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Arsenic on Children's Hands after Playing in Playgrounds

We commend Kwon et al. (2004) for their very interesting study of arsenic on the hands of children in contact with chromated copper arsenate (CCA)-treated wood structures and soil after playing in playgrounds. We would like to comment on some of their cited references and discuss the implications of the reported arsenic concentrations found in their sand/soil samples.

Kwon et al. (2004) accurately stated that previous studies on CCA-treated wood have mostly examined soil and sand samples from playgrounds but have not assessed the amounts of arsenic found on the hands of children playing on the CCA-treated wood structures. However, to justify their statement, they inappropriately cited our previous work that examined arsenic speciation in various synthetic soil samples artificially contaminated with CCA in the laboratory (Balasoiu et al. 2001) and arsenic concentrations in field-collected soils near CCA-treated utility poles (Zagury et al. 2003). Therefore, it is not surprising that the levels of arsenic on the hands of children playing on wood-treated structures were not evaluated during the aforementioned studies.

In our field study, arsenic concentrations found in surface soil collected immediately adjacent to CCA-treated utility poles ranged between 153 ± 49 and 410 ± 150 mg/kg (mean ± SD), although they dropped to between 6.3 ± 1.5 and 61 ± 60 mg/kg at 0.1 m from the pole. Therefore, arsenic concentrations found immediately near CCA-treated utility poles are much higher than the values reported by Kwon et al. (2004) in their study using soil/sand samples collected from playgrounds. Moreover, in a recent study conducted near 217 CCA-treated wood play structures in Toronto, Canada (Ursitti et al. 2004), mean arsenic concentrations in soil samples taken from beneath elevated platforms (mean 20.3; range 12.4–47.5 mg/kg) were significantly greater than background soil samples (mean 2.4; range 0.5–13 mg/kg) and soil from within 1 m (mean 2.1; range 0.5–10 mg/kg). Composite soil samples exceeded the Canadian federal soil guideline (Canadian Council of Ministers of the Environment) of 12 mg/kg at 32 CCA-treated wood play structures. Furthermore, Stilwell and Gorny (1997) reported a mean arsenic concentration of 76 mg/kg in soils collected beneath seven decks built with CCA-treated lumber, compared to a mean concentration of 3.7 mg/kg in control soils (collected at a minimum distance of 5 m from the decks). All these studies suggest that the sampling protocol is crucial in order to obtain a representative pattern of the soil contamination and that the closer the sample is to the CCA-treated wood structure, the higher the arsenic concentration is expected to be.

Therefore, when Kwon et al. (2004) stated that "it is important to point out to the general public that arsenic is naturally present in the soil and sand regardless of whether the playgrounds contain CCA-treated wood structures," they do not adequately refer to previously published studies; therefore, their statement might be misleading. We agree with the authors that there is a natural background concentration of arsenic in soils near CCA-treated utility poles (0.5–7.3 mg/kg) (Zagury et al. 2003; Chirenje et al. 2003), near CCA-treated decks (0.4–2.2 mg/kg) (Stilwell and Gorny 1997; Chirenje et al. 2003), and near CCA-treated play structures (0.5–13 mg/kg) (Ursitti et al. 2004). However, published studies all conclude that arsenic concentrations in soil samples taken from beneath or immediately adjacent (within 01–0.3 m) to CCA-treated wood structures are significantly greater than background arsenic concentrations.

Nevertheless, as the data of Kwon et al. (2004) show, the amount of total arsenic from hand washing suggests that direct contact with CCA-treated wood is a major contributor to arsenic concentration on children's hands. Therefore, oral ingestion of dislodgeable arsenic via hand-to-mouth contact appears to be an important exposure pathway, and we agree with the authors when they recommend that children wash their hands after playing in CCA-treated playgrounds. However, potential ingestion of arsenic from soil under CCA-treated structures should not be neglected based on the unusually low arsenic concentrations found in the soil/sand samples in their study. The importance of this additional exposure pathway can be fully assessed when accurate estimates become available for *a*) soil physicochemical properties and contamination pattern beneath CCA-treated structures, *b*) children's daily soil intake values, and *c*) relative oral bioavailability of arsenic in CCA-contaminated soils.

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Arsenic on Children's Hands: Le et al. Respond

We appreciate the comments of Zagury and Pouschat and their support of our overall conclusions presented in our article (Kwon et al. 2004). In response to their thoughtful comments, we would like to offer the following clarifications.

In the introduction of our article (Kwon et al. 2004), we cited Balasoiu et al. (2001), Zagury et al. (2003), and others (Stilwell and Gorny 1997; Townsend et al. 2003), who examined arsenic in soil and sand samples from the field or from the laboratory. These references provide the readers with useful background information on the sources and levels of potential arsenic exposure. Examining the distribution, partitioning, and concentration of arsenic in the environmental media (e.g., soil, sand, water, and wood surface) appeared to be the primary objectives of these studies. Arsenic levels had not been directly measured on the hands of children after contact with either chromated copper arsenate (CCA)-treated wood or soil in playgrounds until our study (Kwon et al. 2004).

Because the primary objective of our study was to determine the amount of arsenic on the hands of children after playing in playgrounds, we did not focus on the characterization of arsenic in the soil. Although we determined the levels of arsenic

in the composite soil samples from the playgrounds, a detailed characterization of the spatial distribution of arsenic was outside the scope of our study. We agree that the concentration of arsenic in the soil samples varies greatly with the sampling protocols and the location of the samples with respect to the CCA-treated wood structures (Chirenje et al. 2003; Stilwell and Gorny 1997; Zagury et al. 2003; Ursitti et al. 2004). Our composite soil samples could not provide any information on the spatial distribution of arsenic concentration in soil samples collected from the playgrounds. These composite samples were obtained from areas under decks and away from any wood structures. We did not collect soil/sand samples from areas immediately adjacent to the CCA-treated wood. Further studies to understand the distribution of arsenic in playgrounds would benefit from extensive collection and analysis of soil samples from different locations in the playgrounds.

We clearly stated that "children playing in playgrounds constructed with CCA-treated wood have approximately five times more arsenic on their hands than do those playing in playgrounds that do not have CCA-treated wood structures." We also feel that "it is important to point out to the general public that arsenic is naturally present in the soil and sand regardless whether the playgrounds contain CCA-treated wood structures." During our study, we found that many of the parents of the participating children did not know that arsenic was naturally present in the environment, albeit with varying concentrations. They thought that if there was any arsenic, it must have been added to the environment by someone. Conversely, if there was no added "synthetic" arsenic, they did not consider arsenic as a potential health concern. This attitude toward toxic substances (natural versus synthetic) can be counterproductive in the effort to achieve the goal of protecting public health. Properly informing the public that arsenic is naturally present in the soil helps people to understand that it is important for children to wash their hands after playing, regardless of whether the playgrounds contain CCA-treated wood structures. The hand–mouth activities of young children can result in the ingestion of arsenic that may be adsorbed on their hands. Children should wash their hands after playing to reduce their potential exposure to arsenic.

We agree with Zagury and Pouschat that "potential ingestion of arsenic from soil under CCA-treated structures should not be neglected." All efforts need to be made to minimize children's exposure to the toxic species of arsenic.

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Invoking the Precautionary Principle

The article on the precautionary principle, risk perception, and assessment by Wiedemann and Schütz (2005) deserves praise and careful consideration because of the growing awareness that certain human activities could potentially seriously harm human and environmental health.

The precautionary principle (United Nations Conference on Environment and Development 1992) holds forth that a point can presumably be reached when human well-being and environmental health are put at risk by a large-scale human activity or man-made system over which humans have control. At such a point the problem could be identified, a course charted, and precautionary actions taken to ameliorate or prevent a potential threat to human and environmental health on behalf of current and future generations.

Despite the incontrovertible element of uncertainty and other limitations of scientific methods, it is implicitly assumed that science plays the vital role of providing humanity with the best knowledge of how the world works and of the placement of humankind in the natural order of living things. To the extent scientific methods are incorporated and used to inform both the proclamation of a problem and the implementation of its remedy, the precautionary principle affords humanity a mechanism to focus attention and to examine data on potential impacts of human activities and systems upon the natural world. With such attentiveness and knowledge, humans become able to make choices and to engage in the regulation of behaviors that are advantageous rather than detrimental to human and environmental health. As a mechanism of science, the precautionary principle becomes a useful tool in raising awareness and determining aspects of human culture that are and are not sustainable.

Absolute global human population numbers (Hopfenberg 2003; Hopfenberg and Pimentel 2001), increasing human consumption worldwide (Imhoff et al. 2004), and the seemingly limitless expansion of the world's predominant human economy (Czech and Daly 2004; Meritt 2001) point to the existence of a rapidly spreading culture that could be characterized by its proclivity for unlimited growth—growth that increasingly outruns humanity's capabilities to anticipate and address the potential for devastating consequences of growth. Given the current scale and rate of this growth relative to the small, finite, noticeably fragile planet we inhabit, it could be that this cultural predisposition for increasing growth is patently unsustainable and, moreover, could give rise to the potential for recognizable, worst-case scenarios. Global warming; diminishing nonrenewable energy resources; destruction of the ozone layer; biodiversity loss; acid rain; deforestation; solid waste disposal; pollution of the air, water, and land; and desertification are regularly referenced in this context.

Not unexpectedly, the evolution of science gives rise to new approaches for examining large-scale human activities such as human propagation and human consumption and man-made constructions such as the prevailing economic system. Although relatively new, "top-down" research focuses on data acquisition and analysis regarding certain human behaviors and global human systems. This development complements the "bottom-up" research with which scientists are so familiar (Cairns 2003). As the adage goes, scientists have had difficulty "seeing the forest for the trees" because traditional

scientific methods focus primarily on parts of a large system, not on the large system itself.

Another dimension of this change in focus is the development of "joining edge" research, in which leading ideas and best practices from multiple disciplines are brought together in a collaborative effort to examine large, complex systems. This approach complements the more familiar pursuit of progressively narrowing "cutting-edge" research of components of a whole system (Cairns 2003; Kriebel et al. 2001).

Perhaps, scientific data to advance human understanding about why global-scale human activities and systems are sustainable or unsustainable could be vital to protecting humanity from endangerment, biodiversity from extinction, and Earth from irreversible degradation, even in these early years of the 21st century.

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The Precautionary Principle: Schütz and Wiedemann Respond

Salmony rightfully points to various examples for potentially harmful effects of human activities that call for strategies to cope with ambiguous risks. The precautionary principle is seen by many as the answer to this problem. We agree that the precautionary principle formulates a sensible maxim for coping with uncertainty. However, we are less confident that it provides a feasible solution.

There are at least two problems with the application of the precautionary principle. The first, which we addressed in our article (Wiedemann and Schütz 2005), is that applying the precautionary principle might have unintended and unwelcome effects—in our case, increase public concern about radio frequency electromagnetic fields (RF EMFs). At least when precautionary measures are implemented to reassure the public, this runs counter to the original intention.

The second, perhaps more serious, problem of applying the precautionary principle is its "extreme variability in interpretation" (Foster et al. 2000). The decision to apply the precautionary principle depends on three factors: the type of evidence considered as appropriate for decision making, the amount of evidence, and the reference point for triggering the precautionary principle (how much evidence is enough?).

Roughly, three different types of evidence can be distinguished: scientific data, observations of health professionals, and personal experiences of lay people. The problematic issue is that some proponents of the precautionary principle consider scientific information, although necessary and important, not to be the exclusive basis for decision making. However, expanding the data basis beyond scientific information may result in conflicting claims about the significance of the various types of evidence. And there are no efficient procedures to resolve these conflicts.

The key question is whether there is enough scientific evidence to show that the risk potential might be real. Although ignorance and uncertainty exist, at least some evidence is required for triggering precautionary measures (World Health Organization 2000). That is, a hazard must be identified, and some understanding is needed about the conditions under which it is likely to occur. Therefore, a careful assessment of the available evidence is critical.

At present, there is no clear definition of the reference point for the decision to invoke precautionary measures. Without this clarification, any decision on applying the precautionary principle remains arbitrary.

Obviously, the answer to this question cannot be given by science alone (although science can provide important information). It will require value judgments, and it is ultimately a political decision. However, even if policy makers are bold enough to vote for precaution, the question remains about what to do.

Any well-founded decision about precautionary measures will also require some knowledge about the effectiveness of the precautionary measures that are to be taken. Unfortunately, in those situations for which the precautionary principle is intended, this knowledge is usually lacking.

The decision to implement precautionary measures needs to be justified by more than pointing at the possibility that a risk may exist. It needs evidence, and above all, a structured and transparent procedure for evaluating this evidence. For this, a solution is pending.

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Peripheral Arterial Disease and Metals in Urine and Blood

Navas-Acien et al. (2005) recently analyzed the data from the 1999–2000 National Health and Nutrition Examination Survey (NHANES). They suggested that blood lead and blood and urinary cadmium, at levels well below safety standards, were associated with an increased prevalence of peripheral arterial disease (PAD) and that cadmium might partly mediate the detrimental arterial effects of smoking. The authors recognized that their findings needed confirmation and support from mechanistic studies. In line with their suggestion, we analyzed data from 428 participants in the Flemish Study on Environment, Genes, and Health Outcomes (Staessen et al. 1994). As described by Navas-Acien et al. (2005), we included only subjects who were at least 40 years of age and we defined PAD as an ankle brachial index of > 0.9 in at least one leg. Blood lead and blood and urinary cadmium were measured by atomic absorption spectrometry. The geometric mean concentrations were

0.43 nmol/L [5th–95th percentile interval (PI), 0.19–1.03] for blood lead and 11.6 nmol/L (PI, 3.6–31.1) for blood cadmium. The urinary cadmium excretion averaged 11.6 nmol/24 hr (PI, 3.8–35.5).

We adjusted for demographic and cardiovascular risk factors. For blood lead and cadmium, the odds ratios of PAD comparing quartiles 2–4 with the lowest quartile were in line with those of Navas-Acien et al. (2005). However, for the 24-hr urinary cadmium excretion the *p*-value for trend was only 0.72. Urinary cadmium is a more precise biomarker of exposure than blood cadmium, because urinary cadmium reflects lifetime exposure and blood cadmium reflects more recent exposure. Navas-Acien et al. (2005) measured only metal concentrations in spot urine samples (Navas-Acien et al. 2005), whereas we measured the 24-hr excretion of cadmium. We could not demonstrate any relation between cardiovascular disease or the incidence of hypertension in relation to environmental exposure to lead and cadmium (Staessen et al. 2000). We therefore concur with their conclusion that the role of cadmium in the pathogenesis of atherosclerosis needs further research. However, not only are mechanistic studies required but also population studies, for example, that relate pulse wave velocity to biomarkers of cadmium exposure. We currently have similar experiments in progress.

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Peripheral Arterial Disease and Metals: Navas-Acien et al. Respond

We thank Plusquin et al. for their interest in our analysis of the 1999–2000 National Health and Nutrition Examination Survey (NHANES) data on the association of lead, cadmium, and other metals with the prevalence of peripheral arterial disease (PAD) (Navas-Acien et al. 2004; Navas-Acien et al. 2005). After analyzing data from 428 participants in the Flemish Study on Environment, Genes, and Health Outcomes, Plusquin et al. confirmed our findings of a positive and strong association between blood lead or cadmium with PAD. They also reported a nonstatistically significant trend for the association of 24-hr urinary cadmium with the prevalence of PAD; however, in the absence of information on relevant methodologic details, such as the number of subjects with PAD, this nonsignificant result is difficult to interpret. Overall, the findings of Plusquin et al. add to the growing concern about the cardiovascular effects of environmental exposure to low concentrations of metals (Weinhold 2004).

On a more general note, both the NHANES study, which was the basis of our analyses, and the Flemish Study on Environment, Genes, and Health Outcomes (Plusquin et al.) used cross-sectional designs. These designs have important limitations for assessing the causal effects of exposures on cardiovascular risk, even when the outcome is a subclinical marker such as PAD defined using the ankle-braquial blood pressure index. Some limitations of cross-sectional designs include survivor effects for severe cases of cardiovascular disease, potential changes in biomarker levels associated with disease development or with cardiovascular medications, and changes in exposure patterns associated with the development of disease. Although cross-sectional studies are important first steps in evaluating the cardiovascular effects of environmental exposures, prospective studies ultimately will provide more rigorous tests of causality. For cadmium, there are no prospective studies using biomarkers of exposure and adequate measures of cardiovascular disease incidence and mortality, whereas for lead the prospective evidence is limited (Lustberg and Silbergeld 2002; Moller and Kristensen 1992; Pocock et al. 1988). Because of the frequent environmental exposure to lead, cadmium, and other metals; the existence of a biological basis for cardiovascular effects of metals; and the current controversies on safety standards, performing high quality prospective studies with appropriate biomarkers of exposure and standardized cardiovascular outcome definitions is a public health research priority.

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Erratum

The publication date for an article cited by Do et al. [Chlorination Disinfection By-products and Pancreatic Cancer Risk. Environ Health Perspect 113:418–424 (2005)] was incorrectly given as 2004. The correct reference is as follows:

Wilkins JR III, Comstock GW. 1981. Source of drinking water at home and site-specific cancer incidence in Washington County, Maryland. Am J Epidemiol 114:178–190.