EARTH SCIENCE

Plate motion and mantle plumes

A model based on geophysical data from the Indian Ocean suggests that a mantle–plume head may once have coupled the motions of the African and Indian tectonic plates, and determined their respective speeds. SEE ARTICLE P.47

R. DIETMAR MÜLLER

News reports persistently remind us of the effects that plate tectonics has on our daily lives. Earthquakes and tsunamis are consequences of the relentless grinding of tectonic plates past each other. In Earth's deep mantle, rocks flow like warm toffee, and hot, buoyant material can rise up to the surface, causing volcanic eruptions and ash clouds that can bring air traffic to a halt. Our knowledge of what ultimately drives the plates is still incomplete, however, not least when it comes to understanding the possible influence of mantle plumes. On page 47 of this issue, Cande and Stegman[1] offer a fresh perspective on the possible coupling between plate motions and the forces generated by these plumes.

Mantle plumes are hot, cylindrical upwellings capped by an enlarged, mushroom-like head. When a rising plume head arrives at Earth's surface, volcanoes spew out scorching volcanic ashes, gases and voluminous lava. These are quite rare geological events. But when they happen they can cause the break-up of supercontinents, as well as mass extinctions and major ecological changes on land and in the oceans[2]. On the bright side, they are also known to act as a catalyst for emplacing diamonds in the shallow crust[3].

Cande and Stegman[1] extend the catalogue of phenomena attributed to mantle plumes by suggesting that they are able to impart a substantial force on plates — a plume-push force — that can do more than just break up continents. Their hypothesis is that if a rising plume head impinges on the base of a tectonic plate long after supercontinent break-up and dispersal, its pushing force may result in a substantial transient acceleration or deceleration of plates. Whether a plate adopts the speed of a tortoise or a hare after being hit by a plume depends on how the plume-push force balances out with other forces acting on the plate.

Cande and Stegman study the effect of the Réunion plume, whose mushroom head is thought to have reached the surface around 67 million years (Myr) ago, emplacing the huge volumes of volcanic rocks in India known as the Deccan traps[4]. Using a careful analysis of marine geophysical data from the Indian Ocean, they note several phenomena associated with this event: the Indian plate sped up between 68 and 66 Myr ago and subsequently sustained absolute plate-motion speeds of 10–12 centimetres per year for about 15 Myr, with its peak speed reaching about 18 cm yr^{-1}. Between 52 and 45 Myr ago, India's motion slowed dramatically to less than 4 cm yr^{-1}. In addition, Africa's motion slowed down while India's sped up, only to return to its previous path and speed after India had decelerated. Figure 1 shows the state of play at 65 and 40 Myr ago, with respective fast and slower motion of the Indian plate.

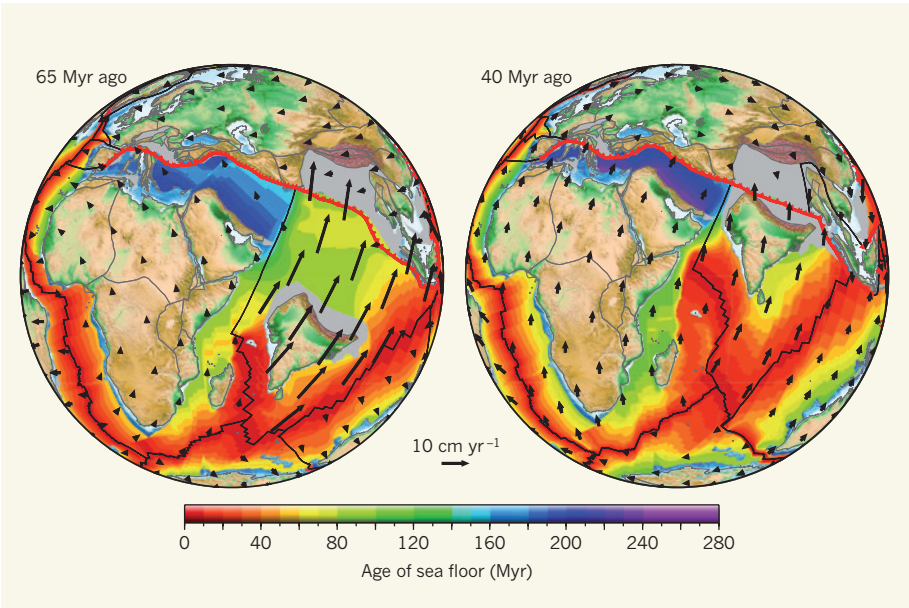

Figure 1 | The Indian and African plates 65 and 40 million years ago. The black arrows represent absolute plate motions, with the fast motion of India 65 Myr ago, and the slow motion of Africa, corresponding to a time shortly after the Réunion plume head is thought to have arrived at Earth's surface; the plume head is hypothesized[1] to have affected the plates' speeds. By 40 Myr ago, the influence of the plume had waned: the Indian plate had slowed down considerably and the African plate had resumed its former direction and speed. Rotated present-day topography is shown on the continents, and sea-floor age is depicted in the ocean basins. The estimated extent of now-destroyed continental margins is represented by the grey areas; mid-ocean ridges and transform faults are shown as black lines; subduction zones are shown as a red line, with teeth on the overriding plate. (Plate reconstructions generated using the GPlates software, data and rotations[11].)

India's superfast motion has been puzzling. One model ascribed it to loss of India's continental roots owing to mantle-plume activity when the supercontinent Gondwana originally broke apart[5]. However, such an erosion and melting away of India's roots, thought to have occurred around 130 Myr ago, cannot be held responsible for the specific acceleration starting 67 Myr ago. Moreover, some geodynamic models suggest that the maximum velocity that tectonic plates typically reach is 8 cm yr^{-1}. The reason for this maximum speed is that plates are largely driven by the pull of slabs subducting in the mantle, especially upper-mantle slabs to which plates are attached[6,7]. The characteristic sinking velocity for upper-mantle slabs is about 7 cm yr^{-1}, with a maximum of 8 cm yr^{-1} for the oldest, thickest, least buoyant slabs[6]. This work has been confirmed by sophisticated numerical models for present-day plate motions[8], which show a near-perfect agreement between observed plate motions and those predicted if plates are driven only by upper-mantle slabs.

Therefore, geodynamic models raise the question of how plates can achieve velocities in such great excess of 8 cm yr^{-1}. Cande and Stegman[1] use their detailed kinematic models to make a conceptual argument that, by adding a plume-push force, India can be sufficiently accelerated while Africa is slowed down. This dichotomy is explained by considering the location of the Réunion plume relative to the two plates. In India's case, the plume-push force would act in unison with slab pull north of India, whereas in Africa's case it would counteract slab pull, slowing the continent down. After exhaustion of the plume head and waning of its presumed pushing force, Africa would exhibit a speeding up synchronous with the slowdown of India.

Of course, this scenario is not without problems. One potential difficulty is that the Deccan traps — emplaced as a direct consequence of the partial melts supplied by the Réunion plume head — were generated over a comparatively short period of time, between 68 and 65 Myr ago[4]. On the other hand, the velocity anomaly of the Indian plate lasted for more than 15 Myr. Additional evidence for a relatively short-lived thermal anomaly under India comes from a combination of seismic images of the crust offshore of southwest India and computer simulations of crustal stretching. This leads to the conclusion that, by 63 Myr ago, relatively thin ocean crust was produced in the vicinity of the Réunion plume, suggesting a significant reduction in its thermal anomaly by that time[9]. One would imagine that, to satisfy Cande and Stegman's conceptual model[1], the plume head and its pushing force had to persist for a further 11 Myr, until 52 Myr ago when India slowed down substantially. After all, plates have no momentum — if a driving force is taken away, they should slow down relatively quickly.

Another potential problem comes from geodynamical modelling. Van Hinsbergen *et al.*[10] have explored the magnitude of the plume-push force and concluded that although a plume-head effect could account for an acceleration of India, it could do so by only a few centimetres per year, certainly not the 10 cm yr^{-1} required to explain the Indian-plate velocities. Equivalently, their models indicate that a combination of reduced plume flux, reduced coupling with the plate and movement of the plate away from the plume can account for only a small portion of India's dramatic slowdown after 52 Myr ago.

Despite these problems, Cande and Stegman's work[1] stands out as an elegant demonstration of how plate kinematic data alone can be used to make inferences about the histories of coupling and synchronous motion of adjacent plates. Whether the plume-push force model for India's superfast motion will stand the test of time remains to be seen, but it offers an intriguing solution to a long-standing dilemma. ■

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STRUCTURAL BIOLOGY

A platform for copper pumps

Copper is vital to most cells, but too much is lethal. The structure of a protein that pumps copper ions out of the cytosol provides insight into both the pumping mechanism and how certain mutations in the protein cause disease. SEE ARTICLE P.59

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Fifty years ago, the neurologist John Menkes described an inherited brain disease in children that was associated with brittle, white, kinky hair. A clue to the origin of the fatal condition came from sheep grazing on copper-deficient pasture in Australia: the animals' steely wool looked like the hair of children with Menkes disease. In both cases, the explanation was found to be inactive copper-dependent enzymes (cuproenzymes). More precisely, affected children have a faulty protein[1] known as the P1B-type ATPase ATP7A (often called the Menkes ATPase), an enzyme that pumps copper ions through membranes and has a vital role in distributing copper around the body.

On page 59 of this issue, Gourdon *et al.*[2] present the much-anticipated crystal structure of a P1B-type ATPase, and reveal a platform in the protein that creates a loading site for copper. A helix peculiar to P1B-type ATPases bends to form the platform, and the authors find that amino-acid residues at the bend of this helix are often mutated in the ATP7A protein of patients with Menkes disease (Fig. 1). More broadly, the structure provides information about the architecture of the P1B-type ATPase family of metal-transporter proteins, which are ubiquitous in bacteria, plants and animals.

Cells handle copper with care. Most cuproenzymes are confined to intracellular compartments or are expelled from cells to perform roles in extracellular fluids and matrices. Other proteins known as metallochaperones 'hand-deliver' copper to where it is needed, relinquishing their load only to those proteins with which they specifically interact and which bind copper most tightly[3]. Proteins such as the Menkes ATPase pump copper from cells to prevent an excess of the metal from accumulating inside. They also pump copper from the cytosol into intracellular compartments where the metal is needed as a cofactor for cuproenzymes[4], as well as into compartments that carry copper and cuproenzymes out of cells. When the Menkes ATPase fails, rather than being loaded into the bloodstream, copper becomes lodged inside cells lining the intestine. This, in turn, leads to a defective supply of copper cofactor for cuproenzymes[5].

Gourdon and colleagues' structure[2] of LpCopA — a P1B-type ATPase from the bacterium *Legionella pneumophila* — is relevant to an eclectic range of sub-disciplines. For example, plant P1B-type ATPases deliver copper into the compartments of chloroplasts, where the metal ions conduct electrons in the process that converts light into chemical energy (the first stage of photosynthesis)[6]. Elevated levels of the Menkes ATPase have been linked to the progression of Alzheimer's disease, and a related protein, ATP7B, limits the efficacy of the anticancer drug cisplatin. P1B-type ATPases are also vital weapons in the immune system's battle against pathogenic bacteria[7]: when immune cells engulf invading microbes, they attack them with bactericidal copper pumped by Menkes ATPases; the bacteria use their own P1B-type ATPases in defence. Microbial P1B-type ATPases also counter the copper used as a fungicide in vineyards, and as a disinfectant in public water systems that can harbour Legionnaires' disease.

Because copper is bound and/or chaperoned