The Thermal State of Earth’s Core

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The current understanding of Earth’s interior has its origins in a landmark paper by Birch in 1952 (1). Using seismological models of Earth’s internal structure and available knowledge of high-pressure equations of state, Birch inferred that the outer core was composed of a liquid iron alloy, whereas the inner core was made of crystalline iron. It followed that the position of the inner-core boundary was defined by the melting temperature of iron and its alloys, providing an important constraint on the thermal state of the core.

Subsequent attempts to determine the melting temperature of iron at high pressure have often produced discordant results (2). Debate among experimentalists continues, but an emerging trend toward high melting temperatures challenges longstanding beliefs about the thermal evolution of the core and the age of the solid inner core.

Experimental studies use shock waves or a combination of static compression and laser heating in diamond cells to achieve high pressures and temperatures. But these experiments cannot fully reproduce the conditions in the core. Melting temperatures must be extrapolated to the pressure of the inner-core boundary (330 GPa) and adjusted for the effects of impurities. Commonly proposed impurities include silicon, sulfur, and oxygen.

Typical estimates for the temperature at the inner-core boundary fall in the range of 5000 to 6000 K (2). Independent estimates from theoretical studies have only recently become feasible, but the results are broadly consistent with the experimental values. A recent calculation that explicitly included the effects of impurities predicted 5500 K (3). Despite the large uncertainties in these estimates, most values are sufficiently high to have important consequences for the rate of cooling of the core.

Heat loss from the core depends on the radial temperature gradient at the boundary between the core and the overlying mantle (see the figure). Temperatures on either side of this boundary are estimated by extrapolating down from the near-surface and up from the inner-core boundary. The difference is thought to represent the temperature drop $\Delta T$ across the thermal boundary layer at the base of the mantle.

By far the largest source of uncertainty in $\Delta T$ is due to the temperature at the inner-core boundary. Current estimates give a temperature drop of 1000 to 1800 K across the boundary layer (4), thought to have a thickness of several hundred kilometers (5). Uncertainties in the thermal conductivity of mantle silicates broaden the bounds on the heat flow, $q$, yielding allowable values of 0.04 to 0.08 W/m$^2$.

These values are surprisingly high, corresponding to a substantial fraction of the heat flow at Earth’s surface.

Integrating $q$ over the area of the core-mantle boundary gives a total heat flow from the core of 6 to 12 TW. Such large values of heat flow have several consequences for the thermal evolution of the core.

Cooling and solidification of the liquid core cause the inner core to grow. Calculations of the thermal evolution of the core suggest that the inner core crystallized from an entirely liquid core about 1000 million years ago (6). Before this time convection was sustained in the core by cold, dense fluid that forms at the top of the core due to rapid cooling. This is important because the persistence of Earth’s magnetic field over the past 3000 million years or more (7) requires continuous regeneration by fluid motion. The power requirements of the magnetic field impose minimum bounds on the heat flow.

At the present time, buoyancy sources due to latent heat release (8) and exclusion of light alloying elements (9) from the inner core drive vigorous fluid motions with relatively modest heat flows. It is estimated that the power required to maintain the magnetic field is roughly 1 TW (10). This rate of power consumption can be sustained at the present time with a heat flow of 5 to 6 TW (11). Such values are at the low end of current estimates, indicating that more than enough power is available to generate the magnetic field.

However, when the inner core was absent during earlier times, the heat flow must have been higher to compensate for the loss of buoyancy sources associated with inner-core growth. Maintaining a power supply of 1 TW before the formation...
of the inner core requires a heat flow of ~15 TW. About 40% of this heat flow is carried by conduction across the core, leaving only 60% to contribute to fluid motions and the maintenance of the magnetic field.

Changes in the heat flow with time are controlled mainly by changes in the thickness of the thermal boundary layer at the base of the mantle, because changes in the temperature across the layer are comparatively small. It is possible to increase the heat flow to 15 TW prior to 1000 million years ago with plausible models for the effects of temperature-dependent viscosity, but the resulting thermal histories invariably produce unrealistically high temperatures in the core by 2000 to 3000 million years ago. So how can we reconcile the high heat flow needed to maintain Earth’s magnetic field with reasonable thermal histories at early times?

There appear to be two ways out of our present difficulties. The first (and perhaps less likely) option is that the power requirements for the magnetic field are much less than 1 TW. With a heat flow sufficient for maintaining a power requirement of just 0.1 TW, we can extend the age of the solid inner core to 3600 million years and avoid high early temperatures (11). However, the present-day heat flow would then have to be substantially lower than 6 to 12 TW. This discrepancy could be explained by accumulating radioactive elements at the base of the mantle, because such additional heat sources decrease the temperature gradient, and hence the heat flow, at the core-mantle boundary. Such an enriched layer could be formed by fractionating radioactive elements into a dense silicate melt at the base of the mantle. A thin zone of partial melt is often invoked to explain anomalously low seismic velocities in this region (12). An alternative source of radioactive material may be a continuous supply of subducted oceanic crust to the core-mantle boundary (13).

A second option requires additional heat sources in the core itself. These heat sources would slow the cooling of the core for a prescribed heat flow and therefore extend the age of the inner core. 40K is most commonly cited as a potential radiogenic heat source in the core (14), although there is little experimental evidence for partitioning large amounts of K into liquid iron (15) during the formation of the core. However, a present-day concentration of 200 ppm K (by mass) is sufficient to avoid high temperatures at early times.

My thermal history calculations show that 200 ppm K has only a small influence on the age of the inner core (increasing it from 950 to 1300 million years for a heat flow of 9 TW). But the effect on the thermal history at earlier times is dramatic because 40K has a short half-life, causing a substantial increase in heat production in the past. A K concentration of 400 ppm could plausibly cause the core to warm at early times before cooling to its present state. The inner core (if present) would shrink during the initial warming and subsequently grow to its current size during cooling.

Advances in experiments and theory have come a long way in our pursuit of Birch’s challenge to determine the thermal state of the core. Yet, the thermal history that brought us to this state remains unclear. Better knowledge of the partitioning of all radiogenic elements between various reservoirs in the planet will help to reduce some of the ambiguity. More reliable estimates of the power requirements for Earth’s magnetic field would place tighter bounds on the allowable heat flow.

Progress on these issues will undoubtedly raise new questions if the past is any guide.

References

C E L L  B I O L O G Y

A Matter of Life or Death

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Photoreceptor cells of both the vertebrate and invertebrate retina have remarkable functional properties that mediate vision. These cells reliably detect single photons under dim light conditions and yet continuously respond to a wide range of light intensities through a finely tuned phototransduction signaling cascade. Rhodopsin, the light-sensitive pigment in photoreceptor cells, is arguably the best understood member of the diverse G protein-coupled receptor (GPCR) family. Visual phototransduction has served as a model for many other G protein signaling systems. Recent work, including the study by Acharya et al. (1) on page 1740 of this issue, reveals new molecular connections between the phototransduction cascade and another crucial cellular signaling process, programmed cell death (apoptosis).

Diseases of retinal degeneration, such as retinitis pigmentosa, age-related macular degeneration, cone dystrophy, and Oguchi’s disease, are all associated with mutations in different components of the visual signaling cascade (2, 3). Despite the diversity of molecular sites affected, these mutations all share a common final outcome: the apoptotic death of photoreceptor cells (3). The compound eye of the fruit fly Drosophila has proved to be a facile model system for dissecting the molecular mechanisms of photoreceptor cell death. Genetic screens that score characteristic changes in eye morphology due to photoreceptor cell loss identify two classes of retinal degeneration mutants. The first class comprises mutations that induce the light-independent death of retinal cells. These mutations are typically found in genes encoding structural components of the rhabdomere (the tightly packed set of microvillar membranes that houses the visual signaling machinery) or genes required for the synthesis of rhodopsin (4). The second class comprises mutations that cause light-dependent photoreceptor cell death and involve mutations in genes encoding several key components of the visual signaling cascade.

The second class of fly eye mutants can be further subdivided according to their requirement for productive visual signaling. For example, arr2 mutants expressing low amounts of the protein arrestin (which shuts off activated rhodopsin) display light-dependent necrotic death of photoreceptor cells, which is suppressed by deleting the Gα protein required for visual signaling (5). A similar form of retinal degeneration is found in rdg4 mutants carrying

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