

Daedalus

Cold porridge

Last week Daedalus presented his new Hibernator. The subject is cooled down strongly enough to stop his heart, but his blood is kept moving by rhythmic hydrostatic pressures applied in sequence to his limbs and regions of his body. Thus his vital organs still receive their greatly reduced demand for oxygen and fuel. Monitoring equipment keeps him stable; at wake-up time, microwave heating swiftly restores his normal body temperature.

The primary beneficiaries of the technology are old people. A municipal Hibernatorium could see thousands of pensioners safely through the winter at far less cost than normal pension and social service provisions. Not only would they dodge the hardships of cold weather; they would live longer. Ageing, and the stealthy advance of degenerative diseases, will almost cease at hibernation temperatures. A pensioner with ten years of life in him could last for twenty years — and all that time would be summer. He would not need to relocate to some warm but dreary southern geriatric resort; he could stay around to follow the progress of his grandchildren, and experience and deplore what the world is coming to, for a much longer period.

The mental effects of the Hibernator remain to be explored. Will six months of hibernation seem like six months to the memory? If not, hibernating pensioners will hardly notice their periods of torpor. But if so, the Hibernator could revolutionize the prison service as well. Most prisoners would be happy to serve their time unconscious; and most prison governors would be only too pleased to let them. At the end of (say) ten years' hibernation, the criminal would wake up to recall his crime and late way of life as a distant folly. He could reasonably vow to leave all that behind, and start life afresh. Unconsciousness would be a far better form of rehabilitation than sharing a gaol with an ever-changing crew of lively expert criminals, all keen to swap ideas.

The Hibernator could serve still other purposes. The long-term unemployed could sleep out an economic downturn; unrequited lovers might embrace it as a way to forget. Those valiantly predicting a religious revival, or the resurgence of Socialism, or invasion by aliens, could use it to await (or sleep safely through) these social revolutions. And the younger child's hopeless threat to his big brother — "Wait till I'm older than you!" — would at last have a chance of coming true.

David Jones

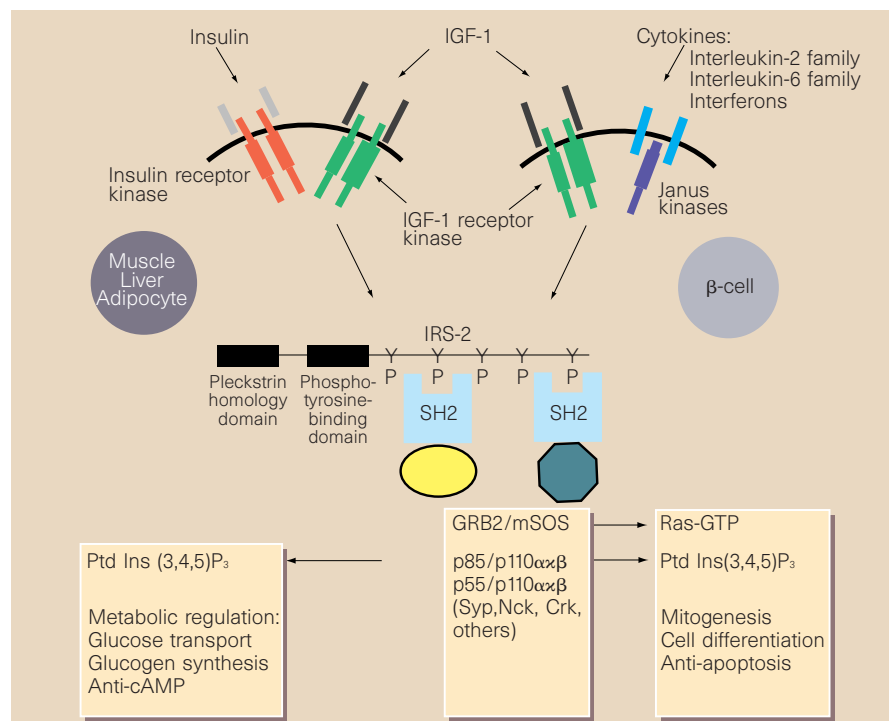


Figure 1 Signal transduction through IRS-2 in the β -cell and in insulin-sensitive tissues. Withers *et al.*² have found that *IRS-2* knockout mice develop a syndrome that closely resembles human type 2 diabetes, implicating *IRS-2* as a candidate diabetes gene. Insulin, IGF-1 and cytokines bind to their respective receptors, resulting in tyrosine phosphorylation of IRS-2 and recruitment of signalling proteins. These include the phosphatidylinositol-3-OH kinases and the Ras-specific guanyl nucleotide exchanger, mSOS, which are likely to be important for the role of IRS-2 in β -cell compensation to insulin resistance. Generation of phosphatidylinositol-3,4,5-trisphosphate (PtdIns(3,4,5)P₃), and perhaps other signals, mediates the characteristic metabolic response to insulin in skeletal muscle, liver and adipose tissue.

the transcriptional basis for β -cell development¹¹. With the exception of hyperglycaemia, the signals that control the fate of β -cells — either during development or in adult life — are poorly defined. A 96-hour glucose infusion in the rat results in a 50% increase in β -cell mass, through increased replication and cellular hypertrophy¹². Nevertheless, the mediators and mechanisms of this response to glucose are not known. Better insight into the control of β -cell fate is necessary to understand what ordinarily limits β -cell compensation in the face of insulin resistance, and to what extent these limitations are genetically programmed or environmentally imposed. The success of β -cell replacement therapies will also depend on such knowledge. For example, long-term β -cell function is much greater when islets are transplanted into human recipients in the context of an intact pancreas, compared with isolated islets. This undoubtedly relates to the capacity for β -cell neogenesis, and the ability of the β -cells to replicate and/or to avoid apoptosis under oxidative or anoxic stress. The contribution of IRS-2 and its partners to these processes will now come under scrutiny. □

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Errata

In the News and Views Feature “Galileo at Jupiter – meetings with remarkable moons” by William B. McKinnon (*Nature* **390**, 23–26; 1997), the heat flow from Io was given as 2.5 mW m⁻². It is actually 2.5 W m⁻², to compare with a terrestrial average of 90 mW m⁻² (Pollack, H. N., Hurter, S. J. & Johnson, J. R. *Rev. Geophys.* **31**, 267–280; 1993). ‘Size’ in Table 1 refers to radius.

In the News and Views article “Fixed hotspots gone with the wind” by Ulrich Christensen (*Nature* **391**, 739–740; 1998), the Web-site address in reference 9 was misspelt. It should have read: <http://www.ngdc.noaa.gov/mgg/image/seafloor.html>

Fixed hotspots gone with the wind

Ulrich Christensen

Island chains such as Hawaii are produced by hotspots – points of volcanic activity driven by plumes of hot rock. New analyses reveal that hotspots are much more mobile than had been thought.

Ever since we have known that the Earth's tectonic plates are in relative motion, the search has been on for fixed points to which absolute plate motion can be referred. For a while it was assumed that hotspots, long-lived centres of volcanic activity, provide such a reference frame. Hotspots are commonly explained by mantle plumes, narrow conduits of upwelling hot rock that probably originate near the core–mantle boundary and rise close to the surface. But it seems unlikely that plumes can be firmly anchored in a convecting mantle. Writing in *Geophysical Journal International*¹, Steinberger and O'Connell now describe how they have used kinematic models to tackle the issue — they show how plumes have swayed, drifted and twisted in the large-scale mantle flow, yet have produced the observed hotspot tracks at the Earth's surface.

The chain of islands and seamounts originating at the Hawaiian hotspot is the best example of such a track. Radiometric dating shows that the rocks become progressively older with increasing distance from Hawaii. The present distances and ages reveal how the Pacific plate has moved, with respect to the hotspot, during the past 80 million years (Myr). For example, the sharp bend at the link between the Hawaiian chain and the Emperor seamount chain (Fig. 1) is thought to have resulted from an abrupt change, at 43 Myr, from northerly plate drift to the current, more westerly, motion.

In 1971, Morgan² recognized that the various hotspot tracks on the Pacific plate can be explained by assuming that the hotspots are fixed relative to one another, and he introduced the concept of deeply anchored mantle plumes. It was later shown, however, that the Pacific hotspots move relative to those in the Atlantic³ at rates of 1–2 cm yr⁻¹. This is less than the speed of fast-moving plates (10 cm yr⁻¹), but enough to make the hotspot frame of reference suspect.

Results from seismic tomography⁴, a technique for imaging the Earth's internal structure using waves generated by earthquakes, imply that much of the return convective flow that balances plate motion occurs in the lower mantle, which therefore cannot be the immobile groundwork through which fixed plumes rise. In a first

step, Steinberger and O'Connell¹ calculate a global mantle-circulation model for the past 68 Myr, using buoyancy forces inferred from tomographic anomalies and taking the present and past plate motions as boundary conditions. In the second step, they insert plume conduits and calculate how these conduits are advected by the 'mantle wind' (a process comparable to the advection of a trail of smoke in the atmosphere). The tendency of tilted plume conduits to straighten out because of their own buoyancy is also accounted for. Strictly speaking, plumes and the large-scale circulation are parts of a single convective system. But because plume conduits are comparatively narrow (50–100 km across), they can probably be considered as separate entities that do not affect the global circulation.

In Steinberger and O'Connell's model, the plume conduits are twisted and substantially tilted from the vertical (it is assumed that plumes break up and become extinct when the tilt gets larger than 60°). The model hotspots migrate at a rate of about 1 cm yr⁻¹ in a reference frame of zero mean plate drift. However, all the Pacific hotspots move more or less consistently towards the south-east. After adjusting the drift of the Pacific plate,

the model explains the observed hotspot tracks equally well as the assumption of fixed plumes does. Because the mantle wind blows differently in other parts of the world, hotspots in the Atlantic and Indian Oceans are predicted to migrate independently from those in Pacific, in agreement with observations.

Plume advection is sensitive to the variation of mantle viscosity with depth. Steinberger and O'Connell's results are consistent with observed hotspot tracks only for a comparatively low viscosity of 1.5×10^{20} pascal seconds (Pa s) between 100 km and 400 km depth, and a high viscosity of the order of 10^{23} Pa s below 1,500 km. If the lower mantle is less viscous than that, hotspots should wander around at a higher rate than is compatible with surface observations. The low viscosity at shallow depth is required to explain the sharpness of the bend in the Hawaii–Emperor chain. In a model with a stiffer upper mantle, the plume conduit reacts to the sudden change of plate drift direction by performing a wide swing, which would result in a smoothly curved hotspot-track.

Although the idea of a viscosity increase with depth is not new, its magnitude has been disputed. A modest rise, less than an order of magnitude, had been inferred from the uplift of the Canadian land surface in response to the removal of the glacial load at the end of the last Ice Age. In contrast, the interpretation of long-wavelength anomalies of the Earth's gravity field related to subducted plates favours a stronger increase of viscosity with depth. This conclusion is now reinforced by the modelling of plume migration.

The new results put some long-standing ideas about plume migration on a more quantitative basis. What are the conse-

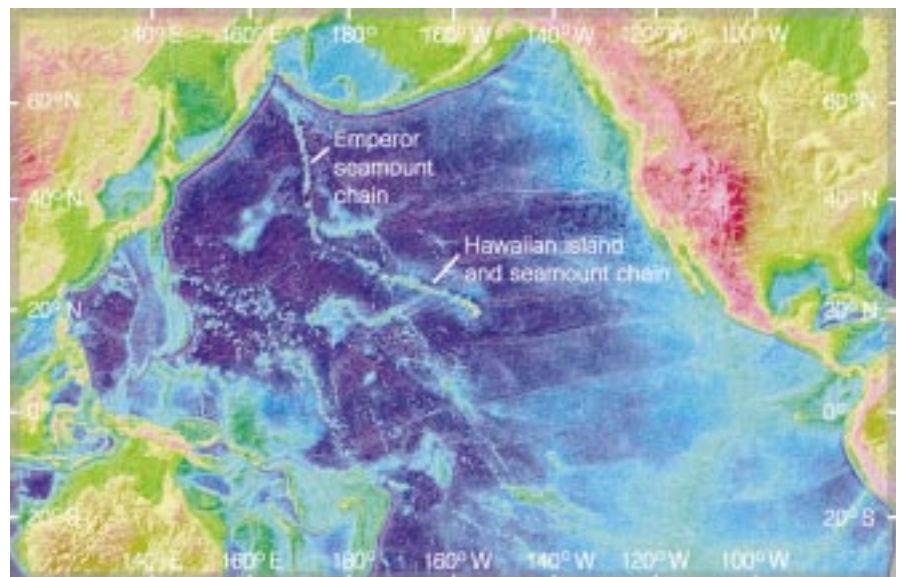


Figure 1 Topography of the northern Pacific seafloor, showing the chain of islands and seamounts originating at Hawaii, and the Emperor seamount chain. The sharp kink between the two is customarily taken to reflect a sudden change in the direction of plate motion about 43 million years ago. (Map reproduced from ref. 9, courtesy of Walter H. F. Smith.)

quences? It seems that we must abandon the convenient concept of fixed hotspots as reference points for past plate motions. Ironically, this jeopardizes some lines of argument in Steinberger and O'Connell's work. The only good evidence that we have for an abrupt change in the motion of the Pacific plate 43 Myr ago is the kink in the Hawaiian hotspot track. No other major tectonic event occurred at that time⁵ and geodynamic models based on buoyancy forces derived from subduction history, which do a good job of explaining present-day plate motions, fail to predict the change⁶. Could it be that the hotspot, rather than the plate, suddenly changed its state of motion?

To decide on this question, we need a reference point that is independent of plates and hotspots. The Earth's magnetic dipole axis, which wanders around but coincides with the rotation pole when averaged over some thousand years, provides just such a reference. The direction of magnetization acquired when a volcanic rock is cooled below its Curie temperature, the temperature at which magnetization becomes fixed, is often preserved over geological time, allowing determination of the rock's palaeolatitude — that is, its latitude at the time that the magnetic field was frozen in.

In another paper, published last year, Tarduno and Cottrell⁷ presented palaeomagnetic data for a 81-Myr-old seamount near the northern end of the Emperor chain, which indicate that it was formed at a latitude of 36 °N. Another seamount further south in the Emperor chain was created 16 Myr later at 27 °N. If the Hawaiian hotspot had remained fixed with respect to the rotation axis, both should have formed at 19 °N, the present latitude of Hawaii. The latitude change could be explained by a movement of the entire Earth relative to the rotation axis, called true polar wander. But although this phenomenon is indeed thought to occur (driven by slight changes in the Earth's moment of inertia), its path, derived from independent data⁸, does not explain the rapid latitude shift of the Hawaiian hotspot in the time between the formation of the two seamounts.

Setting true polar wander aside, the palaeomagnetic data are consistent with a southward drift of the hotspot at a rate of at least 3 cm yr⁻¹ before 43 Myr, much faster than Steinberger and O'Connell's model predicts, and little drift after 43 Myr. These data therefore challenge the usual interpretation, also adopted by Steinberger and O'Connell for calculating their time-dependent circulation model, that the kink in the hotspot track reflects a change of plate motion. Of course, if there is no obvious reason why the motion of the Pacific plate should change, a sudden stop of hotspot migration is equally enigmatic.

Although two data points may not be

enough to make a strong case, and the possibility of true polar wander complicates the interpretation, Tarduno and Cottrell show that the way to disentangle plate drift and hotspot migration is to study the magnetization of volcanic rocks along a hotspot track in some detail. Palaeomagnetic data are abundant for the continents, but most hotspot tracks lie in the oceans and can only be sampled by ocean drilling. So the difficulties in obtaining reliable magnetization directions are far greater for seamounts than for land-based rocks. But it seems well worthwhile for palaeomagnetists to take up the challenge. □

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Immunology

Signal sequences stop killer cells

Eric O. Long

Most proteins destined for the surface of eukaryotic cells possess a signal sequence, which includes a stretch of several hydrophobic amino acids that guide protein translocation into the endoplasmic reticulum during protein syn-

thesis. The fate of amino-terminal signal sequences, found in certain transmembrane proteins, is thought to be much like that of ticket stubs: torn off by an endopeptidase and trashed by further proteolysis.

In an unexpected twist, Braud *et al.*, on

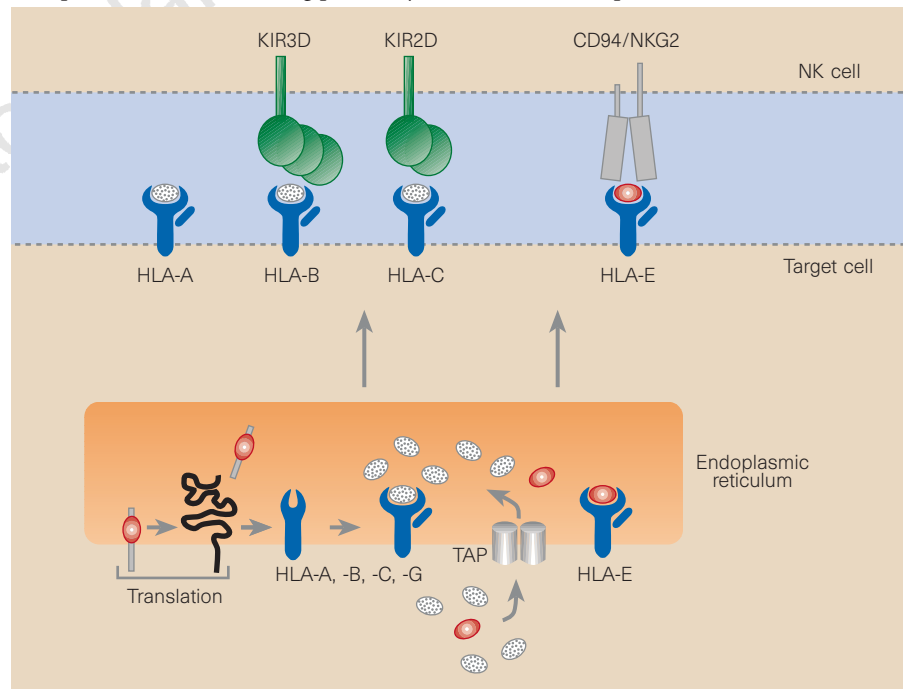


Figure 1 The ABC...E of natural killer (NK) cell tolerance to self. Two types of receptors specific for self HLA class I molecules inhibit NK cells, each having a distinct mode of HLA class I recognition. The lectin-like heterodimer CD94/NKG2 binds to HLA-E molecules¹, whose transport to the cell surface is limited by the availability and binding of a specific peptide (indicated in red) derived from signal sequences of other HLA class I molecules. The killer cell inhibitory receptors with three (KIR3D) or two (KIR2D) immunoglobulin domains recognize HLA-B and -C, respectively. HLA-B and -C bind many different peptides (stippled), most of which are compatible with recognition by KIR3D and KIR2D. The lower left portion of the diagram illustrates the first steps in synthesis of HLA class I molecules. An amino-terminal signal sequence directs translocation into the endoplasmic reticulum during translation of HLA class I messenger RNA. After cleavage of the signal sequence by an endopeptidase, the HLA class I polypeptide folds and assembles with β_2 -microglobulin and a short peptide delivered into the endoplasmic reticulum by the transporter for antigen presentation (TAP). Binding of the signal-sequence-derived peptide to HLA-E is also TAP-dependent⁴. The signal sequence of HLA-E lacks amino acids necessary for binding to HLA-E.