Applied Biological Sciences. In the articles "4,5-Dianilinophthalimide: A protein-tyrosine kinase inhibitor with selectivity for the epidermal growth factor receptor signal transduction pathway and potent *in vivo* antitumor activity" by Elisabeth Buchdunger, Uwe Trinks, Helmut Mett, Urs Regenass, Marcel Müller, Thomas Meyer, Elaine McGlynn, Lorenzo A. Pinna, Peter Traxler, and Nicholas B. Lydon, which appeared in number 6, March 15, 1994, of *Proc. Natl. Acad. Sci. USA* (**91,** 2334–2338) and "Selective inhibition of the platelet-derived growth factor signal transduction pathway by a protein-tyrosine kinase inhibitor of the 2-phenylaminopyrimidine class" by Elisabeth Buchdunger, Jürg Zimmermann, Helmut Mett, Thomas Meyer, Marcel Müller, Urs Regenass, and Nicholas B. Lydon, which appeared in number 7, March 28, 1995, of *Proc. Natl. Acad. Sci. USA* (**92,** 2558–2562), the undersigned authors wish to note the following. "In the interest of scientific accuracy and our mutual desire with the editors that only high quality and reliable data are published, we regret to say that the *in vivo* data for the articles above are not considered reliable. We wish to retract the data until the *in vivo* experiments have been repeated and reevaluated. The validity of *in vitro* and cellular data is not in doubt. We very much regret this situation."

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Genetics. In the article "Mutations in mitochondria cytochrome *c* oxidase genes segregate with late-onset Alzheimer disease" by Robert E. Davis, Scott Miller, Corinna Herrnstadt, Soumitra S. Ghosh, Eoin Fahy, Leslie A. Shinobu, Douglas Galasko, Leon J. Thal, M. Flint Beal, Neil Howell, and W. Davis Parker, Jr., which appeared in number 9, April 29, 1997, of *Proc. Natl. Acad. Sci. USA* (**94,** 4526–4531), the authors wish to note the following: "We recently reported several DNA polymorphisms in cytochrome *c* oxidase subunits 1 and 2 genes, the proportions of which were increased in AD patients relative to controls (1). As we noted in our paper, an unusual feature of these polymorphisms was that these changes also were present in chimpanzee and other great ape mtDNA. We also suggested that these polymorphisms were disease related. Subsequent work by two groups, however, has concluded that these polymorphisms are not present in the mitochondrial genome. These polymorphisms appear to represent a fragment of mtDNA that has been incorporated into the nuclear genome early in evolution (2, 3). Given the similarities in these polymorphisms across the great apes, we also were concerned that our results might be confounded by the presence of a nuclear pseudogene. We were deterred initially from this conclusion because of the unusual length of the gene fragment, as well as its absence of stop codons, multiple deletions, and insertions, and other features that are characteristic of nuclear pseudogenes. In subsequent work, however, we have firmly established that these polymorphisms are present in nuclear pseudogenes. A detailed report of our findings has been submitted for publication.

These nuclear mitochondrial-like DNA (numtDNA) fragments are largely in frame with mitochondrial nucleotide positions 3911–9755. We have confirmed that this nuclear sequence is present in human ρ° SH-SY5Y and ρ° A431 cell lines lacking mtDNA. It is flanked by non-mtDNA sequences, and it does not appear to be expressed. Moreover, we found the sequence in nuclear DNA isolated from a bacterial artificial chromosome library. It was absent in immune-purified mitochondria from SH-SY5Y cell lines and blood cells. Evolutionary analysis suggests that these numtDNAs may have been translocated to the nucleus $\approx$1.2 million years ago.

The presence of these numtDNA sequences is unlikely to cause the CO defects in cells or in humans. However, we believe that the elevation of the ratio of this pseudogene relative to authentic mtDNA in the blood of AD patients still holds. As suggested by Hirano *et al.* (3), this elevation most likely reflects decreased mtDNA content arising from decreased extraction of mtDNA from AD blood relative to control blood by using our heat lysis procedure."

1. Davis, R. E., Miller, S., Herrnstadt, C., Ghosh, S. S., Fahy, E., Shinobu, L. A., Galasko, D., Thal, L. J., Beal, M. F., Howell, N., *et al.* (1997) *Proc. Natl. Acad. Sci. USA* **94,** 4526–4531.
2. Wallace, D. C., Stugard, C., Murdock, D., Schurr, T. & Brown, M. D. (1997) *Proc. Natl. Acad. Sci. USA* **94,** 14900–14905.
3. Hirano, M., Shtilbans, A., Mayeux, R., Davidson, M. M., DiMauro, S., Knowles, J. A. & Schon, E. A. (1997) *Proc. Natl. Acad. Sci. USA* **94,** 14894–14899.

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