Medical Sciences. In the article "Segregation of atrial-specific and inducible expression of an atrial natriuretic factor transgene in an *in vivo* murine model of cardiac hypertrophy" by Howard A. Rockman, Robert S. Ross, Adrienne N. Harris, Kirk U. Knowlton, Mark E. Steinhelper, Loren J. Field, John Ross, Jr., and Kenneth R. Chien, which appeared in number 18, September 1991, of *Proc. Natl. Acad. Sci. USA* (**88**, 8277–8281), the authors request that the following correction be noted. The molecular size of the c-*jun* mRNA (2.5 and 2.7 kb) is higher than indicated in Fig. 2*B*. The c-*jun* mRNA actually migrates between the 28S and 18S subunits.

Medical Sciences. In the article "Accumulation of glycerophosphocholine (GPC) by renal cells: Osmotic regulation of GPC:choline phosphodiesterase" by K. Zablocki, S. P. F. Miller, A. Garcia-Perez, and M. B. Burg, which appeared in number 17, September 1, 1991, of *Proc. Natl. Acad. Sci. USA* (**88**, 7820–7824), the following correction should be noted. Due to a printer's error, Figs. 2 and 3 were exchanged. The correct Figs. 2 and 3 are as follows.

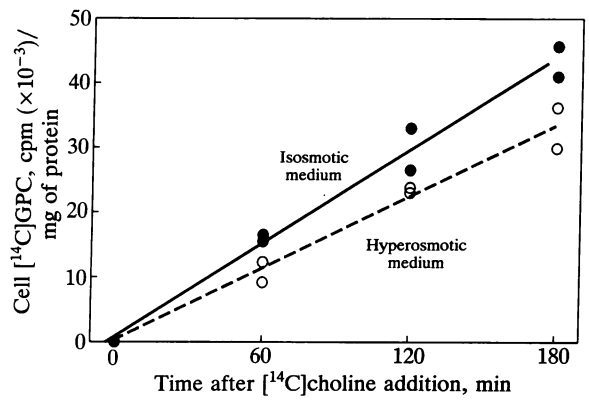

FIG. 2. Incorporation into MDCK cell GPC of ^{14}C from [^{14}C]choline added to the medium. GPC was 28.6 ± 7.5 mmol/kg of protein (n = 3) in cells kept in isosmotic medium and 105 ± 21 mmol/kg of protein (n = 5) 2 days after switching to hyperosmotic medium.

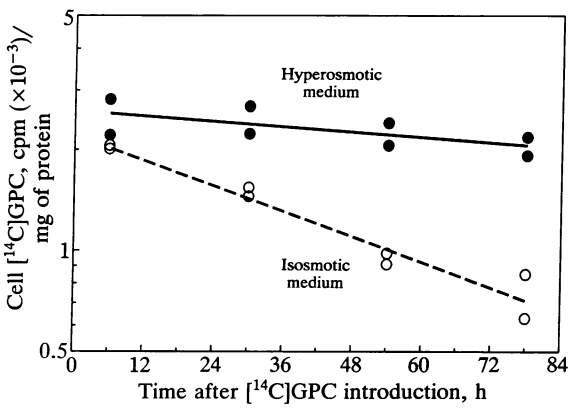

FIG. 3. Effect of NaCl and urea on the rate of disappearance of intracellular [^{14}C]GPC. MDCK cells were grown in isosmotic medium (315 mosmol/kg) and then switched to hyperosmotic medium (715 mosmol/kg with added NaCl and urea) or maintained in the isosmotic medium, as a control. Seven days after the switch, [^{14}C]GPC was loaded into the cells by hypotonic shock. Then, cell [^{14}C]GPC was measured at the intervals shown. GPC was 62 ± 10 mmol/kg of protein (n = 7) in cells kept in isosmotic medium and 163 ± 22 (n = 7) in hyperosmotic medium. Cell GPC did not change significantly from 6 to 72 h in either condition.

Biochemistry. In the article "Splicing-defective mutants of the yeast mitochondrial *COXI* gene can be corrected by transformation with a hybrid maturase gene" by Paul Q. Anziano and Ronald A. Butow, which appeared in number 13, July 1991, of *Proc. Natl. Acad. Sci. USA* (**88**, 5592–5596), the authors request that the following correction be noted. We have subsequently found that the pMIT transformant DNA containing the fusion maturase gene, pMIT-aI1/2, contains upstream of that gene an 800-base-pair fragment of pGEM-7Zf DNA instead of a portion of the noncoding *COXI* transcription initiation region (Fig. 1). The conclusions of the paper, however, are unaffected: the downstream maturase gene present on the pMIT DNA transformed into mitochondria is functionally expressed in pairwise crosses and is active as a trans-acting splicing factor that is readily detectable among the products of mitochondrial protein synthesis.

Biochemistry. In the article "Identification and characterization of a ouabain-like compound from human plasma" by J. M. Hamlyn, M. P. Blaustein, S. Bova, D. W. DuCharme, D. W. Harris, F. Mandel, W. R. Mathews, and J. H. Ludens, which appeared in number 14, July 1991, of *Proc. Natl. Acad. Sci. USA* (**88**, 6259–6263), the authors request that the following correction be noted. On page 6263, paragraph four incorrectly implies that Masugi *et al.* (38, 39) measured ouabain in human plasma. The plasma immunoreactivity measured by Masugi *et al.* was subsequently described as being due to an unstable lipid [Masugi, F., Ogihara, T., Sakaguchi, K., Tomii, A., Hasegawa, T., Chen, Y., Azuma, M. & Kumahara, Y. (1988) *J. Hypertens.* **6**, Suppl. 4, S351–S353]. Ouabain is not an unstable lipid. Thus, the assay of Masugi *et al.* was not specific for ouabain.

Medical Sciences. In the article "Activation of protein kinase C by elevation of glucose concentration: Proposal for a mechanism in the development of diabetic vascular complications" by Tian-Shing Lee, Kirstie A. Saltsman, Hiromi Ohashi, and George L. King, which appeared in number 13, July 1989, of *Proc. Natl. Acad. Sci. USA* (**86**, 5141–5145), we request that the following be noted. Because of questions raised concerning Fig. 1 (*Left*), Table 1, and values of diacylglycerol, phosphatidylinositol, phosphatidylinositol 4-phosphate, and phosphatidylinositol 4,5-bisphosphate in Fig. 3, we now believe that these figures and this table contain data derived from experiments that have flaws in methodologies, execution, and data analysis. As a result, the quantitative aspect of these figures and table are not valid. In further experiments, however, qualitatively similar results have been observed [Fig. 1 *Left*, Shiba *et al.* (1); Table 1, T. Inoguchi, T. Shiba, and G.L.K. (unpublished work)]. Therefore, we believe the mechanism of activation of hyperglycemia causing activation of protein kinase C as postulated in Fig. 2 is consistent with the new data.

1. Shiba, T., Bursell, S. E., Clermont, A., Sportsman, J. R., Heath, W. F. & King, G. L. (1991) *Invest. Ophthalmol. Vis. Sci.* **32** (4), 785.

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