localities, taken individually or in combination, to be aligned on a 3,800 Myr isochron. Rather, they seem to be ancient rocks of uncertain age (probably >,3100 Myr), which, locally, have been partially or nearly completely reset during a phase of metamorphism about 2,500 Myr BP. Ages in the vicinity of 3,300 Myr BP are suggested by zircon data[2,3].

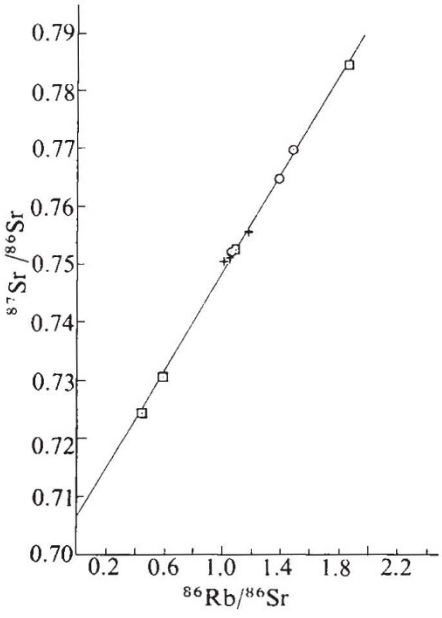
J. S. FARHAT
G. W. WETHERILL
Department of Geophysics and Space Physics,
University of California,
Los Angeles, California 90024

1 Goldich, S. S., and Hedge, C. E., *Nature*, **252**, 467 (1974).
2 Farhat, J. S., and Wetherill, G. W., *Abstr. A. Mt. geol. Soc. Am.*, **6**, 729 (1974).
3 Goldich, S. S., Hedge, C. E., and Stern, T. W., *Bull. geol. Soc. Am.*, **81**, 3671 (1970).
4 Brooks, C., Hart, S. R., and Wendt, I., *Rev. Geophys. Space Phys.*, **10**, 551 (1972).

GOLDICH AND HEDGE REPLY: We wish to correct a procedural error in our original article[1] relating to the age of 3,950±70 Myr (2σ) with an initial ratio of 0.698±0.004. The use of 2σ is not proper because of the small number (5) of samples. The recalculated results give an age of 3,950±130 Myr with an initial ratio of 0.698±0.002 (95% confidence level). We included "sample 339 from the Morton Gneiss as a guide to the initial ratio of 0.700 and an age of 3,800 Myr."

Rb–Sr analyses of the tonalitic phase of the Morton Gneiss of Lund[2] have progressed to a 7-point isochron with an apparent age of 3,630±60 Myr with an initial ratio of 0.6994±0.0004 (95% confidence level). The data will be published in the near future. Additional work is also in progress in the Granite Falls area. The fact that two points do not lie on the isochron in our original diagram means that those samples have had somewhat different histories and is not evidence that an age has not been measured. In this regard some of Farhat and Wetherill's data are relevant.

Fig. 1 Rb–Sr isochron diagram of the massive granitic phase of the Montevideo Gneiss. Localities KA-209 (□) and MV-100 (+) from Farhat and Wetherill[3]; ○, new data.



Four samples of the 'red phase' from locality KA-209 and three samples from locality MV-100, together with three unpublished analyses by Hedge of the more massive granitic phase, define an isochron (Fig. 1) with an apparent age of 3,000±90 Myr with an initial ratio of 0.7065±0.0016 (95% confidence level). Three different localities are represented, but in spite of Farhat and Wetherill's reasoning we feel that the linearity is more than simply fortuitous. The 3,000-Myr age in the Granite Falls area was noted in our original paper[1]. It may represent a regional high-grade metamorphic event that affected both gneissic and more massive phases of the Montevideo Gneiss, but it is possible, if not more likely, that it dates the time of intrusion of granitic magma in a foliated terrain of tonalitic to granodioritic rocks. Both phases were later affected by at least two younger events. We are dealing with a complex geological history in which the effects of interaction between magma and country rock as well as regional metamorphism must be considered.

The 2,470 Myr isochron (MV-102) also is useful, and is not unexpected as we have similar unpublished data. The Montevideo Gneiss has undergone intensive shearing and hydrothermal alteration, and some of the apparent ages in the range 1,850–2,500 Myr BP (ref. 4) may be related to this type of activity. We suggest that Farhat and Wetherill have not considered the variety and complexity of the geological processes and that neither the 3,800 Myr isochron[1] nor the 3,000 Myr isochron are necessarily 'fallacious isochrons'[5].

Northern Illinois University
and
US Geological Survey

1 Goldich, S. S., and Hedge, C. E., *Nature*, **252**, 467 (1974).
2 Lund, E. H., *Bull. geol. Soc. Am.*, **67**, 1475 (1956).
3 Farhat, J. S., and Wetherill, G. W., *Nature*, **000**, 000 (1975).
4 Goldich, S. S., Hedge, C. E., and Stern, T. W., *Bull. geol. Soc. Am.*, **81**, 3671 (1970).
5 Goldich, S. S., *Abstr. geol. Soc. Am.*, **4**, (5), 322 (1972).

Inhibition of Na, K-activated ATPase and release of neurotransmitters

GILBERT *et al.*[1] have raised the question whether a change in the local environment of the neuronal membrane Na, K-activated ATPase (ATPase) could result in a change both in its activity and in its conformation such that an increase in exocytosis occurs; in this manner the activity of ATPase in the nerve terminals and the regulation of neurotransmitter release would be coupled. It has also been suggested that the physiological release of acetylcholine from guinea pig ileum and rat brain cortical slices may be mediated through inhibition of the neuronal membrane ATPase[2,3]. We have shown that several procedures which are known to inhibit ATPase cause a marked release of noradrenaline from sympathetic nerve terminals of the cat spleen[4,5]. Since physiological release of neurotransmitters is dependent on calcium entry into the neuron[6,7] the question arises whether calcium entering the neurone during depolarisation may inhibit ATPase[8], so that inhibition of ATPase is the underlying mechanism in the physiological release of neurotransmitters.

It is generally believed that physiological release of noradrenaline from sympathetic nerve terminals occurs by exocytosis. This belief is mainly based on the demonstration of proportional release of noradrenaline and the soluble form of the vesicular enzyme dopamine-β-hydroxylase (DBH) in response to stimulation of sympathetic nerves[9]. If the physiological release of noradrenaline is mediated through ATPase inhibition, then procedures leading to a decrease in ATPase activity should cause a proportional release of noradrenaline and DBH. We demonstrated however that one such procedure, namely sodium deprivation, caused a pronounced release of noradrenaline (800 ± 85 ng g^{-1}, $n=3$) from cat spleen slices yet the release of DBH over the background level was barely detectable[10]. This result indicates that at least noradrenaline release from sympathetic nerves by sodium deprivation is due to some mechanism other than exocytosis. If the mechanism of release of noradrenaline by sodium depletion is presumed to be due to inhibition of ATPase[4], then physiological release of neurotransmitters by exocytosis is not simply the result of ATPase inhibition.

A. G. GARCIA
S. M. KIRPEKAR
Departamento de Farmacologia,
Facultad de Medicina,
Valladolid, Spain

Received August 22; accepted September 5, 1975.

1 Gilbert, J. C., Wyllie, M. G., and Davison, D. V., *Nature*, **255**, 237–238 (1975).
2 Paton, W. D. M., Vizi, E. S., and Zar, M. A., *J. Physiol., Lond.*, **215**, 819–848 (1971).
3 Vizi, E. S., *J. Physiol., Lond.*, **226**, 95–117 (1972).
4 Garcia, A. G., and Kirpekar, S. M., *Br. J. Pharmac.*, **47**, 729–747 (1973).
5 Garcia, A. G., and Kirpekar, S. M., *J. Physiol., Lond.*, **235**, 693–713 (1973).
6 Rubin, R. P., *Pharmac. Rev.*, **22**, 389–428 (1970).
7 Kirpekar, S. M., *Prog. Neurobiol.*, **4**, 163–212 (1975).
8 Skou, J. C., *Biochim. biophys. Acta*, **23**, 394–401 (1957).
9 Weinshilboum, R. M., Thoa, N. B., Johnson, D. G., Kopin, I. J., and Axelrod, J., *Science*, **174**, 1349–1351 (1971).
10 Garcia, A. G., and Kirpekar, S. M., *J. Pharmac. exp. Ther.*, **192**, 343–350 (1975).