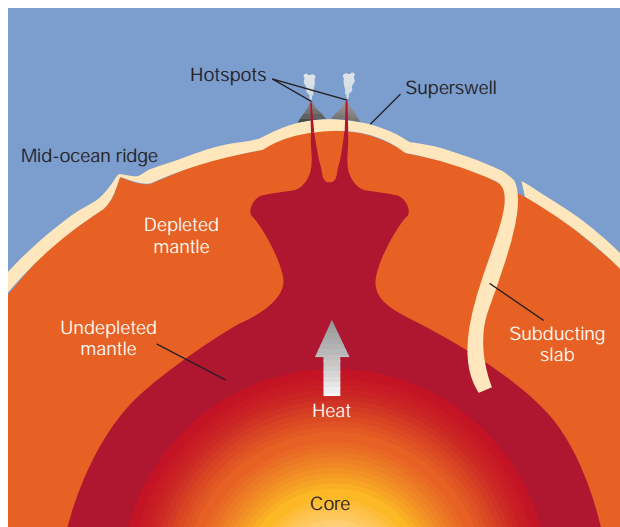


Figure 1 Thermochemical convection in Earth's mantle as it might be now, inferred from Davaille's laboratory simulations<sup>1</sup> with a buoyancy ratio of <math>< 0.5</math>. Heat from the lower mantle and core causes oscillatory doming of an intrinsically denser layer of mantle silicates just above the core. The deeper layer provides a more primitive source of hotspot lavas because, unlike the upper mantle, this region has not lost its volatiles and easily melted radioactive elements during previous plate-tectonic melting cycles.



sity, heated from below, and cooled from above. Results of the experiments depended on the ratio of the intrinsic chemical density difference between the layers to the density anomaly caused by thermal expansion of heated fluid. When this ratio is greater than 1, heating of denser fluid from below does not generate enough thermal buoyancy to offset the chemical stratification, except locally in isolated plumes that rise from the density interface to the surface.

On the other hand, when the buoyancy ratio is between about 0.3 and 0.5, the interface between the stratified fluids deforms into broad domes that rise to the top of the tank, cool and sink back down because of their greater chemical density. This process, which is akin to that seen in a lava lamp<sup>4</sup>, produces an oscillatory pattern of superswell-type features reminiscent of the 100-million-year cycle of superswell formation in the central Pacific. For certain ratios of viscosity contrast between the upper and lower layers, relatively cool plumes rise from the thin thermal boundary layer at the upper surface of the domes (Fig. 1).

Davaille suggests that Earth may have originally formed in the layered-convection regime (buoyancy ratio > 1), but that gradual mixing of the two layers by plume-type upwellings and sheet-type downwellings attenuated the chemical layering to the point that the buoyancy ratio is now less than 0.5. (It is in this sense that the lava-lamp analogy to her model breaks down. Davaille's fluids are completely miscible, unlike those of a lava lamp.) Furthermore, her simulations suggest that the oscillatory doming of superswells rapidly homogenizes the mantle zonation so that there are no more than five cycles of superswell formation. Given evidence from the Pacific that the timescales for superswell rise and fall are of the order of 100,000 years, this would mean that in much less than a billion years Earth will evolve to a well-mixed state of whole-mantle convection.

This model may help to reconcile several conflicting observations as to the extent of layering in mantle convection. The weak stratification thought to represent the present-day situation is insufficient to prevent cold lithospheric slabs, descending at the ocean trenches, from penetrating to depths of 1,000 km or more, as has now been conclusively imaged with seismic tomography<sup>5</sup>. If chemical stratification was indeed stronger earlier in Earth history, it would facilitate the preservation of a deep source for hotspot lavas, more enriched in volatiles and radiogenic isotopes as compared with the mid-ocean-ridge lavas derived from the uppermost mantle<sup>6</sup>.

Of course, much work remains to be done before it will be possible to declare vic-

tory for a model of mantle mixing that is consistent with all geochemical and geophysical observations. Davaille's experiments were conducted in a rectangular tank, not a spherical shell. Her fluids, unlike mantle materials, do not exhibit temperature-dependent variations in viscosity (which influence mixing times) nor realistic depth variations in physical properties, such as the coefficient of thermal expansion. Perhaps most importantly, the lack of temperature-dependent viscosity precludes the formation of more rigid plates in a surface thermal boundary layer, the recycling of which in subduction zones is an important mechanism for reintroducing chemical heterogeneity into the real Earth.

Nevertheless, these experiments point the way for future laboratory and numerical modelling. It can no longer be assumed that chemical density heterogeneities of the order of 1–2% are geodynamically inconsequential. And a snapshot of mantle convection today (as imaged by seismic tomography, for example) may not be representative of its geodynamic behaviour throughout all four billion years of Earth history.

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Lifespan

## The effects of sensory deprivation

James H. Thomas

Once, lifespan genetics was largely the domain of theorists, who tried to explain why an organism's genes so cavalierly allow individual somas to die. But a flood of papers on the nematode worm *Caenorhabditis elegans* has brought the subject into the realm of serious experimental analysis. The latest studies<sup>1,2</sup>, including a report by Apfeld and Kenyon<sup>1</sup> on page 804 of this issue, indicate that the nervous system has a key function in regulating lifespan. Perhaps we are, indeed, only as old as we think we are.

Most of the genes known to regulate adult lifespan also affect formation of the developmentally distinct larval stage called the dauer larva. Formed in response to crowding and other stressors, the dauer larva is adapted to long-term survival under harsh conditions, including the absence of food. Unlike life-

span, dauer formation is very amenable to genetic analysis, and it has been the subject of many studies. Progress in understanding genetic control of lifespan in *C. elegans* began with the finding that mutations in the dauer-formation gene *daf-2* extend lifespan dramatically<sup>3</sup>. This finding allowed researchers to tap into existing information about the genetic pathways that control dauer formation and, more importantly, to use dauer phenotypes to analyse the genes that control lifespan.

This sudden change in the ease of analysis resulted in an explosion of progress, culminating in a fairly complete description of a lifespan-regulating transduction pathway headed by *daf-2*. This gene encodes an insulin-receptor-like protein<sup>4</sup>, and the steps that occur downstream of the DAF-2 receptor parallel the biochemically defined

insulin-signalling pathway in mammals. Specifically, activation of DAF-2 influences the rate of synthesis and degradation of a second messenger called phosphatidylinositol-1,4,5-trisphosphate, PtdIns(1,4,5)P<sub>3</sub> (refs 5,6). The resulting increase in the level of PtdIns(1,4,5)P<sub>3</sub> activates a protein-kinase cascade<sup>7,8</sup>. One output of this insulin-signalling pathway, which was first discovered in *C. elegans*, occurs via the forkhead-like transcription factor DAF-16 (refs 9,10). Mutations that result in activation of DAF-16 lead to a dramatic extension of lifespan (and increased dauer formation), and loss of DAF-16 function prevents this extension.

One gap in our understanding has been what acts upstream of the DAF-2 insulin receptor to regulate its activity. The answer is presumably insulin-like ligands<sup>11</sup>, but what regulates their synthesis and secretion? Dauer formation involves sensory transduction, and the roles of several specific ciliated sensory neurons in dauer formation have been described<sup>12</sup>. Many genes have been identified in *C. elegans* that affect the structure and function of broad sets of sensory neurons, and Apfeld and Kenyon<sup>1</sup> now show that mutations in nearly all of these genes cause increased lifespan, although this effect is much weaker than that caused by mutations in *daf-2*. These sensory mutations do not seem to influence feeding rate, timing of development or fertility, suggesting that their effect on lifespan is relatively direct.

In *C. elegans*, lifespan can also be increased if the germ line is eliminated by laser microsurgery<sup>13</sup>. When Apfeld and Kenyon did this in the sensory-deficient mutants they found that the extension of lifespan was roughly additive, suggesting that the two pathways act independently. Finally, the authors showed that sensory-deficient mutations do not further extend the lifespan of worms mutated in the *daf-2* gene. Moreover, the extended-lifespan effect in sensory-deficient worms is partially suppressed by mutations in the *daf-16* gene. These results indicate that the extension of lifespan caused by the sensory defects results, at least in part, from effects on the insulin-signalling pathway.

What do these striking results mean for the normal process of ageing? The authors' interpretation is simple and sexy — they assert that environmental signals act through sensory neurons to control lifespan. Supporting this view is the fact that there are many insulin-like proteins in *C. elegans*<sup>11</sup>. By analogy with vertebrate insulins, the worm insulins are presumably packaged into secretory vesicles for release from excitable cells. These proteins are a potential mechanistic link between sensory processes and the *daf-2* insulin-signalling pathway. It is possible that insulin release is regulated by sensory neurons that respond to environmental cues,

and that sensory defects reduce this release. The result is a partial failure in activation of the DAF-2 receptor.

But is the link between sensory processes and insulin signalling as direct as a failure to respond to specific sensory cues, as Apfeld and Kenyon suggest? There is reason to doubt this simple idea. In the sensory-deficient mutants, the affected sensory neurons don't just fail to function — they adopt physiologically abnormal states. In particular, the neurons that regulate dauer formation are in an abnormal state that influences dauer formation in ways not characteristic of the wild type<sup>14</sup>. For example, in these sensory mutants affecting the structure of cilia, at least one of the dauer-regulating neurons is thought to release the transforming growth factor- $\beta$ -related protein DAF-7 constitutively<sup>12</sup>. This activity accounts for the dauer-defective phenotype of these mutants. A morphological correlate to this abnormal state is the growth of aberrant axonal processes by many of these neurons<sup>15</sup>. Considering the complexity and variety of the defects, simple interpretations of these results would be incautious.

Nevertheless, Apfeld and Kenyon provide excellent evidence that physiologically abnormal sensory neurons can exert a strong influence on the lifespan of *C. elegans*. This evidence is consistent with a key function for these neurons in the related process of dauer formation, and with the regulation of insulin release by neurons. What it does not provide is specific information about what the normal sensory involvement might be. Discovering whether there are specific environmental modulators of lifespan and, if so, what they are and how they act, is a challenge for the future. ■

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How would the system be paid for? A scientific paper is an advertisement for its authors (indeed, some are little else). So page charges levied on those authors should work well. The tricky problem is refereeing, to keep out (or at least point out) the nutters and incompetents. A democratic database should accept any paper, however mad, provided the author could pay for it. But he would be wise to offer it to a referee, who would 'mark' it, and append comments in the form of a separate note cited in the paper. This citation would neatly reward the referee for his work. The system would multiply the referee's mark, positive or negative, by his own citation score. An author who approached a high-ranking referee would thus be taking a big risk — condemnation would give the paper a very negative mark. Users could scan the database through a filter cutting out papers below a certain mark, thus excluding the low-ranked stuff.

The whole database would probably divide itself into self-contained mutually citing regions. Some would be inhabited by cliques of nutters all citing each other. Some would form rival schools of mutually negative refereeing. But science, alone among philosophies, converges on consensus. The biggest mutually citing region would be the domain of respectable, authoritative scientific opinion. **David Jones**

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