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OSBORNE AND BUTLER REPLY—Our conclusion[1] that a catecholamine (probably dopamine) is continuously released from the glomus cells during normoxia, and that the rate of release decreases during hypoxia, was based on the following: (1) glomus cells store large amounts of catecholamines, in particular, dopamine[2]; (2) catecholamines, especially dopamine, suppress or block the chemoreceptor discharge[3,4]; and (3) α-adrenoreceptor blocking agents greatly increase receptor firing rate (up to tenfold)[3,4] during normoxia and the effectiveness of the these blocking agents decreases during hypoxia[3].

It has since been indicated that there is a high turnover rate of catecholamines in the glomus cells, and that it seems likely that these cells release dopamine at a high rate[5]. This catecholamine activity by the glomus cells is envisaged to occur in normoxic conditions. Indeed, the metabolism of catecholamines requires the presence of molecular oxygen[6]. These observations suggest that as the oxygen availability decreases, the metabolism of catecholamines declines.

In the above article[7], the phrase "the increase in fluorescence in untreated carotid bodies after hypoxia" is puzzling in view of the facts that Mills and Slotkin[8] describe a decrease in catecholamine content of the carotid body during hypoxia, and Sampson *et al.*[9] did not in fact make the straightforward comparison between the amounts of catecholamines present in the glomus cells of untreated normoxic and hypoxic carotid bodies. We do not therefore accept that these two papers[8,9] contain unequivocal evidence regarding the effect of hypoxia on the release of catecholamines from the glomus cells of the carotid body.

Furthermore, Mills and Slotkin[8] state that the decrease in catecholamine content of the carotid body during hypoxia is largely mediated by way of the sinus nerve, whereas Sampson *et al.*[9] report that the catecholamine content of the glomus cells of the carotid body was higher in those organs with an intact sinus nerve. In an attempt to reconcile these apparent differences, Mills *et al.*[7] state more precisely that Mills and Slotkin[8] were referring to noradrenaline and adrenaline only, whereas Sampson *et al.*[9] measured total catecholamines. They now conclude that dopamine levels are increased within the glomus cells, whereas noradrenaline and adrenaline are released in response to efferent nerve activity. Whether this effect is mediated through a direct supply to the glomus cells or via the vasculature is not discussed by these authors, but it does raise the important issue regarding which catecholamine(s) is the major agent of chemoreceptive inhibition.

As has been shown[5,10,11], and as pointed out here, dopamine seems to be the favoured candidate.

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Spectacle haloes

THE nuclear geophysical enigma of the ^{210}Po haloes[1] is quite fascinating, but the explanation put forward on the basis of a novel form of ^{206}Pb is not easy either to understand or to believe.

If no primordial lead is present, all of the ^{206}Pb, ^{207}Pb and ^{208}Pb found in ordinary U–Th haloes will be derived from polonium α decay. To explain all of the polonium haloes observed by Gentry[1–3] without apparent α-emitting precursors it is not useful to assume new types of stable lead daughter products but rather some unknown, long lived isomers either of ^{218}Po and ^{210}Po or of β-emitting precursors of these. The haloes may be explicable without such an assumption. What is needed is that in a usual halo the polonium isotopes must decay while still in the uranium inclusion from which they are derived, whereas in the enigmatic halo they must together have migrated by at least hundreds of micrometres from their ancestral uranium.

Since the enigmatic halo has an intensity comparable with that of an ordinary halo, it must be attributable to the decay of quantities of polonium corresponding to the total output of a normal, halo-producing, uranium inclusion for at least tens of millions of years. That is a very long period compared with the half lives of isotopes of any elements other than uranium and thorium themselves. Conditions must, therefore, have been such as to allow migration from the original parent sources for very long periods.

The most likely migrant would seem to be lead, since emanation or polonium would give larger haloes than are seen. In that case there would be ^{210}Pb with a half life of 22 yr in one series and ^{211}Pb with a half life of 38 min in the other. Further, the ^{232}Th series, which eventually produces the observed ^{208}Pb, has ^{212}Pb with a half life of 10.6 h as a carrier, though it has no polonium with a half life of longer than 0.16 s. I do not know the temperature required for that migration of lead at a suitable rate, but it is, presumably, fairly high. Geological evidence would be interesting: Gentry himself has described[2] short fission tracks which suggest that some mica matrices may have been at a high temperature for a long time.

I do not claim that this explanation is highly probable—the events to be explained are rare—but Gentry's explanation would seem to require a higher order of improbability. Both ^{218}Po and ^{210}Po would need to have unknown precursors with half lives of a few hundred million years; lead or bismuth precursors would presumably have crystallised out with observable quantities of stable isotopes of the same elements. Finally, Gentry has also found[2,3] a variety of dwarf or other haloes for which he assumes different, otherwise unknown, α-emitters. All of those α-emitters apparently have mutually similar half lives or they would either have decayed too soon or would still be around in observable amounts. One such unknown isomer would be unlikely enough; so many would seem very close to impossible.

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GENTRY REPLIES—The crucial question is whether the polonium atoms which formed the polonium haloes were secondarily derived from the decay of uranium and thorium (see, for example, ref. 1) or from unknown isomers[1], or whether they were primordial.

The isomer hypothesis[1] is one way that polonium haloes could have been explained. My present view is that experimental results have ruled out the isomer hypothesis.

As for the postulate that uranium daughter activity provides a source of polonium atoms, I agree that in special