of 10^{-3} M) might still be effective for the synaptic inactivation of GABA. The data of Fonnum *et al.*[4], who estimated the concentration of GABA in 'gabergic' nerve terminals to be "at least 60 mM, probably over 100 mM", seem to support this suggestion.

As to the other comments raised, we are obviously aware of the fact that our present data disagree with previous experiments conducted in our own as well as in other laboratories. This did not seem to us a strong enough reason not to publish the data. Moreover, the disagreement is often more apparent than real, and in most cases concerns the interpretation of the data rather than the data themselves. For example, the finding[5] that cortex slices (which contain structures other than nerve endings) show a net uptake of GABA after 30 min of incubation in the presence of a concentration (200 μM) at which high affinity uptake[3,5,6] and exchange[1] are virtually saturated, does not disprove that homoexchange can account for a large part of the initial rate of accumulation of radioactivity by synaptosomes incubated with 1 or 10 μM ^{3}H-GABA.

Similarly, our data on glycine (detectability of homoexchange in spinal but not in cortical synaptosomes) are not disproven by those of Aprison and McBride[7] who did show a net uptake of unlabelled glycine, but did not assess the contribution of exchange to the accumulation of radioactive glycine. Moreover, these authors may have somewhat overestimated net uptake, as they did not consider the increase in glycine concentration that might have occurred in the tissue on incubation without added glycine[8].

The similarity between radiochemical and chemical uptake of 20 μM GABA that we found in a previous study[9] is difficult to explain at present. In this type of experiment the choice of a correct type of control is very critical, and we are now trying to determine whether an overestimation of net uptake might have resulted from the type of control chosen.

The results of the experiments in which the tissue concentration of GABA was artificially increased[9,10] could have several explanations: for example, the increased intracellular concentration of GABA may cause a concomitant inhibition of net uptake and a stimulation of homoexchange. In appropriate experimental conditions we have shown that the accumulation of radioactive GABA is increased in synaptosomes prepared from aminooxyacetic acid-treated rats.

We take this opportunity to mention that one of the data reported in Fig. 2 of our letter[1] was wrong, because of a trivial technical error. The figure showed a rapid and massive depletion of synaptosomal GABA content and the absence of any detectable homoexchange on superfusion with a sodium-free medium. Our latest data (Raiteri *et al.*, unpublished) indicate that superfusion with sodium-free medium (NaCl replaced by sucrose) does not cause a significant increase in the spontaneous release of ^{3}H-GABA from purified synaptosomes. They do confirm, however, that, in the absence of sodium—homoexchange is 95–100% inhibited. Therefore, the error does not invalidate the conclusion that absence of homoexchange could account for what looks like inhibition of the high affinity uptake of GABA.

Laboratorio di Biologia Cellulare, CNR and Istituto di Farmacologia
Università Cattolica, Rome, Italy

1 Levi, G., and Raiteri, M., *Nature*, **250**, 735 (1974).
2 Henn, F. A., and Hamberger, A., *Proc. natn. Acad. Sci. U.S.A.*, **68**, 2686 (1971).
3 Iversen, L. L., and Johnston, G. A. R., *J. Neurochem.*, **18**, 1939 (1971).
4 Fonnum, F., Grofovà, I., Rinvik, E., Storm-Mathisen, J., and Walberg, F., *Brain Res.*, **71**, 77 (1974).
5 Iversen, L. L., and Neal, M. J., *J. Neurochem.*, **15**, 1141 (1968).
6 Levi, G., and Raiteri, M., *Life Sci.*, **12** (1), 81 (1973).
7 Aprison, M. H., and McBride, W. J., *Life Sci.*, **12**, 449 (1973).
8 Bradford, H. F., and Thomas, A. J., *J. Neurochem.*, **16**, 1495 (1969).
9 Levi, G., Bertollini, A., Chen, J., and Raiteri, M., *J. Pharmac. exp. Ther.*, **188**, 429 (1974).
10 Snodgrass, S. R., and Iversen, L. L., *J. Neurochem.*, **20**, 431 (1973).

Playing possum

In his interesting article on the opossum α haemoglobin chain sequence, Stenzel[1] states that 'a selectionist interpretation of the more rapid rate of molecular evolution in a living fossil is possible' because we may expect more rapid molecular evolution 'accompanying slow morphological changes because the reduced number of loci undergoing substitutions may sustain more intense selection per locus'.

Perhaps this is so, but selectionists can cite scripture for their own purpose. Neutralists, however, are delighted to find that this molecule of a living fossil underwent changes at least as rapidly as the homologous molecules in highly-evolved species. This supports the thesis that 'there seems to be considerable latitude at the molecular level for random genetic changes that have no effect on the fitness of the organism' (ref. 2).

Thomas H. Jukes
University of California, Berkeley

1 Stenzel, P., *Nature*, **252**, 62 (1974).
2 King, J. L., and Jukes, T. H., *Science*, **164**, 788 (1969).

Matters arising

Matters Arising is meant as a vehicle for comment and discussion about papers that appear in *Nature*. The originator of a Matters Arising contribution should initially send his manuscript to the author of the original paper and both parties should, wherever possible, agree on what is to be submitted. Neither contribution nor reply (if one is necessary) should be longer than 300 words and the briefest of replies, to the effect that a point is taken, should be considered.

Sunspot cycle periodicities

The Maximum Entropy Spectral Analysis technique of Burg (unpublished) and Cohen and Lintz[1] has led to the discovery of periodicities in sunspot data and an ability to predict ionospheric reflectivity and climatic features. But the theory that a planetary influence affects sunspot variations[2–4] still cannot be ruled out, as is shown by a further analysis of the data of ref. 1.

I accept the 11.05 ± 1.5, 9.8 ± 0.1, and 8.15 ± 0.15 yr terms found by Cohen and Lintz but wish to choose another way of describing the cause of their 89.6 yr peak. The proposed fundamental beat tone of 179 yr just does not appear in their periodogram nor is it the beat period suggested. At about one half that value (also the true value of the beat period they have proposed) there is a fairly broad resonance. But 89.6 yr (really more like 86 skewed) is the value one obtains when oppositions and conjunctions of Uranus and Neptune are considered as a possible cause. The relative phase of these two planets is at quadrature during the peak sunspot maxima of 1778, 1864 and 1950 and in conjunction or opposition at the least sunspot maxima of 1821, 1907 and 1993 (predicted). I found the same periodicity but different significant phase relationships when I compared the relative positions of Uranus and Neptune to peak periods of energy released by terrestrial earthquakes[2]. Because of the special phase relationships between the sunspot and planetary data, I feel the 86 yr 'tone' is really due to Uranus and Neptune.

Interestingly, the other periodic terms which Cohen and Lintz found can be explained as the resonance of the following planetary mean motions: (1) $J + S + U - N = 1/8.06$ yr; (2) $J + U + N = 1/9.78$ yr; (3) $J + U - N = 1/11.09$ yr.

John P. Bagby
Dept 6504, Bell & Howell Co., 7100 McCormick Road, Chicago, Illinois 60645

1 Cohen, T. J., and Lintz, P. R., *Nature*, **250**, 398–399 (1974).
2 Bagby, J. P., *The Moon*, **6**, 398–404 (1973).
3 Bigg, E. K., *Astr. J.*, **72**, 463 (1967).
4 Wood, K. D., *Nature*, **240**, 91–93 (1972).