pothesis, the missing data are positive HIV-1 samples from human sera from the 1930s and 1940s. However, according to the analysis by Korber *et al.*, even if HIV were already present in humans in 1930, there may have been as few as 10 or so humans infected with HIV-1 M-group strains in all of Africa as late as 1950. Given the small number of predicted infections in humans at this time, it would be numerically surprising if any positive HIV-1 samples were found in old serum samples from before the late 1950s, even if the virus had been slowly diversifying in human populations since 1930. In contrast, the chimp SIV M-group viruses should be relatively easy to find if the OPV hypothesis is correct, because they would be expected to be common in modern chimp populations. Unless direct evidence is found to support or refute the OPV hypothesis (such as a contaminated batch of OPV, or HIV-1 samples from humans from before the OPV trials), a thorough study of SIV variation in wild chimpanzee populations will be the best way to resolve this debate.

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Not-So-Simple Minds

The investigation of brains of people with outstanding abilities has long fascinated neuroanatomists, philosophers, and scientists in other disciplines, as well as the public [a topic discussed in Wang's Essay on Science and Society (*Science*'s Compass, 1 Sept., p. 1477)]. In the ongoing search for an explanation of genius, Witelson, Kigar, and Harvey analyzed the morphology of Albert Einstein's brain, which they described in an article in *Lancet* (*1*).

Witelson and colleagues examined photographs of Einstein's brain taken in 1955 (*2*) and found that there was no parietal operculum, a part of the brain involved in speech. In addition, quantitative measurements (based on calibrated photographs?) revealed that the size of a specific gyral region in the frontal operculum was different in Einstein's brain compared with that of a control group. On the basis of their examination of Einstein's brain (which they describe in their *Lancet* article as morphologically "exceptional") and information gathered from several case studies of the brains of outstanding people, such as Carl F. Gauss (1777–1855), Witleson and colleagues suggested that they had found a new criterion for explaining extraordinary intellectual talents. However, their study was based on the "convolutional morphology," as termed by Critchley in his monograph *The Parietal Lobes* (*3*). No data were given on the architectural structure or connections with other areas, the cerebro-arterial topography, or the white matter. As Critchley mentions in his monograph, such features are of equal importance in studying the complex parietal brain.

There is one medical condition—congenital in children or acquired in adults—where maldevelopment or destruction of the operculum (particularly the frontal operculum) is associated with the failure of speech development or a loss of speech. This syndrome in children has been described by Worster-Drought, and in adults by Foix and Chavany and co-workers (*4*). The former author noticed a correlation of abnormal speech development in children with dysplasias associated with destruction of the operculum.

On the basis of our knowledge of brain development and our own magnetic resonance imaging study of the brain of Gauss (*5*), we suggest that the abnormality observed in Einstein's brain is most likely responsible for the well-known delay of his speech development and the dyslexic features that accompanied him during his life.

Sometimes we forget how limited our current research is. There is the dialectical saying, "If the human brain would be so simple that we could understand it, we would be so simple that we couldn't."

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References and Notes

1. S. Witelson, D. L. Kigar, T. Harvey, *Lancet* **353**, 2149 (1999).
2. Figure 1 in (*1*) had a wrong caption: The photographs were taken in 1955, not in 1995.
3. M. Critchley, *The Parietal Lobes* (Collier MacMillan, London, 1953).
4. H.-J. Christen *et al.*, *Dev. Med. Child Neurol.* **2**, 122 (2000).
5. A. D. Wittmann *et al.*, *Mitt. Dtsch. Gauss Ges.* **36**, 9 (1999).

Data for an Election Year

Albert Einstein was not the only public figure whose brain has been subjected to scientific (or pseudoscientific) analysis (*Science*'s Compass, Essay on Science and Society by Steve C. Wang, "In search of Einstein's genius," 1 Sept., p. 1477). At the December 1933 meeting of the American Association for the Advancement of Science (publisher of *Science*) in Boston, Arthur MacDonald of Washington, D.C., presented a paper that compared the average weight of the brains of members of the U.S. House of Representatives with that of U.S. senators (*1*). The average weight of the 71 representatives' brains studied was 50 ounces, whereas that of 18 senators was 52 ounces, leading one newspaper to headline its story, "More brains needed to get into U.S. Senate than into House, survey shows" (*2*). How MacDonald obtained his data was not reported.

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1. A. MacDonald, "International legislative anthropology," General Program of the Fifth Boston Meeting, American Association for the Advancement of Science (AAAS), Boston, MA, 27 December 1933 to 3 January 1934 (AAAS, Washington, DC), p. 86.
2. "More brains needed to get into U.S. Senate than into House, survey shows," *The Houston Chronicle*, 31 December 1933.

An Early Taste of Things to Come

I was delighted to read about Julie Mennella's research in the Random Samples item "Cultivating tastes in the womb" (21 July, p. 387). She and her colleagues were able to demonstrate that maternal diet does affect the taste preferences of human infants. Theirs may be the first experimental demonstration of this effect in humans, but 25 years ago, my colleagues and I conducted a similar experiment with rat pups (*1*). After parturition, mother rats were fed one of two different diets in a separate feeding cage so that their nursing pups would not encounter solid food. The pups, upon weaning and encountering solid food for the first time, showed a preference for their mother's diet. We speculated that flavor cues were present in the mothers' milk.

At the time, I was an undergraduate at Brooklyn College of the City University of New York enjoying my first experience with scientific research. I expected that as soon as our work with rodents was published, others would rush to replicate our findings in humans. At long last someone has.

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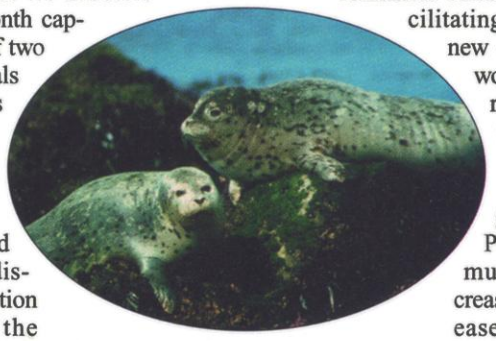
PCBs Are a Health Risk for Humans and Wildlife

Thomas J. O'Shea, in his Letter entitled "PCBs not to blame" (16 June, p. 1965), is technically correct in stating that there is no evidence to implicate polychlorinated biphenyls (PCBs) directly in the virus-associated mass mortality of European seals in 1988. However, the topic of PCBs and associated health risks requires a more encompassing "weight of evidence" approach than he has presented. Current scientific consensus supports the idea that PCBs played a

contributory role in the severity of the event.

About 20,000 European harbor and gray seals died during this event, representing up to 60% of local populations. A newly isolated, identified, and characterized *Morbillivirus* (phocine distemper virus) was assigned as primary cause of the event (*1*). However, concerns lingered that persistent organic pollutants (POPs), many of which are demonstrated immunotoxicants (including dioxins, the "dioxin-like" PCBs, and furans), played a role. We therefore carried out a 30-month captive feeding study of two groups of harbor seals in which 22 seals were fed herring from either the relatively uncontaminated Atlantic Ocean or the contaminated Baltic Sea. POPs disrupted immune function in the seals fed the Baltic Sea herring, causing diminished T cell function in vitro and in vivo and reduced natural killer cell function (*2, 3*), both of which are crucial to antivirus defenses in vertebrates.



Although not the primary cause of the harbor seal die-off in Europe in 1988, PCBs appear to have had a contributory role.

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Our study seals were not directly dosed with pure PCBs, but this would have been unethical and unnecessary. Our aim was to mimic "real world" conditions for free-ranging populations of seals exposed to POPs with a carefully controlled captive feeding study, rather than to create artificial and unrealistic exposure regimes. In the case of the European seal die-off, the following lines of evidence implicate the PCBs: (i) immunotoxicity observed in our captive seals was typical of dioxin-like contaminants, not the other POPs; (ii) PCBs were the overwhelming dioxin-like chemical measured in samples taken from our study seals (93% of total dioxin Toxic Equivalency Quotient) compared with dioxins and furans; (iii) laboratory rat studies using the identical herring diets and a positive control group exposed to dioxin confirmed that the dioxin-like PCBs were largely responsible for the effects observed (*4*); (iv) an extensive laboratory animal literature underscores the immunotoxicity of the dioxin-like compounds in particular; (v) most European seal populations in 1988 had PCB levels that exceeded those found to be immunotoxic in our captive study (*2*) and would have been exposed in a manner (that is, perinatally) that would ensure a more profound immunotoxicity; and (vi) the several *Morbillivirus*-associated mass mortalities observed since 1987 have been largely restricted to the more contaminated marine mammal populations (*5*).

Human and wildlife toxicologists face the challenge of identifying specific chemicals responsible for toxic effects in cases of complex dietary exposures. Ultimately, it is the weight of evidence from laboratory, semi-field, and epidemiological studies that highlights PCBs as a significant threat to the health of wildlife and humans (*6*). Indeed, anthropogenic factors, including environmental contaminants, may be facilitating the emergence of new diseases around the world (*5*). In addition, recent studies of nursing human infants have found that even "background" levels of PCBs lead to immunotoxicity and increased incidence of disease (*7*). The valuable lessons learned from the more highly exposed wildlife species compel us to better integrate human and ecological research to assess the continued health risks of POPs to all consumers.

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* This letter does not reflect EPA policy.

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5. C. D. Harvell *et al.*, *Science* **285**, 1505 (1999).
6. P. S. Ross, *Hum. Ecol. Risk Assess.* **6**, 29 (2000).
7. K. N. Weisglas *et al.*, *Pediatr. Res.* **38**, 404 (1995).

CORRECTIONS AND CLARIFICATIONS

Letters: Corrections and Clarifications (3 Sept. 1999, p. 1493). The trace in Fig. 4C reproduced in this correction of the Report "Molecular identification of a eukaryotic, stretch-activated nonselective cation channel" by M. Kanzaki *et al.* (6 Aug. 1999, p. 882) was offset slightly in the negative direction. A correct panel has been published (26 May 2000, p. 1347).