the FFTF would avoid the major near-term construction costs that are of concern to Agnew.

Several other national needs are fulfilled by FFTF operations. Because it was originally designed to burn mixed-oxide fuel, the FFTF is an ideal facility to reduce the excess stockpile of weapons-grade plutonium. In addition, the core design, target volume, flux, and energy spectrum are suited to production of a broad array of radioisotopes needed for medical, industrial, and agricultural purposes (3). Built to the highest quality standards, the FFTF has a remaining life expectancy of 23 to 30 years.

During its 12 years of operations, the FFTF showed that it could safely operate at full power (400 megawatts thermal) for extended periods (128 days) and achieve high fuel burn-ups (238,000 megawatt days per metric ton). Given these facts, any prudent policy decision regarding tritium production should consider the capabilities of the FFTF.

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What would humorist Tom Lehrer have made of Agnew's proposal that Russia supply the tritium needed (in view of its 12-year half-life) to keep the U.S. hydrogen bomb in existence? Alternatively, the bomb could be allowed to die the most natural of deaths by radioactive decay. Contriving a secure lock on tritium production to enable this would be a worthwhile achievement on the part of the arms control community.

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Science for Nonmajors . . . and Majors

Maureen Scharberg's letter "Importance of teaching" (24 May, p. 1086) appears to miss the point of the News article about the National Science Foundation (NSF) report (*1*) on undergraduate education (J. Mervis, 19 Apr., p. 345). The issue raised by the report has nothing to do with the "scholarship of teaching." Rather, it deals with the perceived need for university faculty to pay more attention, not to teaching, but to undergraduates who are *not* science majors.

NSF's education chief Luther Williams has pointed out that university structures that have evolved in response to an imperative to educate scientists cannot be expected to modify their emphases merely because NSF says to do so.

The situation is not an "either-or" criticism of doctoral institutions. Ample evidence exists for the importance of undergraduate education to doctoral faculty. Doctoral research does not preclude commitment to undergraduate education; it increasingly embraces it. The strength of the U.S. system of higher learning is its enormous scope and its wide-ranging diversity, offering settings for just about any type

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.

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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