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Coal Tar and Paving Products

The National Institute for Occupational Safety and Health (NIOSH) has a partnership relationship with the asphalt paving and roofing industries and their associated unions. Our partners saw in the *EHP* Focus article "Paving Paradise: The Peril of Impervious Surfaces" (Frazer 2005) the statement on page A459: "Asphalt is one concern, as it contains coal tar pitch, a recognized human carcinogen" Our partners asked us if we could help them address this statement.

By definition, asphalt is a petroleum product and contains no coal tar. However, some pavement-repair products and sealants may contain coal tar. NIOSH did not find any evidence of coal tar in U.S. asphalt in our hazard review *Health Effects of Occupational Exposure to Asphalt* (NIOSH 2000).

The author declares he has no competing financial interests.

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Editor's note: The following erratum was published in the January 2006 issue (Environ Health Perspect 114:A21):

EHP regrets the incorrect and unintentional inference in "Paving Paradise: The Peril of Impervious Surfaces" [*Environ Health Perspect* 113:A456–A462 (2005)] that coal tar pitch is used in the actual hot-mix asphalt used to pave roads. Coal tar pitch is instead used in many sealcoat formulations used atop asphalt pavement. Findings published in the 1 August 2005 issue of *Environmental Science & Technology* suggest, in fact, that coal tar-based parking lot sealant may be a major contributor to stream loads of polycyclic aromatic hydrocarbons, including many known carcinogens.

Organic Diets and Children's Health

In their article "Organic Diets Significantly Lower Children's Dietary Exposure to Organophosphorus Pesticides," Lu et al. (2006) used language that is likely to be misused by organic food marketers to promote high-priced foods and could discourage lower-income parents from providing their children with a diet rich in fruits and vegetables.

Regarding their findings that children's median urinary concentrations of two organophosphorus (OP) pesticides dropped to nondetectable levels within 24–48 hr after switching to an organic diet, Lu et al. (2006) state in the "Abstract" that "an organic diet provides a dramatic and immediate protective effect against exposures" and that these results provide "evidence of the effectiveness of this intervention." Later in their article, they admit that they "did not collect health outcome data in this study," but they claim that

It is intuitive to assume that children whose diets consist of organic food items would have a lower probability of neurologic health risks, a common toxicologic mechanism of the OP pesticide class.

This statement, in particular, seems tailor-made to mislead consumers into believing that organic foods will protect against actual neurologic health risks. A previous article by one of the coauthors presenting similar findings also contains potentially misleading wording (Curl et al. 2003). In the "Abstract," Curl et al. (2003) stated that

Consumption of organic fruits, vegetables, and juice can reduce children's exposure levels from above to below the U.S. Environmental Protection Agency's [EPA] current guidelines, thereby shifting exposures from a range of uncertain risk to a range of negligible risk.

Collectively, the wording of both papers strongly implies to consumers and non-specialists that consuming organic foods reduces likely or actual harm caused by residues of OP pesticides. However, evidence of harm from exposure to the low levels of OP pesticide residues in food is completely lacking in children or adults. Although there is some evidence from animal experiments that *in utero* exposures to OP pesticides at high enough doses can cause neurodevelopmental effects (Eskenazi et al. 1999), the doses at which such effects were seen were at least three orders

of magnitude higher than those consumed as food residues by the children in these two studies (Curl et al. 2003; Lu et al. 2006).

Recent measurements of OP metabolites in the U.S. population by two of the authors of the Lu et al. study (Barr et al. 2005) allowed estimations of doses at the 95th and 50th percentiles of the population for chlorpyrifos (the OP exposure closest to the U.S. EPA reference dose). Barr et al. (2005) estimated that at the 95th percentile, children still consumed less than one-half of the U.S. EPA's chronic population-adjusted dose (cPAD), confirming the exposure estimates used in the risk assessment of the Health Effects Division at the U.S. EPA in 2000 (Barr et al. 2005). The cPAD for chlorpyrifos is 1/1,000th of the no observable adverse effect level (NOAEL) in dogs and rats. Thus, children in the 95th percentile consumed < 1/2,000 of the NOAEL, and the median exposure in children was 1/5,000 of the NOAEL.

If it is appropriate to intuitively assume that organic foods pose a lower probability of risk to children, is it not also appropriate to clearly state that all of the risks discussed in these articles are negligible, given that they are tiny fractions of the NOAEL in the most sensitive animal species? It seems that the language chosen by these authors was not appropriate. Already, organic food marketing interests are using these articles as "proof" that organic food is better for you (Organic Consumers Association 2003); Lu et al.'s article is even posted on the Organic Consumers Association website (Organic Consumers Association 2005).

In the future, those of us who communicate with the public on food safety issues should choose our words carefully, not make claims that go beyond the scope of the research, and take the time to accurately place the level of risks being discussed within the context of what is known from animal studies.

The author is employed by the Hudson Institute, a nonprofit organization that has accepted donations from several chemical and pesticide manufacturers over the years, including makers of OP pesticides.

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Organic Diets: Lu et al. Respond

Avery is concerned that the language used in our recent article (Lu et al. 2005), as well as in an earlier article (Curl et al. 2003), may be used to mislead the public regarding the relative safety of organic foods compared with foods derived from crops treated with pesticides.

We agree that communication of scientific information in general, and health risk information in particular, requires a careful choice of words. However, Avery's analysis misconstrues the current scientific debate regarding children's exposure to pesticides and misrepresents our work, thereby contributing to the public misunderstanding of this important issue.

In fact, we did choose our words carefully, and they reflect the essential findings of our studies. In regard to our earlier publication (Curl et al. 2003), we provided a detailed dose estimation to support our conclusion that consumption of organic fruits, vegetables, and juices in the study population would shift exposure from a range of uncertain risk to a range of negligible risk. In regard to the more recent study (Lu et al. 2005), our data clearly support the conclusion that organophosphorus (OP) pesticide exposures are dramatically reduced when organic foods are substituted for conventional foods. Our statement that children who consume organic foods would likely have a lower probability of neurologic health risks is consistent with current understandings of dose–response relationships. In other words, how could we argue that children with OP pesticide exposures have the same neurologic health risks as children whose urine contain no OP pesticide metabolites?

The assessment of health risks associated with neurotoxic chemicals such as OP pesticides is a complex analysis that includes substantial uncertainty. A child may be exposed to dozens of OP pesticides simultaneously

through the diet, as well as through use of these pesticides around the home or in schools. The Food Quality Protection Act (1996) requires the U.S. Environmental Protection Agency to evaluate both children's aggregate exposure (multiple exposure pathways for a single pesticide) and cumulative risk (potential health effects from exposure to multiple compounds that have a common mechanism of toxicity). Thus, current scientific investigations have focused on the relative contributions of specific exposure pathways and have attempted to examine exposure to multiple compounds. Our recent articles provide new information regarding the dietary exposure pathway for several OP pesticides.

Avery's criticism of our work by focusing on a single OP pesticide ignores the central thrust of the Food Quality Protection Act (1996), as well as the scientific advances that have taken place over the past 10 years. We share Avery's concern with the judicious use of language in regard to public communication of pesticide health risks; all of us—including Avery—should follow this advice.

The authors declare they have no competing financial interests.

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Translocation of Ultrafine Particles

We read with great interest the article by Geiser et al. (2005) on the mechanism of

translocation of ultrafine particles (UFPs) across cellular membranes *in vivo* in rats following inhalation and *in vitro* using porcine pulmonary macrophages and human red blood cells.

We are delighted to see this study that vindicates our hypothesis that translocation of UFPs is a possible pathway for the cardiovascular effects of particulate air pollution. A few years ago we reported extrapulmonary translocation of UFPs after intratracheal instillation in hamsters (Nemmar et al. 2001) and after inhalation in healthy human volunteers (Nemmar et al. 2002a), suggesting an alternative and/or a complementary explanation for the extrapulmonary effects of particles.

In their study, Geiser et al. (2005) very elegantly provided novel morphologic data showing the occurrence of translocation of UFPs, and they also reported—for the first time—that this translocation did not occur by endocytic processes but rather by diffusion or adhesive interactions. However, we noted with some surprise that the authors cited Brown et al. (2002) when referring to previous studies on the occurrence of UFP translocation. Indeed, Brown et al. (2002) studied the deposition and clearance of an ultrafine (60 nm) technetium-99m-labeled aerosol in human volunteers after 2 hr, and found no significant radioactivity in the liver ($1.3 \pm 1.2\%$). This activity was attributed to scatter from the lung and/or overlap of lung parenchyma in the liver. Consequently, Brown et al. (2002) excluded the occurrence of translocation and, although they did not measure radioactivity in blood, they challenged our conclusion that UFPs (5–10 nm) could pass from the lungs into blood and extrapulmonary organs (Nemmar et al. 2002a). Therefore, for the sake of accuracy, Geiser et al. (2005) should have referred to our study (Nemmar et al. 2002a) rather than that of Brown et al. (2002).

Geiser et al. (2005) provided micrographs of fluorescent polystyrene particles taken up by macrophages (Figure 3) or red blood cells (Figure 4). It is not clear whether these polystyrene particles contained surface charges, for example, carboxylate-modified (negatively charged) or amine-modified (positively charged), although Geiser et al. (2005) briefly discussed the possible effect of surface charge. This aspect is important; we (Nemmar et al. 2002b) and others (Silva et al. 2005) have reported that hemostasis may be affected by the intravenous or intratracheal administration of UFPs and also established that this phenomenon is dependent on the surface properties of the particles. Thus, only positively charged amine-modified particles led to a marked increase in prothrombotic tendency, which

we showed to result, at least in part, from platelet activation

In conclusion, although the article by Geiser et al. (2005) adds a significant amount of information to the literature related to the extrapulmonary effect of inhaled particles, this issue needs to be clarified in more detail. Therefore, we look forward to this group and others providing more detailed quantification of the proportion of inhaled UFPs that can be found in extrapulmonary organs.

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Ultrafine Particles: Geiser et al. Respond

Nemmar et al. were surprised that we did not cite their study in our article (Geiser et al. 2005) when we referenced state-of-the-art experiments about the translocation of ultrafine particles into secondary organs. We did not cite their human study (Nemmar et al. 2002) because in Figure 2 of their article, they presented clear evidence that a major fraction of the radiolabeled technetium-99 (Tc-99m) came off the Technegas particles. Thus, for methodologic reasons, the fraction of translocated particles could not be determined adequately and was certainly overestimated by Nemmar et al. (2002). This was recently discussed by Kreyling et al. (2004). To briefly illustrate this, we have included Figure 1. Figure 1A

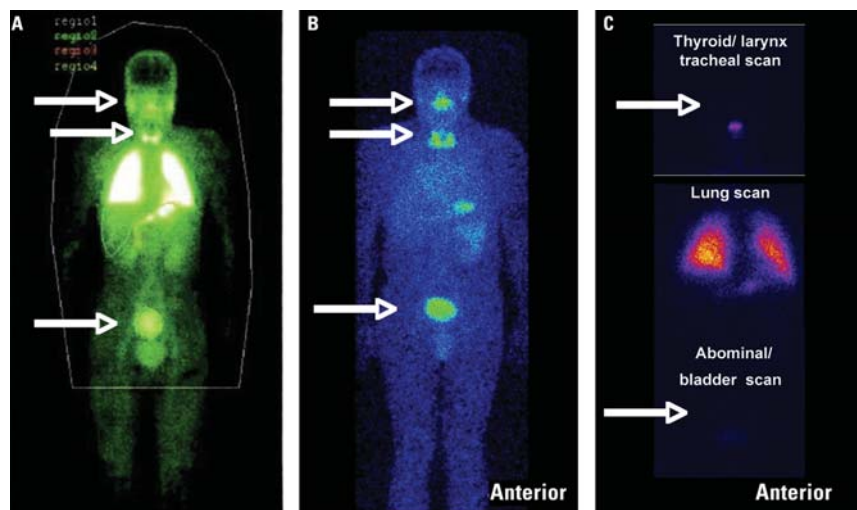


Figure 1. (A) Gamma camera image after the inhalation of Tc-99m radiolabeled ultrafine carbon particles not controlled for leaching. Reproduced from Nemmar et al. (2002) with permission from Lippincott Williams & Wilkins. (B) Gamma camera image after the inhalation of soluble Tc-99m pertechnetate. Reproduced from Kreyling et al. (in press) with permission from *The Journal of Aerosol Medicine*. (C) Gamma camera images after the inhalation of nonleaching Tc-99m radiolabeled ultrafine carbon particles.

shows the original whole-body scintigram published by Nemmar et al. (2002) in which the salivary glands, the thyroid gland, and the urinary bladder are clearly visible, demonstrating that they contain large fractions of the Tc-99m radiolabel. This and the Tc-99m activity in the soft tissue, which shows the contour of the whole body, are clear indications of nonparticulate Tc-99m in the form of pertechnetate. Pertechnetate typically accumulates in these organs, as can be inferred from the Figure 1B, where the same pattern of radiolabel was detected after inhalation of soluble Tc-99m pertechnetate.

In the case of inhalation of nonleaching Tc-99m radiolabeled ultrafine carbon particles (Figure 1C), no activity is detectable in these organs or in the soft tissue. Figure 1C shows three images taken from the head (little larynx retention), the thorax (main carbon particle retention in lungs), and the lower abdomen, with a rather faint image of the urinary bladder. A similar pattern has been reported by Brown et al. (2002).

In addition, Nemmar et al. are interested in the surface charges of the particles we used for the *in vitro* studies, because surface charges are likely to be important determinants for the translocation of ultrafine particles as well as for their biologic effects. The polystyrene particles we used for the studies with the macrophages and erythrocytes were either uncharged, amino-modified, or carboxylate-modified (Rothen-Rutishauser B, Gehr P, Schürch S, unpublished data). The surface charges of the gold and titanium particles are not known. We found nonphagocytic uptake of ultrafine particles of all the different materials and surface charges by

macrophages and erythrocytes. However, because the aim of our study was not to investigate the effects of surface charges on cellular uptake, we did not measure the actual surface charges of the particles or estimate the total number of particles within cells to quantify particle uptake. We certainly agree with Nemmar et al. on the importance of surface charges for particle-cell interaction, and we hope that we will soon find more literature published on this aspect.

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Evaluating Beryllium Exposure Data

We read with great interest “Chronic Beryllium Disease and Sensitization at a Beryllium Processing Facility” (Rosenman et al. 2005). We wish to offer some observations that will broaden the context in which this article is understood.

I agree with the statement by Rosenman et al. (2005) that a limitation of the study is the uncertainty of the exposure estimates. In addition, many statements appear to be unsupported by the data provided. For example, the statement that “most time-weighted averages were below the [Occupational Safety and Health Administration] OSHA (2005) standard of $2 \mu\text{g}/\text{m}^3$ ” (Rosenman et al. 2005) is unsupported by the data in the tables. Table 11 demonstrates that > 91% of the cohort had average daily weighted average (DWA) exposures > $2 \mu\text{g}/\text{m}^3$. Table 12 presents only the highest exposures and shows 56% of the cohort members having exposures > $2 \mu\text{g}/\text{m}^3$ and all but two cohort members exposed to > $0.2 \mu\text{g}/\text{m}^3$. Rosenman et al. (2005) did not explain how the average exposures of the cohort exceed $2 \mu\text{g}/\text{m}^3$ at a rate greater than the peak exposures. This same mysterious artifact of average exposures exceeding peak exposures is also present in Tables 9 and 10. The exposure-estimating process used by these authors could have introduced an erroneous bias in the data set, which causes me to question the “Discussion” and the conclusions drawn from the data.

One point, unstated by Rosenman et al. (2005), is that the DWA represented the daily exposure based on data averaged over 3 months. This is not the same as taking a single 8-hr air sample on 1 day because the 3-month averaging of the task data does not reveal the daily up and down variation in the individual task sample results. The National Institute for Occupational Safety and Health (NIOSH) has found DWAs not comparable to the lapel sampling

method used by OSHA to determine compliance with its permissible exposure limit (PEL) or other 8-hr occupational exposure limits (OELs) (Donaldson and Stringer 1976). In addition, the average exposures presented by Rosenman et al. (2005) in their tables are the result of a second averaging by whole year and finally a third averaging by years of work. Such triple averaging further reduces the standard deviation in the data set, which results in a failure to identify the true high and low ranges of daily exposure. The American Industrial Hygiene Association (AIHA) exposure assessment guide (Mulhausen and Damiano 1998) cautions against ignoring air sample results that comprise the upper tail of an individual’s exposure distribution, especially when comparing it to PELs and OELs.

The flame spectroscopy method of chemical analysis of beryllium used by Rosenman et al. (2005) during the data-collection period of this study had a detection limit of $0.1 \mu\text{g}/\text{filter}$ that translates to $< 0.1 \mu\text{g}/\text{m}^3$ for any lower value. Therefore, Rosenman et al. (2005) cannot make any statements about exposures lower than this value.

Rosenman et al.’s (2005) description of missing and estimated data, the illogical peak versus average data results, the triple averaged DWA exposure estimates, and the limit of analytical detection of the sampling method all combine to make it likely that virtually all members of the study population experienced multiple days of exposure > $2 \mu\text{g}/\text{m}^3$, and hence the study cannot sustain conclusions about the degree of risk associated with lower levels of exposure. This conclusion is supported by the observation that the rates of chronic beryllium disease (CBD) and sensitization were constant across all the categories of exposure used by Rosenman et al. (2005).

Rosenman et al. (2005) made no recommendations regarding how to protect beryllium workers. We cannot change the past, but we can learn from it and change the future. There are two successful models of beryllium safety: one demonstrates effectiveness in preventing clinical CBD (Johnson et al. 2001), and one demonstrates prevention of beryllium sensitization (BeS) using the beryllium blood lymphocyte proliferation test as an index of BeS (Cummings K, unpublished data). Common to both models are *a*) organization and cleanliness of the workplace; *b*) control of the upper range of air level exposure using engineering and respiratory protection; *c*) control of beryllium migration from the work process to the worker, the work area, and outside the facility; *d*) detailed training of workers; and *e*) management and worker commitment to

effective program implementation. In the facility studied by Rosenman et al. (2005), it is not clear that any of the above elements of a beryllium safety management plan were consistently accomplished. Although this is understandable, given prevalent scientific opinion at the time, going forward we should make every effort to effectively disseminate these demonstrated beryllium safety principles to the companies and workers using beryllium.

The author is employed by Brush Wellman Inc., a manufacturer of beryllium-containing products.

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Beryllium Exposure Data: Rosenman et al. Respond

We thank Kolanz for his careful reading of our article (Rosenman et al. 2005). An erratum correcting the problem he noted appears on page A214.

How do these corrected numbers change our results? More chronic beryllium disease (CBD) and sensitization occurred with exposure below the Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) of $2 \mu\text{g}/\text{m}^3$ (OSHA 2005) than we previously reported, but no CBD or sensitization was found below the Department of Energy (DOE) guidelines of $0.2 \mu\text{g}/\text{m}^3$ (DOE 1999) or the even more protective level proposed by the American Conference of Governmental Industrial Hygienists (ACGIH 2005) of $0.02 \mu\text{g}/\text{m}^3$. There was an insufficient number of individuals—only three, and they were all normal—to assess the safety of the DOE guidelines or proposed ACGIH level. Our previously reported findings regarding chemical and physical form—that sensitized individuals compared with individuals with CBD had higher exposure to beryllium in a

soluble form and to fumes of beryllium (Rosenman et al. 2005)—remain unchanged. However, the mean peak exposure for the sensitized group in our Table 8, the mean nonsoluble exposure for the sensitized group in Table 9, and the mean dust exposure for the sensitized group and mean fume exposure for the CBD group in Table 10 were no longer significantly different, whereas the peak fume exposure for the CBD group was now significantly different in Table 10.

Having acknowledged and corrected the error pointed out by Kolan, we take strong exception to his statement that “the study cannot sustain conclusions about the degree of risk associated with lower levels of exposure,” by which he means less than the OSHA PEL of 2 $\mu\text{g}/\text{m}^3$ (OSHA 2005).

One of Kolan's criticisms of our article (Rosenman et al. 2005) is that the daily weighted average (DWA) represented the daily exposure based on data averaged over 3 months. Breslin and Harris (1958) conducted a time study, which was updated as activities and location changed for each job title. During a 3-month period, three or more samples were collected and standardized to represent the general area where each person worked, and in some cases the breathing zone during work activities. The arithmetic average of the samples for each location/type was then calculated and weighted by time (Breslin and Harris 1958). Use of these summed weighted values, divided by the shift duration, is consistent with the American Industrial Hygiene

Association (AIHA) exposure assessment guidance (Mulhausen and Damiano 1998) cited by Kolan.

We agree with Kolan's comment about the use of the flame spectroscopy method of chemical analysis and its general limit of detection of 0.1 $\mu\text{g}/\text{m}^3$. In the job exposure matrix and task exposure matrix developed to support this project (Chen 2001), no exposure estimate was < 0.1 $\mu\text{g}/\text{m}^3$, so this issue would have no effect on our results.

Kolan also takes exception with our estimates of mean exposures, stating that these values were derived by triple averaging. We derived the mean exposures as follows: for a given worker in a given job in a given year, we multiplied the number of days worked in that year for that worker by our

ERRATUM

In the article by Rosenman et al. [Environ Health Perspect 113:1366–1372 (2005)], the authors noted errors in Tables 8–11, which resulted from a programming error. Because of this error, the overall mean daily weighted averages (DWAs) were greater than they should have been. The corrected tables are shown below.

Table 8. Development of definite/probable CBD and sensitization by average cumulative, average mean, and peak exposure (\pm SE).

Disease outcome	No. of individuals	Mean cumulative exposure ($\mu\text{g}\text{-year}/\text{m}^3$)	Mean exposure (days)	Mean average exposure (mg/m^3)	Mean peak exposure (mg/m^3)
Definite/Probable CBD	40	181 \pm 29	3483 \pm 50	1.6 \pm 0.21	3.1 \pm 0.36
Sensitization	37	100 \pm 23 ^a	1934 \pm 5 ^b	1.6 \pm 0.16	2.9 \pm 0.4
Normal	377	209 \pm 16	3359 \pm 76	1.6 \pm 0.06	3.3 \pm 0.13

^a $p = 0.03$ for sensitization vs. definite/probable and $p = 0.0003$ for sensitization vs. normal. ^b $p = 0.047$ for sensitization vs. definite/probable and $p = 0.02$ for sensitization vs. normal.

Table 9. Development of definite/probable CBD and sensitization by chemical form of beryllium, mixed, nonsoluble, and soluble: mean cumulative, mean average, and mean peak exposure levels.

Disease outcome	No.	Mixed			Nonsoluble			Soluble		
		Cumulative ($\mu\text{g}\text{-year}/\text{m}^3$)	Mean ($\mu\text{g}/\text{m}^3$)	Peak ($\mu\text{g}/\text{m}^3$)	Cumulative ($\mu\text{g}\text{-year}/\text{m}^3$)	Mean ($\mu\text{g}/\text{m}^3$)	Peak ($\mu\text{g}/\text{m}^3$)	Cumulative ($\mu\text{g}\text{-year}/\text{m}^3$)	Mean ($\mu\text{g}/\text{m}^3$)	Peak ($\mu\text{g}/\text{m}^3$)
Definite/probable CBD	40	50	0.9	3.7	126	1.7	5.1	5.8 ^a	0.9	1.1
Sensitization	37	20 ^b	0.8	6.8	61 ^c	1.6	5.1	19	0.8	3.3
Normal	377	49	0.9	5.9	128	1.6	6.1	26	0.9	2.7

^a $p < 0.0001$ for definite/probable vs. normal. ^b $p = 0.0005$ for sensitization vs. normal. ^c $p = 0.04$ for sensitization vs. definite/probable, and $p = 0.003$ for sensitization vs. normal.

Table 10. Development of definite/probable CBD and sensitization by physical form of beryllium, dust, fume, and mixed: mean cumulative, mean average, and mean peak exposure levels.

Disease outcome	No.	Dust			Fume			Mixed		
		Cumulative ($\mu\text{g}\text{-year}/\text{m}^3$)	Mean ($\mu\text{g}/\text{m}^3$)	Peak ($\mu\text{g}/\text{m}^3$)	Cumulative ($\mu\text{g}\text{-year}/\text{m}^3$)	Mean ($\mu\text{g}/\text{m}^3$)	Peak ($\mu\text{g}/\text{m}^3$)	Cumulative ($\mu\text{g}\text{-year}/\text{m}^3$)	Mean ($\mu\text{g}/\text{m}^3$)	Peak ($\mu\text{g}/\text{m}^3$)
Definite/probable CBD	40	128	1.6	5.2	4 ^a	0.5	0.8 ^b	49	0.9	3.7
Sensitization	37	66 ^c	1.5	5.1	17	0.9	3.3	17 ^d	0.8	6.8
Normal	377	138	1.5	6.6	20	0.6	1.9	46	0.9	5.9

^a $p = 0.0002$ for definite/probable vs. normal. ^b $p = 0.04$ for definite/probable vs. normal. ^c $p = 0.0021$ for sensitization vs. normal. ^d $p = 0.0004$ for sensitization vs. normal.

Table 11. Development of definite/probable CBD and sensitization by the American Conference of Governmental and Industrial Hygienists notice of intended change, current OSHA, and DOE DWA threshold levels.

Disease outcome	Mean DWA exposure ($\mu\text{g}/\text{m}^3$) [n (%)]			
	0 to < 0.02	0.02 to < 0.2	0.2 to < 2	≥ 2
Definite/probable CBD	0 (0)	0 (0)	33 (10)	7 (6)
Sensitization	0 (0)	0 (0)	24 (7)	13 (11)
Normal	0 (0)	3 (100)	279 (83)	95 (83)

The correct mean exposure levels for the DWA categories in Table 11 were 0.13, 1.15, and 3.13 $\mu\text{g}/\text{m}^3$.

best estimated DWA for that job in that year. We then summed those values over all jobs for that worker to derive that worker's cumulative exposure. Next we divided that worker's cumulative exposure by the total number of days worked in his/her job history to derive that worker's mean exposure. Thus, there is only one averaging on each worker and a subsequent averaging for the population. We used multiple metrics (cumulative, average, peak job, and peak task for total exposure; exposure by chemical form; and exposure by physical form) to characterize not only the central tendencies of the exposures but also their extreme excursions.

We stand by our statement that

The inclusion of genetic data combined with exposure data may better define which individuals in this cohort are at a particularly high risk of development of CBD and/or sensitization and may account for the absence of typical exposure-response seen with other environmental or occupational toxins.

It is also important to note that both peak exposure and the different chemical and physical forms may be important factors in the risk of development of CBD but are not part of the current OSHA standard (OSHA 2005).

Finally, Kolanz states that "Rosenman et al. (2005) made no recommendations

regarding how to protect beryllium workers." Clearly, however, our call to lower the allowable standard is a recommendation we put forth to protect beryllium workers. Despite the limitations of deriving historical exposure estimates, our corrected data continue to point to the inadequacy of the current OSHA standard to protect workers from developing chronic beryllium disease.

M.R. has served as an expert witness both for companies and for workers; he evaluates industrial patients with CBD, serves as the director of a beryllium test laboratory, and is a principal investigator with a grant from Los Alamos. The remaining authors declare they have no competing financial interests.

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Editor's note: It has been clarified by Joseph A. Politch that his letter "Bisphenol A and Risk Assessment" [Environ Health Perspect 114:A16 (2006)] was written while he was employed by the Weinberg Group.