plex, as the article reports: If any agency exceeds an 8% growth rate, it is removed from the base calculation of the bill, which treats civilian research in the aggregate. Therefore, NIH or, for that matter, any other agency that develops political steam, can grow much more rapidly than the annual 5.6% aggregate called out in the bill without harming other accounts.

In addressing the NIH issue, the bill's Senate sponsors worked closely with a number of science, math, and engineering organizations to prevent any disciplinary rifts from developing. They succeeded admirably. Moreover, inspired, in part by intersociety cooperation on the Frist-Rockefeller bill, the presidents of the American Chemical Society, the American Mathematical Society, the American Physical Society, and the Federation of American Societies for Experimental Biology recently presented joint testimony before the House Veterans' Administration–Department of Housing and Urban Development appropriations subcommittee.

The article also states that backers of the Frist-Rockefeller bill were attempting to do an "end run" around House Science Committee Chairman James Sensenbrenner (R–WI), who has shown "distaste" for the bill, by having the Commerce Committee take the lead. The truth is that last year U.S. Representative Heather Wilson (R–NM) submitted the bill in the House, and she is a member of the Commerce Committee. This year, the science and engineering communities have been working with both House committees, since both lay jurisdictional claim to the proposed legislation.

Finally, the article presents pie charts projecting how NIH and other civilian research agencies would fare proportionately in the year 2003 under the Frist-Rockefeller scenario. The charts are now moot, because the amended Frist-Rockefeller bill establishes no constraint on the growth of any agency and no penalty for the remaining ones, if appropriators or authorizers see fit to exceed the 8% threshold established in the legislation. Therefore, no projections can be made, absent assumptions not present in the legislation.

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Einstein's Diploma

In his Letter to the Editor to "set the record straight" about Albert Einstein (*Science*'s Compass, 21 May, p. 1273), Harold I. Brown, citing Abraham Pais's splendid biography (*1*), writes that "Einstein had received a diploma from the Federal Institute of Technology in Zurich, which indicated that he was qualified to teach physics at the university level."

Although Einstein, upon graduation, repeatedly sought a university position (unsuccessfully), the 4-year curriculum, according to Pais (*1*, p. 41), qualified Einstein "as a Fachlehrer, a specialized teacher, in mathematics and physics at a high school." And until he got the patent office job, Einstein did teach at high schools.

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References
1. A. Pais, *Subtle Is the Lord: The Science and the Life of Albert Einstein* (Oxford Univ. Press, Oxford, UK, 1982).

Our Human-Machine Civilization

Diane Proudfoot's review (*Science*'s Compass, 30 Apr., p. 745) of my book *The Age of Spiritual Machines* (Viking, New York, 1999) ignores its salient arguments and instead mires the reader in obscure and misleading factual objections. For example, she says that Univac was installed in April 1951, and "not in 1950 as Kurzweil claims." Actually, I made no such claim, but correctly stated that Univac was developed during 1950. Later (p. 320), I cited 1951 as Univac's installation date. And Univac was installed in March, not April, 1951.

If this all seems petty, then you have a good fix on the spirit of the review. Proudfoot says that Ludwig Wittgenstein's *Tractatus* "says nothing about brains." But Wittgenstein describes it as an examination of what humans can "know," and it is generally accepted that the brain is the organ responsible for knowing.

Proudfoot complains about anthropomorphizing, but there is no harm in using such terms; we routinely speak of the legs of a chair or the hands of a doll. It doesn't follow that it is our intent to endow these objects with human qualities. I clearly state that today's machines do not have the endearing qualities of humans—they are, after all, still a million times simpler, although this disparity is rapidly shrinking. She makes no comment on the book's specific theses, such as reverse engineering the human brain and harnessing its methods in increasingly powerful computational mediums. We are already able to replicate the detailed input-output response of extensive clusters of human neurons, and there is nothing to prevent these efforts from scaling up to the entire human brain.

Instead, she drags out old anti–artificial-intelligence (AI) arguments: some early predictions were wrong (which I discuss); many AI projects have crashed (al-

though I appreciate her acknowledgement that my projects are not among these).

Proudfoot's arguments aside, nonbiological entities, which today have many narrowly focused skills, are going to vastly expand in the breadth, depth, and subtlety of their intelligence and creativity. My book discusses why this is inevitable, the nature of the technologies that will emerge, and the impact this will have on our human-machine civilization, a development no less important than the emergence of human intelligence some thousands of generations ago.

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Neuronal Cell Death: Retraction

We reported that p75 nerve growth factor receptor (p75^{NGFR}) induces the death of a subpopulation of cholinergic medial septum neurons during postnatal development (Reports, 6 Dec. 1996, p. 1729) (*1*). In an analysis of new sets of p75^{NGFR}-deficient and control mice, we find, contrary to our previous report, that (i) the number of choline acetyltransferase (ChAT)–positive (cholinergic) medial septum neurons increases between postnatal days 6 and 15 in 129/Sv and Balb/c control mice; (ii) the number of TUNEL (an indicator of apoptosis)–positive cells in the medial septum is similarly low (one to two cells per 10-micrometer section) in p75^{NGFR}-deficient and control mice at postnatal day 8; and (iii) the number of ChAT-positive neurons is similar in adult p75^{NGFR}-deficient and control mice (*2*). Reanalysis of brain tissue sections from the mice of the previous report (*1*) confirms points (i) and (ii) and reveals that the adult p75^{NGFR}-deficient mice have only approximately 20% more ChAT-positive septal neurons than does the new group of control mice, as compared with the reported 50%. In addition, we fail to confirm that the control mice treated with the p75^{NGFR}-interfering dc28-36 peptide have more ChAT-positive neurons than the mice treated with vehicle.

Thus, there does not appear to be a decrease in cholinergic medial septum neurons during postnatal life, and thus p75^{NGFR} does not appear to cause the death of these neurons. I sincerely apologize for any difficulties that the incorrect information may have caused.

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References

1. C. E. E. M. Van der Zee, G. M. Ross, R. J. Riopelle, T. Hagg, *Science* **274**,1729 (1996).
2. N. L. Ward and T. Hagg, in preparation.