Biochemistry. In the article "Guanidine hydrochloride stabilization of a partially unfolded intermediate during the reversible denaturation of protein disulfide isomerase" by Nihmat A. Morjana, Barry J. McKeone, and Hiram F. Gilbert, which appeared in number 6, March 15, 1993, of *Proc. Natl. Acad. Sci. USA* (**90**, 2107–2111), the authors request the following correction be noted. On p. 2107, right column, lines 22–29 should read as follows: The two forms of PDI appear to represent dimeric and tetrameric species in which a metastable tetramer without intermolecular disulfides is present (M. Kruzel and H.F.G., unpublished observations). Overnight incubation of the preparation at pH 7.5 and 22°C results in essentially complete (>90%) conversion of the tetramer to the dimer; under the conditions of our experiments, the PDI is dimeric.

This correction was necessitated by subsequent experiments that detected an error in the original calibration of the gel-filtration column (M. Kruzel and H.F.G., unpublished observations). Since the thermodynamic parameters are independent of protein concentration, this does not alter the conclusions regarding the stabilization of the folding intermediate by guanidine hydrochloride.

Biochemistry. In the article "Alanine scanning site-directed mutagenesis of the zinc fingers of transcription factor ADR1: Residues that contact DNA and that transactivate" by Sushil K. Thukral, Michael L. Morrison, and Elton T. Young, which appeared in number 20, October 15, 1991, of *Proc. Natl. Acad. Sci. USA* (**88**, 9188–9192), an error was made in sequencing or otherwise characterizing one of the ADR1 mutant plasmids. This mutant, in which glutamate 117 had been mutated to alanine (E117A), has a 10- to 20-fold reduced DNA binding activity whereas it was reported in Table 1, line 15, column 6, that its DNA binding activity was equivalent to that of wild-type ADR1 (1.0). We request that the value 0.05 be inserted at this position in Table 1. Thus, our interpretation that glutamate 117 in finger one is essential for a step in transcription that occurs after DNA binding is unwarranted. We regret this error and any inconvenience that it may have caused the scientific community.

Cell Biology. In the article "Calmodulin-binding domain of recombinant erythrocyte β-adducin" by Dominick A. Scaramuzzino and Jon S. Morrow, which appeared in number 8, April 15, 1993, of *Proc. Natl. Acad. Sci. USA* (**90**, 3398–3402), it is requested that the following corrections be noted. (*i*) On p. 3399, line 15 of the legend to Fig. 2 should read "was a variable amount of proteolytic products between 41 . . .". (*ii*) On p. 3400, the calmodulin-binding sequence shown in Fig. 3 should read "k^{425}qqke . . .".

Medical Sciences. In the article "Platelet-derived growth factor B chain promoter contains a cis-acting fluid shear-stress-responsive element" by Nitzan Resnick, Tucker Collins, William Atkinson, David T. Bonthron, C. Forbes Dewey, Jr., and Michael A. Gimbrone, Jr., which appeared in number 10, May 15, 1993, of *Proc. Natl. Acad. Sci. USA* (**90**, 4591–4595), the authors request that the following correction be noted. Table 2 on p. 4594 should read as follows.

Table 2. Conservation of SSRE core binding sequence in promoters of genes responsive to shear stress

Gene	Species	Location, nt	Sequence	Ref(s).
PDGF-B	Human	−125	TCTCAGAGACC	20, 21
PDGF-B	Feline	−125	TCTCAGAGACC	23
PDGF-B	Murine	−125	TCTCAGAGACC	24
tPA	Human	−945	GGTCTGGTCTC	26
tPA	Rodent	−252	CCTTTGAGACC	26
tPA	Murine	−252	CCTTTGAGACC	26
TGF-β1	Human	−1219	CCTGGGGTCTC	27
TGF-β1	Murine	−401	ACGTCGGTCTC	28
TGF-β1	Murine	−1314	GTGAAGAGACC	28
ICAM-1	Human	−644	GTGGTGAGACC	29

Location of the SSRE core binding sequence is indicated relative to the initiation of transcription. Note that the core binding sequence (GAGACC) of the SSRE identified in this study and its complementary sequence (GGTCTC) are conserved among the PDGF-B promoters of several species and in unrelated genes that are also responsive to shear stress in vascular endothelium.