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QUANTIFYING HUMAN EXPOSURES TO PAHS IN SOIL: STATE OF THE SCIENCE

Decision-making at contaminated upland sites is commonly driven by potential human health risks associated with direct contact with contaminants in soils. For polycyclic aromatic hydrocarbons (PAHs), direct contact risks are dominated by the ingestion route, followed by the dermal route and, several orders of magnitude lower, the inhalation route. Applying standard EPA default exposure values, ingestion accounts for 73 percent and dermal exposure for 23 percent of the potential cancer risks from direct contact with PAHs in soil. EPA guidance makes no provision for reduced oral bioavailability of PAHs in a soil matrix, despite the fact that the literature suggests that exposures are reduced by up to 5-fold when oral bioavailability is considered.[1] If a relative bioavailability adjustment (RBA) of 0.2 (i.e., 5-fold reduction) were applied in a risk assessment for oral exposures, estimated dermal exposure would exceed oral exposure and would drive the risk estimate. Thus, it is important to understand absorption of PAHs from both the ingestion and dermal routes. This article summarizes some of the factors influencing absorption of PAHs from soil; the current state of the science for quantifying the oral bioavailability and dermal absorption of PAHs; and the ongoing research that will allow for cost-effective, site-specific evaluation of direct contact exposures to PAHs in soil.

The Role of Geosorbents in Human Exposure to PAHs in Soil

Carcinogenic PAHs enter soils as discrete source materials, whether they are in the form of solid phases (e.g., soot, chars, cenosphere, coal, or coke) or nonaqueous phase liquids (NAPLs) (e.g., petroleum products, fuel residues, creosote, or coal tar). Soot and chars, which are the remnants of incomplete combustion, are commonly termed "black carbon." These solid phases and NAPLs, in addition to natural materials in soils such as humate and kerogen, offer sorption domains for organic compounds and present a range of affinities for PAHs as characterized by their organic carbon–water partition coefficient (Koc) values (Figure 1). Studies indicate that there are differences of up to two orders of magnitude between the PAH Koc values of natural organic matter in soil and of black carbon forms (Cornelissen et al. 2005). Over time, PAHs will partition to those compounds with the highest Koc values, resulting in PAHs that are both entrained in solid source materials (present in the particle matrix when the PAHs were formed) and sorbed to the particle exteriors (Figure 1). Although there is a lack of studies on the effects of black carbon and other geosorbents on the oral bioavailability and dermal absorption of PAHs in humans, studies using ecological receptors (e.g., earthworms, benthic invertebrates) have

[1] Currently, EPA recommends that risk assessments assume the dermal absorption of PAHs from soils to be 13 percent of the total applied dose.

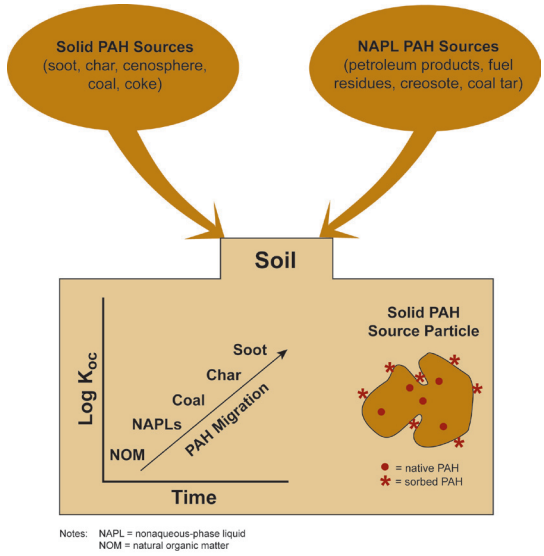

Figure 1. Origin and Fate of PAHs in Soil

demonstrated decreased PAH uptake from soils and sediments containing elevated levels of black carbon, coal, coke, and kerogen. Studies are needed to assess the extent to which black carbon and other geosorbents control the magnitude of human exposures (both oral and dermal) to PAHs in soils.

Oral Bioavailability of PAHs

The oral RBA is an adjustment used to account for the difference in oral absorption of a contaminant between the dosing vehicle used in the critical study on which the toxicity value is based (benzo[a]pyrene [BaP] in diet for PAHs) and the environmental exposure matrix (e.g., soil or sediment). Studies examining the relative bioavailability of PAHs have used a variety of animal models (mouse, rat, minipig) and biological endpoints to estimate absorption (six such studies have been conducted to date and are summarized in Table 1). These methods include measuring excretion of parent PAHs in feces, urinary excretion of one or more PAH metabolites, the extent to which PAHs form DNA adducts in lung or liver tissue and determining the area under the dose extinction curve in blood after a radiolabeled dose of BaP is administered. In general, research efforts have been isolated, focusing on a particular site and evaluating only a few samples in any one study. At this time, there is no generally accepted animal model for assessing the oral bioavailability of PAHs and none of the endpoints mentioned above has been conclusively shown to be a good metric for measuring PAH absorption.

Nevertheless, estimates of the relative bioavailability for carcinogenic PAHs in the six studies to date have ranged from 17 to 76 percent (Table 1), with mean and 90th percentile RBA values of 0.25 and 0.53 (values for each soil in each study treated as individual data points). In aggregate, these results indicate that PAHs from a soil matrix are less well absorbed than those from diet.

Table 1. Summary of Published Studies on the Oral Bioavailability of PAHs from Soil[a]

Study	Site Type/ PAH Source	Test Species	Number of Soils	Particle Size (µm)	Soil Concentration	Endpoint Measured	Relative Bioavailability (%)
Goon et al. 1991 and Magee et al. 1996	Soil sources unknown	Male Sprague-Dawley rat	sandy loam (0.04% TOC) clayey soil (1.4% TOC)	<100	100 mg/kg ^{14}C-labeled BaP (soils weathered for 1, 7, 30 days, 6 or 12 months)	^{14}C AUC in blood	37–49 22–38 (RBA decreasing with time)
Weyand et al. 1996	Manufactured gas plant soils	Female B6C3F1 mouse	2 1	<150	1 and 35 mg/kg pyrene 377 mg/kg total PAHs	Urinary excretion of pyrene metabolite DNA adducts in lung tissue	11 and 36 17
Koganti et al. 1998	Manufactured gas plant soils	Female B6C3F1 mouse	3 3	<150	0.2–627 mg/kg pyrene 8–3,120 mg/kg total PAHs	Urinary excretion of pyrene metabolites DNA adducts in lung tissue	8–100 8–76
Magee et al. 1999	Superfund site	Female B6C3F1 mouse	3	<250	66–388 mg/kg total PAHs	Urinary excretion of BaP metabolite DNA adducts in lung tissue	1–29 7–36
Bordelon et al. 2000	Manufactured gas plant soil	Fischer 344 male rat	1	Unknown	3,500 mg/kg total PAHs	DNA adducts in liver and lung tissue	35 and 40
Gron et al. 2007	Mine waste or household/ construction waste	Minipig	4	Unknown	6–270 mg/kg BaP 0.77–43 mg/kg dibenz[a,h]anthracene	Parent PAH excreted in feces	36–55 27–30

[a] Three publications that address the oral bioavailability of PAHs from soil (Pu et al. 2004; Stroo et al. 2005; James et al. 2011) were reviewed but are not summarized because of inadequate documentation of study methods or results.

Notes: AUC = area under the curve; TOC = total organic carbon

In Vitro Methods for Estimating Oral Bioavailability

One goal of the PAH bioavailability research community has been to develop a simple, reproducible *in vitro* extraction test that correlates with *in vivo* measures of relative bioavailability. Such a test would provide an efficient and inexpensive method to predict the relative bioavailability of PAHs from soil on a site-specific basis. Since 1996, when the first publication regarding *in vitro* testing for PAHs appeared (Hack and Selenka 1996), there have been 31 additional publications addressing some aspect of *in vitro* test development and/or application for PAHs (Figure 2). The rate of publication has increased significantly over the last eight years, indicating a growing interest in the development of *in vitro* tests for PAHs. Four of these publications have presented some form of *in vitro* to *in vivo* correlation, all of which are based on limited *in vivo* data sets. However, there are significant limitations to all four of the different *in vivo* models used for these correlations. Until a generally accepted *in vivo* model becomes available, it will not be possible to validate one of the numerous *in vitro* models currently being proposed.

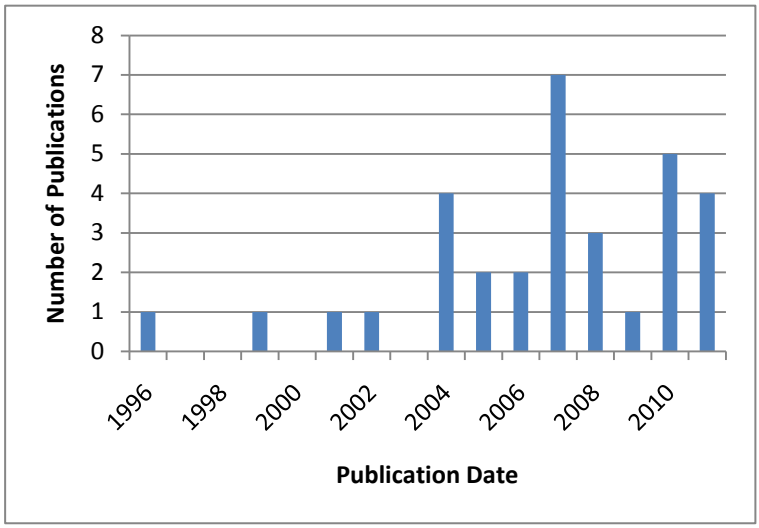

Figure 2. Annual Rate of Publication Regarding *In Vitro* Testing for PAHs

Dermal Absorption of PAHs

The current EPA default value of 13 percent for dermal absorption of BaP (and by extension other PAHs) in soil was derived from a study in which ^{14}C-labeled BaP that had been combined with fine sand was placed on the abdominal skin of four rhesus monkeys (Wester et al. 1990). This value is likely an overestimate of actual dermal absorption in humans due to a number of limitations in the study design. First, the ^{14}C-labeled BaP was mixed with fine sand, not soil. The substrate was prepared by sieving an actual soil to a size range of 48 to 80 mesh (178 to 297 μm, or primarily fine sand) so that no clay or silt was present. Studies have shown that PAHs are primarily bound to clay and silt size particles in soil. Second, sieving out the fine fraction would have removed the black carbon and natural organic matter that would have adsorbed PAHs and might have attenuated PAH absorption. Finally, there was no weathering time for the ^{14}C-labeled BaP on the sand, which might well have resulted in decreased dermal absorption. All of these study

limitations likely result in the 13 percent value being an overestimation of actual dermal absorption of BaP from soil.

Conclusions

In summary, there are currently significant limitations in our ability to quantify PAH exposures to humans from soil via both the oral and dermal exposure routes. These limitations stem from 1) a lack of information on the PAH-soil chemistry that is controlling oral bioavailability and dermal absorption, 2) the lack of a robust *in vivo* model for oral bioavailability, 3) the lack of a "validated" *in vitro* method for inexpensive site-specific estimation of oral bioavailability, and 4) the need for an updated dermal absorption study to derive a more representative estimate of percutaneous absorption of PAHs.

A number of research groups, including Integral Consulting, are currently conducting studies to address these data gaps. Please contact Mike Ruby at 303.404.2944 x14; mruby@integral-corp.com for more information on this topic.

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