Botany. In the article "Isolation, sequence, and bacterial expression of a cDNA for (S)-tetrahydroberberine oxidase from cultured berberine-producing Coptis japonica cells" by Naosuke Okada, Nozomu Koizumi, Toshisori Tanaka, Hiroaki Ohkubo, Shigetada Nakanishi, and Yasuyuki Yamada, which appeared in number 2, January 1989, of Proc. Natl. Acad. Sci. USA (86, 534–538), the authors request that the following correction be noted. The amino acid sequence of part of a 28-kDa protein previously reported to copurify with (S)-tetrahydroberberine oxidase activity was determined, and this sequence was used to design oligonucleotides that were, in turn, used to isolate clones from a cDNA library. Upon further investigation, we found that the "purified" (S)-tetrahydroberberine oxidase was heavily contaminated with triosephosphate isomerase (EC 5.3.1.1) and that the sequence determined was actually that of C. japonica triosephosphate isomerase. Additionally, the DNA sequence of the cDNA clone pTHB201 (p. 536) encodes C. japonica triosephosphate isomerase rather than C. japonica (S)-tetrahydroberberine oxidase, and the RNA hybridization analysis (p. 535) detected triosephosphate isomerase mRNA. Data to support this correction has been presented elsewhere (1).

Medical Sciences. In the article "Point mutations define positions in HLA-DR3 molecules that affect antigen presentation" by Elizabeth Mellins, Benjamin Arp, Devinder Singh, Beatriz Carreno, Laura Smith, Armead H. Johnson, and Donald Pious, which appeared in number 12, June 1990, of Proc. Natl. Acad. Sci. USA (87, 4785–4789), the authors wish that the following corrections be noted. On page 4785, in line 8 under Materials and Methods, DRB3*0301 should be DRB3*0101. On page 4789, ref. 23 should be as follows:


Immunology. With regard to the article "Secretion and cell surface expression of IgG1 are impaired in human B lymphoblasts that lack HLA-A, -B, and -C antigens" by William J. Burlingham, Stephanie S. Ceman, and Robert DeMears, which appeared in number 20, October 1989, of Proc. Natl. Acad. Sci. USA (86, 8005–8009), the authors request that the following correction and retraction be noted. In Fig. 1, SDS/polyacrylamide gel electrophoresis was used to show that surface IgG(κ) was absent from the HLA-A/ B, -C "null" B-LCL mutant 721.221 (lane f) and the pHeBo vector-alone control transfectant (lane g) and was greatly diminished in the pHPT32 vector-alone control transfectant (lane j). In contrast, transfectant cell lines that expressed transgene-encoded HLA-A1 (lane i), -A2 (lane h), -B5 (data not shown), -B8 (lane l), or -C (lane k) also expressed membrane and secretory IgG. In addition, the amount of IgG secreted into the cell culture medium was undetectable or greatly diminished in 721 and the two vector-only control transfectants in comparison to transfectants expressing HLA-A, -B, or -C (Tables 2 and 3). The electrophoretic and secreted IgG observations were highly reproducible. Subsequent pulse-chase and nuclear run-off experiments by S.S.C. showed that the Igγ chain was not made and that the IgG gene was not transcribed in 721. These observations suggested the conclusion proposed in our publication, which was that expression of HLA-A, -B, or -C was needed for expression of the IgG γ gene. However, while data from one experiment that challenged this interpretation was inadvertently over-looked by the first author, the unusual nature of the findings did prompt continued analysis with regard to other possible explanations of the loss of Ig G expression. Subsequent work by S.S.C. now indicates that the proposed interpretation is erroneous. Abundant IgG was observed in newly thawed 721 cells that had been cryopreserved soon after isolation of the mutant. This suggested the possibility that expression of the IgG γ chain might have been lost subsequently for a reason unconnected with the loss of HLA-A, -B, and -C expression during the many doublings that preceded use of 721 in the described experiments. Indeed, newly performed transfers of the HLA-A2 and -B8 genes into IgG−721 did not restore IgG expression. Southern blotting analysis of BamHI-cut DNA with a probe for the IgG γ gene constant region showed that six bands were present in the parental cell line LCL 721 and in the IgG+ early isolate of 721 but that two of the bands were absent in the IgG− version of 721 that was used for our publication. Therefore, we now believe that spontaneous deletion of DNA of the functional Ig γ gene occurred during long-term propagation of mutant 721. One might imagine that the transfersents used for our publication were made at a time when the 721 population was a mixture of IgG+ and IgG- cells and that, by remarkable coincidence, all of the transfersents expressing HLA-A, -B, or -C were derived from IgG+ cells and the vector-alone controls from IgG− cells. According to this interpretation the vector-alone expressing transfersents should express as little Ig γ as 721 itself—i.e., none. The presence of some Ig γ chain (some of abnormal size) in the pHPT32 vector-alone control transfectant (Fig. 1, lane j, and Table 3) suggests the possibility that more than one kind of spontaneous event altering Ig γ expression had occurred during production of the transfectent cell lines used for our publication. We apologize for the mistake in interpretation and for any inconvenience our report may have caused.