Establishing Generic Remediation Goals for the Polycyclic Aromatic Hydrocarbons: Critical Issues

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Polycyclic aromatic hydrocarbons (PAHs) from both natural and anthropogenic sources are ubiquitous in the environment (1,2). Several of the PAHs are considered to be carcinogenic, and for this reason, PAHs are often chemicals of concern at hazardous waste sites. Examination of remediation goals established by different state regulatory agencies for carcinogenic PAHs in soils indicates that values are fairly consistent and uniformly low, generally below 1 mg/kg (Table 1). These remediation goals are either risk based or, if the state has determined that method detection limits (MDLs) are above the risk-based values, are based on the MDLs. However, as a practical matter, reviews of background PAH concentrations show that in many cases, background concentrations are above state goals (1,2). If background soil concentrations are often above established goals and consequently, detailed site-specific evaluation is required on a regular basis, the remediation goals are of limited usefulness.

The health risk-based remediation goals for PAHs have been established using standard procedures and maximum exposure assumptions and are designed to represent an upper bound on likely risks. However, several unresolved issues greatly increase uncertainty in a risk assessment involving PAHs (and in risk-based goals) and, more importantly, make it difficult to determine whether risk is being over- or underestimated. Issues related to regulatory toxicology that affect uncertainty in risk estimates include the lack of a dose–response estimate for site-of-contact tumors caused by dermal exposure, questions regarding the accuracy of the available cancer slope factor for oral exposure, and the lack of an adequate approach for addressing the potency of mixtures of PAHs. Factors that affect uncertainty in exposure estimates include questions regarding the effect of the environmental matrix on the availability of the chemicals to a biological receptor and the lack of information on levels of those PAHs that are not detected using standard analytical procedures. A consideration of these issues is critical to defining the risk posed by PAHs at hazardous waste sites. These unresolved issues and their potential influence on risk assessment results are described here. In addition, cleanup goals for PAHs based on a consideration of both health risks and practicality are proposed.

**Background PAH Levels**

Many authors have measured levels of PAHs in the environment (1,2). In general, the lowest levels are seen in rural areas away from major highways. The International Agency for Research on Cancer (IARC) reported concentrations of 0.01–10 mg/kg for total PAHs (2) and Menzie et al. (3) listed concentrations in forest and rural soils of 0.01–1.3 mg/kg for carcinogenic PAHs (24). It should be noted that Menzie et al. (3) includes benzo(ghi)perylene among the carcinogenic PAHs, although this compound is not commonly considered to be carcinogenic (4). IARC (2) notes that somewhat higher levels of total PAHs are present in urban soils (1–100 mg/kg) and that industry

| Compound | New Jersey a | Michigan b | Oregon c | Illinois d | Washington |
|----------|--------------|------------|----------|------------|------------|
| Benzo[a]anthracene | 0.66 | 0.33 | 0.1 | 0.009 | — |
| Benzo[a]pyrene | 0.66 | 0.33 | 0.1 | 0.009 | — |
| Benzo[b]fluoranthene | ND | 0.33 | 0.1 | 0.009 | — |
| Benzo[k]fluoranthene | 0.66 | 0.33 | 0.1 | 0.009 | — |
| Chrysene | 0.66 | 0.33 | 0.1 | 0.009 | — |
| Dibenz[a,h]anthracene | 0.66 | 0.33 | 0.1 | 0.009 | — |
| Indeno[123-cd]pyrene | 0.66 | 0.33 | 0.1 | 0.009 | — |
| Total carcinogenic PAHs | 4.6 | 2.3 | 0.7 | 0.004 | (0.2) e |

ND, not determined.

aAll states allow adjustments to account for site-specific factors including elevated background concentrations.

bProposed New Jersey values are based on direct contact exposure to surface soils; proposed standards for subsurface soils (below 2 feet) are higher (100–500 mg/kg) (3).

bMichigan values are based on method detection limits for these compounds in soils; risk-based values established based on direct contact exposure were 0.2 mg/kg and were below method detection limits (4).

cOregon values are for residential exposure via direct contact (5).

dThe Illinois Underground Storage Tank Program established a health-based standard of 0.004 mg/kg for carcinogenic PAHs; however, this level is below the allowable detection limit for these compounds of 0.2 mg/kg (6). (Most states consider detection limits of around 0.1 mg/kg as reasonable for individual PAHs in soils.)

Total these values are the sum of individual criteria for the seven carcinogenic PAHs, except for New Jersey (a value of 0.66 mg/kg was used for benzo[b]fluoranthene) and Washington (only a total carcinogenic PAH value was available) (7).

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348 Environmental Health Perspectives
trial areas have even higher levels (1 to >100 mg/kg). Likewise, Menzie et al. (1) report levels of carcinogenic PAHs (plus benzo[ghi]perylene) in urban soils ranging from 0.06 to 5.8 mg/kg, with a median value of 1.1 mg/kg (n = 15). These authors also note that road dust contained very high levels of PAHs, with a median of 137 mg/kg and a range of 8–336 mg/kg (n = 7).

Issues Influencing Risk Assessments for PAHs

The original cancer slope factor of 11.5 (mg/kg/day)\(^{-1}\) developed for oral exposure to benzo[a]pyrene (BaP) and used as the basis for most PAH risk assessments, was derived from a study by Neal and Rigdon (9). Because this study lasted only 140 days out of an expected 2 years (730 days), an adjustment factor of (730/140)\(^{0.5}\) = 140 was incorporated into the cancer slope factor, following EPA policy for less-than-lifetime studies (10). The power function (K value) of 3 is based on a paper that shows that the overall tumor incidence in the human population increases with age by a power of at least 3 (11). Other researchers do not necessarily agree with the EPA adjustment factor of K = 3. For example, Crump et al. (12) assumes K = 4 for tumors in general. Humans do not have a forestomach, the tumor site in the Neal and Rigdon study (9). The human tissues most similar to the forestomach are the esophagus and the stomach, and Doll (11) noted K values of around 6 for both stomach and esophageal cancer. Consequently, an adjustment factor as high as (730/140)\(^{0.5}\) = 20,000 might be more appropriate for the Neal and Rigdon data. If this K value of 6 is appropriate, the corresponding cancer slope factor would be 800 (mg/kg/day)\(^{-1}\), approximately 100 times greater than the current value. Even a K value of 4 based on Crump et al. (12) would mean that the current cancer slope factor for BaP is too low by a factor of 5.

EPA has revised the cancer slope factor for oral exposure to BaP to a value of 7.3 (mg/kg/day)\(^{-1}\) (13). This value is the geometric mean of values from three analyses of the Neal and Rigdon data and one analysis of a study by Brune et al. (14). All cancer slope factors from these four analyses were within a range of 4.5–9.0 (mg/kg/day)\(^{-1}\). The Brune et al. (14) study lasted 2 years, and consequently, no adjustment for length of lifetime was necessary. However, this study by itself is probably insufficient to prove that a K value of 3 is appropriate for the Neal and Rigdon (9) study or that a cancer slope factor of around 7 (mg/kg/day)\(^{-1}\), and not 35 or 800 (mg/kg/day)\(^{-1}\) (the cancer slope factor associated with a K value of 4 or 6, respectively), is appropriate for BaP.

EPA has established an approach for calculating preliminary remediation goals (PRGs) for residential exposure via soil ingestion (15). Using standard EPA assumptions (15), a target risk level of 10\(^{-6}\), a cancer slope factor of 7.3 (mg/kg/day)\(^{-1}\), and EPA's equation for calculating PRGs for residential soil, a PRG for BaP of 0.1 mg/kg is determined. However, if a higher slope factor such as 800 (mg/kg/day)\(^{-1}\) is appropriate, a PRG as low as 0.001 mg/kg can be calculated.

BaP and other carcinogenic PAHs produce skin tumors at the site of contact in mice at very low doses, and evidence suggests that materials containing these compounds are also skin carcinogens for humans (16). The primary route of exposure to PAHs at many hazardous waste sites is likely to be direct dermal contact. There is currently no estimate of the dose-response relationship between dermal exposure and site-of-contact cancer, and this route of exposure is therefore not considered in setting remediation goals such as those listed in Table 1.

Sullivan et al. (17) estimated a dermal site-of-contact cancer slope factor for BaP of 6.6 × 10\(^{-4}\) (mg/cm\(^2\)/day)\(^{-1}\) based on data from a mouse skin-painting bioassay. This value is somewhat questionable because the doses used in the skin-painting study produced over 90% tumor response, and results presented in Clement (18) indicate that at these response levels, a very poor curve fit is achieved (19). Based on discussions with the author of the skin-painting study, Sullivan et al. (17) assumed that the exposed area of the mouse back was 30 cm\(^2\) and that three weekly skin-painting applications led to exposure for 3/7 of a week, or, in other words, that the BaP stays on the skin for 24 hr after application. Using these assumptions, a linearized multistage model (GLOBAL2), and data collected by Schmahl et al. (20) (Table 2), a dermal site-of-contact cancer slope factor of 2.3 × 10\(^{-3}\) (mg/cm\(^2\)/day)\(^{-1}\) is calculated, which is fairly close to the Sullivan et al. (17) estimate. However, information on the number of tumors per animal was not available and therefore it is probably more appropriate to express the cancer slope factor in terms of mg of chemical per whole animal exposed dermally. This approach, essentially eliminating the need to assume that 30 cm\(^2\) of skin was exposed, yields a slope factor of 760 (mg/exposed animal/day)\(^{-1}\). Using the cancer slope factor of 760 (mg/day)\(^{-1}\) under the assumption that exposure of equivalent surface areas of mouse and human skin will lead to the same response, and the further assumption that exposed areas will be similar (approximately 20–25% of the skin is exposed in EPA exposure estimates for humans and in skin painting studies on mice), this value can be used as a site-of-contact cancer slope factor for humans.

EPA has not developed an approach for determining allowable soil concentrations for site-of-contact carcinogens. However, EPA (21) has developed default assumptions for use in evaluating risks associated with dermal absorption of chemicals. Using these assumptions and the preliminary site-of-contact slope factor of 760 (mg/day)\(^{-1}\), a PRG for BaP can be calculated using the formula:

\[
PRG = \frac{TR \times AT \times 365 \text{ days/year}}{SF \times EF \times ED \times CR \times SA \times AB}
\]

where TR is the target excess cancer risk (1 in 1,000,000 or 10\(^{-6}\); AT is the averaging time (70 years); SF is the dermal site-of-contact slope factor (760 (mg/day)\(^{-1}\); EF is the exposure frequency (central estimate: 40 days/year); ED is the exposure duration (upper estimate: 30 years); CR is the contact rate (central estimate: 2 × 10\(^7\) cm\(^2\)/hr); SA is the surface area (central estimate: 25% or 5000 cm\(^2\); and AB is the percentage available from soil relative to availability from solvent used in the skin painting study that is the basis for the slope factor (best professional judgment: 1% or 0.01%)

Based on this equation, a PRG that is protective for direct dermal exposure to BaP is calculated to be 0.003 mg/kg. This value is well below risk-based regulatory limits established for BaP and other carcinogenic PAHs.

### Table 2. Dose levels and results from the Schmahl et al. (20) study

| Compound administered | Dose groups | Dose levels* (μg/treatment) | Cancer incidence, % |
|-----------------------|-------------|----------------------------|---------------------|
| Benzo[a]pyrene        | A1          | 1.0                        | 13 (10/81)          |
|                       | A2          | 1.7                        | 28 (25/88)          |
|                       | A3          | 3.0                        | 53 (43/81)          |
| Control group         |             | 0                          | 1 (1/100)           |

*BaP was administered to the shaved skin of mice twice a week until the natural death of the animals or until the animals developed a tumor. At the start of the study, each dose group consisted of 100 animals, but autolysis limited the total number of animals examined in each group.

#No untreated control group was used in this study. However, based on the results from a group exposed to very low levels of “noncarcinogenic” PAHs, an incidence rate of 1/100 was assumed for an untreated control group in calculations.
cinogenic PAHs, and although preliminary, suggests that further investigation of dermal exposure is warranted. The value is in the same range as the PRG developed based on ingestion exposure and an oral slope factor for BaP of 800 (mg/kg/day)^1, and taken together, these values indicate that current criteria may pose risks above the 10^-6 risk level used as a goal by many regulatory agencies.

Carcinogenic Potency of PAH Mixtures

The PAHs consist of a large family of compounds with a rather large range of toxic potency (2,16). PAHs are seldom found separately in the environment; rather, they occur as complex mixtures of numerous compounds. In calculating site risks, EPA and most state agencies historically separated the PAHs into two categories: carcinogens and noncarcinogens, and treated all the carcinogenic PAHs as equipotent with BaP, one of the more potent PAHs. This approach oversimplifies the situation, as some of the “carcinogenic” compounds are clearly more potent than others, and some of the “noncarcinogenic” compounds appear to have some weak carcinogenic activity or to act as cancer promoters or cocarcinogens (16).

Several authors have evaluated the available data on the carcinogenic potency of different PAHs and developed toxicity equivalency factors (TEFs) for the individual PAHs (18,19,22). These TEFs indicate the carcinogenic potency of each compound relative to BaP, and multiplying the concentration of each PAH by the TEF yields a concentration for the total PAH mixture that is expressed in terms of an equivalent concentration (with regard to toxic potency) of BaP, called BaP equivalents (BaPeq). Table 3 presents the TEFs developed by the various researchers, and Table 4 indicates the BaP equivalent concentrations determined using the historical EPA approach, the current EPA approach (22), and the Nisbet and LaGoy (19) approach on some PAH data from a coal-tar-contaminated soil sample (23). An evaluation of these data indicates that using appropriate TEFs rather than assuming all carcinogenic PAHs are equipotent with BaP would decrease the conservatism in the risk values by a factor of approximately two to three.

Unreported PAHs

The standard EPA analytical methods (methods 625 and 8270) test for the presence of only 17 of the many PAHs likely to occur in environmental samples. The other PAHs may contribute to risk and, by their absence from standard analyses, contribute to the uncertainty in the risk assessment. Over the years, considerable effort has been directed at identifying the carcinogenic components of petroleum products, with much of this effort focused on the PAHs. Consequently, the 17 PAHs that are analyzed in the standard EPA procedures may pose a substantial portion of the risk in most materials. In the one study reviewed in detail (23), the 17 regularly analyzed PAHs accounted for 40% of the total concentration, twice their expected contribution given that a total of 74 PAHs were detected. However, the converse is that 60% of the PAHs in this mixture would be routinely overlooked and consequently not considered in risk estimates. Furthermore, as noted by Poitier (8), certain methylated PAHs and PAHs containing nitrogen or oxygen may be quite potent carcinogens and if present could pose substantial risks.

| Compound                  | Clement (18) | EPA (22) | Nisbet and LaGoy (19) |
|---------------------------|--------------|----------|-----------------------|
| Benzo[a]pyrene            | 1            | 1        | 1                     |
| Dibenzo[a]anthracene      | 1            | 1        | 1                     |
| Benzo[a]anthracene        | 0.145        | 0.1      | 0.1                   |
| Benzo[b]fluoranthene      | 0.140        | 0.1      | 0.1                   |
| Benzo[k]fluoranthene      | 0.066        | 0.01     | 0.1                   |
| Dibenzo[ghi]perylene      | 0.232        | 0.1      | 0.1                   |
| Anthracene                | 0.32         | ND       | 0.01                  |
| Benzo[ghi]perylene        | 0.022        | ND       | 0.01                  |
| Chrysene                  | 0.0044       | 0.001    | 0.01                  |

ND, not determined.

| Detected compound         | Measured | EPA | EPA | Nisbet/Lagoy |
|---------------------------|----------|-----|-----|--------------|
| Benzo[a]pyrene            | 39       | 39  | 39  | 39           |
| Dibenzo[a]anthracene      | 15       | 15  | 15  | 75           |
| Benzo[a]anthracene        | 51       | 51  | 5   | 5            |
| Benzo[b]fluoranthene      | 36       | 36  | 4   | 4            |
| Benzo[k]fluoranthene      | 36       | 36  | 0.2 | 4            |
| Indeno[123-cd]pyrene      | 36       | 36  | 4   | 4            |
| Anthracene                | 12       | 0   | 0   | 0.1          |
| Benzo[ghi]perylene        | 31       | 0   | 0   | 0.3          |
| Chrysene                  | 46       | 46  | 0.05| 0.5          |
| Acenaphthene              | 2        | 0   | 0   | <0.01        |
| Acenaphthylene            | 1        | 0   | 0   | <0.01        |
| Fluoranthe                | 58       | 0   | 0   | 0.06         |
| Fluorene                  | 13       | 0   | 0   | 0.01         |
| Methyl naphthalene        | 3        | 0   | 0   | <0.01        |
| Naphthalene               | 6        | 0   | 0   | <0.01        |
| Phenanthrene              | 19       | 0   | 0   | 0.02         |
| Pyrene                    | 82       | 0   | 0   | 0.08         |
| Subtotal PAHs             | 486      | 47  | 47  | 132          |
| Total BaP equivalent      | 259      | 67  | 67  | 132          |

*Historical approach that assumes all carcinogenic PAHs are as potent carcinogens as BaP.

aEPA (22) toxicity equivalency factors (TEFs) are based in large part on the TEFs developed by Clement (18).
bBased on the TEFs developed by Clement and LaGoy (19).

cIn this study (23), 74 PAHs were detected with a total concentration of 1115 mg/kg. The 17 compounds included in standard analyses represented 486/1115 or over 40% of the total amount of available PAHs.
Bioavailability

To produce toxic effects, PAHs must be available to the target tissue (i.e., must be bioavailable). The carcinogenic PAHs are commonly found in nature in association with other high molecular weight organic compounds (e.g., asphaltene), and these other compounds probably decrease the bioavailability of the PAHs. In addition, PAHs with highly octanol-water partition coefficients tend to bind tightly to most soils, particularly if they have been in contact with the soils for a considerable length of time (21). Consequently, the carcinogenic PAHs may not be readily bioavailable in some situations.

Remediation Goals for Carcinogenic PAHs

Generic, state, or region-wide remediation goals for soils are generally established at the highest of the following: 1) risk assessment-based values, with a cancer risk level of 10−6 commonly used as a target risk level, 2) MDLs, or 3) regional background soil levels. A caveat is that if MDLs are well above risk-based values, regulatory agencies may require the use of more sensitive analytical techniques to achieve remediation goals that are close to risk-based levels. The remediation goals listed in Table 1 were developed with either risk-based values or method detection limits as the criteria. Considering that MDLs and the allowable levels based on previous estimates of residential risks via soil ingestion are reasonably close and that both are above concentrations of carcinogenic PAHs in rural background soils, this approach was appropriate. However, evaluation of the issues affecting risk assessment described above and of the more detailed review of background PAH levels by Menzie et al. (21) suggests that these cleanup criteria are not reasonable.

A preliminary analysis of site-of-contact risks and reevaluation of the oral slope factor suggests that generic remediation goals (10−6 risk) for carcinogenic PAHs may be close to 0.001 mg/kg, well below even rural background PAH levels. In addition, urban soils are probably more representative of background in areas near hazardous waste sites, and, as noted by Menzie et al. (21), background levels of carcinogenic PAHs in urban soils ranged from 0.06 to 6 mg/kg, with a median of 1 mg/kg. Considering that risk-based remediation goals are unachievable, urban background concentrations in soils appear to be the most appropriate basis for setting remediation goals at hazardous waste sites.

In establishing remediation goals based on background chemical concentrations, states such as New Jersey and Washington have generally used upper-bound estimates of likely background (3, 7). Based on the data reviewed by Menzie et al. (21), upper-bound estimates of background levels of carcinogenic PAHs are around 1 mg/kg for rural and forest soils and 6 mg/kg for urban soils. Although the value for urban soils is probably most appropriate, considering the fact that risk-based cleanup goals would be several orders of magnitude below this level, it seems most prudent to set levels “as low as reasonably achievable.” Consequently, a level of 1 mg/kg as BaPeq (rural background) should be used as a target remediation goal. However, because there will clearly be cases where background concentrations exceed this level, a second-tier value of 10 mg/kg (6 mg/kg rounded to reflect the likely uncertainty in this value) is also recommended. This second-tier value should be used if, based on a subjective review, exposure appears unlikely to involve frequent or repeated soil contact. For example, levels below 10 mg/kg would be acceptable as remediation goals at an industrial site or at a grass-covered park and might be acceptable in well-vegetated residential areas. Substantially higher levels would also be allowable if supported by a risk assessment and by site-specific considerations.

Establishing cleanup criteria based on both practicality and risk is similar to the approach used for years to establish goals for radionuclides, namely, setting levels as low as reasonably achievable (ALARA). The concept of ALARA was required for radionuclides because it was recognized that even very low and therapeutic doses had some recognizable health risk. PAHs and radionuclides are similar in that both are naturally and anthropogenically produced materials that regularly occur at levels above health risk-based goals.

Discussion and Conclusions

Because large numbers of sites will contain PAHs and definition of cleanup criteria can affect remediation costs by millions of dollars, it is important to establish reasonably achievable remediation goals for PAHs. Current remediation goals do not consider several factors that are likely to substantially impact risk estimates. The lack of a cancer slope factor for dermal exposure, questions about the oral cancer slope factor, and testing for only 17 of the numerous PAHs are all likely to lead to underestimating risks. On the other hand, not accounting for differences in the relative potency of individual PAHs and ignoring bioavailability issues may lead to overestimating risks. Because of these factors, it may not be possible at this time to firmly establish a risk-based remediation goal. However, it also appears that background soil concentrations may be more appropriately as remediation goals in any case. Considering the toxicity of this class of compounds but also considering their widespread presence in the environment, a two-tiered system is proposed based on the concept of ALARA: a tier 1 level of 1 mg/kg of PAHs measured as BaPeq is recommended as a remediation goal; if PAHs are below this level, no further action is required. A tier 2 level of 10 mg/kg of PAHs measured as BaPeq is recommended as well; if PAHs are below this level, a subjective evaluation of likely current and potential future land use is required. If it appears unlikely that frequent exposure would occur, no further action is required.

Obviously, higher soil levels may also be acceptable if, based on a risk assessment, exposure is determined to be unlikely. For example, levels well over 100 mg/kg BaPeq may not pose a health risk if covered with a surface soil layer and digging into the soils would occur infrequently. Higher levels may require restrictions on future land use to ensure safety. Alternative risk assessment procedures designed to measure the bioavailability of PAHs from soils or the effect of contaminated soils on organisms may also be useful in assessing site risks and in establishing site-specific cleanup criteria (24–29).

Research to address some of the issues raised in this article (e.g., oral cancer slope factors; the effect of mixtures) is underway in both government and private laboratories. However, much work remains to be done, particularly in the areas of determining an appropriate dermal site-of-contact slope factor, evaluating the effects of unanalyzed compounds, and in assessing the bioavailability of PAHs from environmental media.

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