Letter to the Editor

Assessment of Asphalt Workers’ Dermal Exposure

We agree with McClean and his co-workers (McClean et al., 2004) that urinary 1-hydroxypyrene concentration is a useful measure of total absorbed dose of polycyclic aromatic hydrocarbons (PAHs). Many occupational health studies have reported low correlations between inhalation exposures and urinary 1-hydroxypyrene levels, suggesting that other exposure routes (in particular dermal, but also ingestion) play a role, as well as non-occupational PAH sources.

However, we do not agree that the data presented by McClean et al. justify the estimate of an 8-fold higher contribution of the dermal route than the inhalation route during asphalt paving work for the following reasons:

- Only 61% of dermal samples from paving crews contained detectable amounts of pyrene, and many data points were not much above the limit of detection. In fact, the median value presented in Table 1 for paver operators is lower than the detection limit. The authors do not specify how they have treated results below the detection limit in their data analysis. In view of the large proportion (39%) of non-detectable results in this study, the choice of any substitute value in the data analysis, such as the often used figure of half of the detection limit, may have a disproportionate impact on the outcome and it is important to explain to the reader which approach has been chosen.

- The statistical data analysis appears less than rigorous. The authors indicate that ‘statistical significance is reported at the 0.05 level’, but then introduce the wording ‘marginally significant’ for differences with higher P-values, including the difference (P = 0.1) for dermal exposure between pavers (the exposed group) and mixers (the control group) and the effects of age, body mass index and smoking on urinary 1-hydroxypyrene levels (P-values between 0.06 and 0.09). Most importantly, however, the authors indicate that the effect of inhalation in Model 3 is not significant, so in our view this cannot possibly provide the basis for the comparison of contributions from dermal and inhalation routes.

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Reply

We thank Boogaard and Urbanus (2004) for their comments on our article (McClean et al., 2004) and welcome this opportunity to respond.

Boogaard and Urbanus comment that ‘the authors do not specify how they have treated results below the detection limit.’ In the data analysis section of our
article, we state that all values less than detection limits were used in all analyses (p. 568). This approach was selected because many 'non-detected' values were detected above the instrument detection limit, but after blank-correcting were below the method detection limit which was conservatively estimated as three times the standard deviation of the field blanks. The use of actual concentrations, whether or not below the limit of detection, is a valid approach for the statistical analysis of environmental data (Gilbert, 1987, pp. 177–8).

Additionally, Boogaard and Urbanus comment that 'the statistical data analysis appears less than rigorous’ because the term ‘marginally significant’ is used in reference to \( P \)-values between 0.06 and 0.1. We used polynomial distributed lag models and linear mixed-effects models as a novel and rigorous approach to analysing biological exposure data, and provided the \( P \)-values for all results. \( P \)-values between 0.06 and 0.1 are commonly referred to as marginally significant, and this interpretation had no impact on the model building process owing to the a priori decision to control for age, body mass index and smoking status in all analyses of urinary 1-hydroxypyrene.

Lastly, Boogaard and Urbanus comment that the effect of inhalation is not significant and therefore cannot provide the basis for comparing the dermal and inhalation routes. This comment refers to the finding that dermal exposure had a significant cumulative effect on urinary 1-hydroxypyrene levels whereas the cumulative effect of inhalation exposure was not significant. Our estimate that ‘the impact of dermal exposure was approximately eight times the impact of inhalation exposure’ was based on a comparison of the mean estimates for each pathway, a comparison that we made even though the inhalation estimate was not significantly different from zero. We acknowledge that there is uncertainty associated with both the dermal and inhalation effect estimates, but disagree that the effect of inhalation must be significant for us to be justified in our comparison.

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