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, Toshihiro 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).

1. Sato, F., Fitchen, J., Takeshita, N., Hashimoto, T., Okado, N. & Yamada, Y. (1990) *Agric. Biol. Chem.* **54**, 2189–2191.

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:

Bodmer, J. G., Heyes, J. M. & Lindsay, J. (1984) in *Histocompatibility Testing*, eds. Albert, E. D., Baur, M. P. & Mayr, W. R. (Springer, Berlin), pp. 432–438.

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 DeMars, 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 transferent (lane g) and was greatly diminished in the pHPT32 vector-alone control transferent (lane j). In contrast, transferent 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 .221 and the two vector-only control transferents in comparison to transferents expressing HLA-A, -B, or -C (Tables 2 and 3). The electrophoretic and secreted Ig observations were highly reproducible. Subsequent pulse–chase and nuclear run-off experiments by S.S.C. showed that the Ig γ chain was not made and that the Ig gene was not transcribed in .221. 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 Ig γ gene. However, while data from one experiment that challenged this interpretation were inadvertently overlooked 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 γ expression. Subsequent work by S.S.C. now indicates that the proposed interpretation is erroneous. Abundant IgG was observed in newly thawed .221 cells that had been cryopreserved soon after isolation of the mutant. This suggested the possibility that expression of the Ig 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 .221 in the described experiments. Indeed, newly performed transfers of the HLA-A2 and -B8 genes into IgG^- .221 did not restore IgG expression. Southern blotting analysis of *Bam*HI-cut DNA with a probe for the Ig γ-gene constant region showed that six bands were present in the parental cell line LCL 721 and in the IgG^+ early isolate of .221 but that two of the bands were absent in the IgG^- version of .221 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 .221. One might imagine that the transferents used for our publication were made at a time when the .221 population was a mixture of IgG^+ and IgG^- cells and that, by remarkable coincidence, all of the transferents 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 transferents should express as little Ig γ as .221 itself—i.e., none. The presence of some Ig γ chain (some of abnormal size) in the pHPT32 vector-alone control transferent (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 transferent cell lines used for our publication. We apologize for the mistake in interpretation and for any inconvenience our report may have caused.