Correction

MEDICAL SCIENCES. For the article "A rescue factor abolishing neuronal cell death by a wide spectrum of familial Alzheimer's disease genes and Aβ" by Yuichi Hashimoto, Takako Niikura, Hirohisa Tajima, Takashi Yasukawa, Haruka Sudo, Yuko Ito, Yoshiko Kita, Masaoki Kawasumi, Keisuke Kouyama, Manabu Doyu, Gen Sobue, Takashi Koide, Shoji Tsuji, Jochen Lang, Kiyoshi Kurokawa, and Ikuo Nishimoto, which appeared in number 11, May 22, 2001, of *Proc. Natl. Acad. Sci. USA* (**98,** 6336–6341), the following should be noted. First, the GenBank accession no. of HN cDNA is AY029066, not YA029066. Second, the authors admit that the paper lacked sufficient discussion regarding the implications of the findings. There is no proof that the HN cDNA represents a gene, that its origin is nuclear, or that the HN peptide is produced *in vivo*. The information that the long HN cDNA sequence is virtually identical to mitochondrial rRNA should have been put in the *Discussion* rather than published as supplementary material. The *Discussion* should have contained the following:

The long cDNA [1,567 bp including a poly(A) tail] containing the HN ORF is >99% identical (1548/1552) to positions 1679–3230 of mitochondrial DNA (GenBank accession no. AB055387). Because mitochondrial DNA positions 1667–3224 code for mitochondrial 16S rRNA, and mitochondrial 16S rRNA has a short poly(A) tail during transcription (1), the virtual identity of the long HN cDNA to mitochondrial DNA indicates that HN cDNA is mitochondrial 16S rRNA with a poly(A) tail. This makes it unlikely that the peptide encoded by the ORF in HN cDNA is naturally produced. Further, the 75-bp HN ORF is separated from the 5′ end of the long HN cDNA by a 950-bp region containing at least seven ORFs, each with a stop codon. This makes it even more unlikely that HN peptide is produced from the long HN cDNA. It should also be noted that mitochondria-like nuclear sequences occur commonly as pseudogenes (2). Finally, the HN peptide lacks the characteristic N-terminal signal sequence of secreted peptides, although we suggest that a signal peptide-like function may be encoded in the primary sequence of HN peptide. For all of these reasons, it is unlikely that the HN ORF leads to production of the predicted peptide *in vivo*.

Nonetheless, it remains possible that HN cDNA represents a nuclear transcribed mRNA and that the HN peptide is a natural product. Long regions of the HN cDNA are >99% identical to certain registered human mRNAs [1545/1553 at positions 14–1580 of FLJ22981 fis cDNA (AK026634), 925/929 at positions 1–929 of FLJ22517 fis cDNA, 914/919 at positions 1348–2266 of FLJ20341 fis cDNA, and 345/346 at positions 1–346 of PNAS-32 mRNA]. PNAS-32 mRNA is actually expressed to produce NB4 apoptosis-related protein, showing that this mRNA is transcribed from a nuclear gene. In addition, HN cDNA is highly similar to regions of more than 1,000 bp on human chromosomes [positions 245364–244075 of chromosome 11 draft sequence (92%, 1198/1290), positions 65752–66775 of chromosome X draft sequence (95%, 974/1025), and positions 687598–688608 of chromosome 5 draft sequence (93%, 954/1016)]. Also, the HN ORF has a Kozak-like sequence, although it is not canonical.

1. Baserga, S. J., Linnenbach, A. J., Malcolm, S., Ghosh, P., Malcolm, A. D., Takeshita, K., Forget, B. G. & Benz, E. J., Jr. (1985) *Gene* **35,** 305–312.

2. Herrnstadt, C., Clevenger, W., Ghosh, S. S., Anderson, C., Fahy, E., Miller, S., Howell, N. & Davis, R. E. (1999) *Genomics* **60,** 67–77.

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