Public Health England

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Contaminated land information sheet: risk assessment approaches for polycyclic aromatic hydrocarbons (PAHs)*

* This document is an update to the 2010 contaminated land information sheet for PAHs incorporating the category 4 screening level approach

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Executive summary

Public Health England (PHE) currently recommends the use of a surrogate marker approach to assess the risk posed by soil contaminated with the genotoxic polycyclic aromatic hydrocarbons (PAHs). A review of the available data on the variability of the relative PAH profile in UK soils, including 52 contaminated sites, indicates that benzo[a]pyrene (BaP) is a good surrogate marker, being ubiquitous in sites contaminated with PAHs and providing a consistent indicator of the amount of PAHs in contaminated soil.

For the surrogate marker approach to be valid, the PAH profile at potentially contaminated sites should be similar to that seen in the oral carcinogenicity study in mice by Culp et al [1]. BaP appears to be a suitable surrogate marker in the 52 sites evaluated. However, it is conceivable that, at certain sites, the PAH profile may differ from that in the Culp study. Expert judgement would then be required to determine whether it would be appropriate to still use BaP or to consider groups of surrogate marker PAHs, such as the groups of 2, 4 or 8 PAHs outlined in the European Food Safety Authority (EFSA) evaluation of PAHs in food [2]. PHE can be consulted on the interpretation of specific sites when it is uncertain as to whether BaP is sufficiently representative.

Having selected an appropriate surrogate marker (typically BaP alone), the level of the surrogate marker in soil can be compared with a screening value (Generic Assessment Criteria (GAC), Category 4 Screening Level (C4SL) or Site Specific Assessment Criteria (SSAC)), calculated using a toxicological value (Health Criteria Value (HCV) or Low Level of Toxicological Concern (LLTC)) by using the same surrogate marker approach.

Introduction

Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs are a group of organic compounds that contain two or more fused aromatic rings. The toxicology of these substances has been reviewed extensively [3-5]. Several PAHs and mixtures of PAHs have been shown to be genotoxic and to cause cancer in experimental animals [2].

PAHs are formed and released into the environment as the result of combustion/pyrolysis. Emissions can be from natural processes, such as forest fires and also as the result of human activity, such as production and processing of metals, coal, oil and gas. PAHs are also present in car exhaust fumes, cigarette smoke and wood smoke. Humans may be exposed to PAHs in the air, water and food [2]. In addition, humans may be exposed dermally, orally and by inhalation to PAH residues present in soil, particularly in former industrial “brownfield” sites, a common example being former gasworks sites that are often contaminated with coal tar residues. Consequently, it is important to assess the risk posed by PAHs present in soil at such sites so that the risk to health can be reduced to an acceptable level.

Risk assessment challenges

The commonly used chemical risk assessment paradigm entails identification and characterisation of a chemical hazard, which is then compared with an estimate of human exposure to the chemical, in order to assess the risk posed. Risk assessment becomes more complex when it is necessary to assess mixtures of similar chemicals, such as the PAHs since the hazard, mechanism of toxicity and potency may vary between chemicals. One approach would be to determine the hazard posed by each individual PAH and to estimate the dose associated with a minimal risk of adverse health effects, thereby allowing the risk associated with exposure to each individual PAH to be assessed. Unfortunately, the toxicity database and analytical methods available for these chemicals are insufficient to perform such a detailed risk assessment. Furthermore, this approach would not take account of any possible combined effects of a mixture of PAHs. Hence it is necessary to assess the risk posed by the mixture of PAHs using other methods.

Benzo[a]pyrene is the most extensively studied PAH and toxicological data provide a sufficient basis for the risk assessment of this compound. One method of risk assessment would be to assume that the toxicity of all PAHs is equivalent to that of BaP. The sum of the concentrations of the individual PAHs could then be compared to the risk posed by BaP. However, this approach would be extremely conservative since

BaP is considered to be one of the most potent PAHs. Therefore, it would be useful for the risk calculation to take account of the relative potency of the individual PAHs, as is done in the TEF approach. The advantages and disadvantages of both approaches are discussed below.

Toxic Equivalency Factor (TEF) approach

In the TEF approach, each chemical within the group is assigned a TEF (based on toxicity data usually from short-term studies) which estimates the potency of the chemical relative to a reference compound (such as BaP). The reference compound is assigned a TEF of 1. Other chemicals are assigned TEFs that are often order of magnitude estimates of potency; for example, less potent chemicals are assigned a TEF of 0.1 or 0.01. When a mixture of chemicals with the same mechanism of action is encountered, the concentration of each chemical is measured and multiplied by its TEF value, and the results are then summed to arrive at the total toxic equivalent (TEQ) of the mixture.

This TEF approach has been recommended by the World Health Organisation (WHO) for the risk assessment of the dioxins and dioxin-like chemicals [6]. For such chemicals the principal toxic effects are mediated by a common mechanism i.e. binding and activation of the aryl hydrocarbon receptor (AhR) and there is supporting evidence that dose addition occurs upon combined exposure to the various congeners [6].

Surrogate marker approach

The surrogate marker approach is an alternative approach to the risk assessment of PAH mixtures. It assumes that the cancer risk of the PAHs in a complex mixture is proportional to the concentration of a surrogate marker PAH (typically BaP) in the mixture and that this assumption holds for mixtures with a similar relative composition of the component PAHs [7, 8] . Given this proportionality the potency of a PAH mixture can be estimated from the concentration of the surrogate in that mixture and an estimate of the cancer potency of a similar mixture that has been tested in a carcinogenicity assay, such as a coal tar mixture (expressed as risk per unit amount of BaP or other surrogate) [8].

Risk assessment approaches in food, air, water and soil

Numerous organisations and expert bodies have offered advice on the risk assessment of PAHs in specific environmental matrices. Exposure to genotoxic carcinogens, such as certain PAHs, should be as low as reasonably practicable (ALARP). However, it is not possible to avoid PAH exposure entirely since they are widespread in the environment as the result of natural processes as well as human activity. Consequently,

it is necessary to assess the potential health impact posed by environmental exposure to PAHs in order to inform efforts to mitigate the risk posed by these compounds.

Food

In 1994, the UK expert advisory committees on the Carcinogenicity and Mutagenicity of Chemicals in Food, Consumer Products and the Environment (COC and COM) evaluated the limited carcinogenicity data on PAHs and agreed a ranking scheme for the carcinogenic hazard of 25 PAHs, which was based on classification into one of 5 categories. The COC concluded that this could be used to rank priorities for monitoring of PAHs but not for carcinogenic risk assessment [9, 10].

Subsequent evaluations of the available data at the European [2, 11] and international [12] level have supported a surrogate marker approach. The 2 year carcinogenicity study by Culp et al. [1] in which mice were fed two coal mixtures containing several PAHs was identified as the critical study from which a benchmark dose ($BMDL_{10}$)[1] value was derived. The EU Scientific Committee on Food (SCF) and the Joint WHO/FAO Expert Committee on Food Additives (JECFA) recommended the use of BaP as a marker, although a later assessment by the Contaminants in the Food Chain (CONTAM) panel of the EFSA considered that a set of four PAHs[2] provided a more appropriate surrogate for risk assessment. This conclusion was made on the basis of a collation of European food survey data which demonstrated that BaP was absent from 30% of foods found to contain other genotoxic PAHs [13], making it a poor surrogate for risk assessment of PAHs in food when used alone.

Air

In 1999, the UK Expert Panel on Air Quality Standards (EPAQS) considered that BaP provided a suitable basis for setting an air quality standard [14]. Data from limited animal studies were used to estimate the relative potencies of PAHs commonly found in air relative to BaP. Using this approach, the estimated contribution of BaP to the total carcinogenicity of seven PAH compounds was found to be similar in ambient air at two UK municipal sites and at an aluminium smelting plant that was the site of an occupational epidemiological study by Farant and Gariepy [15]. Therefore, the incidence of lung cancer in the studies was considered to form a suitable basis on which to recommend an air quality standard.

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[1] The $BMDL_{10}$ is the lower 95% confidence interval of the modelled benchmark dose (BMD) associated with a 10% benchmark response (BMR), such as a 10% increase in tumour incidence. The $BMDL_{10}$ can be used as a *point of departure* in the calculation of minimum risk levels (MRLs).

[2] PAH4: benzo[a]pyrene(BaP), chrysene, benz[a]anthracene and benzo[b]fluoranthene

The EU target value for PAHs in ambient air is based on BaP as a surrogate marker. The target value is compared with the total content of BaP in the PM_{10} fraction averaged over a calendar year. This value has been adopted in the Air Quality Standards Regulations for England only [16].

Expert evaluations used to set WHO [17, 18] air quality guidelines also support the use of BaP as a surrogate marker.

Water

The WHO proposed a drinking water guideline for BaP in 2003 [19], based on a mouse oral carcinogenicity study by Neal and Rigdon [20], which was supported by subsequent studies by Weyand et al. [21] and Culp et al. [1]. The WHO report discussed many of the uncertainties in the risk assessment of other genotoxic PAHs and reviewed the TEFs derived from mouse skin painting experiments. However, the WHO did not use these TEFs to set drinking water guidelines for the individual genotoxic PAHs.

Soil

In 1993, the United States Environmental Protection Agency (EPA) [22] modelled the data from mouse skin painting studies to derive “estimated orders of potential potency” as temporary guidance for the risk evaluation of PAHs.

In a more recent 2010 draft report the US EPA concluded that the database for PAHs still does not meet the criteria for the derivation of TEFs and therefore the more generalised relative potency factor (RPF) approach would be more appropriate[23] .

In 2001, the Dutch Institute of Public Health and the Environment (RIVM) developed Maximum Permissible Risk Levels (MPRs) for PAHs based on a TEF approach [24]. An excess lifetime cancer risk of 1×10^{-4} was determined for BaP, on the basis of a rat oral carcinogenicity study by Kroese et al. [25]. RIVM then used TEFs proposed by Kalberlah et al. [26] which were order of magnitude estimates of equivalent potency formulated from in vitro and in vivo studies, with a variety of routes of administration. RIVM considered that it would not be suitable to use BaP as a surrogate marker for the carcinogenic risk assessment of PAH mixtures from soil following oral exposure, due to the wide variety in composition of PAH mixtures at Dutch land contamination sites, although no data were provided to support this conclusion in its report. In view of this judgement on variability, and the different physicochemical properties of the PAHs, RIVM considered that it was not possible to set a level for total PAHs and that each individual PAH should be evaluated separately.

In 2008, the Canadian Council of Ministers of the Environment (CCME) derived preliminary human health soil quality guidelines for PAHs [27]. The CCME adopted a

similar approach to RIVM, using cancer potency equivalence factors (PEFs) relative to BaP, combined with the use of a BaP cancer slope factor estimate. This was said to be the best of several carefully evaluated methods, though there is no detailed consideration of the surrogate marker approach in the report. Like RIVM, the CCME stated that contaminated soil is likely to have a diverse compositional range of non-carcinogenic and carcinogenic PAHs of varying potency although no supporting data were provided in its report. The CCME elaborated on the substantial limitations of the equivalency factor approach but used the relative cancer potency estimates detailed in the WHO International Programme on Chemical Safety (IPCS) review of PAHs [4], which were adapted from those proposed by Kalberlah et al. [26]. Cancer risk modelling was applied to a mouse oral carcinogenicity study by Neal and Rigdon [20] to derive an incremental lifetime cancer risk of 10^{-5} to 10^{-6} for BaP. The relative potencies were applied to this risk estimate to calculate guideline values for the carcinogenic risk of 8 genotoxic PAHs.

In 2012 Defra funded a project to develop new screening levels known as category 4 screening levels (C4SLs). C4SLs provide a simple test for deciding when land is definitely not contaminated and suitable for use [28]. The project consortium adopted the surrogate marker approach and derived C4SLs for mixtures of PAHs using BaP as surrogate marker for genotoxic PAHs [29].

Evaluation of the PAH profile in soil

In order to make a robust decision regarding appropriate risk assessment methodologies, it is necessary to understand the variability of the PAH profile within contaminated soil in the UK. The key features of such an evaluation are whether BaP is ubiquitous in soil contaminated with PAHs; whether BaP alone would adequately represent the PAH profile in soil; whether a combination of surrogate markers would be more appropriate, as used by EFSA for food; and whether the PAH profile in soil is adequately represented by the mixtures of PAHs examined in the carcinogenicity study used for the hazard assessment of the surrogate marker. In order to answer these questions, we collated analytical data from 52 contaminated sites (1848 individual soil samples in total) that had been submitted to the Health Protection Agency (HPA)[3] for evaluation.

We found that BaP was present in all sites that were reported to be contaminated with PAHs. The ratio of the soil concentrations of the PAHs relative to the concentration of BaP in soil are shown in table 1.

[3] The functions of the Health Protection Agency transferred to Public Health England on 1 April 2013

Table 1. The ratios of PAH to BaP in soil from potentially contaminated sites in England and Wales.

PAH	Mean ratio to BaP	Minimum	Maximum	Lower confidence limit	Upper confidence limit	Ratio of the upper and lower confidence limits
Benz[a]anthracene	1.03	0.47	2.16	0.95	1.11	1.17
Chrysene	1.15	0.60	2.09	1.07	1.23	1.15
Benzo[b]fluoranthene	1.12	0.54	1.67	1.05	1.19	1.13
Benzo[k]fluoranthene	0.64	0.28	1.15	0.58	0.70	1.21
Dibenz[ah]anthracene	0.37	0.07	1.36	0.30	0.44	1.47
Indeno[123-cd]pyrene	0.53	0.15	1.71	0.45	0.61	1.35
Benzo[ghi]perylene	0.70	0.35	1.74	0.64	0.76	1.19

The concentrations of 7 genotoxic PAHs in soil were measured in PAH-contaminated land sites across England and Wales. The ratios of the mean concentrations of the 7 PAHs relative to BaP in soil were calculated. Data are presented as mean ratio of PAH to BaP with the upper and lower 95% confidence limits and the ratio of the upper and lower confidence limits, where n=52. The ratio of the upper and lower confidence limits was determined by dividing the upper confidence limit by the lower confidence limit (as recommended by IPCS [4]).

It is worth noting that the ratio of the upper and lower confidence intervals for each PAH is less than 2.0, indicating that the levels of the 7 PAHs, relative to the level of BaP, are stable and show little variation among the sources. This suggests that any variation among samples is probably not large enough to alter the estimated risk. The low variation also means that the level of BaP is a good predictor of the levels of the other PAHs that may be present in the soil. The relative profile was also similar to the relative PAH profile of the coal tar mixtures used in the Culp et al [1] study that is pivotal to the risk assessments by EFSA and JECFA (see figure 1).

Categorisation of the data, according to previous industrial use, showed no substantial differences in the relative PAH profiles. Moreover, the PAH profile in contaminated land was similar to that found in industrial, urban and rural UK soil samples [30] and in other surveys of soil within the UK [4, 31-34] .

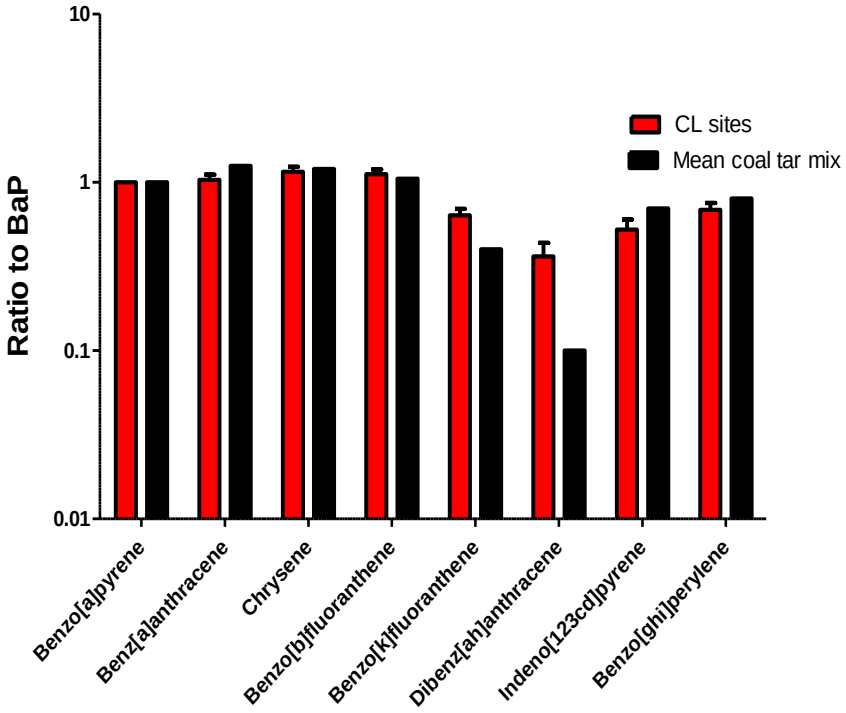


Figure 1. The mean ratio of PAH to BaP in the soil from potentially contaminated sites compared with coal tar mix.
The ratio of the mean concentrations of 7 genotoxic PAHs relative to BaP in soil from PAH-contaminated land sites was compared with the PAH ratio to BaP in two coal tar mixtures derived from a number of coal tar contaminated land sites, as presented by Culp et al. [1]. Data from the contaminated land sites are presented as mean ratio to BaP ± 95% confidence interval, where n = 52. The results for the coal tar mixture are presented as mean of the two coal tar mixtures. Mixture 1 was a composite of seven manufactured gas plant waste sites and mixture 2 was a composite of 2 of the 7 sites, plus a third site that had very high levels of BaP.

Together, these findings indicate that BaP is a suitable surrogate maker to represent the amount of the 8 genotoxic PAHs that are commonly measured in contaminated soil[4].

Discussion

Several expert opinions (including SCF, JECFA, EFSA and WHO) on the risk assessment of PAHs in food, air and water have not supported the use of TEFs due to scientific limitations and underlying uncertainty of this approach. However, reports by CCME [35] and RIVM [24] use TEFs for the risk assessment of soil in preference to the alternative surrogate marker approach due to the large variability in the PAH profile within soil in those countries.

For the TEF approach to be valid for PAHs, they would need to share a common mode of action for carcinogenicity. Although some PAHs bind to the AhR, there are other significant effects in the carcinogenesis of PAHs including DNA binding and induction of

[4] Eight Genotoxic PAHs: benzo[a]pyrene (BaP), benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[ghi]perylene, chrysene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene

mutations. PAHs may be metabolically activated[5] to different extents and may be distributed differently to the various tissues and organs of the body. Furthermore, they may cause distinct DNA adducts[6], potentially at differing sites, and with differing liability for repair; all of which may affect the likelihood of mutation in genes that are critical to cancer development. Studies on mixtures of PAHs have shown the potential for synergistic and/or antagonistic interactions at the metabolic level [11]. In a 2-year carcinogenicity study in mice by Culp et al. [1], administration of BaP alone resulted in a different profile of tumours than those produced by coal tar mixtures of several PAHs. This may indicate underlying differences in the carcinogenic mode of action.

Another limitation of the TEF approach is the lack of route specific carcinogenicity data to evaluate relative potency estimates. The limited available data for PAHs means that the TEFs for PAHs are often derived from in-vivo studies involving direct application to the skin, intraperitoneal injections or instillation into the lungs (i.e. not from the relevant route of oral exposure). Metabolism of PAHs differs according to the route of administration and this also affects carcinogenic potency. Also, enzyme induction by PAHs differs according to the route of administration and this also affects potency[36]. Therefore the relevance of the routes of exposure to PAHs used in animal studies to oral exposure of humans is unclear.

A further source of uncertainty in the TEF approach is the potential presence of PAHs that have greater carcinogenic potency than BaP. For example, dibenzo[a,l]pyrene (DBalP) is not commonly tested for in soil samples in the UK and could be 10-100 times more potent than BaP, depending upon the test system used [37].

These uncertainties are likely to contribute to the apparent under-prediction of carcinogenicity by the TEF method. An opinion on PAHs in food, by the CONTAM panel of the EFSA, provides several examples that generally indicate that the TEF approach would under-predict the carcinogenic potency of a PAH mixture [2].

The surrogate marker approach relies on a number of assumptions. Firstly, it is assumed that the surrogate marker is present in all samples. Secondly, the profile (concentration of the individual PAHs relative to the surrogate marker) is assumed to be similar in all samples. Thirdly, it assumes that the PAH profiles of the samples are similar to those used in the carcinogenicity (and other) studies used in the risk assessment to derive the critical toxicological parameter.

High potency PAHs, such as DBalP, also represent an uncertainty in the surrogate marker approach, as in the TEF approach. As stated above, DBalP is rarely measured

[5] Metabolic activation occurs when metabolism of a compound leads to an increase in its activity, whether beneficial (e.g. activation of a pro-drug) or deleterious (e.g. activation to a toxic metabolite which cause the toxic effect).
[6] DNA adducts arise when a chemical group covalently binds to DNA, resulting in mutations, which, if not repaired, can lead to cancer.

in soil samples during routine sampling. Therefore, it is not known whether the surrogate marker is representative of the unknown levels of DBalP in soil samples and unknown levels in the test mixtures assessed in the relevant toxicity studies. For potent PAHs, such as DBalP, small variations in relative concentration might affect the overall carcinogenicity of the mixture.

An advantage of the surrogate maker approach is that it uses dose response data from a coal tar mixture (PAH mixture) tested in an oral in vivo carcinogenicity study [1], rather than assessing PAHs individually. Therefore, it accounts for potential interactions between the PAHs in the mixture (e.g. synergistic effects). The study is an oral study which is the relevant route of exposure, this is important because the metabolism and carcinogenic potency of PAHs is influenced by the route of exposure. In addition, analysis of UK soil data indicates that the profile of PAHs in UK soils is sufficiently similar to the PAH profile of the coal tar mixture used in the in vivo study.

Conclusion

With the currently available toxicity data the use of a surrogate marker seems to be more appropriate and pragmatic than TEFs, although each approach is subject to uncertainty. Analysis of data from sites around the UK that are contaminated with PAHs indicates that BaP is a suitable surrogate marker for the assessment of PAHs in soil.

Risk assessment using the surrogate marker approach

Oral exposure

Currently, soil samples are generally analysed for eight genotoxic PAHs. We recommend that the ratio of PAHs, relative to BaP, is assessed to ensure the profile is similar to that seen for the test material used in the study by Culp et al. [1]. The International Programme on Chemical Safety (IPCS) considered that "the PAH profile of a tested mixture may deviate from the average profile by about an order of magnitude (up or down)", and that "such small differences are below the resolution of the risk assessment process" [4]. Therefore, the profile of PAHs in a soil sample is considered sufficiently similar to that of the test material if the ratios of each PAH, relative to BaP, are within an order of magnitude above and below that of the test material (see table 2). In such cases BaP would be considered a suitable surrogate marker. The order of magnitude limits are plotted in Figure 2 along with the profiles from each of the 52 sites and data from soil surveys within the UK.

Table 2. Profile of the genotoxic PAHs relative to BaP in the study by Culp *et al.,* along with order of magnitude upper and lower limits.

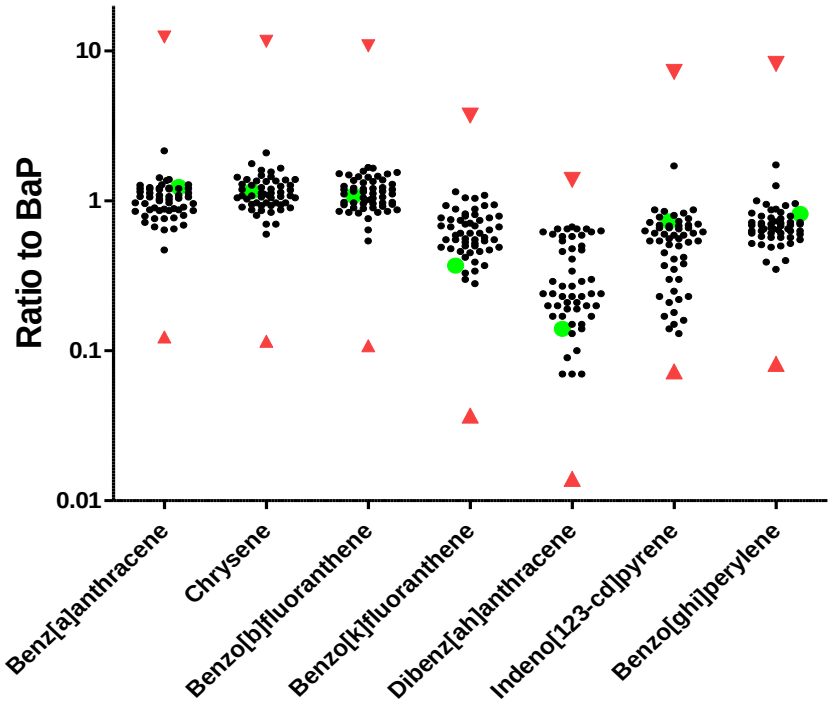
PAH	Mean ratio to BaP	Upper limit	Lower limit
Benz[a]anthracene	1.24	12.43	0.12
Chrysene	1.16	11.61	0.12
Benzo[b]fluoranthene	1.08	10.85	0.11
Benzo[k]fluoranthene	0.37	3.72	0.04
Dibenz[ah]anthracene	0.14	1.38	0.01
Indeno[123-cd]pyrene	0.73	7.27	0.07
Benzo[ghi]perylene	0.82	8.22	0.08

The ratio of the mean concentrations of 7 genotoxic PAHs, relative to BaP, in the test material used in the study by Culp *et al*. were used to determine the upper and lower limits. These limits represent an order of magnitude above and below the mean ratio to BaP of the test material used in the study [1].

In cases where BaP is considered a suitable surrogate marker, levels of BaP measured at the site can be compared with a screening value (GAC, C4SL, SSAC), derived using a toxicological value (HCV/LLTC) for BaP derived from data from the Culp et al study [1].

See below for more information on the recommended HCVs and the LLTCs derived by the C4SLs project consortium.

Figure 2. The ratio of PAH to BaP in the soil from individual potentially contaminated sites



The ratio of the mean concentrations of 7 genotoxic PAHs, relative to BaP, in individual sites are plotted, along with the upper (▼) and lower (▲) limits, which represent an order of magnitude above and below the test material (●) used in the study by Culp *et al.*[1].

Health criteria value

When deriving a value that represents minimal risk it would seem prudent to base the Index Dose (ID) on the $BMDL_{10}$ values proposed by EFSA and JECFA derived from the Culp et al study [1] (0.07 and 0.1 milligrams per kilogram bodyweight per day (mg/kg bw/day), respectively [12, 13].

In order to calculate the ID, the $BMDL_{10}$ is divided by a suitable margin of 10,000[7]. Therefore the ID dose would be 0.007 – 0.01 micrograms per kilogram bodyweight per day (µg/kg bw/day).

In the case of the small number of sites that fall outside of the order of magnitude limits, it might be appropriate to consider using a HCV for groups of surrogate markers, such

[7] Defra recommends at the HCVs for non-threshold substances should be developed on the basis that the "minimal risk level" for non-threshold genotoxic carcinogens in soil is equivalent to a parameter derived from an appropriate animal carcinogenicity study (the BMDL10) divided by a suitable margin of 10,000 [38].

as the groups of 2, 4 or 8 PAHs (see table 3) given in the EFSA evaluation of PAHs in food [2]. The $BMDL_{10}$ values for these groups are based on the summed concentrations of the PAHs in the coal tar mixture tested in the Culp et al. [1] study, and reflect the carcinogenicity of this mixture.

Table 3. EFSA $BMDL_{10}$ values for PAH surrogate marker(s) [2].

	$BMDL_{10}$ (mg/kg bw/day)
PAH1 (BaP)	0.07
PAH2	0.17
PAH4	0.34
PAH8	0.49

PAH2 - BaP and Chrysene; PAH4 – PAH2 + benz[a]anthracene, benzo[b]fluoranthene; PAH8 – PAH4 + benzo[k]fluoranthene, benzo[ghi]perylene, dibenz[a,h]anthracene and indeno[1,2,3-cd]pyrene

In view of the uncertainties in this approach, it would be prudent to select the surrogate marker group that provides the more conservative risk assessment. PHE can be consulted on the interpretation of specific sites where there is uncertainty as to whether BaP is sufficiently representative.

Low level of toxicological concern

The Culp et al study [1] was used to form the basis of the oral LLTC for BaP as a surrogate marker of PAHs. Dose response modelling was performed using the data on the total number of tumour bearing animals administered coal tar mixture 1 in the Culp study. The consortium proposed the use of the BMD_{10}[8] (0.21 mg/kg bw/day) as the point of departure for the derivation of the LLTC. In order to calculate the LLTC the BMD_{10} was divided by a suitable margin of 5000[9] to give an oral LLTC of 0.042 µg/kg bw/day for BaP as a surrogate marker for PAHs [39] .

A small number of sites may fall outside the order of magnitude limits, therefore it might be appropriate to consider using an oral LLTC for groups of surrogate markers, such as the groups of 2, 4 or 8 PAHs used in the EFSA evaluation of PAHs in food [2]. PHE can

[8] The benchmark dose (BMD) is a dose associated with a predetermined change in response rate for an adverse effect. In this case it is 10% increase in tumour incidence.

[9] Defra recommends that a generic margin of 5000 be used for the purposed of deriving Low Levels of Toxicological Concern for non-threshold chemicals when a BMD_{10} from animal data is used as the Point of Departure [31].

be consulted on the interpretation of specific sites where there is uncertainty as to whether BaP is sufficiently representative.

Inhalation exposure

Health criteria value

The recommended inhalation HCV in the 2010 HPA contaminated land information sheet for PAHs was based on the UK Air Quality Objective of 0.25 ng/m^3 proposed by the EPAQS[14, 40].

The Contaminated Land Exposure Assessment (CLEA) framework document on toxicological assessment of contaminants in soil [41] explains that it is disproportionate to recommend a stricter limit for contaminated land when there are less stringent guidelines produced by other regulatory regimes under UK jurisdiction. Therefore, it would be appropriate to use the EU target value of 1 ng/m^3 for BaP, as set out in the Air Quality Standards Regulations 2010, as the basis for the inhalation HCV. Based on a 70 kg adult inhaling 20 m^3 of air per day this equates to an inhalation ID of 0.3 nanograms per kilogram bodyweight per day (ng/kg bw/day) [16].

This inhalation ID for BaP is associated with an excess lifetime cancer risk substantially higher than the minimal risk level (1:100,000) normally considered appropriate, as outlined by Defra [38]. Therefore, caution should be exercised when the ID, and hence the GAC, for BaP are exceeded as the likelihood that significant possibility of significant harm (SPOSH) may occur is greater than if the ID was based on health considerations. Moreover, SPOSH may occur at smaller exceedances of the ID and GAC than usual.

Low level of toxicological concern

The C4SL project consortium also selected the annual ambient air concentration of 1 ng m^{-3}, set as an EU target, as the basis of the inhalation LLTC for BaP. Based on a 70 kg adult inhaling 20 m^3 of air per day this equates to an inhalation LLTC of 0.3 ng/kg bw/day [39].

The inhalation LLTC for BaP is associated with an excess lifetime cancer risk substantially higher than the current low risk level (1:50,000) outlined by Defra [28]. Therefore, caution should be exercised when the LLTC, and hence the Category 4 Screening Level, for BaP are exceeded as the likelihood that SPOSH may occur is greater than if the LLTC was based on health considerations. Moreover, SPOSH may occur at smaller exceedances of the LLTC and Category 4 Screening Level than usual.

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