

Food Additives (JECFA), which considered but did not include naphthalene among genotoxic and carcinogenic PAHs evaluated (FAO/WHO 2006).

Although well intentioned, the LOC values suggested by Rotkin-Ellman et al. (2012) do not appear to take into account the natural background occurrence of PAHs in foods in many categories. Assessments from the body of scientific literature is perhaps best represented by the deliberations of JECFA (which included experts from the FDA), which found benzo(a)pyrene from dietary intake alone to range from 0.16 to 3.3 µg/person/day (Benford et al. 2010; FAO/WHO 1991, 2006). The LOC values proposed by Rotkin-Ellman et al. (2012) would unnecessarily exclude many food groups from consumers, where nutritional benefits far outweighs negligible risk from PAHs.

Public health authorities are responsible for protecting consumers from contaminated commercial and recreational seafood sources, and to that end advisories may be issued to protect consumers. The federal and state interagency risk assessment for seafood safety following the BP oil spill of 2010 was designed and agreed on by all participants to provide conservative criteria that protect the public. The alternative interpretation provided by Rotkin-Ellman et al. (2012) carries a risk of doing more harm than good.

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- “safety” and is concerned that “unnecessarily conservative risk criteria” could harm human health and society as a whole. This viewpoint, which is arguably rooted more in politics than in science, ignores the long history of chemical assessments where new data and approaches have repeatedly demonstrated significantly greater risks than initially believed (Castorina and Woodruff 2003; Grandjean et al. 2010; Hernberg 2000). Furthermore, from a health-cost perspective, there is considerable benefit to assuring that the population is protected from harmful exposures to carcinogens and other toxicants (Landrigan et al. 2002; Trasande et al. 2006).
- A National Research Council (NRC) committee reviewed the status of environmental regulatory risk assessment and concluded that the new science documenting interindividual variability and the vulnerability of the developing fetus and child to chemical contaminants warrants specific changes to risk assessment practices (NRC 2009). These changes were not reflected in the FDA assessment (FDA 2010). The justifications for the FDA’s risk criteria (FDA 2010) that Dickey provides in his letter do not reflect the most current scientific understanding of the health risks from polycyclic aromatic hydrocarbons (PAHs)—or the risk assessment process—and therefore cannot be characterized as biased “on the side of safety.”
- The NRC, the U.S. Environmental Protection Agency (EPA), and the broader scientific community have recognized that children are not just small adults and that calculation of life-stage-specific doses are the most health protective method to ensure public health protection (American Academy of Pediatrics 2011; NRC 1993, 2009; U.S. EPA 2005). This necessitates use of age-specific body weights and intake and specifically refutes the claim that an adult body weight and dose can represent risk across a lifespan.
- The World Health Organization (WHO) and the U.S. EPA have recognized the extremely skewed nature of food consumption curves and the resulting increased health risk to high-end consumers. These agencies recommended that risk assessments be based on either local surveys (if available) or the 95–97th percentile of national surveys (U.S. EPA 2000; WHO 2008). Previous studies that evaluated the utility of dietary data from the National Health and Nutrition Examination Survey (NHANES) against population-specific surveys have concluded that there is a risk of significantly underestimating exposure among children if NHANES data are the sole source of dietary estimates (Riederer et al. 2010). Furthermore, alternative statistical techniques have been shown to allow better characterization of the

FDA Risk Assessment of Seafood Contamination after the BP Oil Spill: Rotkin-Ellman and Solomon Respond

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We thank Dickey for the opportunity to discuss differing approaches to evaluating health risks from chemical contaminants in food, such as those in Gulf seafood after the BP oil spill disaster. As we demonstrate in our commentary, “Seafood Contamination after the BP Gulf Oil Spill and Risks to Vulnerable Populations: A Critique of the FDA Risk Assessment” (Rotkin-Ellman et al. 2012), the choice of parameters and methods can significantly alter the conclusions of a risk assessment, thereby having major impacts on resulting policy decisions. In the example we analyzed, a risk assessment using parameters and methods specifically aimed at protecting vulnerable populations and incorporating the latest risk science differs from the Food and Drug Administration’s (FDA) approach (FDA 2010) by up to four orders of magnitude.

Dickey characterizes chemical risk assessments as inherently biased “on the side of

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.

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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 Flegel 2011; Young and Jan 1977). These studies showed that significant levels of toxic and