



HPA Contaminated Land Information Sheet

Risk Assessment Approaches for Polycyclic Aromatic Hydrocarbons (PAHs)

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Summary

The Health Protection Agency (HPA) currently recommends the use of a surrogate marker approach to assess the risk posed by soil contaminated with the genotoxic polycyclic aromatic hydrocarbons (PAHs). Although the surrogate marker approach is subject to substantial uncertainty, it is preferable to the use of toxic equivalency factors (TEFs) due to the scientific limitations of the latter approach. 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]. The HPA 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 Generic Assessment Criteria (GAC) or Site Specific Assessment Criteria (SSAC), calculated using a health criteria value (HCV) for the same surrogate marker.

Introduction

Polycyclic Aromatic Hydrocarbons (PAHs)

The 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

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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 of the 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 is 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 ^[3].

For the TEF approach to be valid for PAHs, they would need to act through the same mechanism of action at a molecular level. Although some PAHs can bind to the AhR and potentially cause cancer as a result of this, many PAHs are considered to cause cancer by a genotoxic mode of action ^[4]. These PAHs may be metabolically activated^a to different extents and may be distributed differently to the various tissues and organs of the body. Furthermore, they may cause distinct DNA adducts^b, potentially at differing sites, and with differing liability for repair; all of which may affect the likelihood of mutation in genes that are

^a 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).

^b DNA adducts arise when a chemical group covalently binds to DNA, resulting in mutations, which, if not repaired, can lead to cancer.

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critical to cancer development. Overall, the PAHs cannot be considered to have a common mechanism of action. Indeed, studies on mixtures of individual PAHs have shown the potential for synergistic and/or antagonistic interactions at the metabolic level ^[4]. 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.

As well as the differences in toxic mechanism of action of the individual PAHs, other uncertainties complicate the use of TEFs for the assessment of PAH contaminated soil. Many of the proposed TEF schemes, such as that proposed by Kalberlah *et al.* ^[5], are based on a variety of study types and model systems including *in-vitro* and *in-vivo* assays. The combination of *in-vivo* data from several routes of exposure adds further uncertainty to the use of TEFs to assess contaminated land. Mouse skin painting studies have provided much of the evidence base and these studies may not be directly applicable to other routes of exposure. Also, the TEF approach requires individual compounds to be tested against a reference compound; little can be drawn from those studies that do not provide a direct comparison to a reference compound.

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 (DBaP) is not commonly tested for in soil samples and could be 10-100 times more potent than BaP, depending upon the test system used ^[6]. Without information on the level present in soil samples and suitable data for the derivation of a TEF, such high potency PAHs cannot be evaluated using the TEF approach.

These uncertainties are likely to contribute to the apparent under-prediction of carcinogenicity by the TEF method. A recent 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].

Overall, based on these uncertainties, the TEF approach would seem to be unsuitable for the risk assessment of PAH mixtures in soil. However, in spite of concerns regarding the appropriateness of the TEF approach, several organisations^c have endorsed it as the best available method for assessing mixtures of PAHs in soil ^[10-12]. This endorsement is, in part, due to uncertainties in the alternative *surrogate marker* approach to PAHs.

Surrogate marker approach

The surrogate marker approach estimates the toxicity of a mixture of PAHs in an environmental matrix by using data from toxicity studies in which a PAH mixture of known composition was tested. This is in contrast to the TEF method which uses concentration and toxicity data on individual PAHs. Exposure to the surrogate marker (usually BaP) is assumed to represent exposure to all the PAHs in the environmental matrix. Thus, the level of toxicity ascribed to the surrogate represents the toxicity of the PAH mixture.

The marker approach relies on a number of assumptions. Firstly, it is assumed that the surrogate marker is present in all samples. Secondly, the profile of the various PAHs relative

^c The TEF approach is broadly analogous to the COT ranking scheme ^[14], EPAQS relative potencies ^[17], EPA estimated orders of potential potency ^[12], CCME cancer potency equivalence factors ^[10] and RIVM potency equivalence factors ^[11].

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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 and lastly, it assumes that the carcinogenic potency of the total PAH mixture increases linearly with dose.

High potency PAHs, such as DBaIP, represent an uncertainty in the surrogate marker approach, as in the TEF approach. As stated above, DBaIP is rarely measured in soil samples during routine sampling. Therefore, it is not known whether the surrogate marker is representative of the unknown levels of DBaIP in soil samples and unknown levels in the test mixtures assessed in the relevant toxicity studies. For potent PAHs, such as DBaIP, small variations in relative concentration might affect the overall carcinogenicity of the mixture.

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 exposure entirely since PAHs 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 [7, 8].

Subsequent evaluations at the European [2, 4] and international [9] level have supported a surrogate marker approach based on benchmark dose (BMDL₁₀) values^d derived from the 2 year carcinogenicity study by Culp *et al.* [1] in which mice were fed two coal tar mixtures containing several PAHs. 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 CONTAM panel of the EFSA considered that a set of four PAHs^e 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 [10], making it a poor surrogate for risk assessment of PAHs in food when used alone.

^d The BMDL₁₀ 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 BMDL₁₀ can be used as a *point of departure* in the calculation of minimum risk levels (MRLs)

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

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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 ^[11]. 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 Garipey ^[12]. Therefore, the incidence of lung cancer at the study was considered to form a suitable basis on which to recommend an air quality standard. Expert evaluations used to set WHO ^[13] and EU ^[14] 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 ^[15], based on a mouse oral carcinogenicity study by Neal and Rigdon ^[16], which was supported by subsequent studies by Weyand *et al.* ^[17] 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) ^[18] 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 2001, the Dutch Institute of Public Health and the Environment (RIVM) developed Maximum Permissible Risk Levels (MPRs) for PAHs based on a TEF approach ^[19]. 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.* ^[20]. RIVM then used TEFs proposed by Kalberlah *et al.* ^[5] 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 ^[21]. 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 ^[22], which were adapted from those proposed by Kalberlah *et al.* ^[5]. Cancer risk modelling was applied to a mouse oral carcinogenicity study by Neal and Rigdon ^[16] to derive an incremental lifetime cancer risk of 10^{-5} to 10^{-6} for BaP. The relative potencies were

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applied to this risk estimate to calculate guideline values for the carcinogenic risk of 8 genotoxic PAHs.

Evaluation of the PAH profile in Soil

As outlined in the previous section, several recent expert opinions on the risk assessment of PAHs in food, air and water have not supported the use of TEFs due to the scientific limitations and underlying uncertainty of this approach. However, two recent reports by CCME [21] and RIVM [19] do use TEFs for the risk assessment of soil, on the basis that the variability in the PAH profile within soil precludes the use of other approaches.

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 HPA for evaluation.

We found that BaP was present in all sites that were reported to be contaminated with PAHs. Although the absolute concentrations of the individual PAHs were highly variable, the variability was substantially reduced when they were expressed relative to the measured concentration of BaP (see table 1).

Table 1. The mean ratio 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	Confidence range
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 confidence range, where n=52. The confidence range was determined by dividing the upper confidence limit by the lower confidence limit (as recommended by IPCS [22])

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It is worth noting that the confidence range 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 ^[23] and in other surveys of soil within the UK ^[4, 26-29].

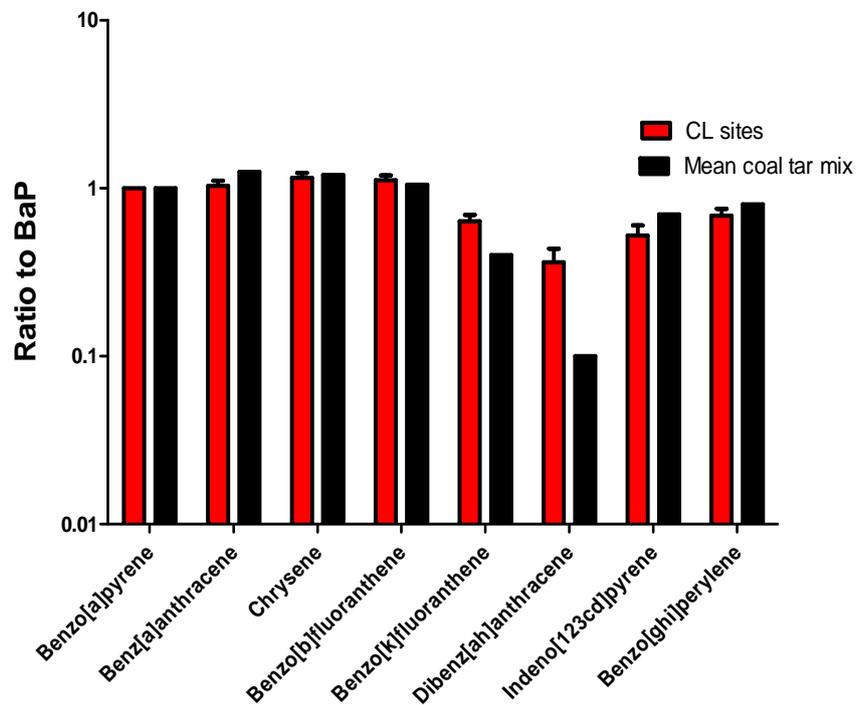


Figure 1. The mean ratio of PAH to BaP in the soil from potentially contaminated sites compared with coal tar mix.

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 \pm 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.

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Together, these findings indicate that BaP is a suitable surrogate marker to represent the amount of the 8 genotoxic PAHs that are commonly measured in contaminated soil^f. Our detailed findings will be published as soon as possible.

Risk Assessment

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” ^[22]. 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].

^f 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

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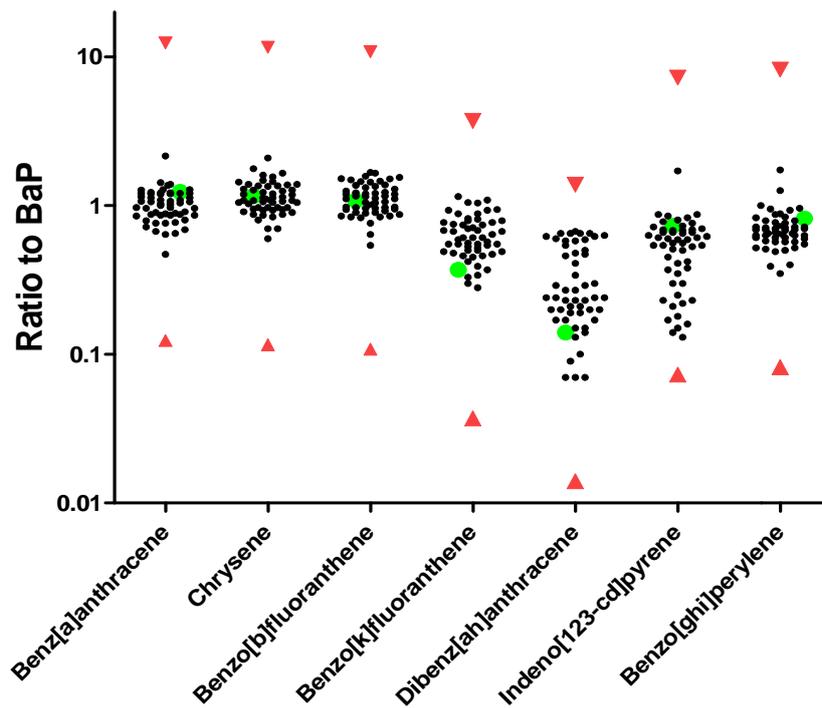


Figure 2. The ratio of PAH to BaP in the soil from individual potentially contaminated sites

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

In cases where BaP is considered a suitable surrogate marker, levels of BaP measured at the site can be compared with a GAC or SSAC, derived using a HCV for BaP derived from data from the Culp *et al* study^[1] (see table 3).

The Environment Agency are reviewing the available toxicology data on PAHs and will endorse an appropriate point of departure from which to derive the oral HCV. In the interim, it would seem prudent to base the index dose (ID) on the BMDL₁₀ 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^[9, 10]. Both EFSA and JECFA have recognised BMD expertise and are likely to represent the state of the art in this methodology.

In order to calculate the ID the BMDL₁₀ is divided by an uncertainty factor of 10,000. Therefore the ID dose would be 0.007 – 0.01 micrograms per kilogram bodyweight per day ($\mu\text{g}/\text{kg}$ 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 an HCV for groups of surrogate markers, such as the groups of 2, 4 or 8 PAHs (see table 3) given in the EFSA evaluation of PAHs in food^[2]. The BMDL₁₀ 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.

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Table 3. EFSA BMDL₁₀ 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 provided the more conservative risk assessment. The HPA can be consulted on the interpretation of specific sites where there is uncertainty as to whether BaP is sufficiently representative.

Inhalational exposure

The Environment Agency are reviewing the available toxicology data on PAHs and will endorse an appropriate point of departure from which to derive an inhalation HCV. The Contaminated Land Exposure Assessment (CLEA) framework document on toxicological assessment of contaminants in soil ^[24] explains that it is disproportionate to recommend a stricter limit for contaminated land when there are less stringent guidelines produced by regulatory regimes under UK jurisdiction. Therefore, in the interim it would be appropriate to continue to use the UK Air Quality Standard of 0.25 ng m⁻³ proposed by the EPAQS as a basis for deriving the inhalation HCV. Based on a 70 kg adult inhaling 20 m³ of air per day this equates to a inhalation ID of 0.07 nanograms per kilogram bodyweight per day (ng/kg bw/day) ^[11, 25].

Conclusion

With the currently available toxicity data, no approach to risk assessment of PAHs is ideal. The use of a surrogate marker seems to be more appropriate than TEFs, although each approach is subject to substantial uncertainty. Analysis of data from sites from around the UK that are contaminated with PAHs indicates that BaP is a good surrogate marker.

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