suture will be further south. Fourth, geophysical evidence strongly suggests an Upper Triassic–Lower Jurassic Gondwana model in which the Falkland Plateau as part of the South American plate borders the African plate[12–14]. This model is consistent with Truswell's considerations and with the hypothesis that the Falkland islands were to the north of the inferred subduction zone. The position of the subduction zone with respect to Patagonia is unclear. If it was situated to the east of Patagonia, the suture could lie in the basement structures in the continental edge and in the Malvinas Basin[15]. The suture could then cross the continent in the Upper Permian Patagonian Massif[8] or the Middle Triassic Deseado Massif[16]. Geological evidence, however, strongly supports the existence of a subduction zone that bordered the western rim of Patagonia from Devonian to Triassic times[7,17].

Fifth, from the Gondwana model it is clear that parts of Patagonia and the Antarctic Peninsula formed the southern land mass that approached southern Africa during Palaeozoic times in a process peripheral to the main Gondwana land mass. Even this accreted land mass should show affinities with Gondwanaland from at least Lower Carboniferous times.

This first order extension of our very elementary model inferring a continental collision origin for the Cape Fold Belt shows that the constraints proposed by Truswell can indeed be reconciled with a continent–continent (or at least a continent–island arc) collision south of Africa.

Our explanation of the origin of the Beattie magnetic anomalies is more contentious and should perhaps be related to processes causing the accretion of the present-day basement in the Karroo and further south rather than to processes causing the formation of the fold belt.

National Physical Research Laboratory, Pretoria, 0001, South Africa and PO Box 43, Half Way House, 1685, South Africa

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Chocolate, β-phenethylamine and migraine re-examined

ON the basis of an industrial analytical report indicating that chocolate might contain as much as 3 mg of β-phenethylamine (PEA) per 2-ounce bar, Sandler *et al.*[1] administered 3 mg of this amine to each of 36 chocolate-sensitive migraine-prone subjects and found that 18 suffered attacks within 12 h, as opposed to six attacks with placebo.

Having previously developed a sensitive procedure for the analysis of PEA in urine[2], we applied it with minor modifications to the analysis of one US brand of milk chocolate. We found only 21 μg per 2-ounce bar, or about $^1/_{150}$ of that quoted above. Various brands and types of chocolate were then examined.

In the method, 1-^{14}CPEA, 8.86 mCi mmol^{-1}, was used as an internal standard. About 10^5 d.p.m. were added to 1–2 g chocolate melted in 5 ml water. With some samples, 1–50 μg of unlabelled PEA was also added in tests to demonstrate recovery. Briefly (see ref. 2 for details), bases were extracted with chloroform. After concentration, the residue was applied to a silica gel plate and developed in ethylacetate–methanol–diethylamine (9:1:1). The labelled area, eluted and derivatised with heptafluorobutyric anhydride, was chromatographed in benzene–ethyl acetate (9:1). A portion of the eluted radioactive zone was counted and a smaller portion was injected on to a column containing 3% Poly A-103 on Gas Chrom Q (F and M Model 400, fitted with Hewlett-Packard ^{63}Ni electron capture detector). With the use of external standards prepared from crystalline N-heptafluorobutyryl-PEA and an estimate of losses provided by recovered ^{14}C, a simple calculation provided the native PEA level.

As Table 1 shows, the highest level, 780 μg per 2 ounce, was found in the unsweetened variety. Semi-sweet forms contained intermediate amounts and milk chocolate was lowest in PEA. The decline may be related to the amount of time the raw chocolate is subjected to conching, a mellowing process during manufacture which includes rolling and heating. PEA, a relatively volatile amine, could escape during this step. The modest rise in PEA after relatively severe hydrolysis indicates that presumed conjugates do not contribute substantially to the total potential PEA pool.

Table 1 PEA levels in various brands of chocolate

Brand	Country	Type*	μg PEA-HCl per 2 ounce: First analysis	μg PEA-HCl per 2 ounce: Second analysis	Recovery of PEA-HCl added to 1 g chocolate: Added (μg)	Native level (μg)	Total recovered (μg)
A	England	Milk	38	41	—	—	—
		Mild sweet	285	315	1.0	5.0	6.2
B	USA	Milk	78	85	50.0	1.4	57.0
		Mildly sweet	330	—	—	—	—
C	USA	Milk	21	—	—	—	—
		Semi-sweet	360	390	1.0	6.4	8.2
D	USA	Unsweetened	780	978†	20.0	13.8	35.7
E	Switzerland	Milk	86	—	—	—	—
		Bittersweet	260	—	—	—	—
F	Switzerland	Milk	103	—	—	—	—
		Bittersweet	340	—	—	—	—

*Manufacturer's description.
†Sample subjected to 100 °C hydrolysis in 6 M HCl for 0.5 h.

These data suggest that chocolate-induced migrainous attacks are either not related to PEA-induced headaches, or that patients suffering from migraine are sensitive to very low levels of PEA.

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JACK W. SCHWEITZER
ARNOLD J. FRIEDHOFF
RALPH SCHWARTZ

Millhauser Laboratories of the Department of Psychiatry, New York University School of Medicine, 550 First Avenue, New York, New York 10016

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