Risk Analysis, Vol. 25, No. 1, 2005

Response to Letter to the Editor

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Dear Editors,

The recent "Letter to the Editor" in response to our article, "Children's Exposure to Arsenic from CCA-Treated Wooden Decks and Playground Structures," not only reflects differences of judgment between us and the writers of this letter, but also contains errors of fact or interpretation. Among the more important issues are the following:

1. The writers of this letter are biased in their suggestion that our paper must be in error because it does not agree with results of Gradient (2001), CPSC (2003), Dubé *et al*. (2004). The writers cite studies that represent the low end of the range, while failing to cite others that suggest that larger exposures may occur (e.g., EWG, 2001; CalDHS, 1987; Maas, *et al*. 2003; Roberts & Ochoa, 2001). Moreover, there is no logic by which the writers can support their implication that our paper must be in error because we suggest that children's exposures to As from CCA-treated wood could exceed exposures to As from food or water. Instead, we believe that the results of such a comparison underscore the need for further studies of children's As exposure from CCA-treated wood.
2. The writers of this letter seem to argue that the validity of data can be judged on the basis of its age, and that observations become "outdated" after a few years and should be "replaced or enhanced by considering more recently collected information." This view is contrary to the tenets of modern science, in which the acceptability of data is judged not on its age, but on the adequacy of the methods, taken in context of the questions to be answered. In our article, we were exhaustive in searching the literature for all available skin-loading data, and we also devoted over two pages to critical review of the procedures used in each study. Some of the "more recently collected information" that the writers of the letter refer to is not yet published (e.g., Dubé *et al*., 2004), and can more appropriately be addressed via open scientific debate once it is in the public domain.
3. Although the writers assert that there are not important differences between moist versus dry hand loadings, in each study where both are measured, the result is that the former are systematically higher, sometimes by a large margin, as is evident by inspection of our Table I. Carlson-Lynch and Smith (1998) specifically conclude that wet hands acquire more As than dry hands, and both CPSC (2003) and Arsenault (1975) data show the same result. We chose to use moist hand data in our sample order-of-magnitude exposure calculations for the simple reason that children's mouths are moist, and we are considering ingestion via the hand-to-mouth pathway. The writers are in error in claiming that we "discarded" a number of results for dry hands; those data are all presented clearly in our Table I. Their statement that we used four moistened hand samples in our sample calculations is misleading. While the grammar *technically* says that we used four data from the CPSC study, the sentence can easily be

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0272-4332/05/0100-0007$22.00/1

misinterpreted that we used four data points total, whereas in fact we used a total of 37 data points, deleting with explanation two of the *highest* in calculating an average hand loading.

4. The statement that the studies we cite "provide almost no information regarding measures taken to control for background hand loading or contamination of the hand rinsates" is wrong. Systematic field blank procedures are described, for example, by CalDHS (1987)(these workers actually tablulate their blank values) and by Carlson-Lynch and Smith (1998).
5. The writers argue that the studies that we cite are inadequate because they are based on only one or a few wood samples or wood structures. Indeed, even a single playset or deck is constructed of many pieces of wood, thus inherently providing a significant amount of sampling diversity.
6. Despite an extensive search, we found only one report of measurement of urinary excretion of arsenic from an individual exposed to CCA-treated wood (CalDHS, 1987). Of course additional data are desirable; this is one of the statements that we make in our abstract. The writers are incorrect, however, in criticizing this one experiment as having limited documentation; in fact, as we state, urinary concentrations of arsenic were measured over a period of four days. A total of 27 data points documenting the As to creatinine ratio are reported.

   Further, it is a matter of curiousity that the letter writers, who make a point of "the complexities of living organisms and differences between dislodgeable material and pure chromium arsenate," would make the claim that we should have given precedence to *in vivo* animal studies over theoretical solubility considerations in our discussion of bioavailability of dislodgeable As. We stand by our approach, which is to critically evaluate evidence both ways, and not to claim precedence of one over the other at this time. The use of animal models for evaluating bioavailability of As in humans has been questioned for reasons that include differences in gut microflora, differences in diet, and species-specific metabolism, which results in variable degrees of methylation of arsenic (SAP, 2001). As a final and somewhat ironic point, the writers of this letter cite Casteel (2003) as their recommended authority on dislodgeable As bioavailability, but Casteel in turn cites a 1991 ("outdated"?) article, on the topic of *lead* bioavailability, as justification for use of the swine model. Given the major differences in the chemistry of As and Pb, the assertion that such bioavailability experiments deserve "precedence" is unfounded. They should be weighed, together with all evidence, as part of an ongoing effort to understand the As uptake process.
7. The writers of the letter go on to claim that the 30% bioavailability results of Casteel (2003) invalidate our use of 50% as an estimate of relative bioavailability of dislodgeable As. In fact, even if the issues of interspecific comparison (humans vs. swine, as noted above in Point 6) are discounted, various additional sources of uncertainty are likely larger than the difference between 30% and 50%. For example, the material that Casteel *et al*. (2003) fed to swine was removed from wood by brush, concentrated by rotary evaporation, dried, and irradiated using Cobalt-60 prior to dosage. Was the resulting material really the same as material dislodged by children at play on CCA-treated wood, given the mode of collection and subsequent possible alteration of physical-chemical form? The fact that the dried material contained only 3.5 mg/g As suggests that it was rich in wood particles still containing fixed As, whereas the material that renews itself on the surface of CCA-treated wood and comes off on children's hands might be more soluble and hence bioavailable. Finally, the precision and accuracy that can be anticipated in bioavailability studies is easy to overstate. For example, in another report Casteel *et al*. document nearly identical experiments on the bioavailability of As in soil, and as part of each bioavailability study they independently measured a urinary excretion factor for soluble inorganic arsenic. Results were 68% in one study versus 81% (about 20% higher) in the other.
8. The writers cite a new study by Wester *et al*. (2004) (not available when we submitted our article), in which a dermal absorption factor of 0.1% was found. This is compared with our estimate, whose uncertainty we discuss thoroughly, of 2%. Although we suggested that dermal absorption was indeed

significantly less important than ingestion in the case of CCA, we pointed out the large uncertainty. We also feel that further dismissal of this exposure pathway is premature. First, Wester *et al*. (2004) use dry-processed material removed from CCA wood with a brush. Apart from the concerns about sample processing (see Point 7), dry material may also result in less absorption than may occur when skin is moist, as it may be in children at play in warm weather. Second, other recent evidence suggests that toxicity of dermally absorbed As may result directly to the skin by arsenic that does not enter the circulatory system or become excreted in urine. Bernstam *et al*. (2002) demonstrate strong skin absorption of As(III) and associated cytotoxicity at levels as low as 10 μg/L for aqueous exposure. Further, Khan *et al*. (2004) have recently found that a significant fraction of arsenic leaching from weathered wood is in the form of As(III), not As(V) as commonly assumed. Further, other uninvestigated pathways, such as As entry through the skin via splinters and abrasions, could yet prove to be significant. Much further research is needed, but it is still possible that in considering only percutaneous As(V) absorption we have underestimated rather than overestimated the significance of dermal exposure.

9. The writers object to our use of a mechanistic model for estimating ingestion of As from estimates of hand loading, hand-to-mouth contact frequency, and transfer efficiency from the hand to the oral cavity. This approach, however, is the norm in risk analysis approaches for assessing children's exposures to CCA-treated playgrounds. Although additional data would be highly desirable, the authors consider that sufficient data are available to make informed estimates for each of the parameters needed for this risk analysis approach. The mouthing frequency rate we used in a sample calculation was based upon the work of Reed *et al*. and is consistent with several other studies, as well as with the mean value utilized by Zartarian *et al*. (2003) in the most recent U.S. EPA risk assessment, which is used as the basis of comparison by the authors of the "Letter to the Editor."
10. Rates for children's hand-to-mouth contact as discussed by Freeman *et al*. (2001) are certainly relevant, but do not suffice to make a case that estimates such as we make are necessarily too high. Data from children under three years are lacking, and in fact Freeman *et al*. point out that this group has higher hand-to-mouth activity than do the older children that they studied. Freeman *et al.* also point out that hand washing by children is infrequent. From this we infer that As accumulated on a child's hands may continue to be transferred to the mouth after they stop playing on a deck or playset, thus increasing the effective duration of As ingestion beyond play time. Other factors that could lead to greater ingestion, but that have not yet been quantified and are not included in exposure estimates, include: foot-to-mouth activity of young children (Freeman *et al*., 2001), who presumably may walk or crawl barefoot on CCA-treated structures, hand-to-food-to-mouth transfer during snacks and meals (Freeman *et al*., 2001), or even direct mouthing of CCA-treated wood, as observed by one of us (Solo-Gabriele).
11. The writers are in error regarding their interpretation of our use of a 100% removal efficiency. As we discuss, what is relevant is the effectiveness of removal of As by mouthing *relative to the efficiency of the rinsing processes employed in hand-loading experiments*. One hundred percent efficiency thus means that mouthing is taken to be about equal in effectiveness to the various rinsing protocols. The hand-cleaning processes employed in loading experiments are typically not very rigorous, and indeed CalDHS (1987) demonstrated that their procedure left measurable levels of As on the hands. There is no current basis for judging whether hand-cleaning procedures used in dislodgeable As experiments are more or less effective than children's mouthing activity in removing dislodgeable As from hands. Thus a 100% removal efficiency is not only reasonable to assume, but could even be an underestimate.
12. We did in fact consider an analogous approach similar to the "benchmarking" calculation the writers of the letter suggest (see section called "Calculations Based on Analogy with Soil Ingestion"). Such an approach is, however, characterized by its own considerable set of uncertainties, not the least of which is the likelihood of major differences in both adhesion and

ingestion of soil particles and CCA residues. Thus, in the specific case of CCA residue, we judged a mechanistic approach to be not only more straightforward, but also more realistic, more readily testable, and more able to be refined in future studies.

13. The authors of the "Letter to the Editor" reiterate in their summary statement the claim that our estimates are a magnitude greater than those reflected by studies that they assert are "more recent and more comprehensive." Again, we point out that several risk assessments have been conducted showing a wide range of variability (e.g., Gradient, 2001; EWG, 2001, etc.), but the authors of the letter have chosen to cite only studies, including one not yet published, that provide exposures on the low end. This does not advance the science, which should proceed by reasoned debate rather than selective citation. In the case of studies not yet published, such debate becomes more productive after the work becomes available in the public domain for all parties to access.
14. The finding that our exposure assessment is greater than the estimated dietary and drinking water uptake in young children in no way discredits our estimates. Rather, it emphasizes the need to evaluate exposures from CCA-treated wood further. To reiterate, the exposure assessment we provided only considers children's exposures to CCA-treated playsets. Exposures of arsenic from CCA-treated wood can occur throughout a lifetime from many different structures (decks, marinas, playsets, picnic tables, etc.) due to the prevalence of CCA within residential, commercial, and industrial settings. Potential risks also continue long after the product has been taken out of service for disposal.

In our article, we argue the need for a focus on integrated studies in which human exposure is assessed using measures of As excretion in urine, hair, and nails. Indeed, a good case can be made that the scope of studies should be expanded to consider lifetime exposures and risks associated with CCA-treated products throughout the product lifecycle. The writers of this "Letter to the Editor" have expressed an opinion that we have overestimated the magnitude of As exposure from CCA-treated wood, but they bring no additional facts. Such opinion could, however, be taken within the regulatory realm to argue that no further research on children's arsenic exposure from CCA-treated wood is necessary. In neither case does this letter contribute to science or the protection of public health.

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