Taking the core temperature

Mark S. T. Bukowinski

The Earth’s core extends from its centre to about 55% of Earth’s radius, or about 2,900 km below our feet. And yet its effects on humanity are immense. The liquid outer core generates the Earth’s magnetic field that shields us, and all other living things, from the solar wind. And by some estimates it provides sufficient heat to the bottom of the mantle to influence mantle convection and hence tectonic motions, earthquakes and volcanism. The extent of these and other influences depends on just how hot the core is relative to the mantle.

This is a matter that continues to challenge geophysicists, and is the subject tackled by Alfè et al. on page 462 of this issue. The authors have applied new developments in computational physics to devise a virtual thermometer that may refine estimates of the temperature of the Earth’s core. The technique involved is innovative and powerful, and should have much broader applications.

To appreciate the geophysical significance of Alfè and colleagues’ contribution, consider first the temperatures in the Earth’s mantle. Imagine going down a deep mine shaft. As you descend you start getting uncomfortably warm. Were you to go as deep as one kilometre, you would find that the temperature exceeds that at the surface by 20 to 30 °C. Vertical conduction along this temperature gradient is one mechanism by which the Earth rides itself of about $4.2 \times 10^{13}$ joules of heat every second. How does the temperature trend continue with depth and how much of the surface heat flow comes from the core?

So, we may have to watch out for a backward-stepping dynein as well.

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Meanwhile, this new study by Alfè et al. provides a benchmark for future computations and challenges the experimental community to try to converge on tighter limits on the melting temperatures. Further stimulus will probably come from computations of chemical potentials that should yield theoretical estimates of the solubility and thermal effects of sulphur, oxygen, hydrogen and other candidate alloying elements in the core. Such calculations should also allow estimates of the density change across the inner core boundary, as well as of the heat of fusion.

Combining ab initio computations with geophysical data it will then be possible to test the assumptions underlying the gravitational dynamo models: that the light component of the core is largely excluded from the inner core, leaving behind a buoyant residue that can drive convection in the outer core. The Earth can be thought of as a high-pressure experiment, a vast arena for the interplay of geophysical observation with experimental and computational materials science. For research, it is a clear win–win situation.

**Apoptosis**

**Cutting red-cell production**

Stuart H. Orkin and Mitchell J. Weiss

For the proper delivery of oxygen to our tissues we need to have enough circulating red blood cells (erythrocytes). As Olympic officials have learned, artificially boosting the number of erythrocytes enhances athletic performance. But a potential downside of having excessive red blood cells is sluggish circulation and stroke. So, for good health, we need finely tuned regulation of red-cell production, and on page 489 of this issue DeMaria and colleagues propose a new mechanism for how this might be accomplished.

The maturation of immature red-blood-cell precursors (proerythroblasts) into mature erythrocytes is positively controlled by a polypeptide hormone called erythropoietin, which promotes both proliferation and survival of erythroid precursors. The idea that the formation of red blood cells might also be negatively regulated has only recently been considered. In cell culture, binding of ligands to so-called death receptors on the surface of erythroid precursors activates death-promoting enzymes called caspases (cysteine proteases with aspartate specificity), and culminates in cell suicide (apoptosis). DeMaria and colleagues now provide evidence that activation of death receptors on erythroid cells — or, alternatively, deprivation of erythropoietin — leads to caspase-induced cleavage (and, hence, inactivation) of a nuclear regulatory protein called GATA-1. This transcription factor is crucial for the maturation and survival of erythroid precursors. The authors’ observations suggest a hitherto unknown negative-control mechanism by which life, death and cellular maturation decisions converge on a single protein target (Fig. 1, overleaf).

In mammals, red blood cells form in the bone marrow within anatomical units called erythroblastic islands. These units comprise macrophages (white blood cells that engulf foreign particles and microorganisms) surrounded by erythroblasts at different stages of maturation. The proliferation and survival of these erythroblasts depend on the presence of erythropoietin. Erythroblasts have erythropoietin receptors on their surface and, if deprived of erythropoietin, they succumb to apoptosis. In part, this reflects a requirement for signals from the erythropoietin receptor in inducing or stabilizing the expression of an anti-apoptotic protein called bcl-\(X_1\) (ref. 2). Indeed, as shown by a study of embryonic stem cells\(^3\), bcl-\(X_1\) is also necessary for normal maturation of erythroid cells. So, as with many other types of cell, apoptosis must be held in check for normal development to ensue.

As well as extracellular anti-apoptotic signals, erythroblasts use internal programmes to ensure their own survival. The