

Biochemistry. In the article “Characterization of residual structure in the thermally denatured state of barnase by simulation and experiment: Description of the folding pathway” by Chris J. Bond, Kam-Bo Wong, Jane Clarke, Alan R. Fersht, and Valerie Daggett, which appeared in number 25, December 9, 1997, of *Proc. Natl. Acad. Sci. USA* (**94**, 13409–13413), one of the authors regrets that she inadvertently omitted references to the computer program and protein potential function that the authors used for their simulations of barnase cited above. The following sentence should have been the first sentence of the *Methods* section: Molecular dynamics simulations were performed with the program ENCAD (44) and the potential energy function of Levitt *et al.* (45).

- 44. Levitt, M. (1990) ENCAD, Energy Calculations and Dynamics. (Molecular Applications Group, Palo Alto, CA and Yeda, Rehovot, Israel).
- 45. Levitt, M., Hirshberg, M., Sharon, R. & Daggett, V. (1995) *Comp. Phys. Commun.* **91**, 215–231.

Biochemistry. In the article “Negative control of bacterial DNA replication by a cell cycle regulatory protein that binds at the chromosome origin” by Kim C. Quon, Bing Yang, Ibrahim J. Domian, Lucy Shapiro, and Gregory T. Marczyński, which appeared in number 1, January 6, 1998, of *Proc. Natl. Acad. Sci. USA* (**95**, 120–125), the authors wish to note that the institutional affiliations in the author line were incorrectly attributed. The correct affiliations are as follows. Bing Yang and Gregory T. Marczyński are at McGill University in Montreal, and Kim C. Quon is now at the Netherlands Cancer Institute.

Biochemistry. In the article “The 30-kDa C-terminal domain of the RecB protein is critical for the nuclease activity, but not the helicase activity, of the RecBCD enzyme from *Escherichia coli*” by Misook Yu, Jehanne Souaya, and Douglas A. Julin, which appeared in number 3, February 3, 1998, of *Proc. Natl. Acad. Sci. USA* (**95**, 981–986), the following correction should be noted. The symbols in the graph (Fig. 3C) were identified incorrectly in the manuscript. The corrected legend and graph with accompanying symbols are printed below.

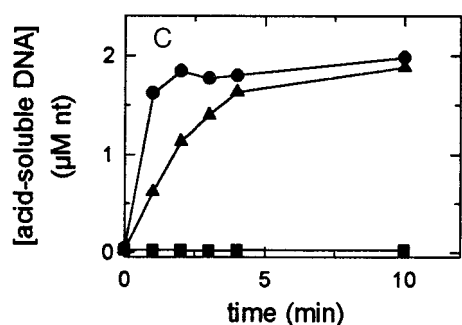


FIG. 3. Nuclease assays. All reaction mixtures contained buffer A and 10 mM MgCl₂. (A) ssDNA exonuclease assay with 100 nM single-stranded linear 5′-³²P-labeled 25-mer as the substrate. (B) ssDNA endonuclease assay using 6 nM single-stranded circular M13 phage DNA as the substrate. (C) dsDNA exonuclease assay using *Ava*I-digested [³H]pTZ19R. Each reaction contained 2.3 µM (nucleotides) double-stranded [³H]pTZ19R, 250 µM ATP, and 9 nM RecB1-929CD (■), 0.2 nM RecBCD (▲), or 9 nM RecBCdD (●).

Biochemistry. In the article “*Escherichia coli* RNA polymerase terminates transcription efficiently at rho-independent terminators on single-stranded DNA templates” by Susan M. Uptain and Michael J. Chamberlin, which appeared in number 25, December 9, 1997, of *Proc. Natl. Acad. Sci. USA* (**94**, 13548–13553), the authors request that the following correction be noted. It is critical that the bands in lanes 6 and 8 of Fig. 3 indicated by the T7Te arrow be visible. The existence of these terminated bands is a major point on which the conclusions of the paper depend. Therefore, to enhance their visibility, Fig. 3 and its accompanying legend are reprinted below with greater contrast.

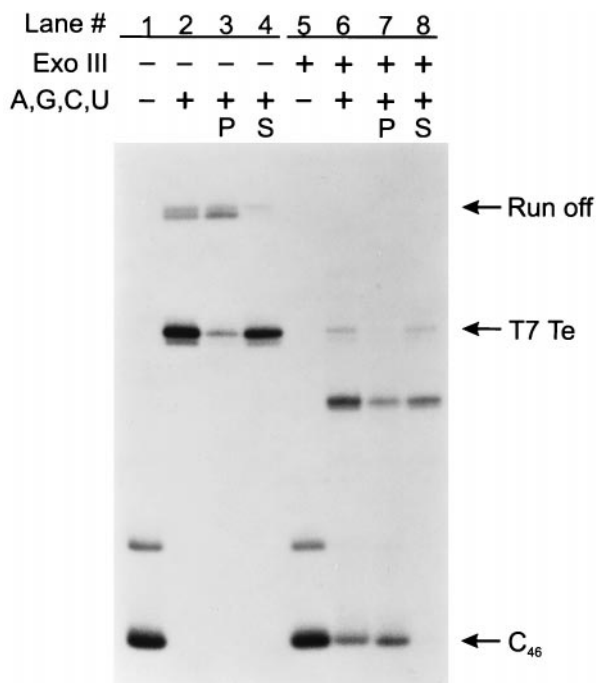


FIG. 3. Assaying for intrinsic transcript termination at T₇Te on ssDNA. C₄₆ complexes bound to Ni²⁺-NTA agarose in TGK-B₄₀M₄ were chased with 500 µM ATP, 500 µM GTP, 500 µM CTP, and 500 µM UTP for 10 min at 37°C in the presence of rifampicin at 20 µg/ml and yeast *Torula* RNA at 0.8 mg/ml. The lanes are assigned as for Fig. 2A. The T₇Te terminator is at positions +95 and +96, whereas transcription to the end of the DNA template generates a run-off RNA of +146 nucleotides. C₄₆ complexes in lanes 5–8 were digested with Exo III at 5,000 units/ml for 5 min at 37°C. Unlike C₄₇, some of the C₄₆ complexes failed to resume elongation after treatment with Exo III (see lanes 6 and 7). The mechanism of this inactivation is unknown but similar observations have been made by others (29, 30).

Cell Biology. In the article “Scavenger receptor class B, type I (SR-BI) is the major route for the delivery of high density lipoprotein cholesterol to the steroidogenic pathway in cultured mouse adrenocortical cells” by Ryan E. Temel, Bernardo Trigatti, Ronald B. DeMattos, Salman Azhar, Monty Krieger, and David L. Williams, which appeared in number 25, December 9, 1997, of *Proc. Natl. Acad. Sci. USA* (**94**, 13600–13605), the following correction should be noted. The equation on page 13601 should be as follows:

$$P_{\text{total}} = \frac{[P_{\text{max}}][\text{HDL}]}{K_{\text{HA}} + [\text{HDL}]} + C[\text{HDL}]$$

Cell Biology. In the article "Identification of a family of low-affinity insulin-like growth factor binding proteins (IGFBPs): Characterization of connective tissue growth factor as a member of the IGFBP superfamily" by Ho-Seong Kim, Srinivasa R. Nagalla, Youngman Oh, Elizabeth Wilson, Charles T. Roberts, Jr., and Ron G. Rosenfeld, which appeared in number 24, November 25, 1997, of *Proc. Natl. Acad. Sci. USA* (94, 12981–12986), the authors request that the following corrections be noted. In Fig. 8, the units on the scale should indicate the number of substitution events, rather than "million years." The lengths of the branches represent the relative distance between the sequences of mammalian IGFBPs compared in this figure. In the *Discussion*, the statement "The dendrogram depicted in Fig. 8 indicates that, based upon structural similarities, all ten members of the superfamily can be traced back to an ancestor gene 60 million years ago" should read: "The dendrogram depicted in Fig. 8 indicates that all ten members of the superfamily share a common ancestral gene based upon their sequence similarities."

Neurobiology. In the article "Hair cell-specific splicing of mRNA for the α_{1D} subunit of voltage-gated Ca^{2+} channels in the chicken's cochlea" by Richard Kollmar, John Fak, Lisa G. Montgomery, and A. J. Hudspeth, which appeared in number 26, December 23, 1997, of *Proc. Natl. Acad. Sci. USA* (94, 14889–14893), the authors wish to note that the quality of reproduction of Fig. 1 was below standard. In all three panels,

Immunology. In the article "Parasite-mediated nuclear factor κ B regulation in lymphoproliferation caused by *Theileria parva* infection" by Guy H. Palmer, Joel Machado, Jr., Paula Fernandez, Volker Heussler, Therese Perinat, and Dirk A. E. Dobbelaere, which appeared in number 23, November 11, 1997, of *Proc. Natl. Acad. Sci. USA* (94, 12527–12532), the following correction should be noted. The concentration of *N*-acetylcysteine used in the experiments was 30 mM, not 25 μ g/ml as erroneously reported on page 12528, lines 14 and 15 of the paragraph entitled "Cell Lines and Cultures" in the *Materials and Methods* section.

the middle parts were affected. Specifically, the reverse (white-on-black) type denoting exons 9a, 22a, and 30a was illegible; parts of the arrows that represented primers such as F₉ were missing; and the outlines of several of the boxes that depicted exons such as 9, 10, and 20 were defective. The figure and its legend are reproduced below.

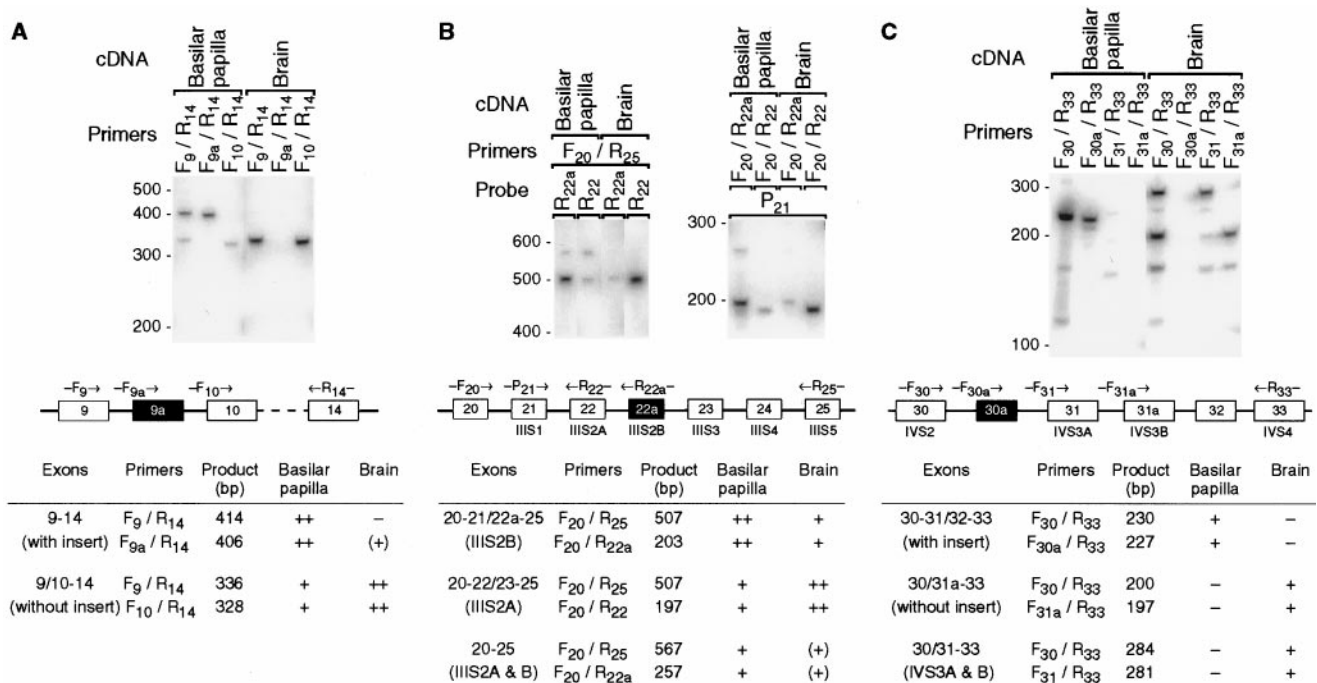


FIG. 1. Alternative splicing of the α_{1D} mRNA in the basilar papilla and the brain. (A) Southern blot of PCR products amplified with primers flanking the insert in the I-II loop (exon 9a). Marker sizes in base pairs are indicated on the left. The diagram below of the putative genomic structure (not drawn to scale) depicts exons as rectangles, introns as horizontal lines, and PCR primers as arrows. To amplify all isoforms together, we used primers F₉ and R₁₄. To amplify rare isoforms without interference from more abundant ones, we used exon-specific primers: primer F_{9a} binds across the splice junction of exons 9 and 9a, and primer F₁₀ binds across that of exons 9 and 10. The table at the bottom lists product size and occurrence for each splice variant and primer pair. ++, abundant; +, detectable; (+), barely so; -, not detectable. (B) Same as A, but for the alternative IIIS2 segment (exon 22a). Note the abundance in the basilar papilla of mRNAs with exons for both IIIS2 segments. (C) Same as A, but for the insert in the IVS2–3 loop (exon 30a). Primer F_{30a} binds across the splice junction of exons 30 and 30a, primer F₃₁ binds across that of exons 30 and 31, and primer F_{31a} binds across that of exons 30 and 31a. For the basilar papilla, the lengths of even the minor products were consistent only with splice isoforms containing exon 30a; for the brain, they were consistent only with isoforms lacking exon 30a. Note the abundance in the brain of mRNAs with exons for both IVS3 segments.