Biochemistry. In the article "Structure, characterization, and expression of the rat oxytocin receptor gene" by Florence Rozen, Caterina Russo, Denis Banville, and Hans H. Zingg, which appeared in number 1, January 1995, of *Proc. Natl. Acad. Sci. USA* (**92,** 200–204), the authors request that the sequence shown in Fig. 2 of the original report should be corrected as follows. As shown schematically here in Fig. 1*A*, nucleotides 1452–1504 of the original report have to be replaced by a novel sequence shown here in Fig. 1*B*. This correction pertains to the 5′-flanking region of the gene and does not affect the coding region nor any of the conclusions drawn in the original report. The fact that there was a missing sequence element was discovered and pointed out to us by Tracy L. Bale in the laboratory of Daniel M. Dorsa, Departments of Psychiatry and Behavioral Sciences and Pharmacology, University of Washington, Seattle. As illustrated in Fig. 1*A*, the novel sequence has to be inserted at the location of a dinucleotide repeat, $(GT)_{26}$, located 89 nucleotides 5′ to the main transcriptional initiation site. Resequencing of a newly generated phage subclone as well as Southern blot and PCR analyses (not shown) confirmed that the sequence presented here is indeed part of the genomic sequence. Since this novel sequence element is itself flanked by two dinucleotide repeats, $(GT)_{20}$ and $(GT)_{24}$, respectively, a likely explanation is that this segment was spliced out during subcloning due to recombination between the two dinucleotide repeats. This idea is further supported by the fact that dinucleotide repeats that have the potential of forming Z-DNA structures have been shown to enhance recombination in extrachromosomal DNA up to 20-fold (1). Despite the recurrence of dinucleotide repeats around chromosomal rearrangement breakpoints, their role in mediating recombination on intact chromosomes remains, however, uncertain (2).

We thank Tracy L. Bale and Daniel M. Dorsa for pointing out the error and for their help and collaboration in its correction.

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Chemistry. In the article "Sequence verification of human creatine kinase (43 kDa) isozymes by high-resolution tandem mass spectrometry" by Troy D. Wood, Lorenzo H. Chen, Camille B. White, Patricia C. Babbitt, George L. Kenyon, and Fred W. McLafferty, which appeared in number 25, December 5, 1995, of *Proc. Natl. Acad. Sci. USA* (**92,** 11451–11455), the authors request that the following corrections be noted. The Asp vs. His difference in the human brain creatine kinase sequences of ref. 18 vs. ref. 17 is at residue 295, not 296, as this is the N-terminal Met-truncated isoform. Its predicted masses should be 42,533, not 42,622 (ref. 18), and 42,355, not 42,699 (ref. 19); however, these also are different from the value 42,512 (ref. 17) that agreed with our mass spectrometry measurement. The multiply-charged isotopic distributions in Fig. 3, *A* and *C*, are of $(M + 33H)^{33+}$.

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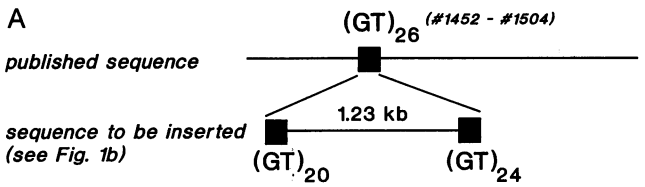

B

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          10         20         30         40         50
          |          |          |          |          |
   1 GTGTGTGTGT GTGTGTGTGT GTGTGTGTGT GTGTGTGTGT GATCAAACTA
  51 CCTAAAATAA AGATTTCTCT CAAGGCTGAT AGGAGGAACA GGTATCTCCA
 101 ATTCATCCTT AATAGGAATG TGAGCAAAGC CATTCCAAAG AATGAACTTA
 151 CCTGAGCTCT CTGAAAAAAA AAAAAGATGT AAAGAAAAAA AAAAAACCAA
 201 AAAGGCGAAG AAATGCAACT TTATTAAAAA TGTACTCAGG GAAAAACAAA
 251 CGTCACTTTC CAAGGTTCCA GTATCTCTGA GAACGTAAAG AGCTAAACCA
 301 AGAAAGGAAG AAGTTAGGTG GGTTGTTTGC GAGTTTCAAG CCACTAATGA
 351 AATAAATCTG ACCTAAGGGT TAGCTGACCA GGCTCTCTGG GATCGCCGCG
 401 GGCGACTTGC TCTGCCCTCT AGCGGACATA CGTGGTGCTG CAGCCTCCGG
 451 CTCCAGCTGT GGACCCGCAC CTTCAGACAG CACCGCGTGC TGACGCCGGC
 501 GGCTCACAGC CTCGCTGGCT CTCTCTTGTC TTCCTACGGC ATCCAGCCAC
 551 CAGCAAAGCC GCAAGCTAGG GGATTTCAAA GACGCCGAGA TCTACCCCGT
 601 GGTAGCACGG AGGGTGGCGC CAGCAGCTCT CCCATCCTGG GCGGAGAGTC
 651 CACGCATCTC TGAGGCCATC CACTGCAGTG CAACTTCCCC GTTGGCAAGG
 701 CAACTTTGGG CTAACCTGCA GTCTCGCTTT AGGTGGCTGG GACCTTTGGA
 751 CCGTGGGGAC AGGATGGCTA GGACCTACCT GTAGGACCAT CAGGTTCGGA
 801 GGGAAGGATC TGGCGCGTCT CGCACCTCTG GTTCCCGGAG GAGGTGCTCA
 851 GGATGTTCTG AGAGCCTGAA AAGCTTGGAC GAAGATGCAA AGTCTCTGAC
 901 TGTCCGGGCT CCTGGGCTGG GGGGATTCCT GTGAAAGGCG TCCCCTGGGG
 951 TCTCTGGCAC AGCAGCCTTG CGTTGCGCTG GGGTAGGAGG TAAGGTGGGG
1001 GCACTACGCT TGTAGGCAGG AGTCCCTCCG GAAGCAAGCG GCGTCGGGAA
1051 GTTCTGGAAG CCGGGGTTGA GTCTTGGAAG CAGGAGGTGA AGAAGGATGG
1101 GCTTGAGCTG GAGCTTTTTG GGGGGACCCC AGGAAGATGT ACTTGGAGTA
1151 AAGGCTATTA AGATGGGGGC TGGCTTCGAT CCCCTAGGAG GTGGTTGTTC
1201 TGTGTCCGGA TGGGATAGGT ATAGGTATAG GTTTGGGGGT TCTCTGCAGG
1251 AGAGGCCGCT GGTGTGTGTG TGTGTGTGTG TGTGTGTGTG TGTGTGTGTG
1301 TGTGTGTGT
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FIG. 1. (*A*) Schematic diagram for sequence correction. (*B*) The sequence shown replaces nucleotides 1452–1504 of Fig. 2 of the original report.

Pharmacology. In the article "Tityustoxin Kα blocks voltage-gated noninactivating K^+ channels and unblocks inactivating K^+ channels blocked by α-dendrotoxin in synaptosomes" by Robert S. Rogowski, Bruce K. Krueger, John H. Collins, and Mordecai P. Blaustein, which appeared in number 3, February 1, 1994, of *Proc. Natl. Acad. Sci. USA* (**91,** 1475–1479), the authors request that the following be noted. The molecular weight of tityustoxin Kα (TsTX-Kα) is 3941, and not 3997 as stated in the legend to Fig. 5.