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of student. Expecting all institutions to adopt an educational model in place at one type of institution weakens, not strengthens, the enterprise.

Decrying "publish or perish," Scharberg convolutes the message of the NSF report with her stated objective for universities to recognize the "scholarship of teaching" as a legitimate scholarly activity. If this change is to occur, the same standards for faculty evaluation, proposal review, and publication of science-education research have to be applied to those enterprises as to "traditional" research, or they will continue to remain peripheral.

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Hippocampal Neurodegeneration in Aging

Recent articles (I. Wickelgren, Horizons in Aging, News, 5 July, p. 48; Research News, 1 Mar., p. 1229) have addressed the question of hippocampal cell death in normal aging. Although newly developed stereological methods have demonstrated cell loss in some regions of aged mammalian brain, including areas associated with the hippocampus in humans, these methods have not detected a loss of principal neurons in the CA1-CA3 regions of hippocampus in human, monkey, and rodent brain (1-3). These findings contrast with earlier reports of age-related cell loss in those regions of hippocampus based on decreases in neuron density (4-6). Because the new stereological techniques are not confounded by many factors, such as changes in brain volume or size and orientation of cells within the structure, the discrepancy could be due to methodology alone. A letter (P. W. Landfield et al., 31 May, p. 1249) and commentary by Wickelgren and elsewhere (7) touch on other issues that might account for the discrepancy, apart from differences in neuroanatomical methods. The presentation of this debate has given the impression of different perspectives and disagreement where little may actually exist. In subsequent communications among the various investigators, we find broad agreement on a number of issues.

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Although differences in sampling procedures and counting methods cannot be excluded as contributing factors, it is unlikely that age-related changes in the volume of the hippocampus or strain differences can account for the discrepancies. Increases in the volume of the rodent hippocampus are observed into adulthood (8), but the magnitude of this change is small relative to the decreases in cell density (30 to 40%) reported in comparisons of aged rats with young rats in the 6- to 8-month range (4, 6). Moreover, stereological studies have now reported no hippocampal neuron loss in the Long-Evans rat strain used in a number of the earlier studies reporting cell density measures (3). In addition, a behavioral characterization for hippocampal-dependent cognitive decline in that study (3) demonstrated no cell loss in aged rats with cognitive impairment. Similar findings have been reported for Wistar rats (2). On the basis of these results, we are in substantial agreement that neuron loss of the type reported earlier is not required for age-associated cognitive impairment.

It is also possible that aging may not lead to neurodegeneration in the hippocampus, but rather may increase vulnerability to other factors that do cause cell death (9); these factors may have differed across study populations.

We all agree that the effects of aging on neuronal function in the hippocampus that precede neuron loss are likely to play an important role in cognitive decline and may also render neurons more vulnerable to degeneration precipitated by other causes.

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Earth's Carrying Capacity

The News article "Ecologists look at the big picture" (13 Sept., p. 1490) by Anne Simon Moffat states that I estimated the Earth could support 1 trillion people (1), "assuming improved agriculture." Although I cited deWit's calculation (2) that "1,000 billion people could live from the earth if photosynthesis is the limiting factor," and Marchetti (3) has analyzed how 10^{12} people could live, I analyzed how much land 10 billion people might spare for nature (1).

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Corrections and Clarifications

In the picture accompanying the article "Universities step up to the challenge" (News, Science in Japan, 4 Oct., p. 44), the person identified as Hiroyuki Yoshikawa, president of the University of Tokyo, was actually Hiroshi Yoshikawa, a professor of molecular biology at Nara Institute of Science and Technology. Hiroyuki Yoshikawa is shown below. *Science* apologizes for the error.



Figure 2A (p. 356) and figure 5, A and B (p. 357), in the report "The secreted product of *Xenopus* gene *lunatic Fringe*, a vertebrate signaling molecule" by J. Y. Wu *et al.* (19 July, p. 355) were printed too darkly. The correct figures appear below. In figure 2A there are two bands in lane 4 near 50 kD; in figure 5A there is one band in lane 7 of the Xbra panel; and in figure 5B there is one band in lane 7 of the Globin panel.



In the ScienceScope item "Gulf's 'dead zone' worries agencies" (18 Oct., p. 331), Terry Nelsen's name was incorrectly spelled.

Letters to the Editor

Letters may be submitted by e-mail (at science_letters@aaas.org), fax (202-789-4669), or regular mail (*Science*, 1200 New York Avenue, NW, Washington, DC 20005, USA). Letters are not routinely acknowledged. Full addresses, signatures, and daytime phone numbers should be included. Letters should be brief (300 words or less) and may be edited for reasons of clarity or space. They may appear in print and/or on the World Wide Web. Letter writers are not consulted before publication.

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