Biochemistry. In the article "Cloning of an intracellular receptor for protein kinase C: A homolog of the β subunit of G proteins" by Dorit Ron, Che-Hong Chen, Jeremy Caldwell, Lee Jamieson, Elisha Orr, and Daria Mochly-Rosen, which appeared in number 3, February 1, 1994, of *Proc. Natl. Acad. Sci. USA* (**91,** 839–843), the following correction should be noted. In Fig. 4*A*, page 842, the sequence for rPKC-δ should be corrected to Y-g-K-I-W-E (332–337).

Cell Biology. In the article "Interactions among members of the Bcl-2 protein family analyzed with a yeast two-hybrid system" by Takaaki Sato, Motoi Hanada, Sharon Bodrug, Shinji Irie, Natsuko Iwama, Lawrence H. Boise, Craig B. Thompson, Erica Golemis, Linda Fong, Hong-Gang Wang, and John C. Reed, which appeared in number 20, September 27, 1994, of *Proc. Natl. Acad. Sci. USA* (**91,** 9238–9242), the authors wish that the following correction be noted. The error is in the description of the Bax sequences expressed in yeast, which include the entire mouse Bax protein, amino acids 1–192. The corrected figure with legend is shown here.

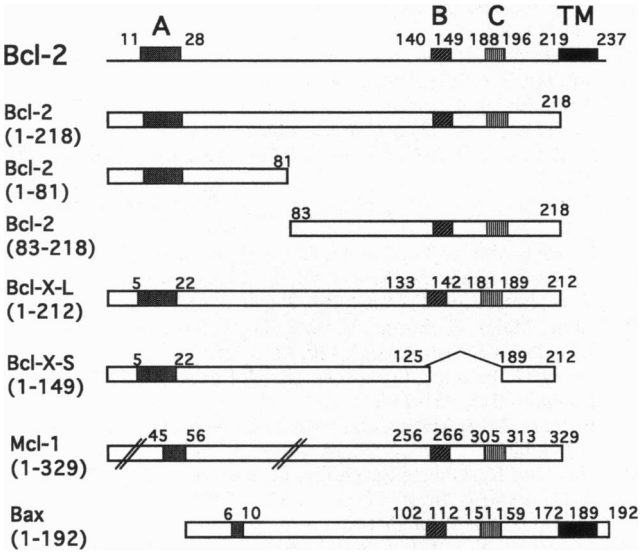

FIG. 1. Schematic depiction of regions of Bcl-2 and related proteins expressed as LexA or B42 fusion proteins. The structure of the human Bcl-2 protein is presented in linear form, indicating the three regions that are conserved among many of the family members (domains A, B, and C) and the corresponding amino acid positions. The transmembrane (TM) domain was excluded from all fusion proteins, except Bax, by introduction of a stop codon. The Bcl-X-S protein has a 63-aa deletion relative to Bcl-X-L because of a splicing event. The Mcl-1 protein is the longest of the family members (denoted by slashes).

Medical Sciences. In the article "Incipient and overt stages of neoplastic transformation" by Harry Rubin which appeared in number 25, December 6, 1994, of the *Proc. Natl. Acad. Sci. USA* (**91,** 12076–12080), the following correction should be made. In the Abstract, p. 12076, the sentences beginning on line 31 and ending on line 38 should read: The barely perceptible nature of the early morphological changes suggests an analogy to incipient neoplasia. Their graded nature, relatively high frequency, regularity of occurrence, and uniformity of appearance are characteristic of an epigenetic process which Foulds [Foulds, L. (1969) *Neoplastic Development, Vol. I* (Academic, New York), preface, and pp. 41–89] cited as the most plausible basis for incipient neoplasia.

Neurobiology. In the article "Two separate areas of the brain differentially guide the development of a song control nucleus in the zebra finch" by Eugene Akutagawa and Masakazu Konishi, which appeared in number 26, December 26, 1994, of *Proc. Natl. Acad. Sci. USA* (**91,** 12413–12417), the authors request that the following corrections be noted. In the Abbreviations footnote, RA should be defined as robust nucleus of the archistriatum. In the legend to Fig. 1, DLM should be defined as medial portion of the dorsolateral nucleus of the thalamus.

Neurobiology. Concerning the article "Advanced Maillard reaction end products are associated with Alzheimer disease pathology" by Mark A. Smith, Shinji Taneda, Peggy L. Richey, Satoshi Miyata, Shi-Du Yan, David Stern, Lawrence M. Sayre, Vincent M. Monnier, and George Perry, which appeared in number 12, June 7, 1994, of *Proc. Natl. Acad. Sci. USA* (**91,** 5710–5714), the authors request that the following be noted: Since publication it has come to our attention that a previous publication (1) should have been cited in our article. We acknowledge the contribution of this work in which the possible involvement of advanced Maillard reaction end products in Alzheimer disease was proposed.

1. Pappolla, M. A., Alzofon, J., McMahon, J. & Theodoropoulos, T. J. (1990) *Eur. Arch. Psychiatr. Neuro. Sci.* **239,** 314–319.