

but there are strategic (policy) reasons why I believe that establishing a separate genome institute was inopportune. Congress has generally been sympathetic to NIH, but they have become understandably irritated at the cacophony, perceived as self-serving, that they have been hearing from different sectors of the scientific community. NIH should also get its act together and agree on priorities. Three priorities for NIH extramural funding exceed all others. First, there should be funding of individual investigator-initiated grants to about the 30th percentile. Currently, this means somewhere between 6000 or 7000 new and competing grants each year. The level should be stable, and it should be protected from earmarked projects. Second, there must be adequate funds for training of new scientists at the graduate and postdoctoral level. Third, there must be special attention to young scientists so that the best and brightest are launched on their independent careers as early as possible.

The genome project was the result of skillful persuasion on the part of distinguished scientists. I believe their resourcefulness was misdirected, and I regret that genome scientists did not see fit to add their powerful voices to strengthen the common enterprise with the full confidence that genome research would surely and inevitably progress right along with other exciting initiatives in the life sciences.

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Two serious distortions in Roberts' recent Research News article require comment. First, it was implied that critics of the Human Genome Initiative (HGI) who support mapping the human genome, or various of its other "short term goals," actually support the initiative. This is incorrect. The relatively inexpensive and comparatively modest-scaled effort to map the human genome does not justify a \$3-billion big science project; indeed, it is a project that can be completed without the need for any new "funding experiments." The predisposition of HGI supporters to claim that critics who support mapping have somehow diminished their opposition to the HGI is just one example of the distortion that the initiative's advocates have used in their campaign, a campaign whose tactics include the direct lobbying of Congress.

Many of us oppose brute force sequencing of the human genome because we believe it is an inefficient use of scarce research dollars. We have argued that biomedical research dollars are generally most efficiently spent on investigator-initiated research. We

believe that innovation from scientists in the field produces better science than does narrowly targeted, top-down directed big science projects like the HGI. This is a position taken by many responsible scientists.

I must also protest the implication in Roberts' article that the opposition can be divided into two camps—one position represented by Bernard Davis and the other by myself and Martin Rechsteiner. My substantive position is virtually indistinguishable from that of Davis. The difference is only one of tactics—Rechsteiner and I have advocated writing to Congress as the only effective response to the direct lobbying efforts of the initiative's advocates.

It is an ominous development for American science when those who carry out not only their right, but also their obligation, to speak our freely on issues of such importance to science and society are subjected to unseemly personal attack by the leading general science journal in the United States.

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## Aging Studies

The cellular-molecular approach to the problem of the causes of aging received almost all the attention in Ann Gibbons' article "Gerontology comes of age" (Research News, 2 Nov., p. 622), and only one other approach was mentioned. However, not all of us believe that the main causes of aging will be found by studies of the molecular biology of cells. Cells do not exist in isolation in the body, and their functions are regulated by circulating hormones, nutrients, immunologic agents, blood gases, pH changes, waste products of metabolism, hemodynamic fluxes, and cell-to-cell communication. Studies of cells in vitro can tell us only of intrinsic changes, but not of the many important external factors that control cellular functions.

The late Nathan Shock, dean of American gerontology, expressed the view that aging is regulated principally by the integrative mechanisms of the body (1), which means mainly the brain, the endocrine glands, and the immune tissues, or what is now collectively termed the neuroendocrinimmune system. This system acts in coordination to control and integrate all body functions. We and others have presented evidence that dysfunctions that develop with age in the hypothalamic portion of the brain, a critical component of the neuroendocrinimmune system, lead to declines in body functions and to

reduced ability to maintain body homeostasis (2). These result in cessation or reductions in reproductive functions, a decline in protein synthesis by body tissues (resulting from the decrease in growth hormone secretion), deficiencies in immune competence, and development of numerous mammary and pituitary tumors. Correction of neurotransmitter deficiencies in the hypothalamus of old rats has been shown to inhibit or reverse these and other aging declines in body functions and to even prolong their lifespan (2). The complex functions of the neuroendocrinimmune system cannot be explained by studying cells in vitro, least of all by studies of fibroblasts (connective tissue cells). I believe the knowledge gained thus far from studies of neuroendocrinimmune functions during aging has yielded far more basic information about the fundamental causes of aging than cellular studies have. They have also provided a basis for possible interventions to inhibit or reverse aging changes in elderly human subjects.

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## Adaptive Optics

M. Mitchell Waldrop, in his Research News article "Astronomers try to put Mauna Kea 'into space'" (31 Aug., p. 987), describes the growing interest in adaptive optics in astronomy, as shown by our recent work in France (1) and in Chile (2). Nevertheless, a few quotes might mislead the reader.

The current prototype, in work on the European Southern Observatory (ESO) 3.6-meter telescope, functions with reference stars as faint as magnitude 10 to 13 (3) and is still being improved. The Hawaii approach also uses a reference star. The practical method of deriving from it the wavefront instantaneous distortion differs from ours, but this minor difference is not related to the system's sensitivity, which is set only by the particular photon detector being used. Because the use of adaptive optics in astronomy is still in its infancy, the proper evaluation of its capabilities and limits is of prime importance. It would be correct to state that there is no universal value for the magnitude of a reference star needed to inform the "rubber mirror." It all depends on the degree of correction one wants to achieve or, to put it differently, on the fraction of the total light

one wants to put in the sharp, diffraction-limited image. This, in turn, depends on the wavelength of operation—changing by a factor of 20 from the visible to the infrared range at 10  $\mu\text{m}$ —and on the strength of the turbulence (the astronomical “seeing”). Limited correction in first-class sites in the near infrared may be possible with stars of magnitude 16 to 17, while more ambitious correction may require stars of magnitude 10 to 12. The former are available almost anywhere in the sky, while the availability of the latter is more restricted, but would lead to exceptional images. In the long run, artificial reference laser stars may allow these limitations to be overcome. According to a French saying, “Qui peut le plus peut le moins” (“One who can do the most can do the least”); the Hawaii approach aims, through innovative methods, to reach very faint stars with a limited correction. The European Very Large Telescope, while capable of a similar performance, has an approach that aims to make more ambitious corrections on particular astronomical sources whenever possible.

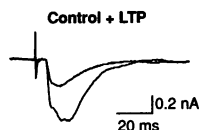
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2. European Southern Observatory, press release, May 1990.
3. F. Rigaut *et al.*, in preparation.

#### Correction

In reviewing the data in our Research Article “Comparison of two forms of long-term potentiation in single hippocampus neurons” (29 June, p. 1619) [*Science* **248**, 1619 (1990)], we discovered that we made an error in composing one of the figures. The trace examples in figure 4C (p. 1622) were said to be from the mossy fiber pathway to a single cell from the experiment summarized in 4B. The “during tetanus” trace was as stated, but the “control” and “LTP” traces were not from the same recording, as is obvious from close inspection of the waveform and latencies. We show below the “control” and “LTP” traces from that cell.



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**Erratum:** In the report “Toward protein tertiary structure recognition by means of associative memory Hamiltonians” by M. S. Friedrichs and P. G. Wolynes (20 Oct. 1989, p. 371), the second sentence of first full paragraph in the second column on page 372 was printed incorrectly. The sentence should have read, “The square well widths were set to  $0.5 \sqrt{|i-j| - 1} \text{ \AA}$ , where  $|i-j|$  is the sequence distance, and the depths . . . .”

**Erratum:** Equation 2 (p. 477) in the report “Ostwald ripening of clays and metamorphic minerals” by D. D. Eberl *et al.* (27 Apr. 1990, p. 474) was printed incorrectly. The correct equation appears below.

$$f(\omega) = \left[ \frac{1}{\omega \beta (2\pi)^{1/2}} \right] \times \exp \left\{ - \left( \frac{1}{2\beta^2} \right) [\ln(\omega) - \alpha]^2 \right\}$$

**Erratum:** In table 1 (p. 737) of the report “Peptide immunogen mimicry of a protein-specific structural epitope on human choriongonadotropin” by J.-M. Bidart *et al.* (11 May, p. 736), the peptide sequence corresponding to batch 4 should have read, “hCG $\beta$ (46-55)-hCG $\beta$ (106-116).”

## 1991 Bower Awards

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—Prof. Paul Christian Lauterbur, University of Illinois at Champaign-Urbana—magnetic resonance imaging.

—James Edward Burke, Chairman, Partnership for a Drug Free America, and Retired CEO of Johnson & Johnson—health related technological developments, and leadership in the war on drugs.

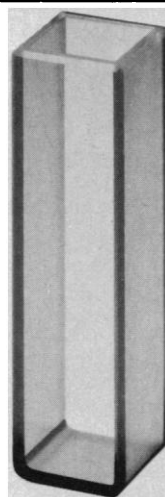
Awards will be announced in September and conferred in December in a major convocation. Nominations for both awards must be postmarked by April 17, 1991. For guidelines and official nomination forms please contact:

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