A superplume in the mantle

K. G. Cox

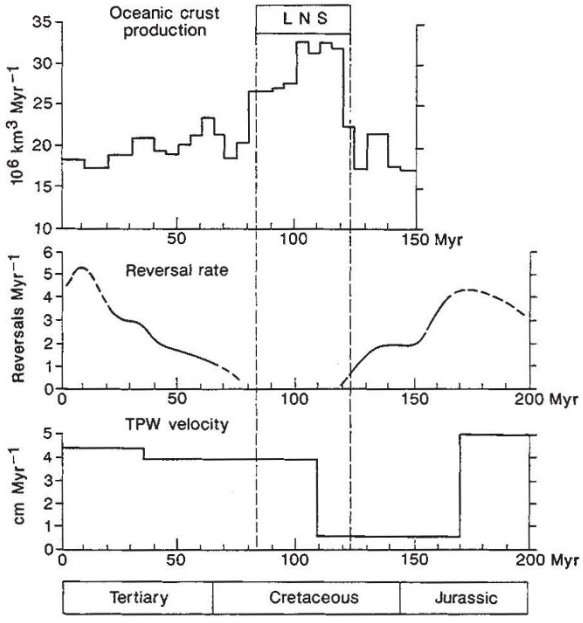
THE Earth's mantle, R. L. Larson suggests in *Geology*[1], may have undergone a vast hiccough in circulation 120 million years ago, the last effects of which may still be apparent in the Tahiti 'superswell'. Perhaps related to events in the Earth's core, the hiccough, Larson argues, caused an outburst of production of oceanic crust, and it may even have been responsible for global climate warming at the time.

The idea that rising hot jets of mantle material (plumes) constitute an important, even possibly the most important, component of the convection of the Earth's mantle, has been growing in popularity among earth scientists since its first appearance nearly 30 years ago. The original concept[2] sought to explain chains of volcanic islands which appeared to migrate across oceanic plates. These were interpreted as representing the volcanic expression of the movement of plates across relatively fixed hotspots or plumes — in other words, the mantle, as well as undergoing convection associated with plate movements, contains a long-lived, and probably deep-seated, stable convective pattern of plumes, over which plates independently move. The plumes were, by implication, pencil-like in form — narrow jets giving rise to narrow chains of islands such as the Hawaiian islands and the Emperor sea-mount chain.

Then evidence of larger plumes, or at least with much more extensive effects at the surface, began to emerge, so that by 1989 some plumes were postulated as having active heads 2,000 km across[3], explaining large continental flood basalt provinces and oceanic plateaux. R. L. Larson's new paper[1] carries this trend one step further, in identifying a so-called superplume which affected areas mainly in the Pacific during the mid-Cretaceous (120–80 million years ago) and had effects over lateral distances of perhaps 6,000 km, a significant fraction of the circumference of the Earth.

Larson's hypothesis is based on a careful estimate of variations in the rate of production of new oceanic crust with time, over the past 150 million years. He finds a 50–75 per cent increase in the production rate during the mid-Cretaceous (top part of figure), which he attributes to the activity of a superplume, originating at the core–mantle boundary about 125 million years ago, and still perhaps represented, though in a near-exhausted state, by the present-day 'superswell' under Tahiti.

If superplumes do exist, however, they are not likely to be manifestations of just mantle convection. Convection on this scale, if originating in the lower mantle, may be related to events in the core, such as reversals of the Earth's magnetic field. As evidence of this, Larson demonstrates that the period of maximum oceanic crust production coincides very closely with the long period of normal polarity (the 'long normal superchron' or LNS) during the Cretaceous, although he does not speculate in detail about the physics of the core–mantle interaction. But the implications of superplumes do not stop here. If unusually large convective and melting events take place, the resultant surface volcanism, involving massive additions of CO_2 to the atmosphere, may have dramatic effects on climate, and on the biosphere in general[4].



Variation with time of (top) estimated world oceanic crust production rate (excluding Tethys)[1], (middle) magnetic reversal rate[5], and (bottom) velocity of true polar wander[5]. Vertical broken lines enclose the long normal superchron (LNS).

The general hypothesis that mantle convection — the main mechanism of heat loss from the Earth's interior — may control major events both at the surface and in the core has always been exciting and plausible. But what is the nature of the new evidence, how compelling is it, and indeed, in terms of general scientific method, how should such speculations be conducted?

Larson's calculations of rates of production of oceanic crust are based on a number of clearly stated assumptions. Little is known, for example about the Cretaceous crust of the eastern Pacific, most of which no longer exists, having been subducted under the Americas. Symmetrical spreading has to be assumed for an estimate of the missing volume. It also has to be assumed that most of the large oceanic plateaux of the western Pacific (which have much thicker crust) had eastern counterparts that are no longer available. Similarly, nothing can be said about production of oceanic crust, if any, in the vanished ocean of Tethys. All the same, the calculated increase in crust production during the mid-Cretaceous is so large that the conclusions deserve serious attention.

More problematic is the question of whether crust production at mid-ocean ridges (as opposed to oceanic plateaux) is a safe measure of the vigour of deep-seated mantle convection. The amount of sea-floor spreading at a particular period clearly may be influenced by plumes, but it is also a function of the amount of concurrent subduction. Larson argues that the latter did not vary significantly during the period in question, but the evidence is difficult to assess.

This raises the question, what is the best thing to measure if we wish to monitor deep-seated convective activity in the mantle? Perhaps in the future, using Larson's type of approach, detailed knowledge of basalt geochemistry may permit the argument to be restricted to the volume of volcanism suspected to be purely plume-related (and that would include continental flood basalts), while leaving out production at ridges. Courtillot and Besse[5] have taken a quite different approach, however, using the velocity of true polar wander (TPW — the migration of the hotspot reference frame in the mantle relative to the spin axis of the Earth) as their indicator of important changes in mantle convection. In their analysis (middle and bottom parts of the figure), they found a period of very slow TPW from about 170 to 110 million years ago, coinciding with a significant burst of continental break-up and largely preceding the mid-Cretaceous LNS.

Their important correlation was thus between very slow TPW, continental fragmentation (which was endemic during the same period), and the fairly consistent diminution in the geomagnetic reversal rate during the late-Jurassic and early-Cretaceous. Then, shortly after the mid-Cretaceous LNS had started, TPW velocity increased suddenly to more-or-

less its present high rate. Not surprisingly, their model of core–mantle interaction shows some significant differences from Larson's, which directly relates heat loss from the lower mantle to simultaneous stabilization of the core. From their evidence, slow TPW — that is, reduced convective activity — corresponds to a decreasing reversal rate. Conversely, fast TPW is accompanied by an increasing reversal rate, so, as in Larson's model, increased mantle activity is somehow related to core stabilization. But as the figure shows, some substantial time-lags have to be built into Courtillot and Besse's model, with events in the mantle significantly preceding changes in the core.

There is no doubt that the issues under discussion are of supreme importance to the earth sciences. If the coupling of core, mantle and surface events (including climatic) can be firmly established, a substantial unifying framework will be available for the study of most major geological processes, though events of extraterrestrial origin may yet muddy the waters. The comparison of Larson's and Courtillot and Besse's papers, however, illustrates some of the obstacles to further progress.

First, at present it is permissible to postulate many different things about the physical nature of happenings at the core–mantle boundary, within the mantle itself, and within the core. The generally acceptable theoretical models, against which the observational facts ultimately have to be tested (because many of the phenomena are outside the range of direct observation), do not yet exist in sufficient detail. Superplumes demand supercomputers.

Second (within the observational field) is the problem of correlation of events in time, which is so often (and not always wisely) the basis of geological hypothesis. Larson, for example, demonstrates a superb one-to-one correlation, but unfortunately there is only one period of increased production of oceanic crust to correlate with one period of geomagnetic stability. What is the level of significance? Ultimately, if time correlation is the key to the argument, we need to define as many separate volcanic events as possible, or whatever other events are used to measure mantle convective activity. □

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IMMUNOLOGY

Disease and evolution

J. C. Howard

"Now every species of mammal and bird so far investigated has shown a quite surprising biochemical diversity revealed by serological tests. The antigens concerned seem to be proteins to which polysaccharide groups are attached. We do not know their functions in the organism, though some of them seem to be part of the structure of the cell membrane. I wish to suggest that they may play a part in disease resistance, a particular race of bacteria or virus being adapted to individuals of a certain range of biochemical constitutions, while those of other constitutions are relatively resistant" (J. B. S. Haldane, 1949; ref. 1).

THIS passage is typical Haldane — clear, original, synthetic, precocious. But was he right? Haldane was aiming to explain not the existence of membrane-bound glycoproteins as such, but rather their polymorphic diversity. By virtue of their overwhelming advantage in rate of evolution, microbes seemed always to have the upper hand against host attempts to evolve a resistance mechanism common to all members of the species. Haldane conceived of a form of equilibrium in which host resistance factors vary from individual to individual. Infectious pathogens would then tend to learn to deal with the commonest type, leaving the way clear for rare types to persist, and eventually flourish for a time.

The membrane glycoproteins coded by the major histocompatibility complex (MHC) were some of the main contributors to Haldane's "quite surprising biochemical diversity". We now know that the MHC indeed seems to function as a resistance system, apparently exactly as Haldane conceived it. Polymorphic glycoproteins encoded by the MHC play a central role in the immune system; peptide fragments of infectious origin can be captured in the peptide-binding grooves of both class I and class II MHC molecules and presented by them to the T-cell immune system. Furthermore, the astonishing polymorphism of MHC molecules largely centres upon those residues which interact with peptides[2], and the large excess of coding substitutions at exactly these positions[3] suggests, exactly as Haldane's theory predicts, that there is strong selective pressure in favour of variation.

All this is good physiology and good genetics, but does it constitute proof of Haldane's theory? Surely, what is needed is explicit evidence that MHC allele frequencies are driven by pathogens? On pages 595 and 619, *Nature* now publishes two outstanding studies on the selective pressures operating on MHC polymorphism in the wild[4,5], and they reach what at first sight seem to be opposed conclusions, one wholly for Haldane, the other wholly against. I shall try to reconcile these two studies, and will argue that they should in fact both be seen as strengthening rather than weakening the generalization that infectious disease is the principal motor for polymorphism.

First, for Haldane, Hill and colleagues[4] face the problem directly. Find a disease associated with a high mortality in the wild, a disease having a high prevalence and in a species in which it is possible to identify most histocompatibility alleles with precision. In addition, it must be possible to assess disease-related mortality at all stages in the life cycle. The species is the challenge of course. Only the human qualifies. The disease is falciparum malaria, hyperendemic in West Africa, a potent killer of the young and already established as the exemplar for natural selection through its effect in elevating the frequency of HbS, the sickle cell haemoglobin allele[6] (still, after more than 30 years, the only balanced polymorphism in man for which we have an explanation).

Hill *et al.* compared human leukocyte antigen types in children desperately ill with severe malarial anaemia or cerebral malaria against a number of control groups. This was a brave and correct analysis, for the story is about natural selection, and natural selection in this context means life-threatening illness. Out of 45 class I alleles (assayed first by serology, then confirmed in a different sample by the polymerase chain reaction), one alone, HLA-Bw53, was significantly reduced in frequency in the severely ill children. The hypothesis that HLA-Bw53 is indeed a protective allele against the severest forms of malaria is confirmed by its geographical distribution; vanishingly rare elsewhere in the world, HLA-Bw53 reaches a frequency as high as 25% in malarial regions of Africa. Out of 13 class II haplotypes assayed, again one, DRB1*1302-DQB1*0501, was significantly reduced in frequency in one category of the severely ill children, namely those with severe malarial anaemia. The class I and class II protective mechanisms are presumably different, and suggest new approaches to vaccine development.

Although the degree of protection conferred on an individual by the protective HLA alleles was less than that provided by HbS, the higher frequency