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September 17, 2008

Ligia Mora-Applegate
Bureau of Waste Cleanup
Florida Department of Environmental Protection
2600 Blair Stone Road
Tallahassee, FL 32399

Re: Human Health Risk Assessment, Cabot Carbon/Koppers Superfund Site

Dear Ms. Mora-Applegate:

I have reviewed at your request two documents related to the proposed human health risk assessment by AMEC. One is titled "Description of Cabot Carbon/Koppers, Gainesville, Florida On-Site Worker Microexposure Event Model and Example of Model Calculation." This document was produced as a follow-up to a request made during the June 30, 2008 meeting in Tallahassee for more information on the microexposure event model. The second is titled "Relative Absorption Factors (RAFs) for Oral and Dermal Absorption of Compounds in Soil, Cabot Carbon/Koppers Site, Gainesville, Florida." The latter document reviews the literature on relative bioavailability of PAHs, dioxins, and arsenic, and provides distributions for RAFs to incorporate in the risk assessment. My comments on these documents are outlined separately below.

Microevent Exposure Model

This document is very helpful in explaining how the microevent exposure model works, but greater clarity on the approach has led to more questions and comments:

1. The current model default is to develop risk calculations for male workers (see page 1). This affects the choice of body weight, surface area, inhalation rate, and perhaps other distributions. What is the rationale for restricting the analysis to male workers?

2. The document isn't clear how many variability loops and how many uncertainty loops will be run. How will the number of loops be selected to insure stable output?

3. Dermal adherence factors, relative bioavailability factors, and toxicity values are considered uncertain parameters in the model and will be entered as distributions.

    a) As noted previously, current U.S. EPA guidance recommends using point estimates rather than distributions for toxicity values in probabilistic risk assessments. Risk distributions could be produced with different toxicity values as point estimates as part of an uncertainty analysis. However, risk management decisions will be guided primarily by risk values generated using toxicity values for which FDEP has the most confidence (i.e., as taken from their hierarchy of toxicity value sources).

b) Risk calculations are performed with a default oral relative bioavailability factor of 1[1] unless there is compelling evidence for an alternative, site-specific value. With the exception of lead, for which an *in vitro* method of estimating relative bioavailability from soil has been approved by the U.S. EPA, adjustment of the relative bioavailability factor in human health risk assessments requires an *in vivo* study using a suitable animal model. Risk calculations using a distribution of relative bioavailability factors from the literature can be used to ask "what if" questions typically found in the uncertainty section of a risk assessment, but risk calculations using the default relative bioavailability factor as a point value need to be provided as a basis for regulatory decision-making.

c) Uncertainty contributed by the choice of dermal adherence factors could be addressed in the risk assessment, although it is doubtful that the dermal route will be a substantial contributor to total risk at this site. It will be most useful to show the risk result when using the default dermal adherence factor, and a comparison with risk using other dermal adherence assumptions can be presented and discussed in the context of an uncertainty analysis.

4. Because the risk assessment is focused on current operations by Koppers, Inc., the exposure frequency and duration distributions should be based on employment information specific to the Koppers Gainesville site rather than literature values.

5. The COPC concentration in soil is treated as a variable; that is, the concentration to which an individual will be exposed on a given day will vary. It is not clear how the distribution of exposure point concentrations for a chemical will be derived so that it reflects the spatial distribution of contaminants and activity patterns of workers. More information is needed to be sure that this distribution accurately reflects what is intended.

6. The upper 75th percentile soil ingestion rate from Stanek et al. (1997) of 50 mg/d is used as the maximum soil ingestion rate. The Exposure Factors Handbook (US EPA, 1997 Table 4-23) lists 50 mg/d as the median ingestion value for outdoor workers. It is questionable whether the incidental soil ingestion rate distribution chosen for the model is relevant for outdoor workers with significant soil/dust exposure.

7. Table 1 shows some example distributions for the model. Concerns about the soil concentration, incidental ingestion, exposure frequency, and exposure duration distributions are noted above. For example, the distribution for exposure duration indicates that half of the workers at the facility are there for only about 2.5 years or less, and 25% of the workers last 6 months or less. Job turnover rates this high at Koppers would need to be documented.

8. The microexposure event model utilizes six hours per day as the minimum daily exposure duration and eight hours per day as the median. This results in the model choosing a workday less than eight hours half of the time. It is unclear if this distribution reflects current workday durations at Koppers Inc. The maximum, minimum, and median values for exposure time should be obtained from site-

[1] In Florida, the default oral relative bioavailability factor for arsenic in soil is 0.33.

specific Koppers, Inc. data. Documentation that specifies the minimum and maximum length of shifts at the site should be included.

9. As has been discussed previously, FDEP [and presumably the U.S. EPA] will need to have access to software to confirm model outputs. AMEC has expressed a willingness to provide this software with appropriate restrictions to protect their intellectual property.

Relative Absorption Factors (RAFs)

This document provides a summary of the literature on the oral and dermal bioavailability of PAHs, dioxin, and arsenic from soil. While there are some technical comments on the interpretation of some of the studies (below), the principal limitation of the data overall is that they provide no insight specifically with regard to bioavailability of these chemicals from soil at the Koppers Gainesville site. There is ample evidence from the literature that RAF values for chemicals can vary from site to site due to factors that are still not completely understood. At best, literature summary values can provide some indication of the range of RAF possibilities, but even in this respect they are limited. For example, the most extensive literature summary is for the PAHs. Studies have found RAFs ranging from 0.08 to 1.0 for pyrene (Table 7) and 0.07 to 0.76 for benzo(a)pyrene and carcinogenic PAHs (Table 8). This means, in effect, that almost any relative bioavailability from <0.1 to 1.0 is possible for the PAHs. If a large data set of RAF measurements was available for a variety of sites, it might be possible to consider what the RAF for PAHs might be at the Koppers facility strictly as a matter of chance. Conceptually, that is the approach proposed for the Koppers human health risk assessment using a probability distribution of RAFs from the literature survey. The problem with this approach is that a large data set of PAH RAFs doesn't exist. Results presented in Tables 7 and 8 vary to some extent because they represent different soil samples, but can also vary because they were measured using different models and approaches. There aren't enough measurements taken using consistent methodology to determine with any confidence what a distribution of RAFs for the universe of PAH sites should look like, yet the shape of the distribution will have a pronounced effect on the PAH risk distributions. The distribution chosen for PAH oral RAFs (Figure 2) will use values most extensively from the lower end of the range of possibilities (e.g., 50% of iterations will use a value of 0.3 or less). I think that this has the potential to bias the risk calculations for PAHs low.

Even less data are available for RAFs for dioxin and arsenic, making it unclear to what extent the true range of possible RAFs has been characterized. In the case of arsenic, a distribution of values is proposed based on a single measurement of the RAF for soil arsenic from a wood treatment facility. It is not clear to me how a distribution of RAFs for arsenic for wood treatment facilities can be generated from an N=1.

It is reasonable to propose that the RAF for PAHs, dioxins, and arsenic from soil at the Koppers site may be less than 1 (or less than the FDEP default of 0.33 in the case of arsenic), and the literature summarized in this report make that point strongly from a qualitative standpoint. However, to make an adjustment in the risk estimates for a specific site, the RAF(s) applicable to that site must be determined with a reasonable level of confidence. Currently, because the factors that control RAF are not well understood, that means site-specific RAF measurements.

Specific comments related to the document are as follows:

1. The authors point out that assessment of gastrointestinal absorption of PAHs by measuring unchanged PAH in the feces is potentially affected by the excretion of absorbed, unchanged PAH in the bile (see, for example, page 4). It should also be noted that metabolism of PAHs by gastrointestinal microflora (e.g., van de Wiele et al., Environ. Health Perspect. 113:6-10, 2005) can also influence this measurement.

2. In the first complete paragraph on page 5, fecal excretion of unchanged B(a)P at 94-98% is taken to mean a high degree of absorption, when of course it indicates the opposite. The presentation of data must be in error.

3. The study by Withey et al. (1991) is criticized because bioavailability is measured using blood data (area under the curve) (see pages 5 and 6). It is claimed that the fraction that leaves the blood and distributes to tissues is not properly counted. Unless there is reason to believe that the distribution of labeled PAH is different following oral administration than following intravenous administration, this criticism is not valid. Blood data and urinary excretion data gave very different values for the bioavailability of PAH. It is not clear to me why the best solution in this situation is to simply average the values for the two different approaches, as was done for this analysis.

4. Among the data used for RAF development are results from the study of Bartosek et al. (1984) (page 6). In this study, PAH was dosed in Pluronic F68 emulsifier to fasted animals. Given that the information needed is absorption of PAH from food, results from this study do not appear to be relevant.

5. In the study by Weyland et al. (1996) (page 9), different diets were used for soil versus extract. This might have influenced the results. Also, it is interesting to note the relatively low excretion of pyrene in this study. Possible explanations are substantially different biliary excretion or lower absorption from diet compared to other studies, although there is no obvious reason for either. The inconsistency does raise questions about the study.

6. The report by Magee et al. (1999) (page 17) is available only as an abstract. I'm not sure if sufficient information is available to properly evaluate the quality of this study. Also, the approach used to estimate bioavailability [based on lung DNA adducts] assumes linear kinetics for a myriad of processes between the absorption in the gut and DNA adduct formation in the lungs. Without evidence for linearity, the reliability of these estimates is suspect.

7. The RAF values presented by Gron et al. (2007) are based on a comparison of the absolute bioavailability of PAH from soil versus PAH in hexane solution, rather than diet.

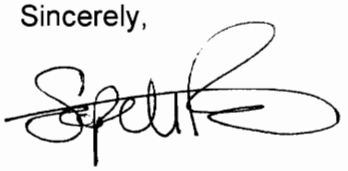
8. The RAF analysis for dioxin uses data from fly ash: "AMEC assumed that the RAF (oral-soil) equals the oral fly-ash RAF." No explanation why bioavailability from soil and fly ash should be considered equivalent is provided.

9. The paper by Budinsky et al. (Chemosphere 70:1774-1786, 2008) is an important study of relative bioavailability of dioxin from soil, but is not included in the analysis. In addition to providing RAF values for one site, it offers information about challenges in measuring bioavailability of dioxins *in vivo* that are useful in evaluating other studies.

10. The uncertainty distribution for the RAF for oral exposure to arsenic in soil is based on the mean and standard deviation of five measurements from a single sample in the primate study of Roberts et al. (2002). The sample is from a wood treatment facility. This mean and standard deviation is an expression of variability among individual experimental subjects. It might be considered representative of potential variability in RAF among individuals (although it is likely an underestimate, given that the number of subjects is small and the population relatively homogenous), but provides no indication of how the RAF might vary among different wood treatment sites. Consequently, it might be considered applicable to an evaluation of variability, but not uncertainty, associated with the RAF assumption for arsenic.

Please let me know if I can be of addition assistance in planning the human health risk assessment for this site.

Sincerely,



Stephen M. Roberts, Ph.D.