, the incidence of forestomach and lung tumours is presented.

Table 19: Incidence of forestomach and lung tumours in female A/J mice fed pure BaP

BaP Conc. in food	**0**	**16 ppm**	**98 ppm**
BaP (mg/kg bw/d)[1] **Tumour site**	**0**	**1.624**	**10.264**
Forestomach	0/21 (0 %)	**5/25 (20 %)**	**27/27 (100 %)**
Lung	4/21 (19 %)	**9/25 (36 %)**	**14/27 (52 %)**

[1] The values were calculated according to REACH Guidance (Appendix R.8-2, Table R.8-17) for female mice: 25 g bw, daily food consumption: 130 g food/kg bw/d; Note: Bold print means significantly different ($p<0.05$) from control; determined by x^2 test

<u>MGP</u>

MGP, which contained BaP at a concentration of 2760 mg/kg, was given at concentrations of 0.1 or 0.25 % in the diet (6.9 or 16.3 µg BaP/mouse/d, equivalent to doses of 0.276 or 0.652 mg BaP/kg

bw/d). The survival rate for the both treatment groups was 27/30 and 29/30, respectively. Treatment with MGP induced development of tumours in the lung. No local tumours in the forestomach were noted. The effect of MGP ingestion on the development of lung tumours is given in Table 20.

Table 20: Incidence of lung tumours in female A/J mice fed MGP

MGP Conc. in food (%)	**0**	**0.10**	**0.25**
BaP (mg/kg bw/d)[1] **Tumour site**	**0**	**0.276**	**0.652**
Lung	4/21 (19 %)	**19/27 (70 %)**	**29/29 (100 %)**

[1] The values were calculated according to REACH Guidance (Appendix R.8-2, Table R.8-17) for female mice: 25 g bw, daily food consumption: 130 g food/kg bw/d; Bold print means significantly different ($p<0.05$) from control; determined by x^2 test

In the study with female A/J mice increased numbers of tumours in lungs were found after treatment with MGP (statistically significant at 0.10 % MGP, containing ≥ 0.276 mg BaP/kg bw/d) in feed for 260 days. Lung tumour induction in mice fed pure BaP was considerably lower than for those fed MGP diet. A diet containing pure BaP at 98 ppm (10.264 mg/kg bw/d) produced a statistically significant incidence of tumour-bearing mice with 52 % developing lung tumours. In the controls, 19 % showed pulmonary adenomas. The ingestion of 16 or 98 ppm BaP in the diet resulted in 20 and 100 % of the mice developing forestomach tumours, respectively. The incidence of forestomach carcinomas in mice was 8 and 52 %, respectively. However, neither in animals fed MGP nor in controls, forestomach tumours were found.

In summary, the lowest dose level associated with a significantly increased tumour response in female A/J mice was identified at:

Lung: 0.276 mg BaP/kg bw/d
Forestomach: 1.642 mg BaP/kg bw/d

B.5.8.2 Carcinogenicity: inhalation

In reviews, reference to carcinogenic effects of PAH-containing mixtures or pure BaP by the inhalation route was limited to a small number of studies. Inhalation of PAH or BaP caused lung tumours in rats and mice and respiratory tract tumours in hamsters. Regarding the possible use for calculating cancer risk values and for derivation of DMELs for the inhalation route of exposure, the most relevant studies are presented in the following paragraph. The studies in rats, mice, and hamsters were evaluated as adequate animal cancer data and were selected for further estimations.

B.5.8.2.1 Rat

B.5.8.2.1.1 Chronic inhalation study in rats (Heinrich et al., 1994)

In a chronic inhalation study, female Wistar rats were exposed to coal tar pitch (CTP) aerosol in order to estimate lifetime unit lung cancer risk for BaP. A total of 5 experimental groups, each consisting of 72 females, 7 weeks of age at the start of the experiment, were used in this inhalation study. Two different concentrations of CTP aerosol were used, 1.1 and 2.6 mg/m^3, which contained a. o. 20 and 46 µg BaP/m^3, respectively, resulting in cumulative doses of inhaled BaP of 71 mg BaP/m^3/h (43-wk exposure), 142 mg BaP/m^3/h (86-wk exposure), 158 mg BaP/m^3/h (43-wk exposure), and 321 mg BaP/m^3/h (86-wk exposure), respectively. The concentrations of some other

particle-bound and gaseous PAHs, as they occurred in the 2.6 mg CTP/m3 exposure atmosphere, are listed in Table 21.

Table 21: Concentration of some particle-bound and gaseous PAHs in the exposure chamber containing 2.6 mg CTP aeroosol/m^3

PAHs and PAH-related compounds	Concentration of particle-bound PAHs (µg/m^3)	Concentration of gaseous PAHs (µg/m^3)
Acenaphthene		38
Acenaphthylene		9
Anthracene	11	3
Benz[*a*]antracene	58	
Benzo[*a*]fluorine	20	
Benzo[*a*]pyrene	46	
Benzo[*b*]fluorine	23	
Benzo[*c*]phenanthrene	7	
Benzo[*e*]pyrene	39	
Benzo[*g,h,i*]fluoranthene	8	
Benzo[*g,h,i*]perylene	27	
Benzofluoranthenes	93	
Benzonaphthothiophene	12	
Carbazole	6	
Chrysene	59	
Coronene	7	
Cyclopenta[*d,ed]*phen.	8	
Cyclopenta[*d,ef*]phen		1
Dibenzofurane		14
Dibenzothiophene	3	2
Fluoranthene	87	1
Fluorene		17
Indeno[*1,2,3-c,d*]pyrene	29	
Methylbiphenyl		7
Methyldibenzofurane		5
Methylfluorene		3
Perylene	11	
Phenanthrene	50	
Phenanthrene		13
Pyrene	67	

Animals were exposed to filtered clean air or the aerosols of CTP, free of any carbon black carrier particles, for 17 hours per day, 5 days per week for 10 or 20 months, followed by a clean air period

of up to 20 or 10 months, respectively. Thus, the total experimental time for all groups was 30 months. The mass median aerodynamic diameter of the CTP aerosol was 0.5 µm (Heinrich et al., 1994).

A clear dose-dependent increase in lung tumour incidence was observed. Most tumours were classified as keratinising squamous cell tumours, but also some bronchio-alveolar adenomas and adenocarcinomas were found. No exposure-related tumours were observed in organs other than the lung, however other organs were not evaluated for neoplasms. The incidence of lung tumours in female rats chronically exposed to the aerosols of CTP is given in Table 22.

Table 22: Incidence of lung tumours in female Wistar rats chronically exposed to CTP by inhalation

BaP (µg/m³)	BaP (mg/kg bw/d)[1]	Exposure (months)	Post exposure (months)	Cumulative Exposure (mg BaP/m³ x h)	Lung tumour incidence
0	0	0	30	0	0/72 (0 %)
20	0.0148	10	20	71	3/72 (4.2 %)
20	0.0148	20	10	142	24/72 (33.3 %)
46	0.0341	10	20	158	28/72 (38.9 %)
46	0.0341	20	10	321	70/72 (97.2 %)

[1] The values were calculated according to REACH Guidance (Appendix R.8-2, Table R.8-17) for female rats: 275 g bw, respiratory rate: 200 mL/min

When compared to controls, increased mortality rates were observed in the groups exposed to 2.6 mg/m^3 CTP aerosol (46 µg/m^3 BaP) for 10 or 20 months. In particular, the animals exposed for 20 months had to be sacrificed because of the development of large, multiple tumours in the lung. No lung tumours were found in control animals.

In summary, the lowest tested concentration of 1.1 mg/m³ CTP containing 20 µg/m^3 BaP, associated with a significantly increased tumour response in the lung after exposure for 10 (4.2 %) or 20 months (33.3 %) in female Wistar rats, should be used as a point of departure/starting point for further estimations.

The concentration of 20 µg BaP/m^3 (≙0.00002 mg/L) is equivalent to 0.0148 mg/kg bw/d.

B.5.8.2.2 Mouse

B.5.8.2.2.1 Long-term inhalation study in newborn mice (Schulte et al., 1994)

In a long-term inhalation study, newborn female NMRI/BR mice were used to study carcinogenic effects of PAH-rich exhausts. The use of newborn animals was explained by their lower spontaneous lung tumour incidence and greater susceptibility to tumour induction. Exposure started at the first day after birth. The animals (n=40/group) were exposed to filtered room air or coal tar pitch volatile aerosols (mass median aerodynamic diameter, MMAD of 0.55 ± 0.03 µm), containing 50 or 90 µg BaP/m^3 (0.05 or 0.09 mg/m^3), for 16 hours per day, 5 days per week during 44 exposure weeks. The PAH-rich exhaust was produced by pyrolysing preheated (80 °C) coal tar pitch under a nitrogen atmosphere at 750-800 °C, which was then diluted with fresh air and was transferred into the exposure chambers. BaP served as the lead compound for standardising the exposure concentrations. The animals of the control group were exposed to filtered room air (Schulte 1994).

The number of surviving mice at termination of the experiment was slightly lower in the PAH-rich exhaust exposure groups [50 µg/m^3: 38/40 (95 %); 90 µg/m^3: 35/40 (87.5 %)] as compared to controls [39/40 (97.5 %)], but average lifetime was nearly the same in all groups (44.2 - 44.4 weeks).

Results of the macroscopic and microscopic analysis of the lung clearly demonstrated that exposure to PAH-rich exhausts caused a dose-dependent increase in lung tumours. As in the previous study, tumours in organs other than the lung were not investigated. Bronchiolo-alveolar adenomas were observed in all mice exposed to 50 or 90 µg BaP/m^3 BaP (40/40 each) as compared to 5/40 in the control group. Bronchiolar-alveolar adenocarcinomas had developed in 10/40 and in 33/40 mice (see detailed study results in Table 23.

Table 23: Macroscopic and microscopic results of long-term exposure of female NMRI/BR mice to PAH-rich exhausts

Exposure	BaP (mg/kg bw/d)[1]	Lung cancer mortality abs. (%)	Average no. of nodules per lung	Number of adenomas abs. (%)	Number of adeno-carcinomas abs. (%)	No. of squamous cell carcin-omas abs. (%)	No. of adeno-squamous carc .
0	0	0/40	0.1	5/40 (12.5)	0/40	0/40	0/40
50 µg BaP/m^3	0.0768	1/40 (2.5)	24.7	40/40 *** (100)	10/40** (25)	0/40	0/40
90 µg BaP/m^3	0.1382	4/40 (10.0)	37.1	40/40 *** (100)	33/44*** (75)	6/40* (15)	1/40 (2.5)

Note: *, p<0.05; **, p<0.01; ***, p<0.001 (compared to controls, pair wise Fisher test).; [1] The values were calculated making the following assumptions for female mice: 25 g bw, respiratory rate: 40 mL/min.

The lowest tested concentration of PAH-enriched exhaust containing 50 µg BaP/m^3, associated with a significant increase in lung adenomas and a dose-dependent increase in malignant lung tumours for female NMRI/BR mice exposed for 44 weeks (5 d/wk, 16 h/d), should be used as a point of departure/starting point for further estimations.

The concentration of 50 µg BaP/m^3 (≙0.00005 mg/L) is equivalent to 0.0768 mg/kg bw/d.

B.5.8.2.3 Hamster

B.5.8.2.3.1 Chronic inhalation study in hamsters (Thyssen et al., 1981)

In another chronic inhalation study, groups of male Syrian golden hamsters (n = 25-27) were exposed by inhalation to BaP concentrations of 2.2, 9.5, and 46.5 mg/m^3 air for 4.5 h/d, 7 days/week for the first 10 weeks, then for 3 h/d for up to 2 years. Exposure was by nose breathing only. The total average dose of BaP per animal was 29, 127, and 383 mg, respectively. Controls were exposed to aerosol with 240 µg NaCl/m^3 air. The particle sizes were reported to be within the respirable range: more than 99 % of the BaP particles had diameters between 0.2 and 0.5 µm, and over 80 % were between 0.2 and 0.3 µm (Thyssen et al., 1981).

The chronic inhalation study in hamsters provides clear-cut evidence of a dose-response relationship between inhaled BaP particles and respiratory tract tumourigenesis. Survival time was significantly decreased from 96 weeks for controls to 59.5 weeks for animals in the 46.5 mg/m^3 BaP exposure group; survival times were not altered in the other exposure groups. Respiratory tract tumours were induced in the nasal cavity, pharynx, larynx, and trachea in hamsters exposed to 9.5 or 46.5 mg BaP/m^3. Exposure-related tumours were also found in the oesophagus and forestomach following exposure to 9.5 or 46.5 mg/m^3 (presumably as a consequence of mucocilliary particle clearance and swallowing of particles). These tumours were papillomas, papillary polyps, and squamous cell carcinomas. No respiratory tract tumours and no upper digestive tract tumours were found in the controls and in animals exposed to 2.2 mg BaP/m^3.

In contrast to lung tumours observed in rats and mice, no BaP-related tumours were found in the lungs of hamsters. An overview of tumour incidences in hamsters exposed to BaP is presented in Table 24.

Table 24: Incidence of tumours in male Syrian golden hamsters after long-term exposure to BaP

Conc. (µg BaP/m^3)	**0**	**2200**	**9500**	**46500**
Dose level (mg BaP/kg bw/d) [1] **Site of tumour/Parameter**	**0**	**0.572**	**2.4624**	**12.0528**
Nasal cavity	0/27	0/27	3/26 (12 %)	1/25 (4 %)
Larynx	0/27	0/27	8/26 (31 %)	13/25 (52 %)
Trachea	0/27	0/27	1/26 (4 %)	3/25 (12 %)
Pharynx	0/27	0/27	6/26 (23 %)	14/25 (56 %)
Oesophagus	0/27	0/27	0/26	2/25 (8 %)
Forestomach	0/27	0/27	1/26 (4 %)	1/25 (4 %)
Tumour incidence (all tumours)	14/26 (52 %)	16/26 (68 %)	20/26 (77 %)	15/25 (60 %)
No. of tumours/tumour-bearing animal	1.1	1.3	1.7	2.5

[1] The values were calculated based on mg BaP/animal data taken from the original study report and assuming a body weight of 125 g according to the REACH Guidance on Information Requirements (Appendix R.8-2, Table R.8-17) for male hamsters:, respiratory rate: 120 mL/min

In male Syrian golden hamsters long-term inhalation exposure to ≥ 9.5 mg/m^3 BaP induced tumours in the respiratory and the upper digestive tract. The data from the highest concentration group should not be used for further calculation since these animals had an appreciably shortened life span (59.5 vs. 96 weeks in other groups). Therefore, the concentration of 9.5 mg BaP/m^3 associated with tumour induction in the respiratory and upper digestive tract should be used as a point of departure/starting point for further estimations, if applicable.

The concentration of 9.5 mg BaP/m^3 (≙0.0095 mg/L) BaP is equivalent to 1.016 mg/kg bw/d.

B.5.8.3 Carcinogenicity: dermal

The majority of carcinogenicity studies in laboratory animals to examine tumour formation following exposure to BaP and various PAHs were conducted as skin painting studies. In various strains of mice the ability of the PAHs to induce benign and malignant skin tumours after repeated exposure to comparatively low doses was demonstrated (Knafla et al., 2006).

In addition, a number of chronic animal studies used dermal application of condensates containing various PAH compounds. Tested condensates were obtained from tobacco smoking, diesel and gasoline engine exhaust, carbon blacks, coal tar and coal gasification derived products, etc. The following PAHs were tumourigenic in rats and mice following dermal exposure: benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[l,2,3-c,d]pyrene. Although many studies were not conducted according to current EU or OECD guidelines, they clearly indicate that PAHs induce skin tumours (HCN 2006).

BaP is a potent experimental skin carcinogen, and it is often used as a positive control in bioassays on other substances. BaP produced skin tumours after dermal application in mice, rats, rabbits, and guinea pigs. Mixtures of PAHs that include BaP such as coal tar were also found to be dermal carcinogens in experimental animals.

Dermal studies with PAHs and/or BaP were examined to identify the most relevant animal long-term studies. The following three studies in NMRI mice, one in CD-1 mice, and one in C3H/HeJ mice were evaluated as adequate animal cancer data and were selected for further estimations for the dermal route of exposure. The studies selected for analysis also involved the application of BaP to skin in acetone or toluene as vehicle.

B.5.8.3.1 Mouse

B.5.8.3.1.1 Dermal lifetime study in mice (Schmähl et al., 1977)

The carcinogenic action of PAH mixtures predominantly found in condensates of automobile exhaust were studied in this study. A total of four different test items was administered: pure BaP, a mixture of known carcinogenic PAHs ('C PAH', including BaP), a mixture of PAHs not considered carcinogenic by the study authors ('NC PAH'), and a combination of the latter two ('C PAH + NC PAH').

Female NMRI mice were dermally exposed (back area) to these test items (dissolved in 0.02 mL acetone) twice weekly for their entire lifespan. Concentrations were adjusted in a way that treated animals of the BaP, C PAH, and C PAH + NC PAH groups received 1.0, 1.7, or 3.0 µg BaP (corresponding to 0.04, 0.068, or 0.12 mg BaP/kg bw/d) regardless of the test item used. For the NC PAH group, concentrations were used which corresponded to the proportions (by weight) of the respective PAHs relative to BaP as encountered in real-life exhaust gas condensates. In addition, a concurrent control group was treated with the vehicle acetone alone.

The test articles were administered to the shaved skin of mice until the natural death of the animals or until the animals developed a tumour. At the start of the study, each dose group consisted of 100 animals, but spontaneous deaths and autolysis reduced the total number of animals examined in each group (Schmähl et al., 1977).

Lifetime exposure of female NMRI mice to 1.0, 1.7, and 3.0 µg BaP/animal from various mixtures produced a dose-related increase in carcinomas and other tumours of the skin at the site of application. In Table 25 the findings are presented in detail.

Table 25: Incidence of skin tumours in female NMRI mice topically administered BaP from various matrices (cf. text)

Dose level (µg BaP/animal/d) [1]	**0**	**1**	**1.7**	**3**
Dose level (mg BaP/kg bw/d) [1]	**0**	**0.04**	**0.068**	**0.12**
Pure BaP				
Skin carcinoma	0/81	10/77 (13 %)	25/88 (28 %)	43/81 (53 %)
Any skin tumour	1/81 (1 %)	11/77 (14 %)	25/88 (28 %)	45/81 (56 %)
C PAH				
Skin carcinoma	0/81	25/81 (31 %)	53/88 (60 %)	63/90 (70 %)
Any skin tumour	1/81 (1 %)	29/81 (36 %)	57/88 (65 %)	65/90 (72 %)
NC PAH				
Skin carcinoma	1/85 (1 %)	0/84	1/88 (1 %)	15/86 (17 %)
Any skin tumour	1/85 (1 %)	0/84	1/88 (1 %)	16/86 (19 %)
C PAH + NC PAH				
Skin carcinoma	0/81	44/89 (49 %)	54/93 (58 %)	64/93 (69 %)
Any skin tumour	0/81	46/89 (52 %)	57/93 (61 %)	65/93 (70 %)

[1] It is important to note that 'per day' refers to treatment days only (treatment schedule: 2 d/wk). Body weight for female mice was assumed to be 25 g/animal, in accordance with the REACH Guidance on Information Requirements (Appendix R.8-2, Table R.8-17)

The results given in the above table show clearly that PAH mixtures containing BaP and certain other PAHs will cause a higher incidence of neoplasms when administered at the same BaP exposure level.

In this study, induction of local tumours was observed at all tested concentrations. The lowest tested concentration of 1.0 µg BaP/animal was equivalent to 0.04 mg BaP/kg bw/d.

B.5.8.3.1.2 Dermal lifetime study in mice (Habs et al., 1980)

In a dermal lifetime study, pure BaP was tested with regard to local carcinogenicity by topical application to mouse skin. Groups of female NMRI mice (n=40) were topically administered (2d/wk) with 1.7, 2.8, or 4.6 µg BaP/animal dissolved in acetone for up to 130 weeks. Controls received the vehicle alone. The solutions were applied by topical dropping to the clipped dorsal skin in the interscapular area. Each application comprised 0.02 mL. All experimental animals were checked twice daily and the occurrence of tumours at the site of application was recorded. Animals at an advanced stage of macroscopically clearly infiltrative tumour growth were killed prior to their natural death (Habs et al., 1980).

A clear dose-response relationship could be established for the carcinogenic activity of pure BaP at the site of application. Control animals did not develop tumours at the site of application. Study results are summarised in Table 26.

Table 26: Incidence of skin tumours in female NMRI mice topically administered with BaP

Dose level (µg BaP/animal/d) [1]	**0**	**1.7**	**2.8**	**4.6**
Dose level (mg BaP/kg bw/d) [1]	**0**	**0.068**	**0.112**	**0.184**
No. of tumour-bearing animals	0/35	8/34 (24 %)	24/35 (69 %)	22/36 (61 %)
Age-standardised tumour frequencies (%)	0	24.8	89.3	91.7

[1] It is important to note that 'per day' refers to treatment days only (treatment schedule: 2 d/wk). Body weight for female mice was assumed to be 25 g/animal, in accordance with the REACH Guidance on Information Requirements (Appendix R.8-2, Table R.8-17)

The lowest tested concentration of 1.7 µg BaP/animal topically administered (2d/wk) for up to 130 weeks and associated with significant increase in local tumours (x^2 test, probit analysis) in female NMRI mice should be used as a point of departure/ starting point for further estimations.

The concentration of 1.7 µg BaP/animal is equivalent to 0.068 mg/kg bw/d.

B.5.8.3.1.3 Dermal lifetime study in mice (Habs et al.,, 1984)

In a third life-time study, the carcinogenicity of condensates of different origin was examined. BaP was used as positive control. Only results from the positive control compared to controls were presented in this report. Groups of female NMRI mice were treated with 2 or 4 µg BaP/mouse in acetone (2 d/wk) and one solvent–treated control, each group containing 20 animals. The individual dose in the control group was 0.01 mL acetone. The solutions (0.01 mL) were applied by topical dropping to the clipped dorsal skin in the interscapular area twice a week for life. All animals were monitored twice daily and the occurrence of skin tumours was recorded. Animals in an advanced stage of macroscopically clearly invasive tumour growth were killed, all other animals were observed until their natural death (Habs et al., 1984).

Treatment was tolerated without signs of acute or subacute toxicity. Weight development in test compound-treated mice did not differ from that in controls. Mean survival time was 691 (95 % CI: 600-763) days in the acetone control, 648 (440-729) days in the 2 µg BaP/mouse, and 528 (480-555) days in the 4 µg BaP/mouse groups, respectively.

BaP was found to be clearly carcinogenic in both tested concentrations. No skin tumours were seen in vehicle controls. The carcinogenic activity of BaP after chronic epicutaneous application to female NMRI mice is presented in Table 27.

Table 27: Incidence of skin tumours in female NMRI mice topically administered with BaP

Dose level (µg BaP/animal/d) [1]	**0**	**2**	**4**
Dose level (mg BaP/kg bw/d) [1]	**0**	**0.08**	**0.16**
No. of animals with skin carcinoma	0/20	7/20 (35 %)	17/20 (85 %)
No. of animals with skin papilloma	0/20	2/20 (10 %)	0/20
Total no. of skin tumour-bearing animals	0/20	9/20 (45 %)	17/20 (85 %)

[1] It is important to note that 'per day' refers to treatment days only (treatment schedule: 2 d/wk). Body weight for female mice was assumed to be 25 g/animal, in accordance with the REACH Guidance on Information Requirements (Appendix R.8-2, Table R.8-17)

In summary, the lowest topically administered concentration of 2 µg BaP/mouse to female NMRI mice throughout their lifetime induced statistically significant (comparing age-adjusted expected with actually observed incidences, Peto et al., 1980) skin tumours in 9/20 animals (45 %). The

concentration of 2 µg BaP/animal should be used as a point of departure/starting point for further estimations.

The concentration of 2 µg BaP/animal is equivalent to 0.08 mg/kg bw/d.

B.5.8.3.1.4 Dermal lifetime study in mice (Warshawsky and Barkley 1987)

In a further study, relative carcinogenic potencies of three combustion products of fossil fuels and BaP were compared in carcinogenicity mouse skin bioassays (skin painting studies). The BaP group was used as positive control. Only the results of the positive BaP control group in comparison to controls were presented in this report. In the positive control group, 50 male C3H/HeJ mice were treated twice a week with a 0.025 % BaP solution (12.5 µg BaP/animal delivered in 50 µl of acetone) applied to the interscapular region of the back for up to 99 weeks. The animals of the control group were treated with 50 µL of distilled acetone twice weekly. Hair from the backs of mice was removed with electric clippers at least two days before the first treatment and every two weeks after the first treatment. During the course of the experiment animals were observed twice daily (Warshawsky and Barkley 1987).

Male C3H/HeJ mice administered with 12.5 µg BaP/animal for 99 weeks produced skin tumours in 48/50 mice. While in one instance a benign tumour was found, tumours were malignant in all other cases. The mean latency period was 32.4 weeks. The concentration of 12.5 µg BaP/animal should be used as a point of departure/starting point for further estimations.

Assuming a body weight of 30 g/male mouse, the concentration of 12.5 µg BaP/animal is equivalent to 0.417 mg/kg bw/d.

B.5.8.3.1.5 Dermal lifetime study in mice with creosote (FhI 1997)

The carcinogenic potential of two coal tar oil preparations has been examined by a dermal carcinogenicity study performed by the Fraunhofer Institute of Toxicology and Aerosol Research in Hannover/Germany (FhI 1997). It should be noted that not the full study report, but only a summary was available to the authors of this dossier, which was prepared by the RMS Sweden for their Competent Authority Report (CAR) in the context of the process of including creosote into annex I of Dir. 98/8/EC (Biocidal Products Directive, BPD).

The study followed the principles of OECD TG 451, but examinations were restricted to the investigation of local carcinogenicity (skin). Therefore, the list of examined endpoints was considerably limited as compared to a long-term guideline study. These deviations were in general not considered to impair the validity of the study performance and the reliability of test results with respect to skin tumour formation.

However, toluene was used as vehicle in order to be able to apply sufficiently low doses and the choice of this vehicle turned out to be problematic, as it displayed strong irritant properties in the course of the study (cf. following paras).

The 78-week dermal carcinogenicity study was conducted with two samples of coal tar creosote. Creosote is a coal tar product, which is variable in composition and contains not only BaP as sole carcinogenic constituent but also varying amounts of other PAHs and other potentially mutagenic and carcinogenic substances. Two different creosote samples were tested: creosote 1 (CTP1) and creosote 2 (CTP 2) containing 10 and 275 mg BaP/kg, respectively. The concentrations of BaA

were 25 and 1190 ppm, respectively, and another potent carcinogen, DBAhA, was determined at 1 and 19.2 ppm, respectively. Both samples were diluted with toluene before being applied to male [Crl:CD®-1(ICR)BR] CD-1 mice (n=62).

The animals were treated with 25 µL of solution twice a week over a period of 78 weeks. The solutions were applied to the interscapular skin area (about 10 % of the body surface). For the sake of clarity, administered dose levels are given in Table 28 and Table 29.

Table 28: FhI dermal carcinogenicity study in mice with creosote, CTP 1 dose groups

Dose level (mg CTP 1 /animal/d) [1]	**0**	**0.3**	**1**	**3**	**9**
Dose level (µg BaP/animal/d) [1]	0	0.003	0.01	0.03	0.09
Dose level (mg BaP/kg bw/d) [1]	0	0.0001	0.0003	0.001	0.003

[1] It is important to note that 'per day' refers to treatment days only (treatment schedule: 2 d/wk). Body weight for male mice was assumed to be 30 g/animal, in accordance with the REACH Guidance on Information Requirements (Appendix R.8-2, Table R.8-17)

Table 29: FhI dermal carcinogenicity study in mice with creosote, CTP 2 dose groups

Dose level (mg CTP 2 /animal/d) [1]	**0**	**0.1**	**0.3**	**1**	**3**	**9**
Dose level (µg BaP/animal/d) [1]	0	0.027	0.08	0.27	0.8	2.4
Dose level (mg BaP/kg bw/d) [1]	0	0.0009	0.0027	0.009	0.027	0.08

[1] It is important to note that 'per day' refers to treatment days only (treatment schedule: 2 d/wk). Body weight for female mice was assumed to be 25 g/animal, in accordance with the REACH Guidance on Information Requirements (Appendix R.8-2, Table R.8-17)

A positive control group receiving 7.5 µg BaP/treatment dissolved in 25 µL toluene (equivalent to 0.25 mg BaP/kg bw/d) was run concurrently.

Mean lifespan was significantly reduced in animals treated with the highest CTP 1 dose (447 days) and with the three highest dose levels of CTP 2 (444, 407, 252 days, respectively) when compared with the negative control group (483 days). The highest CTP 2 dose group was terminated at 39 weeks (274 days) in view of the high death rate seen (cf. comment to the above table).

The following non-neoplastic local effects were observed: Treatment-related inflammatory changes of the skin were observed in all groups and consisted either of slight to severe ulcerative dermatitis (ulceration) or superficial purulent dermatitis, epidermal erosion and inclusion cysts. More than 70 % of the solvent control and oil-treatment animals suffered from dermatitis. In the solvent control group, ulcerative dermatitis was noted in 27/62 mice, purulent dermatitis in 25/62 mice, and skin erosion in 9/62 mice. In all CTP 1 dose groups, the frequency of ulcerative dermatitis was comparable to that of the negative control group (27/62, 23/62, 21/62, 28/62, and 31/62 mice in controls, 0.3, 1, 3 and 9 mg dose groups, respectively). The incidence of epidermal hyperplasia without cellular atypia occurred in almost all of the solvent control and all CTP1 treated groups in dose-related grade from very slight to severe.

The following pre-neoplastic lesions were noted. The incidence of epidermal hyperplasia with cellular atypia occurred incidentally in the solvent control (1/62) and CTP 1 groups (1/62 and 2/62 at 3 and 9 mg, respectively), but to a significantly higher extent in the CTP 2 treated groups (3 mg: 11/62; 9 mg: 13/62) in moderate to severe grades. Nodules (‘apparent papillomas’) were

macroscopically identified in CTP 1 groups (1/62; 2/62; and 4/62 in the 1, 3, and 9 mg group, respectively). No statistical significance was obtained for this change.

Treatment with CPT1 and CPT2 resulted in neoplastic effects as squamous cell carcinomas and papillomas at the site of application. Other organs were not examined by microscopy. In the CPT 1 groups no skin tumours were seen at 1 mg and 3 mg which corresponded to the application of 0.01 and 0.03 µg BaP. At 3 mg (0.03 µg BaP) one solitary squamous cell carcinoma was observed, two solitary squamous cell papillomas in the 9 mg (0.09 µg BaP) group. However, those incidences were not statistically different from the control group. In the 3 and 9 mg dose groups, mean life span was not statistically significantly lower (447 days) than in the untreated control group (494 days).

In the CPT 2 groups, a dose-related, statistically significant ($p < 0.05$) increase in tumour rate was found. An increased incidence of squamous cell skin carcinomas occurred after treatment with 3 mg (0.8 µg BaP) and above, skin papillomas were noted from 1 mg (0.27 µg BaP) and multiple papillomas were observed at the highest dose of 9 mg (2.4 µg BaP). The mean life span was significantly decreased after treatment with 1 mg, 3 mg and 9 mg CPT 2 from 494 days in the control to 444, 407, and 252 days, respectively.

In the BaP positive control group, a high number of animals carrying squamous papillomas or carcinomas was observed (32/62 with malignant squamous cell carcinoma and 27/62 with benign squamous cell papillomas). Only in this group, additional skin tumour types were detected (basal cell carcinoma, benign basal cell tumour, sebaceous carcinoma, sebaceous adenoma, malignant fibrous histiocytoma and malignant Schwannoma).

An overview of the results from the dermal carcinogenicity study in mice using the two coal tar oil preparations and the BaP positive control group is given in Table 30.

Table 30: Results from the dermal carcinogenicity study in CD-1 male mice using BaP and two coal tar oil preparations CTP1 and CTP2

Test item	**Ctrl 1**	**Ctrl 2**	**BaP**	**CTP1**				**CTP2**				
Dose level mg test item/animal/d	**0**	**0**	**0.0075**	**0.3**	**1**	**3**	**9**	**0.1**	**0.3**	**1**	**3**	**9** [1]
Dose level (µg BaP/kg bw/d)	0	0	0.25	0.0001	0.0003	0.001	0.003	0.027	0.08	0.27	0.8	2.4 [1]
No. of tumour-bearing animals	1 [2]	0	47/62	0/62	0/62	1/62	2/62	1/62	3/62	9/62	23/62	n.e. [1]

[1] terminated after 274 days because of skin ulcerations; [2] skin tumour considered atypical of PAH by the study authors (cavernous haemangioma)

Overall, the study showed a dose-dependent statistically significant ($p < 0.05$) increase in the number of animals with skin tumours after CTP 2 treatment, while for CTP 1, such a relationship could not be established, putatively due to the very low doses of BaP administered.

B.5.8.4 Carcinogenicity: human data

Evidence that mixtures of PAHs are carcinogenic to humans is primarily derived from occupational studies of workers following inhalation and dermal exposure. No data were located regarding

cancer in humans following inhalation of individual PAH compounds. Exposure of humans to PAHs is characterised by a mixture of these compounds and other substances in either occupational or environmental situations. Therefore, it is difficult to ascertain the carcinogenicity of a single PAH component or a given mixture of PAHs, as presumably amplification of carcinogenicity may have occurred through the presence of other carcinogenic substances in the mixtures. In addition, PAH levels were not quantified in most reports and confounding factors such as smoking may have contributed to the effects, but were not adequately considered. For human oral exposure to single PAHs or PAH mixtures in humans no adequate long-term studies are available.

There is a large body of epidemiological studies of PAH-exposed workers, especially in coke ovens and aluminium smelters supporting a clear excess of lung cancer, and highly suggestive of an excess of bladder cancer. Skin cancer in man is well known to have occurred following exposure to poorly refined lubricating and cutting oils.

The epidemiological studies include cohort and case-control studies with various PAH-rich sources. Exposure–response relationships for occupational PAH exposure and cancer in humans have been reviewed by several working groups of the IARC (2006), the US EPA (1984), the WHO (1987, 1998, 2000, and 2003), and by the UK Health and Safety Executive (HSE, Armstrong et al., 2003). Several additional studies have been published (Armstrong et al., 1994, 2004, 2009; Boffetta et al., 1997; Bosetti et al., 2007; Costantino et al., 1995; Mastrangelo et al., 1996; Moolgavkar et al., 1998). All of them confirm that heavy occupational exposure to mixtures of PAHs entails a substantial risk of lung, skin, or bladder cancer. BaP has been chosen as a lead compound for carcinogenic PAHs, although the limitations and uncertainties of the approach were recognised. The epidemiological data relevant for this report are summarised in the following section.

In the 1980s, the IARC reviewed numerous epidemiological studies on PAH-exposed workers whose occupational exposure was assessed on the basis of type of employment or industrial process involved. Given the long latency between first exposure and cancer, these workers were exposed mainly during the first half of the century, when data on industrial hygiene were scarce. A definite risk of cancer was found in workers employed in the coke (lung cancer), aluminium (lung and bladder cancer), and steel industries (lung cancer), which were subsequently considered Group 1 carcinogens along with coal tar pitch, untreated and mildly treated mineral oils, and soot. On the other hand, inconsistencies between studies, lack of control of confounding factors, potential bias, and uncertainty regarding a dose-response relationship precluded any definitive conclusions for other occupational settings: roofers and asphalt workers, mechanics exposed to engine exhaust, bus and truck drivers, railroad workers, and excavator operators exposed to diesel exhaust in mines and tunnels (IARC 1983, 1984, 1985, and 1989).

An increased risk of lung cancer among coke-oven workers was used for the quantitative risk assessment of PAHs with BaP as the lead substance in the Air Quality Guidelines for Europe (WHO 1987, 2000). According to WHO (1987), a strongly increased risk of death from cancer of the respiratory system had been demonstrated among workers at coke ovens in Allegheny County, Pennsylvania, USA, for 1953–1970, especially in top-oven workers (relative risk [RR] = 6.6–15.7 for 300 topside, full-time workers, divided into different categories according to the years of exposure). WHO (1987) further refers to a risk assessment by the US EPA in 1984 which applied a linearised multistage mathematical model to the individual exposure estimates, thereby generating an upper-bound risk estimate expressed in terms of benzene-extractable material. The US EPA estimate was converted in terms of BaP levels by assuming a 0.71 % content of BaP in the benzene extract, thus estimating the lung cancer risk from a lifetime exposure to PAHs in ambient air at 8.7×10^{-5} per ng BaP/m^3 BaP (WHO 1987, 2000).

A well-performed meta-analysis study on lung and bladder cancer risk following PAH-exposure and funded by the UK HSE (Armstrong 2003, 2004) was performed based on published reports, in which relationships between occupational PAH exposure and lung and bladder cancer was studied quantitatively. All exposures concerned airborne PAHs emitted from incomplete combustion of organic matter. Risk estimates on lung and bladder cancer were determined because of the clear positive or at least highly suggestive association between these cancers and occupational PAH exposure.

The analysis included only studies which did meet certain criteria, i. e.:

- original epidemiological studies of occupational exposure by inhalation,
- studies on workplaces in which PAHs were considered as the predominant carcinogens (this means exclusion of rubber industry, diesel exhaust, foundries and part of steel works),
- studies in which misclassification of exposure was considered unlikely, and
- only the most recently reported results from the same workforce reported in several papers.

The meta-analysis included 39 occupational cohorts (35 cohorts, 1 case-cohort and 3 nested case-control samples from within a cohort) exposed to PAHs for which risk estimates for lung cancer could be estimated and 27 cohorts for which risk estimates were published for bladder cancer.

The underlying studies, however, showed a substantial variation in exposure definition, ranging from no explicit definition to quantitative assessment of exposure to BaP. Exposure was measured as BaP, as a proxy (benzene soluble matter, BSM; total PAH; carbon black) that could be converted to BaP, or no measure of exposure was provided. For the studies lacking information on exposure, the authors defined supplementary estimates for exposure to BaP for each industry/workgroup combination, based on available published exposure estimates in the same industries. Furthermore, the exposure variables were converted to cumulative exposure (duration × time-weighted mean concentration), if necessary. Where risk by cumulative exposure was not published, it was derived as the product of mean estimated concentration of exposure in each group for which risk was reported and the mean duration of exposure in that group. In the absence of information on duration of exposure, 20 years were assumed, representing the average found in studies for which the duration was reported. Concerning studies with cumulative exposure, the mean cumulative exposure in each group or the midpoint of interval was chosen as an estimate for the average cumulative BaP exposure. Overall, the cumulative exposure in the highest exposure groups ranged across three orders of magnitude, from 0.75 to 805 µg BaP /m^3 years (≈ average air concentration of 0.04 to 40 µg/m^3 BaP).

The unit relative risk (URR) was estimated for each study for a benchmark exposure level of 100 µg BaP/m^3 years, in which 100 µg/m^3 BaP years corresponds to a concentration of 2.5 µg BaP/m^3 over 40 years. The authors had chosen this benchmark level such that it was within the exposure ranges of the studies included in the meta-analyses. Concerning data on effects, the authors preferred mortality outcomes over morbidity outcomes within a same study. Furthermore, other preferences were formulated, such as smoking-adjusted data over unadjusted data. The URR was estimated by fitting an exposure-risk model to the data with Poisson regression. For determining URR two models were used: the log-linear relative risk model (exponential) as normally used in epidemiological studies and meta-analyses [$RR=\exp(b_{login}x)$] and the linear relative risk model $(RR)=1+b_{lin}x$), where "*x*" is the cumulative exposure (µg/m^3-years) and "b" is the slope of the

exposure-risk relationship. Meta-regression was applied to assess the impact of study characteristics on the final risk estimate.

Lung cancer

The RR for lung cancer, predicted at 100 µg/m^3 BaP years from the log-linear models, ranged from 0 to over 1000 among the studies, with standard errors ranging between 0.02 and 1000. This is a substantial variation in precision, which is well explained by variations in the degree of exposure in the studies (some studies have only low exposures) and by variations in size of cohort populations and duration of follow-up. The overall mean URR of 1.20 (95 % CI, 1.11-1.29) per unit of 100 µg/m^3 x year cumulative BaP exposure was calculated for lung cancer. This implies that the risk for lung cancer was 20 % higher in workers exposed to a cumulative 100 µg BaP/m^3 x year. In a meta-analysis, it is common practice to investigate whether the data from the studies included are sufficiently in agreement with each other by testing for heterogeneity. A statistically significant heterogeneity in URR between the individual studies was observed, indicating that some studies (mainly the smallest, i.e. least precise studies) had deviating estimates. However, meta-regression analysis revealed that the URR for coke ovens, gas works and aluminium smelters were consistent and relatively precisely estimated (combined URR 1.17, 95 % CI: 1.12-1.22). For other characteristics (such as study design, region or type of exposure measurement, duration, dust exposure, smoking habits) no statistically significant heterogeneity was detected. However, whether the differences between industries are caused by chance or by true unknown variations is not exactly known, because scientific data that reported on the presence of true variations was not available or insufficient to draw conclusions.

In summary, lung cancer risk at other exposures can be estimated from URR under log-linear model assumptions: $URR_{cum\ exp\ X} = [URR_{cum\ exp\ 100}]^{(x/100)}$. In the UK, the lifetime lung cancer risk in males from the general population is 8 % (year 1997). This means a lifetime excess risk of 1.9×10^{-4} for coke oven workers (URR 1.17), who were exposed to 1.5 µg/m^3 BaP for 40 years (8 cases among 1000 coke oven workers).

Bladder cancer

There were 27 cohorts for which risk estimates were published for bladder cancer. The overall mean URR for bladder cancer was calculated as 1.33 (95 % CI: 1.16-1.52), with no statistically significant variation by industry or other putative determinants. Although the results support a PAH-bladder cancer association, this finding was less robust than that for lung cancer. The causal relationship between PAH exposure and bladder cancer was considered weak for several reasons. Firstly, in epidemiological studies less cases of bladder (and renal) cancer than lung cancer were reported. This limits the power of the analysis and results in a less precise estimate than for the estimates for lung cancer. Secondly, bladder cancer mortality in the general population is much lower than for lung cancer. Thirdly, it cannot be excluded that bladder cancer is induced by other substances (not PAHs), which are suspected to be specific bladder carcinogens (e.g., 2-naphthylamine), and are found in the types of industries under investigation. The investigators estimated that the number of expected bladder cancer cases after 40 years of occupational exposure to 1.5 µg/m^3 corresponds to 3.3 cases among 1000 coke-oven workers.

In addition, uncertainties as to the exposure-response relationship were discussed in more detail. The authors concluded that the results for coke-ovens, gasworks, and aluminium production are relatively well supported by others, although potential biases should be considered. These biases include: smoking, which was uncontrolled in most studies; other occupational exposures, although

the authors excluded studies in which PAHs were judged unlikely to be the predominant carcinogen (except dust); and inaccurate exposure measurement estimates (uncertainty in past exposure).

Overall, the meta-analysis supports the conclusions of previous reviews that lung cancer and bladder cancer are associated with PAH exposure. On average, relative risk predicted for lung cancer at 100 mg/m^3 BaP years (URR) was 1.20 (95 %CI:1.11-1.29), but this varied significantly across industries. Coke ovens (1.17; CI:1.12-1.22), gasworks (1.15; CI:1.11-1.20), and aluminium production (1.16; CI:1.05-1.28) were slightly lower than the mean, and asphalt (17.50; CI:4.21-72.78) and chimney sweeping (16.2; CI:1.64-160.7) were much higher but imprecisely estimated. There was also an association of PAHs with bladder cancer (mean URR=1.33; 95 %CI: 1.16-1.52, no significant heterogeneity), but this finding was less robust than that for PAH-lung cancer, being largely dependent on two studies of aluminium production workers. The overall URR represents risk at fairly common exposures historically, but high for today. Risks at other exposure levels can be estimated from URR under (loglinear) model assumptions: e.g. at 1 mg/m^3 BaP for 40 years, cumulative exposure is 40 mg/m^3 BaP years, and the average lung cancer risk is 1.20(40/100)=1.076.

In a continuative study, the exposure–response function associating PAH exposure and lung cancer, with consideration of smoking, was estimated. Mortality, occupational exposure and smoking histories were ascertained for a cohort of 16431 persons (15703 men and 728 women) who had worked in one of four aluminium smelters in Quebec from 1950 to 1999. A variety of exposure–response functions were fitted to the cohort data using generalised relative risk models. In 677 lung cancer cases there was a clear trend of increasing risk with increasing cumulative exposure to PAH measured as BaP. A linear model predicted a relative risk of 1.35 (95 % CI 1.22 to 1.51) at 100 mg/m^{-3} BaP years, but there was a significant departure from linearity in the direction of decreasing slope with increasing exposures. Among the models tried, the best fitting were a two-knot cubic spline and a power curve (RR= $(1+bx)^p$), the latter predicting a relative risk of 2.68 at 100 mg/m^{-3} BaP years. Additive and multiplicative models for combining risks from occupational PAHs and smoking fitted almost equally well, with a slight advantage to the additive.

Despite the large cohort with a long follow-up, the shape of the exposure–response function and the mode of combination of risks due to occupational PAHs and smoking remain uncertain. If a linear exposure–response function is assumed, the estimated slope is broadly in line with the estimate from a previous follow-up of the same cohort and somewhat higher than the average found in a recent meta-analysis of BaP exposed populations in a variety of industries (Armstrong et al., 2009).

In a former study by Armstrong and co-workers (1994), the quantitative lung cancer risk for aluminium production workers from a large cohort study in Quebec, Canada was estimated. The follow-up period started in 1950 and continued through 1988. The study was performed in one plant that used two types of pots to melt aluminium, namely Söderberg and prebake pots. In particular in the Söderberg process high amounts of coal tar pitch volatiles are emitted in the air. The workers were exposed to substantial quantities of coal tar pitch volatiles, expressed as cumulative exposure to BSM or BaP. In the study 338 lung cancer cases and 1138 controls from about 16000 subjects who had worked in an aluminium refinery plant for at least one year during the period 1950-1979 were selected. Exposure to BSM or BaP in these subjects was estimated by the industrial hygienists of the factory by integrating jobs with historical (5 year) average PAH levels; exposure was estimated on the basis of sparse measurements carried out from 1970. Cumulative exposure was the sum of products (years of exposure per PAH level) in each job. Reference workers were exposed to less than 1 mg/m^3 x years of BSM, or less than 10 µg/m^3 x years of BaP. In this study, a 50 % increase in the risk of lung cancer was demonstrated, with a cumulative exposure to BaP as

indicator substance at levels of 10–100 μg/m^3 × years (equivalent to an exposure to 0.25–2.5 μg/m^3 BaP for 40 years). Two mathematical models, linear and nonlinear, were fitted to the data. According to the mathematical model expressing the association between lung cancer risk and cumulative BSM exposure, after controlling for the effect of smoking, the risk in a subject exposed for 40 years to 0.2 mg/m^3 of BSM is 1.25 (linear model) or 1.42 (nonlinear model), with respect to non-exposed subjects. It was concluded that the exposure to PAHs in an aluminium production plant gave a quantitative risk estimate of 1 x 10^{-5} per ng BaP/m^3 as workplace exposure for 40 years. If converted to lifetime continuous exposure, the corresponding lifetime unit risk for respiratory cancer would be approximately 9 × 10^{-5} per ng/m^3 (70/40 years × 365/220 days × 24/8 hr). This risk figure is identical to the WHO risk estimate for coke oven workers.

An update of the WHO cohort data and the mortality among other coke-oven workers in Pennsylvania, USA, providing 30 years of follow-up, has been presented by Costantino et al. (1995). The cause-specific mortality patterns of two large steelworker cohorts, called the Allegheny County and the non-Allegheny County cohort (a total of 15818 workers), were studied. The lung cancer mortality in 5321 coke workers was compared with that in 10497 non-coke workers in a large steel company. The workers were grouped into three exposure categories: 3.15 mg/m^3 for topside (i. e. workplace at the top of the oven) full-time jobs, 1.99 mg/m^3 for topside part-time jobs, and 0.88 mg/m^3 for side jobs. During the follow-up mortality was registered. No information on smoking habits concentrations was available. The results presented in this review were consistent with the findings reported from previous updates of both the Allegheny County and the non-Allegheny County cohorts. Occupational exposure to coke oven emissions was associated with excess mortality from cancer of the respiratory system. The non-exposed group included subjects with cumulative BSM exposure equal to 0. For overall lung cancer, the RR value was estimated at 1.95; 95 % CI, 1.6-2.3.

In a further study, the unit risk for lung cancer due to exposure to coke oven emissions was estimated based on information on the cohorts described by Costantino et al. (1995). The data from the non-Allegheny County non-white cohort were analysed, using standard techniques of survival analysis and the two-mutation expansion model of carcinogenesis. Results of analyses of the coke oven cohorts using this approach are consistent with the conclusions of earlier analyses that emissions from coke ovens are associated with increased risk of lung cancer. The best estimate for unit risk for lung cancer at 70 years of age for continuous exposure to coke oven emissions at a concentration of 1 μg/m^3 starting at birth was 1.5 x 10^{-4} (exponential dose-response model without birth cohort effects, adjusted for competing causes of mortality; 95 % confidence interval, = 1.2-1.8 x 10^{-4}) (Moolgavkar et al., 1998).

As a result of extensive research of epidemiological studies (1991-1996) 10 epidemiological studies (2 cohort studies; 2 case-cohort studies, 2 case-control studies nested in cohorts, and 4 hospital- or population-based case-control studies) reporting direct evidence of the carcinogenic effects of PAHs in occupationally exposed subjects were selected for the cancer risk estimation. Risks of lung and bladder cancer were dose dependent when PAHs were measured quantitatively and truly nonexposed groups were chosen for comparison. It was concluded that the more accurate recent studies, which reveal an increase in lung and bladder cancer with the occupational exposure to PAHs, support the earlier findings provided by the qualitative job title-based epidemiological studies. It was further suggested that the threshold limit value of 0.2 mg/m^3 of BSM (which indicates PAH exposure) is unacceptable because, after 40 years of exposure, it involves a RR of 1.2-1.4 for lung cancer and 2.2 for bladder cancer (Mastrangelo et al., 1996).

In a further review, several industries and occupations were included of which data were published before 1997 (Boffetta et al., 1997). Heavy exposure to PAHs entails a substantial risk for lung, skin and bladder cancer, which is not likely to be due to other carcinogenic exposure present in the same industries. The major target organ of PAH carcinogenicity was found to be the lung. The increased risk for lung cancer was present in most industries and occupations (aluminium production, coal gasification, coke production, iron and steel foundries, tar distillation, shale oil extraction, wood impregnation, roofing, road paving, carbon black production, carbon electrode production, chimney sweeping, and calcium carbide production). An increased risk for skin cancer was related to high dermal exposure. However, increased risk for bladder cancer was less consistent; positive associations were mainly found in industries where workers were exposed to coal tars and coal tar pitch volatiles (e.g., aluminium production, coal gasification and tar distillation).

In another, recent review, the results from cohort studies conducted on workers exposed to PAHs in several industries, including those of the aluminium production, coal gasification, coke production, iron and steel foundries, coal tar and related products, carbon black and carbon electrodes production, were evaluated, with a focus on cancers of the respiratory and urinary tract (Bosetti et al., 2007). The main results from cohort studies conducted on workers from PAH-related occupations, with emphasis on study results reported after the review by Boffetta et al. (1997) were described. An excess risk of lung/respiratory cancer was found in most of the examined industries, whereas the pooled relative risk (RR) for workers in coal gasification was extensively increased. The evidence for cancer of the bladder and of the urinary system is less consistent, with a modest increase in risk only for workers of aluminium production, coal gasification, iron and steel foundries.

B.5.8.5 Other relevant information

The production and use of toxic chemicals pose potentially significant environmental threats to the health of consumers, and especially of children. A wide range of chemicals can affect children's health, but one chemical class is of particular concern - PAHs. Chemicals with a DNA-damaging mode of action may cause cancer already during childhood, if the children themselves, or their mothers during pregnancy, suffer from sufficiently intense or prolonged exposure. Infants are at particular risk because mouthing or play behaviour can lead to the ingestion of toxic chemicals that accumulate on surfaces (e.g. toys) or in soil.

The younger child and toddler are susceptible to dermal exposure. It is prudent to regard any exposure to such substances as potentially carcinogenic. Whether children are likely to be more or less susceptible than adults to such chemicals is likely to depend on how a given chemical is absorbed, distributed in the body, and metabolised in younger versus older individuals, at exposure levels encountered by children and pregnant women (Neri et al., 2006).

Therefore, the focus in the following section was put on the question whether children are more susceptible to carcinogens than adults and on how children differ from adults in their susceptibility to chemical exposure.

B.5.8.5.1 Relative susceptibility to carcinogens of children vs. adults

There is currently international scientific consensus that children might represent a vulnerable sub-population with respect to potentially adverse effects of chemical substances.

Toxic response in infants and children can differ markedly from that seen in adults, both in severity and in the nature of the adverse effects (EPA, 1996, 1998; Schneider et al., 2002; APUG, 2004; IPCS, 2006). Potential differences in inherent biological susceptibility as well as in exposure to environmental chemicals might have the consequence that a child will experience risks from exposure to chemicals differently compared to an adult, which are further explained below (Charnley and Putzrath, 2001).

Probable higher susceptibility of children might be due to the fact that

- children are in a dynamic state of growth,

- children have more time to develop diseases with long latency, more years of life to be lost and more suffering to endure as a result of impaired health (IPCS, 2006).

Publications on this topic by various institutions (e.g. IPCS, EPA) put forward the theory that children might be particularly vulnerable to a broad range of agents of environmental pollution and that exposure to carcinogens early in life can result in the development of cancer (Anderson et al., 2000).

Due to differences in the age-related behaviour of children, which determines exposure, and due to age-related physiological differences, which might have an impact on toxicokinetics and toxicodynamics of a compound, these factors (either alone or in combination) can form the basis for differences in sensitivities towards certain toxicants during different life stages.

Developmental stages are defined as temporal intervals with distinct anatomical, physiological, behavioural, or functional characteristics that contribute to potential differences in vulnerability to environmental exposure. The following different stages in human development were distinguished:

Table 31: Definitions for stages in human development (IPCS, 2006)

Developmental stage/event	**Time period**
Preconception	Prefertilisation
Preimplantation embryo	Conception to implantation
Postimplantation embryo	Implantation to 8 weeks of pregnancy
Fetus	8 weeks of pregnancy to birth
Preterm birth	24–37 weeks of pregnancy
Normal-term birth	40 ± 2 weeks of pregnancy
Perinatal stage	29 weeks of pregnancy to 7 days after birth
Neonate	Birth to 28 days of age
Infant	28 days of age to 1 year
Child	
- Young child	1–4 years of age
- Toddler	2–3 years of age
- Older child	5–12 years of age
Adolescent	Beginning with the appearance of secondary sexual characteristics to achievement of full maturity (usually 12–18 years of age)

B.5.8.5.1.1 Vulnerability of children to harmful compounds

There is extensive literature demonstrating concern about the particular vulnerability of infants and children to environmental exposure, particularly to carcinogens (Thomas, 1995; EPA, 1996, 1998; Buffler and Kyle, 1999; Anderson et al., 2000; Charnley and Putzrath, 2001; Ginsberg, 2003; Wild and Kleinjans, 2003; Barton, 2005; Hattis et al., 2005; Whyatt, 2006); further, vulnerability of infants and children might be different for distinct stages of development.

The timing of exposure to chemicals is critical for the development of effects (either in childhood age or later in development). Because of the differing windows of susceptibility, the same dose of the same chemical during different periods of development can have very different consequences.
The postulated increased vulnerability of children involves many factors, which can be grouped into the following broad areas (Wild and Kleinjans, 2003):

- Children are exposed to relatively higher exogenous doses of environmental toxins, i.e., intakes are increased as compared to adults, which can be attributed both to lifestyle and physiology.

- A child's response to a hazardous substance in the environment can differ from that of an adult for a number of physiological reasons, related to differences in disposition, bioavailability, toxicokinetics, and toxicodynamics.

- If exposure begins in childhood, there is a greater likelihood that exposure becomes chronic, and further, that adverse health effects become manifest, even for those with a long latency period such as cancer.

In the following section, the major differences in susceptibility of children compared to chemical exposure to adults are reviewed.

B.5.8.5.1.2 Age-related differences in exposure and in susceptibility to hazardous compounds

Children are not 'little adults'. Profound differences exist between children and adults.

The most distinguishing characteristic between adults on the one side and infants and children on the other is that the latter grow and develop. Different systems and organs develop at different rates and at different stages of development. The metabolic rates of children and infants are higher compared to those of adults. From conception through adolescence, rapid growth and developmental processes occur that can be disrupted by exposures to environmental chemicals. The developing organism experiences many complex, integrated events involving the regulation of cell growth, differentiation, and morphogenesis. Chemically-induced disturbances or interferences of those events (e.g. through mutation, altered mitosis, nucleic acid biosynthesis, membrane function, enzyme function, or energy sources) can have significant adverse impacts on development (Faustman et al., 2000; Thomas, 1995; Wilson, 1977).

Thus, children might be more susceptible to chemicals than adults due to rapid growth and development as well as anatomical and physiological and metabolic changes in various organs and biological systems, and also in behavioural differences during childhood (Armstrong et al., 2002; Scheuplein et al., 2002; Schneider et al., 2002).

The three main aspects of differences between children and adults with respect to adverse affects of compounds, i. e.

- age-related differences in exposure
- differences in toxikokinetics (absorption, distribution, metabolism, excretion), and
- differences in susceptibility of development of cancer

will be discussed subsequently.

Age-related differences in exposure

Children can be more susceptible to chemicals than adults due to different patterns of activity and behaviour in the different age groups. For that reason, children can also not be regarded as a homogeneous group; rather, they are exposed to particular harmful substances to differing degrees throughout their development.

Compared to adults, children have particular pathways and scenarios of exposure (e.g., breast milk ingestion, hand-to-mouth behaviour, time spent outdoor/indoor). Exposure can occur *in utero* through transplacental transfer of environmental agents from mother to foetus or in nursing infants via breast milk. Because of differences in physiology, behaviour, body weight, and body surface area, the exposure levels in children may be different from and – dependent on compound-specific characteristics - often higher than in adults. Infants and children differ from adults in their exposure both qualitatively and quantitatively, in part because they eat more food, drink more water, and breathe more air per unit of body weight than adults do (Roberts, 1992).

Furthermore, an increased possibility and frequency of chemical exposure of infants and children might be due to certain particularities in behaviour, e.g., hand-to-mouth activity and crawling on the ground (Carroquino et al., 1998). In this context, the various age- and development-dependent activity and behaviour patterns are of prime importance.

Children have different nutritional patterns than adults and this can also influence exposure. For instance, children may consume more milk and other dairy products on a daily basis than adults, and their dietary patterns are different and often less variable during different developmental stages. At the extreme, during early infancy, children may solely ingest breast milk or artificial milk diluted with drinking water (Wild and Kleinjans, 2003; IPCS, 2006).

Additional particularities refer to an often intensive dermal exposure due to playing behaviour, differences in food composition (breast milk, baby food) and handling child-specific objects (toys etc.) (Schneider et al., 2002).

Parental exposure before conception may also affect health outcomes during later stages of development of the progeny. Adverse health effects may be detected during the same life stage as the exposure is occurring, or they may be expressed later in life (IPCS, 2006).

Differences in toxicokinetics (absorption, distribution, metabolism, excretion)

Children present unique biological characteristics (e.g. volumes of the organs, degree of enzymatic maturation, body composition…) which may affect the toxicokinetics and the toxicodynamic behaviour of substances (IPCS, 2006).

This section concentrates on differences in toxicokinetics, as specific data demonstrating differences in toxicodynamics of PAHs in children as compared to adults were not available.

Absorption

A number of physiological changes during childhood can affect absorption of exogenous agents. There is sufficient evidence to indicate that absorption of environmental toxins will differ with age and development (Benedetti and Baltes, 2003).

Absorption from the stomach may be affected by gastric pH. Practically neutral at birth, the gastric pH falls to about 1–3 within the 24 h following birth and then gradually returns to neutrality by day 8. It slowly declines again thereafter (e.g. pH = 2–3 by the age of 2–3 years) to reach adult values. By the age of 3 years the amount of gastric acid excreted per kilogram of body weight is similar to that excreted in adults (Stewart and Hampton, 1987).

Absorption by inhalation is relatively higher than in adults due to the fact that the respiratory minute volume per kg of body weight is on average twice as high, which means that the same concentration in the ambient air (indoor and outdoor air) will produce a higher inhaled dose relative to body weight and consequently a higher internal concentration (given a comparable concentration of a toxin in the inspired air). Further factors increasing inhalation uptake in children might be explained by a higher body surface area to body weight ratio, higher activity patterns, immaturity of the lungs and the immune system (Schneider et al., 2002).

Dermal absorption may be increased in the child for whom the surface area relative to the body weight is larger than in the adult, and particularly in neonates and infants where reduced epidermal and stratum corneum thickness also results in more efficient absorption by this route. Further the larger relative surface area of children means that body heat loss will be more rapid, requiring a higher rate of metabolism (Routledge, 1994; WHO-Europe, 2005; APUG, 2004).

Distribution

A number of physiological changes during childhood can affect disposition of exogenous agents. There is sufficient evidence to indicate that the distribution of environmental toxins will differ with age and development (Benedetti and Baltes, 2003). Key factors explaining differences in drug distribution between children and adults are membrane permeability, plasma protein concentration and plasma protein characteristics, endogenous substances in plasma, total body and extracellular water, fat content, regional blood flow and probably p-glycoprotein (Pgp), mainly that present in the gut, liver and brain. With respect to plasma protein binding it is known that the young are less capable of binding many compounds to proteins in the blood. Protein binding can be a significant detoxification mechanism, since it can segregate a chemical foreign to the body, making it less likely that the compound will reach its site of toxic action (Calabrese, 1986). Especially with respect to water-soluble substances, it can be stated that children of all age groups show a higher volume of distribution on a body weight basis. This is caused by an increased proportion of extracellular water and/or reduced protein binding compared to adults (Schneider et al., 2002).

Metabolism

Biotransformation enzymes are an important component in the metabolism of foreign substances. Several enzymatic systems are not fully developed at birth and mature during the child's development. Important differences have been found in children compared with adults both for

phase I enzymes [e.g. different cytochrome P450 (CYP) enzymes, mainly CYP3A7, CYP3A4 and CYP1A2), reductive and hydrolytic enzymes] and phase II enzymes (e.g. N-methyltransferases and UDP glucuronosyltransferases). Depending on the substance considered, this can either lead to increased or decreased toxicity compared to adults. Maturation of these enzymatic systems mainly takes place during the first year after birth (Calabrese, 1986; Benedetti and Baltes, 2003).

Elimination

Biliary and renal clearances do not reach the functional capacity of the adult organism until one or several months after birth, respectively, which resulted in a delayed elimination of foreign substances (Schneider et al., 2002).

During the growth and maturation process there is an evolution of membranes, including receptors, in infants and children as they approach adulthood. These changes represent a potential for a very different environment for chemical interactions with receptors (Roberts, 1992). The elimination mechanisms in children are not fully developed before the age of six months. Thus, the same dose relative to body weight cannot be eliminated at the same rate as later in life. Particularly in the case of repeated exposure, the reduced rate of elimination means that the same intake relative to body weight leads to a higher internal concentration and consequently to the possibility of a more pronounced effect (APUG, 2004).

Differences in susceptibility of children in relation to carcinogens

The increased susceptibility of infants and children to carcinogens when compared to adults is of particular importance. It is difficult to make generalisations about the effect of age on susceptibility to chemical carcinogens due to the heterogeneous nature of the possible influences – with regard to the individual age groups and individual substances – and the response mechanisms of the organism.

Age can, for example, affect metabolism, cell proliferation rates, and hormone levels, which can in turn affect tumour incidence, latency, and tumour type, as can a myriad of other interactions that are genetically, behaviourally, and environmentally determined.

In particular, two parameters expected to modulate the effects of exposure to an environmental carcinogen are DNA repair and cell proliferation (Wild and Kleinjans, 2003). The balances between DNA adduct formation, DNA repair, and cell replication will be critical to the development of tumours. There are relatively few direct data on changes in cell replication rate with age. However, inference can be made from the rate of organ weight gain, decreased in many organs from birth to 4–6 years of age. Although for organs such as the brain, this rate then remains stable, for others such as the liver, another increase in growth rate occurs towards puberty (Ginsberg, 2003). Additional periods of high cell replication rates exist for reproductive organs around puberty. Despite the absence of precise data, periods of rapid growth *in utero* and during infancy will be accompanied by greater cell division rates than in adults for many organs.

In the recent scientific literature, there is an increased number of publications stating that risks of cancer from exposure to carcinogens occurring from conception through puberty can be different compared to risks from exposure occurring in adulthood.

Induction of genetic damage early in life may increase risk of carcinogenesis later (Anderson et al., 2000; Birnbaum and Fenton, 2003; Ginsberg, 2003; Wild and Kleinjans, 2003; Miller et al., 2002; Scheuplein et al., 2002).

Multiple biological modes of action are involved in carcinogenesis. Thus, many factors may account for differences in sensitivity. Some aspects regarding a greater lifetime risk of cancer for infants and children due to exposure to a carcinogen early in life are summarised subsequently (Barton et al., 2005; Budroe et al., 2009):

- Cancer is a multistage process and the occurrence of the first stages in childhood increases the chance that the entire process will be completed, and a cancer produced, within an individual's lifetime.

- Tissues undergoing rapid growth and development may be particularly vulnerable to carcinogenic agents. More frequent cell division during development can result in enhanced fixation of mutations due to the reduced time available for repair of DNA lesions and clonal expansion of mutant cells gives a larger population of mutants (Slikker et al, 2004). In addition, more opportunity for incorrect repair of damage (e.g., DNA breaks, crosslinks, adducts) or alterations to result in permanent changes to the DNA (e.g., mutations, altered DNA methylation) could occur that may ultimately lead to cancer.

- Some embryonic cells, such as brain cells, lack key DNA repair enzymes.

- During early development, a greater proportion of the body's cells consists of relatively undifferentiated stem cells, and as such represent a large target population of somatic cells capable of passing along permanent changes to the DNA during future cell divisions.

- Some components of the immune system are not fully functional during development (Holladay and Smialowicz, 2000; Holsapple et al., 2003).

- Hormonal systems operate at different levels during different life stages (Anderson et al., 2000).

- There may be greater sensitivity to hormonal carcinogens early in life since the development of many organ systems is under hormonal control (e.g., male and female reproductive systems, thyroid control of CNS development).

- Induction of developmental abnormalities can result in a predisposition to carcinogenic effects later in life (Anderson et al., 2000; Birnbaum and Fenton, 2003; Fenton and Davis, 2002).

- Other factors that may play a role in increased cancer risk from exposures during critical developmental periods include differences in immunological activity, intestinal absorption, biliary and kidney excretion, blood and fat distribution, and expression of enzyme systems that activate or detoxify carcinogens.

Another reason that the young may be more susceptible to carcinogens than adults is simply because they have a much longer life over which the carcinogenic action may occur (IPCS 2006).

Conclusion

There are potential differences in inherent biological susceptibility and in exposures to environmental carcinogens of children as compared to adults. Throughout the course of their development, children go through different stages of specific exposure and vulnerability to environmental influences of carcinogens.

Children have different susceptibilities during different life stages owing to their dynamic growth and developmental processes as well as physiological, metabolic, and behavioural differences. Young children's immune systems, including the blood-brain barrier, are not fully developed, making them less able to fight and control the effects of biological and chemical assault on their bodies.

From conception through adolescence, rapid growth and developmental processes occur that can be disrupted by exposures to environmental chemicals. These include anatomical, physiological, metabolic, functional, toxicokinetic, and toxicodynamic processes. Children's bodies and brains are still developing, so lifelong structural or functional changes in their bodies are more likely than in adults.

Due to many of the specific characteristics and behaviour patterns described, children might be subject to higher exposure than adults and further, exposure pathways and exposure patterns may also be different in different stages of childhood. Exposure can occur *in utero* through transplacental transfer of environmental agents from mother to foetus or in nursing infants via breast milk.

Children consume more food and beverages per kilogram body weight than do adults, and their dietary patterns are different and often less variable during different developmental stages. They have a higher inhalation rate and a higher body surface area to body weight ratio, which may lead to increased exposures.

Children's normal behaviours, such as crawling on the ground and putting their hands in their mouths, can result in exposures not faced by adults.

Children's metabolic pathways may differ from those of adults.

Children have more years of future life and thus more time to develop chronic diseases that take decades to appear and that may be triggered by early environmental exposures. Because children generally have many more years ahead of them than adults, their risks from cumulative exposures are also greater. Diseases and conditions that are slow to develop are more likely to result from childhood exposures or exposures that are repeated over many years.

Overall, children are a vulnerable population subgroup with special susceptibilities and unique exposures to environmental chemicals, especially cancer-causing agents such as PAHs that have important implications for public health practices and risk assessment approaches.

There is currently no international scientific consensus on what is the best approach for assessing the risk of substances that are both genotoxic and carcinogenic and different approaches are used around the world. In many countries and especially within the EU, the advice is given to reduce the exposure to such substances – in particular that of children - to a level that is as low as reasonably achievable (known as the ALARA principle).

B.5.8.6 Summary and discussion of carcinogenicity

B.5.8.6.1 Animal studies

In numerous animal studies, the carcinogenic effects of PAHs, as single compounds or as various complex PAH-containing mixtures to which humans may be exposed, were examined by various routes of exposure. BaP is the best-studied PAH. It is carcinogenic by all routes tested in a number of animal species and is used as the main indicator of carcinogenic PAHs. The majority of carcinogenicity studies in experimental animals was conducted as skin painting studies, a limited number of studies following ingestion were available, and only a few animal studies have been published on inhalation exposure. Oral studies with pure BaP or PAH mixtures resulted in increased tumour incidences in the gastrointestinal tract, liver, and respiratory tract in rats and mice. Long-term inhalation of PAH mixtures or pure BaP induced tumours in the lung in rats and mice. In hamster inhalation of BaP caused tumours in the respiratory tract, but not in the lung. Dermal exposure to relative low BaP or various PAH concentrations induced benign and malign skin tumours in various strains of mice.

A tabular overview of all animal carcinogenicity studies is given again in Appendix 1 to this dossier.

B.5.8.6.2 Human data

No data are available on the carcinogenic effects of single PAHs in humans. In contrast, most of the human studies have addressed the carcinogenicity of PAH mixtures with BaP as lead compound. A considerable number of epidemiological studies have demonstrated that occupational exposure to soot, coal tar, and other PAH-containing mixtures is carcinogenic to humans. However, interpretation of these data is hampered by the many variables present in assessing work-related health effects in humans. These include the study design, exposure measurements, lifestyle factors, co-exposure to other toxic compounds and data presentation. Nevertheless, despite these confounding factors, the majority of the epidemiological data associated airborne PAH exposures with increased lung cancer risk. In addition, exposed workers, particularly at coke ovens and aluminium smelters, have shown excess bladder cancer for which a relationship to PAH exposure was highly suggestive. Skin cancer has been reported to be positively associated with dermal, but not with inhalation PAH exposure.

Some investigators estimated (excess) lifetime lung cancer risk. For instance, the results of a well-performed meta-analysis have been published (Armstrong et al., 2003, 2004), which included 39 different cohorts. Exposure in all these cohorts concerned coal-derived PAH sources from various industries (e.g., coke oven, gas works, aluminium production). The unit relative lung cancer risk (URR) at 100 $\mu g/m^3$ years BaP, in which 100 $\mu g/m^3$ BaP years corresponds to a concentration of 2.5 g/m^3 BaP over 40 years, was estimated at 1.20 (95 % CI, 1.11-1.29, $p<0.001$; log-linear model). This risk value was not driven by any particular cohort and was not dependent on analysis method. There was also an association of PAHs with bladder cancer (mean URR=1.33; 95 %CI: 1.16-1.52, no significant heterogeneity), but this finding was less robust than that for PAH-lung cancer, being largely dependent on two studies of aluminium production workers. The overall URR represents risk at fairly common exposures historically, but high for today. Risks at other exposures can be estimated from URR under (log-linear) model assumptions: e.g. at 1 mg/m^3 BaP for 40 years, cumulative exposure is 40 mg/m^3 BaP years, and the average lung cancer risk is 1.20 (40/100) = 1.076.

All in all the available human data were considered as helpful in supporting the view on PAHs as human carcinogens. However, as they were not considered suitable for being used as a basis for quantitatively assessing the risk potentially caused by the dermal exposure of consumers to PAH-contaminated consumer articles, (semi-)quantitative risk characterisation was based on the results from animal studies.

The increased susceptibility and vulnerability of infants and children compared to adults to carcinogens is of special importance. Children are not "little adults". There are potential differences in inherent biological susceptibility and in exposures to environmental carcinogens of children as compared to adults. Children have different susceptibilities during different life stages owing to their dynamic growth and developmental processes as well as physiological, metabolic, and behavioural differences. Therefore, it is difficult to make generalizations about the effect of age on susceptibility to chemical carcinogens due to the heterogeneous nature of the influences – with regard to the individual age groups and individual substances – and the reaction mechanisms of the organism.

Probable higher susceptibility of children might be due to the fact that children are in a dynamic state of growth and have more time to develop diseases with long latency, more years of life to be lost and more suffering to be endured as a result of impaired health (Charnley and Putzrath, 2001; IPCS 2006).

The case for the increased vulnerability of children involves many factors, which can be grouped into three broad areas. The first is that children are exposed to relatively higher exogenous doses of environmental toxins, i.e., intakes are increased compared with adults, which can be attributed both to lifestyle and physiology. The second area of concern relates to the manner in which compounds are dealt with in the body of a child. A child's response to a hazardous substance in the environment can differ to that of an adult's for a number of physiological reasons, relating to differences in the disposition, bioavailability, toxicokinetics and toxicodynamics. Finally, if exposure begins in childhood, there is a greater likelihood that exposure becomes chronic, and further, that adverse health effects become manifest, even for those with a long latency period such as cancer (Wild and Kleinjans, 2003).

B.5.9 Toxicity to reproduction

BaP is a known and legally classified reproduction toxicant (both in terms of fertility impairment and developmental toxicity, cf. section B.3). However, the observed effects are considered to be threshold effects and it is assumed that these thresholds will be orders of magnitude higher than potential DMELs for carcinogenicity.

A similar conclusion was drawn by the Swedish CA when preparing their assessment of creosote under the BPD (EC 1998).

B.5.10 Other effects

Not relevant for this dossier

B.5.11 Derivation of DMELs

The PAHs treated in this dossier are known genotoxic carcinogens. Even if it is acknowledged that the potential mechanisms of chemical carcinogenesis are certainly more complex, in general the central paradigm of the risk assessment of genotoxic carcinogens, i. e. that a threshold for tumorigenic activity cannot be set, is still generally accepted and applied in the characterisation of the potential risk caused by genotoxic carcinogens.

As no thresholds for carcinogenicity can be set, also no 'Derived *No* Effect Levels' (DNELs) but only so-called 'Derived *Minimum* Effect Levels' (DMELs) can be derived.

B.5.11.1 Procedure followed in the derivation of DMELs

The current dossier closely follows the procedure as laid out in the REACH Guidance on Information Requirements and Chemical Safety Assessment (IR/CSA) (ECHA 2008), Section R.8.5, which can be summarised as follows:

- Gather all available relevant data, extract dose-response relationships and identify dose levels with a significant increase in tumour incidence. This should be done separately for all studies available, along all routes and for all different types of substance-related tumours.

- Out of this database, select suitable studies for DMEL derivation.

- Identify the most suitable dose metric.

- Derive suitable dose descriptors. In general, a T25 (dose level with a net 25 % increase in tumour incidence) and, if suitable, a benchmark dose (BMD; most often the BMD_{10} - 10 % increase in tumour incidence - or its lower 95 % confidence band, the $BMDL_{10}$) are calculated.

- Modify – if necessary - the dose descriptor(s) to the correct starting point thereby reflecting on differences between the animal experiment and the human exposure scenario.

- Extrapolate from the high- to the low-dose range. Different methods such as the so-called 'linearised' or the 'large assessment factor' approach may be used. Apply - if necessary – suitable assessment/extrapolation factors in this process.

It should be stressed that the detailed steps taken within the above procedure, the different methodologies used and their inherent uncertainty, as well as the overall meaningfulness of the extrapolation results have been the subject of a still ongoing, intense debate among regulators in Europe. The whole DMEL concept has also been criticised for introducing a *de facto* threshold for non-threshold carcinogens by the back door.

Even within the Guidance on IR/CSA, different approaches for the derivation of DMELs are offered which lead to substantially different results. Therefore, rather than determining a single DMEL for risk characterisation, in this dossier an overview will be given of the range of possible DMELs derived from different studies and for different carcinogenicity endpoints by using the different approaches without prejudice.

The uncertainty associated with the derivation of a DMEL may well be in the order of ± one magnitude or even more. Considering all of these points, risk characterisation has to be performed

with some sense of proportion. As a consequence, in Section B.10 below, the range of DMELs derived in the present section will be compared with exposure estimates for highly PAH-contaminated consumer products as well as for the same products with theoretical PAH contents at the limit values proposed in this dossier. In addition, the resulting risk characterisation will try and integrate the results with those of other published risk assessments of PAHs.

B.5.11.2 Assumptions made in deriving DMELs for benzo[a]pyrene and the other PAHs in this dossier

B.5.11.2.1 Selection of toxicological studies for T25 and/or BMD/BMDL calculations

From a greater range of available studies, mainly those were chosen which were considered as key studies in previous risk assessments of BaP or PAHs. Given the fact that most of these evaluations have been performed before 2003, the published literature since then has been searched in order to find out, whether more recent carcinogenicity experiments were reported (with the exception of one dermal study performed with creosote, this was not the case).

The following criteria were then applied to further limit the number of selectable studies and – within these studies, endpoints [1] - for DMEL calculation:

1. Only studies, in which BaP was administered as the component of a mixture of PAHs, were considered for the following two reasons:

    a. As regards the carcinogenic potential of PAH mixtures of varying composition, studies with BaP alone are not considered representative of the problems addressed in this dossier, aiming at regulating a total of eight PAHs in articles which even contain a multitude of other PAHs (some of which may be even more potent carcinogens).

    b. T25 or BMD values obtained from carcinogenicity studies performed with BaP alone tended to be clearly higher than those from studies using mixtures (mostly tar preparations of different origin), even when both were based on BaP content.

2. Studies with strong deficits in experimental design and/or reporting were only considered, if meaningful results could be obtained in spite of these flaws.

3. Within a study that had been selected in step 1 only endpoints were considered, for which a 'meaningful' dose-response relationship could be established, which was assumed to be the case if:

    a. at least 3 dose levels in addition to a concurrent control could be obtained. Studies with only two dose levels + control were ruled out for BMD calculation by definition

[1] For the sake of clarity, the term 'endpoint' in this subsection is without distinction used to designate either a certain type of tumour in a certain location, e.g. 'skin carcinoma' or 'skin (benign tumours only)', all types of observed tumours at a certain site (e.g. 'lung tumours'), or even 'number of tumour-bearing animals (i.e. regardless of tumour type or location)'.

and were only used for T25 calculations, if the range of net tumour incidences observed included the net 25 increase, i. e. when an experimental value was obtained above the potential T25. Studies with only one dose level in addition to control were not used.

b. ideally, response increased monotonously with dose (although small deviations were tolerated, e.g. cf. ID 16 in Appendix 1)

c. ideally, if the highest observed substance-related net increase in incidence was above 50 %.

When from a given study the endpoint 'any tumour' (no. of tumour-bearing animals) would qualify to the same degree as a more specific endpoint (e.g. 'skin carcinoma'), the latter was chosen, if both the incidences across all groups and the calculated T25 or BMD values were comparable.

In a further selection step, BMD_{10}/$BMDL_{10}$ values were deselected for DMEL calculation, when appropriate quality indicators pointed at a poor goodness-of-fit (cf. section B.5.11.3.3).

Finally, out of the remaining endpoints from a given study, the one resulting in the lowest T25/BMD values was selected.

B.5.11.2.2 Mechanistical considerations – selection of the most suitable dose metric

Graffi and Bielka (1959, as cited by Habs et al., 1980) have summarised the pharmacodynamic mechanism of action in PAH-induced carcinogenesis in the following way:

'[…] The number of 'clones of transformed cells' is directly proportional to the dose applied, the number of 'clones of transformed cells' produced by a given dose is limited. A certain amount of a certain carcinogen will always induce about the same number of 'clones of transformed cells'.

The activities even of smallest individual doses are added up without loss as far as the 'number of clones of transformed cells' is concerned.

In the case of low concentrations in the solution and a not too rapid sequence of applications, the number of 'clones of transformed cells' is completely independent of the time distribution and the size of partial doses and is proportional only to the carcinogenic total dose.

A threshold value cannot be determined in the formation of a 'clone of transformed cells' or is at least very low. […]'

Although today the highly complex multi-stage process of chemical carcinogenesis is still far from being understood, the basic assumption of the above description from 50 years ago can still be supported for certain steps in carcinogenesis such as initiation and/or promotion, i. e.:

For every cell in the body, the statistical likelihood of being turned into a cancer cell by a chemical carcinogen is expected to be increasing with an increasing number of contacts between the cell and the carcinogenic agent. Or, in terms of the body as a whole, the likelihood for suffering from

chemically induced cancer should be in some way dependent on the cumulative lifetime dose per number of body cells in contact with the carcinogenic agent

When evaluating the tumours caused by BaP or PAH mixtures (cf. section 5.8 above) notably tumours are often found to be located along the route of entry (oral: tongue, oesophagus, forestomach, liver; dermal: skin; inhalation: lung and upper airways).

At first glance, one might be tempted to declare these tumours as 'local' and assume a concentration-dependency and – consequently – a threshold concentration below which no carcinogenicity would be expected to occur. However for the following reasons this view is not supported:

In the oral studies, also tumours in remote organs such as the lung were detected, which can only be explained by systemic distribution of the carcinogenic agent(s). As BaP and the other PAHs are proven to be bioavailable to a significant degree along all three routes of uptake, similar effects cannot be ruled out for an exposure via dermal contact or inhalation.

In the dermal or inhalation studies summarised in section B.5.8, only the 'local' tumours were evaluated, while other organs were not examined and thus, reliable data on additional 'systemic tumours' is missing.

B.5.11.3 DMEL calculation

The focus of this dossier is placed on dermal exposure of consumers towards PAH-contaminated articles and exposure assessment (section 0) was therefore almost exclusively restricted to the dermal route.

Therefore, while all studies fulfilling the criteria given in section B.5.11.2.1 were accounted for, only dermal DMELs were calculated (it is noted that for other routes, slightly different DMELs would result when using different assessment factors for correction of the chosen for route-specific bioavailability).

B.5.11.3.1 Selection of the most suitable dose metric

As mentioned in the previous section, in a first approximation the likelihood of tumourigenesis can be assumed to correlate with the lifetime cumulative dose per number of body cells.

This dose metric is easily converted into a lifetime average dose per kg body weight and day, which - for ease of calculation and in line with the recommendations of the REACH Guidance on Information Requirements - was therefore chosen as the relevant dose metric for DMEL derivation in this dossier.

B.5.11.3.2 Calculation of the dose descriptors

In accordance with the REACH Guidance on IR/CSA, Chapter R.8 (ECHA 2008) T25 and BMD_{10}/$BMDL_{10}$ values were calculated as follows:

B.5.11.3.2.1 Calculation of T25

T25 values were calculated by the method described by Dybing and co-workers (Dybing et al., 1997), i. e. first the lowest dose level with a significant increase in tumour incidence vs. control was identified. Then higher dose levels were examined in order to identify levels which – when used for

T25 calculation – would result in lower values than would be generated using the originally selected dose level. Significance was established by performing a variant of the χ^2 test routine ('Test of Equal or Given Proportions', using the statistical software 'R' cf. http://stat.ethz.ch/R-manual/R-patched/library/stats/html/prop.test.html, as of March 20, 2010).

T25 calculation was performed after subtracting background incidence observed in the controls (if applicable) and resetting the remaining animal number to a hundred percent. Then a simple 'rule of three' was applied: the selected dose level was multiplied by a factor of $25/I_{n,S}$, where $I_{n,S}$ designates the net incidence above control.

The equation used can be formulated as follows:

$T25 = D_S \times 0.25 \times 1/I_{n,S} = D_S \times 0.25 \times (n_S-I_C)/(I_S-I_C)$

With

D_S	=	Dose level selected as basis for T25 calculation
I_C	=	Tumour incidence (number of animals with tumours) in the control group
$I_{n,S}$	=	Net tumour incidence at dose level D_s corrected for background tumour incidence in controls
I_S	=	Tumour incidence (number of animals with tumours) at the dose level selected as basis for T25 calculation
n_S	=	Number of animals at the dose level selected as basis for T25 calculation.

B.5.11.3.2.2 Calculation of BMD_{10}/$BMDL_{10}$

BMD_{10}/$BMDL_{10}$ values were calculated using the benchmark dose software 'BMDS' (version 2.1.1) as issued by the United States Environmental Agency (US EPA, as of March 20, 2010, the software can be downloaded from cf. http://www.epa.gov/ncea/bmds/).

Two different models, i. e. 'Probit' and 'Multistage Cancer' were used in the calculations. In addition, both a χ^2 test p-coefficient and the so-called Akaike Information Criterion (AIC) were calculated in order to obtain figures of merit for evaluating the quality of the models (basically, the goodness-of-fit of the model curve fitted to the dose-response data).

B.5.11.3.3 Correction factors applied to the dose descriptors

In accordance with the REACH Guidance on IR/CSA, Chapter R.8 (ECHA 2008) correction factors were applied in order to correct the nominal (external) dose levels for deviations from a hypothetical 'reference design' of substance administration for 7 d/wk and lifetime. In addition, possible differences in bioavailability between the animal experiment and the expected human exposure scenario were accounted for.

When deriving DMELs for dermal exposure of humans from sweat, a correction factor of 2.5 was used in order to account for the lower dermal absorption rate assumed for this scenario (20 %) as compared to the absorption rates deduced from the animal experiments (50 % for all routes).

Furthermore, when a DMEL was derived based on an inhalation study, different respiratory rates of animals and humans were accounted for by using the appropriate allometric scaling factors suggested by the REACH Guidance on IR/CSA.

B.5.11.3.4 Results of T25 and BMD calculations

An overview of the calculated T25 and BMD_{10}/$BMDL_{10}$ values and the correction factors applied to these dose descriptors is provided in Appendix 2.

B.5.11.3.5 Assessment factors

In the linearised approach, only an assessment factor for allometric scaling (i. e. 4 for rats and 7 for mice) was applied. In accordance with section R.8 of the REACH Guidance on Information Requirements, the use of further assessment factors was not deemed necessary.

B.5.11.3.6 High- to Low-Dose extrapolation

Basically two different methods are available: 'Linearised' and 'Large assessment factor' approach. In the REACH Guidance on IR/CSA, Chapter R.8 (section 8.5.2.1), the strategy recommended for assessment of consumer risk is using the linearised approach and calculate DMELs at the extra risk level of 10^{-6}. However in addition, for the sake of comparison, DMELs were also calculated by the linearised approach at the 10^{-5} risk level and by the large assessment factor approach.

B.5.11.3.7 Results of the DMEL calculations

In summary, the following results were obtained:

For the calculations based on T25 as dose descriptor, a total of 11 dose-response relationships from as many different experiments were evaluated, including 5 studies with oral, 3 with dermal, and 3 with inhalation application.

The three inhalation studies could not be used for the calculations based on BMD_{10}/$BMDL_{10}$, as for these descriptors, a study design using 3 treated groups + control is required.

For each of the selected studies (where appropriate) T25, BMD_{10}, and $BMDL_{10}$ estimates were used as dose descriptors. For all of these descriptors (thus, for each of the studies) DMELs were calculated applying both the 'Large Assessment Factor' and the 'Linearised' approach (the latter at both the 10^{-5} and 10^{-6} risk levels and using the 'Probit' as well as the 'Multistage Cancer' algorithms for curve fitting). A commonly used approach would have been to derive a final DMEL from that study considered the most sensitive (or otherwise most relevant), i. e. the 'key study'. In this dossier, a different approach was chosen: rather than performing quantitative risk characterisation on the results of only a single study, a number of selected carcinogenicity studies were taken into account.

The obtained range of derived DMELs was then compared with the range of dermal doses that consumers can be expected to be exposed to when having skin contact to consumer articles.

A detailed representation of the results of the DMEL calculations can be found in Table 60 - Table 64 in Appendix 2. The associated uncertainties as well as their meaning in the context of risk characterisation for consumers are discussed in section B.10.

The range of values obtained is also given in Table 32

Table 32: Overview of dermal DMEL ranges obtained by using the different calculation methods featured in the REACH Guidance on Information Requirements (section R.8). Cf. Appendix 2 for details. All values in ng/kg bw/d

Method used	Lowest value	Highest value	High/Low ratio
T25, linearised approach, 10^{-5} risk level	0.047	9.921	211
T25, linearised approach, 10^{-6} risk level	0.005	0.992	198
T25, large assessment factor approach	0.133	27.780	209
BMD_{10}, Probit model, linearised approach, 10^{-5} risk level	0.026	10.493	404
BMD_{10}, Probit model, linearised approach, 10^{-6} risk level	0.003	1.049	350
BMD_{10}, Probit model, large assessment factor approach	0.074	29.379	397
$BMDL_{10}$, Probit model, linearised approach, 10^{-5} risk level	1.3×10^{-4}	4.657	35800
$BMDL_{10}$, Probit model, linearised approach, 10^{-6} risk level	1.3×10^{-5}	0.466	35800
$BMDL_{10}$, Probit model, large assessment factor approach	4×10^{-4}	13.040	32600
BMD_{10}, Multistage Cancer model, linearised approach, 10^{-5} risk level	0.046	10.629	231
BMD_{10}, Multistage Cancer model, linearised approach, 10^{-6} risk level	0.005	1.063	213
BMD_{10}, Multistage Cancer model, large assessment factor approach	0.129	29.761	231
$BMDL_{10}$, Multistage Cancer model, linearised approach, 10^{-5} risk level	0.035	8.511	243
$BMDL_{10}$, Multistage Cancer model, linearised approach, 10^{-6} risk level	0.004	0.851	213
$BMDL_{10}$, Multistage Cancer model, large assessment factor approach	0.099	23.829	241
All calculations	1.3×10^{-5}	29.761	229

It can be seen that even for the same calculation methods calculated values sometimes span a range of > 2 orders of magnitude, when all available studies were considered. Lowest DMELs represent the outcome of the most sensitive studies, the highest DMELs were obtained from the least sensitive ones.

In the particular case of the calculations using the BMDL10 dose descriptor and the Probit fitting model, even values from the upper fg/kg bw/d range up to about 30 ng/kg bw/d were obtained (4 orders of magnitude). However, as the Multistage Cancer model is the approach recommended by the REACH IR/CSA guidance, the very low values obtained by the Probit approach are not considered further in this dossier. They do also not have any practical regulatory relevance/impact (i. e. they could not be regulated due to lack of availability of suitable analytical technology) and also far below inevitable exposure of consumers via the environmewnt or food/drinking water.

Table 32 above shows, that of the two main approaches presented in the REACH IR/CSA guidance R.8, i. e. the T25-based linearised approach at the 10^{-6} risk level and the BMD_{10}-based large assessment factor (or 'EFSA') approach using the Multistage Cancer model, the latter leads to estimates which are less conservative by a factor of ca. 30 in the present case (when comparing the upper limits of the corresponding DMEL ranges).

Conversely, the choice of dose descriptor (T25/BMD_{10}/$BMDL_{10}$) did not impact significantly on the outcome of the calculations.

Admittedly, the range of results presented in Table 32 could have been perhaps narrowed down to a greater extent when a closer scrutiny of the selected carcinogenicity studies would have been performed. On the other hand, appropriate criteria for such an operation cannot be easily applied without the danger of manipulating the database towards a particular outcome.

Given that Article 68 (2) already implicitly considers the presence of CMR substances of DSD categories 1 or 2 in consumer articles as an (unwanted) risk, it was felt by the authors of this dossier that the DMEL ranges obtained here would suffice to perform a quantitative risk characterisation.

The variation of DMEL results should best be seen in light of all the factors of uncertainty listed below in section B.10.1.2.2. It should also be noted that these DMEL ranges do by and large correspond to levels to which consumers are inevitably exposed in everyday life (e. g. via food and other environmental sources). They are also situated in the range of what others have termed the 'Threshold of Toxicological Concern (TTC)' for genotoxic carcinogens (i. e. 2.5 ng/kg bw/d, Kroes et al., 2007) .

Finally it should be kept in mind, that as DMELs are not DNELs and thus cannot be used to make a statement on the total absence of a carcinogenic risk, they should rather be seen as a means to identify risk levels calling for urgent action in risk management.

Summing up, the following DMEL results ranges (excluding the Probit calculations) were obtained:

Large Assessment Factor approach: 0.1 – 30 ng/kg bw/d

Linearised Approach, 10^{-5} risk level: 0.03 – 10 ng/kg bw/d

Linearised Approach, 10^{-6} risk level: 0.004 – 1 ng/kg bw/d

The boundaries of these ranges roughly represent the results from the most (lower boundaries) and least (upper boundaries) sensitive studies, respectively.

Instead of taking the most sensitive DMEL from a single study forward to quantitative risk characterisation, the whole range of DMELs using different approaches were accounted for.

B.6 Human health hazard assessment of physico-chemical properties

B.6.1 Explosivity

Not relevant for this dossier

B.6.2 Flammability

Not relevant for this dossier

B.6.3 Oxidising potential

Not relevant for this dossier

B.7 Environmental hazard assessment

B.7.1 Aquatic compartment (including sediments)

In the effect assessment below the ecotoxicity data has been evaluated separately for the eight EPA-PAHs in question.

B.7.1.1 Benzo[a]pyrene

Results from studies on the aquatic toxicity of benzo[a]pyrene are shown in Table 33. The water-solubility of benzo[a]pyrene is 1.2-1.8 µg/L (Mackay et al., 2000). Relevant to assess the acute toxicity is a test with Daphnia magna under exposure to UV radiation. Here the EC_{50} is 1.2 µg/L for immobility of Daphnia magna after exposure for 24 h with a 16:8 light:dark photoperiod, then two hours exposure to UV (295-365 nm; peak 340 nm) with an intensity of 370 ± 20 µW/cm^2, followed by one hour of recovery in the test medium (Wernersson, 2003).

Chronic toxicity of benzo[a]pyrene was reported for the alga Pseudokirchneriella subcapitata with an E_rC_{10} of 0.78 µg/L, and for reproduction of Ceriodaphnia dubia with an EC_{10} of 0.5 µg/L in a 7-d study when exposed to laboratory light without UV (Bisson et al.,, 2000).

In a 28-d early life stage (ELS) study with Brachydanio rerio no effects were observed up to the highest test concentration of 4.0 µg/L, which is already above the water solubility of benzo[a]pyrene (Hooftman and Evers-de Ruiter, 1992).

In another ELS study with Oncorhynchus mykiss a NOEC of 1.5 µg/L was obtained for developmental abnormalities as endpoint (Hannah et al., 1982). Evaluation of the data presented by Hannah *et al.* with a log-logistic relationship resulted in the derivation of an EC_{10} of 2.9 µg/L (The Netherlands - Bureau REACH, 2009), which again is above the water solubility of benzo[a]pyrene. Furthermore, it has been shown that UV radiation increases the long term toxicity of benzo[a]pyrene. For shell development of Crassostrea gigas, when exposed to UV radiation, the calculated EC_{10} was 0.22 µg/L whereas under UV-lacking fluorescent laboratory lighting conditions the resulting EC_{10} was 1.1 µg/L (Lyons et al., 2002).

As the study on shell development of the marine mollusc Crassostrea gigas resulted in the lowest reliable chronic EC_{10} value (0.22 µg/L) it was chosen as key study for T-assessment.

Table 33: Overview of studies concerning the aquatic toxicity of benzo[a]pyrene.

Species	Duration	Endpoint	Value	Comment	References
Freshwater organisms, acute					
Daphnia magna	27 h	EC_{50}	1.2 µg/L	16:8 hour light:dark followed by 2 hour UV-A B radiation and 1 hour recovery	Wernersson, 2003

Species	Duration	Endpoint	Value	Comment	References
Freshwater organisms, chronic					
Pseudokirchneriella subcapitata	72 h	EC_{10} growth	0.78 µg/L	Light intensity 6000 - 8000 lux, cool white fluorescent lamps	Bisson et al., 2000
Oncorhynchus mykiss	36 d	NOEC EC_{10} abnormalities	1.5 µg/L (2.9 µg/L, calculated, above WS)	ELS	Hannah et al., 1982
Ceriodaphnia dubia	7-d	EC10 reproduction	0.5 µg/L	Laboratory light photoperiod 16:8 h light:dark at less than 500 lux	Bisson et al., 2000
Marine organismus, chronic					
Crassostrea gigas	48 h	NOEC shell development EC_{10}	0.5 µg/L 0.22 µg/L	Embryos 12:12 hour light:dark UV-A B radiation	Lyons et al., 2002

B.7.1.2 Benzo[e]pyrene

The water solubility of benzo[e]pyrene is about 6.3 µg/L at 25°C (Pearlman et al., 1984). Data on aquatic toxicity are scarce, as only one study was published: Newsted and Giesy determined the median lethal time of neonates of Daphnia magna (Newsted and Giesy, 1987). In this study already mentioned above, the daphnids were exposed to one concentration of benzo[e]pyrene (measured concentration of 0.7 µg/L). The test was performed as a static renewal acute toxicity test. After 24 hours of exposure with a 16:8 light : dark photoperiod, the animals were exposed to UV-light with an intensity of 25 ± 3 µW/cm^2 UV-B (310 ± 36 nm), 120 ± 5 µW/cm^2 UV-A (365 ± 36 nm), and 680 ± 10 µW/cm^2 visible light (400 to 700 nm). The median lethal time after UV-radiation started was 9 hours 16 min. This type of study is however not designed to determine dose-response relationships and hence quantitative data on toxicity or toxicity threshold values cannot be derived from the result.

Table 34: Overview of studies concerning the aquatic toxicity of benzo[e]pyrene.

Species	Duration	Endpoint	Value	Comment	References
Freshwater organisms, acute					
Daphnia magna	Until 50 % of the test animals died	LT_{50}	9 h 16 min (after commencement of irradiation and exposure to 0.7 µg/L benzo[e]pyrene)	Static renewal test: exposure to 0.7 µg/L benzo[e]pyrene After 16:8 hours light:dark photoperiod irradiation with UV A and B + visible light	Newsted and Giesy, 1987

B.7.1.3 Benzo[a]anthracene

Data on acute toxicity of benzo[a]anthracene are available for algae, crustaceans and amphibians. Effects within the water solubility of the substance have been observed for the crustacean Daphnia pulex upon exposure to mixed fluorescent and natural light and for larvae of the amphibian Pleurodeles waltl irradiated throughout the experiment with UV-A light (see Table 34). The 96-h EC_{50} of Daphnia pulex exposed under a 12:12 h photoperiod to mixed fluorescent and natural light was 10 µg/L (Trucco et al., 1983). The 48-h EC_{50} of Daphnia magna from a test in the dark was higher than 9.1 µg/L (Bisson et al., 2000). Also under artificial light with a photoperiod of 16:8 h light:dark 50% immobility was not reached in the highest concentration when Daphnia magna was exposed for 24 hour. The same test followed by irradiation with UV (295-365 nm; peak 340 nm) with an intensity of 370 ± 20 µW/cm^2 for two hours and one hour of recovery in the test medium lead to an LC_{50} of 3.4 µg/L. UV-radiation thus increases the toxicity of benzo[a]anthracene.

Larvae of the amphibian Pleurodeles waltl irradiated throughout the experiment with UV-A light (320-400 nm with a maximum at 365 nm) at 250 µW/cm^2 survived at 3.1 µg/L and died all at 6.3 µg/L (Fernandez and L'Haridon, 1992).

In a study with larvae of Pimephales promelas the median lethal time was determined (Oris and Giesy, 1987). 7-d old larvae were exposed to a measured concentrations of 1.8 µg/L benzo[a]anthracene for an incubation period of 24 hour in the absence of UV-radiation and thereafter exposed to UV-light with an intensity of 20 µW/cm^2 UV-B (290-336 nm), 95 µW/cm^2 UV-A (336-400 nm). After the incubation time of 24 hours, the medium was renewed every twelve hours. The median lethal time after UV-radiation started was 65 hours. Thus, after 89 hours, of which the last 65 hours were with UV radiation, 50% mortality of the fish larvae occurred at 1.8 µg/L.

Chronic toxicity data are available for algae, cyanophyta, aquatic plants and crustaceans. The lowest chronic toxicity has been observed for the alga Pseudokirchneriella subcapitata with an EC_{10} of 1.2 µg/L for growth inhibition (Bisson et al., 2000). This study was therefore chosen as key study.

The aqueous solubility of benz[a]anthracene is around 10 µg/L (Mackay et al., 2000). The light intensity may play an important role in the lower EC_{10} from the study by Bisson et al. (2000).

Table 35: Overview of studies concerning the aquatic toxicity of benzo[a]anthracene.

Species	Duration	Endpoint	Value	Comment	References
Freshwater organisms, acute					
Daphnia pulex	96 h	EC_{50}	10 µg/L	12:12 h photoperiod to mixed fluorescent and natural light	Trucco et al., 1983
Daphnia magna	48 h	EC_{50}	> 9.1 µg/L	Dark	Bisson et al., 2000
Pleurodeles waltl (larvae)	6 d	LC_{50}	at 3.1 µg/L 100% survival; at 6.3 µg/L 100% mortality	Irradiation with UVA light throughout experiment	Fernandez and L'Haridon, 1992

Species	Duration	Endpoint	Value	Comment	References
Freshwater organisms, chronic					
Pseudokirchneriella subcapitata	72-h	EC_{10} growth	1.2 µg/L	Light intensity 6000 - 8000 lux	Bisson et al., 2000

B.7.1.4 Chrysene

The water solubility of chrysene is about 1.6 µg/L, with a range between 1.0 and 3.3 µg/L (Mackay et al., 2000). Around or below this value, no significant effects were observed for any species in a regular toxicity experiment, although chronic toxicity studies were performed with algae, crustaceans (including Daphnia) and fish. The only study, that showed a considerable effect of chrysene, was a determination of the median lethal time of neonates of Daphnia magna (Newsted and Giesy, 1987). In this experiment, the daphnids were exposed to one concentration of chrysene (measured concentration of 0.7 µg/L). The test was performed as a static renewal acute toxicity test. After 24 hours of exposure with a 16:8 light:dark photoperiod, the animals were exposed to UV-light with an intensity of 25 ± 3 µW/cm^2 UV-B (310 ± 36 nm), 120 ± 5 µW/cm^2 UV-A (365 ± 36 nm), and 680 ± 10 µW/cm^2 visible light (400 to 700 nm). The median lethal time after UV-radiation started was 24 hours. Thus, after 48 hours, of which the last 24 hours were with UV radiation, 50% mortality of the daphnids occurred at 0.7 µg/L. This type of study is however not designed to determine dose-response relationships and hence quantitative data on toxicity or toxicity threshold values cannot be derived from the result.

Table 36: Overview of studies concerning the aquatic toxicity of chrysene.

Species	Duration	Endpoint	Value	Comment	References
Freshwater organisms, acute					
Daphnia magna	Until 50 % of the test animals died	LT50	24 h (after commencement of irradiation and exposure to 0.7 µg/L Chrysene)	Static renewal test: exposure to 0.7 µg/L chrysene after 16:8 hours light:dark photoperiod irradiation with UV A and B + visible light	Newsted and Giesy, 1987

B.7.1.5 Benzo[b]fluoranthene

No effects have been observed at concentrations within the water solubility of benzo[b]fluoranthene, i.e. up to 1.1 - 1.5 µg/L (Mackay et al., 2000). An acute study with Daphnia magna in the dark showed no effects at the tested concentration of 1.1 µg/L (Bisson et al., 2000). In a 24-h study with the same organism and a photoperiod of 16:8 h light: dark, extended by two hours of irradiation with UV light followed by two hours of recovery, the EC_{50} for immobilisation was determined as 4.2 µg/L, which is above the water solubility of benzo[b]fluoranthene (Wernersson and Dave, 1997).

Table 37: Overview of studies concerning the aquatic toxicity of benzo[b]fluoranthene.

Species	Duration	Endpoint	Value	Comment	References
Freshwater organisms, acute					
Daphnia magna	24 h	EC_{50}	4.2 µg/L	16:8 hour light:dark followed by 2 hour UV-A B radiation and 2 hour recovery	Wernersson and Dave, 1997

B.7.1.6 Benzo[j]fluoranthene

The water solubility of benzo[j]fluoranthene is about 2.5 µg/L (Yalkowsky and Dannenfelser, 1992). Acute toxicity studies are reported for Daphnia magna (EC_{50} for immobilization 2.3 µg/L) and Oryzias latipes where the LC_{50} is above the concentration for water solubility (>4.2 µg/L). Also there were several chronic studies conducted. The most sensitive one was done on Pseudokirchneriella subcapitata where a NOEC value of 0.15 µg/L was found. All data were reported on the Chemical Risk Information Platform of the Japanese National Institute of Technology and Evaluation (CHRIP) and are shown in Table 38.

Table 38: Overview of studies concerning the aquatic toxicity of Benzo[j]fluoranthene.

Species	Duration	Endpoint	Value	Comment	References
Freshwater organisms, acute					
Daphnia magna	48 h	EC_{50} (acute immobilization)	2.3 µg/L		NITE, 2010
Oryzias latipes	96 h	LC_{50}	>4.2 µg/L		
Freshwater organisms, chronic					
Daphnia magna	21 d	EC_{50} (Reproduction)	>2.7 µg/L		NITE, 2010
		NOEC (Reproduction)	>=2.7 µg/L		
Pseudokirchneriella subcapitata	72 h	EC_{50} (growth)	0.26 µg/L		
		NOEC (growth)	0.15 µg/L		
Pseudokirchneriella subcapitata	72 h	EC_{50} (cell augmentation)	2.4 µg/L		
		NOEC (cell augmentation	0.85 µg/L		

B.7.1.7 Benzo[k]fluoranthene

Acute toxicity data for benzo[k]fluoranthene are only available for Daphnia magna. However, in the two available studies (Bisson et al., 2000; Verrhiest et al., 2001) no effects were observed. However, due to the low solubility of benzo[k]fluoranthene of about 1 µg/L (Mackay et al., 2000), acute effects are not anticipated. For algae no EC_{50} is presented. However, in the 72-h study with Pseudokirchneriella subcapitata the EC_{10} for growth is larger than 1 µg/L (Bisson et al., 2000) and hence the EC_{50} must also be higher than this value. In the 7-d reproduction study with Ceriodaphnia dubia no effects were observed either (Bisson et al., 2000). In two studies, the effects of benzo[k]fluoranthene in an ELS test with Brachydanio rerio were examined. In the first 28-d study one concentration of 0.58 µg/L was tested. At this concentration 52 % mortality occurred (Bisson et al., 2000). In a second 42-d study a dose-response relationship was examined. The mentioned concentrations here are based on measured concentrations per concentration and not on average recovery times the nominal concentration as given in the report. The LC_{50} estimated from the presented data with a log-logistic relationship was 0.65 µg/L. From the data for weight and length EC_{10} are derived of 0.31 and 0.17 µg/L. Due to the good fit of the log-logistic equation, these estimates have a low uncertainty.

The most sensitive endpoint is length of Brachydanio rerio in an ELS test. The EC_{10} for this endpoint is 0.17 µg/L. With an assessment factor of 10, the PNEC for fresh water is 0.017 µg/L.

Table 39: Overview of studies concerning the aquatic toxicity of benzo[k]fluoranthene.

Species	Duration	Endpoint	Value	Comment	References
Freshwater organisms, chronic					
Brachydanio rerio	28 d	LC_{50}	0.58 µg/L	ELS	Bisson et al., 2000
Brachydanio rerio	42 d	LC_{50} EC_{10} weight EC_{10} length	0.65 µg/L 0.31 µg/L 0.17 µg/L	ELS	

B.7.1.8 Dibenzo[a,h]anthracene

Results of studies of the aquatic toxicity of dibenzo[a,h]anthracene are provided in Table 40. Chronic toxicity studies with fresh water species are available for crustaceans, aquatic plants, and algae.

The 72-h EC_{10} for the growth rate of Pseudokirchneriella subcapitata was 0.14 µg/L (Bisson et al., 2000). In both the test with C. dubia and P. subcapitata concentrations were measured. As the study with Pseudokirchneriella subcapitata resulted in the lowest reliable chronic EC_{10} value it was chosen as the key study.

Table 40: Overview of studies concerning the aquatic toxicity of dibenzo[a,h]anthracene.

Species	Duration	Endpoint	Value	Comment	References
Freshwater organisms, acute					
Daphnia magna	27 h	EC_{50}	1.8 µg/L	16:8 hour light:dark followed by 2 hour UV-A B radiation and 1 hour recovery	Wernersson, 2003
Daphnia magna	28 h	EC_{50}	4.6 µg/L	16:8 hour light:dark followed by 2 hour UV-A B radiation and 2 hour recovery	Wernersson and Dave, 1997
Freshwater organisms, chronic					
Pseudokirchneriella subcapitata	72 h	EC_{10} growth	0.14 µg/L	Light intensity 6000 – 8000 lux	(Bisson et al., 2000)

B.7.1.9 Summary and discussion on aquatic toxicity

An overview on the aquatic toxicity obtained in the key studies (chronic tests) selected for the PAHs is provided in Table 41.

Table 41: Aquatic toxicity of the PAH-constituents observed in the selected key studies (chronic effects)

Substance	Value	Key study endpoint
Benzo[a]pyrene	0.22 µg/L	EC_{10} shell development, *Crassostrea gigas larvae,* 48-h ELS
Benzo[e]pyrene	9 h 16 min (after commencement of irradiation and exposure to 0.7 µg/L benzo[e]pyrene)	LT_{50} - Until 50 % of the test animals died, *Daphnia magna*
Benz(a)anthracene	1.2 µg/L	EC_{10} growth, Pseudokirchneriella subcapitata, 72-h
Chrysene	24 h (after commencement of irradiation and exposure to 0.7 µg/L Chrysene)	LT_{50} - Until 50 % of the test animals died, *Daphnia magna*
Benzo[b]fluoranthene	-	No toxicity has been observed up to the water solubility limit of the substance
Benzo[j]fluoranthene	0.15 µg/L	NOECgrowth, Pseudokirchneriella Subcapitata, 72 h
Benzo[k]fluoranthene	0.17 µg/L	EC_{10} growth (length), *Brachydanio rerio,* 42-d

Substance	Value	Key study endpoint
Dibenzo[a,h]anthracene	0.14 µg/L	EC_{10} growth, Pseudokirchneriella subcapitata, 72-h

The experimental data indicate a very high acute and chronic toxicity of the PAHs for aquatic organisms. NOEC/EC10 values < 10 µg/L have been observed for the following PAHs: benzo[a]-pyrene, benzo[a]anthracene, benzo[j]fluoranthene, benzo[k]fluoranthene and dibenzo[a,h]anthrax-cene. No toxicity to aquatic organisms has been observed within the water solubility limit of benzo[b]fluoranthene. The same is the case for benzo[e]pyrene and chrysene, with the exception that in one study aimed at determining the mean lethal time (LT50) of Daphnia magna upon exposure to 0.7 µg/L chrysene considerable toxic effects have been observed. This type of study is however not designed to determine dose-response relationships and hence quantitative data on toxicity like EC10 or EC50 values or toxicity threshold values like long-term no effect levels cannot be derived from the results. All eight PAHs in this dossier are categorised as carcinogenic (category 2 / Carc. 1B), BaP and chrysene as mutagenic (category 2 /Muta 1B and 3 / Muta. 2, respectively) and furthermore BaP as toxic to reproduction (category 2 /Repr. 1B) according to DSD/CLP. Therefore the T criterion is nevertheless met for all eight substances, including benzo[b]fluoranthene, benzo[e]pyrene and chrysene.

B.7.2 Terrestrial compartment

Not relevant for this dossier

B.7.3 Atmospheric compartment

Not relevant for this dossier

B.7.4 Microbiological activity in sewage treatment systems

Not relevant for this dossier

B.7.5 Non compartment specific effects relevant for the food chain (secondary poisoning)

Not relevant for this dossier

B.8 PBT and vPvB assessment

B.8.1 Assessment of PBT/vPvB Properties – Comparison with the Criteria of Annex XIII

In chapters B.4.1, B.4.3, and B.7.1, data for Persistence, potential for Bioaccumulation and aquatic Toxicity were presented. The comparison with the Criteria of Annex XIII of the REACH Regulation is shown in Table 42.

In chapter B.4.1 it was shown, that for benzo[a]pyrene, benzo[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene and dibenzo[a,h]anthracene half life-times in soil larger than 120 days were measured. Therefore the Persistence criterion is met. Since the half life times are also larger than 180 days the criterion “very persistent” is also met. In the field study performed by Wild et al (1991), for example, half life times of several years were found. For benzo[a]anthracene and benzo[j]fluroanthane there is only one study by Bossert et al. (1994) that is no clear degradation study. Nevertheless the results found are similar to those of the other PAHs in question and seem to indicate half-life times that are longer than the trigger values for the P and vP-criterion.

Experimentally obtained BCF values above 5,000 are reported for the following PAH: benzo[a]pyrene, benzo[e]pyrene, benzo[a]anthracene, chrysene, benzo[k]fluoranthene and dibenzo[a,h]anthracene. As the BCF values of the above PAHs substances exceed the B- and vB criteria (measured BCF values in aquatic species > 2000, respectively > 5000), it is concluded that the vB-criterion is fulfilled by all six substances. No experimental data on the bioaccumulation potential of benzo[b]fluoranthene and benzo[j]fluoranthene were available. A final conclusion on the B- or vB-properties of these substances is therefore not possible. However, from a weight of evidence approach on the basis of similarities of the log K_{OW}-values and molecular sizes with other PAHs for which BCFs above the Annex XIII bioaccumulation criteria have been experimentally confirmed, it can be assumed that these compounds will at least fulfill the B criterion (i.e. BCF >2000) as well.

In chapter B.7.2 a summary of the studies concerning aquatic toxicities for the PAHs in question was given. Moreover all the eight PAHs in this dossier are categorised as carcinogenic (category 1 or 2), BaP and CHR as mutagenic (category 2 and 3, respectively) and furthermore BaP as toxic for reproduction (category 2). Therefore the T criterion is met for all eight substances.

Table 42: Comparison of the fulfilment of the criteria of Annex XIII REACH Regulation.

	Criterion fulfilled?				
	P	**vP**	**B**	**vB**	**T**
Benzo[a]pyrene	Yes	Yes	Yes	Yes	Yes
Benzo[e]pyrene	No definitive conclusion possible	No definitive conclusion possible	Yes	Yes	Yes
Benzo[a]anthracene	Yes	Yes	Yes	Yes	Yes
Chrysene	Yes	Yes	Yes	Yes	Yes
Benzo[b]fluoranthene	Yes	Yes	No definitive conclusion possible	No definitive conclusion possible	Yes

	Criterion fulfilled?				
	P	**vP**	**B**	**vB**	**T**
Benzo[j]fluoranthene	No definitive conclusion possible	No definitive conclusion possible	No definitive conclusion possible	No definitive conclusion possible	Yes
Benzo[k]fluoranthene	Yes	Yes	Yes	Yes	Yes
Dibenzo[a,h]anthracene	Yes	Yes	Yes	Yes	Yes

In conclusion the available data shows that the criteria of Annex XIII of the REACH Regulation for substances with PBT and vPvB substances are met for benzo[a]pyrene, benzo[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene and dibenzo[a,h]anthracene. This conclusion was already drawn in the Annex XV dossier on CTPHT (European Chemicals Agency, 2009).

For benzo[e]pyrene, benzo[b]fluoranthene and benzo[j]fluoranthene no definitive conclusion on their PBT/vPvB properties is currently possible, due to lack of data relevant for P/vP and/or B/vB assessment in accordance with Annex XIII of the REACH Regulation. When considering the similarity of these three substances to the other five PAHs, from a weight of evidence approach it seems reasonable that they will also meet the necessary trigger values for the persistence and the bioaccumulation criterion.

B.8.2 Emission Characterisation

Not relevant for this dossier

B.9 Exposure assessment

B.9.1 General discussion on releases and exposure

B.9.1.1 Summary of the existing legal requirements

There are currently no legal requirements limiting the PAH content of consumer articles in general. For mixtures, a specific concentration limit of 0.01 % for BaP is stipulated in Regulation (EC) 1272/2008 with regard to classification & labelling for carcinogenicity.

With respect to toys, Directive 2009/48/EC, the EU Toy Safety Directive, states under Annex II 3.3 that:

> *'3. […]substances that are classified as carcinogenic, mutagenic or toxic for reproduction (CMR) of category 1A, 1B or 2 under Regulation (EC) No 1272/2008 shall not be used in toys, in components of toys or in micro-structurally distinct parts of toys.'*

However, this strong rule is contrasted by several exemptions under Annex II 3.4, in particular in bullet (a):

> *4. By way of derogation from point 3, substances or mixtures classified as CMR of the categories laid down in Section 3 of Appendix B may be used in toys, in components of toys or micro-structurally distinct parts of toys provided that one or more of the following conditions is met:*
>
> *(a) these substances and mixtures are contained in individual concentrations equal to or smaller than the relevant concentrations established in the Community legal acts referred to in Section 2 of Appendix B [comment: this refers to CLP and to Dir. 1999/45/EC, respectively] for the classification of mixtures containing these substances;[…]'*

B.9.1.2 Summary of the effectiveness of the implemented operational conditions and risk management measures

For consumer products, neither RMMs specifically aiming at a reduction of consumer PAH exposure, nor OCs fostering such a reduction could be identified.

B.9.2 Manufacturing

B.9.2.1 Occupational exposure

Not relevant for this dossier which focuses on consumer products. The IARC (2006) concluded, that occupational PAH exposure associated with the following industrial processes belongs to classification Group 1 ('carcinogenic to humans'): coal gasification, coke production, coal-tar distillation, work as chimney sweep, paving and roofing with coal-tar pitch, aluminium production.

In the same publication, occupational exposure to PAHs during carbon electrode manufacture has been rated as 'probably carcinogenic to humans' (Group 2A) while exposure during calcium carbide production was 'not classifiable as to its carcinogenicity to humans' (Group 3).

B.9.2.2 Environmental release

Not relevant for this dossier

B.9.2.3 General information

None

B.9.3 Exposure estimation

B.9.3.1 Workers exposure

Cf. section B.9.2.1

B.9.3.2 Consumer exposure

B.9.3.2.1 Chemical analysis of consumer products for BaP and PAHs

Data on the content of PAHs of more than 5300 samples were available. In almost all cases the concentration of the 16 EPA-PAHs were analysed. The list of EPA-PAHs includes the following compounds: acenaphthene, acenaphthylene, anthracene, benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[ghi]perylene, benzo[k]fluoranthene, chrysene, dibenzo[ah]anthracene, fluoranthene, fluorene, indeno[1,2,3-cd]pyrene, naphthaline, phenanthrene, and pyrene.

But, data for the compounds benzo[e]pyrene and benzo[j]fluoranthene, which are legally classified as carcinogens and included in this restriction proposal are not available. Both substances are not included in the list of 16 EPA-PAHs. For further data evaluation additionally the sum of the legally classified carcinogens benzo[a]pyrene, benz[a]anthracene, dibenz[ah]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, and chrysene was quantified as a surrogate for the sum of the 8 legally classified PAHs ('EU-PAHs'). In the dossier the sum of these 6 PAHs is abbreviated as PAH-6. In those cases, where only the concentrations of BaP and the sum of EPA-PAHs were reported, the content of PAH-6 could not be calculated.

No information about the analytical methods used was given by the data suppliers. But at least in Germany a harmonised method is generally used by the accredited laboratories for quantifying EPA-PAHs in elastomers and other plastic materials (ZEK 01.2-08, cf. Appendix 8). Using this method, articles were analysed for testing whether they fulfil the criteria of the German ‘GS’ (proofed safety) mark concerning the PAHs. After extraction of the material with toluene in an ultrasonic bath for 1 hour at 60 °C, PAHs are quantified by gaschromatography with MS-detection (GC-MSD). The default limit of quantification (LOQ) defined in the method is 0.2 mg/kg for every single PAH. Well-experienced laboratories reported that a lower LOQ of 0.1 mg/kg bw/d is achievable in routine analysis. From an analytical point of view the results, of the different suppliers can be combined for further assessment.

The samples analysed cover different kinds of consumer products. Depending on the occurrence of skin contact they have been divided in the following 8 categories:

1. Electrical devices such as cable insulations, plugs, chinch-plugs, covers of domestic electrical appliances or compressors, printers, switches, adapters, remote controls, handheld electrical tools, rubber sleeves on electrical devices, cable sheathings,

2. Grips: grips/handlea made of rubber or plastic, e.g. of tools (hammer, screwdriver, knives, pliers) bicycles, gardening tools, buggies, stationary bicycles, walking frames, torches, handle coatings

3. Skin contact areas of sports equipment or other consumer products: mostly grips and handles of sports equipment, e. g. a dumb-bell set,

4. Toys: balls, figures, toy cars, run bikes, coloured pencils, shuttlecocks, toy guns, electrical parts of toy train set

5. Materials with close contact to the body: shoes, gloves, underwear, working clothes, flip-flops,

6. Other products with skin contact: e.g. ski goggles, headphones, pulse monitors, eye-cups, desk pads, bracelets, steering wheel covers, computer mice, mouse pads, furniture handles, watch straps,

7. Tyres and rolls: e.g. car tyres, tyre cover, transport wheel, rolls of transport aid,

8. Other products: O-ring-seals, seals for sanitary devices, doormats, door stopper, plastic hook coating, measuring tape, rubber hems, foam, rubber band, bulb horn, rubber lips.

A detailed summary of the results from the statistical evaluation of all analytical data as well as of the separated categories is given in Table 65 in Appendix 3.

The highest concentrations of all data are 1200 mg/kg for BaP, 25400 mg/kg for 16 EPA-PAHs and 6930 mg/kg for EU 8. In 91.8 % of all samples BaP was not detectable and in 95 % of all samples the concentration was below 1 mg/kg. EPA-PAHs were not detectable in 22 % of all samples. The sum of the concentrations of all 16 EPA-PAHs below 1 mg/kg was quantified in 50 % and a concentration below 10 mg/kg in 85 % of the samples. In comparison the corresponding values for PAH-6 are 83.9 % not detectable, 90.7 % of the values below 1 mg/kg and 94.8 % of the values below 10 mg/kg.

A short overview is given in Table 43.

Table 43: Overview on the analytical data, maximum levels (mg/kg) and fractions of samples (%) with no detectable level, with a level below 1 mg/kg and with a level above 10 mg/kg

Category	Analytes	Max content (mg/kg)	Fraction of samples (%)		
			n. d. [1]	< 1 mg/kg	> 10 mg/kg
All data (n = 5278)	BaP	1200	91.9	95.3	2.8
	EPA-PAHs	25400	22	50	14.9
	PAH-6	6930	83.9	90.7	5.2
Electrical devices (n = 1705)	BaP	195	91.9	94.8	3
	EPA-PAHs	4516	16.2	46	17.2
	PAH-6	1915	87.8	92	5.3

Category	Analytes	Max content (mg/kg)	Fraction of samples (%)		
			n. d. [1]	< 1 mg/kg	> 10 mg/kg
Grips, handles (n = 541)	BaP	98	90.4	92.8	5.6
	EPA-PAHs	3699	20.7	46.6	18.5
	PAH-6	2483	81.7	89.3	7.5
Contact areas of sports equipment and other articles (n = 120)	BaP	129	87.5	90	5.8
	EPA-PAHs	1801	5.8	24	22.3
	PAH-6	995	73.1	83.3	9.3
Toys (n = 340)	BaP	65.9	94.7	97.1	0.9
	EPA-PAHs	1992	18.5	50.6	9.7
	PAH-6	447	87.8	94.3	3.7
Materials with close contact to the body (n = 535)	BaP	111	88.2	97.6	1.3
	EPA-PAHs	1503	18.1	37.8	19.7
	PAH-6	412	61.4	79.6	4.7
Other products with skin contact (n = 460)	BaP	530	94.8	96.5	2.6
	EPA-PAHs	9300	23.3	60.4	8.1
	PAH-6	3380	90.8	94.1	4
Tyres, rolls (n = 35)	BaP	1200	60	65.7	22.9
	EPA-PAHs	25400	2.9	20	42.9
	PAH-6	6930	45.7	60	34.4
Other products (n = 1519)	BaP	380	93.2	96.1	1.9
	EPA-PAHs	9574	32.5	59.1	11.4
	PAH-6	1994	85.7	92.5	4

[1] 'not detected'; the exact LOD might have varied between the different laboratories whose results were included in the present evaluation. However, with some certainty it can be assumed that the LOQ was always 0.2 mg PAH/kg sample, or lower.

The highest concentrations of BaP, EPA-PAHs and of PAH-6 are reported in the category tyres and rolls. This is valid not only for the maximum values, but for the whole category as demonstrated by the frequency distribution. Even though there might be only a few events with direct skin contact, e.g. changing the car tyres two times a year, these products may contribute to the exposure as well as to the contamination of the environment.

In toys, BaP was not detected in the majority of samples analysed (ca. 95 %). Nevertheless, also some very high BaP values were found in samples from this group. In addition, a high fraction (50 %) of toys contained a sum concentration of EPA-PAHs > 1 mg/kg.

Concerning BaP as a lead substance, < 10 % of samples from all product categories contained comcentrations > 1 mg BaP/kg (with the exeception of tyres/rolls). Taking into account the EPA-PAHs, significantly higher percentages of samples revealed concentrations > 1 mg EPA-PAHs/kg sample. These findings support the view that it is generally preferable to restrict the whole group of carcinogenic PAHs rather than BaP alone.

These data demonstrate that in every product category there are some samples of unacceptable high levels of BaP, EPA-PAHs and of PAH-6. Therefore regulations should include all products.

B.9.3.2.2 Estimation of migration rates for BaP and PAHs

B.9.3.2.2.1 Release into fine dust particles (in air) and house dust

Air concentrations

PAHs have been found in fine dust particles in air exhibiting a great range of concentrations. Low values of 10 to 35 pg/m³ for BaP and 500 pg/m³ for PAH have been found in Sweden (Johannesson et al., 2008; Gustavson et al., 2008). Ohura et al. (2005) described the fluctuation of PAHs in indoor rooms between 0.1 and 1.4 ng/m³. In Greek houses maximum concentrations of 4 ng/m³ were found. Taking into account an inhalation rate of 20 m³ per day, a rough estimate of inhalation exposure to PAHs from fine house dust particles in air of 0.2 up to 60 ng per day can be estimated.

House dust

Lewis et al (1999) detected PAHs in house dust in dust fractions ranging from < 4 to 500 µm diameter. PAHs were detected in one or more size fractionated samples. The concentrations reported ranged from 0.02 to 22 pg/g. From these results a raw estimate of PAH intake of 0.02 pg/kg bw/d can be estimated by using the dust intake standard value of 110 mg per day (AUH, 1995).

B.9.3.2.2.2 Data on migration into aqueous media/ static design

For investigation of PAH migration out of articles into aqueous media such as artificial sweat simulant, artificial saliva and water were used as migration media in a static study design. Dynamic effects during the dermal contact with the product were not incorporated in the study design.

Stiftung Warentest conducted studies on the migration of EPA-PAHs into water as sweat simulant using the grip of a window wiper with a PAH content of 10707 mg/kg (BfR, 2009). The released amount of PAHs in 120 minutes related to the surface area was 33.97 µg/dm^2.

Assuming a weight of 100 g and a surface of 1 dm^2 for the window grip, a migration rate per hour of 0.0015 % (1.5×10^{-5}) for the PAHs can be calculated under these test conditions.

The 'Wirtschaftsverband der deutschen Kautschukindustrie' (WDK), an association of the German cautchouk industry, investigated the migration of BaP and of 8 PAHs regulated in the tyre directive (Dir. 2005/69/EC) from an elastomer containing plasticiser oil and an elastomer containing carbon black as PAH sources into aqueous sweat simulant (40°C, 2 hours) (WDK, 2007). By comparing the concentrations of BaP and the PAHs in both elastomers with the concentrations in the aqueous sweat simulants, migration rates of 0.001 % (10^{-5}) to 0.01 % (10^{-4}) for both BaP and the 8 EU-PAHs regulated by the Directive on Polycyclic Aromatic Hydrocarbons in Extender Oils and Tyres (Dir. 2005/69/EC) were estimated. This migration rates are similar to the rates from Stiftung Warentest using aqueous sweat stimulant.

Hamm et al. published results on the migration of PAHs from cured rubber materials containing carbon black as reinforcing agent into aqueous media (Hamm et al., 2009). They produced seven rubber materials containing different but defined amounts of carbon black. Four of these additionally contained 10 % extender oil, resulting in concentrations of BaP between < 0.1 and

10.8 mg/kg. Concentrations of the 8 EU PAHs regulated in the tyres directive (Dir. 2005/69/EC), varied between < LOQ and 23.6 mg/kg and that of EPA-PAHs between 0.9 and 367 mg/kg. No migration of BaP and of the EU-PAHs was detectable from these artificial samples after a migration time of 7 days when using drinking water, rain water, artificial sweat and artificial saliva as aqueous simulants. Although no migration of BaP or EU-PAHs were detectable, the migration of EPA-PAHs into aqueous simulants after 7 days varied between 0.0002 and 0.001 mg/dm^2 corresponding to a migrated fraction of between 0.0002 % ($2x10^{-6}$) and 0.001 % (10^{-5}).

B.9.3.2.2.3 Data on migration into lipophilic solvents/ static design

Taking into account the lipophilicity of PAHs, in two studies isooctane and 95 % ethanol instead of aqeous simulants have been used for migration investigations. Both solvents are usually used as simulants for fatty food (EN 1186-14, 2002).

Stiftung Warentest conducted a study on the migration of PAHs into isooctane, using a window wiper grip with a PAH content of 10707 mg/kg (BfR, 2009). The amounts released into isooctane were determined for the 16 EPA-PAHs including BaP. The quantified area-related migration during 2 hours was 131.014 µg/dm^2 for PAHs and 5.56 mg/dm^2 for the individual compound BaP. If one assumes for the handle a mass of 100 g and a surface of 1 dm^2, approximately 6.1 % of the PAHs migrated per hour. These data are distinctly higher than the migration quantified for the same article by using water as stimulant.

Isooctane as well as 95 % ethanol have been used for migration investigations on beach sandals made of soft PVC containing 8.7 mg/kg BaP and 546 mg/kg PAHs (sum of 16 EPA-PAHs) (UBA, 2010) The migration rates have been estimated by the following approach: the area being in contact with the foot was wetted with 100 mL simulant for 24 h at a temperature of 20 °C. The migration resulted in a BaP concentration of 0.2 and 0.3 mg/kg and in a concentration of the sum of EPA-PAH of 23.7 and 44.9 mg/kg in the simulants 95 % ethanol and isooctane, respectively. For a contact time of one hour, migration rates related to the concentration of BaP in the product were calculated to be 0.16 % when using ethanol and 0.24 % with isooctane. The corresponding migration rates for the sum of EPA-PAH are 0.30 % and 0.57 %. In addition, migration rates have been reported based on contact areas, which were 0.035 mg/dm^2 and 0.055 mg/dm^2 for BaP and 4.57 mg/dm^2 and 8.31 mg/dm^2 for the sum of EPA-PAH in the simulants 95 % ethanol and isooctane, respectively. Migration into isooctane is about 50 % higher than into ethanol.

The detailed data of the studies using aqueous or organic solvents as simulants and the static migration design are summarised in Table 66 (Appendix 4).

B.9.3.2.2.4 Data on migration tests with latex gloves/ dynamic design

A different experimental approach for estimating the migration of PAHs by dermal contact was chosen in some further migration studies. For simulating the skin a latex glove was used as surrogate. The PAH-containing article was held by hand, which was covered by a latex glove and wetted by aqueous sweat simulant. Simulated contact conditions also included the dynamic and mechanical effects under use conditions. The migrated amount of BaP was quantified after extracting the glove. In all experiments, artificial acidic sweat simulant corresponding to EN ISO 105-E04 (2009) was used. For determination of BaP and EPA-PAHs in the products or extracts the method ZEK 01.2-08 (2008, cf. Appendix 8) was used. The detailed data and results of the described studies are summarised in Table 67 (Appendix 4).

For estimating the fraction of BaP migration per hour to the glove, the following equation was applied.

$$Fc_{migr}[\%] = \frac{M_{g/h}}{Fc_{prod} * Q_{prod} * F_{cont}} * 100$$

The parameters of this equation are defined as follows:

Fc_{migr} = rate (fraction) of substance migrating to skin per unit time (kg substance migrated/kg substance contained in article/h)

$F_{contact}$ = fraction of contact area for skin, to account for the fact that the product is only partially in contact with the skin

Fc_{prod} = weight fraction of substance in product (mg substance/kg product)

$M_{g/h}$ = amount of substance migrated into glove per hour (µg/h)

Q_{prod} = amount of product used (g)

$T_{contact}$ = contact duration between article and skin

TÜV Rheinland provided data on the migration of PAHs from a rubber grip of a hammer into a latex glove which covered the hand of a man holding and moving the hammer. The glove was wetted every 10 minutes by aqueous sweat simulant during a total contact time of one hour. The mechanics of the use conditions such as strongly holding, moving, and fractioning of the grip were simulated (Sander, 2006). The concentration of BaP in this rubber grip was 42 mg/kg. After this procedure, the amounts of BaP and of the EPA-PAHs in the glove were quantified. By comparing the content of PAHs and BaP in the tool grip with the amount quantified in the glove, a migration rate of 9.7 % per hour for BaP was estimated.

The same test design was also used for migration experiments with further products such as a cover of a steering wheel made of rubber and containing high amounts of 35 mg BaP/kg (TÜV Rheinland, 2010). The contact scenario was designed similar to realistic use conditions, wetting the glove with sweat simulant every 10 minutes. During the contact time of one hour the hand was not fixed but moved around the whole surface of the steering wheel cover. Consequently, it has to be realized that under these conditions the total surface of the steering wheel cover was not permanently in direct contact with the glove during the total time. Therefore, the fraction of migration was calculated for two different cases. First a fixed contact area of the glove with the steering wheel cover over 1 hour was assumed and the migrated fraction was based on the contact area of a hand resulting in a migrated fraction of 10.2 % per hour. Then assuming the whole surface of the steering wheel as contact area for 1 hour resulted in a migrated fraction of 0.95 %. While the first arrangement leads to an overestimation, the second outcome is a clear underestimation of the migrated fraction. Probably, the real situation would be reflected by a migration fraction between these both values.

The migration of BaP from a bulb of a hooter presumably made of synthetic butyl rubber and containing 530 mg/kg BaP was tested by the same design. A contact time of 1 hour with the latex

glove wetted with aqueous sweat simulant, holding and pressing the bulb of the hooter resulted in a comparably low migration rate of 0.02 % per hour.

Hutzler (2009) used a similar design simulating dermal contact by a latex glove wetted with aqueous sweat simulant. A torch handle with a cover and angles of PAH containing rubber was held and fractioned strongly with the glove for a shorter contact time of 1 minute. In addition, a measuring tape was examined, the case and angles of which were made of rubber. No information on the material of the torch is available. The concentrations of BaP in these products were 11.8 mg/kg in the measuring tape and 12.8 mg/kg in the torch handle. For extrapolation of the migration rate for 1 minute to a 1 hour-period, a linear correlation between migration and time was assumed. For every experiment the skin contact area was detected by fluorescence of the PAHs on the glove and quantified. Calculated migration rates for BaP were 5.4 % per hour for the measuring tape and 1.8 % per hour for the torch handle.

This experiment was repeated with the same products and a 10 minute-contact time, but without fractioning. A linear correlation between migration rate and contact time was assumed for extrapolating migration to a 1 hour-period. Under these conditions the calculated migration rates for BaP were 0.15 % per hour for the torch handle and 0.23 % per hour for the measuring tape.

B.9.3.2.2.5 Discussion of migration results

Different experimental approaches for the determination of the PAH migration rates have been performed and different materials have been included. Summarising all available data on migration rates estimated in different study designs, there is a large difference of several orders of magnitude. The migration rate depends on several factors which are discussed in the following.

Under static conditions either aqueous migration media or lipohilic solvents such as isooctane and 95 % ethanol have been used. The results demonstrate, that migration rates are strongly influenced by the simulant used.

The lowest migration rates below 0.001 % to 0.01 % have been observed using aqueous sweat simulant under static conditions. But in the case of highly lipohilic PAHs, lipophilic sweat components and skin fat, are assumed to increase migration. Aqueous simulants are therefore not considered to adequately reflect the real-life situation, as the migration rates estimated presumably underestimate the real migration of PAHs from articles in contact with skin.

PAHs are highly lipophilic substances. It has been demonstrated by experiments of TÜV Rheinland, that skin fat and organic or fatty constituents of the sweat may enlarge the migration of PAHs. Wennemer detected PAHs on skin by its fluoresecence under UV light after a very short contact of a hand creamed with moisturising creme with a tool grip containing high amounts of PAHs (Wennemer, 2009; Sander, 2006). The relevance of skin fat on dermal exposure has also been demonstrated by human biomonitoring results of Angerer et al. (2007). After use of fatty skin protection cream, PAH-exposed workers showed a higher excretion of PAH metabolites in urine than workers who did not use skin protection cream. Consequently, the influence of lipophilic sweat components and skin fat on PAH migration (and possibly penetration) by dermal contact are not negligible and have to be considered in a realistic way by suitable migration conditions in an experimental design. It has to be considered that skin fat and organic or fatty constituents of the sweat increase the migration of the lipohilic PAHs.

Taking into account the lipohilicity of PAHs, in two studies isooctane and 95 % ethanol have been used for migration investigations instead of aqueous simulants. Both solvents are usually used as simulants for fatty food (EN 1186-14, 2002) and should better reflect the influence of skin fat and of other organic or fatty sweat constituents on the migration of PAHs by dermal contact.

The results of Stiftung Warentest, who investigated the same article using both water and isooctane, clearly demonstrate that the migration rates estimated for the lipohilic PAHs strongly depend on the simulant used in the experimental design. Consequently, the migration rate of EPA-PAHs increased by more than four orders of magnitude from 0.0015 % using aqueous simulant to 6.1 % using the highly lipophilic solvent isooctane. In comparison to isooctane the migration rates into ethanol were slightly lower as demonstrated by the study on beach sandals.

Ethanol and particularly isooctane are highly lipohilic solvents which are not inert. They may change the outer layer of polymer and elastomer products by swelling and influence the migration rate distinctly. This may increase migration, but also other processes such as dissolving may contribute to the release of the analyte. It is therefore questionable whether the solvents ethanol and isooctane are suitable for reflecting the skin surface and the composition of sweat by aqueous and fatty components. Presumably the use of these highly lipohilic solvents may result in an overestimation of PAH migration rates.

Furthermore, as a general problem of the static study design, in which the product is enclosed by the simulant, dynamic processes are not reflected. Dynamic processes like moving and fractioning the hand on the surface or a strong skin contact pressure are relevant under use conditions and influence the migration rate.

A more realistic scenario, which better reflects the dynamic aspects, is the use of latex gloves wetted with sweat simulant and simulating the use conditions. Different from the lipohilic solvents the latex gloves have a lipophilic character, but are chemically inert. They do not affect the surface of the product. Furthermore, flexible contact and the contact pressure between the surface and the skin can be simulated.

The migration rates of BaP estimated by the contact of latex gloves wetted with sweat simulant under use conditions had values between 1 and 10 % for rubber materials. For synthetic butyl cautchouc (bulb of a hooter) with the highest BaP concentration the lowest migration rate of 0.02 % has been estimated. No correlation between the concentration of BaP and the migration rate can be derived. Migration mainly depends on the composition of the materials. This is well known and generally accepted for food contact materials. Not only the type of polymer or elastomer but also the type and amount of additives, plasticisers, and further components influence the diffusion coefficient in the material and, consequently, migration. The large variability of the migration rates estimated for BaP can be explained by a different composition of the materials investigated. Also on the market a large variability of materials used for consumer products is available. Therefore it is assumed that the spectrum of estimated migration rates may reflect the situation of the consumer products on the market.

Aside from the material itself, also mechanical effects like friction have a strong influence on the resulting migration rate. This has been demonstrated by Hutzler (2009). In the case of the torch handle and the measuring tape migration rates of BaP decreased without friction by a factor of 10, which might be explained by additional abrasion and adhesion of particles on the glove, when friction is present. This effect of abrasion by friction may result in an overestimation of the migration rates. Furthermore, linear extrapolation from shorter contact times in some studies (1 min

and 10 min) to a 1 hour-contact time may lead to a slight overestimation. However, the extent of both aspects can not be appraised exactly.

For the exposure estimation of consumers, migration rates quantified under dynamic conditions are used. In order to cover the different materials on the market and the influence of dynamic conditions on exposure, three different migration rates for BaP are used for the calculation:

- migration rate of 10 % as the worst case of dynamic migration with friction
- migration rate of ca. 1.5 % as the geometric mean of the dynamic migration with friction
- migration rate of 0.2 % as mean of dynamic migration without friction.

B.9.3.2.3 Estimation of dermal exposure for BaP and PAHs

B.9.3.2.3.1 Exposure of children by contact with rubber granula

Study results on PAH migration from crumb rubber into watery media are compatible with the assumption of higher migration rates for PAHs with higher volatility such as naphthalene, pyrene, and fluoranthene. BaP was not detected in most leachates (with some relatively high detection limits), but BaP migration into gastric fluid of 2.95 % in one hour has also been reported.

Nilsson et al (2005) conducted a migration experiment with six tyre samples, which were exposed to artificial sweat for one hour at 30 °C and then washed. The only PAHs detected in the filtered leachate were fluoranthrene (0.029-0.277 ng/cm² from sample concentrations of 2.1 -16.0 mg/kg) and pyrene (0.032-0.487 ng/cm² from sample concentrations of 11.4 - 34.0 mg/kg).

Plesser et al (2004) determined leaching of PAHs from two samples of artificial turf from recycled SBR crumb rubber into deionised water. Leaching procedures in this study were based on pr EN 12457-4 and NT ENVIR 005. The water to solid material ratio L/S was 10 L/kg. The container with water and solid material was rotated at a speed of 10 r.p.m. for 24 hours, the leachate was then filtered (normal pressure, ash-free paper filter) and the filtrates were analysed. Benzo[a]pyrene in the leachate was below the detection limit of 0.01µg/L, PAH(16) were 0.87 µg/L and 0.44 µg/L. Considering the PAH (16) contents of the samples, which were 51 mg/kg and 74 mg/kg, a leached percentage of 0.4 % and 0.3 % per 24 h can be calculated (corresponding to about 0.01 %/h).

Zhang et al (2008) determined bioaccessibility of PAHs from a sample of artificial turf from recycled crumb rubber into artificial saliva (2.5 g sample with 8 ml synthetic saliva, incubation at 37°C with constant shaking at 90 rpm for 15 min). 45.7 % of the naphthalene content was detected in the saliva fluid, but BaP was not detectable (LOD 0.023 ng/mL, BaP content in the sample 0.78 mg/kg). Tests on gastric bioaccessibility (2.5 g sample with 8 ml synthetic saliva and 100 ml synthetic gastric fluid, incubation at 37 °C for 2 h) were performed with the same sample and 6.38 % of the naphthalene content and 2.95 % of the BaP content were determined in the leachate. The same test done with another sample revealed 45.7 % of the naphthalene content in the leachate, but BaP was not detectable (LOD 0.023 ng/ml, BaP content in the sample 0.41 mg/kg). This latter sample was also tested on intestinal bioaccessibility (2.5 g sample with 8 ml synthetic saliva and 100 ml synthetic gastric fluid, incubation at 37 °C for 2 h, addition of 100ml intestinal fluid and incubation at 37 °C for another 2h). 50.9 % of the naphthaline content was detected in the leachate, but BaP remained not detectable.

The Office of Environmental Health Hazard Assessment, OEHHA (2007) conducted a simulated stomach leaching study with three samples of recycled rubber (40 g with 200 mL simulated gastric fluid and paraffin seal, incubation at 37 °C in a rotary shaker for 21 h). PAHs in the filtered leachates were below the (relatively high) reporting limits of 190 or 360 µg/L.

From these experimental data it can be concluded, that PAHs are present in materials such as rubber granules and rubber turf preparations. Also, there is concern that they will be released with different release rates and at potentially considerable amounts.

Rubber mats and granules are used as ground coverings on playgrounds, either in form of granulate or as rubber pavement. These materials may contain PAHs or BaP as measured by different authors (Edeskär, 2006; Hofstra, 2006; Byggforsk, 2004; Zhang 2008). Concentrations of PAHs in rubber granulates ranged between 45 and 76 mg/kg, that of BaP between 0.06 and 8.58 mg/kg.

Preparing an estimate of exposure due to rubber turf (granules) exposure

Preparation of an estimate of exposure should take into account the uncertainties due to the heterogeneity of the results of migration studies finding different PAHs. Therefore, for this dossier a rough estimate has been made which is based on the recommendations of the ECHA consumer exposure guidance document (2010) and the ECETOC TRA guidance document (2009). Also, a second approach has been applied that takes into consideration the release rates derived above.

Children playing on these grounds may be exposed by the dermal route. Two scenarios have been choosen to characterise the exposure:

- Granules may be put under a playing slide or a swing. It may happen that a playing child falls down on this ground (which is normal for slides). The basic estimates for this scenario are as follows: The contact area is 5000 cm², which is half of the body surface of a 6 to 10 year-old child. The frequency of falling down and thus coming into contact 50 times, the duration of each contact, however is less than 30 seconds.

- The second scenario is due to walking or standing on the ground for about two hours. The contact area is represented by the bare feet area of 340 cm² (see footnote).

Estimation by use of the ECETOC TRA approach

A basic and raw estimate has been performed based on the assumptions and algorithms of the ECETOC TRA, which is also part of the ECHA guidance for consumer exposure estimation. Release rates of PAHs as described above have been applied.

In the ECETOC TRA approach, contact times will not be considered. In this scenario, the highest value of PAH content of 76 mg/kg as reported by Hofstra (2006) has been used as well as the standard default values as given in the guidance documents.

The exposure estimate performed by using very raw estimates and a very simple model approach. The intention of taking the proposal of ECHA guidance is to overestimate exposure. For the scenario 1 which describes short contact times with greater parts of the body a value of 25 µg/kg has been estimated. Scenario 2 reveals an estimate of 1.7 µg/kg.

Table 44: Estimation of PAH exposure of consumers from rubber granules using the ECETOC TRA approach

ECETOC TRA / ECHA guidance				
	Slide, swing	**Standing, walking**		
Content	76		mg/kg	
Contact area	5000	340	cm²	
Thickness of layer	0.001	0.001	cm²	
Mass	0.005	0.00034	g	(Density = 1)
100 % release	0.38	0.02584	mg	
Body weight	15			
Exposure	0.0253	0.0017	mg/kg	
	25.3333	1.7227	µg/kg	

Approach which considers the migration of PAHs

To consider the overall uncertainties in this evaluation, a second approach has been applied to estimate exposure from PAHs in rubber granules on e.g. playgrounds. This approach considers the migration data which have been derived above. There are no migration rates available for rubber granules. In these scenarios the 10 % migration rate (worst case estimate) has been considered for calculation. The other rates derived for characterising migration of 1.5 and 0.2 % per hour were used to calculate respective exposures.

The mass of the product that is in contact with skin is a key issue of the estimation. In this scenario, we have a bigger area covered with rubber granules on and in which the child is walking, running standing, or lying. The two scenarios try to point out the limits of this behaviour. The mass of the product may be best characterised by the contact area multiplied with a small layer to build a fictive volume which contents the substance. It is important to note that this construct is different to the thickness of layer approach used in the ECETOC TRA concept where a 100 % release is assumed from the 0.001 cm-layer. Calculated release which is expressed per mass of product is then related to the surface (contact) area. The values displayed in Table 45 are approximately in a similar range as the experimental values referenced above.

In this case, the mass of the product is calculated by the area (in cm) and a layer of 0.1 cm. In addition, the amount released from this layer will be related to the area of contact.

Table 45: Estimation of PAH exposure of consumers from rubber granules using an approach which considers migration

		Scenario 1 (10 % release)	Scenario 2 (10 % release)	
Migration default data as derived in chapter B.9.3.2.2				
Contact area	**A**	5000.00	340.00[1]	cm²
Layer (Expert Judgment)	**L**	0.10	0.10	cm
Mass of product	**M**	500.00	34.00	g
Content BAP (RIVM)	**C**	76.00	76.00	mg/kg
Content available for exposure	**CE**	38.00	2.58	mg
Release rate per hour (derived default)	**RR**	0.1000	0.1000	%
Amount released per hour	**R**	3.80	0.26	mg/h
Time of contact	**TC**	25	120	min
Amount released in time of contact	**RT**	1.58	0.52	µg
Amount released related to area	**RA**	0.32	1.52	µg/cm²
Exposure data due to the scenarios described above, extrapolation of 3,05 migration				
		Swing, slide	**Standing and walking**	
Area of contact scenario	**Ac**	5.000.00	340.00	cm²
Amount of exposure	**E**	1.583.33	516.80	µg
Amount of exposure per body weight 15 kg	**Ebw**	105.56	34.45	µg/kg

The equations used in this scenario are as follows

$$M = A * L$$
$$CE = M * C$$
$$RT = CE * RR / (60 * TC)$$
$$RA = RT / A * 1000$$
$$E = Ac * RA$$

This estimation reveals an exposure ranging from a low estimate of 34 µg per kg of bodyweight up to 105 µg/kg of body weight.

Taking the migration rates of 1.5 and 0.2 %/h into consideration and hold the other parameters constant, respective exposures of 15.75 and 0.19 µg/kg (scenario 1) as well as 5.5 and 0.069 µg/kg can be calculated.

[1] [1] Mean estimate, ref. to AUH. Values on areas of soles of feet are missing, but have been derived from available data for whole feet areas in children (400 cm²; 9 to 10 years, 235 cm²; 3 to 4 years; no further data)

The range(s) estimated by different approaches and taking different migration rate data demonstrate the uncertainties of this evaluation, which are due to the defaults taken, and the model approach, in particular the default for layer thickness and the extrapolation of released amounts to the contact area. In addition, the behaviour data may be criticised as too conservative and unrealistic. It is obvious that the basis for migration data is very heterogenous and inappropriate to prepare a sound exposure estimate. Interestingly, the ECHA / ECETOC TRA approach does **not** calculate estimates that are considerable higher than values calculated otherwise. The appropriateness of the approaches applied here, however, can only by evaluated by the use of appropriate release data which are currently not available.

Taken all together, the results clearly show that rubber products may contain high amounts of PAHs. The knowledge about release of these compounds needs to be improved, but there is evidence that considerable amounts can be released. Those studies should be performed by standardised procedures that cover typical uses of the tools.

B.9.3.2.3.2 Other consumer products

The external dermal exposure by consumer products was estimated in two different ways:

1. The results of the dynamic migration tests described in chapter B.9.3.2.2.4 were used for estimating external exposure to BaP by those products tested under dynamic conditions

2. The external dermal exposure was estimated based on the estimated migration rates and on the available data on BaP concentrations in consumer products.

B.9.3.2.3.3 Exposure estimation based on the migration test with dynamic conditions

Models

In the dynamic test design for estimation of migration rates the amounts of BaP migrated into gloves after contact with the consumer products under use condition were quantified. Based on these values the external dermal doses for the single consumer products which were included in these studies have been estimated.

For the calculation the following equation was used:

$$D_{der} = \frac{M_{g/h} \times T_{contact} \times n}{BW} * 1000$$

The parameters used in this equation are defined as follows:

BW = body weight (kg)

D_{der} = dermal dose (ng/kg bw/d)

$M_{g/h}$ = Amount of BaP migrated into the glove per hour (µg BaP/h)

n = mean number of exposure events per day

$T_{contact}$ = contact duration between article and skin (h)

Scenarios

As the default value for the body weight of adults, 60 kg was used for the calculation. For the necessary parameters contact duration ($T_{contact}$) and number of events per day (n), no default values were available. Therefore the values were estimated for every specific sample based on the experience of users. These values reflect the ordinary behaviour of the consumers and should correspond to a realistic case scenario. They do not reflect a worst case scenario.

Corresponding to REACH IR Guidance R.15 (Chapter 15.2.1.2) for estimation of the chronic external exposure for products used frequently, the exposure was calculated for the actual duration of the event, expressed as dose per day.

Table 46: External dermal dose estimated from the results of dynamic migration with gloves

Sample	Simulated contact conditions		Amount of BaP migrated into glove per hour $M_{g/h}$ (µg/h)	Contact duration per event $T_{contact}$ (h)	Number of events per day n (1/d)	External dermal dose $D_{der\ BaP}$ (ng/kg bw/d)
	Simulant	Mechanics				
Hammer grip	Glove and sweat simulant	Hold and move. strong power	320	1	1	5333.3
Cover of steering wheel		Hold and move	100 [1]	1	2	6666.7
Bulb of a hooter		Hold and move	4	0.017	2	2.3
Torch handle		Hold and friction	22.8	0.17	1	64.6
Measuring tape		Hold and friction	15	0.17	1	42.5
Torch handle		Hold	1.92	0.17	1	5.4
Measuring tape		Hold	1.074	0.17	1	3.0

[1] the exposure was estimated for the contact of two hands with the cover of steering wheel

The dynamic migration data reflect use conditions. The calculated external dermal doses vary between 2.3 and 6667 ng/kg bw/d. The highest doses were estimated for skin contact with a tool grip and a cover of a steering wheel both made of rubber. The contact conditions between the product and the skin, e.g. the intensity of pressure, movement and friction were very powerful during the experiment, reflecting realistic worst case conditions. But for some users with less power an overestimation of external dermal dose might be possible.

B.9.3.2.3.4 Exposure estimation based on the estimated migration rates

Models

For estimation of the external dermal dose to be expected from exposure towards the products for which the results of chemical analysis were given in Appendix 4, equation 15-8 on page 24 of the REACH IR Guidance R.15 was used (for practical use, it was combined with the equation for calculating the external dermal dose given on page 25 of the same document):

$$D_{der} = \frac{Q_{prod} \times Fc_{prod} \times Fc_{migr} \times F_{contact} \times T_{contact} \times n}{BW} * 1000$$

The parameters used in this equation are defined as follows:

BW	=	body weight (kg)
D_{der}	=	dermal dose (ng/kg bw/d)
Fc_{migr}	=	rate (fraction) of substance migrating to skin per unit time (µg substance migrated/µg substance contained in article/h)
$F_{contact}$	=	fraction of contact area for skin, to account for the fact that the product is only partially in contact with the skin
Fc_{prod}	=	weight fraction of substance in product (mg substance/kg product)
n	=	mean number of exposure events per day
Q_{prod}	=	amount of product used (g)
$T_{contact}$	=	contact duration between article and skin per event (h)

Corresponding to REACH IR Guidance R.15 (Chapter 15.2.1.2) for estimation of the chronic external exposure for products used frequently the exposure was calculated for the actual duration of the event and expressed as dose per day.

As described in chapter B.9.3.2.2 the calculation of the exposure of consumers to BaP by articles three different values for the migrating fraction has been used. These values should reflect the large variability of the migrating fractions due to different materials of the products and the considerable influence of the dynamic conditions on the release by dermal contact. No information on the materials of the products analysed for PAH concentrations are available. So by using 3 values of migration fractions the results should characterize the range of exposure.

Scenarios

For the estimation of the external dermal dose products with foreseeable skin contact and with measured concentrations of BaP were selected. Separate exposure estimations were made for adults and for children.

For adults the following product groups were included:

- Grips of different handheld tools or bicycles.
- Skin contact areas of sports equipment or other consumer products
- Footwear
- Protective gloves
- Cover of a steering wheel
- Watch strap

For children, the following consumer products were selected for the estimation of the external dermal dose:

- Grips of bicycles
- Skin contact areas of sports equipment or other consumer products

- Footwear (sandals)
- Rubber boots for children
- Watch strap
- Toys

The scenarios and assumptions for the estimation of the external dermal dose are described subsequently.

Grips of different handheld tools or of bicycles

In most cases information on the type of grip (e.g. tool, bicycle and knife) were available. In some cases this information was missing. Theses samples were assumed as grips for bicycles.

For grips of bicycles, trolley bags and walking frames the contact with two hands and in all other cases a one hand contact were assumed. The masses of some tool grips were reported by the laboratory and used for all other tool grips. Masses of grips of bicycles for adults and for children were based on information on web sites of bicycle shops. For children, bicycle grips have a lower mass. The fraction of contact area for skin for adults and for children was estimated by own observations. No default values for describing the contact conditions under use, the contact time, and the number of contact events per day were available. Therefore for the parameters of contact time and the number of contact events per day assumptions based on user experience were made and should reflect conditions under use.

Skin contact areas of sports equipment or other consumer products

No detailed information on the type of products was reported. It was clarified, that in most cases grips of tools as well as of sports equipments, such as bicycles, were analysed. Therefore as a standard scenario all samples of this product type were handled as grips of bicycles, reflecting a higher dermal dose because of the two hand-contact. The same parameters were used as described before.

Footwear

Several data were available for footwear or parts of footwear made of plastics or material combinations. Further detailed information was not reported. For the exposure scenario it has been assumed, that beach sandals have been analysed and used as slippers daily at home. The masses of beach sandals for adults and for children were determined by weighing. The direct foot contact area with the upper side of the sandals was estimated by own observations. A common use of one hour twice daily should reflect user behaviour for adults as well as for children, even thought this might underestimate daily contact time in some cases.

Protective gloves

The mass of a pair of protective gloves was weighed. The contact time per event day is based on user experience but may deviate to lower as well as to higher values.

Cover of a steering wheel

Data on the mass and surface of the cover of a steering wheel have been reported by the laboratory. Generally two hands are in contact with the steering wheel. The contact time is based on user experience but may deviate to lower as well as to higher values.

Watch strap

The mass of a watch strap made of plastics was weighed and the daily contact time estimated by use experience.

Rubber boots for children

A pair of rubber boots was weighed and the mass for the lower foot part was estimated. It was assumed, that the foot of the child is in direct contact with these lower foot parts of the boots, and that during a rainy day, children are using rubber boots for about 2 hours.

Toys

Most samples with measured BaP concentrations were tyres of toy cars or of training bikes, a few samples were outdoor toys. Only a list of examples for outdoor toys such as a shovel or watering can made of plastics for playing in the sand or swings or a frisbee was reported without any further detailed information. In these cases the article was assumed to be a grip of a shovel or watering can.

A contact time of 10 minutes was assumed for the tyres of car toys or of the training bike (inflate the tyres) and of 0.5 hour for the outdoor toys.

As default values for the body weight of adults 60 kg and for the body weight of a 5 year old child 20 kg were used for the calculation.

All detailed parameters used for the estimation of external dermal dose as well as the results for the three different migration rates are summarised in Appendix 5 in Table 68 for adults and in Table 69 for children.

The results indicate that in every product group included in this evaluation very high dermal doses were estimated for adults and for children, even in the case when the lowest migration rate was used.

In comparison the dermal doses for these product groups using the same scenarios were estimated assuming a hypothetical BaP concentration of 0.2 and of 1 mg/kg and using the three values for migration rates. An overview on the results is given in Table 47 for adults and in
Table 48 for children.

Depending on the fraction of migration used for the calculation and on the products the lowest obtained value for dermal exposure of adults is 0.5 ng/kg bw/d and the highest 150 ng/kg bw/d based on the BaP content of 0.2 mg/kg. For children the lowest value for dermal exposure is 0.1 ng/kg bw/d and the highest 300 ng/kg bw/d based on the BaP content of 0.2 mg/kg.

Table 47: Comparison of dermal exposure to BaP at current contamination levels with hypothetical limit values for the same products and scenarios for adults

Category	Sample	Number of measured values included	Type	Concentration in product Fc_{prod} BaP (mg/kg)	Q_{prod} (g)	$F_{contact}$	$T_{contact}$ (h)	n (1/d)	$D_{der\ BaP}$		
									Fc_{migr} (%/h) = 10	Fc_{migr} (%/h) = 1.5	Fc_{migr} (%/h) = 0.2
									(ng/kg bw/d)	(ng/kg bw/d)	(ng/kg bw/d)
Grip	Tool	27	Measured values	0.1-98	100	0.8	1	1	13-13067	2-1960	0.3-261
			Hypothetical limit values	0.2					26.67	4	0.53
				1					133.33	20	2.67
Grip black	Assumed as bicycle grip	17	Measured values	0.2-94	200	0.8	1	2	106-50133	16-7520	2-1002
			Hypothetical limit values	0.2					106.67	16	2.13
				1					533.33	80	10.67
Skin contact areas of sports equipment or other consumer products	Assumed as grip	15	Measured values	0.2-128	200	0.8	1	2	106-68613	16-10292	2-1372
			Hypothetical limit values	0.2					106.67	16	2.13
				1					533.33	80	10.67
Footwear (shoes, boots, sandals)	Assumed as sandals used as slipper	36	Measured values	0.01-111	200	0.4	2	1	3-29680	0.4-4452	0.05-593
			Hypothetical limit values	0.2					53.33	8	1.07
				1					266.67	40	5.33
Protective gloves		12	Measured values	0.001-0.5	100	0.9	1	2	0.3-150	0.05-22.5	0.01-3
			Hypothetical limit values	0.2					60	9	1.2
				1					300	45	6

Category	**Sample**	**Number of measured values included**	**Type**	**Concentration in product Fc $_{prod}$ BaP (mg/kg)**	**Q_{prod} (g)**	**$F_{contact}$**	**$T_{contact}$ (h)**	**n (1/d)**	**D $_{der\ BaP}$**		
									Fc $_{migr}$ (%/h) = 10	**Fc $_{migr}$ (%/h) = 1.5**	**Fc $_{migr}$ (%/h) = 0.2**
									(ng/kg bw/d)	**(ng/kg bw/d)**	**(ng/kg bw/d)**
Cover of a steering wheel		3	Measured values	14-35	300	0.2	1	2	2800-7000	420-1050	56-140
			Hypothetical limit values	0.2					40	6	0.8
				1					200	30	4
Watch strap		2	Measured values	0.3-43	30	1	15	1	202-32250	30-4837	4-645
			Hypothetical limit values	0.2					150	22.5	3
				1					750	112.5	15

Table 48: Comparison of dermal exposure to BaP at current contamination levels with hypothetical limit values for the same products and scenarios for children

Category	Sample	Number of measured values included	Type	Concentration in product Fc $_{prod}$ BaP (mg/kg)	Q_{prod} (g)	$F_{contact}$	$T_{contact}$ (h)	n (1/d)	D $_{der\ BaP}$ (ng/kg bw/d)		
									Fc $_{migr}$ (%/h) = 10	Fc $_{migr}$ (%/h) = 1.5	Fc $_{migr}$ (%/h) = 0.2
Grip black	Assumed as bicycle grip	27	Measured values	0.2-94	100	0.6	1	1	60-28200	9-4230	1.2-564
			Hypothetical limit values	0.2					60	9	1.2
				1					300	45	6
Skin contact areas of sports equipment or other consumer products	Assumed as bicycle grip	15	Measured values	0.2-128	100	0.6	1	1	60-38595	9-5789	1.2-772
			Hypothetical limit values	0.2					60	9	1,2
				1					300	45	6
Footwear (shoes, boots, sandals)	Assumed as sandals used as slipper	36	Measured values	0.01-111	150	0.4	1	2	6-66780	0.9-10017	0.1-1336
			Hypothetical limit values	0.2					120	18	2.4
				1					600	90	12
Rubber boots	For children	11	Measured values	0.04-1.4	200	0.80	2	1	62-2272	9-341	1.2-45
			Hypothetical limit values	0.2					320	48	6.4
				1					1600	240	32

Category	Sample	Number of measured values included	Type	Concentration in product Fc_{prod} BaP (mg/kg)	Q_{prod} (g)	$F_{contact}$	$T_{contact}$ (h)	n (1/d)	$D_{der\ BaP}$ (ng/kg bw/d)		
									Fc_{migr} (%/h) = 10	Fc_{migr} (%/h) = 1.5	Fc_{migr} (%/h) = 0.2
Watch strap		2	Measured values	0.3-43	20	1	10	1	270-43000	40-6450	5-860
			Hypothetical limit values	0.2					200	30	4
				1					1000	150	20
Toy car	Tyre	3	Measured values	0.2-66	30	0.60	0.17	1	3-1008	0.5-151	0.1-20.2
			Hypothetical limit values	0.2					3.1	0.5	0.1
				1					15.3	2.3	0.3
Training bike	Tyre	8	Measured values	1-34	100	0.10	0.17	1	6.3-283	1-42.5	0.1-5.7
			Hypothetical limit values	0.2					1.7	0.3	0.03
				1					8.5	1.3	0.2
Outdoor toys	Grip of a shovel or watering can	2	Measured values	0.07-0.4	50	0.6	0.5	1	5.3-30.2	0.8-4.5	0.1-0.6
			Hypothetical limit values	0.2					15	2.3	0.3
				1					75	11.3	1.5

B.9.3.2.3.5 Estimation of oral exposure for BaP and PAHs

It is noted that, in principle, consumer exposure to PAH-contaminated articles may well occur along all three major exposure routes, i. e. via dermal or oral uptake, or by inhalation.

Based on the low volatility of the 8 PAHs discussed here and considering the product categories investigated in more detail in this dossier (cf. section B.9.3.2.1), inhalation was not assumed to contribute significantly to the overall exposure when compared with the dermal route. For house dust exposure, please cf. chapter B.9.3.2.2.1.

With respect to oral uptake, it is noted that small children typically display intensive mouthing behaviour with respect to toys, possibly also those designed for use by older children living in the same household. Mouthing by children should also be taken into consideration when using rubber granulates on playgrounds. Granules may be swallowed by small children and PAHs may be released during the gastro-intestinal (GI) passage, potentially leading to considerable amounts of PAH exposure by the oral pathway.

However, in view of the greater available database with regard to dermal exposure, incl. migration experiments, and of the fact that, from a practical point of view, an additional consideration of mouthing behaviour could not have lead to a more rigorous restriction proposal, it was decided that the focus of this dossier should be placed exclusively on exposure assessment and subsequent risk characterisation for the dermal route.

B.9.3.3 Indirect exposure of humans via the environment

Aside from exposure towards PAH-burdened consumer products, possible sources of non-occupational exposure to PAHs are (acc. to WHO, 1998):

- polluted ambient air (main emission sources: vehicle traffic, industrial plants, and residential heating with wood, coal, mineral oil),
- polluted indoor air (main emission sources: open stoves and environmental tobacco smoke),
- tobacco smoking,
- contaminated food and drinking-water,
- use of products containing PAHs (coal-tar skin preparations and coal-tar-containing hair shampoos) ingestion of house dust, and
- dermal absorption from contaminated soil and water.

B.9.3.3.1 Exposure via smoking

Tobacco smoke contains thousands of chemical compounds, including many carcinogenic PAHs. Usually, BaP will be determined in cigarette smoke, but there are also values available for BaA, DBahA, BbFA and BjFA (cf. Table 49).

Table 49: PAH content in cigarette smoke (Values are given in ng/cigarette)

Details	BaP	BaA	DBahA	BbFA	BjFA	Reference
UK, mean of 25 brands	9.3	n.d.	n.d.	n.d.	n.d.	Gregg et al., 2004
USA, 9 brands, range	10-16	38-67	3.6-6.2	5.1-12	14-24	Ding et al., 2007

n.d. not determined

It is plausible, that BaP values in the US are higher compared to the EU since the directive 2001/37/EC limits the maximum tar level in cigarettes to 10 mg tar per cigarettes whereas no such restrictions were in place in the US.

A recent study has investigated the retention of PAHs by the smoker (Moldoveanu et al., 2008) and, applying these values to an average smoker who consumes 30 cigarettes per day, some estimates can be made for the exposure (cf. Table 50).

Table 50: PAH exposure of smokers (PAH content in smoke is based on Ding et al., 2007, retention values derived from Moldoveanu et al., 2008)

Details	BaP	BaA	DBahA	BbFA
USA, 9 brands, range in ng/cig	10-16	38-67	3.6-6.2	5.1-12
Consumption of 30 cigarettes per day PAH values in ng	300 - 480	1140 -2010	108 - 186	153 - 360
Retention value in the smoker in %	72	88	65	63
Exposure of the smoker values in ng per smoker	216 - 346	1003 - 1769	70 - 121	96 - 227

Biomonitoring of 3-hydroxybenzo[a]pyrene (3-OHBaP) has been performed scarcely in smokers and non-smokers, only one French study showed results since the amount of metabolite excreted is so low: The 3-OHBaP amount excreted in urine (in 24 h) averaged to 131 pg in smokers (< 40 to 367 pg) and 59 pg (< 44 pg to 224 pg) in non-smokers (Lafontaine et al., 2006). This shows, that only a minute part of the hundreds of ng BaP the smoker retain is excreted as 3-OHBaP.

B.9.3.3.2 Exposure via food and drinking water

An extensive description can be found in WHO (1998) or, specifically with regard to exposure via food of the general population (incl. children), also in WHO/JECFA (2005) and as part of a comprehensive evaluation by EFSA (2008). The latter two sources provide comparable reference estimates for mean daily BaP intake via food in the range of up to 10 ng BaP/kg bw/d.

BaP exposure has also been reviewed for drinking water (WHO 2003) and barbecued meat (FEHD 2004) to name just two out of a wealth of publications.

In this dossier, the background PAH burden of consumers via food is only considered in order to obtain a basic idea of comparative orders of magnitude of exposure (cf. section B.10).

B.9.3.3.3 Summary

In the view of the authors of this dossier, any inevitable environmental or even deliberate, intentional (smoking) exposure to PAHs does not preclude the necessity to reduce PAH exposure

from consumer products. Given that risk characterisation is able to show a potential risk and impact as well as socio-economic analysis indicate practicality, any opportunity should be taken to reduce exposure to genotoxic carcinogens.

B.9.3.4 Environmental exposure

Not relevant for this dossier

B.9.4 Other sources (for example natural sources, unintentional releases)

Not relevant for this dossier

B.9.5 Overall environmental exposure assessment

Not relevant for this dossier

B.9.6 Combined human exposure assessment

Due to lack of suitable data, a combined human exposure assessment has not been performed in this dossier.

From the analytical data presented here as well as from everyday experience it is clear that consumers are exposed to a multitude of potentially PAH-containing articles along several routes of exposure (cf. section B.9.3.2.3.5) and will most likely come in contact with more than just one of these products on a daily basis.

Significantly higher risk characterisation ratios (RCR) than those calculated in this report could be expected for combined exposure. However, in this dossier, concern could already be demonstrated for several exemplary exposure scenarios involving just one PAH source. For this reason, performing a combined exposure assessment did not appear necessary.

B.10 Risk characterisation

Please note that risk characterisation (RC) is only performed for the scenarios directly relevant for this dossier, i. e. use of PAH-contaminated consumer articles. Furthermore, only the dermal route has been taken forward to quantitative risk characterisation (cf. section B.9.3). A significant risk is demonstrated.

No RC has been carried out with regard to exposure during manufacturing of PAH-contaminated consumer articles or for indirect exposure via the environment.

B.10.1 Human health

B.10.1.1 Workers

Not relevant for this dossier

B.10.1.2 Consumers

B.10.1.2.1 Risk characterisation for consumers exposed to consumer articles via the dermal route

The DMELs for dermal BaP exposure of consumers derived from the relevant toxicological studies can be found in Appendix 2. A summary of the obtained ranges is given in Table 32 in section B.5.11.3.7.

These DMELs (or rather, DMEL ranges) have to be contrasted with the exposure estimates given in Table 47 and Table 48 for adults and children, respectively, for those consumer articles which have been tested positive for PAHs

At the same time it should also be noted that for most product categories 90 % of the articles were tested negative with respect to BaP, i. e. below an LOD/LOQ to be assumed in the order of 0.1 or 0.2 mg PAH/kg article). For the 6 EU-PAHs contained in the EPA PAH list, a slightly lower rate of negatives (ca. 84 %) was found when looking at all product groups, but a higher degree of variability between product groups (45.7 – 90.8 %) then for BaP was seen (cf. Table 43 below).

B.10.1.2.1.1 Risk characterisation for contaminated articles

For contaminated products, however, the following conclusions can be drawn when comparing DMELs and exposure estimates: The current situation (without restriction) is characterised by the following findings:

- when using the most conservative estimate of a migration rate of 10 %/h, estimated external dermal exposure of *adults* may amount to as much as 70 µg/kg bw/d. The corresponding RCR would thus range as high as > 2000-fold above the highest (i. e. least conservative) DMEL values given in Table 32.

- still, when using the least conservative migration assumption of only 0.2 %/h, adult exposure could be assumed to up to nearly 1.5 µg/bw/d with the most highly contaminated articles.

- for children, comparably high exposure estimates of ca. 70 µg/kg bw/d (assuming 10 % migration/h) and 1.3 µg/kg bw/d (0.2 %/h) obtained (corresponding to RCRs of ca. 2300 and 43, respectively).

B.10.1.2.1.2 Risk characterisation for articles containing 1 mg BaP/kg article

In addition, Table 47 and Table 48 above also give esposure estimates for the hypothetical situation that BaP would be restricted to a limit of 1 mg/kg article or down to the LOQ of 0.2 mg/kg bw/d as achieved by the analytical method:

- a limit of 1 mg BaP/kg product (part) would – depending on the exposure scenario - reduce exposure of adults to levels of up to 750 ng/kg bw/d (10 % migration/h), or 15 ng/kg bw/d (0.02 %/h). In light of the uncertainties discussed in the subsequent section, the latter value might seem acceptable, however, it is again underlined that this would only be true if the least conservative assumptions were made.

  Nevertheless for children, on top of their postulated higher vulnerability, a clearly higher exposure of up to 1600 ng/kg bw/d would be expected at a BaP level of 1 mg/kg product (10 % migration). Assuming a low migration rate of 0.2 %/h, exposure could be clearly reduced, the highest estimate still amounting to as much as 32 ng/kg bw/d, i. e. in the order of the least conservative DMELs derived, but clearly above those DMEL ranges obtained by the calculation methods recommended in the REACH guidance.

B.10.1.2.1.3 Risk characterisation for articles containing 0.2 mg BaP/kg article

Finally, a hypothetical limit set at the LOQ of 0.2 mg/kg can be shown to bring exposure down to a level that could be considered as tolerable for the bulk of products/uses examined, although for some extremely contaminated articles still an exposure up to one order of magnitude above the least conservative DMELs is found when assuming the most conservative migration rate:

- according to the calculations performed, adults would be at most exposed to levels of 150 ng/kg bw/d when assuming 10 % migration/h, or 3 ng/kg bw/d, when using 0.2 %/h.

- for children, maximum exposure levels of 320 or 6,4 ng/kg bw/d are calculated, when migration rates are set to 10 or 0.2 %, respectively.

As 0.2 mg BaP/kg article (component) constitute the official LOQ of the currently available analytical method, there is at present no sense in postulating a restriction below this level. However, at least for articles accessible to or used by children, future progress in analytical methodology should be frequently monitored and the technological feasibility of a further reduction should be considered.

Exposure is still modeled to be somewhat above even the least conservative DMELs. The results presented in this section therefore suggest that the ALARA principle (in the form of setting the limit value to the analytical LOQ) should also be applied equally to the other known carcinogenic PAHs addressed in this dossier. Moreover, it should be kept in mind, that these substances are regulated as surrogates/placeholders for a group of hundreds of congeners, some of which are known to (and many more might) be even more carcinogenic than BaP.

B.10.1.2.2 Discussion of uncertainties

The following points relevant for risk characterisation are each associated with (sometimes considerable) uncertainty (the following list is not considered exhaustive):

B.10.1.2.2.1 Toxicology

Uncertainties with respect to toxicological data exist due to:

- biological variability,
- absence of guideline- and GLP-conform tests, use of non-standardised test protocols, poor experimental conduct, incomplete reporting etc. resulting in limited reliability of some of the toxicity studies,
- uncertainty in intra- and interspecies extrapolation, e.g. as regards bioavailability along different routes, and
- using one compound as a surrogate toxicity indicator for mixtures of potentially several hundred congeners with potencies ranging over more than two orders of magnitude (cf. e. g. WHO 1998).

B.10.1.2.2.2 Exposure

With respect to exposure, uncertainty

- due to variability in results of chemical analysis of the products (both regarding the choice of the analytical method and the analytical results themselves),
- in estimating migration rates,
- in modelling exposure scenarios, and
- about the list of products being representative for the respective product groups

has to be considered.

B.10.1.2.2.3 Conclusion

Most of the uncertainty discussed above is inherently inevitable; the length of the list of potential sources of error is quite impressive (and certainly not exhaustive) and leads to the conclusion that statements based on this report should be carefully weighed. Some of this uncertainty is of course accounted for by applying assessment factors and/or by sometimes using worst-case assumptions. In addition, a range of DMELs was deduced rather than to rely on just one experiment.

However, in the light of the above list of sources of uncertainty, the authors of this dossier have decided to consider internal systemic exposure as probably uncritical from the viewpoint of consumer protection, if it is within a range of not more than one order of magnitude from the centre of the DMEL range established.

In other words, considering that the DMEL ranges calculated each span a range of about two orders of magnitude, also the conclusion can be drawn that dermal exposure at levels only slightly above the upper limit of a particular DMEL range would not urgently call for regulatory action.

B.10.1.2.3 Discussion of risk characterisation for consumers

The results of the risk characterisation are summarised in Figure 1.

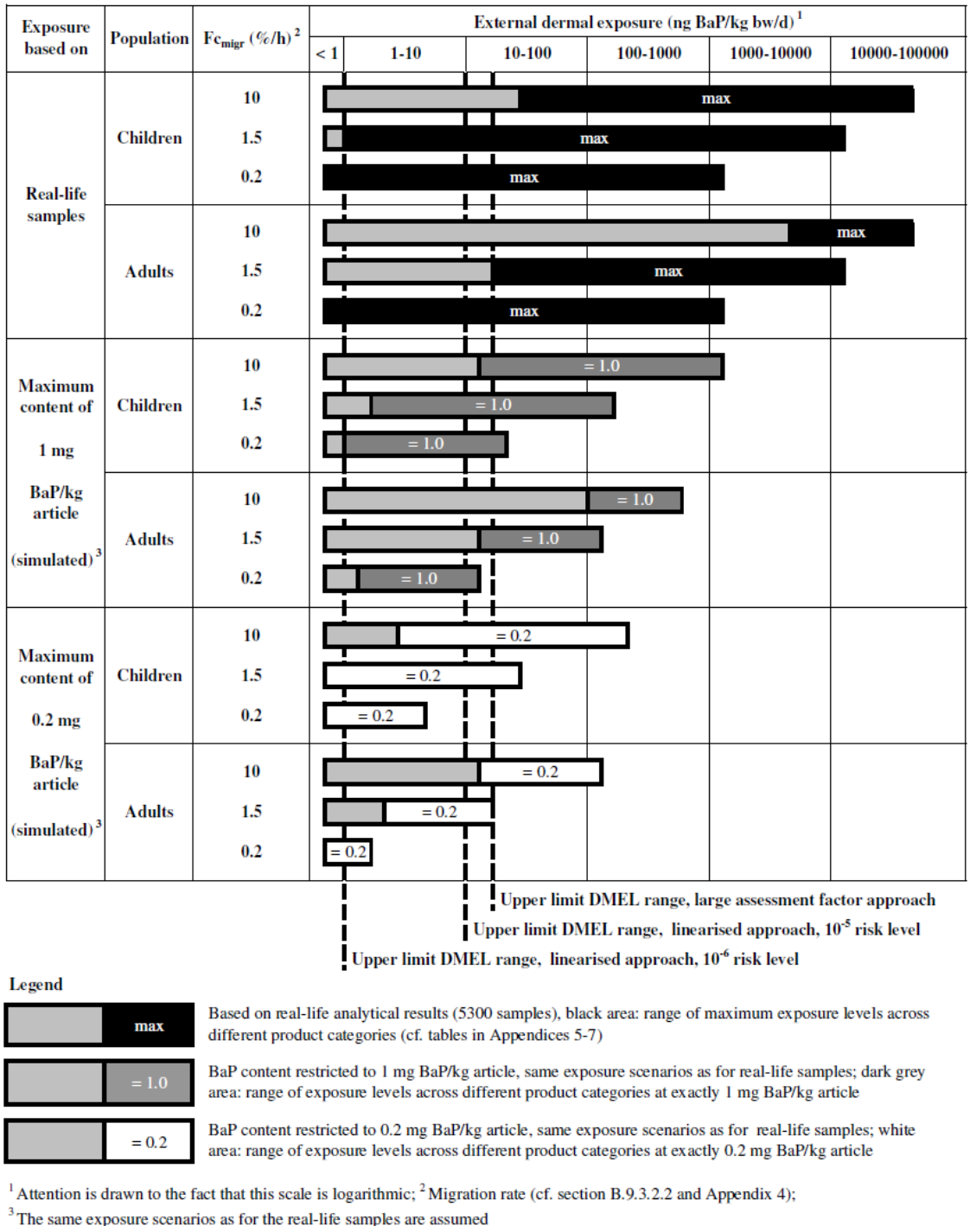


Figure 1: Overview of the results of risk characterisation

B.10.1.2.3.1 Real-life samples

Regardless of the migration rate chosen within a range of 0.2 – 10 %, a massive exceedance of even the upper limit of the least conservative DMEL range was found when calculating dermal exposure for real-life consumer article samples tested positive for PAHs (for a discussion of the number of articles with PAH non-detects vs. the number of positive ones, the reader is referred to section B.9.3.2.1).

Depending on the method chosen for DMEL derivation, dermal exposure of children estimated for the most highly contaminated samples was found to range up to 2-4 orders of magnitude above these upper limits of the corresponding DMEL range with a peak value of ca. 70000 ng/kg bw/d. For adults, similar peak values of up to 70000 ng/kg bw/d were found.

It is noteworthy that in spite of these alarming values, none of the samples from the group of toys was found to exceed the specific concentration limit of 100 mg BaP/kg as currently set by Dir. 2009/488/EC (EU Toy Safety Directive).

B.10.1.2.3.2 Risk characterisation for articles restricted to 1 mg BaP/kg article

In addition, Table 47 and Table 48 also give exposure estimates for the hypothetical situation that BaP would be restricted to a limit of 1 mg/kg article or down to the LOQ of 0.2 mg/kg bw/d as achieved by the analytical method:

- a limit of 1 mg BaP/kg product (part) would – depending on the exposure scenario - reduce exposure of adults to levels of up to 750 ng/kg bw/d (10 % migration/h), or 15 ng/kg bw/d (0.02 %/h). In light of the uncertainties discussed in the subsequent section, the latter value might seem acceptable, however, it is again underlined that this would only be true if the least conservative assumptions were made.

- Nevertheless for children, on top of their postulated higher vulnerability, a clearly higher exposure of up to 1600 ng/kg bw/d would be expected at a BaP level of 1 mg/kg product (10 % migration). Assuming a low migration rate of 0.2 %/h, exposure could be clearly reduced, the highest estimate still amounting to as much as 32 ng/kg bw/d, i. e. in the order of the least conservative DMEL derived, but clearly above those DMEL ranges obtained by the calculation methods recommended in the REACH guidance.

B.10.1.2.3.3 Risk characterisation for articles containing 0.2 mg BaP/kg article

Finally, a hypothetical limit set at the LOQ of 0.2 mg/kg can be shown to bring exposure down to a level that could be considered as tolerable for the bulk of products/uses examined, although for some extremely contaminated articles still an exposure up to one order of magnitude above the least conservative DMELs is found when assuming the most conservative migration rate:

- according to the calculations performed, adults would be at most exposed to levels of 150 ng/kg bw/d when assuming 10 % migration/h, or 3 ng/kg bw/d, when using 0.2 %/h.

- for children, maximum exposure levels of 320 or 6.4 ng/kg bw/d are calculated, when migration rates are set to 10 or 0.2 %, respectively.

As 0.2 mg BaP/kg article (component) constitute the official LOQ of the currently available analytical method, there is at present no sense in postulating a restriction below this level.

However, at least for articles accessible to or used by children, future progress in analytical methodology should be frequently monitored and the technological feasibility of a further reduction should be considered.

Likewise, as exposure is still modeled to be somewhat above even the least conservative DMELs, the results presented in this section suggest that the ALARA principle (in the form of setting the limit value to the analytical LOQ) should also be applied to the other known genotoxic and carcinogenic PAHs addressed in this dossier.

B.10.1.2.4 Indirect exposure of humans via the environment

Not relevant (cf. section B.4.3)

B.10.1.2.5 Combined exposure

Due to lack of suitable data, a combined human exposure assessment has not been performed in this dossier.

B.10.2 Environment

B.10.2.1 Aquatic compartment (including sediment and secondary poisoning)

Not relevant for this dossier

B.10.2.2 Terrestrial compartment (including secondary poisoning)

Not relevant for this dossier

B.10.2.3 Atmospheric compartment

Not relevant for this dossier

B.10.2.4 Microbiological activity in sewage treatment systems

Not relevant for this dossier

B.11 Summary on hazard and risk

B.11.1 Hazard

B.11.1.1 General information on hazard with regard to human health

Consumer products containing one or more polycyclic aromatic hydrocarbons (PAHs) as listed in entry 50 of Annex XVII of the REACH regulation (Reg. (EC) 1907/2006) are considered severely hazardous based on their carcinogenic and mutagenic properties, as well as their potential for being toxic to reproduction.

All eight PAHs discussed in this dossier are classified carcinogens of category 2 (DSD) or 1B (CLP), respectively. Benzo[a]pyrene (DSD Cat. 2) and chrysene (DSD Cat. 3) also are legally classified mutagens. Lack of classification for the other congeners does not necessarily reflect absence of genotoxicity, but may rather be attributed to the comparatively limited database available for these compounds.

The focus of this dossier is placed on the carcinogenicity of BaP and the other PAHs under question. The observed mutagenic properties by themselves do not allow for a quantitative risk characterisation but are considered in a qualitative way, i. e. in establishing the mechanism behind PAH carcinogenicity.

The potential of at least BaP to cause toxicity to reproduction is noted. However, for this type of toxicity usually threshold dose levels can be assumed, below which no substance-related adverse effects are expected. Consequently, it was considered unlikely that the resulting DNELs for reproduction toxicity would fall below the DMELs proposed in this dossier on the basis of non-threshold carcinogenicity. Therefore reproduction toxicity was not evaluated further in this dossier.

It is noted that with respect to carcinogenicity, children have to be considered a particularly sensitive sub-population, both in terms of an inherent greater sensitivity and their longer remaining life-span (increasing the statistical risk for the development of cancer following exposure towards PAHs).

A number of comprehensive toxicological evaluations of PAHs is available in the published literature. Therefore many other aspects of PAH toxicity have not been treated in-depth or even were omitted in this dossier, if they were not considered relevant for its purpose.

Evaluation of the database of animal tests regarding the dermal absorption of BaP resulted in the establishment of rough working estimates of 50 and 20 % from acetone or aqueous media (such as sweat), respectively. Likewise, coarse grain estimates of 50 % each were used to cover absorption along the oral route and by inhalation.

B.11.1.2 Derivation of DMELs for carcinogenicity

DMELs were calculated from a set of available animal studies using specified selection criteria and following the scheme as provided in the REACH IR/CSA guidance on dose-response assessment (R.8). T25, BMD_{10}, and $BMDL_{10}$ were all used as dose descriptors. DMELs were calculated applying both the 'Large Assessment Factor' and the 'Linearised’ approach' (the latter at both the 10^{-5} and 10^{-6} risk levels and using the 'Probit' as well as the 'Multistage Cancer' algorithms for curve fitting).

The following DMEL results ranges (excluding the Probit calculations) were obtained:

Large Assessment Factor approach: 0.1 – 30 ng/kg bw/d

Linearised Approach, 10^{-5} risk level: 0.03 – 10 ng/kg bw/d

Linearised Approach, 10^{-6} risk level: 0.004 – 1 ng/kg bw/d

B.11.2 Exposure

B.11.2.1 Emissions

The present dossier is based on the evaluation of more than 5300 samples from consumer articles analysed for their PAH content. The samples analysed covered a multitude of different consumer products, which – based on similar exposure patterns - were subdivided into the following eight broader categories: 1. Electrical devices, 2. Grips/handles, 3. Skin contact areas of sports equipment or other consumer products, 4. Toys, 5. Materials with close contact to the body, 6. Other products with skin contact, 7. Tyres and rolls, and 8. Other products.

Calculated over all product groups, in 91.9 % of the cases BaP was not detectable and in 95 % of all samples the concentration was below 1 mg/kg. The corresponding values were 83.9 % not detectable, 90.7 % of the values below 1 mg/kg for the sum of the 6 EU-PAHs contained in the EPA-PAH list. However, detected levels vary considerably between different product groups.

In the remaining samples, the highest PAH levels found were 1200 mg/kg for BaP, 25400 mg/kg for the sum of all EPA-PAHs and 6930 mg/kg for the sum of all EU-PAHs.

B.11.2.2 Migration rates

For the exposure estimation of consumers in this dossier, migration rates quantified under dynamic conditions were used. These are strongly influenced by the material and the contact conditions such as mechanical friction and intensity of contact (pressure) between skin and product.

Three different migration rates have been estimated. In order to cover the different materials on the market and the influence of the dynamic conditions on the realistic expectancy range of dermal exposure, all of the following three migration rates for BaP were used for the calculations:

- 10 % as the worst case of dynamic migration with friction,
- 1.5 % as the approximate geometric mean of dynamic migration with friction,
- 0.2 % as the mean of dynamic migration for materials with low migration, when friction is not considered.

The knowledge about release of these compounds needs to be improved. Standardised migration methods that cover the typical uses of the consumer articles investigated should be developed by further projects.

B.11.2.3 Exposure assessment

Exposure of children to rubber granules

The assessment of dermal exposure of children to rubber granules from synthetic turf was performed using a) the ECETOC TRA approach b) a similar approach, but considering migration. The results clearly show that rubber products may contain high amounts of polycyclic hydrocarbons. The knowledge about release of these compounds needs to be improved, but there is evidence that considerable amounts can be released.

External exposure to other consumer articles

The bulk of exposure assessment was carried out on the articles listed in section B.9.3.2.1 above. The focus of this evaluation was on the dermal route. The external dermal exposure by consumer products was estimated in two different ways:

1. The results of the dynamic migration tests described in chapter B.9.3.2.2.4 were used for estimating the external exposure to BaP by those products included in these tests.

2. The external dermal exposure was estimated based on the estimated migration rates and on the available data on BaP concentrations in consumer products.

By approach 1, external dermal exposure levels from a total of 7 products (grips, handle of a torch, a hooter, a steering wheel cover, and a hammer grip) were estimated to range from ca. 2 ng/kg bw/d up to 6.7 µg/kg bw/d.

Approach 2 was applied to > 100 articles assumed to be used by adults and ca. 100 articles assumed to be used by children. In each case, exposure estimates were calculated using all three of the above migration rates. Using the highest (most conservative) assumption of 10 % migration/h, exposure estimates of 0.3-68613 ng/kg bw/d for adults and 3-66780 ng/kg bw/d for children were obtained. Estimates using 1,5 or 0.2 % migration/h ranged lower by a factor of 6.67 or 50, respectively.

Summarising the results, there is clear evidence that under normal use conditions considerable amounts of BaP can be released even when a comparatively low migration rate is assumed.

Simulation of the proposed restriction conditions

In order to simulate the effect of the proposed restriction, another set of calculations was carried out, this time assuming that the same products used with approach 2 before would have contained 1 or only 0.2 mg BaP/kg product (but otherwise making the same assumptions regarding exposure scenarios and migration rates):

For the 1 mg BaP/kg article simulation, this resulted in a dermal exposure range of 133.3 – 750 ng/kg bw/d for adults assuming 10 % migration/h (children: 8.5 – 1600 ng/kg bw/d). Again, estimates using 1,5 or 0.2 % migration/h can be obtained by dividing by a factor of 6.67 or 50, respectively.

For the 0.2 mg BaP/kg article simulation, corresponding dermal exposure values could be obtained by dividing the results from the 1 mg/kg simulation by a further factor of 5, i. e. a range of 26.67 – 150 ng/kg bw/d for adults assuming 10 % migration/h was calculated (children: 1.7 – 320 ng/kg

bw/d). Estimates using 1,5 or 0.2 % migration/h again would be lower by factors of 6.67 or 50, respectively.

Other exposure scenarios

Exposure at the workplace or indirect exposure via the environment via food or smoking have not been considered in this dossier due to lack of relevance for the problem addressed by this restriction dossier.

Combined/aggregate exposure assessment

Owing to lack of suitable data, a combined/aggregate human exposure assessment has not been performed in this dossier. From the analytical data presented in this dossier as well as from everyday experience it is clear that consumers are exposed to a multitude of potentially PAH-containing articles via several routes and will most likely come in contact with more than just one of these products on a daily basis. However, in this dossier, concern could already be demonstrated for several exemplary exposure scenarios involving just one PAH source. For this reason, a combined exposure assessment did not appear necessary.

B.11.3 Risk

Risk characterisation (RC) was only performed for the scenarios directly relevant for this dossier, i. e. use of PAH-contaminated consumer articles. Furthermore, only the dermal route has been taken forward to quantitative risk characterisation (cf. section B.9.3).

B.11.3.1 Risk characterisation for contaminated articles

DMELs (or rather, DMEL ranges) were contrasted with the exposure estimates given in Table 47 and Table 48 for adults and children, respectively, for those consumer articles which had been tested positive for PAHs.For these products, the following conclusions with respect to the current risk situation (without restriction) were drawn when comparing DMELs and exposure estimates:

- when using the most conservative estimate of a migration rate of 10 %/h, estimated external dermal exposure of *adults* may amount to as much as 70 µg/kg bw/d and would thus range as high as > 2000-fold above the highest (i. e. least conservative) DMEL values given in Table 32.

- still, when using the least conservative migration assumption of only 0.2 %/h, adult exposure could be assumed to reach up to nearly 1.5 µg/bw/d with the most highly contaminated articles.

- for children, comparably high exposure estimates of up to ca. 70 µg/kg bw/d (assuming 10 % migration/h) and 1.3 µg/kg bw/d (0.2 %/h) were obtained.

B.11.3.1.1 Risk characterisation for the effect of simulated restrictions

Table 47 and Table 48 above give esposure estimates for the hypothetical situation that BaP would be restricted to a *limit of 1 mg/kg article* or down to the LOQ of 0.2 mg/kg bw/d as achieved by the analytical method:

- a limit of 1 mg BaP/kg product (component) would – depending on the exposure scenario - reduce exposure of adults to levels of up to 750 ng/kg bw/d (10 % migration/h), or 15 ng/kg

bw/d (0.02 %/h). In light of the uncertainties discussed, the latter value might seem acceptable, however, it is underlined that this would only be true if the least conservative assumptions were made.

- for children, nevertheless, on top of their postulated higher vulnerability, a clearly higher exposure of up to 1600 ng/kg bw/d would be expected at a BaP level of 1 mg/kg product (10 % migration). Assuming a low migration rate of 0.2 %/h, exposure could be clearly reduced, the highest estimate still amounting to as much as 32 ng/kg bw/d, i. e. in the order of the least conservative DMELs derived, but clearly above those DMEL ranges obtained by the calculation methods recommended in the REACH guidance.

Finally, a hypothetical *limit set at the LOQ of 0.2 mg/kg* can be shown to bring exposure down to a level that could be considered as tolerable for the bulk of products/uses examined, although for some extremely contaminated articles still an exposure up to one order of magnitude above the least conservative DMELs is found when assuming the most conservative migration rate:

- according to the calculations performed, adults would be at most exposed to levels of 150 ng/kg bw/d when assuming 10 % migration/h, or 3 ng/kg bw/d, when using 0.2 %/h.

- for children, maximum exposure levels of 320 or 6.4 ng/kg bw/d are calculated, when migration rates are set to 10 or 0.2 %, respectively.

As 0.2 mg BaP/kg article (component) constitute the official LOQ of the currently available analytical method, there is at present no sense in postulating a restriction below this level. However, at least for articles accessible to or used by children, future progress in analytical methodology should be frequently monitored and the technological feasibility of a further reduction considered.

Likewise, as exposure is still modeled to be somewhat above even the least conservative DMELs, the results presented in this section suggest that the ALARA principle (in the form of setting the limit value to the analytical LOQ) should also be applied to the other known genotoxic and carcinogenic PAHs addressed in this dossier even though some of these are regarded to be of a lower carcinogenic potency than BaP. After all it should be kept in mind, that these substances are regulated as surrogates/placeholders for a group of hundreds of congeners, some of which are known to (and many more might) be even more carcinogenic than BaP.

C. Available information on alternatives

C.1 Identification of potential alternative substances and techniques

C.1.1 Product Data

The most important argument for the availability of alternatives comes from the product tests. As PAH-levels found in most products prove, it is technically very well possible to produce consumer products with PAH levels below LOQ. The analytical data of various tested product groups show that most products do not contain PAHs while only some products of the same group contain PAHs. In fact, in the majority of products tested, the problem lies in the low-price sector products. This indicates that the use of PAH–containing additives is mostly due to economical reasons and not for reasons concerning the technical properties of the PAH. Nonetheless, it should be noted that high PAH concentrations were also found in premium products.

C.1.2 Experience with the restriction of PAHs in extender oils for tyres

In extender oils used for tyres placed on the European market the PAH-content is restricted since 01.01.2010 according to the REACH regulation, Annex XVII, No 50 to 1 mg/kg BaP or to 10 mg/kg of the sum of all eight listed PAHs. Industry therefore already experienced the replacement of PAH-containing extender oils for a high volume product which has to comply with high technical requirements. The European Tyre & Rubber Manufacturer Association (ETRMA, formerly BLIC) coordinated the activities to do research regarding the necessary technical properties of the alternative oils, to ensure the availability of the alternative oils, to define new quality requirements also for oil extended rubber compounds and to deliver analytical methods not only for the extender oils but also for the tyres as (imported) end product. Today extender oils with reduced or no PAH content are used in tyres (details see below). This example shows that alternatives exist and can be used. It is hard to argue, that consumer product should comply with a lower protection level than tyres regarding the content of hazardous PAH.

C.1.3 Statements of industrial organisations

The Statements of important Industrial Organisations also implicate, that PAHs in consumer products are not necessary. For example Plastics Europe wrote in a questionnaire conducted when preparing this dossier: “The member companies of Plastics Europe do not use PAHs intentionally in their products. Only in plastics which use carbon black as filling material technical contaminations with PAHs cannot be excluded. For critical applications like food packaging materials or toys carbon black is used which complies with the purity requirements of the ’plastics recommendation IX on colorants for food packaging and commodities’ of the German Federal Institute for Risk Assessment (BfR)” (statement translated from German to English).

This statement is backed by the answers of different single companies in the questionnaire. It therefore seems that the problem of PAHs in consumer products might be mainly a problem of imported goods.

C.1.4 Alternative 1: Extender oils with reduced PAH content

The first alternative is to use extender oils which have a reduced PAH content or which are free of PAH. These alternatives have been tested for the use in tyre rubbers but are also suitable for other rubber applications. They are also suitable to replace extender oils in plastics like soft PVC.

The original extender oils for tyres were DAE (distilled aromatic extracts) with a high content of PAH, therefore commonly named "labelled oils" since they have to be classified and labelled according to directive 67/548/EC or regulation (EC) No 1272/2008. Different alternative oils could be used for its replacement, commonly named "unlabelled oils" with no labelling required. The limit values of the PAH-restriction in extender oils for tyres under REACH "are regarded as kept, if the polycyclic aromatics (PCA) extract is less than 3 % by mass, as measured by the Institute of Petroleum standard IP346: 1998 (Determination of PCA in unused lubricating base oils and asphaltene free petroleum fractions – Dimethyl sulphoxide extraction refractive index method), provided that compliance with the limit values of BaP and of the listed PAHs, as well as the correlation of the measured values with the PCA extract, is controlled by the manufacturer or importer every six month …" (REACH, Annex XVII, No. 50). Therefore the maximum content of the alternative oils is 3% extractable PCA, but can be less, too. Beyond this there is a wide variety in the exact chemical composition of the different oils. The following types of unlabelled oils can be distinguished (Shaw, 2008; Shaw, 2009; Neau and Rangstedt, 2009; Joona, 2003; Null, 1999). A process scheme is given in Neau and Rangsted (2009):

- **MES (mild extract solvates)** consist mainly of paraffinic oils, i.e. linear alkanes, which result from the lube base oil production and where aromatic components with two and more rings have been removed by extraction. Further refining can lead to so called "white oils".

- **Treated distilled aromatic extracts (TDAE)** are produced from distilled aromatic extracts (DAE) which are further treated by a solvent extraction to remove the aromatic components with three rings and upwards.

- **Treated Residual Aromatic Extracts (TRAE)** are produced from residual aromatic extracts (RAE), a heavy oil fraction produced from the de-asphalting of heavy petroleum residues, by solvent extraction of aromatic components.

- **Naphthenic oils** consist mainly of cyclic alkanes. They are produced by hydro-treatment of heavy vacuum petroleum distillates.

Nevertheless the aromatic extracts may contain alkylated polyaromatic constituents other than PAHs, which has been demonstrated by chemical analysis of these multi-component mixtures. There is scientific concern that also several alkylated polyaromatic compounds possess carcinogenic potency. As a consequence, for consumer products the use of extender oils that are free of aromatic compounds (e. g. "white oils") is recommended.

C.1.5 Alternative 2: Carbon black with reduced PAH content

Carbon Black with a reduced content of PAHs is available on the market. In order to produce Carbon Black with a low PAH content several methods of removing the PAHs after the initial production of Carbon Black are possible. The most common methods seem to be thermal treatment under vacuum or inert gas atmosphere at temperatures >300°C and solvent extraction of the Carbon Black. This is mainly done using a Soxhlet Extractor and common organic solvents like hexane or toluene.

C.1.6 Alternative 3: Thermoplastic elastomers (TPE)

Depending on the intended use of the product and the required flexibility and mechanical properties, thermoplastic elastomers which do not need extender oils or plasticizers at all can also be an alternative. Thermoplastic elastomers contain a hard, thermoplastic compound and a soft, elastomeric compound which are physically bound to each other to form the final elastic polymer. Different types of thermoplastic elastomers are TPE-O or TPO (thermoplastic olefins), TPE-S or TPS (styrenic block copolymer), TPE-U or TPU (thermoplastic polyurethanes), TPE-A or TPA (thermoplastic polyamides), TPE-E or TPC (thermoplastic copolyester) and TPE-v or TPV (thermoplastic vulcanizates). In this field of different types of thermoplastic elastomers a big variety of specific thermoplastic elastomers is available.

C.1.7 Alternative 4: Surrender of use

In some cases the best alternative might be not to use any extender oils or carbon black at all. In many cases extender oils are necessary because of the high amount of economically cheap filling materials. A reduction of the amount of filling material could make it unnecessary to use extender oils in some cases.

C.1.8 Alternative 5: Specific uses as binding agents for clay pigeons and coal briquetting

- **Clay pigeons:** As alternatives to the PAH containing CTPHT (Coal tar pitch high temperature) normally petrochemical binders like bitumen are used. Bitumen has a lower PAH content than CTPHT but still leads to a PAH-burden of around 30 mg/kg of EPA-PAHs in clay pigeons (UVEK 2008). In Austria and the Netherland the use of CTPHT in clay pigeons is already forbidden. Another, better alternative for coal tar pitch could be the use of a mixture of several clays only, without any binder, as reported for one company in the RAR of CTPHT (The Netherlands - Bureau REACH, 2009).

- **Coal briquetting:** For coal briquettes the possibility to use more environmental friendly binders like starch and molasses is proposed in the RAR of CTPHT (The Netherlands - Bureau REACH, 2009).

C.2 Assessment of alternatives

Attention has to be paid to the fact, that the suitability of an alternative can only be assessed finally on the level of a specific product. Different articles like tyres, shoes, handholds or floorings might need different alternatives due to their different technical and use characteristics. Therefore the following section provides only a very general assessment of the alternatives 1 to 3. The alternative 4 (surrender of use) would have to be discussed in detail for each different usage in the articles where a surrender would be possible. Alternative 5 is a very specific usage that has been discussed already in the RAR for CTPHT (The Netherlands - Bureau REACH, 2009).

Furthermore the use of extender oils containing polyaromatic compound other than PAHs for consumer products should be avoided because of their possible carcinogenic potency. The use of extender oils that are free of aromatic compounds (e. g. white oils) is recommended.

C.2.1 Availability of alternatives

C.2.1.1 Alternative 1: Extender oils with reduced PAH content

Much attention has been paid by the manufactures of tyres to the question of the availability of alternative extender oils, because they need a huge quantity of extender oils of around 1 million tonnes per year. The industry had several years time to prepare for the coming restriction. Nowadays the conversion to unlabelled oils has started not only in Europe but worldwide, with varying preferences for the different alternative extender oils between the different companies (Shaw, 2008; Shaw, 2009; Anonymus, 2009).

C.2.1.2 Alternative 2: Carbon black with reduced PAH content

The alternative is available, because it is already required for special applications. No market shares are known, but they are probably still low.

C.2.1.3 Alternative 3: Thermoplastic elastomers

Thermoplastic elastomers are well established products on the plastics market. There is no reason known for a lack of availability.

C.2.2 Human health risks related to alternatives

At the time this dossier was compiled, no specific data on human health risks potentially arising from the use of the above alternatives as compared to using PAH-rich articles was available to the authors of this dossier.

C.2.3 Environment risks related to alternatives

Concerning the PAH-content and the environmental risks related with PAH, all the mentioned alternatives are clearly preferable.

C.2.4 Technical and economic feasibility of alternative

C.2.4.1 Alternative 1: Extender oils with reduced or no PAH content

- **Technical feasibility:** The technical feasibility of alternative extender oils for tyre rubbers has been extensively studied by the tyre industry with the result that the now available alternatives show good technical characteristics. During the process some reformulation of the rubber had to be made to obtain the same wet grip, a necessary characteristic for tyres (Shaw, 2009; Neau and Rangstedt, 2009); Joona, 2003; Null, 1999). Taking into account that the technical requirements for consumer products are in most cases considerable lower than for tyres, there is no doubt about the technical feasibility of the alternative extender oils. In the questionnaire performed when preparing the dossier some manufacturers only raised some concern for product parts which have to endure high mechanic stress at high temperatures.

  Alternative oils might also technically serve as secondary plasticiser for Soft-PVC as shown in the product information at the homepage of Nynas and also shortly explained in an article there (Nynas, 2003).

- **Economic feasibility:** Unlabelled oils are more expensive than the labelled ones, but the burden is the same for all producers for the European market. Since a restriction also addresses imports the same rules and burdens also exist for all actors in the market.

C.2.4.2 Alternative 2: Carbon black with reduced PAH content

- **Technical feasibility:** The technical feasibility is assumed, because there are already some applications which require carbon black with reduced PAH content.

- **Economic feasibility:** The product price might increase slightly, because an additional processing step for the reduction of the PAH content would be necessary.

C.2.4.3 Alternative 3: Thermoplastic elastomers

- **Technical feasibility:** Thermoplastic elastomers are materials which behave in a wide temperature range like rubbers and show elastic properties. Above this temperature range they loose their elasticity, start to melt and can be moulded. After cooling down they become elastic again. Thermoplastic elastomers show some advantages during the manufacture compared with rubber: they consist of few compounds which are easy to control and to mix, energy consumption during the manufacture is relatively low and they are easy to mould by extrusion or injection moulding. Furthermore they can be produced in different hardness grades, are easy to dye and recyclable. The disadvantages of thermoplastic elastomers lay in their poor chemical and heat resistance and their lower compression and thermal stability.

  Today thermoplastic elastomers already replace rubber or soft-PVC in applications like coatings for wires and cables, tubes, pipes, spring elements, joints and gaskets, tool handles, shoe soles, conveyor belts, kitchenware and consumer electronics.

- **Economic feasibility:** Thermoplastic elastomers are presumed to be engineering plastics with a higher price than the usual commodity plastics (like soft-PVC). It is not possible to load them with cheap filling materials like it is possible with rubber. In conclusion it depends on the

required characteristics and the special purpose of the end products, if an elastomeric plastic might be an economically feasible alternative.

C.2.5 Other information on alternatives

None

D. Justification for action on a Community-wide basis

D.1 Considerations related to human health and environmental risks

The PAHs considered in this dossier are identified as carcinogens (Carc.Cat. 2/Carc. 1B according to the DSD/CLP). Furthermore BaP and CHR are classified for their mutagenic effects (Muta. Cat. 2 / Muta. 1B or Muta. Cat. 3 / Muta 2, respectively; cf. Table 2)., and BaP for toxicity to reproduction.

In the view of the authors of this dossier, exposure of consumers to genotoxic carcinogens should be minimised to the maximum extent reasonably possible (ALARA principle).

The present dossier clearly shows that consumers might be put at risk by a considerable fraction of consumer articles made of rubber or other plasticised materials, which are currently on the market. This has been demonstrated in the section on risk characterisation (RC, section B.10), where the toxicological assessment has been contrasted with findings from analytical examinations performed by independent laboratories on a large collective of samples from many different consumer article categories, including toys.

Based on the results of RC, it is deemed necessary that corresponding action should be taken to limit the content of PAHs in consumer articles down to the limit of quantification (LOQ, cf. section A.1.2) achievable by monitoring laboratories in the EU.

D.2 Considerations related to the internal market

It is clear, that marketing of the consumer products dealt with in this report is not confined to any national market, but that such products are spread evenly over the Community market instead, thus justifying Community-wide action.

In addition, a restriction of PAHs in consumer products via Art. 68 (2) of the REACH regulation allows for the inclusion of imported articles in the same way as for such products produced within the EU.

D.3 Other considerations

Based on a statistical evaluation of analytical samples, it is noted that a great majority of the analysed articles already today comply with the limit proposed in this dossier. Thus, the proposal appears also to be technically feasible at an arguable cost.

D.4 Summary

Community-wide measures to restrict PAHs in consumer products are considered necessary based on considerations of human health risk, marketing profile of the articles under question, and technical feasibility.

E. Justification why the proposed restriction is the most appropriate Community-wide measure

E.1 Identification and description of potential risk management options

E.1.1 Risk to be addressed – the baseline

E.1.1.1 Introduction

There are two main sources of PAHs in consumer products:

- carbon black containing PAHs used as black colouring agents in rubbers, plastics, and coatings and
- extender oils containing PAHs used in soft rubbers and plastics.

PAHs themselves fulfil no functional purposes but occur as by-products or impurities in various production processes and materials. Due to the nature of potentially contaminated materials (soft or dark plastic surfaces), PAHs are wide-spread in a multitude of goods which are intended to be used by or could under foreseeable circumstances be used by consumers (hereafter referred to as consumer goods).

As a result of the ubiquity of PAHs, it is not possible to list all affected products or product groups exhaustively. Typical examples for consumer products with elevated PAH levels are tools with rubber grips, various types of rubber wheels, plastic shoes, textiles and sports equipment as well as plastic toys. The latter category is particularly worrying as many toys are aimed at small children who may be highly exposed through prolonged handling or placing into the mouth of contaminated articles.

While a robust figure for the number of PAH-contaminated products, either in the consumer market as a whole or in a specific product group (for example toys), cannot be given, the below summary of recent testing results may provide some insights into the severity of the problem.

E.1.1.2 Results of PAH tests in consumer products

E.1.1.2.1 TÜV Rheinland

TÜV Rheinland is a provider of technical, safety and certification services. In 2009, TÜV Rheinland published the results of a series of tests for PAHs in inflatable water toys[1] (purchased in popular European vacation destinations in the Mediterranean) and various types of consumer products[2] (purchased in bargain and home improvement stores). Products were tested for

[1] TÜV Rheinland (2009a). TÜV Rheinland LGA testet aufblasbare Schwimmartikel: Vergifteter Badespaß. (http://www.tuv.com/de/news_mittelmeertest.html)

[2] TÜV Rheinland (2009b). Risikofaktor PAK: Konzentration in Produkten alarmierend hoch. (http://www.tuv.com/de/news_pak_in_produkten.html)

compliance with the PAH criteria set forth by the GS mark ('Geprüfte Sicherheit', a voluntary safety and quality certification mark) which are as follows:[1]

Table 51: PAH criteria set forth by the German GS quality mark

Parameters	Category 1	Category 2	Category 3
	Materials intended to be put in the mouth and toys for children under 36 months intended to come into contact with skin	Materials not covered by category 1 with foreseeable contact with skin for more than 30 seconds (long-term skin contact)	Materials not covered by categories 1 or 2 with foreseeable contact with skin for up to 30 seconds (short-term skin contact)
Benzo[a]pyrene (mg/kg)	Not detectable (< 0.2)	1	20
Sum of 16 PAHs (EPA) (mg/kg)	Not detectable (< 0.2)	10	200

Table 52 summarises the number of products tested in each category as well as the number and percentage of products whose PAH levels were above these limits.

Table 52: Results of tests by TÜV Rheinland for compliance with the PAH criteria cited in GS mark

Product category	Number of products tested	Number of products with PAH levels above limits	Percentage of products with PAH levels above limits	Source
Wheelbarrow/cart wheels	5	5	100 %	TÜV Rheinland, 2009b
Bicycle horns/grips	4	4	100 %	TÜV Rheinland, 2009b
Plastic clogs/sandals	2	2	100 %	TÜV Rheinland, 2009b
Hand tools	12	7	58 %	TÜV Rheinland, 2009b
Inflatable water toys	25	6	24 %	TÜV Rheinland, 2009a

E.1.1.2.2 'test'

'test' is a monthly magazine published by Stiftung Warentest, a consumer product testing organisation. As part of their reviews, 'test' routinely checks for PAH levels in potentially affected products. PAH levels are rated on a scale of 1 to 5 where 5 is the lowest rating and indicative of a "non satisfactory" PAH concentration. Specifically, a rating of 5 was awarded to products with PAH concentrations exceeding GS mark limits (see Table 51). A rating of 4 was given to products which, although barely satisfying GS mark criteria, were still judged to be significantly contaminated with PAHs.

Table 53 shows a summary of tests in which PAH-contaminated products were found. Each test usually compares a cross-section of products within a given category (for example children's

[1] ZLS (2009). ZEK 01.2-08: PAH test and evaluation required for GS certification, p. 4. (http://www.zls-muenchen.de/de/left/aktuell/pdf/zek_01_2-08_pak_verbindlich_mindermengen.pdf)

bicycles), covering both lower and higher end products from a variety of manufacturers and retailers.

Table 53: Overview of PAH-contamined products (test, 2006-2009)

Product category	Number of products tested	Number of products with PAH levels rated 4	Number of products with PAH levels rated 5	Percentage of products with PAH levels rated 4 or 5	Percentage of products with PAH levels rated 5	Source
Home improvement products	33	2	26	85 %	79 %	test 4/2006
Children's trainer bikes	15	0	9	60 %	60 %	test 7/2008
Baby strollers	14	0	6	43 %	43 %	test 9/2009
Thermos bottles	16	0	5	31 %	31 %	test 8/2008
Baby buggies	15	0	2	13 %	13 %	test 7/2006
Colour markers	20	4	2	30 %	10 %	test 9/2008
Flashlights and headlights	16	2	1	19 %	6 %	test 1/2006
Ski goggles	17	1	1	12 %	6 %	test 1/2009
Power drills	18	1	1	11 %	6 %	test 2/2006
Erasers	23	1	0	4 %	0 %	test 9/2008

E.1.1.2.3 'ÖKO-TEST'

'ÖKO-TEST' is a consumer magazine focusing on health and environment related aspects of consumer products. Similarly to test, ÖKO-TEST publishes reviews of consumer products which are tested, among others, for PAH levels. PAH levels found by testers to be concerning are labeled as "elevated" (sum of 24 PAHs exceeding 0.1 mg/kg) or "strongly elevated" (sum of 24 PAHs exceeding 1 mg/kg). Table 54 shows the occurrence of such PAH concentrations in some product categories relevant to the proposed restriction. The tested products are largely representative of typical offerings marketed to consumers in a given product category.

Table 54: Overview of PAH-contamined products (ÖKO-TEST, 2006-2009)

Product category	Number of products tested	Number of products with elevated PAH levels	Number of products with strongly elevated PAH levels	Percentage of products with elevated or strongly elevated PAH levels	Percentage of products with strongly elevated PAH levels	Source
Children's bicycles	9	0	9	100 %	100 %	ÖKO-TEST 5/2009
Children's rubber boots	15	1	14	100 %	93 %	ÖKO-TEST 10/2007
Swimming aids	16	4	11	94 %	69 %	ÖKO-TEST 7/2008
Children's sneakers	18	6	12	100 %	67 %	ÖKO-TEST 8/2008
Cordless screwdrivers	9	3	6	100 %	67 %	ÖKO-TEST 3/2009
Work gloves	20	8	12	100 %	60 %	ÖKO-TEST 2/2009
Plastic toy figures	13	6	7	100 %	54 %	ÖKO-TEST 12/2009
Carnival masks	10	5	4	90 %	40 %	ÖKO-TEST 2/2010
Children's trainer bikes	10	4	4	80 %	40 %	ÖKO-TEST 5/2007
Erasers	20	3	5	40 %	25 %	ÖKO-TEST 1/2007
Plastic clogs	22	16	5	95 %	23 %	ÖKO-TEST 7/2008
Vacuum cleaners	11	5	2	64 %	18 %	ÖKO-TEST 9/2008
Children's mud pants	13	10	2	92 %	15 %	ÖKO-TEST 3/2008
Children swimming pools	13	7	2	69 %	15 %	ÖKO-TEST 7/2009
Power drills	11	4	1	45 %	9 %	ÖKO-TEST 8/2008
Teething rings	20	7	1	40 %	5 %	ÖKO-TEST 3/2007
Changing pads	18	6	0	33 %	0 %	ÖKO-TEST 1/2008
Children's tooth brushes	18	6	0	33 %	0 %	ÖKO-TEST 2/2008
Doll buggies	6	2	0	33 %	0 %	ÖKO-TEST 12/2009
Children's bicycle seats	14	4	0	29 %	0 %	ÖKO-TEST 4/2008
Tooth brushes	16	0	0	0 %	0 %	ÖKO-TEST 9/2008

E.1.1.3 Assessment of the baseline situation

As different organisations may use different test methods, a precise assessment of the number of PAH-contaminated consumer products on the market is not possible. Despite this, the following observations can be made:

In some product categories (such as children's bicycles and some hand and power tools), significant contaminations in tyres, gripping surfaces etc. are common in more than 50 % of tested products.

In other product categories (for example water toys and certain types of shoes), the percentage of highly contaminated products is lower (often in the range of 10 – 20 %). Nevertheless, speaking in

absolute terms and taking into account the large market volume of products in these categories, elevated PAH levels are still likely to be present in a considerable number of goods on the market.

The extent to which some product groups targeted at children and babies are affected is particularly worrisome. Samples found to be contaminated even include articles specifically meant to be placed into the mouths of small children (for example teething rings or children's tooth brushes which had higher PAH levels than adults' tooth brushes).

Looking more closely at testing results, it becomes apparent that high PAH levels are not exclusive to low-priced or no-name products but are also present (perhaps to a somewhat lesser extent) in higher-priced goods, sometimes even sold under reputable brands.[1]

Also, while there appears to be some concentration of PAH contaminations on goods imported from low-cost-labour countries (such as China, among other predominantly Asian countries), the problem does not appear to be limited to products manufactured in any specific geographical area and affects, to some degree, countries with presumed higher production standards as well.[2]

E.1.1.4 Regulatory status quo and need for action

At present, no legally binding limits on PAH content are in effect for most types of consumer products. In toys, PAH concentrations are limited by the European Toy Safety Directive (2009/48/EC) in connection with the CLP Regulation (Reg. (EC) No.1272/2008). For example, the limit for BaP in toys is 100 mg/kg. Furthermore, extender oils containing more than 1 mg/kg of BaP or more than 10 mg/kg of all PAHs combined may not be used in tyres according to Directive 2005/69/EC, amending Directive 76/769/EC. It has been the source of much criticism that this limit is applicable to tyres of, among others, automobiles but not to tyres of toys and other consumer products.[3] As the existing PAH limits on toys are widely regarded as too high to provide a reasonable degree of protection and no other specific provisions exist for consumer products, the regulatory status quo is highly inadequate.

In summary, the available data show an unacceptable baseline situation for European consumers with respect to PAH contaminations in a variety of widely used consumer products. As present provisions in chemicals and consumer protection legislation are insufficient to solve the problem, swift regulatory action is needed in order to better protect consumers, especially children, from long-term adverse health effects of PAHs.

E.1.2 Options for restrictions

E.1.2.1 RMO 1: Authorisation under REACH

In this option, PAHs would be added to the list of substances subject to authorisation (Annex XIV of REACH Regulation 1907/2006/EC). As substances in Annex XIV, PAHs as substances as such

[1] For example, in a review of 18 pairs of children's sneakers, some of the most expensive and well-known brand name products featured "strongly elevated" PAH levels. See ÖKO-TEST (8|2008, pp. 60-66). Kinderturnschuhe: Passt uns gar nicht.

[2] For example, in a review comprising 15 pairs of work gloves, 8 out of 12 products manufactured in China were found to have "strongly elevated" PAH levels. However, "strongly elevated" PAH levels were also present in 1 out of 2 products manufactured in Germany. See ÖKO-TEST (2|2009, pp. 130-135). Arbeitshandschuhe: Hände hoch!

[3] See for example F.A.Z (2010, March 12). Gefährliches Kinderspielzeug soll verschwinden.

or in mixtures would thereafter be banned from use or placing on the market unless a specific authorisation has been granted.

E.1.2.2 RMO 2: Restriction under REACH via Art. 68(1)

In this option, manufacture and import of articles containing PAHs would be restricted by means of amending Annex XVII of REACH in line with the process laid out in Art. 68 (1).

The restriction in this option could, in principle, extend to all types of articles but would likely be limited to the category of consumer products. Details regarding timing and scope (such as transitional periods and specific PAH limits for different kinds of products) of the restriction would be specified in the Annex XVII entry.

E.1.2.3 RMO 3: Restriction under REACH via Art. 68(2)

In this option, manufacture, import and consumer use of articles containing PAHs would be restricted by means of amending Annex XVII of REACH in line with the process laid out in Art. 68(2).

Contrary to RMO 2, which could be initiated by a member state, this option would need to be proposed by the Commission. Compared to Art. 68(1), Art. 68(2) offers a simplified restriction procedure for substances classified as CMR which could be used by consumers as such, in mixtures or in articles. This route appears to be available as the criteria are met.

In analogy to RMO 2, the restriction in this option would extend to all consumer goods (including toys) while its timing and exact scope would be defined in Annex XVII.

E.1.3 Community-wide risk management options other than restriction

E.1.3.1 RMO 4: Restriction under the Toy Safety Directive

As set out in Annex II of the Toy Safety Directive (2009/48/EC), substances classified as CMR of category 1A, 1B or 2 may not be used in toys. This means that PAHs are permissible in toys up to the specific concentration limits included in their classification in Annex VI of the CLP Regulation (Reg. (EC) No. 1272/2008), e. g. 100 mg/kg for BaP. However, in addition to this, Art. 46(2) of the Toy Safety Directive allows for adoption of specific limit values for chemicals used in toys intended for use by children under 36 months or in other toys intended to be placed into the mouth.

In this option, Annex II would be amended in line with the provisions of Art 46(2) by specifying safe PAH limits (lower than those already in place) for toys. The scope of this option would be limited to the types of toys referred to in Art. 46(2).

E.1.3.2 RMO 5: Restriction under the General Product Safety Directive

Art. 13 of the General Product Safety Directive (2001/95/EC) provides that the European Commission may adopt a decision to deal with serious risks from certain products to the health and safety of consumers. Recent examples for this are the decisions on cigarette lighters (Decision 2006/502/EC) and the ban of dimethylfumarate in consumer products (Decision 2009/251/EC). In this option, a decision in accordance with Art. 13 would be adopted to address risks to consumers from PAHs in consumer products.

Regarding scope, this option would likely cover consumer products in general but not toys specifically as the Directive does not apply directly to products and/or risks covered by sector-specific product safety legislation (Art. 13(1) b). Also, this option would extend only to products sold in the EU and not those manufactured in the EU for export. Regarding timing, the decision would be valid for one year only and would have to be confirmed after that period (Art. 13(2)).

E.1.3.3 RMO 6: Voluntary action by industry

In this option, industry would voluntarily reduce PAH levels in consumer products, including toys. Administration of a voluntary industry program, i.e. setting up standards and enforcing compliance of participating companies, would probably be carried out by industry trade groups (possibly with support by government authorities). This option could be combined with a certification scheme where a consumer safety label would be awarded to products meeting certain minimum quality standards, one such criterion being low PAH content (comparable to the GS mark).

Scope and timing of this option would not be pre-defined as such a program would be voluntarily set up by industry and companies would be free to decide whether or not to participate in the program and any optional certification scheme.

E.2 Assessment of risk management options

E.2.1 Overall assessment of RMO 1 (authorisation under REACH)

E.2.1.1 Advantages

RMO 1 would have the effect that, according to REACH, identification of PAHs as SVHCs would trigger certain duties of suppliers to communicate information on PAHs in articles to recipients and consumers (Art. 33(1) and 33(2), respectively). This could lead to higher transparency regarding PAHs in relevant supply chains and on the consumer market.

E.2.1.2 Drawbacks

Firstly, it is uncertain whether PAHs could be successfully identified as Substances of Very High Concern (SVHC) subject to authorisation under REACH. This is because a multitude of substances may be relevant to PAHs as a group, and the chemical characteristics of these substances would need to be explored further to withstand the vigorous prioritisation process involved in adding substances to Annex XIV. Specifically, since PAHs are not produced as substances per se but occur as by-products in various production processes (mainly combustion), it is doubtful whether subjecting PAHs to authorisation would be an appropriate route.

Secondly, assuming PAHs could be identified and prioritised as required, their inclusion in Annex XIV would likely require a considerable amount of time during which risks to consumers associated with the status quo would persist.

Thirdly and most significantly, even after the eventual inclusion of PAHs in Annex XIV, articles (such as toys) containing PAHs could still be legally imported and sold to consumers in the EU as imports of SVHC substances may be subject to authorisation, but not imports of articles containing these substances. Furthermore, articles containing PAHs could still be produced in the EU as PAHs are impurities or by-products and not used as such or in mixtures to produce articles

E.2.2 Overall assessment of RMO 2 (restriction under REACH via Art. 68 (1))

E.2.2.1 Advantages

RMO 2 would be able to address risks arising from PAHs in the most comprehensive fashion as it is based on chemicals legislation (REACH) and therefore not limited to any specific type of articles (i.e., potentially covering both toys and other consumer and non-consumer products as well as covering both imports and goods produced in the EU). Also, an Art. 68 (1) restriction process would possess a high degree of transparency because of involvement of RAC, SEAC, Member States and stakeholders.

E.2.2.2 Drawbacks

RMO 2 has the main drawback that a considerable lead time would likely pass before a restriction would come into effect because of the complex restriction process under REACH. Also, this option has high procedural barriers as sufficient scientific evidence would have to be provided to prove that PAHs pose an unacceptable risk to human health or the environment.

E.2.3 Overall assessment of RMO 3 (restriction under REACH via Art. 68(2))

E.2.3.1 Advantages

RMO 3 would enable the introduction of a thorough and effective restriction for all relevant consumer products, including toys. As PAH related risks clearly concern consumer use of articles containing PAHs, pursuing this option would incur the benefits of the streamlined process afforded by Art. 68 (2) for CMR substances used by consumers. Therefore, it is expected that RMO 3 would lead to a restriction, and thereby to the needed protection of consumers, in a timelier manner than RMO 2.

E.2.3.2 Drawbacks

As RMO 3 would be proposed by the European Commission and member states or industry stakeholders could be involved less directly in the process, transparency could be somewhat reduced in comparison to RMO 2.

E.2.4 Overall assessment of RMO 4 (restriction under the Toy Safety Directive)

E.2.4.1 Advantages

RMO 4 would stipulate more stringent PAH limits for toys and, as a result, lead to better protection of children against health risks.

E.2.4.2 Drawbacks

RMO 4 is applicable not to all toys but only to toys intended for use by children under 36 months or toys intended to be placed in the mouth. Further, consumer products other than toys would not be covered at all by RMO 4. Consequently, this option would only address a relatively small segment of consumer goods and toys potentially contaminated with PAHs.

E.2.5 Overall assessment of RMO 5 (restriction under the General Product Safety Directive)

E.2.5.1 Advantages

RMO 5 could be used to establish legally binding and sanctionable requirements regarding PAH levels in consumer goods sold in the EU.

E.2.5.2 Drawbacks

As RMO 5 is limited to consumer products that are not covered by specific sector legislation, it is probable that toys would not be covered by RMO 5 but would have to be addressed separately, adding regulatory complexity. Also, the limited validity of RMO 5 for a time period of one year would necessitate further action in the near future in order to ensure a long-term solution regarding PAHs in consumer products. Lastly, in this option, PAH-contaminated goods meant for export could still be produced in the EU, thus leaving workers in the EU involved in the production of export goods unprotected.

E.2.6 Overall assessment of RMO 6 (Voluntary action by industry)

E.2.6.1 Advantages

RMO 6, especially when accompanied by a certification scheme, could improve market transparency with respect to PAH levels in consumer goods. This might strengthen incentives for importers and manufacturers to observe lower limits on a voluntary basis. Moreover, regulatory costs would be low in this option as little or no action by legislators or authorities would be required initially.

E.2.6.2 Drawbacks

Effectiveness of RMO 6 is in heavy doubt as voluntary agreements and certification schemes already exist within the EU (either applying to consumer goods in general or to specific sectors such as textiles) but have failed to solve the problem of PAHs in a satisfactory manner.[1] Also, setting up, monitoring, and enforcing voluntary standards could lead to considerable administrative costs which would ultimately burden companies and industry associations participating in such a program or running it. For imported goods, it appears questionable whether voluntary action agreed by European producers would have any impact on importers and producers outside the EU market.

[1] Examples of existing voluntary schemes which define limits and test for PAH concentrations in consumer products include the before mentioned GS mark, VDE certification marks, the EU Eco-Label and Oeko-Tex Standard 100.

E.3 Comparison of the risk management options

Table 55 compares the six risk management options identified in section E.1.2 using the criteria of effectiveness and efficiency. An RMO is denoted as effective if it will likely lead to a significant reduction of human health risks from PAH-contaminated consumer goods. An RMO is denoted as efficient if its regulatory and legislative costs are expected to be proportionate to its human health benefits.

Table 55: Comparison of different risk management options

Option	Effectiveness	Efficiency
RMO 1 (Authorisation under REACH)	**Low:** Not fully effective for goods produced in EU and ineffective for imported articles. Some incentives by duty to communicate information	**Average:** Complex and time-consuming process of subjecting substances to authorisation under REACH.
RMO 2 (Restriction under REACH via Art. 68(1); common approach for all target groups)	**High:** Comprehensive restriction of PAHs in all affected goods (applies to toys and other consumer products, covers EU production and imports)	**Average:** Complex and time-consuming process of introducing restriction under REACH.
RMO 3 (Restriction under REACH via Art. 68(2) ; direct approach for CMR substances in consumer products)	**High:** Similar to RMO 2, comprehensive restriction of PAHs in consumer products (including toys), whether produced in EU or impo rted.	**High:** Simplified and more expeditious regulatory process than RMO 2.
RMO 4 (Restriction under Toy Safety Directive)	**Average:** Effective for certain toys (intended for children up to 36 months or intended to be placed into mouth), ineffective for other toys and other consumer products.	**Average:** Regulatory process less complex than RMO 1 or RMO 2, comparable to RMO 3.
RMO 5 (Restriction under General Product Safety Directive)	**Average:** Effective only for products not covered by specific sector legislation, therefore likely not applicable to toys. Valid only for one year.	**Low:** Initially, similar to RMO 4. However, restriction must be renewed due to time limitation.
RMO 6 (Voluntary action by industry)	**Low:** Some positive incentives for industry to reduce PAH levels but commitment of all relevant companies unlikely.	**Low:** Significant administrative costs for setting up, enforcing and monitoring voluntary program.

E.4 Main assumptions used and decisions made during analysis

With regard to the baseline scenario, it is assumed that the present situation would not improve considerably if no regulatory action were taken. For a number of years, a fair amount of public attention has been drawn to the PAH problem through multiple reports in news media. Some industry representatives have stated that, as a result, many retailers have installed stricter voluntary policies, forcing suppliers and manufacturers to observe lower PAH limits than required by law. They argue that for this reason, occurrence of contaminated consumer goods has already decreased dramatically or is about to do so in the near future. While it is likely that companies marketing goods to consumers will undertake some effort in this direction to avoid negative publicity, recently published reports and product tests suggest that the situation has not improved sufficiently to warrant delaying legislative action.

E.5 The proposed restriction(s) and summary of the justifications

Based on the comparison of risk management options given in section E.3, RMO 6 (voluntary action by industry) should be eliminated because of its low effectiveness in dealing with human health risks to consumers arising from PAHs.

RMO 1, RMO 2, and RMO 5 would be partially effective, but all suffer from notable drawbacks. RMO 1 (authorisation under REACH) would fail to cover imports. RMO 4 (restriction under the Toy Safety Directive) is limited to a small percentage of toys and would therefore not impact on a large portion of potentially PAH-contaminated consumer products on the market. Similarly, RMO 5 (restriction under the General Product Safety Directive) is restricted in scope, applying by design only to consumer products not covered by sector-specific legislation. This would probably leave out toys, which is unacceptable given the particularly urgent need for better protection of children. It might be possible to pursue RMO 4 and RMO 5 concurrently; this option would however add unnecessary regulatory complexity and would still have the disadvantage of the limited period of validity (one year) associated with RMO 5.

RMO 2 (restriction under REACH via Art. 68 (1)) and RMO 3 (restriction under REACH via Art. 68 (2)) remain as the only options which would address health risks to consumers in a satisfactory manner. Between the two, RMO 3 is regarded as superior because of its simplified legislative process designed to allow for a more expeditious route to deal with hazards to consumers. In summary, RMO 3 clearly emerges as the most favorable option and is therefore the basis of the proposed restriction.

F. Socio-economic assessment of the proposed restriction

F.1 Human health and environmental impacts

F.1.1 Human health impacts

F.1.1.1 Impacts on consumers

With regard to the wide range of relevant products and the high content of PAH constituents that has been found in some products, it is expected that the proposed restriction will reduce PAH-related risks to consumers substantially, which would lead to significant benefits to human health. Because the exact number of affected products and affected consumers as well as the length and frequency of exposure of consumers to these products cannot be determined with certainty, health-related benefits cannot be quantified. However, particularly where risks of illness and premature death due to cancer as a result of dermal exposure of consumers to PAH-contaminated materials are concerned, the proposed restriction would have substantial long-term health benefits.

The proposed restriction would have a particularly positive impact with regard to health-related risks of PAHs to children. Children may handle PAH-contaminated toys for prolonged periods of time, playing an average of 15,000 hours up to the age of 6.[1] Also, children may place toys and other products into their mouths, implying a possible oral exposure in addition to the dermal exposure relevant in most cases to consumers. Taking into account the high susceptibility of children to PAH-exposure via contaminated toys and other consumer products, the human health benefits of the proposed restriction would apply in large part to a highly sensitive group with a particular need for protection. In this regard, the proposed restriction, when compared to other risk management options, has the added benefit of applying not only to toys per se but to other products, which are not intended as toys but could be used by children as such.

F.1.1.2 Impacts on workers

In addition to consumers, the proposed restriction would have health-related benefits to workers involved in the manufacturing of consumer goods featuring potentially PAH-contaminated materials. This effect primarily concerns workers producing or assembling products (for example, combining several plastic parts to form a tool or a toy) but also, to some degree, workers employed in upstream or downstream parts of relevant supply chains (such as manufacturers of raw materials or retailers, respectively). It should be noted that workers could be exposed to PAH-contaminated parts of consumer goods for prolonged periods of time where consumers would, under foreseeable conditions, be exposed either only for shorter periods or not at all (examples could be tyres of a garden cart or internal or non-accessible parts of articles). While these effects cannot be quantified, the proposed restriction would clearly reduce PAH-related risks to workers' health resulting from handling potentially PAH-contaminated goods in the workplace.

F.1.2 Environmental impacts

Not relevant for this proposal.

[1] BfR (2009) BfR Opinion No 051/2009: Polycyclic aromatic hydrocarbons (PAHs) in toys, p. 1. (http://www.bfr.bund.de/cm/230/polycyclic_aromatic_hydrocarbons_pahs_in_toys.pdf)

F.2 Economic impacts

F.2.1 Impacts on manufacturers of technical rubber products

One group which would be affected economically by the proposed restriction is formed by manufacturers of technical rubber products. Sealings and tubes used in automobiles or sanitary facilities and cables and plugs used as electrical components are examples of potentially PAH-contaminated technical rubber products that could pose a threat to consumers.

F.2.1.1 Costs

Costs of the proposed restriction to manufacturers of technical rubber products would, for the most part, arise from increased costs of raw materials. Varying from article to article, costs of raw materials relative to the total costs of a product such as a sealing could typically be 30 to 40 percent. According to information received from a consulted manufacturer, costs for low-PAH alternatives (in particular, special carbon blacks and white oils) may be approximately 1.3 to 1.5 times higher than traditional raw materials and intermediates:[1]

Table 56: Additional costs and availability of substitutes for traditional extender oils and carbon black

Original substance	Substitute	Approximate change in cost	Availability
Traditional carbon blacks	Special carbon blacks	Higher by factor of 1.5	Relatively new to market, improving availability
Traditional extender oils	Low-PAH extender oils (e.g. white oils)	Higher by factor of 1.3	Multiple alternatives widely available

Further costs to manufacturers of technical rubber could result from increased storage costs of raw materials. If manufacturers were to employ low-PAH alternatives next to existing substances, these would need to be stored separately. To that end, manufacturers might have to reserve or set up additional silos (tanks) which could result in significant one-off investment costs as well as higher running costs to companies.

The market for technical rubber products comprises a multitude of different articles (often of relatively low value) for different applications. Therefore, this market segment is fragmented and populated by a host of specialised vendors. Furthermore, it is difficult to determine the relevance of this segment to the category of consumer goods because it is unclear which specific articles consumers come into direct contact with. For these reasons, the impacts of the proposed restriction of PAHs in consumer products on manufacturers of technical rubber products cannot be quantified or monetised in a robust way.

Where manufacturers of technical rubber products act as suppliers to mature industries (for example automotive and plant building), there is generally a high degree of competition both on a European and on a global level and, to some degree, excess capacity among suppliers. Therefore it is to be

[1] These cost ratios of low-PAH alternatives to traditional materials should be seen as a general estimate only and may vary by specific substances, applications and by market fluctuations. According to one study, for example, the costs of mineral oils used in textile processing with a content of less than 1.0% by weight are three three times higher than those of conventional oils. See ASQUAL, IFTH (2007). Revision of the Textile Eco-label: Draft Final Report, p.54. (http://ec.europa.eu/environment/ecolabel/ecolabelled_products/categories/pdf/rapport_2007.pdf)

expected that suppliers will not be able to pass on a large portion of the above mentioned cost increases for raw materials and storage to downstream companies.

F.2.1.2 Benefits

The proposed restriction would benefit manufacturers of technical rubber products which already employ advanced production materials and processes that lead to lower PAH concentrations. These companies' competitive situation would improve as competitors could no longer gain a cost advantage from lower production standards. This can be expected to translate into a net positive effect on the competitiveness and innovation of quality-oriented European companies.

F.2.2 Impacts on manufacturers of consumer goods

Manufacturers of a wide array of consumer goods form a second group likely to be impacted economically by the proposed restriction. Judging by PAH-contaminated products found in tests, manufacturers of toys, tools and shoes would be among those most affected.

F.2.2.1 Costs

As a result of the proposed restriction, manufacturers of consumer goods would be forced to reduce PAH concentrations in articles to meet the prescribed limits. In many cases, manufacturers may already observe similar limits to conform to distributors' and retailers' requirements, certification schemes or voluntary standards. Where this is not the case, manufacturers would likely face some increase in costs for improved production processes or alternative raw materials, in the order of the figures presented in section F.2.1. It is also conceivable that manufacturers would replace plastic or rubber surfaces in some products with different materials altogether, thereby making the use of more costly low-PAH carbon blacks and extender oils unnecessary (for example, hammers can be equipped with wooden handles instead of rubber handles).

The available market data is insufficient for a precise valuation of the total costs to manufacturers of consumer goods associated with substitution of high-PAH materials. However, industry insiders generally predict any cost increases to be relatively minor in relation to the total value of the affected consumer goods. One reason for this is the fact that parts contaminated with PAHs usually constitute only a relatively small and low-value portion of a larger article. For example, handlebar grips which are commonly made of high-PAH materials will in all likelihood represent less than 1 percent of the total value of a complete bicycle.

Other evidence seems to substantiate the conclusion that the costs of the proposed restriction to manufacturers of consumer goods would be limited. The available data shows that even in product groups where high PAH concentrations are widespread (for example plastic sandals or plastic toy figures), there are normally at least some product options with acceptable PAH concentrations which offer comparable functionality at similar prices to consumers. In addition, contrary to what might be intuitively expected, low PAH levels do not seem to be limited to high-priced articles and vice versa (i.e., a relatively inexpensive tool may have a low PAH concentration whereas a premium tool may be contaminated).[1]

[1] For example, in a review of 11 power drills, no PAHs were found in the handles of the two least expensive products whereas "elevated" and "strongly elevated" PAH levels were present in far higher priced products. See ÖKO-TEST (8|2008, pp. 148-155). Bohrmaschinen: Ganz schön durchgedreht.

F.2.2.2 Benefits

At the moment, there is a lack of consistent and transparent EU-wide requirements regarding PAHs in consumer goods. This leads to a situation which is characterized by a large degree of uncertainty among manufacturers as to which limits (limits of voluntary certification schemes, requirements set by retailers etc.) should be observed and how these limits apply to specific articles (e.g. do weight ratios apply to parts of an article or to the whole article?). The proposed restriction would benefit manufacturers by reducing these uncertainties and setting clear rules regarding what PAH levels are acceptable.

Additionally, under the proposed restriction, companies in Europe voluntarily observing higher consumer safety standards would be better protected against unfair competition from low-quality overseas production as highly PAH-contaminated imports would be banned.

F.2.3 Impacts on importers, distributors and retailers of consumer goods

Besides manufacturers, the proposed restriction would impact companies which buy consumer goods in bulk (either from European or, in the case of importers, from overseas manufacturers) in order to place them on the European market. Importers, distributors and retailers dealing with toys, tools, shoes, and other affected products fall into this category.

F.2.3.1 Costs

The proposed restriction would prohibit the import and placing on the market of consumer goods with PAH levels above the prescribed limits. According to trade groups, many retailers already employ stricter policies on PAHs than required by law. These companies would not face any additional costs as a result of the proposed restriction.

Where this is not the case, importers, distributors, and retailers would need to communicate to their suppliers modified product specifications reflecting lower maximum PAH levels as required by the restriction. For reasons stated above, it is estimated that for most consumer goods, observing lower limits is possible for suppliers of consumer goods and associated with relatively little extra costs.

The worst case scenario would be suppliers being unable to reduce PAH concentrations or charging a high price premium for higher quality specifications. This would affect importers, distributors and retailers in so far as those goods would then have to be offered at significantly higher prices to consumers, leading to costs in the form of reduced turnover and profits. However, consultations have not revealed any areas where such a worst case scenario is to be expected so that these potential costs cannot be quantified.

Significant costs to importers, distributors and retailers would arise if placing on the market of goods with certain PAH levels were to be immediately banned following a decision on the proposed restriction. This would lead to companies being unable to sell goods already imported or stocked, resulting in possibly large write-offs on inventory. This problem could be cured by an appropriate transitional period during which companies would be allowed to sell off inventory.

F.2.3.2 Benefits

The proposed restriction would provide some benefit to importers, distributors, and retailers by setting clear and unambiguous standards on PAH levels. For retailers, this would reduce or

eliminate the need to introduce and communicate to suppliers voluntary policies on PAHs, thereby lowering coordination and administration costs. In the long run, responsible importers, distributors and retailers of consumer goods can be expected to benefit from a, in comparison to the status quo, more level playing field and more transparent marketplace afforded by the proposed restriction.

F.2.4 Impacts on manufacturers of raw materials and intermediates

As carbon blacks and extender oils are the main source PAHs in consumer products, the proposed restriction would to some extent affect manufacturers of these materials as suppliers to makers of various consumer goods.

F.2.4.1 Costs

Representatives of manufacturers in Germany and Europe have stated that, to their knowledge, highly PAH-contaminated carbon blacks and extender oils used in consumer goods originate not from European production but from imports of overseas production. Some European manufacturers have also released press statements indicating they no longer produce or use high-PAH oils.[1] These claims cannot be verified due to a lack of independent data but would imply that the majority of manufacturers of carbon blacks and extender oils located in Europe would not face any significant costs resulting from a restriction of PAHs in consumer products.

F.2.4.2 Benefits

It is to be expected that, concerning the relevant supply chains for consumer goods, the proposed restriction will lead to more demand for innovative low-PAH carbon blacks and extender oils and less demand for traditional high-PAH raw materials and intermediates. However, as the exact share of the total production of these substances used in applications relevant to consumer goods is unknown, it is not possible to predict whether the proposed restriction will impact total demand of carbon blacks and extender oils in a significant way.

F.2.5 Impacts on consumers

Economic impacts of the proposed restriction on consumers purchasing and using a wide array of consumer products could include changes in availability, functionality, and prices of goods as well as changes in the transparency of consumer markets.

F.2.5.1 Costs

As the proposed restriction would require manufacturers of consumer goods to abandon materials with very high PAH concentrations, one possible effect could be reduced availability of certain products to consumers. This effect is unlikely to materialise in a noticeable way because in nearly all affected product groups, a percentage (differing between product groups) of products already displays low PAH content. Specific products disappearing from the market altogether are unlikely but conceivable in a very limited number of cases (such as small and very low-value plastic toys typically offered in bargain bins and discount stores).

[1] See for example LANXESS (2009). LANXESS to stop using oils containing high levels of PAH worldwide: Safe ingredients for improved rubber formulations. (http://corporate.lanxess.com/en/media/press-releases/detail/1871/)

Next, the required substitution of materials could potentially lead to costs to consumers in terms of reduced functionality of products. Some manufacturers have stated that high-PAH materials (like certain carbon blacks) are necessary for the safe and sustained functioning of thermally and mechanically stressed parts and that substitution is of little or no benefit in internal parts hidden from end users. To account for the latter point, the proposed restriction is limited to parts of consumer articles to which direct (dermal or oral) contact is foreseeable. Concerns regarding functionality, while potentially relevant to some technically advanced products, seem to be directed at a relatively small portion of consumer goods as PAH contaminations are, in most cases, present in rubber grips and surfaces which serve no technically demanding purposes. With respect to the limited number of consumer goods which pose high functional demands on rubber materials, experiences from the substitution of extender oils in (automotive) tyres in the wake of Directive 2005/69/EC appear to suggest that suitable low-PAH alternatives can be used at reasonable costs even in applications where high-performance rubber compounds are needed.[1]

A third potential source of costs of the proposed restriction for consumers would be increases in retail prices. In general, some price increases can be expected for products which currently feature PAH-contaminated materials that would need to be substituted with higher-cost alternatives as a result of the restriction. For reasons explained above and taking into account the generally high downward price pressures in the consumer retail space, these increases are expected to be rather low although no reliable quantitative estimate can be given based on the available data. Further, some experts contend that any price increases would primarily concern imported no-name products at the low end of the market which would, given a possible moderate price hike, still retail at price points far below comparable brand-name products following a restriction of PAHs (for example, the cost of a no-name hammer imported from overseas might climb from 2 Euro to a still relatively inexpensive 2.50 Euro).

F.2.5.2 Benefits

At present, consumers have, for the most part, no means available to them to assess whether a given product has a worryingly high PAH content and are therefore unable to make informed buying decisions. By setting clear mandatory minimum standards, the proposed restriction would contribute to a more transparent market. This would benefit consumers who would need to exert less time and effort researching and comparing buying options in order to avoid PAH-contaminated goods.

F.3 Social impacts

Not relevant for this proposal

F.4 Wider economic impacts

Not relevant for this proposal

[1] See for example Rubber World (2007, January 1). Non-carcinogenic tire extender oils providing good dynamic performance.

F.5 Distributional impacts

Regarding the distribution of the identified impacts among consumers and industry, the proposed restriction's positive effects are primarily human health-related, benefitting primarily consumers and secondarily workers. In contrast, a large part of the restriction's economic costs would likely be borne by industry as manufacturers' ability to charge higher prices following the substitution of high-PAH materials is expected to be limited owing to pricing pressures of the market. Children are believed to benefit in particular from the improved protection against PAH-contaminated consumer products that will be achieved with the restriction.

Geographically, no major distributional impacts are expected within the EU. It should be noted, however, that a considerable part of the costs and benefits of the proposed restriction will occur in countries outside of the EU. Specifically, as high-PAH products would not be allowed to be imported into the EU subsequent to the proposed restriction, manufacturers in exporting countries would need to substitute high-PAH materials in order to continue exports, thereby experiencing some economic costs. Based on the assumption that a relatively high percentage of PAH-contaminated consumer goods is imported from overseas where production quality is partly below EU standards, a notable portion of the proposed restriction's costs may be expected to fall onto companies outside of the EU.

Finally, the proposed restriction's benefits and costs would be somewhat unevenly distributed throughout time. A significant portion of costs to industry associated with the necessary substitution of high-PAH materials (such as one-time costs for redesigning of products and modification of production processes) would be incurred in the near term following the restriction. In contrast, health benefits to consumers, mainly in the form of reduced risks of cancer, in connection with the carcinogenic properties of PAHs would accrue over the long term.

F.6 Main assumptions used and decisions made during analysis

Due to the presence of PAHs in a vast variety of materials and products, it is not possible to identify all affected products, product groups and manufacturers, thereby rendering a robust monetary valuation of the economic impacts of the proposed restriction unfeasible. Similarly, due to the long-term nature and the wide dispersion of the proposed restriction's human health impacts, benefits to consumers cannot be quantified or monetised in a reliable way. For these reasons, the analysis focuses on a qualitative presentation and assessment of the socio-economic impacts of the proposed restriction. Thus, no advanced economic valuation or discounting methods requiring specific methodological choices were used in the analysis.

As the analysis was prepared by the REACH Competent Authority of Germany, much of the data obtained from authorities and stakeholders may reflect more closely the situation in Germany than that in other member states. Nonetheless, based on feedback from and experiences in other Member States[1] and taking into account the free movement of goods within the EU, it is assumed that consumers in virtually all member states are equally affected by the identified risks with regard to PAHs in consumer products.

[1] Cf. for example BM für Land- und Forstwirtschaft, Umwelt und Wasserwirtschaft (2009). Schriftl. parl. Anfr. d. Abg. z. NR Mag. Johann Maier, Kolleginnen und Kollegen vom 10. Juli 2009, Nr. 2786/J, betreffend Krebsgefahr durch Polyzyklische aromatische Kohlenwasserstoffe in Konsumgütern?. (http://www.parlament.gv.at/PG/DE/XXIV/AB/AB_02665/fnameorig_167152.html)

F.7 Uncertainties

While there is wide agreement among experts regarding health hazards of PAHs, some scientific uncertainty remains in terms of toxicology. For example, incidence of cancer and cancer related deaths has risen among children in recent years.[1] Although many PAHs are classified as CMR and migration is likely to take place in humans handling consumer products, there is little concrete evidence of a direct causal link between PAHs and cancer in children and other persons. Also, there have been some questions whether extraction methods commonly used in analytical testing (especially for PAHs in carbon blacks) are appropriate to study risks from dermal exposure of consumers to PAHs in various products under realistic circumstances.[2]

Secondly, some uncertainty may arise from concerns whether testing results of samples can be interpreted as representative of the whole market. Because PAHs may potentially affect an extremely large number of consumer products, comprehensive testing for PAHs is practically impossible. Where results of single or multiple samples for a given product group (for example hand tools) are available, it is often unclear whether products were randomly selected for testing or whether they were selected specifically for some reason (for example, a high PAH concentration was already suspected before testing). The latter would obviously undermine the representativity of samples with respect to PAH levels in the consumer market as a whole.

On a similar note, it is doubtful whether a PAH-contaminated sample of a given product is a reliable indicator that all identical articles of the same model and manufacturer are equally contaminated. One industry representative noted that manufacturing irregularities at production facilities could lead to higher contamination of some articles or charges of articles despite production specifications prescribing lower PAHs levels. It is uncertain whether such a variance of PAH levels in identical products exists to a significant degree, how it would evolve if no regulatory action were taken and whether it would persist under the proposed restriction.

Lastly, there is some uncertainty as to how the proposed restriction's economic costs would be affected by incremental variations of the envisaged regulatory limits on PAH concentrations in consumer goods. It can be inferred from analytical results of actual product samples (cf. Appendix 3) and statements of industry stakeholders that, for the vast majority of consumer goods, products with acceptable PAH levels are either already available on the market or possible at reasonable costs. Still, some experts have suggested that, while limits in line with existing voluntary standards (namely the GS mark) are reasonable, significantly lower limits would lead to disproportionate costs to some manufacturers. It has also been contended by industry that completely PAH-free products are not technically realistic in some cases where PAHs emerge unavoidably in production processes (vulcanisation of tyres, for example)[3]. Presently available independent data is insufficient to assess definitively whether a specific regulatory PAH limit would be feasible and proportionate without exception when applied to all types of consumer goods. Therefore, derogations of PAH limits for a limited number of products or product groups might be appropriate pending further information and analysis.

---

[1] BfR (2009) BfR Opinion No 051/2009: Polycyclic aromatic hydrocarbons (PAHs) in toys, p. 1. (http://www.bfr.bund.de/cm/230/polycyclic_aromatic_hydrocarbons_pahs_in_toys.pdf)

[2] See for example Evonik, ICBA (2009). Statement regarding the presence of Polycyclic Aromatic Hydrocarbons (PAH) in Carbon Black. (http://kraiburg-belmondo.de/belmondo/wp-content/uploads/pak/icba_pah_statement_05_09.pdf)

[3] See for example Ralf Bohle GmbH (2008). Keine Gefährdung durch Fahrrad-Reifen. (http://www.puky.de/media/Stellungnahme_Reifenhersteller.pdf)

F.8 Summary of the socio-economic impacts

The proposed restriction's main impacts are economic and human health-related. Economic costs may arise from the substitution of high-PAH materials in consumer goods with somewhat more expensive (in the order of a 1.3- to 1.5-fold cost increase) alternatives such as special carbon blacks and white oils. This burden would have to be shared between industry (in the form of economic losses from higher costs of raw materials and perhaps slightly lower quantities sold for some goods) and consumers (in the form of potentially higher retail prices of consumer products). Economic benefits of the proposed restriction include improved transparency and reduced uncertainty in consumer markets which would benefit innovative European manufacturers observing higher quality and safety standards than low-cost, low-quality overseas manufacturers exporting goods to the EU.

The proposed restriction's primary human health benefit would be greatly reduced risks to consumers from exposure to high concentrations of PAHs which are worryingly wide-spread in the baseline situation. As this affects a vast range of products handled regularly in day-to-day use by large numbers of consumers, and risks associated with PAHs include potentially severe cancer-related illness or death, the proposed restriction would be highly beneficial to consumers throughout the EU. In particular, it addresses the urgent need for better protection of children who are, for behavioural and biological reasons, particularly vulnerable to the risks of PAH-contaminated toys. The reduced risk from handling PAH-contaminated materials to workers involved in the production of consumer goods can be seen as a secondary human health benefit of the proposed restriction.

The proposed restriction's human health benefits in combination with its economic benefits are found to clearly outweigh its economic costs. Thus, the restriction is considered to be proportionate with respect to its total socio-economic impacts.

G. Stakeholder consultation

G.1 Consultation of Member States

The consultation was conducted in order to gather additional information about specific products and exposures regarding PAH in consumer products and to contribute transparency and representativeness of the restriction proposal. Therefore, a consultation/background paper and a field survey document were circulated to MS via CIRCA (see Appendix 6 and 7).

The documents were passed on December 21, 2009 and feedback was sought until January 31, 2010. Responses were received from 6 MS (Denmark, Sweden, Ireland, Austria, Finland, the Netherlands), what represented a 22 % response rate. Notwithstanding this response rate, MS who responded provided some significant information and insights.

Some of the key themes emerging from MS responses:

G.1.1 General agreement

Almost all respondents agreed on a need of action in respect of PAH in consumer products and the fact that child protection in terms of PAHs is currently insufficient.

G.1.2 Need for discussion

Considerable support was expressed for the proposed restriction of PAHs in consumer products via article 68 (2) of the REACH-Regulation. However, some stakeholders expressed concern about the relevance of PAH uptake from consumer products in terms of negative health effects compared to all global PAH sources that influence peoples daily life (polluted ambient air, polluted indoor air, tobacco smoking, etc.). Besides that, the question was raised whether the “normal” restriction procedure outlined in Article 68 (1), which would include consultation of RAC, SEAC, and the forum, would be the more appropriate/transparent way to regulate PAHs in a broader range in order to expand the project's scope.

G.1.3 Current state/national measures

Different situations in EU countries exist. Some countries do not have any provisions concerning PAHs in general, while others do have voluntary measures or legal regulations for some specific scopes.

G.1.4 Identified products burdened by PAH

Paints, tools, toys, discuses, etc. (cf. section B.9.3).

G.2 Consultation of Industry and NGOs

In addition to the MS consultation, 11 designated German NGOs, companies, industry associations were contacted by mail (see Appendices 6 and 7). As for the MS consultation, the documents were passed on December 21, 2009 and feedback was sought until January 31, 2010. All of them did reply with the exception that 2 companies did not answer the questionnaire themselves but referred to the 'Wirtschaftsverband der deutschen Kautschukindustrie' (association of the German cautchouk industry), as they are members of this association.

In January 2010, 6 German NGOs/companies/industry associations and one Federal Institute were accessorily contacted by mail (see Appendix 6) in order to pass the questionnaire. Again, all of them answered to our query.

Some of the key themes emerging from German NGOs, companies, industry associations responses:

G.2.1 General agreement

Most of the consultation participants pointed out that PAH-burdened consumer products are mainly imported products from outside the EU.

G.2.2 Need for discussion

Views varied about the usefulness of the proposed restriction. In this context, the question was raised which method can be applied to evaluate the amount of PAHs in consumer products and the migration rate of PAHs from consumer products.

Finally, some of the already consulted German NGOs, companies, and industry associations were contacted once more in February 2010 by telephone to substantiate and confirm some comments and notations.

H. Other information

None available

Abbreviations

Abbreviation	Explanation
3-OHBaP	3-Hydroxybenzo[a]pyrene
ADME	Absorption, Distribution, Metabolism, and Excretion
AF	Assessment factor
AIC	Akaike Information Criterion
ALARA	As Low As Reasonably Achievable
APUG	Aktionsprogramm Umwelt und Gesundheit
Art.	Article
AS	Allometric scaling
ATSDR	US Agency for Toxic Substances and Disease Registry
AUH	Ausschuss für Umwelthygiene
BA	Bioavailability
BaA	Benzo[a]anthracene
BaP	Benzo[a]pyrene
BAuA	Bundesanstalt für Arbeitsschutz und Arbeitsmedizin
BbFA	Benzo[b]fluoranthene
BCF	Bioconcentration factor
BeP	Benzo[e]pyrene
BeP	Benzo[e]pyrene
BfR	Bundesinstitut für Risikobewertung / German Federal Institute for Risk Assessment
BghiP	Benzo[g,h,i]perylene
BjFA	Benzo[j]fluoranthene
BkFA	Benzo[k]fluoranthene
BMD	Benchmark dose
BMD10	Benchmark dose for the effect in 10 % of a given population
BMDL10	Lower confidence limit of the benchmark dose for the effect in 10 % of a given population
BMDS	Benchmark dose software
BPD	Biocidal Products Directive
BSM	Benzene soluble matter
CA	Competent Authority (of an MS under REACH)
CAR	Competent Authority Report (under the EU Biocidal Products Directive 98/8/EC)
CAS	Chemical Abstracts System
CHR	Chrysene
CHRIP	Chemical Risk Information Platform
CI	Confidence Interval

Abbreviation	Explanation
CIRCA	Communication & Information Resource Centre Administrator (collaborative internet workspace of EU and MS authorities)
CLP	Regulation (EC) 1272/2008 on the classification, Labelling, and Packaging of Chemicals
CMR	Carcinogenic, Mutagenic, and/or toxic to Reproduction
CNS	Central Nervous System
CSA	Chemical safety assessment (under REACH)
CTM	Coal tar mixture
CTP	Coal tar pitch
CTPHT	Coal tar pitch high temperature
Ctrl	Control
CYP	Cytochrome P enzyme
DAE	Distilled aromatic extracts
DBAhA	Dibenzo(a,h)anthracene
DIMDI	Deutsches Institut für Medizinische Dokumentation und Information
DMEL	Derived Minimum Effect Level
DNA	Desoxyribonucleic acid
DNEL	Derived No-Effect Level
DSD	Dangerous Substances Directive
EC	European Communities
EC/HC	Health Canada / Environment Canada
ECETOC TRA	European centre for ecotoxicology and toxicology of chemicals …
ECHA	European Chemicals Agency
EEC	European Economic Community
EFSA	European Food Safety Authority
EINECS	European Inventory of Existing Chemical Substances
ELS	Early life stage
EN	European Norm / Europäische Normen
EN ISO	European Norm
EPA-PAHs	16 PAHs selected by the United States Environmental Agency to characterise environmental PAH pollution
ETRMA	European Tyre & Rubber Manufacturer Association
EU	European Union
EU-PAHs	8 PAHs listed in entry 50 of annex XVII to Reg. (EC) 1907/2006
F	Female
F.A.Z.	Frankfurter Allgemeine Zeitung
FA	Fluoranthene
FAO/WHO	Food and Agriculture Organization of the United Nations / World Health Organization
FEHD	Food and Environmental Hygiene Department (of the Government of Hong Kong)

Abbreviation	Explanation
FhI	Fraunhofer Institute of Toxicology and Aerosol Research
GC-MSD	Gas chromatography with Mass-Selective Detection
GI	Gastro-intestinal
GLP	Good Laboratory Practice
GS	Geprüfte Sicherheit
HBMD10	Benchmark dose for the effect in 10 % of a given population, corrected for differences between the animal experiment and the human exposure situation
HBMDL10	Lower confidence limit of the benchmark dose for the effect in 10 % of a given population, corrected for differences between the animal experiment and the human exposure situation
HCN	Health Council of the Netherlands
HSE	UK Health and Safety Executive
HT25	Dose level corresponding to a net increase in tumour incidence as compared to untreated controls, corrected for differences between the animal experiment and the human exposure situation
IARC	International Agency for Research on Cancer
ICBA	International Carbon Black Association
IFTH	Institut Français du Textile et de l'Habillement
IP	Indenopyrene
IPCS	International Programme on Chemical Safety
IR	Information requirements (section of the REACH guidance)
IR / CSA	Information Requirements and Chemical Safety Assessment
ISI	Institute for Scientific Information
IUCLID	International Uniform Chemical Information Database
IUPAC	International Union of Pure and Applied Chemistry
JECFA	Joint (FAO/WHO) Expert Committee on Food Additives
LOD	Limit of detection
LOQ	Limit of quantitation
M	Male
MES	mild extract solvates
MGP	Manufactured Gas Plant Residue
MITI	Japanese Ministry of International Trade and Industry
MMAD	Mass Median Aerodynamic Diameter
MS	Member State (of the European Union)
NGO	Non-governmental organisation
NITE	Japanese National Institute of Technology and Evaluation
OC	Operational Condition
OECD TG	Organisation for Economic Cooperation and Development …
OEHHA	Office of Environmental Health Hazard Assessment (of the Canadian Government)
PAH	Polycyclic Aromatic Hydrocarbon
PAH-6	6 PAHs common to both the EPA-PAH and EU-PAH lists

Abbreviation	Explanation
PBT	Persistent, Bioaccumulative, and Toxic
PCA	Principal Component Analysis
Pgp	p-Glycoprotein
PVC	Polyvinylchlorid
PY	Pyrene
R	Risk phrases
RAC	Risk Assessment Committee (of the European Chemicals Agency)
RAE	Residual aromatic extracts
RAPEX	Rapid Exchange of Information System
RAR	Risk Assessment Report
RC	Risk Characterisation
RCR	Risk Characterisation Ratio
REACH	Regulation (EC) 1906/2007 on the Registration, Evaluation, Authorisation and restriction of Chemicals
RIVM	Rijksinstitut voor Volksgezondehit en Milieu (Dutch National Institute for Pubic Health and the Environment
RMM	Risk Management Measure
RMO	Risk Management Option
RMS	Rapporteur Member State
SBR	Styrene-Butadiene Rubber
SCTEE	Scientific Committee on Toxicity, Ecotoxicity and the Environment of the European Commission
SEAC	Socio-Economic Analysis Committee (of the European Chemicals Agency)
SIM	Single Ion Monitoring (a GC-MSD mode)
STP	Sewage Treatment Plant
SVHC	Substances of Very High Concern
T25	Dose level corresponding to a net increase in tumour incidence as compared to untreated controls
TDAE	Treated distilled aromatic extracts
TEF	Toxicity Equivalence Factor
TOXNET	Toxicology Data Network
TPA	thermoplastic polyamides
TPC	thermoplastic copolyester
TPE	Thermoplastic elastomers
TPO	Thermoplastic olefins
TPS	Styrenic block copolymer
TPU	Thermoplastic polyurethanes
TPV	Thermoplastic vulcanisates
TRAE	Treated Residual Aromatic Extracts
TTC	Threshold of Toxicological Concern

Abbreviation	Explanation
TÜV	Technischer Überwachungs-Verein
UBA	Umweltbundesamt
UK	United Kingdom
URR	Unit Relative Risk
US EPA	US Environmental Protection Agency
UV	ultraviolet radiation
UVCB	(Substance of) Unknown or Variable Compüosition or Biological Origin
UVEK	Eidgenössische Departement für Umwelt, Verkehr, Energie und Kommunikation
vB	Very Bioaccumulative
VDE	Verband der Elektrotechnik, Elektronik und Informationstechnik
vP	Very Persistent
vPvB	Very Persistent and very Bioaccumulative
WDK	Wirtschaftsverband der deutschen Kautschukindustrie
WHO	World Health Organization
ZEK	Zentrale Erfahrungsaustauschkreis der ZLS
ZLS	Zentralstelle der Länder für Sicherheitstechnik

References

Anderson LM, Diwan B A, Fear N T, and Roman E (2000). Critical windows of exposure for children's health: cancer in human epidemiological studies and neoplasms in experimental animal models. Environ. Health Perspect., 108 (suppl. 3): 573–594.

Angerer J (2007). Presentation "PAK-Belastung -Human Biomonitoring" second meeting of the ad hoc PAH working group of the provisional BfR Plastics Committee, 10.12.2007, BfR, Berlin (http://www.bfr.bund.de/cm/216/2_sitzung_der_ad_hoc_arbeitsgruppe_pak_der_vorlaeufigen_kunststoff_kommission.pdf

Anonymus (2009). Demand for non-labelled oils increases worldwide, as EU deadline approaches. European Rubber Journal(July/August):14.

APUG (2004). Action Programme Environment and Health, What are the differences between children and adults? (www.apug.de and www.umweltbundesamt.de)

Armstrong B, Tremblay C, Baris, D, and Theriault, G (1994). Lung cancer mortality and polynuclear aromatic hydrocarbons: A case- cohort study of aluminum production workers in Arvida, Quebec, Canada. AM. J. EPIDEMIOL. 139(3), 250-262.

Armstrong TW, Zaleski RT, Konkel WJ, and Parkerton TJ (2002). A tiered approach to assessing children's exposure: a review of methods and data. Toxicol. Lett., 127, 111–119.

Armstrong B, Hutchinson E, and Fletcher T (2003). Cancer risk following exposure to polycyclic aromatic hydrocarbons (PAHs): A meta analysis. Report No. Research Report 068,

Armstrong B, Hutchinson E, Unwin J, and Fletcher T (2004). Lung cancer risk after exposure to polycyclic aromatic hydrocarbons: A review and meta-analysis. Environmental Health Perspectives 112(9), 970-978.

Armstrong BG & Gibbs G (2009). Exposure-response relationship between lung cancer and polycyclic aromatic hydrocarbons (PAHs). Occup. Environ. Med. 66(11), 740-746.

ATSDR (1995). Toxicological profile for polycyclic aromatic hydrocarbons. 1-487

(AUH) Ausschuss für Umwelthygiene (1995). Standards für Expositionsabschätzung. Bericht des Ausschusses für Umwelthygiene. Behörde für Arbeit, Gesundheit und Soziales (Hrsg.), Hamburg 1995

Baird WM, Hooven LA, and Mahadevan B (2005). Carcinogenic polycyclic aromatic hydrocarbon-DNA adducts and mechanism of action. Environmental and Molecular Mutagenesis 45(2-3), 106-114.

Baker JE & Eisenreich SJ (1990). Concentrations and fluxes of polycyclic aromatic hydrocarbons and polychlorinated biphenyls across the air-water interface of lake superior. Environmental Science and Technology 24(3):342-352.

Barton H, Cogliano J, Firestone MP, Flowers L, Setzer RW, Valcovic L, and Woodruff T (2005). Supplemental guidance for assessing susceptibility from early-life exposure. Report No. EPA/630/R-03/003F, 1-126.

Barton HA, Cogliano VJ, Flowers L, Valcovic L, Setzer RW, and Woodruff TJ (2005a). Assessing Susceptibility from Early-Life Exposure to Carcinogens. Environ Health Perspect 113:1125–1133

Bayona JM, Fernandez P, Porte C, Tolosa I, Valls M, and Albaiges J. (1991). Partitioning of urban wastewater organic microcontaminants among coastal compartments. Chemosphere 23(3):313-326.

Behymer TD, Hites RA (1988). Photolysis of polycyclic aromatic hydrocarbons adsorbed on fly ash. Environ Sci Technol 22(11):1311-1319.

Benedetti MS, and Baltes EL (2003). Drug metabolism and disposition in children. Fundam. Clin. Pharmacol., 17: 281–299.

BfR (2009). Data of Stiftung Warentest, reported in BfR Expert Opinion No. 025/2009, 8 June 2009, "PAHs in consumer products must be reduced as much as possible" (http://www.bfr.bund.de/cm/230/pahs_in_consumer_products_must_be_reduced_as_much_as_possible.pdf)

Birnbaum LS & Fenton SE (2003). Cancer and developmental exposure to endocrine disruptors. Environ Health Perspect 111:389–394.

Bisson M, Dujardin R, Flammarion P, Garric J, Babut M, Lamy MH, Porcher JM, Thybaud É, and Vindimian É (2000). Complément au SEQ-Eau: méthode de détermination des seuils de qualité pour les substances génotoxiques. Verneuil-en-Halatte, France: Institut National de l'Environnement Industriel et des Risques (INERIS), Agence de l'eau Rhin-Meuse.

Bleeker EAJ & Verbruggen EMJ (2009). Bioaccumulation of polycyclic aromatic hydrocarbons - DRAFT. RIVM.

Boffetta P, Jourenkova N, and Gustavson P (1997). Cancer risk from occupational and environmental exposure to polycyclic aromatic hydrocarbons. Cancer Causes and Control 8, 444-472.

Bossert I, Kachel WM, and Bartha R (1984). Fate of hydrocarbons during oily sludge disposal in soil. Appl Environ Microbiol 47(4):763-767.

Bosetti C, Boffetta P, and La Vecchia C (2007). Occupational exposures to polycyclic aromatic hydrocarbons, and respiratory and urinary tract cancers: a quantitative review to 2005. Annals of Oncology 18(3), 431-446.

Bruner KA, Fisher SW, and Landrum PF (1994). The role of the zebra mussel, Dreissenia polymorpha, on contaminant cycling: I.The effect of body size and lipid content on the bioconcentration of PCBs and PAHs. J Great Lakes Res 20(4):725-734.

Budroe JD, Brown JP, Collins JF, Marty MA, and Salmon AG (2009). Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment Air Toxicology and Epidemiology Branch (OEHHA)

Buffler PA & Kyle AD (1999). Carcinogen Risk Assessment Guidelines and Children. Environmental Health Perspectives Volume 107, Number 6, June. http://www.envirohealthpolicy.net/Publications/KidsAssessment.html

Bulder AS, Hoogenboom LAP, Kan CA, Raamsdonk LWD, van Traag WA, and Bouwmeester H (2006). Initial Risk Assessment of Polycyclic Aromatic Hydrocarbons (PAHs) in Feed (materials). Report No. Report 2006.001; Addendum to Report: 03/0027745.

Cailleaud K, Budzinski H, Le Menach K, Souissi S, and Forget-Leray J. (2009). Uptake and elimination of hydrophobic organic contaminants in Estuarine copepods: An experimental study. etc 28(2):239-246.

Calabrese EJ (1986). Age and susceptibility to toxic substances. John Wiley & Sons, NewYork Chichester Brisbane Toronto Singapore:239-285

Calvert JG, Atkinson JR, Becker KH, Kamens RM, Seinfeld JH, Wallington TJ, and Yarwood G (2002). The mechanisms of atmospheric oxidation of aromatic hydrocarbons. Oxford University Press, Oxford.

Carroquino MJ, Galson SK, Licht J, Amler RW, Perera FP, Claxton LD, and Landrigan PJ (1998). The U.S. EPA Conference on Preventable Causes of Cancer in Children: a research agenda. Environ Health Perspect 106(suppl 3):867

Carslaw N, Carpenter LJ, Plane JMC, Allan BJ, Burgess RA, Clemitshaw KC, and Penkett SA (1997). Simultaneous observations of nitrate and peroxy radicals in the marine boundary layer. J Geoph Res 102(D15):18917-18933.

Charnley G & Putzrath RM (2001). Children's Health, Susceptibility, and Regulatory Approaches to Reducing Risks from Chemical Carcinogens. Environ Health Perspect 109(2): 187-192.

Coover MP & Sims RC (1987). The effect of temperature on polycyclic aromatic hydrocarbon persistence in an unacclimated agriculture soil. Hazard Waste Hazard Mater 4(1).

Costantino JP, Redmond CK, and Bearden A (1995). Occupationally Related Cancer Risk Among Coke-Oven Workers - 30 Years of Follow-Up. Journal of Occupational and Environmental Medicine 37(5), 597-604.

Culp S, Gaylor DW, Sheldon WG, Goldstein LS, and Beland FA (1998). A comparison of the tumors induced by coal tar and benzo[a]pyrene in a 2-year bioassay. Carcinogenesis 19(1), 117-124.

Dean SW, Coates A, Brooks TM, and Burlinson B (1998). Benzo[a]pyrene site of contact mutagenicity in skin of Muta (TM) Mouse. Mutagenesis 13(5), 515-518.

de Maagd PG-J, de Poorte J, Opperhuizen A, and Sijm DTHM (1998). No influence after various exposure times on the biotransformation rate constants of benzo[a]anthracene in fathead minnow (Pimephales promelas). Aquatic Toxicology 40(2-3):157-169.

Dermentzoglu M, Manoli E, Voutsa D, and Samara V (2003). Sources and patterns of polycyclic aromatic hydrocarbons and heavy metals in fine indoor particular matter of greek houses PSP Volume 12, Fresenius Environmentaö Bulletin 12, 1511 – 1519

Ding YS, Ashley DL, and Watson CH (2007). Determination of 10 carcinogenic polycyclic aromatic hydrocarbons in mainstream cigarette smoke. J Agric Food Chem. 55: 5966-5573

Duxbury CL, Dixon DG, and Greenberg BM (1997). Effects of simulated solar radiation on the bioaccumulation of polycyclic aromatic hydrocarbons by the duckweed Lemna gibba. etc 16(8):1739-1748.

Dybing E, Sanner T, Roelfzema H, Kroese D, and Tennant, RW (1997). T25: a simplified carcinogenic potency index: description of the system and study of correlations between carcinogenic potency and species/site specificity and mutagenicity. Pharmacol Toxicol 80(6), 272-279.

EC (1998). Directive 98/8/EC of the European Parliament and of the Council of 16 February 1998 - concerning the placing of biocidal products on the market. Official Journal of the European Communities, L 123-1-L 123/63.

EC (2002). Opinion of the Scientific Committee on Food on the risks to human health of Polycyclic Aromatic Hydrocarbons in food. Report No. SCF/CS/CNTM/PAH/29 Final, 1-84

EC (2008). Regulation (EC) No. 1272/2008 of the European Parlament and of the Council of 16 December 2008 on classification, labelling and packaging of substances and mixtures, amending and repealing Directives 67/548/EEC and 1999/45/EC, and amending Regulation. Official Journal of the European Union, L 353-1-L 353/1355.

EC/HC (1994). Canadian Environmental Protection Act. Priority Substances List Assessment Report: Polycyclic aromatic hydrocarbons. 1-68

ECHA (2008). Guidance on information requirements and chemical safety assessment, Chapter R.8: Characterisation of dose [concentration]-response for human health. 1-150

EEC (1967). COUNCIL DIRECTIVE 67/548/EEC of 27 June 1967 on the approximation of laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances. 1-357

EFSA (2008). Polycyclic aromatic hydrocarbons in food. Scientific Opinion of the Panel on Contaminants in the Food Chain. The EFSA Journal 724, 1-114.

EN 1186-14 (2002). Materials and articles in contact with foodstuffs - Plastics - Part 14: Test methods for 'substitute tests' for overall migration from plastics intended to come into contact with fatty foodstuffs using test media iso-octane and 95 % ethanol, Beuth Verlag

EN ISO 105-E04 (2009). Textiles - Tests for colour fastness - Part E04: Colour fastness to perspiration.

European Centre for Ecotoxicology and Toxicology of Chemicals (ECETOC, 2008). ECETOC technical report No 93, Targetet Risk Assessment. ECETOC AISBL, 4 Avenue E. Van Nieuwenhuyse (Bte 6), B-1160 Brussels, Belgium.

European Centre for Ecotoxicology and Toxicology of Chemicals (ECETOC, 2010). Addendum to ECETOC Targeted Risk Assessment Report No. 93. ECETOC AISBL, 4 Avenue E. Van Nieuwenhuyse (Bte 6), B-1160 Brussels, Belgium.

European Chemicals Agency (ECHA, 2008). Chapter R.7a: Endpoint specific guidance. In: Guidance On Information Requirements And Chemical Safety Assessment. 1-428.

European Chemicals Agency (ECHA, 2009). Support document for identification of Coal Tar Pitch, High Temperature as a SVHC because of its PBT and CMR properties. ECHA.

European Chemicals Agency (ECHA, 2010). Guidance on information requirements and chemical safety assessment. Chapter R.15: Consumer exposure estimation. Version 1.2, Draft under preparation.(http://guidance.echa.europa.eu/docs/guidance_document/information_requirements_r15_en.pdf?vers=20_08_08

European Commission (EC, 2007). European Union Risk Assessment Report, Draft of November 2007, Coal Tar Pitch High Temperature, CAS No: 65996-93-2, EINECS No: 266-028-2.

Evans MS & Landrum PF (1989). Toxicokinetics of DDE, benzo[a]pyrene, and 2, 4, 5, 2GÇ¦, 4GÇ¦, 5GÇ¦-hexachlorobiphenyl in Pontoporeia hoyi and Mysis relicta. Journal of Great Lakes Research 15(4):589-600.

FAO/WHO (JECFA, 2006).. Safety evaluation of certain contaminants in food. Prepared by the Sixty-fourth meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). 1-5. 2006. World Health Organization WHO; Food and Agriculture Organization of the United Nations FAO. WHO Food Additives Series 55; FAO Food and Nutrition Paper 82.

Faustman EM, Silbernagel SM, Fenske RA, Burbacher TM, and Ponce RA (2000). Mechanisms underlying children's susceptibility to environmental toxicants. Environ Health Perspect 108:13–21.

FEHD (2004). Polycyclic Aromatic Hydrocarbons In Barbecued Meat. Report No. Risk Assessment Studies Report No. 14.

Fenton SE & Davis CC (2002). Atrazine exposure in utero increases dimethylbenz[a]anthracene-induced mammary tumor incidence in Long Evans offspring [Abstract]. Toxicol Sci 66(1–2):185.

Fernandez M & L'Haridon J (1992). Influence of lighting conditions on toxicity and genotoxicity of various PAH in the newt in vivo. Mutation Research - Genetic Toxicology Testing and Biomonitoring of Environmental or Occupational Exposure 298(1):31-41.

FhI (1997). Fraunhofer Institute of Toxicology and Aerosol Research (FhI) (1997). Dermal Carcinogenicity Study of Two Coal Tar Products (CTP) by Chronic Epicutaneous Application in Male CD-1 Mice (78 Weeks). Report No. Final Report, 1-11

Foth H, Kahl R, and Kahl GF (1988). Pharmacokinetics of low doses of benzo[a]pyrene in the rat. Food and Chemical Toxicology 26(1), 45-51.

Frank AP, Landrum PF, and Eadie BJ (1986). Polycyclic aromatic hydrocarbon rates of uptake, depuration, and biotransformation by Lake Michigan Stylodrilus heringianus. Chemosphere 15(3):317-330.

Garry S, Nesslany F, Aliouat E, Haguenoer JM, and Marzin D (2003). Assessment of genotoxic effect of benzo[a]pyrene in endotracheally treated rat using the comet assay. Mutation Research-Genetic Toxicology and Environmental Mutagenesis 534(1-2), 33-43.

Ginsberg GL (2003). Assessing cancer risks from short-term exposures in children. Risk Anal., 23: 19–34.

Gossiaux DC, Landrum PF, Fischer SW (1996). Effect of Temperature on the Accumulation Kinetics of PAHs and PCBs in the Zebra Mussel, Dreissena polymorpha. J Great Lakes Res 22(2):379-388.

Gregg E, Hill C, Hollywood M, Kearney M, McAdam K, McLaughlin D, Purkis S, and Williams M (2004). The UK Smoke Constitutents Testing Study. Summary of Results and Comparison with Other Studies. Beiträge zur Tabakforschung International 21: 117-138

Greim H (2008). Gesundheitsschädliche Arbeitsstoffe; Toxikologisch-arbeitsmedizinische Begründungen von MAK-Werten und Einstufungen. (534) Weinheim, DFG, Deutsche Forschungsgemeinschaft, WILEY-VCH Verlag.

Gustavson P, Östmann C, and Sällsten G (2008). Indoor Levels of Polycyclic Aromatic Hydrocarbons in Homes with or without Wood Burning for Heating. Environ. Sci. Technol. 42, 5074–5080

Habs M, Schmahl D, and Misfeld, J (1980). Local Carcinogenicity of Some Environmentally Relevant Polycyclic Aromatic-Hydrocarbons After Lifelong Topical Application to Mouse Skin. Archiv fur Geschwulstforschung 50(3), 266-274.

Habs M, Jahn SAA, and Schmahl D (1984). Carcinogenic Activity of Condensate from Coloquint Seeds (Citrullus-Colocynthis) After Chronic Epicutaneous Administration to Mice. Journal of Cancer Research and Clinical Oncology 108(1), 154-156.

Hakura A, Tsutsui Y, Sonoda J, Kai JK, Imade T, Shimada M, Sugihara Y, and Mikami, T (1998). Comparison between in vivo mutagenicity and carcinogenicity in multiple organs by benzo[a]pyrene in the lacZ transgenic mouse (Muta (TM) Mouse). Mutation Research-Fundamental and Molecular Mechanisms of Mutagenesis 398(1-2), 123-130.

Hakura A, Tsutsui Y, Sonoda J, Mikami T, Tsukidate K, Sagami F, and Kerns WD (1999). Multiple organ mutation in the lacZ transgenic mouse (Muta (TM) Mouse) 6 months after oral treatment (5 days) with benzo[a]pyrene. Mutation Research-Fundamental and Molecular Mechanisms of Mutagenesis 426(1), 71-77.

Hamm St, Frey Th, Weinand R, Moninot G, and Petiniot N (2009). "Investigations on the extraction an migration behaviour of polycyclic aromatic hydrocarbons (PAHs) from rubber formulations containing carbon black as reinforcing agent", Rubber Chemistry and Technology, 2009, Vol. 82 No. 2

Hannah JB, Hose JE, and Landolt ML (1982). Benzo[a]pyrene-induced morphologic and developmental abnormalities in rainbow trout. aect 11(6):727-734.

Hashimoto AH, Amanuma K, Hiyoshi K, Takano H, Masumura K, Nohmi T, and Aoki, Y (2005). In vivo mutagenesis induced by benzo[a]pyrene instilled into the lung of gpt delta transgenic mice. Environmental and Molecular Mutagenesis 45(4), 365-373.

Hattis D, Goble R, and Chu M (2005). Age-Related Differences in Susceptibility to Carcinogenesis. II. Approaches for Application and Uncertainty Analyses for Individual Genetically Acting Carcinogens. Environ Health Perspect 113:509–516

HCN (2006). BaP and PAH from coal-derived sources. Health-based calculated occupational cancer risk values of benzo[a]pyrene and unsubstituted non-heterocyclic polycyclic aromatic hydrocarbons from coal-derived sources. Health Council of the Netherlands (Gezondheidsraad), 1-204.

Heinrich U, Roller M, and Pott F (1994). Estimation of a lifetime unit lung cancer risk for benzo[a]pyrene based on tumour rates in rats exposed to coal tar/pitch condensation aerosol. Toxicol Lett 72(1-3), 155-161.

Hoff RM & Chan KW (1987). Measurement of polycyclic aromatic hydrocarbons in the air along the niagara river. Environmental Science and Technology 21(6):556-561.

Holladay SD & Smialowicz RJ (2000). Development of the murine and human immune system: differential effects of immunotoxicants depend on time of exposure. Environ Health Perspect 108(suppl 3):463–473.

Holsapple MP, West LJ, and Landreth KS (2003). Species comparison of anatomical and functional immune system development. Birth Defects Res B Dev Reprod Toxicol 68:321–334.

Hooftman RN & Evers-de Ruiter A (1992). Early life stage tests with Brachydanio rerio and several polycyclic aromatic hydrocarbons using an intermittent flow-through system (draft OECD guideline).TNO-report IMW-R 92/210. The Netherlands Organisation for Applied Scientific Research (TNO), Environmental and Energy Research, Institute of Environmental Sciences, Delft, The Netherlands.

Hutzler C (2009). Presentation „Zur Problematik von polycyclischen aromatischen Kohlenwasserstoffen (PAK) in verbrauchernahen Produkten und Spielzeug – Migrationsuntersuchungen“, First meeting of the ad hoc panel on toys of the BfR Committee for consumer products, 26.06.2009, BfR, Berlin

International Agency for the Research of Cancer (IARC, 1983). Polynuclear Aromatic Compounds, part 1, chemical, environmental and experimental data summary of data reported and evaluation. (Monographs on the Evaluation of Carcinogenic Risks to Humans - Volume 32)

International Agency for the Research of Cancer (IARC, 1984). Polynuclear Aromatic Compounds, Part 2: Carbon Blacks, Mineral Oils (Lubricant Base Oils and Derived Products) and Some Nitroarenes. (Monographs on the Evaluation of Carcinogenic Risks to Humans - Volume 33)

International Agency for the Research of Cancer (IARC, 1984). Polynuclear Aromatic Compounds, Part 3: Industrial Exposures in Aluminium Production, Coal Gasification, Coke Production and Iron and Steel Founding. (Monographs on the Evaluation of Carcinogenic Risks to Humans - Volume 34)

International Agency for the Research of Cancer (IARC, 1985). Polynuclear Aromatic Compounds, Part 4: Bitumens, Coal-Tars and Derived Products, Shale-Oils and Soots. (Monographs on the Evaluation of Carcinogenic Risks to Humans - Volume 35)

International Agency for the Research of Cancer (IARC, 1989). Diesel and Gasoline Engine Exhausts and Some Nitroarenes. (Monographs on the Evaluation of Carcinogenic Risks to Humans - Volume 46)

International Agency for the Research of Cancer (IARC, 2006). Polycyclic aromatic hydrocarbons. Report No. 92.

International programme on chemical safety (IPCS, 2006), “Principles for evaluating health risks in children associated with exposure to chemicals”, Environmental health criteria Nº237, World Healh Organisation (WHO)., Geneva, Switzerland.

Jimenez BD, Cirmo CP, and McCarthy JF (1987) Effects of feeding and temperature on uptake, elimination and metabolism of benzo[a]pyrene in the bluegill sunfish (Lepomis macrochirus).. Aquatic Toxicology 10(1):41-57.

Johannesson S, Bergemalm-Rynell K, Strandberg B, and Sällsten G (2008). Indoor concentrations of fine particles and particle-bound PAHs in Gothenburg, Sweden Journal of Physics:ConferenceSeries 151 (2009) 012006

Joona M (2003). Making more eco-friendly tyres. Naphtenics Magazine(2).

Kamens RM, Guo J, Guo Z, and McDow SR (1990). Polynuclear aromatic hydrocarbon degradation by heterogeneous reactions with N2O5 on atmospheric particles. Atmospheric Environment - Part A General Topics 24 A(5):1161-1173.

Kamens RM, Perry JM, Saucy DA, Bell DA, Newton DL, and Brand B (1985). Factors which influence polycyclic aromatic hydrocarbon decomposition on wood smoke particles. Environment International 11(2-4):131-136.

Kao J, Patterson FK, and Hall, J (1985). Skin penetration and metabolism of topically applied chemicals in six mammalian species, including man: an in vitro study with benzo[a]pyrene and testosterone. Toxicology and Applied Pharmacology 81(3 Pt 1), 502-516.

Knafla A, Phillipps KA, Brecher RW, Petrovic S, and Richardson M (2006). Development of a dermal cancer slope factor for benzo[a]pyrene. Regulatory Toxicology and Pharmacology 45(2), 159-168.

Kroes R, Renwick AG, Feron V, Galli CL, Gibney M, Greim H, Guy RH, Lhuguenot JC, and van de Sandt,JJ (2007). Application of the threshold of toxicological concern (TTC) to the safety evaluation of cosmetic ingredients. Food and Chemical Toxicology 45(12), 2533-2562.

Kroese ED, Muller JJA, Mohn GR, Dortant PM, and Wester PW (2001). Tumorigenic effects in Wistar rats orally administered benzo[a]pyrene for two years (gavage studies). Implications for human cancer risks associated with oral exposure to polycyclic aromatic hydrocarbons. Report No. RIVM 658603 010, 1-138.

Lafontaine M, Champmartin C, Simon P, Delsaut P, and Funck-Brentano C (2006). 3-Hydroxybenzo[a]pyrene in the urine of smokers and non-smokers.Toxicol Lett. 162:181-185.

Landrum PF & Poore R. (1988). Toxicokinetics of selected xenobiotics in Hexagenia limbata. J Great Lakes Res 14(4):427-437.

Landrum PF (1988). Toxicokinetics of organic xenobiotics in the amphipod, Pontoporeia hoyi: Role of physiological and environmental variables. Aquatic Toxicology 12(3):245-271.

Leversee GJ, Giesy JP, Landrum PF, Bartell S, Gerould S, Bruno M, Spacie A, Bonling J, Haddock J, and Fannin T (1981). Disposition of benzo(a)pyree in aquatic system components: periphyton, chironomids, daphnia, fish. In: Analysis and Biological Fate: Polynuclear Aromatic Hydrocarbons. Cooke M, Dennis AJ, editors, Batelle Press, Columbus, OH, USA. 357-366.

Leversee GJ, Giesy JP, and Landrum PF (1982). Kinetics and biotransformation of benzo[a]pyrene in Chironomus riparius. aect 11(1):25-31.

Lewis RG, Fortune CR, Willis RD, Camann ED, and Antlet JT (1999). Distribution of Pesticides and Polycyclic Aromatic Hydrocarbons in House Dust as a Function of Particle Size. Environmental Health Perspectives * Volume 107, Number 9, 721 – 726

Lyons BP, Pascoe CK, and McFadzen IRB (2002). Phototoxicity of pyrene and benzo[a]pyrene to embryo-larval stages of the pacific oyster Crassostrea gigas. Mar Environ Res 54(3-5):627-631.

Mackay D, Shiu WY, and Ma K (2000). Illustrated handbook of physical-chemical properties and environmental fate for organic chemicals. Vol. II: Polynuclear aromatic hydrocarbons, polychlorinated dioxins and dibenzofurans. Lewis Publishers, Chelsea.

MAK (2008). Policyclische Aromatische Kohlenwasserstoffe (PAH). In Gesundheitsschädliche Arbeitsstoffe: Toxikologisch-arbeitsmedizinische Begründungen von MAK-Werten und Einstufungen. 45. Lieferung (H.Greim, Ed.), pp. 1-214. John Wiley & Sons.

McCarthy JF, Jimenez BD, and Barbee T (1985). Oct. Effect of dissolved humic material on accumulation of polycyclic aromatic hydrocarbons: Structure-activity relationships. Aquatic Toxicology 7(1-2):15-24.

McVeety BD & Hites RA (1988). Atmospheric deposition of polycyclic aromatic hydrocarbons to water surfaces: A mass balance approach. Atmospheric Environment 22(3):511-536.

Mastrangelo G, Fadda E, and Marzia V (1996). Polycyclic aromatic hydrocarbons and cancer in man. Environmental Health Perspectives 104(11), 1166-1170.

Meylan WM & Howard PH (1991). Bond contribution method for estimating Henry's law constants. etc 10(10):1283-1293.

Miller MD, Marty MA, Arcus A, Brown J, Morry D, and Sandy M (2002). Differences between children and adults: implications for risk assessment at California EPA. Int J Toxicol 21:403–418.

Moldoveanu SC, Coleman W, and Wilkins JM (2008). Determination of Polycyclic Aromatic Hydrocarbons in Exhaled Cigarette Smoke. Beiträge zur Tabakforschung International 23: 85-97

Moolgavkar, SH, Luebeck, EG, and Anderson, EL (1998). Estimation of unit risk for coke oven emissions. Risk Anal. 18(6), 813-825.

Neau A & Rangstedt M (2009). Naphthenic extender oils in natural rubber tyre tread compounds. Rubber World(11):21-26.

Neri M, Ugolini D, Bonassi S, Fucic A, Holland N, Knudsen L, Srám R, Ceppi M, Bocchini V, and Merlo DF (2006). Children's exposure to environmental pollutants and biomarkers of genetic damage. II. Results of a comprehensive literature search and meta-analysis. Mutat Res, 612(1): 14–39.

Newsted JL & Giesy JP (1987). Predictive models for photoinduced acute toxicity of polycyclic aromatic hydrocarbons to Daphnia magna Strauss (cladocera, crustacea). Environ Toxicol Chem 6(6):445-461.

Ng KM, Chu I, Bronaugh RL, Franklin CA, and Somers, DA (1992). Percutaneous absorption and metabolism of pyrene, benzo[a]pyrene, and di(2-ethylhexyl) phthalate: comparison of in vitro and in vivo results in the hairless guinea pig. Toxicology and Applied Pharmacology 115(2), 216-223.

Nilsson NH, Feilberg A, and K (2005). Pommer, Emissions and evaluation of health effects of PAH's and aromatic mines from tyres, Danish Ministry of the Environment.

Nishikawa T, Haresaku M, Fukushima A, Nakamura T, Adachi K, Masuda M, and Hayashi M (2002). Further evaluation of an in vivo micronucleus test on rat and mouse skin: results with five skin carcinogens. Mutation Research-Genetic Toxicology and Environmental Mutagenesis 513(1-2), 93-102.

NITE (2010). Chemical Risk Information Platform (CHRIP).

Null V (1999). Safe Process Oils Tires with Low Environmental Impact. Kunstoffe Gummi Kautschuk 52(12):799-805.

Nynas (2003). Trade fair highlights naphthenic benefit for PVC. Naphtenics Magazine(2).

Office of Environmental Health Hazard Assessment (OEHHA, 2007). Evaluation of Health Effects of Recycled Waste Tires in Playground and Track Products. California Integrated Waste Management Board (CIWMB) publication #622-06-013. http://www.calrecycle.ca.gov/ publications/Tires/62206013.pdf retrieved on 07 April 2010

Ohura T, Noda T, Amagi T, and Fusaya M (2005). Prediction of Personal Exposure to PM2.5 and Carcinogenic Polycyclic Aromatic Hydrocarbons by Their Concentrations in Residential Microenvironments. Environ. Sci. Technol. 2005, 39, 5592-5599

Oris JT & Giesy JP (1987). The photo-induced toxicity of Polycyclic aromatic hydrocarbons to larvae of the fathead minnow (Pimephales promelas). Chemosphere 16(7):1395-1404.

Park KS, Sims RC, Dupont RR, Doucette WJ, Matthews JE. 1990 Feb. Fate of PAH compounds in two soil types: influence of volatilization, abiotic loss and biological activity. Environ Tox Chem 9(2):187-195.

Payan JP, Lafontaine M, Simon P, Marquet F, Champmartin-Gendre C, Beydon D, Wathier L, and Ferrari E (2009). 3-Hydroxybenzo[a]pyrene as a biomarker of dermal exposure to benzo[a]pyrene. Archives of Toxicology 83(9), 873-883.

Pearlman RS, Yalkowsky SH, and Banerjee S (1984). Water Solubilities of Polynuclear Aromatic and Heteroaromatic Compounds. J Phys Chem Ref Data 13(2):555-562.

Peto R, Pike MC, Day NE, Gray RG, Lee PN, Parish S, Peto J, Richards S, and Wahrendorf, J (1980). Guidelines for simple, sensitive significance tests for carcinogenic effects in long-term animal experiments. IARC Monogr Eval. Carcinog. Risk Chem Hum Suppl(2 Suppl), 311-426.

Plesser TS, & Lund OJ (2004). Potential health and Environmental Effects Linked to Artifical Turf Systems – Final Report. Prepared for BYGGFORSK, Oslo, Norway

PORG PORG (1997). Ozone in the United Kingdom. Official Report (Fourth report).

Richardson BJ, Tse ESC, De Luca-Abbott SB, Martin M, and Lam PKS (2005). Uptake and depuration of PAHs and chlorinated pesticides by semi-permeable membrane devices (SPMDs) and green-lipped mussels (Perna viridis). Marine Pollution Bulletin 51(8-12):975-993.

Roberts RJ (1992). Overview of similarities and differences between children and adults: implications for risk assessment. In: Similarities and Differences between Children and Adults (Guzelian PS, Henry CJ, Olin SS, eds). Washington, DC:ILSI Press, 1992;11

Routledge PA (1994). Pharmacokinetics in children. J. Antimicrob. Chemother. 34: 19–24.

Sagredo C, Ovrebo S, Haugen A, Fujii-Kuriyama Y, Baera R, Botnen IV, and Mollerup S (2006) Quantitative analysis of benzo[a]pyrene biotransformation and adduct formation in Ahr knockout mice. Toxicology Letters 167(3), 173-182.

Sander K, (2006). TÜV-Rheinland, Presentation “PAK - Migration im Hautkontakt”, first meeting of the ad hoc PAH working group of the provisional BfR Plastics Committee, 13.12.2006, BfR, Berlin

Sanders CL, Skinner C, and Gelman, RA (1986). Percutaneous absorption of 7, 10 14C-benzo[a]pyrene and 7, 12 14C-dimethylbenz[a]anthracene in mice. Journal of Environmental Pathology, Toxicology and Oncology 7(1-2), 25-34.

Scheuplein R, Charnley G, and Dourson M (2002). Differential sensitivity of children and adults to chemical toxicity. I. Biological basis. Regul. Toxicol. Pharmacol., 35, 429–447.

Schmähl D, Schmidt KG, and Habs M (1977). Syncarcinogenic action of polycyclic hydrocarbons in automobile exhaust gas condensates. IARC scientific publications(16), 53-59.

Schneider K, Roller MF, Kalberlah F, and Schuhmacher-Wolz, U (2002). Cancer Risk Assessment for Oral Exposure to PAH Mixtures. J. Appl. Toxicol. 22, 73-83.

Schnell JV, Gruger J, and Malins DC (1980). Mono-oxygenase activities of coho salmon (Oncorhynchus kisutch) liver microsomes using three polycyclic aromatic hydrocarbon substrates. Xenobiotica 10(3):229-234.

Schulte A, Ernst H, Peters L, and Heinrich U (1994). Induction of squamous cell carcinomas in the mouse lung after long-term inhalation of polycyclic aromatic hydrocarbon-rich exhausts. Experimental and Toxicologic Pathology 45, 415-421.

Scientific Committee on Toxicity, Ecotoxicity and teh Environment (SCTEE, 2003). Opinion of the Scientific Committee on Toxicity, Ecotoxicity and teh Environment (SCTEE) on "Questions to the CSTEE relating to scietific evidence of risk to health and the environment from polycyclic aromatic hydrocarbons in extender oils and tyres". Report No. C7/GF/csteeop/PAHs/12-131103 D(03), 1-11

Shaw D (2008). Tyre makers prepare for EU ban on labelled oils from 2010. European Rubber Journal(October/November):30.

Shaw D (2009). Tyre companies begin to switch to non-labelled oils ahead of EU legislation. European Rubber Journal(October/November):22-23.

Slikker W, Mei N, and Chen T (2004). N-Ethyl-N-nitrosourea (ENU) increased brain mutations in prenatal and neonatal mice but not in the adults. Toxicol Sci 81(1):112–120

Southworth GR, Beauchamp JJ, and Schmieder PK (1978). Bioaccumulation potential of polycyclic aromatic hydrocarbons in Daphnia pulex. Water Research 12(11):973-977.

Southworth GR (1979). The role of volatilization in removing polycyclic aromatic hydrocarbons from aquatic environments. Bulletin of Environmental Contamination and Toxicology 21(4-5):507-514.

Stewart CF & Hampton EM (1987). Effect of maturation on drug disposition in pediatric patients. Clin. Pharm. 6, 548–564

Takeuchi I, Miyoshi N, Mizukawa K, Takada H, Ikemoto T, Omori K, and Tsuchiya K (2009). Biomagnification profiles of polycyclic aromatic hydrocarbons, alkylphenols and polychlorinated biphenyls in Tokyo Bay elucidated by 13C and 15N isotope ratios as guides to trophic web structure. Marine Pollution Bulletin 58(5):663-671.

The Netherlands - Bureau REACH (2009). Annex XV transitional report: coal tar pitch, high temperature (CTPHT).

Thomas RD (1995). Age-Specific Carcinogenesis - Environmental Exposure and Susceptibility. Environ Health Perspect 103(6):45-48

Thyssen J, Althoff J, Kimmerle G, and Mohr U (1981). Inhalation studies with benzo[a]pyrene in Syrian golden hamsters. Journal of the National Cancer Institute 66, 575-577.

Trucco RG, Engelhardt FR, and Stacey B (1983). Toxicity, accumulation and clearance of aromatic hydrocarbons in Daphnia pulex. Environmental Pollution Series A, Ecological and Biological 31(3):191-202.

TÜV Rheinland (2010). Dr. Wennemer, personal communication

Umweltbundesamt (UBA, 2010). Cancerogene, mutagene, reproduktionstoxische (CMR) und andere problematische Stoffe in Produkten - Identifikation relevanter Stoffe und Erzeugnisse, Überprüfung durch Messungen, Regelungsbedarf im Chemikalienrecht. UFOPLAN-Projekt 3707 61 300, Dessau, 2008-2010.

US EPA (1984). Health effects assessment for benzo(a) pyrene (BaP). Cincinnati, EPA, Environmental Criteria and Assessment Office. Report No. EPA-540/1-86-022: 1-42

US EPA (1996). Environmental Health Threats to Children. EPA 175-F-96-001. Washington, DC:U.S. Environmental Protection Agency.

US EPA (1996). Proposed Guidelines for Carcinogen Risk Assessment. EPA/600/P-92/003C. Washington, DC:U.S. Environmental Protection Agency, Office of Research and Development.

US EPA (1998). Recommendations from the Children's Health Advisory Committee Regarding Evaluating Environmental Standards. Washington, DC:U.S. Environmental Protection Agency, Office of Children's Health Protection.

US EPA (1998). Comparison of the Effects of Chemicals with Combined Perinatal and Adult Exposure vs. Adult Only Exposure in Carcinogenesis Bioassays. Washington, DC:U.S. Environmental Protection Agency, Office of Pesticide Programs.

US EPA (2002). Peer consultation workshop on approaches to polycyclic aromatic hydrocarbon (PAH) health assessment. Report No. EPA/635/R-02/005, 1-182

US EPA (2007). Risk Characterization for Carcinogens that have a Mutagenic Mode of Action.

Verrhiest G, Clement B, and Blake G (2001). Single and combined effects of sediment-associated PAHs on three species of freshwater macroinvertebrates. Ecotoxicology 10(6):363-372.

Warshawsky D and Barkley W (1987). Comparative Carcinogenic Potencies of 7H-Dibenzo[C,G]Carbazole, Dibenz[A,J]Acridine and Benzo[A]Pyrene in Mouse Skin. Cancer Letters 37(3), 337-344.

Wayne RP, Barnes I, Biggs P, Burrows JP, Canosa-Mas CE, Hjorth J, Le Bras G, Moortgat GK, Perner D, Poulet G, Restelli G, and Sidebottom H (1991). The nitrate radical: Physics, chemistry, and the atmosphere. Atmospheric Environment - Part A General Topics 25(1):1-203.

Wirtschaftsverband der Deutschen Kautschukindustrie (WDK, 2007). Presentation „PAK in verbrauchernahen Produkten “second meeting of the ad hoc PAH working group of the provisional BfR Plastics Committee, BfR, Berlin

http://www.bfr.bund.de/cm/216/2_sitzung_der_ad_hoc_arbeitsgruppe_pak_der_vorlaeufigen_kunststoff_kommission.pdf

Wennemer, A. (2009). TÜV Rheinland Presentation “PAK-Konzentration in Produkten erschreckend hoch”, Pressekonferenz TÜV Rheinland Group, Köln, 31. März 2009, http://www.tuv.com/de/news_pak_in_produkten.html?lan=1

Wernersson AS & Dave G (1997). Apr. Phototoxicity identification by solid phase extraction and photoinduced toxicity to Daphnia magna. Arch Environ Contam Toxicol 32(3):268-273.

Wernersson AS (2003). Predicting petroleum phototoxicity. Ecotoxicology and Environmental Safety 54(3):355-365.

Wester RC, Maibach HI, Bucks DA, Sedik L, Melendres J, Liao C, and DiZio S (1990). Percutaneous absorption of [14C]DDT and [14C]benzo[a]pyrene from soil. Fundamental and Applied Toxicology 15(3), 510-516.

Weyand EH, Chen YC, Wu Y, Koganti A, Dunsford HA, and Rodriguez LV (1995). Differences in the Tumorigenic Activity of A Pure Hydrocarbon and A Complex Mixture Following Ingestion - Benzo[A]Pyrene Vs Manufactured-Gas Plant Residue. Chemical Research in Toxicology 8(7), 949-954.

World Health Organization (WHO, 1987). Air Quality Guidelines for Europe. WHO Regional Publications, European Series No 23. Copenhagen, WHO Regional Office for Europe.

World Health Organization (WHO, 1998). Selected non-heterocyclic policyclic aromatic hydrocarbons. 1-701. Geneva, World Health Organization (WHO) / International Programme on Chemical Safety (IPCS). Environmental Health Criteria 202.

World Health Organization (WHO, 2000). Air quality guidelines for Europe. Second Edition (WHO Regional Publications, European Series, No. 91), 1-288. Copenhagen / Denmark, World Health Organization (WHO) / Regional Office for Europe Copenhagen.

World Health Organization (WHO, 2003). Polynuclear aromatic hydrocarbons in drinking-water. Background document for development of WHO guidelines for drinking-water quality. WHO/SDE/WSH/03.04/59, 1-27. Geneva, World Health Organization (WHO) / International Programme on Chemical Safety (IPCS).

World Health Organization (WHO, 2004). Regional Office for Europe: Children's environment and health action plan for Europe (CEHAPE). Available online at: http://www.euro.who.int/eprise/main/WHO/Progs/BUD/resolutions/20030328_2 (as of: 2003)

World Health Organization (WHO-Europe, 2005). Effects of Air Pollution on Children's Health and Development. A Review of the Evidence. Special Programme on Health and Environment. World Health Organization, European Centre for Environment and Health, Bonn Office, Bonn, Germany.

Whyatt R (2006). Intolerable Risk: The Physiological Susceptibility of Children to Pesticides. Journal of Pesticide Reform http://eap.mcgill.ca/MagRack/JPR/JPR_06.htm

Wild CP & Kleinjans J (2003). Children and increased susceptibility to environmental carcinogens: evidence or empathy? Cancer Epidemiol. Biomarkers & Prev., 12, 1389–1394

Wild SR, Berrow ML, and Jones KC (1991). The persistence of polynuclear aromatic hydrocarbons (PAHs) in sewage sludge amended agricultural soils. Environ Pollut 72:141-157.

Wild SR & Jones KC (1993). Biological and abiotic losses of polynuclear aromatic hydrocarbons (PAH) from soils freshly amended with sewage sludge. Environ Toxicol Chem 12:5-12.

Wilson J (1977). Current status of teratology; general principles and mechanisms derived from animal studies. In: Handbook of Teratology; General Principles and Etiology (Wilson J, Fraser F, eds). New York:Plenum Press.

Wu Q, Suzuki JS, Zaha H, Lin TM, Peterson RE, Tohyama C, and Ohsako, S (2008). Differences in gene expression and benzo[a]pyrene-induced DNA adduct formation in the liver of three strains of female mice with identical AhR(b2) genotype treated with 2,3,7,8-tetrachlorodibenzo-p-dioxin and/or benzo[a]pyrene. Journal of Applied Toxicology 28(6), 724-733.

Xue W, & Warshawsky D (2005). Metabolic activation of polycyclic and heterocyclic aromatic hydrocarbons and DNA damage: a review. Toxicol Appl. Pharmacol 206(1), 73-93.

Yalkowsky SH & Dannenfelser RM. 1992. Aquasol Database of Aqueous Solubility, Version 5. College of Pharmacy, Univ. of Ariz, Tucson, Az.

ZEK 01.2-08 (2008). Prüfung und Bewertung von Polycyclischen Aromatischen Kohlenwasserstoffen (PAK) bei der GS-Zeichen-Zuerkennung"
http://www.zls-muenchen.de/de/left/aktuell/pdf/zek_01_2-08_pak_verbindlich_mindermengen.pdf

Zhang J, Han IK, Zhang L, and Crain W (2008). Hazardous Chemicals in Synthetic Turf Materials and their Bioaccessibility in Digestive Fluids. Journal of Exposure Science & Environmental Epidemiology, Vol. 18, No. 6, 600-607.

Appendix 1: Tabular overview of the carcinogenicity studies evaluated for this dossier

Table 57: Overview of oral carcinogenicity studies

ID *	Reference	Route, Schedule	Species, Strain, Sex	Test item, Vehicle	Site/type of tumour	Dose level (mg BaP/kg bw/d)	No. affected animals	Total no. animals
1	Kroese et al., 2001	Oral, gavage, 5 d/wk x 104 wk	Rat, Wistar, M+F	BaP, soy oil	Liver	0	0	104
						3	6	103
						10	77	103
						30	100	104
2	Kroese et al., 2001	Oral, gavage, 5 d/wk x 104 wk	Rat, Wistar, M+F	BaP, soy oil	Forestomach	0	1	104
						3	14	103
						10	82	103
						30	102	104
14	Kroese et al., 2001	Oral, gavage, 5 d/wk x 104 wk	Rat, Wistar, M+F	BaP, soy oil	Auditory Canal	0	0	104
						3	0	104
						10	2	104
						30	37	104
3	Culp et al., 1998	Oral, diet, 7 d/wk x 104 wk	Mouse, B6C3F1, F	BaP, diet	Forestomach	0.000	1	48
						0.650	3	46
						3.250	36	46
						13.000	46	47
4	Culp et al., 1998	Oral, diet, 7 d/wk x 104 wk	Mouse, B6C3F1, F	BaP, diet	Oesophagus	0.000	0	48
						0.650	0	48
						3.250	2	45
						13.000	27	46

ID *	Reference	Route, Schedule	Species, Strain, Sex	Test item, Vehicle	Site/type of tumour	Dose level (mg BaP/kg bw/d)	No. affected animals	Total no. animals
5	Culp et al., 1998	Oral, diet, 7 d/wk x 104 wk	Mouse, B6C3F1, F	BaP, diet	Tongue	0.000	0	48
						0.650	0	48
						3.250	2	46
						13.000	23	48
6	Culp et al., 1998	Oral, diet, 7 d/wk x 104 wk	Mouse, B6C3F1, F	CTM1, diet	Forestomach	0.000	0	47
						0.029	2	47
						0.086	6	45
						0.286	3	47
						0.858	14	46
						1.742	15	45
						2.860	6	41
7	Culp et al., 1998	Oral, diet, 7 d/wk x 104 wk	Mouse, B6C3F1, F	CTM1, diet	Lung	0.000	2	47
						0.029	3	48
						0.086	4	48
						0.286	4	48
						0.858	27	47
						1.742	25	47
						2.860	21	45

ID *	Reference	Route, Schedule	Species, Strain, Sex	Test item, Vehicle	Site/type of tumour	Dose level (mg BaP/kg bw/d)	No. affected animals	Total no. animals
8	Culp et al., 1998	Oral, diet, 7 d/wk x 104 wk	Mouse, B6C3F1, F	CTM1, diet	Liver	0.000	0	47
						0.029	4	48
						0.086	2	46
						0.286	3	48
						0.858	13	45
						1.742	1	42
						2.860	5	43
16	Schneider 2002	Oral, diet, 7 d/wk x 104 wk	Mouse, B6C3F1, F	CTM1, diet	No. of tumour-bearing animals	0.000	5	48
						0.029	12	48
						0.086	14	48
						0.286	12	48
						0.858	40	48
						1.742	42	48
						2.860	43	48
9	Culp et al., 1998	Oral, diet, 7 d/wk x 104 wk	Mouse, B6C3F1, F	CTM2, diet	Forestomach	0.000	0	47
						0.143	3	47
						0.481	2	47
						1.443	13	44
10	Culp et al., 1998	Oral, diet, 7 d/wk x 104 wk	Mouse, B6C3F1, F	CTM2, diet	Lung	0.000	2	47
						0.143	4	48
						0.481	10	48
						1.443	23	47

ID *	Reference	Route, Schedule	Species, Strain, Sex	Test item, Vehicle	Site/type of tumour	Dose level (mg BaP/kg bw/d)	No. affected animals	Total no. animals
11	Culp et al., 1998	Oral, diet, 7 d/wk x 104 wk	Mouse, B6C3F1, F	CTM2, diet	Liver	0.000	0	47
						0.143	7	47
						0.481	4	47
						1.443	10	45
17	Schneider 2002	Oral, diet, 7 d/wk x 104 wk	Mouse, B6C3F1, F	CTM2, diet	No. of tumour-bearing animals	0.000	5	48
						0.143	17	48
						0.481	23	48
						1.443	44	48
12	Weyand et al., 1995	Oral, diet, 7 d/wk x 260 d	Mouse, A/J, F	BaP, diet	Forestomach	0.000	0	21
						1.624	5	25
						10.264	27	27
13	Weyand et al., 1995	Oral, diet, 7 d/wk x 260 d	Mouse, A/J, F	BaP, diet	Lung	0.000	4	21
						1.624	9	25
						10.264	14	27
15	Weyand et al., 1995	Oral, diet, 7 d/wk x 260 d	Mouse, A/J, F	MGP, diet	Lung	0.000	4	21
						0.276	19	27
						0.652	29	29

* Internal ID for identification of studies across appendices in this dossier

Table 58: Overview of inhalation carcinogenicity studies

ID *	Reference	Route, Administration	Schedule	Species, Strain, Sex	Test item	Site/type of tumour	Atmospheric concentration (µg BaP/m^3 air)	Dose level (mg BaP/kg bw/d)	No. affected animals	Total no. animals
44	Heinrich et al., 1994	Inhalation, aerosol, exposure: 10 mo., post-exposure: 20 mo.	17 h/d x 5 d/wk	Rat, Wistar, F	CTP	Lung	0	0	0	72
							20	0.0148	3	72
							46	0.0341	28	72
45	Heinrich et al., 1994	Inhalation, aerosol, exposure: 20 mo., post-exposure: 10 mo.	17 h/d x 5 d/wk	Rat, Wistar, F	CTP	Lung	0	0	0	72
							20	0.0148	24	72
							46	0.0341	70	72
46	Schulte et al., 1994	Inhalation, aerosol, whle-body, exposure: 44 wk, post-exposure: 0 wk	16 h/d x 5 d/wk	Mouse, NMRI/BR, F	CTP	Lung-adenomata	0	0	5	40
							50	0.0768	40	40
							90	0.1382	40	40
47	Schulte et al., 1994	Inhalation, aerosol, whle-body, exposure: 44 wk, post-exposure: 0 wk	16 h/d x 5 d/wk	Mouse, NMRI/BR, F	CTP	Lung-adenocarcinomata	0	0	0	40
							50	0.0768	10	40
							90	0.1382	33	40
48	Schulte et al., 1994	Inhalation, aerosol, whle-body, exposure: 44 wk, post-exposure: 0 wk	16 h/d x 5 d/wk	Mouse, NMRI/BR, F	CTP	Lung-squamous cell carcinomata	0	0	0	40
							50	0.0768	0	40
							90	0.1382	6	40
49	Schulte et al., 1994	Inhalation, aerosol, whle-body, exposure: 44 wk, post-exposure: 0 wk	16 h/d x 5 d/wk	Mouse, NMRI/BR, F	CTP	Lung - average number of nodules per animal	0	0	0,1	40
							50	0.0768	24,7	40
							90	0.1382	37,1	40

ID *	Reference	Route, Administration	Schedule	Species, Strain, Sex	Test item	Site/type of tumour	Atmospheric concentration (µg BaP/m^3 air)	Dose level (mg BaP/kg bw/d)	No. affected animals	Total no. animals
50	Schulte et al., 1994	Inhalation, aerosol, whle-body, exposure: 44 wk, post-exposure: 0 wk	16 h/d x 5 d/wk	Mouse, NMRI/BR, F	CTP	Lung - no. of tumour-bearing animals	0	0	5	40
							50	0.0768	40	40
							90	0.1382	40	40
51	Thyssen et al., 1981	Inhalation, aerosol, nose-only, exposure: 104 wk, post-exposure: 0 wk	4.5 h/d x 7 d/wk for the first 10 wk, then 3 hr/d x 7 d/wk	Hamster, Syrian golden, M	BaP	Nasal cavity	0	0	0	27
							2200	0.232	0	27
							9500	1.016	3	26
							46500	12.0528	1	25
52	Thyssen et al., 1981	Inhalation, aerosol, nose-only, exposure: 104 wk, post-exposure: 0 wk	4.5 h/d x 7 d/wk for the first 10 wk, then 3 hr/d x 7 d/wk	Hamster, Syrian golden, M	BaP	Larynx	0	0	0	27
							2200	0.572	0	27
							9500	2.4624	8	26
							46500	12.0528	13	25
53	Thyssen et al., 1981	Inhalation, aerosol, nose-only, exposure: 104 wk, post-exposure: 0 wk	4.5 h/d x 7 d/wk for the first 10 wk, then 3 hr/d x 7 d/wk	Hamster, Syrian golden, M	BaP	Trachea	0	0	0	27
							2200	0.572	0	27
							9500	2.4624	1	26
							46500	12.0528	3	25
54	Thyssen et al., 1981	Inhalation, aerosol, nose-only, exposure: 104 wk, post-exposure: 0 wk	4.5 h/d x 7 d/wk for the first 10 wk, then 3 hr/d x 7 d/wk	Hamster, Syrian golden, M	BaP	Pharynx	0	0	0	27
							2200	0.572	0	27
							9500	2.4624	6	26
							46500	12.0528	14	25

ID *	Reference	Route, Administration	Schedule	Species, Strain, Sex	Test item	Site/type of tumour	Atmospheric concentration (µg BaP/m^3 air)	Dose level (mg BaP/kg bw/d)	No. affected animals	Total no. animals
55	Thyssen et al., 1981	Inhalation, aerosol, nose-only, exposure: 104 wk, post-exposure: 0 wk	4.5 h/d x 7 d/wk for the first 10 wk, then 3 hr/d x 7 d/wk	Hamster, Syrian golden, M	BaP	Oesophagus	0	0	0	27
							2200	0.572	0	27
							9500	2.4624	0	26
							46500	12.0528	2	25
56	Thyssen et al., 1981	Inhalation, aerosol, nose-only, exposure: 104 wk, post-exposure: 0 wk	4.5 h/d x 7 d/wk for the first 10 wk, then 3 hr/d x 7 d/wk	Hamster, Syrian golden, M	BaP	Forestomach	0	0	0	27
							2200	0.572	0	27
							9500	2.4624	1	26
							46500	12.0528	1	25
58	Thyssen et al., 1981	Inhalation, aerosol, nose-only, exposure: 104 wk, post-exposure: 0 wk	4.5 h/d x 7 d/wk for the first 10 wk, then 3 hr/d x 7 d/wk	Hamster, Syrian golden, M	BaP	No. of tumours/tumour-bearing animal	0	0	1,1	27
							2200	0.572	1,3	27
							9500	2.4624	1,7	26
							46500	12.0528	2,5	25
59	Thyssen et al., 1981	Inhalation, aerosol, nose-only, exposure: 104 wk, post-exposure: 0 wk	4.5 h/d x 7 d/wk for the first 10 wk, then 3 hr/d x 7 d/wk	Hamster, Syrian golden, M	BaP	No. of tumour-bearing animals	0	0	14	27
							2200	0.572	17	27
							9500	2.4624	20	26
							46500	12.0528	15	25

* Internal ID for identification of studies across appendices in this dossier

Table 59: Overview of dermal carcinogenicity studies

ID *	Reference	Route, Schedule	Species, Strain, Sex	Test item, Vehicle	Site/type of tumour	Dose level (µg BaP/animal/d)	Dose level (mg BaP/kg bw/d)	No. affected animals	Total no. animals
18	Schmähl et al., 1977	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	BaP, acetone	Skin - any tumour	0	0	1	81
						1	0.04	11	77
						1.7	0.068	25	88
						3	0.12	45	81
19	Schmähl et al., 1977	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	BaP, acetone	Skin carcinoma	0	0	0	81
						1	0.04	10	77
						1.7	0.068	25	88
						3	0.12	43	81
20	Schmähl et al., 1977	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	C PAH, acetone	Skin - any tumour	0	0	1	81
						1	0.04	29	81
						1.7	0.068	57	88
						3	0.12	65	90
21	Schmähl et al., 1977	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	C PAH, acetone	Skin carcinoma	0	0	0	81
						1	0.04	25	81
						1.7	0.068	53	88
						3	0.12	63	90
23	Schmähl et al., 1977	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	C PAH + NC PAH, acetone	Skin-any tumour	0	0	0	81
						1	0.04	46	89
						1.7	0.068	57	93
						3	0.12	65	93

ID *	Reference	Route, Schedule	Species, Strain, Sex	Test item, Vehicle	Site/type of tumour	Dose level (µg BaP/animal/d)	Dose level (mg BaP/kg bw/d)	No. affected animals	Total no. animals
24	Schmähl et al., 1977	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	C PAH + NC PAH, acetone	Skin - carcinoma	0	0	0	81
						1	0.04	44	89
						1.7	0.068	54	93
						3	0.12	64	93
25	Habs et al., 1980	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	BaP, acetone	Skin - any tumour	0	0	0	35
						1.7	0.068	8	34
						2.8	0.112	24	35
						4.6	0.184	22	36
60	Habs et al., 1980	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	BaP, acetone	Skin - age-standardised tumour frequency (%)	0	0	0	35
						1.7	0.068	24,8	34
						2.8	0.112	89,3	35
						4.6	0.184	91,7	36
26	Habs et al., 1984	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	BaP, acetone	Skin - any tumour	0	0	0	20
						2	0.08	9	20
						4	0.16	17	20
27	Habs et al., 1984	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	BaP, acetone	Skin - carcinoma	0	0	0	20
						2	0.08	7	20
						4	0.16	17	20
28	Habs et al., 1984	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	Coloquint seed tar, acetone	Skin - any tumour	0	0	0	20
						0.138	0.00552	1	20
						0.552	0.02208	5	20

ID *	Reference	Route, Schedule	Species, Strain, Sex	Test item, Vehicle	Site/type of tumour	Dose level (µg BaP/animal/d)	Dose level (mg BaP/kg bw/d)	No. affected animals	Total no. animals
29	Habs et al., 1984	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	Coloquint seed tar, acetone	Skin - papilloma	0	0	0	20
						0.138	0.00552	0	20
						0.552	0.02208	2	20
30	Habs et al., 1984	Dermal, 2/wk, entire lifespan	Mouse, NMRI, F	Coloquint seed tar, acetone	Skin - carcinoma	0	0	0	20
						0.138	0.00552	1	20
						0.552	0.02208	3	20
31	Warshawsky & Barkley 1987	Dermal, 2/wk x 99 wk	Mouse, C3H/HeJ, F	BaP, acetone	Skin - any tumour	0	0	0	50
						12.5	0.5	48	50
42	Warshawsky & Barkley 1987	Dermal, 2/wk x 99 wk	Mouse, C3H/HeJ, F	BaP, acetone	Skin tumours (malignant only)	0	0	0	50
						12.5	0.5	47	50
32	FhI 1997	Dermal, 2/wk x 78 wk	Mouse, CD-[Crl:CDR-1(ICR)BR], F	Creosote, toluene	Skin - any tumour	0	0	0	62
						0.003	0.0001	0	62
						0.01	0.000333333	0	62
						0.03	0.001	1	62
						0.09	0.003	2	62
35	FhI 1997	Dermal, 2/wk x 78 wk	Mouse, CD-[Crl:CDR-1(ICR)BR], F	Creosote, toluene	Skin tumours (benign only)	0	0	0	62
						0.003	0.0001	0	62
						0.01	0.000333333	0	62
						0.03	0.001	0	62
						0.09	0.003	2	62

ID *	Reference	Route, Schedule	Species, Strain, Sex	Test item, Vehicle	Site/type of tumour	Dose level (µg BaP/animal/d)	Dose level (mg BaP/kg bw/d)	No. affected animals	Total no. animals
33	FhI 1997	Dermal, 2/wk x 78 wk	Mouse, CD-[Crl:CDR-1(ICR)BR], F	Creosote, toluene	Skin - any tumour	0	0	0	62
						0.027	0.0009	1	62
						0.08	0.002666667	3	62
						0.27	0.009	9	62
						0.8	0.026666667	23	62
34	FhI 1997	Dermal, 2/wk x 78 wk	Mouse, CD-[Crl:CDR-1(ICR)BR], F	Creosote, toluene	Skin - any tumour	0	0	0	62
						7.5	0.25	47	62
36	FhI 1997	Dermal, 2/wk x 78 wk	Mouse, CD-[Crl:CDR-1(ICR)BR], F	Creosote, toluene	Skin tumours (benign only)	0	0	0	62
						0.027	0.0009	0	62
						0.08	0.002666667	2	62
						0.27	0.009	6	62
						0.8	0.026666667	7	62
37	FhI 1997	Dermal, 2/wk x 78 wk	Mouse, CD-[Crl:CDR-1(ICR)BR], F	Creosote, toluene	Skin tumours (benign only)	0	0	0	62
						7.5	0.25	15	62

* Internal ID for identification of studies across appendices in this dossier

Appendix 2: Calculation of DMELs from T25 and BMD values

Table 60: Dermal DMELs calculated from T25 values for BaP based on the results obtained in animal carcinogenicity studies. All dose levels in mg BaP/kg bw/d, DMELs in ng BaP/kg bw/d

ID	Reference	Test Item	Route	Schedule	Species	Site/type of tumour	Dose level used for T25 calculation	T25	Modification factors		HT25	Assessment factors		Linearised		Large AF
									Lifetime	Sche-dule		AS*	BA**	DMEL 10^{-5}	DMEL 10^{-6}	DMEL
7	Culp et al., 1998	CTM 1	Oral	7 d/wk x 104 wk	Mouse	Lung	0.858	0.386	1	1	0.386	7	1/2.5	5.516	0.552	15.444
10	Culp et al., 1998	CTM 2	Oral	7 d/wk x 104 wk	Mouse	Lung	0.481	0.694	1	1	0.694	7	1/2.5	9.921	0.992	27.780
15	Weyand et al., 1995	MGP	Oral	7 d/wk for 260 d	Mouse	Lung	0.276	0.108	260/730	1	0.039	7	1/2.5	0.554	0.055	1.551
16	Schneider et al., 2002	CTM 1	Oral	7 d/wk x 104 wk	Mouse	Any site	0.086	0.103	1	1	0.103	7	1/2.5	1.467	0.147	4.109
17	Schneider et al., 2002	CTM 2	Oral	7 d/wk x 104 wk	Mouse	Any site	0.143	0.128	1	1	0.128	7	1/2.5	1.830	0.183	5.124
21	Schmähl et al., 1977	C PAH	Dermal	2 d/wk, entire lifespan	Mouse	Skin carcinoma	0.068	0.028	1	2/7	0.008	7	1/2.5	0.115	0.012	0.323
24	Schmähl et al., 1977	C PAH + NC PAH	Dermal	2 d/wk, entire lifespan	Mouse	Skin carcinoma	0.040	0.020	1	2/7	0.006	7	1/2.5	0.083	0.008	0.231
33	FhI 1997	Creosote	Dermal	2 d/wk x 78 wk	Mouse	Skin – any tumour	0.009	0.016	78/104	2/7	0.003	7	1/2.5	0.047	0.005	0.133
44	Heinrich et al., 1994	CTP	Inhalation	5 d/wk, 10 mo. Exposure	Rat	Lung	0.034	0.022	1/3	5/7	0.005	4	1/2.5	0.130	0.013	0.209
45	Heinrich et al., 1994	CTP	Inhalation	5 d/wk, 20 mo. Exposure	Rat	Lung	0.034	0.009	2/3	5/7	0.004	4	1/2.5	0.104	0.010	0.167

ID	Reference	Test Item	Route	Schedule	Species	Site/type of tumour	Dose level used for T25 calculation	T25	Modification factors		HT25	Assessment factors		Linearised		Large AF
									Lifetime	Sche-dule		AS*	BA**	DMEL 10^{-5}	DMEL 10^{-6}	DMEL
46	Schulte et al., 1994	CTP	Inhalation	5 d/wk x 44 wk	Mouse	Lung adenoma	0.077	0.019	44/104	5/7	0.006	7	1/2.5	0.083	0.008	0.232

* Allometric scaling; ** Bioavailability factor in order to account for the assumption of 50 % absorption across all routes in animal experiments using organic solvents as vehicle vs. 20 % absorption in the human exposure situation (dermal absorption from a sweat matrix)

Table 61: Dermal DMELs calculated from BMD_{10} values for BaP based on the results obtained in animal carcinogenicity studies and using the Probit model. All dose levels are given as rounded values in mg BaP/kg bw/d

ID	Reference	Route	Schedule	Species	Site/type of tumour	BMD_{10} (Probit)	Modification factors		$HBMD_{10}$ (Probit)	Assessment factors		Linearised		Large AF
							Life-time	Sche-dule		AS*	BA**	DMEL 10^{-5}	DMEL 10^{-6}	DMEL
7	Culp et al., 1998	Oral	7 d/wk x 104 wk	Mouse	Lung	0.159	1	1	0.159	7	1/2.5	5.679	0.568	15.901
10	Culp et al., 1998	Oral	7 d/wk x 104 wk	Mouse	Lung	0.294	1	1	0.294	7	1/2.5	10.493	1.049	29.379
16	Schneider et al., 2002	Oral	7 d/wk x 104 wk	Mouse	Any site	0.146	1	1	0.146	7	1/2.5	5.214	0.521	14.600
17	Schneider et al., 2002	Oral	7 d/wk x 104 wk	Mouse	Any site	0.089	1	1	0.089	7	1/2.5	3.161	0.316	8.850
21	Schmähl et al., 1977	Dermal	2 d/wk, entire lifespan	Mouse	Skin carcinoma	0.015	1	2/7	0.004	7	1/2.5	0.156	0.016	0.438
24	Schmähl et al., 1977	Dermal	2 d/wk, entire lifespan	Mouse	Skin carcinoma	0.003	1	2/7	0.001	7	1/2.5	0.026	0.003	0.074
33	FhI 1997	Dermal	2 d/wk x 78 wk	Mouse	Skin - any tumour	0.005	78/104	2/7	0.001	7	1/2.5	0.040	0.004	0.112

* Allometric scaling; ** Bioavailability factor in order to account for the assumption of 50 % absorption across all routes in animal experiments using organic solvents as vehicle vs. 20 % absorption in the human exposure situation (dermal absorption from a sweat matrix)

Table 62: Dermal DMELs calculated from $BMDL_{10}$ values for BaP based on the results obtained in animal carcinogenicity studies and using the Probit model. All dose levels are given as rounded values in mg BaP/kg bw/d

ID	Reference	Route	Schedule	Species	Site/type of tumour	$BMDL_{10}$ (Probit)	Modification factors		$HBMDL_{10}$ (Probit)	Assessment factors		Linearised		Large AF
							Life-time	Sche-dule		AS*	BA**	DMEL 10^{-5}	DMEL 10^{-6}	DMEL
7	Culp et al., 1998	Oral	7 d/wk x 104 wk	Mouse	Lung	0.069	1	1	0.069	7	1/2.5	2.479	0.248	6.941
10	Culp et al., 1998	Oral	7 d/wk x 104 wk	Mouse	Lung	0.130	1	1	0.130	7	1/2.5	4.657	0.466	13.040
16	Schneider et al., 2002	Oral	7 d/wk x 104 wk	Mouse	Any site	0.048	1	1	0.048	7	1/2.5	1.713	0.171	4.797
17	Schneider et al., 2002	Oral	7 d/wk x 104 wk	Mouse	Any site	0.035	1	1	0.035	7	1/2.5	1.259	0.126	3.524
21	Schmähl et al., 1977	Dermal	2 d/wk, entire lifespan	Mouse	Skin carcinoma	0.007	1	2/7	0.002	7	1/2.5	0.074	0.007	0.207
24	Schmähl et al., 1977	Dermal	2 d/wk, entire lifespan	Mouse	Skin carcinoma	0.000	1	2/7	0.000	7	1/2.5	$1.3x10^{-4}$	$1.3x10^{-5}$	$4x10^{-4}$
33	FhI 1997	Dermal	2 d/wk x 78 wk	Mouse	Skin - any tumour	0.003	78/104	2/7	0.001	7	1/2.5	0.026	0.003	0.072

* Allometric scaling; ** Bioavailability factor in order to account for the assumption of 50 % absorption across all routes in animal experiments using organic solvents as vehicle vs. 20 % absorption in the human exposure situation (dermal absorption from a sweat matrix)

Table 63: Dermal DMELs calculated from BMD_{10} values for BaP based on the results obtained in animal carcinogenicity studies and using the Multistage Cancer model. All dose levels are given as rounded values in mg BaP/kg bw/d

ID	Reference	Route	Schedule	Species	Site/type of tumour	BMD_{10} (Multi-stage Cancer)	Modification factors		$HBMD_{10}$ (Multi-stage Cancer)	Assessment factors		Linearised		Large AF
							Life-time	Sche-dule		AS*	BA**	DMEL 10^{-5}	DMEL 10^{-6}	DMEL
7	Culp et al., 1998	Oral	7 d/wk x 104 wk	Mouse	Lung	0.298	1	1	0.298	7	1/2.5	10.629	1.063	29.761
10	Culp et al., 1998	Oral	7 d/wk x 104 wk	Mouse	Lung	0.282	1	1	0.282	7	1/2.5	10.081	1.008	28.228
16	Schneider et al., 2002	Oral	7 d/wk x 104 wk	Mouse	Any site	0.102	1	1	0.102	7	1/2.5	3.639	0.364	10.189
17	Schneider et al., 2002	Oral	7 d/wk x 104 wk	Mouse	Any site	0.084	1	1	0.084	7	1/2.5	2.984	0.298	8.356
21	Schmähl et al., 1977	Dermal	2 d/wk, entire lifespan	Mouse	Skin carcinoma	0.010	1	2/7	0.003	7	1/2.5	0.098	0.010	0.275
24	Schmähl et al., 1977	Dermal	2 d/wk, entire lifespan	Mouse	Skin carcinoma	0.009	1	2/7	0.002	7	1/2.5	0.088	0.009	0.246
33	FhI 1997	Dermal	2 d/wk x 78 wk	Mouse	Skin - any tumour	0.006	78/104	2/7	0.001	7	1/2.5	0.046	0.005	0.129

* Allometric scaling; ** Bioavailability factor in order to account for the assumption of 50 % absorption across all routes in animal experiments using organic solvents as vehicle vs. 20 % absorption in the human exposure situation (dermal absorption from a sweat matrix)

Table 64: Dermal DMELs calculated from BMDL10 values for BaP based on the results obtained in animal carcinogenicity studies and using the Multistage Cancer model. All dose levels are given as rounded values in mg BaP/kg bw/d

ID	Reference	Route	Schedule	Species	Site/type of tumour	$BMDL_{10}$ (Multi-stage Cancer)	Modification factors		$HBMDL_{10}$ (Multi-stage Cancer)	Assessment factors		Linearised		Large AF
							Life-time	Sche-dule		AS*	BA**	DMEL 10^{-5}	DMEL 10^{-6}	DMEL
7	Culp et al., 1998	Oral	7 d/wk x 104 wk	Mouse	Lung	0.238	1	1	0.238	7	1/2.5	8.511	0.851	23.829
10	Culp et al., 1998	Oral	7 d/wk x 104 wk	Mouse	Lung	0.183	1	1	0.183	7	1/2.5	6.552	0.655	18.347
16	Schneider et al., 2002	Oral	7 d/wk x 104 wk	Mouse	Any site	0.083	1	1	0.083	7	1/2.5	2.977	0.298	8.336
17	Schneider et al., 2002	Oral	7 d/wk x 104 wk	Mouse	Any site	0.057	1	1	0.057	7	1/2.5	2.031	0.203	5.687
21	Schmähl et al., 1977	Dermal	2 d/wk, entire lifespan	Mouse	Skin carcinoma	0.008	1	2/7	0.002	7	1/2.5	0.085	0.009	0.239
24	Schmähl et al., 1977	Dermal	2 d/wk, entire lifespan	Mouse	Skin carcinoma	0.008	1	2/7	0.002	7	1/2.5	0.077	0.008	0.216
33	FhI 1997	Dermal	2 d/wk x 78 wk	Mouse	Skin - any tumour	0.005	78/104	2/7	0.001	7	1/2.5	0.035	0.004	0.099

* Allometric scaling; ** Bioavailability factor in order to account for the assumption of 50 % absorption across all routes in animal experiments using organic solvents as vehicle vs. 20 % absorption in the human exposure situation (dermal absorption from a sweat matrix)

Appendix 3: Analytical Data Tables

Table 65: Statistical evaluation of the analytical data on BaP, 16 EPA-PAHs and PAH-6 for all samples and for the product categories

Category	Parameter		BaP			EPA-PAH			PAH-6 [1]		
All data	**Total number of values**		5278			5346			4961		
	Number of positive values		428 (8.1 %)			4170 (78 %)			798 (16.1 %)		
	Mean of positive values (mg/kg)		24.1			66.9			107.7		
	Maximum (mg/kg)		1200			25400			6930		
	Frequency distribution	**Range**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**
		n.d.	4850	91.9	91.9	1176	22.0	22.0	4163	83.9	83.9
		<0.1	37	0.7	92.6	140	2.6	24.6	56	1.1	85.0
		0.1-<0.5	102	1.9	94.5	794	14.9	39.5	186	3.7	88.8
		0.5-<1	40	0.8	95.3	563	10.5	50.0	95	1.9	90.7
		1-<5	73	1.4	96.7	1396	26.1	76.1	172	3.5	94.2
		5-<10	29	0.5	97.2	482	9.0	85.1	32	0.6	94.8
		10-<100	128	2.4	99.6	540	10.1	95.2	116	2.3	97.2
		> 100	19	0.4	100.0	255	4.8	100.0	141	2.8	100.0

Category	Parameter		BaP			EPA-PAH			PAH-6 [1]		
Category 1: Electrical devices	**Total number of values**		1705			1705			1705		
	Number of positive values		138 (8.1 %)			1428 (83.8 %)			208 (12.2 %)		
	Mean of positive values (mg/kg)		16.4			59.9			130.8		
	Maximum (mg/kg)		195			4516			1915		
	Frequency distribution	**Range**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**
		n.d.	1567	91.9	91.9	277	16.2	16.2	1497	87.8	87.8
		<0,1	5	0.3	92.2	36	2.1	18.4	7	0.4	88.2
		0.1-<0.5	32	1.9	94.1	250	14.7	33.0	43	2.5	90.7
		0.5-<1	13	0.8	94.8	222	13.0	46.0	21	1.2	92.0
		1-<5	25	1.5	96.3	481	28.2	74.3	43	2.5	94.5
		5-<10	11	0.6	97.0	146	8.6	82.8	3	0.2	94.7
		10-<100	45	2.6	99.6	197	11.6	94.4	38	2.2	96.9
		> 100	7	0.4	100.0	96	5.6	100.0	53	3.1	100.0

Category	Parameter		BaP			EPA-PAH			PAH-6 [1]		
Category 2: Grips, handles	**Total number of values**		541			541			496		
	Number of positive values		52 (10 %)			429 (79 %)			91 (18 %)		
	Mean of positive values (mg/kg)		25.5			90.7			164.6		
	Maximum (mg/kg)		98			3699			2482.8		
	Frequency distribution	**Range**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**
		n.d.	489	90.4	90.4	112	20.7	20.7	405	81.7	81.7
		<0.1	1	0.2	90.6	19	3.5	24.2	4	0.8	82.5
		0.1-<0.5	12	2.2	92.8	76	14.0	38.3	26	5.2	87.7
		0.5-<1	0	0.0	92.8	45	8.3	46.6	8	1.6	89.3
		1-<5	7	1.3	94.1	142	26.2	72.8	14	2.8	92.1
		5-<10	3	0.6	94.6	47	8.7	81.5	2	0.4	92.5
		10-<100	29	5.4	100.0	60	11.1	92.6	9	1.8	94.4
		> 100	0	0.0	100.0	40	7.4	100.0	28	5.6	100.0

Category	Parameter		BaP			EPA-PAH			PAH-6 [1]		
Category 3: Contact areas of sports equip-ment and other articles	**Total number of values**		120			121			108		
	Number of positive values		15 (12.5 %)			114 (94.2 %)			29 (27 %)		
	Mean of positive values (mg/kg)		38.3			88.9			138.9		
	Maximum (mg/kg)		128.65			1801.5			995.1		
	Frequency distribution	**Range**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**
		n.d.	105	87.5	87.5	7	5.8	5.8	79	73.1	73.1
		<0.1	0	0.0	87.5	2	1.7	7.4	3	2.8	75.9
		0.1-<0.5	3	2.5	90.0	14	11.6	19.0	6	5.6	81.5
		0.5-<1	0	0.0	90.0	6	5.0	24.0	2	1.9	83.3
		1-<5	3	2.5	92.5	46	38.0	62.0	6	5.6	88.9
		5-<10	2	1.7	94.2	19	15.7	77.7	2	1.9	90.7
		10-<100	4	3.3	97.5	16	13.2	90.9	2	1.9	92.6
		> 100	3	2.5	100.0	11	9.1	100.0	8	7.4	100.0

Category	Parameter		BaP			EPA-PAH			PAH-6 [1]		
Category 4: Toys	**Total number of values**		340			340			246		
	Number of positive values		18 (5.3 %)			277 (81.5 %)			30 (12.2 %)		
	Mean of positive values (mg/kg)		9.56			21.47			37.10		
	Maximum (mg/kg)		65.90			1992.50			446.85		
	Frequency distribution	**Range**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**
		n.d.	322	94.7	94.7	63	18.5	18.5	216	87.8	87.8
		<0.1	2	0.6	95.3	5	1.5	20.0	0	0.0	87.8
		0.1-<0.5	4	1.2	96.5	74	21.8	41.8	14	5.7	93.5
		0.5-<1	2	0.6	97.1	30	8.8	50.6	2	0.8	94.3
		1-<5	4	1.2	98.2	95	27.9	78.5	4	1.6	95.9
		5-<10	3	0.9	99.1	40	11.8	90.3	1	0.4	96.3
		10-<100	3	0.9	100.0	24	7.1	97.4	6	2.4	98.8
		> 100	0	0.0	100.0	9	2.6	100.0	3	1.2	100.0

Category	Parameter		BaP			EPA-PAH			PAH-6 [1]		
Category 5: Materials with close contact to the body	**Total number of values**		535			574			427		
	Number of positive values		63 (11.8%)			470 (81.9%)			165 (38.6%)		
	Mean of positive valuesmg/kg		4.0			24.4			11.1		
	Maximummg/kg		111.3			1503			411.9		
	Frequency distribution	**Range**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**
		n.d.	472	88.2	88.2	104	18.1	18.1	262	61.4	61.4
		<0.1	20	3.7	92.0	11	1.9	20.0	7	1.6	63.0
		0.1-<0.5	15	2.8	94.8	57	9.9	30.0	43	10.1	73.1
		0.5-<1	15	2.8	97.6	45	7.8	37.8	28	6.6	79.6
		1-<5	5	0.9	98.5	160	27.9	65.7	56	13.1	92.7
		5-<10	1	0.2	98.7	84	14.6	80.3	11	2.6	95.3
		10-<100	6	1.1	99.8	96	16.7	97.0	15	3.5	98.8
		> 100	1	0.2	100.0	17	3.0	100.0	5	1.2	100.0

Category	Parameter		BaP			EPA-PAH			PAH-6 [1]		
Category 6: Other products with skin contact	**Total number of values**		460			467			425		
	Number of positive values		24 (5 %)			358 (77 %)			39 (9 %)		
	Mean of positive values mg/kg		41.4			59.0			215.1		
	Maximummg/kg		530			9300			3380		
	Frequency distribution	**Range**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**
		n.d.	436	94.8	94.8	109	23.3	23.3	386	90.8	90.8
		<0.1	0	0.0	94.8	12	2.6	25.9	2	0.5	91.3
		0.1-<0.5	6	1.3	96.1	106	22.7	48.6	6	1.4	92.7
		0.5-<1	2	0.4	96.5	55	11.8	60.4	6	1.4	94.1
		1-<5	3	0.7	97.2	123	26.3	86.7	6	1.4	95.5
		5-<10	1	0.2	97.4	24	5.1	91.9	2	0.5	96.0
		10-<100	10	2.2	99.6	22	4.7	96.6	8	1.9	97.9
		> 100	2	0.4	100.0	16	3.4	100.0	9	2.1	100.0

Category	Parameter		BaP			EPA-PAH			PAH-6 [1]		
Category 7: Tyres, rolls	**Total number of values**		35			35			35		
	Number of positive values		14 (40 %)			34 (97 %)			19 (54 %)		
	Mean of positive values mg/kg		198.8			1652.2			867.7		
	Maximummg/kg		1200			25400			6930		
	Frequency distribution	**Range**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**
		n.d.	21	60.0	60.0	1	2.9	2.9	16	45.7	45.7
		<0.1	1	2.9	62.9	0	0.0	2.9	3	8.6	54.3
		0.1-<0.5	1	2.9	65.7	3	8.6	11.4	2	5.7	60.0
		0.5-<1	0	0.0	65.7	3	8.6	20.0	0	0.0	60.0
		1-<5	1	2.9	68.6	10	28.6	48.6	2	5.7	65.7
		5-<10	3	8.6	77.1	3	8.6	57.1	0	0.0	65.7
		10-<100	4	11.4	88.6	4	11.4	68.6	4	11.4	77.1
		> 100	4	11.4	100.0	11	31.4	100.0	8	22.9	100.0

Category	Parameter		BaP			EPA-PAH			PAH-6 [1]		
Category 8: Other products	**Total number of values**		1519			1540			1514		
	Number of positive values		104 (7 %)			1040 (67 %)			217 (14 %)		
	Mean of positive valuesmg/kg		18.7			47.7			54.7		
	Maximummg/kg		380			9574			1994		
	Frequency distribution	**Range**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**	**Number**	**Percent**	**Cumulative percent**
		n.d.	1415	93.2	93.2	500	32.5	32.5	1297	85.7	85.7
		<0.1	8	0.5	93.7	48	3.1	35.6	30	2.0	87.6
		0.1-<0.5	29	1.9	95.6	207	13.4	49.0	46	3.0	90.7
		0.5-<1	8	0.5	96.1	155	10.1	59.1	28	1.8	92.5
		1-<5	25	1.6	97.8	335	21.8	80.8	41	2.7	95.2
		5-<10	5	0.3	98.1	119	7.7	88.6	11	0.7	96.0
		10-<100	27	1.8	99.9	121	7.9	96.4	34	2.2	98.2
		> 100	2	0.1	100.0	55	3.6	100.0	27	1.8	100.0

[1]By chemical analysis only the content of 6 out of the 8 PAHs adressed in this restriction proposal were available. The sum of these 6 are assumed as surrogate for the sum of 8 and abbreviated as EU 8.

Appendix 4: Calculation of migration rates

Table 66: Estimation of migration rates in aqueous and lipohilic simulants/ static conditions

Sample	Analyte	Simulant	Contact Time $T_{contact}$	Sample weight Q_{prod} (g)	Product area A_{prod} (cm^2)	Concentration in the sample Fc_{prod} (mg/kg)	Concentration in the simulant (mg/kg)	Migration related to sample area (mg/dm^2)	Rate (fraction) of migrating substance Fc_{migr} (%/h)	Ref.
Beach sandals	BaP	95 % ethanol	24 h	60	54	8.7	0.2	0.035	0.16	UBA, 2010
		isooctane					0.3	0.055	0.24	UBA, 2010
	EPA-PAH	95 % ethanol	24 h	60	54	546	23.7	4.57	0.3	UBA, 2010
		isooctane					44.9	8.31	0.57	UBA, 2010
Grip of a window wiper	BaP	water	2 h	100	100			0.0013		BfR, 2009
		isooctane						5.56		BfR, 2009
	EPA-PAH	water	2 h	100	100	10707		0.034	0.0015	BfR, 2009
		isooctane						131.014	6.1	BfR, 2009
Elastomer 1	BaP	aqueous sweat simulant	2 h			355	0.045		0.01	WDK, 2007
	EU-PAH					1077	0.112		0.01	WDK, 2007
Elastomer 2	BaP	aqueous sweat simulant	2 h			3.85	< 0.001		< 0.01	WDK, 2007
	EU-PAH					10.36	< 0.001		< 0.001	WDK, 2007

Sample	Analyte	Simulant	Contact Time $T_{contact}$	Sample weight Q_{prod} (g)	Product area A_{prod} (cm^2)	Concentration in the sample Fc_{prod} (mg/kg)	Concentration in the simulant (mg/kg)	Migration related to sample area (mg/dm^2)	Rate (fraction) of migrating substance Fc_{migr} (%/h)	Ref.
Cured rubber	BaP	artificial saliva	7 d		100	10.8		n.d. (< 0.000002)		Hamm et al., 2009
	EU-PAH					23.6		n.d. (< 0.0002)		Hamm et al., 2009
	EPA-PAH					367		0.001	0.001	Hamm et al., 2009
	BaP	aqueous sweat simulant	7 d		100	10.8		n.d. (< 0.000002)		Hamm et al., 2009
	EU-PAH					23.6		n.d. (< 0.0002)		Hamm et al., 2009
	EPA-PAH					367		0.001	0.001	Hamm et al., 2009
	BaP	rain water	7 d		100	10.8		n.d. (< 0.000002)		Hamm et al., 2009
	EU-PAH					23.6		n.d. (< 0.0002)		Hamm et al., 2009
	EPA-PAH					367		0.001	0.001	Hamm et al., 2009
	BaP	drinkung water	7 d		100	10.8		n.d. (< 0.000002)		Hamm et al., 2009
	EU-PAH					23.6		n.d. (< 0.0002)		Hamm et al., 2009
	EPA-PAH					367		0.0002	0.0002	Hamm et al., 2009

Table 67: Data on migration tests with latex gloves/ dynamic design (glove and sweat stimulant)

Sample	Material	Contact conditions		Sample weight Q_{prod} (g)	BaP concentration in the sample Fc_{prod} (mg/kg = µg/g)	Product area A_{prod} (cm^2)	Glove contact area A_g (cm^2)	Fraction of contact area $F_{contact}$	Migrated amount of BaP during contact time M_g (µg)	Migrated amount of BaP per hour $M_{g/h}$ (µg/h)	Rate (fraction) of migrating BaP Fc_{migr} (%/h)	Ref.
		Mechanics	Contact Time T_{cont}									
Hammer grip	Rubber	hold and move, strong power	1 h	90	42	160	140	0.88	320	320	9.67	Sander, 2006
Cover of steering wheel/ case 1	Rubber	hold and move		300	35	1500	140	0.09	100	100	10.20	Sander, 2006
Cover of steering wheel/ case 2	Rubber	hold		300	35	1500	140	1 [1)]	100	100	0.95	Sander, 2006
Bulb of a hooter	Butyl rubber	hold and move		35	530	130	140	1.08	4	4	0.022	Sander, 2006
Torch handle	Unknown	hold and friction	1 min	150	12.8	119	78	0.66	0.38	22.8	1.81	Hutzler, 2009
Measuring tape	Rubber			39	11.8	90	54	0.60	0.25	15	5.43	Hutzler, 2009
Torch handle	Unknown	hold	10 min	150	12.8	119	78	1 [1)]	0.32	1.92	0.15	Hutzler, 2009
Measuring tape	Rubber			39	11.8	90	54	1 [1)]	0.179	1.074	0.23	Hutzler, 2009

Appendix 5: Calculation of external dermal exposure against PAH-contaminated articles

Table 68: Calculation of external dermal exposure of adults by BaP containing articles

Category	Sample	Concentration in product Fc_{prod} (mg/kg)			Q_{prod} (g)	$F_{contact}$	$T_{contact}$ (h)	n	$D_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc_{migr} (%/h) = 10	Fc_{migr} (%/h) = 1.5	Fc_{migr} (%/h) = 0.2
Grip	Tool	98	2393.0	354.0	100	0.8	1	1	13066.67	1960.00	261.33
Grip	Tool	95	2100.0	1453.8	100	0.8	1	1	12666.67	1900.00	253.33
Grip black	No information, assumed as bicycle grip	94	3699.0	2482.8	200	0.8	1	2	50133.33	7520.00	1002.67
Grip	Tool	75.7	1792.0	375.6	100	0.8	1	1	10097.78	1514.67	201.96
Grip black	No information, assumed as bicycle grip	72.0	1523.0	765.0	200	0.8	1	2	38400.00	5760.00	768.00
Grip	Kitchen knife	61	1743.0	1202.0	100	0.8	1	2	16266.67	2440.00	325.33
Grip	Window wiper	60	930.0	340.0	100	0.8	8	1	64000.00	9600.00	1280.00
Grip black	No information, assumed as bicycle grip	52	>2226	1015.0	200	0.8	1	2	27733.33	4160.00	554.67
Grip	Tool	48	1636.0	244.0	100	0.8	1	1	6400.00	960.00	128.00
Grip	Tool	44	1689.0	244.0	100	0.8	1	1	5866.67	880.00	117.33
Grip	Tool	40	552.0	210.0	100	0.8	1	1	5333.33	800.00	106.67
Grip	Tool	35	556.0	332.0	100	0.8	1	1	4666.67	700.00	93.33
Grip black	No information, assumed as bicycle grip	34	>1262	632.0	200	0.8	1	2	18133.33	2720.00	362.67
Grip black	No information, assumed as bicycle grip	33	876.0	501.0	200	0.8	1	2	17600.00	2640.00	352.00

Category	Sample	Concentration in product Fc $_{prod}$ (mg/kg)			Q $_{prod}$ (g)	F $_{contact}$	T $_{contact}$ (h)	n	D $_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc $_{migr}$ (%/h) = 10	Fc $_{migr}$ (%/h) = 1.5	Fc $_{migr}$ (%/h) = 0.2
Grip black	No information, assumed as bicycle grip	32	1057.0	495.6	200	0.8	1	2	17066.67	2560.00	341.33
Grip	Tool	32	678.0	173.0	100	0.8	1	1	4266.67	640.00	85.33
Grip	Tool	30	610.0	155.5	100	0.8	1	1	4000.00	600.00	80.00
Grip	Tool	30	549.3	296.0	100	0.8	1	1	4000.00	600.00	80.00
Grip	Tool	30	328.0	197.9	100	0.8	1	1	4000.00	600.00	80.00
Grip black	No information, assumed as bicycle grip	27	778.0	251.0	200	0.8	1	2	14400.00	2160.00	288.00
Grip	Tool	26	351.5	188.0	100	0.8	1	1	3466.67	520.00	69.33
Grip	Tool	24	1779.0	120.0	100	0.8	1	1	3200.00	480.00	64.00
Grip	Tool	24	454.0	114.0	100	0.8	1	1	3200.00	480.00	64.00
Grip black	No information, assumed as bicycle grip	21	278.0	124.0	200	0.8	1	2	11200.00	1680.00	224.00
Grip	Tool	18	330.8	165.0	100	0.8	1	1	2400.00	360.00	48.00
Grip	Tool	18	239.0	100.0	100	0.8	1	1	2400.00	360.00	48.00
Grip	Tool	14	754.0	317.7	100	0.8	1	1	1866.67	280.00	37.33
Grip black	Kitchen knife	10	240.0	99.2	100	0.8	1	2	2666.67	400.00	53.33
Grip black	No information, assumed as bicycle grip	9	210.0	59.0	200	0.8	1	2	4800.00	720.00	96.00
Grip black	No information, assumed as bicycle grip	4.7	199.1	18.1	200	0.8	1	2	2497.42	374.61	49.95
Grip	Tool	4.6	288.0	8.2	100	0.8	1	1	613.33	92.00	12.27

Category	Sample	Concentration in product Fc $_{prod}$ (mg/kg)			Q $_{prod}$ (g)	F $_{contact}$	T $_{contact}$ (h)	n	D $_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc $_{migr}$ (%/h) = 10	Fc $_{migr}$ (%/h) = 1.5	Fc $_{migr}$ (%/h) = 0.2
Grip	Tool	4	86.0	28.0	100	0.8	1	1	533.33	80.00	10.67
Grip black	No information, assumed as bicycle grip	4.0	90.2	29.9	200	0.8	1	2	2133.33	320.00	42.67
Grip black	Torch	3.9	126.0	48.2	100	0.5	1	1	325.00	48.75	6.50
Grip	Walking frame	2.5	110.0	34.2	200	0.8	2	2	2666.67	400.00	53.33
Grip black	No information, assumed as bicycle grip	2.3	39.4	13.1	200	0.8	1	2	1251.56	187.73	25.03
Grip	Tool	1.3	25.4	8.2	100	0.8	1	2	346.67	52.00	6.93
Grip black	Bicycle	0.5	22.0	0.5	200	0.8	1	2	266.67	40.00	5.33
Grip	Trolley bag	0.3	64.1	1.1	200	0.8	1	2	160.00	24.00	3.20
Grip black	No detailed information	0.3	5.5	1.8	100		1	2	0.00	0.00	0.00
Grip black	bicycle	0.2	0.2	0.2	200	0.8	1	2	106.67	16.00	2.13
Grip	Tool	0.2	10.0	1.0	100	0.8	1	1	26.67	4.00	0.53
Grip	Tool	0.2	136.1	1.6	100	0.8	1	1	26.67	4.00	0.53
Grip	Tool	0.2	9.5	0.2	100	0.8	1	1	26.67	4.00	0.53
Grip black	No information, assumed as bicycle grip	0.2	19.2	2.9	100	0.8	1	2	53.33	8.00	1.07
Plastic grip	No detailed information	0.2	14.1	0.2	100	0.8	1	1	26.67	4.00	0.53
Grip	Tool	0.2	10.0	1.0	100	0.8	1	1	26.67	4.00	0.53
Grip black	Bicycle	0.2	0.2	0.2	200	0.8	1	2	106.67	16.00	2.13
Grip	Tool	0.2	5.9	0.8	100	0.8	1	1	21.33	3.20	0.43

Category	Sample	Concentration in product Fc $_{prod}$ (mg/kg)			Q $_{prod}$ (g)	F $_{contact}$	T $_{contact}$ (h)	n	D $_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc $_{migr}$ (%/h) = 10	Fc $_{migr}$ (%/h) = 1.5	Fc $_{migr}$ (%/h) = 0.2
Grip	Tool	0.11	16.0	1.3	100	0.8	1	1	14.67	2.20	0.29
Grip	Tool	0.1	6.1	1.4	100	0.8	1	1	13.33	2.00	0.27
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	128.65	1543.0	995.1	200	0.8	2	1	68613.33	10292.00	1372.27
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	127	1774.0	783.0	200	0.8	2	1	67733.33	10160.00	1354.67
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	116.95	1329.5	792.8	200	0.8	2	1	62373.33	9356.00	1247.47
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	80.3	1801.5	403.3	200	0.8	2	1	42826.67	6424.00	856.53
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	43	604.9	194.7	200	0.8	2	1	22933.33	3440.00	458.67
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	41.5	1003.5	522.5	200	0.8	2	1	22133.33	3320.00	442.67

Category	Sample	Concentration in product Fc $_{prod}$ (mg/kg)			Q $_{prod}$ (g)	F $_{contact}$	T $_{contact}$ (h)	n	D $_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc $_{migr}$ (%/h) = 10	Fc $_{migr}$ (%/h) = 1.5	Fc $_{migr}$ (%/h) = 0.2
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	14.15	682.0	108.2	200	0.8	2	1	7546.67	1132.00	150.93
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	7.5	128.3	36.1	200	0.8	2	1	4000.00	600.00	80.00
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	6.18	119.3	105.7	200	0.8	2	1	3296.00	494.40	65.92
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	4.85	285.0	57.5	200	0.8	2	1	2586.67	388.00	51.73
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	3	88.4	7.2	200	0.8	2	1	1600.00	240.00	32.00
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	1.2	22.0	7.3	200	0.8	2	1	640.00	96.00	12.80
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	0.2	65.0	2.7	200	0.8	2	1	106.67	16.00	2.13

Category	Sample	Concentration in product Fc $_{prod}$ (mg/kg)			Q $_{prod}$ (g)	F $_{contact}$	T $_{contact}$ (h)	n	D $_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc $_{migr}$ (%/h) = 10	Fc $_{migr}$ (%/h) = 1.5	Fc $_{migr}$ (%/h) = 0.2
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	0.2	11.0	0.7	200	0.8	2	1	106.67	16.00	2.13
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	0.2	13.1	1.1	200	0.8	2	1	106.67	16.00	2.13
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	111.3	1198.0	411.9	200	0.4	1	2	29680.00	4452.00	593.60
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	34.7	812.0	160.1	200	0.4	1	2	9253.33	1388.00	185.07
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	19.5	319.6	173.8	200	0.4	1	2	5200.00	780.00	104.00
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	19.5	1004.5	325.7	200	0.4	1	2	5200.00	780.00	104.00
Beach sandals	Slipper	13	340.0		200	0.4	1	2	3466.67	520.00	69.33
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	12.4	202.1	62.9	200	0.4	1	2	3306.67	496.00	66.13
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	10.55	612.0	75.1	200	0.4	1	2	2813.33	422.00	56.27
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	10.1	547.0	59.7	200	0.4	1	2	2693.33	404.00	53.87
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	9.9	313.2	146.0	200	0.4	1	2	2640.00	396.00	52.80

Category	Sample	Concentration in product Fc $_{prod}$ (mg/kg)			Q $_{prod}$ (g)	F $_{contact}$	T $_{contact}$ (h)	n	D $_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc $_{migr}$ (%/h) = 10	Fc $_{migr}$ (%/h) = 1.5	Fc $_{migr}$ (%/h) = 0.2
Beach sandals	Slippers	8.7	546.0		200	0.4	1	2	2320.00	348.00	46.40
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	1.1	41.0	11.3	200	0.4	1	2	293.33	44.00	5.87
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	1	44.0	12.1	200	0.4	1	2	266.67	40.00	5.33
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.9	25.2	5.1	200	0.4	1	2	240.00	36.00	4.80
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.85	21.5	1.7	200	0.4	1	2	226.67	34.00	4.53
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.84	0.8	0.8	200	0.4	1	2	224.00	33.60	4.48
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.8	40.5	10.2	200	0.4	1	2	213.33	32.00	4.27
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.8	1503.0	11.3	200	0.4	1	2	213.33	32.00	4.27
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.7	22.4	6.2	200	0.4	1	2	186.67	28.00	3.73
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.67	13.3	4.8	200	0.4	1	2	178.67	26.80	3.57
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.6	82.2	20.1	200	0.4	1	2	160.00	24.00	3.20
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.6	50.4	8.7	200	0.4	1	2	160.00	24.00	3.20
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.5	74.0	7.2	200	0.4	1	2	133.33	20.00	2.67

Category	Sample	Concentration in product Fc $_{prod}$ (mg/kg)			Q $_{prod}$ (g)	F $_{contact}$	T $_{contact}$ (h)	n	D $_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc $_{migr}$ (%/h) = 10	Fc $_{migr}$ (%/h) = 1.5	Fc $_{migr}$ (%/h) = 0.2
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.5	32.8	4.2	200	0.4	1	2	133.33	20.00	2.67
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.35	210.5	0.4	200	0.4	1	2	93.33	14.00	1.87
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.3	15.5	1.9	200	0.4	1	2	80.00	12.00	1.60
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.3	31.8	1.7	200	0.4	1	2	80.00	12.00	1.60
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.2	138.5	0.7	200	0.4	1	2	53.33	8.00	1.07
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.2	135.5	3.0	200	0.4	1	2	53.33	8.00	1.07
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.077	3.1	0.1	200	0.4	1	2	20.53	3.08	0.41
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.07	12.1	3.9	200	0.4	1	2	18.67	2.80	0.37
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.063	2.0	0.1	200	0.4	1	2	16.80	2.52	0.34
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.048	4.9	0.9	200	0.4	1	2	12.80	1.92	0.26
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.043	3.4	0.0	200	0.4	1	2	11.47	1.72	0.23
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.032	0.2	0.2	200	0.4	1	2	8.53	1.28	0.17

Category	Sample	Concentration in product Fc $_{prod}$ (mg/kg)			Q $_{prod}$ (g)	F $_{contact}$	T $_{contact}$ (h)	n	D $_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc $_{migr}$ (%/h) = 10	Fc $_{migr}$ (%/h) = 1.5	Fc $_{migr}$ (%/h) = 0.2
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.011	0.1	0.0	200	0.4	1	2	2.93	0.44	0.06
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.01	1.6	0.0	200	0.4	1	2	2.67	0.40	0.05
Protective gloves		0.5	26.3	5.3	100	0.9	1	2	150.00	22.50	3.00
Protective gloves		0.4	49.0	24.8	100	0.9	1	2	120.00	18.00	2.40
Protective gloves		0.01	0.9		100	0.9	1	2	3.00	0.45	0.06
Protective gloves		0.007	1.3		100	0.9	1	2	2.10	0.32	0.04
Protective gloves		0.005	2.0		100	0.9	1	2	1.50	0.23	0.03
Protective gloves		0.003	273.8		100	0.9	1	2	0.90	0.14	0.02
Protective gloves		0.003	12.7		100	0.9	1	2	0.90	0.14	0.02
Protective gloves		0.003	2.0		100	0.9	1	2	0.90	0.14	0.02
Protective gloves		0.002	2.1		100	0.9	1	2	0.60	0.09	0.01
Protective gloves		0.002	2.4		100	0.9	1	2	0.60	0.09	0.01
Protective gloves		0.002	10.5		100	0.9	1	2	0.60	0.09	0.01
Protective gloves		0.001	1.7		100	0.9	1	2	0.30	0.05	0.01
Cover of a steering wheel		35	1430.0	736.4	300	0.2	1	2	7000.00	1050.00	140.00
Cover of a steering wheel		25	648.0	161.7	300	0.2	1	2	5000.00	750.00	100.00
Cover of a steering wheel		14	570.0	314.0	300	0.2	1	2	2800.00	420.00	56.00

Category	Sample	Concentration in product Fc $_{prod}$ (mg/kg)			Q $_{prod}$ (g)	F $_{contact}$	T $_{contact}$ (h)	n	D $_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc $_{migr}$ (%/h) = 10	Fc $_{migr}$ (%/h) = 1.5	Fc $_{migr}$ (%/h) = 0.2
Watch strap		43	1250.0	766.6	30	1	15	1	32250.00	4837.50	645.00
Watch strap		0.27	26.6	0.3	30	1	15	1	202.50	30.38	4.05

Table 69: Calculation of external dermal exposure of children by BaP containing articles

Category	Sample	Concentration in productFc $_{prod}$ (mg/kg)			Q $_{prod}$ (g)	F $_{contact}$	T $_{contact}$ (h)	n	D $_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc $_{migr}$ (%/h) = 10	Fc $_{migr}$ (%/h) = 1.5	Fc $_{migr}$ (%/h) = 0.2
Grip black	No information, assumed as bicycle grip	94	3699.0	2482.8	100	0.60	1	1	28200.0	4230.0	564.0
Grip black	No information, assumed as bicycle grip	72.0	1523.0	765.0	100	0.60	1	1	21600.0	3240.0	432.0
Grip black	No information, assumed as bicycle grip	52	>2226	1015.0	100	0.60	1	1	15600.0	2340.0	312.0
Grip black	No information, assumed as bicycle grip	34	>1262	632.0	100	0.60	1	1	10200.0	1530.0	204.0
Grip black	No information, assumed as bicycle grip	33	876.0	501.0	100	0.60	1	1	9900.0	1485.0	198.0
Grip black	No information, assumed as bicycle grip	32	1057.0	495.6	100	0.60	1	1	9600.0	1440.0	192.0
Grip black	No information, assumed as bicycle grip	27	778.0	251.0	100	0.60	1	1	8100.0	1215.0	162.0

Category	Sample	Concentration in productFc $_{prod}$ (mg/kg)			Q_{prod} (g)	$F_{contact}$	$T_{contact}$ (h)	n	$D_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc_{migr} (%/h) = 10	Fc_{migr} (%/h) = 1.5	Fc_{migr} (%/h) = 0.2
Grip black	No information, assumed as bicycle grip	21	278.0	124.0	100	0.60	1	1	6300.0	945.0	126.0
Grip black	No information, assumed as bicycle grip	9	210.0	59.0	100	0.60	1	1	2700.0	405.0	54.0
Grip black	Bicycle	1.3	25.4	8.2	100	0.60	1	1	390.0	58.5	7.8
Grip black	Bicycle	0.3	5.5	1.8	100	0.60	1	1	90.0	13.5	1.8
Grip black	Bicycle	0.2	10.0	1.0	100	0.60	1	1	60.0	9.0	1.2
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	128.65	1543.0	995.1	100	0.60	1	1	38595.0	5789.3	771.9
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	127	1774.0	783.0	100	0.60	1	1	38100.0	5715.0	762.0
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	116.95	1329.5	792.8	100	0.60	1	1	35085.0	5262.8	701.7
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	80.3	1801.5	403.3	100	0.60	1	1	24090.0	3613.5	481.8
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	43	604.9	194.7	100	0.60	1	1	12900.0	1935.0	258.0
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	41.5	1003.5	522.5	100	0.60	1	1	12450.0	1867.5	249.0

Category	Sample	Concentration in product Fc_{prod} (mg/kg)			Q_{prod} (g)	$F_{contact}$	$T_{contact}$ (h)	n	$D_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc_{migr} (%/h) = 10	Fc_{migr} (%/h) = 1.5	Fc_{migr} (%/h) = 0.2
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	14.15	682.0	108.2	100	0.60	1	1	4245.0	636.8	84.9
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	7.5	128.3	36.1	100	0.60	1	1	2250.0	337.5	45.0
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	6.18	119.3	105.7	100	0.60	1	1	1854.0	278.1	37.1
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	4.85	285.0	57.5	100	0.60	1	1	1455.0	218.3	29.1
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	3	88.4	7.2	100	0.60	1	1	900.0	135.0	18.0
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	1.2	22.0	7.3	100	0.60	1	1	360.0	54.0	7.2
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	0.2	65.0	2.7	100	0.60	1	1	60.0	9.0	1.2
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	0.2	11.0	0.7	100	0.60	1	1	60.0	9.0	1.2
Skin contact areas of sports equipment or other consumer products	No detailed information, assumed as grip	0.2	13.1	1.1	100	0.60	1	1	60.0	9.0	1.2

Category	Sample	Concentration in productFc $_{prod}$ (mg/kg)			Q_{prod} (g)	$F_{contact}$	$T_{contact}$ (h)	n	$D_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc_{migr} (%/h) = 10	Fc_{migr} (%/h) = 1.5	Fc_{migr} (%/h) = 0.2
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	111.3	1198.0	411.9	150	0.40	1	2	66780.0	10017.0	1335.6
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	34.7	812.0	160.1	150	0.40	1	2	20820.0	3123.0	416.4
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	19.5	319.6	173.8	150	0.40	1	2	11700.0	1755.0	234.0
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	19.5	1004.5	325.7	150	0.40	1	2	11700.0	1755.0	234.0
Beach sandals		13	340.0		150	0.40	1	2	7800.0	1170.0	156.0
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	12.4	202.1	62.9	150	0.40	1	2	7440.0	1116.0	148.8
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	10.55	612.0	75.1	150	0.40	1	2	6330.0	949.5	126.6
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	10.1	547.0	59.7	150	0.40	1	2	6060.0	909.0	121.2
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	9.9	313.2	146.0	150	0.40	1	2	5940.0	891.0	118.8
Beach sandals		8.7	546.0		150	0.40	1	2	5220.0	783.0	104.4
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	1.1	41.0	11.3	150	0.40	1	2	660.0	99.0	13.2
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	1	44.0	12.1	150	0.40	1	2	600.0	90.0	12.0
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.9	25.2	5.1	150	0.40	1	2	540.0	81.0	10.8
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.85	21.5	1.7	150	0.40	1	2	510.0	76.5	10.2
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.84	0.8	0.8	150	0.40	1	2	504.0	75.6	10.1
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.8	40.5	10.2	150	0.40	1	2	480.0	72.0	9.6
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.8	1503.0	11.3	150	0.40	1	2	480.0	72.0	9.6
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.7	22.4	6.2	150	0.40	1	2	420.0	63.0	8.4
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.67	13.3	4.8	150	0.40	1	2	402.0	60.3	8.0
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.6	82.2	20.1	150	0.40	1	2	360.0	54.0	7.2

Category	Sample	Concentration in product Fc_{prod} (mg/kg)			Q_{prod} (g)	$F_{contact}$	$T_{contact}$ (h)	n	$D_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc_{migr} (%/h) = 10	Fc_{migr} (%/h) = 1.5	Fc_{migr} (%/h) = 0.2
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.6	50.4	8.7	150	0.40	1	2	360.0	54.0	7.2
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.5	74.0	7.2	150	0.40	1	2	300.0	45.0	6.0
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.5	32.8	4.2	150	0.40	1	2	300.0	45.0	6.0
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.35	210.5	0.4	150	0.40	1	2	210.0	31.5	4.2
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.3	15.5	1.9	150	0.40	1	2	180.0	27.0	3.6
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.3	31.8	1.7	150	0.40	1	2	180.0	27.0	3.6
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.2	138.5	0.7	150	0.40	1	2	120.0	18.0	2.4
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.2	135.5	3.0	150	0.40	1	2	120.0	18.0	2.4
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.077	3.1	0.1	150	0.40	1	2	46.2	6.9	0.9
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.07	12.1	3.9	150	0.40	1	2	42.0	6.3	0.8
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.063	2.0	0.1	150	0.40	1	2	37.8	5.7	0.8
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.048	4.9	0.9	150	0.40	1	2	28.8	4.3	0.6
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.043	3.4	0.0	150	0.40	1	2	25.8	3.9	0.5
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.032	0.2	0.2	150	0.40	1	2	19.2	2.9	0.4
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.011	0.1	0.011	150	0.00	1	2	6.6	1.0	0.1
Footwear (shoes, boots, sandals)	Assumed as sandals used as slippers	0.01	1.6	0.010	150	0.40	1	2	6.0	0.9	0.1
Rubber boots	For children	1.4200	31.5		200	0.80	2	1	2272.0	340.8	45.4
Rubber boots	For children	1.0800	22.1		200	0.80	2	1	1728.0	259.2	34.6
Rubber boots	For children	0.8510	26.5		200	0.80	2	1	1361.6	204.2	27.2
Rubber boots	For children	0.5710	13.8		200	0.80	2	1	913.6	137.0	18.3

Category	Sample	Concentration in product Fc_{prod} (mg/kg)			Q_{prod} (g)	$F_{contact}$	$T_{contact}$ (h)	n	$D_{der\ BaP(ng/kg\ bw/d)}$		
		BaP	EPA-PAH	PAH-6					Fc_{migr} (%/h) = 10	Fc_{migr} (%/h) = 1.5	Fc_{migr} (%/h) = 0.2
Rubber boots	For children	0.3810	19.9		200	0.80	2	1	609.6	91.4	12.2
Rubber boots	For children	0.2590	10.1		200	0.80	2	1	414.4	62.2	8.3
Rubber boots	For children	0.2190	6.3		200	0.80	2	1	350.4	52.6	7.0
Rubber boots	For children	0.1630	6.4		200	0.80	2	1	260.8	39.1	5.2
Rubber boots	For children	0.1060	5.2		200	0.80	2	1	169.6	25.4	3.4
Rubber boots	For children	0.0439	5.2		200	0.80	2	1	70.2	10.5	1.4
Rubber boots	For children	0.0387	8.0		200	0.80	2	1	61.9	9.3	1.2
Watch strap		43	1250.0	766.6	20	1.00	10	1	43000.00	6450.00	860.00
Watch strap		0.27	26.6	0.3	20	1.00	10	1	270.00	40.50	5.40
Toy car	Tyre	65.9	1992.5	446.9	30	0.60	0.17	1	1008.3	151.2	20.2
Training bike	Tyre	34	764.1	209.5	100	0.10	0.17	1	283.3	42.5	5.7
Training bike	Tyre	32	968.0	227.0	100	0.10	0.17	1	266.7	40.0	5.3
Training bike	Tyre	10	257.1	53.8	100	0.10	0.17	1	83.3	12.5	1.7
Training bike	Tyre	6.9	103.0	21.4	100	0.10	0.17	1	57.5	8.6	1.2
Training bike	Tyre	5.7	164.0	46.9	100	0.10	0.17	1	47.5	7.1	1.0
Training bike	Tyre	4.9	84.4	24.0	100	0.10	0.17	1	40.8	6.1	0.8
Training bike	Tyre	4.3	120.6	30.9	100	0.10	0.17	1	35.8	5.4	0.7
Training bike	Tyre	0.76	83.0	6.9	100	0.10	0.17	1	6.3	1.0	0.1
Outdoor toys	Swing, frisbee, shovel, watering can,	0.4030	7.8		50	0.60	0.5	1	30.2	4.5	0.6
Toy car	Tyre	0.25	6.0	0.3	30	0.60	0.17	1	3.8	0.6	0.1

Category	**Sample**	**Concentration in productFc $_{prod}$ (mg/kg)**			**Q $_{prod}$ (g)**	**F $_{contact}$**	**T $_{contact}$ (h)**	**n**	**D $_{der\ BaP(ng/kg\ bw/d)}$**		
		BaP	**EPA-PAH**	**PAH-6**					**Fc $_{migr}$ (%/h) = 10**	**Fc $_{migr}$ (%/h) = 1.5**	**Fc $_{migr}$ (%/h) = 0.2**
Toy car	Tyre	0.2	6.0	1.2	30	0.60	0.17	1	3.1	0.5	0.1
Outdoor toys	Swing, frisbee, shovel, watering can,	0.0700	14.4		50	0.60	0.5	1	5.3	0.8	0.1

Appendix 6: Background to the restriction recommendation for PAH in consumer products (annex 1 of the letter sent to MS)

baua:

21.12.2009

Annex 1: PAH Restriction Background

Background to the Restriction Recommendation for PAH in Consumer Products

PAH in Consumer Products and Toys

Polycyclic aromatic hydrocarbons (PAH) are complex mixtures of organic compounds that are often found as environmental pollutants. They are formed during incomplete combustion of organic material e. g. coal and other fossil fuels, tobacco, or in barbecued food. Numerous PAH are also natural components of crude oil. Environmental exposure of humans to PAH occurs mainly via food, breathing air, or, in particular, by smoking. PAH are also well absorbed through the skin, thus the dermal route constitutes another potentially relevant pathway.

More than 100 individual PAH are known. Already in the 1980s, the US Environmental Protection Agency (EPA) established a list of 16 PAH ('EPA-PAH') that have been most frequently detected in environmental samples. The reference/lead compound in this substance group is benzo[a]pyrene (BaP), which has been classified as a human carcinogen by the IARC and is also a suspected human mutagen and reprotoxicant. Many other PAH are also under suspicion or even known to be CMR substances.

Contamination of consumer products such as tool handles or beach sandals can occur e.g. through the use of PAH-containing extender (plasticiser) oils in the production of rubber and plastics or by addition of carbon black. Materials used for construction work (e.g. flooring material) may contain PAH when produced via recycling of used tyres.

The German Federal Institute for Risk Assessment (BfR) has recently (June 2009) evaluated a series of data on PAH concentrations in various consumer products which were made available by a commercial enterprise. The evaluation revealed that no PAH could be detected in approximately 23 % of the > 1,400 samples examined, whereas 27 % contained up to 1 and 37 % between 1 and 10 mg/kg PAH (sum of 16 EPA-PAH). Thirteen percent of the products were found to contain > 10 mg/kg, with peak contents exceeding 4000 mg PAH/kg product.

A more extensive description can be found in a BfR statement on PAH in consumer products on the institute's website under the following link:

http://www.bfr.bund.de/cm/230/PAH_in_consumer_products_must_be_reduced_as_much_as_possible.pdf

The BfR has also evaluated a number of chemical-analytical studies on the PAH content of different toys. Ca. 70 % of the analysed samples contained no PAH, a further 19 % were in the range of 1-10 mg PAH/kg product, another 7 % contained 10-100 mg PAH/kg product, while the top 3 % displayed peak PAH levels of up to 1000 mg/kg product. A summary in English[1] of the BfR position on PAH in toys is available under:

http://www.bfr.bund.de/cm/230/polycyclic_aromatic_hydrocarbons_pah_in_toys.pdf

[1] The full statement is expected to be available in English soon

baua:

21.12.2009

Annex 1: PAH Restriction Background

Current Regulation at the EU Level

Entry no. 50 of Annex XVII of the REACH regulation lists eight PAH, which are all legally classified carcinogens, i. e. they have been placed in Carc. Cat. 2 acc. to Dir. 67/48/EEC (DSD) or Cat. 1B acc. to Reg. (EC) 1272/2008 (CLP regulation), respectively. A limit value of 1 mg/kg (0.0001 %) BaP or 10 mg/kg (0.001 %) for the sum of all of the above 8 PAH is set for extender/plasticiser oils when used in the manufacture of tyres. Depending on the amount of extender oil used, even considerably lower PAH contents result for the tyre as a whole. In contrast, Title III of Annex II (Chemical Properties) of the revised EU Toy Safety Directive 2009/48/EC states that

> *'[...] Substances that are classified as carcinogenic, mutagenic or toxic for reproduction (CMR) of category 1A, 1B or 2 under Regulation (EC) No 1272/2008 shall not be used in toys, in components of toys or in micro-structurally distinct parts of toys.*
>
> *By derogation [...]* ***substances or mixtures*** *classified as CMR of the categories laid down in Section 3 of Appendix B [Comment: Cat. 2 acc. to DSD or Cat. 1 A/B acc. to CLP] may be used in toys, in components of toys or micro-structurally distinct parts of toys provided that [...]*
>
> *(a)* ***these substances and mixtures are contained in individual concentrations equal to or smaller than the relevant concentrations established in the Community legal acts referred to in Section 2 of Appendix B [Comment: specific concentration limits acc. to DSD/CLP] for the classification of mixtures containing these substances [...]';***

In practice, this results in tolerated amounts of up to 100 mg/kg product for each of the individual PAH. The German Federal Institute for Risk Assessment (BfR) has calculated that such levels could lead to an exposure of children 300 times above the Threshold of Toxicological Concern (TTC) for genotoxic carcinogens (0.15 µg/person/d) and thus, under adverse circumstances, in a far higher exposure than the uptake of PAH by adults via food or even smoking. It also appears quite paradox, that a tyre for a toy car, potentially mouthed by a small infant, would be allowed to contain more than a 100 times higher PAH levels than the tyre used in a normal car.

For consumer products in general, no specific limit values for PAH have been set so far at the EU level.

Motivation of the German CA for Suggesting a Restriction of PAH in Consumer Products

As for genotoxic carcinogens no safe threshold level for exposure can be set, the German CA is of the opinion that for CM substances of Cats. 1 A or B, the ALARA (As Low As Reasonably Achievable) principle should be followed, i. e. exposure to these substances should be minimised to the greatest extent possible. Nevertheless, this has to be done on a scientific basis, i. e. aspects such as migration rates and exposure scenarios have to be accounted for, and differently so for different product groups.

This position especially holds for children's toys. Over the last decades, a general increase in the number of cases of cancer in children has been reported and requires urgent action to minimise exposure of children to CMR substances as much as possible. Not only do children constitute a population subgroup particularly sensitive to chemical hazards; also the threshold levels currently set in the EU Toy Safety Directive for CMR Cat. 1 A/B substances neither protect children's health adequately, nor do they meet the requirement on exposure minimisation for CMR substances.

baua:

21.12.2009

Annex 1: PAH Restriction Background

On the other hand, analytical investigations of the PAH content of toys and other consumer products have shown that while in these articles sometimes alarming PAH levels are found, a majority of manufacturers uses production technology resulting in products with no or only a low PAH burden.

Therefore, in the view of the German CA, the aspired restriction of PAH in consumer products is not only clearly indicated from the point of view of consumer safety, it also appears to be accomplishable at a reasonable economic cost.

Appendix 7: Questionnaire sent to MS via CIRCA

baua:

21.12.2009

Annex 2: PAH Restriction Questionaire

Member State Questionnaire for Improving the Data Basis on PAH in Consumer Products

Thank you very much for aiding us by completing this questionnaire! Please give your answers directly in this MS Word file. In some instances you might find there is too little space for some of the answers/information that you might wish to include. In this case, please feel free to adjust table column widths/add new rows as appropriate and to otherwise take all the space you need for your answer.

Your contact information (please note that at most the name and country of your organisation will be shared with any other interested party):

Name:

Name of your organisation:

Address:

Telephone number:

E-Mail address:

baua:

21.12.2009

Annex 2: PAH Restriction Questionaire

Question 1: Data protection

We are seriously committed to protecting your personal data and any confidential business information (CBI). As regards the data protection/CBI status of the information that you kindly provide us with, please indicate the way we may handle this information by ticking the appropriate box in the following table:

The information I have provided in this questionnaire…	
☐	… is not confidential and may be shared with any other interested party for any given purpose
☐	… is not confidential and may be shared with any other interested party for any given purpose, with the exception of those sections specifically marked as confidential (please indicate the way by which you marked these sections, e. g. different colour: ……...)
☐	… is confidential and may only be shared in anonymised form with other Member State CAs, the European Commission, and/or ECHA, and only so for the specific purpose of generating a restriction proposal for PAH in consumer products incl. toys
☐	… should – with respect to confidentiality/data protection - be handled as follows:

Question 2: Sources for PAH in consumer products

The German CA has so far identified three main sources of PAH in consumer products (including toys): mineral oil- or coal-based extender/plasticiser oils, carbon black, and recycled tyres. Please help us by providing in the table below any data on such substances with respect to the tonnage produced, imported or consumed and future developments related to the stated substances/mixtures. If you know of additional sources, please add this information, too.

Please note that this question only refers to substances/mixtures used in products, not the products themselves.

Substance/mixture name(s) (also trade name(s), if available)	Identifiers (such as CAS or EC nos.)	PAH-content in mixture (mg/kg or %)	Manufacture (t)	Import (t)	Amount of applica-tion (t)	Future develop-ment

Are you aware of any other relevant source for PAH in consumer products, e. g. a specific production process? Please provide this information here:

baua:

21.12.2009

Annex 2: PAH Restriction Questionaire

Question 3: Products containing PAH

Typically, PAH are contained in certain elastomer/rubber materials, but potentially also in other plastics, lacquers/varnishes, or coatings that may be encountered in or as part of consumer products, including toys. Numerous examples of such products include e. g. tool handles, bicycle handlebars, slippers/flip-flops/beach sandals, toy car tyres, and many others. They may also be contained in materials used for construction work, e. g. flooring material.

Please specify in the table below product (group)s, for which you have information on content of PAH, on relevant exposure routes for consumers, and whether the general population and, more specifically, children might be exposed to these products.

Product group	Exposure of GP [1]	Exposure of children	Route of exposure (o/d/i) [2]	Market volume (t)	Analytical content of PAH (mg/kg product) [2]
	☐	☐			
	☐	☐			
	☐	☐			

[1] General Public; [2] Oral/dermal/inhalation, multiple entries possible. In case of toys, please consider that mouthing of articles by small children might not be restricted to toys designed for their own age group; [3] If such information is available to you, please give averages, but also maximum values separately for benzo(a)pyren and other PAH

Question 4: Alternatives

Are you aware of any alternative technology or substitute substances which could lead to a reduction or replacement of PAH in consumer products?

Alternative technology / substance / mixture	Might replace/substitute the following technology / substance / mixture…	…in the following product (group)s

Question 5: Analytical data

Has any organisation in your country undertaken scientific research or are you aware of any recent research performed elsewhere on the following topics:

- chemical-analytical determination of the content of PAH in marketed substances/ mixtures/products including toys,
- the release of PAH from consumer products including toys, e. g. emission from materials used in construction work, migration from handles into sweat, migration into saliva for products in contact with oral mucosa (e.g. after mouthing by toddlers etc.), and/or
- data on external or internal (biomonitoring data) human exposure towards PAH from consumer products?

baua:

21.12.2009

Annex 2: PAH Restriction Questionaire

Studies or summary reports of studies using well-documented scientific methodology and with comprehensive documentation are of particular relevance. Please enter a short description into the following table and attach any relevant study or detailed summary reports available to you to this questionnaire.

Product type	Type of study	Reference

Question 6: Regulation

Is there currently – or has there been before the entry into force of the REACH legislation – national legislation or voluntary agreement in your country regulating the content of carcinogenic PAH in specific products (not necessarily consumer products)?

Regulation	Product type	Details on regulative measures, e. g. limit values

If your or any other organisation in your country is currently preparing a similar or alternative regulation proposal for PAH in consumer products at the EU level, we would be grateful, if you could provide us with a short description of that proposal:

Question 7: Your opinion

From your own point of view, do you feel that the current level of protection of children by the concentration limit of 0.01 % (100 mg/kg product) is sufficient?

Children: ☐ yes ☐ no ☐ don't know

Do you think that the current situation for consumers in general, i. e. no specific EU regulation on the content of PAH in consumer products, poses a potential health risk to consumers?

☐ yes ☐ no ☐ don't know

baua:

21.12.2009

Annex 2: PAH Restriction Questionaire

What is your opinion on the strategic approach to regulate PAH in consumer products (incl. toys) by using the simplified restriction procedure of Article 68 (2) of the REACH regulation to amend Annex XVII accordingly?

☐ There is no need for the proposed restriction

☐ Yes, I agree with the proposed strategy in general[1]

☐ I have no specific opinion on this issue

☐ No, I would favour a different approach, i. e.:

Question 8: Any other comments/information

If you feel that we have missed anything important, or would like to comment on any of the issues raised by this questionnaire, or present a particular position of your organisation on the regulation of PAH in consumer products, please provide this here:

[1] It is important to understand that, as we are currently reviewing the available database on toxicology and exposure, we cannot yet quantify the specific limit values which will be proposed in the end.

baua:

21.12.2009

Annex 2: PAH Restriction Questionaire

In addition to the Annex XV Dossier, we intend to prepare an Impact Assessment of the proposed restriction of PAH in consumer products and toys. Any insights regarding the socio-economic impacts of the restriction provided below are greatly appreciated and will help to improve the quality of the data available for the Impact Assessment.

Question 9: Prices and sales of sources for PAH

Please indicate prices per ton and sales per year (in Euro terms) of substances and mixtures listed under Question 2 which may occur as sources for PAH in consumer products. Also, please indicate any significant changes over the last years and expected future trends.

Substance or mixture	Price per ton in Euro terms	Sales per year in Euro terms	Changes over last years / expected future trends

Question 10: Manufacture and import of PAH contaminated products

Please indicate units sold and sales per year (in Euro terms) of PAH contaminated products listed under Question 3 either manufactured or imported from non-EU countries. Please indicate figures for your country and, if available, for the EU in parentheses. Also, please indicate any significant changes over the last years and expected future trends.

	Manufactured in your country (in the EU) per year			Imported from non-EU countries to your country (to the EU) per year		
Product (group)	Units sold	Sales in Euro terms	Changes / future trends	Units sold	Sales in Euro terms	Changes / future trends

baua:

21.12.2009

Annex 2: PAH Restriction Questionaire

Question 11: Economic impacts of alternatives on consumers

Please provide your view on the likely impacts of alternatives listed under Question 4 on consumers.

Alternative and related product (group)	Impacts on characteristics and functionality of consumer products	Impacts on availability of consumer products	Impacts on prices of consumer products

Question 12: Economic impacts of alternatives on businesses

Please provide your view on the likely impacts of alternatives listed under Question 4 on businesses in relevant supply chains.

Alternative and related product (group)	Extent of change in costs for manu-facturers, importers, distributors etc.	Types of costs afflicted (purchasing, production, switching costs etc.)	Impacts on product portfolio	Opportunities / threats related to substitution (e.g. competitiveness in-/outside of EU)

Question 13: Further socio-economic data

If available, please provide any further information or insights relevant to the socio-economic assessment of the proposed restriction of PAH in consumer products and toys.

Thank you again for having taken the time to complete this questionnaire!

Appendix 8: Analytical method proposed as the reference method for determination of PAHs in consumer products

ZEK 01.2-08

Prüfanweisung

Harmonisierte Methode zur Bestimmung von polycyclischen aromatischen Kohlenwasserstoffen (PAK) in Kunststoffproben

1 Ziel und Zweck

Bestimmung von polycyclischen aromatischen Kohlenwasserstoffen (PAK) in Kunststoffproben.

2.1 Kurzbeschreibung des Verfahrens

2.1.1 Standardverfahren

Aus dem Kunststoff wird eine repräsentative Teilprobe entnommen und mittels Schere, Seitenschneider, etc. in maximal 2 - 3 mm Stücke zerkleinert. Davon werden 500 mg eingewogen und mit 20 ml Toluol, versetzt mit internem Standard, 1 h bei 60 °C im Ultraschallbad extrahiert. Nach Abkühlung auf Raumtemperatur wird aus dem Extrakt ein Aliquot entnommen. Bei Kunststoffen bzw. Gummiprodukten, bei denen während der Untersuchung Matrixprobleme auftreten, wird zusätzlich ein säulenchromatographischer Aufreinigungsschritt durchgeführt. Die Quantifizierung erfolgt am Gaschromatographen mit massenspezifischem Detektor (GC-MSD) im SIM-Verfahren.

2.1.2 Verfahren bei Mindermengen

Sollte die **Gesamt**masse des zu untersuchenden Materials 500 mg unterschreiten, gilt Folgendes: Identische Materialien des Produkts können vereint und als eine Probe betrachtet werden. Zusätzliche Produktmuster dürfen jedoch nicht verwendet werden.

Ist für einzelne Proben **weniger als 50 mg** Material verfügbar, werden diese nicht geprüft.

Beträgt die verfügbare Masse des zerkleinerten Materials nur zwischen 50 mg und 500 mg, dann ist die Probe nach 2.1.1 zu prüfen, und die Toluolmenge ist proportional umzurechnen bzw. anzupassen. Die tatsächliche Masse der Probe ist entsprechend im Prüfbericht aufzuführen.

2.2 Geräte

- Ultraschallbad: Mindest-Leistung 200 W bei einer Badfläche von 706 cm^2 entspricht 0,28 W/cm^2 ohne Korb mit internem oder externem Thermostat

Prüfanweisung

Harmonisierte Methode zur Bestimmung von polycyclischen aromatischen Kohlenwasserstoffen (PAK) in Kunststoffproben

- Gaschromatograph mit massenselektivem Detektor

2.3 Chemikalien und Lösungen

2.3.1 Chemikalien

- Toluol
- Interne Standards
  - Standard 1: Naphthalin-d8
  - Standard 2: Pyren-d10 oder Anthracen-d10 oder Phenanthren-d10
  - Standard 3: Benzo[a]pyren-d12 oder Perylen-d12 oder Triphenylbenzol

  Es sind mindestens drei interne Standards zu verwenden, welche dem Extraktionsmittel Toluol zugesetzt werden.
- Externer Standard: 16 PAK-Substanzen nach EPA, als Mix oder einzeln
- Petrolether
- Kieselgel
- Natriumsulfat.

2.3.2 Kalibrierlösungen

Die Konzentrationen der Kalibrierlösungen sind so zu wählen, dass mit einer 3-Punktkalibrierung ein Arbeitsbereich bei den Proben von 0,1 bis 10 mg/kg abgedeckt ist. Dies entspricht einem Konzentrationsbereich bei den Kalibrierlösungen von 2,5 ng/ml bis 250 ng/ml.

3.1 Probenaufarbeitung

Aus dem Material wird eine repräsentative Teilprobe entnommen. Die Abmessungen der Teilchen, die aus der zu untersuchenden Probe durch Zerkleinerung (Scheren, Seitenschneider, Zangen etc.) erzeugt werden, sollen max. 2 - 3 mm betragen.

ZEK
01.2-08

Prüfanweisung

Harmonisierte Methode zur Bestimmung von polycyclischen aromatischen Kohlenwasserstoffen (PAK) in Kunststoffproben

3.1.1 Extraktion

500 mg Probe werden im Bördelglas mit 20 ml Toluol, welchem bereits die internen Standards zugesetzt sind, versetzt und 1 h im Ultraschallbad bei durchgehenden 60 °C extrahiert. Hierzu werden die Bördelgläser ohne Verwendung eines Korbes in das Ultraschallbad hineingestellt oder -gehängt. Anschließend werden die Bördelgläser herausgenommen und ein Aliquot des Extrakts nach Abkühlung auf Raumtemperatur und kurzem Aufschütteln entnommen und direkt oder nach Verdünnung mit Toluol gemessen.

3.1.2 Säulenchromatographischer Extraktreinigungsschritt

Bei einigen Kunststoff- bzw. Gummiprodukten, insbesondere solchen, die unter den beschriebenen Extraktionsbedingungen mit Toluol weitestgehend gelöst werden, ist eine Reinigung des Extrakts durch Kieselgel-Adsorptionschromatograhie erforderlich.

Hierzu wird eine Clean-up Säule mit Hahnschliff (ca. 220 x 15 mm) mit Glaswolle, 4 g Kieselgel und 1 cm Natriumsulfat gefüllt.

Das Silicagel ist zuvor durch Zugabe von 10 % Wasser zu desaktivieren (das Kieselgel wird im Glaskolben mit der entsprechenden Menge Wasser versetzt und anschließend 1 h am Rotations-verdampfer bei 760 Torr und Raumtemperatur homogenisiert. Das Kieselgel kann dann im verschlossenen Glaskolben bei Raumtemperatur gelagert werden).

Die Konditionierung der gepackten Säule erfolgt mit 10 ml Petrolether.

Danach wird der Toluolextraktaliquot im Rotationsverdampfer auf ca. 1 ml eingeengt und auf die Säule gegeben. Der Spitzkolben wird mit ca. 20 ml Elutionsmittel ausgespült, was ebenfalls auf die Clean-up Säule überführt wird. Die Elution erfolgt mit 50 ml Petrolether. Der aufgefangene Petrolethereluat wird mit 1 ml Toluol versetzt und am TurboVap mit Stickstoff auf ca. 1 ml einge-engt. Anschließend wird mit Toluol auf ein definiertes Volumen aufgefüllt und der Extrakt dann mittels GC-MS analysiert.

3.2 Messverfahren

Die anzuwendende Bestimmungsmethode ist die Gaschromatographie mit massenselektivem Detektor im SIM-Modus.

ZEK
01.2-08

Prüfanweisung

Harmonisierte Methode zur Bestimmung von polycyclischen aromatischen Kohlenwasserstoffen (PAK) in Kunststoffproben

Zu bestimmen sind die 16 PAK nach EPA:

- Naphthalin
- Acenaphthylen
- Acenaphthen
- Fluoren
- Phenanthren
- Anthracen
- Fluoranthen
- Pyren
- Chrysen
- Benzo[a]anthracen
- Benzo[b]fluoranthen
- Benzo[k]fluoranthen
- Benzo[a]pyren
- Indeno[1,2,3-cd]pyren
- Dibenzo[a,h]anthracen
- Benzo[g,h,i]perylen

3.2.1 Gaschromatographische Messbedingungen

Die Geräteparameter (Temperaturen, Säulen, Massenspuren) sind dem jeweiligen Labor freigestellt bzw. durch die Analyten festgelegt.

3.2.2 Auswertung

- Interne Standards: Verwendung von mindestens drei internen Standards. Die internen Standards sowie die Korrekturbereiche sind für diese drei Standards wie folgt festgelegt:

ZEK 01.2-08

Prüfanweisung

Harmonisierte Methode zur Bestimmung von polycyclischen aromatischen Kohlenwasserstoffen (PAK) in Kunststoffproben

Parameter	Interne Standards mit empfohlener Bezugnahme
Naphthalin	Naphthalin-d8
Acenaphthylen	pyren-d10 oder Anthracen-d10 oder Phenanthren-d10
Acenaphthen	Pyren-d10 oder Anthracen-d10 oder Phenanthren-d10
Fluoren	pyren-d10 oder Anthracen-d10 oder Phenanthren-d10
Phenanthren	Pyren-d10 oder Anthracen-d10 oder Phenanthren-d10
Anthracen	pyren-d10 oder Anthracen-d10 oder Phenanthren-d10
Fluoranthen	pyren-d10 oder Anthracen-d10 oder Phenanthren-d10
Pyren	pyren-d10 oder Anthracen-d10 oder Phenanthren-d10
Benzo[a]anthracen	pyren-d10 oder Anthracen-d10 oder Phenanthren-d10
Chrysen	pyren-d10 oder Anthracen-d10 oder Phenanthren-d10
Benzo[b]fluoranthen	Benzo[a]pyren-d12 oder Perylen-d12 oder Triphenylbenzol
Benzo[k]fluoranthen	Benzo[a]pyren-d12 oder Perylen-d12 oder Triphenylbenzol
Benzo[a]pyren	Benzo[a]pyren-d12 oder Perylen-d12 oder Triphenylbenzol
Indeno[1,2,3-cd]pyren	Benzo[a]pyren-d12 oder Perylen-d12 oder Triphenylbenzol
Dibenzo[a,h]anthracen	Benzo[a]pyren-d12 oder Perylen-d12 oder Triphenylbenzol
Benzo[g,h,i]perylen	Benzo[a]pyren-d12 oder Perylen-d12 oder Triphenylbenzol

- Externe Kalibrierung: Für jeden Einzel-PAK ist eine mindestens 3-Punkt-Kalibrierung mit Bezug auf die oben aufgeführte interne Standardisierung durchzuführen. Hierbei wird ein Arbeitsbereich von 0,1 bis 10 mg/kg empfohlen.
- Konzentrationen oberhalb des Kalibrierbereichs können durch Verdünnen des Extrakts bestimmt werden.

3.2.3 Bestimmungsgrenze

Die Bestimmungsgrenze liegt für Materialproben bei 0,2 mg/kg pro Parameter.

3.3 Besonderheiten

Auf Grund seiner relativen Flüchtigkeit gegenüber den anderen 15 PAK nach EPA stellt Naphthalin einen schwierig zu beurteilenden Parameter bei hautnahen Produkten dar.

Erfahrungen der Prüfinstitute zeigen, dass sowohl Abreicherungen von Naphthalin aus Materialien als auch Sekundärkontamination feststellbar sind.

ZEK
01.2-08

Prüfanweisung

Harmonisierte Methode zur Bestimmung von polycyclischen aromatischen Kohlenwasserstoffen (PAK) in Kunststoffproben

Das erhaltene Naphthalinergebnis gibt daher immer nur die momentane Situation des Prüfstücks zum Zeitpunkt der Messung wieder.

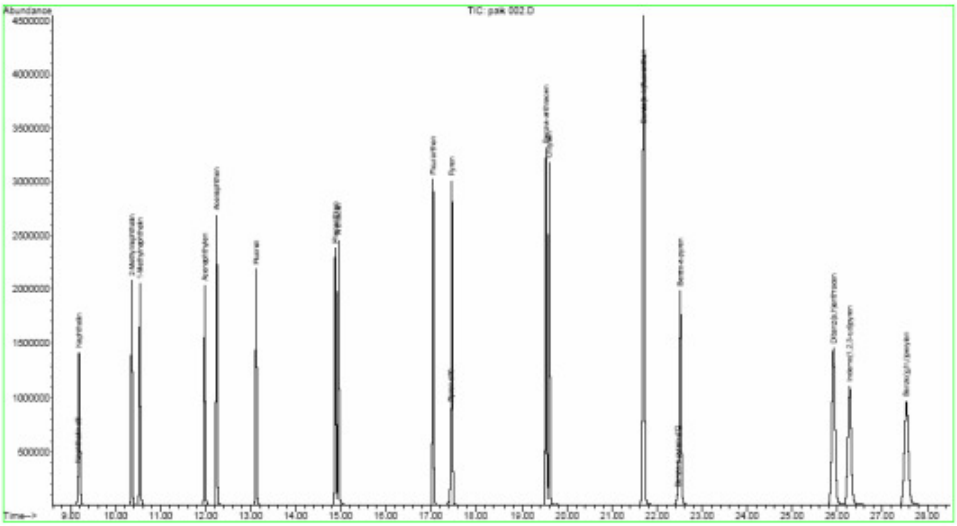
ZEK 01.2-08

Prüfanweisung

Harmonisierte Methode zur Bestimmung von polycyclischen aromatischen Kohlenwasserstoffen (PAK) in Kunststoffproben

Anhang: Gaschromatographische Messbedingungen (informativ)

Injizierte Menge: 1µl, pulsed splitlos
Säule: HT8 25m, ID 0,22mm, Filmdicke 0,25µm
Injektortemperatur: 280°C
Transferlinetemperatur: 260°C
Initialtemperatur: 50°C
Initialtime: 2 min
Heizrate: 11°C/min
Finaltemperatur: 320°C
Finaltime: 8 min



Parameter	RT ca.min	MOL-MASSE
Naphthalin	10,5	128
Acenaphthylen	14,5	152
Acenaphthen	14,8	153
Fluoren	16,0	166
Phenanthren	18,3	178
Anthracen	18,4	178
Fluoranthen	21,1	202
Pyren	21,7	202
Chrysen	24,5	228
Benzo(a)anthracen	24,6	228
Benzo(b)fluoranthen	27,0	252
Benzo(k)fluoranthen	27,0	252
Benzo(a)pyren	27,8	252
Indeno(1-,2,3-cd)pyren	31,2	276
Dibenzo(ah)anthracen	31,5	278
Benzo(ghi)perylen	32,7	276

Datum: 03.12.2008

Seite 13 von 13