Robert Harriss
Texas A&M University, College Station, TX 77843–3136, USA
E-mail: harriss@tamu.edu

■

Polio Vaccine Production

In the debate between Alan W. Dove and Vincent R. Racaniello (Policy Forum, 8 Aug., p. 779) and Harry F. Hull and R. Bruce Aylward (Policy Forum, 8 Aug., p. 780) about whether or not to convert polio vaccination from oral to inactivated vaccine during the last stages of eradication, the supply of enhanced-potency inactivated vaccine is an important consideration. Currently, Pasteur Mérieux Connaught supplies most of the inactivated polio vaccine (IPV) used in the world. In our opinion, expanded production to 500 million doses per year would be feasible if enough advance notice were given, particularly in view of the likelihood that other manufacturers would enter into production of IPV.

Stanley A. Plotkin
Emeritus Professor of Pediatrics, University of Pennsylvania, and Consultant, Pasteur Mérieux Connaught, 4650 Wismer Road, Doylestown, PA 18901, USA

■

Clean Air Skepticism

The article by Jocelyn Kaiser about the debate over tightened ambient air quality standards (News & Comment, 25 July, p. 466) does a good job of presenting the U.S. Environmental Protection Agency's (EPA's) side of the story, but does not mention several of the scientific issues that make up the basis for widespread skepticism. Most of the new epidemiological studies have examined short-term (daily) responses, including mortality. However, because of temporal colinearity among pollutants and uncertain exposures of the putative victims, it is not possible to apportion blame among potential environmental agents with certainty (*1*, *2*). The effects of carbon monoxide have often been neglected, and the effect of particle size (if any) remains unclear (*3*). As Kaiser points out, there are also questions about the degree of prematurity of death.

Two recent mortality studies considered long-term survival rates of defined cohorts in relation to the average air pollution concentrations, as measured during periods of follow-up. Kaiser describes the first of these studies (*4*) as "convincing"; it reported that about 26% of all deaths in six U.S. cities were attributable to air pollution, thus putting air pollution on a par with smoking and implying that eliminating air pollution could have about the same health benefit as eliminating all human cancers, for example. Kaiser quotes me as allowing that a systematic gradient in lifestyle across the six cities "might" account for the mortality gradient that was attributed to air pollution. Such a regional gradient in physical activity exists (*5*), and its implied effect on longevity is almost exactly the same as that shown in an independent study of individuals in California (*6*). Accounting for this confounding variable would leave a mortality excess of only about 5% (in the most polluted city), and this excess could well be a result of the much higher historic exposures that were present in that city (*6*). My concern is thus much more than a hypothetical "what if."

Studies that conclude that current air pollution is as lethal as smoking or cancer have omitted known confounders such as diet, physical exercise, income, and employment status, and treat nonlinear factors (for example, body mass and education) as if they were linear. The second cohort study

(7) also suffered from most of these design faults, and it considered only two pollutants, neglecting the influence of their correlates. These two studies (4, 7) are thus a shaky basis on which to attempt to interpret the ambiguous daily studies.

Finally, the scientific skepticism about this issue runs much deeper than just pro forma industrial opposition. A recent invited critical review of the particulate matter standards expressed doubt about the validity of both the short- and long-term mortality studies (8), and other academics have expressed similar opinions (9, 10). EPA would be well advised to demonstrate the actual public health benefits already accrued from its existing air quality regulations before mandating the hefty additional investments that meeting the new regulations will require.

Frederick W. Lipfert
Environmental Consultant,
23 Carll Court,
Northport, NY 11768, USA

References

1. J. M. Samet, S. L. Zeger, J. E. Kelsall, J. Xu, "Air pollution, weather, and mortality in Philadelphia, 1973–88, Report to the Heath Effects Institute on Phase 1B: Particle Epidemiology Evaluation Project" (Johns Hopkins Univ. Press, Baltimore, MD, 1996).
2. R. T. Burnett, S. Cakmak, J. R. Brook, D. Krewoki, *Environ. Health Perspect.* **105**, 614 (1997).
3. F. W. Lipfert and R. E. Wyzga, *J. Air Waste Manag. Assoc.* **47**, 517 (1997).
4. D. W. Dockery *et al.*, *N. Engl. J. Med.* **329**, 1753 (1993).
5. E. L. Frazier, C. A. Okoro, C. Smith, D. V. McQueen, *Morbid. Mortal. Wkly. Rep.* **45** (no.556), 1 (1996).
6. F. W. Lipfert, in *Particulate Matter: Health and Regulatory Issues* (Air and Waste Management Association, Publ. VIP 49, Proceedings of the International Specialty Conference, Pittsburgh, PA, April 1995), pp. 78–102.
7. C. A. Pope, III, M. J. Thun, M. M. Namboodiri, D. W. Dockery, J. S. Evans, *Am. J. Resp. Crit. Care Med.* **151**, 669 (1995).
8. S. Vedal, *J. Air Waste Manag. Assoc.* **47**, 551 (1997).
9. R. F. Phalen, testimony before subcommittees on health and environment and on oversight and investigations, Committee on Commerce, U.S. House of Representatives, 8 May 1997.
10. A. A. Moghissi, *Environ. Intl.* **23**, 147 (1997).

■

Genetic Evolution of Morphology

Two commentaries in the 4 July issue, by their contrast, inadvertently point to a missing element in most discussions of the evolution of animal form. In his Perspective "Which came first, the hypha or the yeast?" (p. 52), P. T. Magee draws the lesson from a report by B. R. Braun and A. D. Johnson (4 July, p. 105) that the existence of a simple genetic switch between the budding yeast and the thread-like hypha morphologies of *Candida albicans* suggests that "there is no 'default' form for this organism." This seems reasonable: C. *albicans* is thought of as polymorphic, with numerous forms being consistent with a single genetic constitution. The choice between alternative forms in such cases may depend on epigenetic or environmental factors, although, in principle, heritable genetic change could bias such choices, leading to distinct morphological varieties.

This view may be compared with one presented in the Special News Report by Elizabeth Pennisi and Wade Roush "Developing a new view of evolution" (4 July, p. 34), in which they discuss, among other things, recently published evidence that a gene called *manx* distinguishes a species of tunicates whose larvae lack tails from a related species whose larvae develop them (B. J. Swalla and W. R. Jeffery, Reports, 15 Nov., 1996 p. 1205). We are told in the News article that the result "raises the possibility that a single genetic change could be responsible for the innovation that led to a tail in primitive vertebrates."

The attribution of ineffable creative power to individual genes is not an isolated instance, but can be traced back at least to the erroneous "unit character" model of Mendelism propounded by some early ge-