using more recent data is unlikely to change that conclusion (see Appendix of Jerrett et al. 2009). Finally, national risk estimates are more applicable globally than city-specific estimates because they include larger and more diverse populations.

However, because the evidence for chronic ozone mortality is more limited than the large body of evidence demonstrating mortality associations with short-term ozone exposure, we present here estimates of the global burden of ozone on mortality using RR estimates from Bell et al. (2004), a large multicity study of short-term ozone mortality. We estimated mortalities daily using the difference between preindustrial and present-day 8-hr maximum ozone, and sum mortalities over the 1-year simulation. We used the reported relationship for cardiopulmonary mortality and daily average ozone [0.64% (95% posterior interval, 0.31–0.98%) for a 10-ppb increase], and corrected to 8-hr ozone using the reported ratio between daily 8-hr and 24-hr average ozone associations with nonaccidental mortality.

Using these methods, we estimated 362,000 (95% confidence interval, 173,000–551,000) annual global premature cardiopulmonary deaths attributable to ozone, approximately 50% of the 700,000 premature deaths we calculated in our original study (Anenberg et al. 2010). Since estimated deaths due to $PM_{2.5}$ (3.7 million) are an order of magnitude larger, using a short-term rather than long-term RR estimate for ozone has only a minor effect on the overall global burden of disease due to outdoor air pollution. As RRs for chronic ozone mortality are not as strongly supported as those for $PM_{2.5}$, we expect that estimates of mortality burden will improve as research on chronic ozone exposure and mortality continues globally.

S.C.A. and J.J.W. received unrestricted funding from the Merck Foundation to support a related project, and the Merck Foundation was unaware of this research. D.Q.T. is an employee of Science and Technology Corporation, working at NOAA facilities. L.W.H. declares he has no competing financial interests.

Susan C. Anenberg
J. Jason West
The University of North Carolina at Chapel Hill
Chapel Hill, North Carolina
E-mail: jasonwest@unc.edu

Larry W. Horowitz
NOAA Geophysical Fluid Dynamics Laboratory
Princeton, New Jersey

Daniel Q. Tong
Science and Technology Corporation at NOAA Air Resources Laboratory
Silver Spring, Maryland

References

Anenberg SC, Horowitz LW, Tong DQ, West JJ. 2010. An estimate of the global burden of anthropogenic ozone and fine particulate matter on premature human mortality using atmospheric modeling. Environ Health Perspect 118:1189–1195.

Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F. 2004. Ozone and short-term mortality in 95 US urban communities, 1987-2000. JAMA 292:2372–2378.

Jerrett M, Burnett RT, Pope CA III, Ito K, Thurston G, Krewski D, et al. 2009. Long-term ozone exposure and mortality. N Engl J Med 360:1085–1095.

Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, et al. 2009. Extended Follow-up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality. Research Report 140. Boston:Health Effects Institute.

National Research Council, Committee on Estimating Mortality Risk Reduction Benefits from Decreasing Tropospheric Ozone Exposure. 2008. Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution. Washington, DC:National Academies Press. Available: http://www.nap.edu/catalog.php?record_id=12198#toc [accessed 11 March 2011].

Influence of Selenium and Mercury on Age-Related Cataracts in the Brazilian Amazon

doi:10.1289/ehp.1003242

In their article, Lemire et al. (2010) provided important data on the frequency of age-related cataracts among adults in the Brazilian Amazon and found a correlation between age-related cataracts and whole-blood total mercury (Hg) concentrations and selenium (Se) levels in plasma and whole blood. However, in the "Discussion" of their paper, they stated that they "observed no adverse effects although Se concentrations were very high, reaching 1,500 μg/L for [blood]-Se and 913 μg/L for [plasma]-Se" (Lemire et al. 2010). However, they did not mention the potential and substantial adverse health effects associated with a high body Se burden. There are potentially adverse consequences to Se body burden, such as hair loss (alopecia), tooth decay, nail changes, peripheral paresthesias, weakness, skin lesions, and diabetes (Hira et al. 2004; Nuttall 2006; Shearer 1975; Stranges et al. 2010; Sutter et al. 2008; Yang et al. 1983). It would be valuable to know what Se-related adverse effects were observed by Lemire et al. (2010). Most studies, taken together, suggest a possible attenuation of Hg toxicity, probably as insoluble form of Hg selenide (Clarkson 2002; International Programme on Chemical Safety 1990).

Selenium may be able to delay the onset of toxic effects in animal models exposed to methylmercury in the diet (Clarkson 2002; Ganther et al. 1972). However, the Hg–Se interaction may not have an equivalent effect in some animals. There is evidence that coadministration of methylmercury and Se may lead to an important synergistic effect (Heinz and Hoffman 1998). In studies of persons who have been co-exposed to Hg and Se, toxic effects of Se should be taken into account to discover the potential synergistic effect between Se and Hg.

The authors declare they have no competing financial interests.

Claudio Minoia
Anna Ronchi
Laboratory of Environmental and Toxicology Testing
"S. Maugeri" IRCCS
Pavia, Italy

Paolo D. Pigatto
Department of Technology for Health
Dermatological Clinic
IRCCS Galeazzi Hospital
University of Milan
Milan, Italy

Gianpaolo Guzzi
Italian Association for Metals and Biocompatibility Research–AIRMEB
Milan, Italy
E-mail: gianpaolo_guzzi@fastwebnet.it

References

Clarkson TW. 2002. The three modern faces of mercury. Environ Health Perspect 110(suppl 1):11–23.

Ganther HE, Goudie C, Sunde ML, Kopecky MJ, Wagner P. 1972. Selenium: relation to decreased toxicity of methylmercury added to diets containing tuna. Science 175:1122–1124.

Heinz GH, Hoffman DJ. 1998. Methylmercury chloride and selenomethionine interactions on health and reproduction in mallards. Environ Toxicol Chem 17:139–145.

Hira CK, Partal K, Dhillon KS. 2004. Dietary selenium intake by men and women in high and low selenium areas of Punjab. Public Health Nutr 7:39–43.

International Programme on Chemical Safety. 1990. Methylmercury. Environmental Health Criteria 101. Geneva:International Programme on Chemical Safety, World Health Organization. Available: http://www.inchem.org/documents/ehc/ehc/ehc101.htm [accessed 2 March 2011].

Lemire M, Fillion M, Frenette B, Mayer A, Philibert A, Passos CJ, et al. 2010. Selenium and mercury in the Brazilian Amazon: opposing influences on age-related cataracts. Environ Health Perspect 118:1584–1589.

Nuttall KL. 2006. Evaluating selenium poisoning. Ann Clin Lab Sci 36:409–420.

Shearer TR. 1975. Developmental and postdevelopmental uptake of dietary organic and inorganic selenium into the molar teeth of rats. J Nutr 105:338–347.

Stranges S, Laclaustra M, Ji C, Cappuccio FP, Navas-Acien A, Ordovas JM, et al. 2010. **Higher selenium status is associated with adverse blood lipid profile in British adults.** J Nutr 140:81–87.

Sutter ME, Thomas JD, Brown J, Morgan B. 2008. Selenium toxicity: a case of selenosis caused by a nutritional supplement. Ann Intern Med 148:970–971.

Yang GQ, Wang SZ, Zhou RH, Sun SZ. 1983. Endemic selenium intoxication of humans in China. Am J Clin Nutr 37:872–881.

Influence of Selenium: Lemire et al. Respond

doi:10.1289/ehp.1003242R

We agree with the comments made by Minoia et al. in their letter. In this study population, we did evaluate the sentinel signs and symptoms of selenosis (Lemire M, Philibert A, Fillion M, Passos CJS, Guimarães JRD,