The practicability of a provision in the new National Institutes of Health Guidelines for Stem Cell Research that requires institutional assurance of stem cell derivation is discussed. Regarding the origin of AIDS, "a thorough study of [simian immunodeficiency virus] variation in wild chimpanzee populations will be the best way to resolve this debate." The abnormal morphology of Albert Einstein's brain, it is suggested, is more likely an explanation for his delayed speech development than his genius. A 1933 study of the average brain size of members from the U.S. House of Representatives and Senate is offered as snack food for thought this election year. And the difficulties of deciphering the health risks of persistent organic pollutants to both humans and wildlife are examined.

The NIH Guidelines on Stem Cell Research

Donald Kennedy is appropriately cautionary in his Editorial "Two cheers for new stem cell rules" (*Science*'s Compass, 1 Sept., p. 1469) in calling attention to both the political shoals that lie in the path of the newly issued National Institutes of Health (NIH) guidelines on human embryonic stem cell research, and the unsatisfactory balance beam on which the guidelines rest in distinguishing ethically between derivation and use of these cells. Patient advocacy groups and the scientific community have welcomed the new guidelines, yet the document contains one requirement that is problematic and could impede the intent to broaden scientific participation in this promising technology.

The troublesome provision requires that applications for federal funding be accompanied by a signed assurance by "the responsible institutional official" that the stem cells were derived in accordance with the NIH guidelines, as well as an abstract of the protocol used to derive the cells. The assurance requirement might be appropriate in those infrequent circumstances in which stem cell preparations will be freshly derived in temporal and spatial proximity to their actual use. But as dissemination of these cells and cell lines becomes widespread, how will "responsible institutional officials" be able to assure the details of their distant derivation? Moreover, because private funds, which will often be corporate, must be used to derive the cells and may be used to establish cell lines, details of preparation may be considered proprietary and not made fully accessible.

Institutional assurances are legally binding commitments that the behaviors of investigators who conduct federally sponsored research within an institution comply with applicable law and regulation. Such assurances are not intended to deal with nor be effective in addressing behaviors that take place beyond the institution's reach. Scrupulous compliance with assurances is always expected, but especially so in an area of research that is certain to remain contentious and intensely scrutinized. Therefore, the new assurance requirement needs to be modified.

Granted that knowledge about the derivation of embryonic stem cells will continue to be necessary for biological and political reasons, would it not be sensible to recognize that investigators and institutions share responsibility for establishing a realistic chain of custody over stem cells that must ultimately rest on accepting the assertions of the originating providers? And that the institutional assurance itself can be no more than an attestation that due diligence has been exercised? A relatively simple way to exercise this shared responsibility would be to require investigators to obtain from embryonic stem cell providers certification that the cells were derived in compliance with the NIH guidelines. The certification ("package insert") would follow the stem cells as they are propagated, shared, and disseminated. The NIH could develop a model form to ensure that content is standardized and all relevant concerns addressed. For their part, the institutions would establish the requirement as policy and assure the government that it was enforced.

Although these changes would not shield institutions from their ultimate accountability in overseeing this research, they would provide clarification and credibility to the mandated institutional assurance, as well as a practicable way to meet it. They would also help to ensure that institutions do not shy away from participation in this research from fear of assuming damaging liability for distant behaviors beyond their control.

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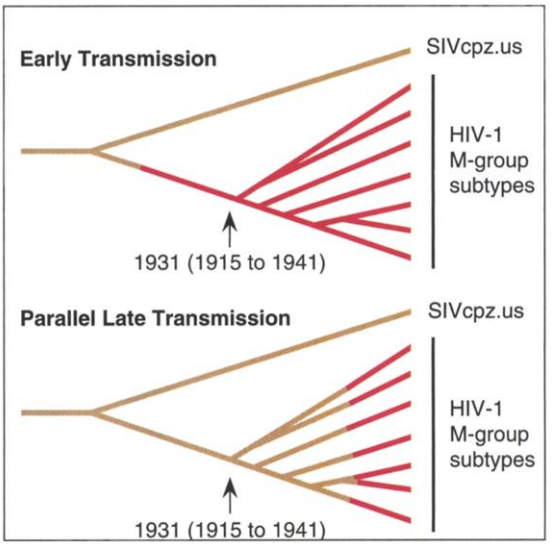
How to Resolve the Debate on the Origin of AIDS

Although I agree with Edward Hooper (Letters, 18 Aug., p. 1140) that the analysis by Korber *et al.* (Research Article, "Timing the ancestor of the HIV-1 pandemic strains," 9 June, p. 1789) is technically consistent with either an early or a late transmission of simian immunodeficiency virus (SIV) from chimps to humans (the principal origin of AIDS), we disagree on which of the two hypotheses is more likely in light of the presented data. In both cases, there are as-yet-uncollected data that could provide support for the respective hypotheses.



More data are needed to resolve which hypothesis on the origin of AIDS, early or late transmission of SIV to humans, is correct. Beige, SIV in chimpanzee populations; red, HIV-1 M-group viruses in humans.

If Hooper is correct that chimp SIVs were transmitted to humans from contaminated oral polio vaccines (OPVs) in the 1950s, then we should expect to see a large diversity of M-group viruses in present-day chimpanzee populations. Sampling to date has not revealed any such viruses, although more detailed sampling obviously should be undertaken. Finding diverse chimp SIVs that are phylogenetically embedded in the M group would support late, parallel transmission of the viruses into humans, consistent with Hooper's OPV hypothesis.

In the case of the early transmission hy-

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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