development. Lead at high doses kills children by causing an encephalopathy, so the question is not whether lead is toxic to the brain, but at what dose the toxicity is measurable. Lead is the best studied of the environmental chemical agents thought to damage the brains of children at relatively low levels of exposure. Because lead poisoning occurs in environments that offer other challenges to the families, isolation of that effect has been difficult. Responsible reviewers, though, including the authors of the meta-analysis Ernhart cites (*2*), have taken confounding factors into account and judged it to be more likely than not that lead causes the defects. Claims of deliberate misrepresentation have not been verified (*3*); moreover, such claims were directed at a specific investigator and would not in any case affect interpretation of the weight of the literature. From our point of view, causality was sufficiently established so that we and our advisors thought that a trial, testing whether such damage could be prevented or reduced, was justified.

Finally, it is hardly "curious" that we do not report associations between lead level and IQ from trial data. Therapeutic trials and observational studies of etiology are designed very differently. In order that we could best make comparisons between children given the active drug and children given placebo, we selected children from a relatively high, narrow range of blood lead levels and, of course, we treated them, both of which factors make the trial data less suitable for studying etiology. There have been many other studies designed specifically to do that, as noted above (*2*).

WALTER J. ROGAN,[1]* JAMES H. WARE,[2] KIM N. DIETRICH,[3] JERILYNN RADCLIFFE,[4] GEORGE G. RHOADS[5]

For the Treatment of Lead-Exposed Children Trial Study Group: [1]National Institute of Environmental Health Sciences, Research Triangle Park, NC 27709, USA. [2]Harvard School of Public Health, Boston, MA 02115, USA. [3]University of Cincinnati, Cincinnatti, OH 45267, USA. [4]Children's Hospital of Philadelphia, Philadelphia, PA 19104, USA. [5]Environmental and Occupational Health Sciences Institute, University of Medicine and Dentistry of New Jersey, Piscataway, NJ 08854, USA

*To whom correspondence should be addressed. E-mail: rogan@niehs.nih.gov

References and Notes

1. W. J. Rogan *et al., N. Engl. J. Med.* **344**, 1421 (2001).
2. S. J. Pocock, M. Smith, P. Baghurst, *Br. Med. J.* **309**, 1189 (1994).
3. J. Palca, *Science* **256**, 1389 (1992).
4. The authors note the passing of our senior colleague, J. Julian Chisolm Jr., a month after the appearance of our paper. His intellectual contribution to the study continued until the end, and we will miss him.

CORRECTIONS AND CLARIFICATIONS

PERSPECTIVES: "One for all?" by B. E. Ellis and G. P. Miles (15 Jun., p. 2022). Reference 5 [S. Plakidou-Dymock *et al.*, *Curr. Biol.* **8**, 315 (1998)] has been corrected by its authors. In the 3 April 2001 issue of *Current Biology* (p. 535) Kanyuka, Couch, and Hooley stated that their claim of a connection between the GCPR in *Arabidopsis* and the cytokinin response is wrong; a mutation independent of the antisense construct for the seven-trans-membrane receptor GCR1 caused this phenotype. Therefore, the function of GCR1 remains an open question. The main thrust of the Perspective is unaffected by this situation.

REPORTS: "Differentiation of embryonic stem cells to insulin-secreting structures similar to pancreatic islets" by N. Lumelsky *et al.* (18 May, p. 1389). In the first line at the top of page 1392, the average protein content of a cell is given as about 20 pg. It should have been given as about 200 pg per cell.