mental design may underlie observed differences in sensitivity to detect neurotoxicity, possibly because of differences in biokinetics and exposure during sensitive windows of development. Fortunately, much effort is taking place in the scientific community to optimize experimental designs at different levels of biological complexity, including (developmental) neurobehavioral studies. Although a critical review on the impact of different experimental designs for *in vivo* (developmental) neurotoxicity studies would be very useful, it was beyond the scope of our review (Dingemans et al. 2011).

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

Milou M.L. Dingemans
Martin van den Berg
Remco H.S. Westerink
Institute for Risk Assessment Sciences
Utrecht University
Utrecht, the Netherlands
E-mail: m.dingemans@uu.nl

References

Alcock RE, Macgillivray BH, Busby JS. 2011. Understanding the mismatch between the demands of risk assessment and practice of scientists—the case of deca-BDE. Environ Int 37:216–225.

Dingemans MML, van den Berg M, Westerink RHS. 2011. Neurotoxicity of brominated flame retardants: (in-)direct effects of parent and hydroxylated polybrominated diphenyl ethers on the (developing) nervous system. Environ Health Perspect 119:900–907; doi:10.1289/ehp.1003035 [Online 18 January 2011].

Eriksson P. 2008. Response to: Use of the pup as the statistical unit in developmental neurotoxicity studies: overlooked model or poor research design? [Letter]. Toxicol Sci 103:411–413.

Gee JR, Moser VC. 2008. Acute postnatal exposure to brominated diphenylether 47 delays neuromotor ontogeny and alters motor activity in mice. Neurotoxicol Teratol 30:79–87.

Hardy M, Stedeford T. 2008. Developmental neurotoxicity: when research succeeds through inappropriate statistics. Neurotoxicology 29:476.

Williams AL, DeSesso JM. 2010. The potential of selected brominated flame retardants to affect neurological development. J Toxicol Environ Health B Crit Rev 13:411–448.

Three Criteria for Ecological Fallacy

doi:10.1289/ehp.1103768

In a large cohort study published in *Environmental Health Perspectives*, Brenner et al. (2011) confirmed previous results on I-131 exposure and thyroid cancer among a Ukranian population. According to the authors, one motivation to study this association was based on evidence from ecological studies (Jacob et al. 1999) with two methodological limitations: use of grouped doses and poor control of confounding. With these new findings, evidence from ecological, case–control, and cohort studies are consistent; thus, an interesting question is whether there was an ecological fallacy.

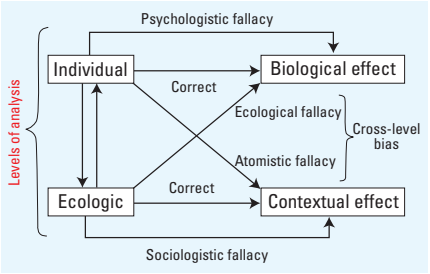

Figure 1. Levels of analysis in epidemiologic studies and potential fallacies during causal inference.

Although ecological studies are important to epidemiology (especially in environmental and social epidemiology), public health practitioners seem afraid of ecological studies. It is a common practice to assume the presence of ecological fallacy (Robinson 1950) and low-level validity when analyzing an ecological study. Most epidemiologists prefer an exclusive individualistic approach, although the importance of a multilevel causal approach is widely recognized (Diez-Roux 2002). In this sense, some authors suggest that it is as important to recognize the presence of ecological fallacy as to recognize psychologistic or individualistic fallacy (Subramanian et al. 2009) (Figure 1).

Thus, it is necessary to have clear guidelines on when there is or not an ecological fallacy. In this sense, I propose three criteria for the identification of ecological fallacy; all three of these should be present to confirm its existence:

- Results must be obtained with ecological (population) data.
- Data must be inferred to individuals. One use of ecological studies is to explore individual-level association when individual data are not available. When the focus of the study was contextual or based on population effects and there is no inference to individuals, ecological fallacy is not possible. When only the first two criteria are present—which is insufficient to affirm ecological fallacy—it is appropriate to acknowledge that there is a possible relationship and that further study is required.
- Results obtained with individual data are contradictory.

Only when empirical data are available is it possible to confirm that an ecological fallacy is present.

The author declares that he has no competing financial interests.

Alvaro J. Idrovo
Center for Health Systems Research
National Institute of Public Health
Cuernavaca, Morelos, Mexico
E-mail: javier.idrovo@insp.mx

References

Brenner AV, Tronko MD, Hatch M, Bogdanova TI, Oliynik VA, Lubin JH, et al. 2011. I-131 dose response for incident thyroid cancers in Ukraine related to the Chornobyl accident. Environ Health Perspect 119:933–939; doi:10.1289/ehp.1002674 [Online 17 March 2011].

Diez-Roux AV. 2002. A glossary for multilevel analysis. J Epidemiol Community Health 56(8):588–594.

Jacob P, Kenigsberg Y, Zvonova I, Goulko G, Buglova E, Heidenreich WF, et al. 1999. Childhood exposure due to the Chernobyl accident and thyroid cancer risk in contaminated areas of Belarus and Russia. Br J Cancer 80(9):1461–1469.

Robinson WS. 1950. Ecological correlations and the behavior of individuals. Am Sociol Rev 15(3):351–357.

Subramanian SV, Jones K, Kaddour A, Krieger N. 2009. Revisiting Robinson: the perils of individualistic and ecologic fallacy. Int J Epidemiol 38(2):342–360.

Carbon Black

doi:10.1289/ehp.1103444

In "Research Recommendations for Selected IARC-Classified Agents," Ward et al. (2010) identified research gaps for 20 occupational agents "based on evidence of widespread human exposures and potential carcinogenicity in animals or humans." (Ward et al. 2010) For carbon black, the authors suggested that

> Research needs include updating epidemiology cohorts with data on work histories and exposures in relation to particle size and surface area, and recruitment of additional carbon black facilities. The relationship between occupational exposure to carbon black and validated biomarkers of oxidative stress should be examined and exposure–response relationships in humans and rodents quantified, including the role of particle size.

Ward et al. (2010) referred to a study of British carbon black workers in which carbon black was suggested as a possible "late stage carcinogen" (Sorahan and Harrington 2007). In that study, Sorahan and Harrington (2007) called for similar analyses of other carbon black cohorts (i.e., evaluating the possibility of carbon black acting as a late stage carcinogen via the concept of "lugging," which considers only recent exposures and not historical exposures). In response to suggestions made by Sorahan and Harrington, we conducted such analyses on a large German carbon black cohort (Morfeld and McCunney 2007, 2009). We were unable to reproduce the results of the British analysis, despite the elevation noted in lung cancer among German cohort workers, thus providing no support for the late stage-lugging hypothesis. Results of a detailed analysis of the German cohort using Bayesian methodology showed smoking and exposure to occupational carcinogens prior to work at the carbon black plant as confounders probably responsible for the lung cancer excess (Morfeld and McCunney 2010).

Ward et al. (2010) called for enhanced exposure–response assessments in humans. Currently, a dose–response exposure analysis is under way on the U.S. carbon black cohort (> 5,000 production workers). An earlier evaluation of this cohort showed no increase in any type of cancer (Dell et al. 2006).