upper percentiles in an exposure distribution (Chatterjee et al. 2008). The 90th percentile NHANES dietary values used by the FDA (2010) therefore cannot be characterized as biased toward safety.

The National Toxicology Program (2005) and the California Environmental Protection Agency Office of Environmental Health Hazard Assessment (2005) have determined that there is sufficient evidence to consider naphthalene a carcinogen. The FDA's reliance on an outdated determination by the U.S. EPA (1998) does not constitute a conservative assessment of the health risks associated with exposures to naphthalene.

Dickey offers the example of the cancer potency factor for benzo(*a*)pyrene (BaP) as specifically demonstrating a "bias toward safety" based on his assertion that it reflects the "95% upper confidence limit of the dose–response curve." This characterization does not match the description of the cancer potency factor on the Integrated Risk Information System (IRIS) website (U.S. EPA 1994). In fact, the cancer potency factor was based on the "geometric mean of four slope factors obtained by differing modeling procedures" (U.S. EPA 1994). Dickey further asserts that the cancer potency factor "could be as low as zero," which implies no cancer risk and therefore contradicts the designation of BaP as a carcinogen by multiple authoritative bodies including the FDA (2010), U.S. EPA (1994), Food and Agriculture Organization of the United Nations (FAO)/WHO (2006), and the International Agency for Research on Cancer (IARC 1998).

Last, Dickey cites estimates of annual BaP dietary intake, which he attributes to natural occurrence, as a rationale for not considering the lower acceptable exposure levels we proposed in our commentary (Rotkin-Ellman et al. 2012). Unfortunately this logic is severely flawed and does not comport with the FDA's charge to protect public health. For an adult, with values based on standard risk assessment methods, the range of total dietary intake Dickey describes (0.16–3.3 µg/person/day) corresponds to a lifetime cancer risk ranging from 1.7×10^{-5} to 3.4×10^{-4}—the upper value exceeding what Dickey cites as an acceptable risk range of 1×10^{-4} to 1×10^{-6}. An appropriate FDA response to this finding would be to investigate sources of dietary exposure to PAHs and enact policies to reduce unsafe exposures. This is what the European Union has done in setting standards for BaP in foods of concern (oils and fats, smoked meats, smoked fish, fish, crustaceans, mollusks, baby food, and infant formula) (European Food Safety Authority 2008). To argue that the presence of existing (and potentially unsafe) exposures precludes a thorough assessment of risk for vulnerable populations—because it might identify further risks—runs counter to the tenet of disease prevention inherent in public health protection.

The FDA's assessments of the risks from contaminants in seafood (e.g., PAHs, mercury), food additives (e.g., bisphenol A, phthalates), and chemicals used in personal care products (e.g., triclosan) have implications for the health of millions of Americans. We hope that our commentary and these letters are the beginning of a fruitful dialogue on how to incorporate advances in the scientific understanding of the impacts of chemical contaminants on vulnerable populations into all risk assessments and policies at the FDA.

M.R.-E. and G.M.S. are employed by the Natural Resources Defense Council, a nonprofit environmental advocacy group.

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Fields and Forests in Flames: Lead and Mercury Emissions from Wildfire Pyrogenic Activity

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In the article "Fields and Forests in Flames," Weinhold (2011) addressed the toxic health effects associated with fire smoke. Although he acknowledged the limited data on the toxicity of wildfires, several important studies on environmental emissions from fire events and their consequences were omitted.

Weinhold (2011) listed multiple compounds from wildfires, back burning, and incinerated buildings, but listed only four elements: potassium, chlorine, sulfur, and silicon. Significant omissions were the toxic elements lead and mercury. Lead has been identified as one of the most environmentally pervasive and damaging metals to human health (Patterson 1965).

Several studies have detailed the remobilization of metals from fire events (e.g., Finley et al. 2009; Nriagu 1989; Odigie and Flegal 2011; Young and Jan 1977). These studies showed that significant levels of toxic and

nontoxic metals are emitted into the environment during fires. Young and Jan (1977) found that smoke from a 1975 Californian wildfire emitted various metals, including cadmium, chromium, copper, iron, lead, manganese, nickel, silver, and zinc, up to 100 km from the fire. Contamination of local marine waters with lead, iron, and manganese from the wildfire exceeded the polluting effects of the local municipal wastewater, the main source of metals.

Nriagu (1989) and Finley et al. (2009) estimated that the amount of lead (plus other trace metals) and mercury, respectively, from fires were comparable to emissions from anthropogenic sources such as industrial processes and city pollution. Nriagu (1989) estimated that global emissions of lead from wildfires ranged from 60,000 to 3,800,000 kg/year, with an average of 1,900,000 kg/year. Global mercury emissions from wildfires are also significant, estimated at 890,000 ± 490 kg/year for gaseous elemental mercury and 170 ± 100 kg/year for particulate-bound mercury (Finley et al. 2009). Until recently it was not known whether lead released by wildfires is from natural and or industrial sources. Odigie and Flegal (2011) measured the isotopic lead composition of ash from the 2009 Jesusita Fire in Southern California. Their work showed clearly that the ash from the wildfire contained industrial lead primarily from leaded gasoline used in Southern California during the 1960s through the 1980s.

Environmental media, such as air, dust, sediment, soil, and water, have well-defined and strict environmental and human health guidelines because of their damaging effect on natural and anthropogenic systems. Even low levels of atmospheric lead emissions are known to cause adverse human health effects, including irreversible neurological damage. For example, the U.S. Environmental Protection Agency (EPA) recently reduced the lead-in-air guideline by an order magnitude—from 1.5 μg/m^3 to 0.15 μg/m^3—after reviewing > 6.000 human health–lead-related studies (U.S. EPA 2008). Although pyrogenic activity affects environmental quality, its effects remain ill-defined, despite evidence of harmful human health effects from exposure to toxicants, even at very low levels (Lanphear et al. 2005). The risk from fires is likely to increase as the frequency of climatically driven fire events rises in response to predicted global warming (Intergovernmental Panel on Climate Change 2007). The predicted environmental changes present a significant research opportunity for those interested in monitoring the biogeochemical cycling of metals and their potential risk of harm to human and environmental health systems.

The authors declare they have no actual or potential competing financial interests.

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Editors' note: We thank Kristensen and Taylor for their comments. They are a useful addition to the information provided in Weinhold's news article, which addressed an extensive list of other toxic substances and mentioned mercury only briefly.

Errata

In the article "Temporal Variability of Tungsten and Cobalt in Fallon, Nevada" by Sheppard et al. [Environ Health Perspect 115:715–719 (2007)], "the Mann-Whitney test of medians" should have been "the Mann-Whitney test of differences in cumulative distribution functions." This term was used in the last paragraph of the "Materials and Methods" and in Table 1.

Davis et al. have reported an error in their article "A Retrospective Assessment of Occupational Exposure to Elemental Carbon in the U.S. Trucking Industry" [Environ Health Perspect 119:997–1002 (2011)]. On p. 1001 of their article (the next to last paragraph of the "Discussion"), there was a coding error in the original calculation: The percentage of person-years prior to 1971 was 8% and not 1.1%, as stated in the article.

The corrected sentence is as follows:

Although this extrapolation period does not cover the entire period of exposure relevant to the epidemiologic cohort, the missing years before 1971 represent a small percentage of the person years (8%) in the epidemiologic cohort. We are currently exploring options for assigning exposure levels for periods before 1971.

The authors apologize for the error.