to have demonstrated the existence of a mechanism, presumably enzymatic, causing the rapid degradation of fragments *in vivo*, and discuss at some length the physiological implications of this finding. Neither the authors nor the reviewer consider an alternative explanation, that the fragments might have been released from the bacteria into the medium and in this way have escaped detection by the methods used. There is in fact some evidence for this explanation, from earlier experiments with alkaline phosphatase nonsense mutants of *E. coli*; phosphatase fragments were found to be rapidly released into the medium, an average time of about 20 minutes being required for a fragment to appear in the medium after being synthesized intracellularly[4,5]. The behaviour of phosphatase fragments could represent a special case, because the phosphatase enzyme in a standard strain of *E. coli* is localized in the region between the outer cell wall and the inner membrane (the periplasmic space); consequently phosphatase fragments might escape from a cell more readily than fragments of other proteins. Nevertheless, in the absence of evidence to the contrary, the intracellular disappearance of fragments of any protein might result, not from degradation, but rather from the passage of the fragments into the surrounding medium. Until this possibility has been tested, the conclusion that *E. coli* protein fragments are degraded *in vivo* must be regarded as speculative.

Yours faithfully,

ALAN GAREN

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1 Goldschmidt, R., *Nature*, **228**, 1151 (1970).
2 Platt, T., Miller, J. H., and Weber, K., *Nature*, **228**, 1154 (1970).
3 *Nature*, **228**, 1137 (1970).
4 Suzuki, T., and Garen, A., *J. Mol. Biol.*, **45**, 549 (1969).
5 Natori, S., and Garen, A., *J. Mol. Biol.*, **49**, 577 (1970). Reference to the loss of phosphatase fragments from cells is specifically made on page 587 of the second article, and the experimental details have been submitted for publication in *J. Mol. Biol.*

THIS letter has been shown to our correspondent, who replies :

The idea that cells might extrude unwanted nonsense fragments is appealing, but seems unlikely to apply for β-galactosidase and lactose repressor mutants. Neither of these proteins is located in the periplasmic space; this must imply that a special (enzyme) system would be needed to transport the nonsense fragments out of the cell. This seems more complicated than merely degrading the fragments, and would in any case demand the type of recognition mechanism which the authors discussed to discriminate mutant from normal proteins. An extrusion mechanism does not, therefore, seem to possess any theoretical advantage, and experimentally Goldschmidt was able to detect and identify a protein fragment which is probably produced by the postulated endopeptidase degradation of the β-galactosidase nonsense fragment.

Neuromythology?

SIR,—Is the large daily loss of neurones from the brain, which figures in all the textbooks and provides the basis for Dawkins's[1] ingenious model of memory, as well established as he suggests? I write because the matter, which was the subject of a fair amount of controversy at a recent American research course on ageing, is of considerable importance for models of psychological and psychiatric ageing as well.

Leaving aside insect work, the prime authority for a massive loss is Brodie[2], whom Dawkins quotes, though the original mammalian claim was made by Hatai[3] for the rat, and repeated by Vogt and Vogt[4]. The loss of Purkinje cells from rat brain found by Inukai[5] has not been discovered in ageing hamsters[6]. With regard to the cortex, which would appear to be the critical site for Dawkins's purpose, some loss is reported[7] but in the rat it appears to be trifling[8]. The most recent title indicates no loss whatever with age from the human cochlear nucleus[9].

In view of the importance of the subject and the categorical nature of the textbook statement, the matter of cell loss with age among fixed postmitotics is due for careful re-examination, in spite of the tiresome task of cell counting which it involves. It would be of interest to know whether studies of this kind in mammals are already in progress.

Yours faithfully,

ALEX COMFORT

Director of Research, Gerontology, University College London

1 Dawkins, R., *Nature*, **229**, 118 (1971).
2 Brodie, H., *J. Comp. Neurol.*, **102**, 511 (1955).
3 Hatai, S., *J. Comp. Neurol.*, **12**, 107 (1902).
4 Vogt, C., and Vogt, O., *Nature*, **158**, 304 (1946).
5 Inukai, T., *J. Compar. Neurol.*, **45**, 1 (1928).
6 Wilcox, H. H., *J. Gerontol.*, **11**, 442 (1956).
7 Wright, E. A., and Spink, J. M., *Gerontologia*, **3**, 277 (1959).
8 Brizzee, K. R., Sherwood, N., and Timiras, P. S., *J. Gerontol.*, **23**, 289 (1968).
9 Konigsmark, B. W., and Murphy, E. A., *Nature*, **228**, 1355 (1970).

ESRO Satellite

SIR,—It was not our intention to imply that a satellite needs to be stabilized to the same accuracy with which one desires to locate the X-ray sources. There is, however, a relation between the two as illustrated by the example of Sco X-1 given by Dr Gursky[1]. As he correctly states, the requirement is to know, with high precision, the attitude, at the same time maintaining a stability commensurate with the field of view of the instrument. For the HEAO mission to which Dr Gursky refers (that carrying modulation collimators, not the large X-ray telescope planned for a later mission) he claims a limiting accuracy in location of several arc seconds.

If this can be achieved, the two missions would give complementary results in the location of sources since the occultation satellite, although in principle capable of higher precision, locates a smaller fraction of the observed sources. On the other hand, an important characteristic of the occultation method is that the angular dimensions and relative structure of extended sources can be obtained with even higher accuracy than that of location, down to small fractions of an arc second.

We should finally note that the terms "complex" and "expensive" are, of course, relative rather than absolute, and should be read in the context of the ESRO budget which does not at present envisage satellites of the HEAO type.

Yours faithfully,

J. ORTNER

The ESRO Mission Definition Group for the Highly Eccentric X-ray Astronomy Lunar Occultation Satellite

1 Gursky S., H., *Nature*, **228**, 1121 (1970).

Neural Nomenclature

SIR,—In 1933, Dale[1] suggested the terms "adrenergic" and "cholinergic" for the two types of autonomic nerve fibre known at that time. He introduced these terms "to assist clear thinking, without committing us to precise chemical identifications, which may be long in coming".

In the early 1960s powerful nerves were found to supply that gut which were neither cholinergic nor adrenergic[2-4]. Since then, there have been a number of reports concerning details of their distribution, structure and function[5-7], but their description as non-adrenergic, non-cholinergic nerves is clumsy and somewhat negative. Evidence has recently been presented that the transmitter substance released from these nerves may be ATP or some related purine nucleotide[8]. It would therefore seem suitable, for the same reasons put forward by Dale in 1933, to propose that the new nerves be termed "purinergic".

Yours faithfully,

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1 Dale, H. H., *J. Physiol.*, **80**, 10 (1933)
2 Burnstock, G., Campbell, G., Bennett, M. and Holman, M. E., *Nature*, **200** 581 (1963).
3 Burnstock, G., Campbell, G., Bennett, M., and Holman, M. E., *Intern. J. Neuropharmacol.*, **3**, 163 (1964).
4 Burnstock, G., Campbell, G., and Rand, M. J., *J. Physiol.*, **182**, 504 (1966).