

also produces very fine-grained basalt, and small crystals retain their magnetization much better than do large ones. Where crust is forming above sea level, as it is in Iceland, molten rock often flows tens of kilometres before solidifying in thin horizontal sheets, which do not produce linear magnetic anomalies related to the field reversals. No one I know had expected that we would have the same luck on other planets, which is why the new observations<sup>1,2</sup> are so unexpected.

What do the magnetic anomalies tell us about Mars? The planet must have had a core that acted as a dynamo early in its history, and so provides only the second unambiguous example of dynamo action inside a rocky planet<sup>1</sup>. It must have had a liquid-iron core at this time. But the absence of a present-day magnetic field cannot be used to argue that the core has now solidified. We also cannot estimate the field's strength or the reversal frequency.

The magnetic anomalies themselves are surprisingly large (Fig. 1). Connerney *et al.*<sup>2</sup> remark that they require an intensity of magnetization that is an order of magnitude greater than that of the largest magnetic anomaly found on Earth — which is in Russia, and is  $\pm 10$  nanotesla at 100–200 km (the height of the Mars Global Surveyor). At that height, the magnitude of the martian anomalies is similar to that of anomalies produced by plate separation, but their width is much greater.

If the magnetization is similar, the anomalies require a magnetized layer that is 20–50 times thicker than that within the Earth's oceanic crust, which is generally believed to consist of a layer of quenched basalt about 2 km thick. On Mars, molten rock produced by plate separation will contain about twice the iron, but only half the titanium, concentration of terrestrial basalt<sup>4</sup>. Because of its smaller gravity field, plate separation on Mars will produce about 2.6 times the thickness of melt as it does on Earth if the mantle temperature is the same. So the martian crust is likely to be about 20 km thick, with twice the Earth's concentration of iron. Such a crust is adequate to generate the observed magnetic anomalies if it is magnetized as strongly as it is on Earth. Connerney *et al.*<sup>2</sup> suggest that iron sulphides are required as well, which is certainly possible. The shape of the martian anomalies suggests that the magnetization can resist remagnetization when the main field reverses. On Earth only the top couple of kilometres of the oceanic crust have the required magnetic stability, and it is hard to understand how the cooling on Mars can be more rapid than it is on Earth.

Another surprising feature of the martian anomalies is the lack of associated surface features. Lineated fault blocks parallel to the anomalies should be present if they are produced by plate separation, but there are no obvious topographic features of this kind. The anomaly profiles produced by Conner-

ney *et al.*<sup>2</sup> do not show obvious symmetry, as do those from terrestrial spreading ridges, but the coverage of Mars is still poor. Presumably it will remain so for some time, because the present spacecraft orbit is too far from the planet to be able to make accurate maps of the anomalies.

Am I convinced that the martian anomalies result from plate motions? Not yet. The shapes of the anomalies are not distinctive, they do not form obvious symmetric patterns and they are not clearly offset by fracture zones. A major problem with the

present data results from the variable height of the spacecraft: data are needed from a low circular orbit. Hopefully, the height will be reduced before the spacecraft fails! □

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Ageing

## A message from the gonads

Donald L. Riddle

Animal evolution is commonly viewed as producing diverse, environmentally adapted bodies to propagate the germ line. The evolutionary theory of ageing suggests that genetic limits to lifespan may be inadvertent consequences of evolutionary selection for maximizing that propagation. In other words, trade-offs occur that favour reproductive success over post-reproductive longevity<sup>1</sup>; lifespan should be inversely correlated with fecundity when progeny production diverts resources from the maintenance of somatic (non-reproductive) cells. The germ line contains all the genetic information to specify the soma. But it is also possible that there are other, environmentally modulated instructions for life history that the germ line conveys to the soma to maximize reproduction.

In their intriguing paper on page 362 of this issue<sup>2</sup>, Hsin and Kenyon explore this possibility. From their data, it seems that the reproductive system of the nematode *Caenorhabditis elegans* regulates longevity by means of an insulin or insulin-like growth-factor (IGF-1) signalling pathway. Some general functional parallels between the nematode pathway and insulin signalling in vertebrates have been inferred from the phenotypes of the mutant nematode receptor<sup>3</sup>, but the functions of insulin-like sequences in the *C. elegans* genome have not yet been determined. Nevertheless, down-regulation of the pathway in *C. elegans* was known to result in long life<sup>3–5</sup>, and now it appears that part of that signal is under the control of the gonads.

In *C. elegans*, sterile mutants do not live longer than their wild-type counterparts, implying that, under laboratory conditions, there is no trade-off between reproduction and longevity in this species. This was confirmed by Kenyon *et al.*<sup>4</sup>, who observed no effect on lifespan when they killed the four gonadal cells present at hatching — two germline precursors (cells that generate the sperm and eggs) plus two somatic-gonadal

precursors (cells that generate the reproductive organs) — with a laser microbeam. Hsin and Kenyon<sup>2</sup> have revisited this issue by destroying only the two germline cells, leaving the somatic gonad intact (see their Fig. 1 on page 363). Remarkably, this surgery extended life by about 60%, an observation they replicated in other wild isolates of *C. elegans* and in *Pristionchus pacificus*, a nematode thought to have diverged from *C. elegans* over 100 million years ago.

Removal of both somatic gonad and germ line has no effect, yet the intact germ line normally shortens life. So, what is happening? One explanation is that there is an equivalent signal from the somatic gonad to promote longevity in competition with the life-limiting germline signal. Killing the two somatic precursor cells alone is not a useful experiment, because the germ line will not proliferate in the absence of a somatic gonad. Instead, Hsin and Kenyon turned to a genetic argument using *daf* (dauer larva formation) mutants to address the question of a somatic-gonadal signal.

It has been known for some time that nematodes can regulate their lifespan. In an adverse environment, *C. elegans* can halt larval development to form the dauer (enduring) dispersal stage<sup>6</sup>. Dauer larvae do not feed, and can survive for months until food is found, whereupon they resume development. Post-dauer adults have the same two-week lifespan as adults that never entered the dauer stage, indicating that dauer larvae do not age<sup>7</sup>. Some of the *daf* genes encode components of the insulin/IGF-1 signalling pathway. Signalling inhibits dauer formation and also limits adult longevity. Insulin-like signalling may therefore regulate both development and ageing in response to nutritional status. This resembles the effect of caloric restriction on the lifespan of mice and rats, in which animals with restricted diets live longer than those fed as much as they want.

Mutants of *C. elegans* with reduced DAF-2 insulin-receptor activity live up to twice as

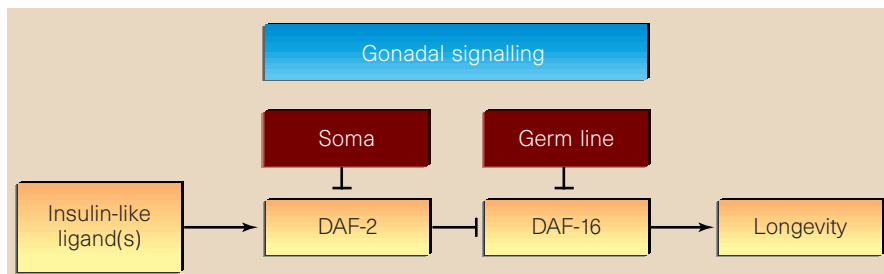


Figure 1 A model for gonadal influences on lifespan arising from the results of Hsin and Kenyon<sup>2</sup>. Arrows, activating signals; T-bars, inhibitory signals. Insulin-like ligands are proposed to activate the DAF-2 receptor, which then deactivates the DAF-16 transcription factor through a phosphorylation cascade. DAF-16 increases longevity (by promoting a process that requires CTL-1 catalase activity) unless inhibited by DAF-2 or by a separate signal from the germ line. Somatic-gonadal cells enhance longevity by reducing the level of DAF-2 inhibition. The role of the DAF-12 protein is uncertain. Destruction of the whole gonad has a similar effect to *daf-12* mutations, so DAF-12 might be required for signalling from both somatic gonad and germ line. However, DAF-12 is not required for somatic-gonad signalling in a wild-type genetic background. Hsin and Kenyon suggest that DAF-12 activity is influenced by DAF-2, an idea that is supported by other genetic data<sup>5,8</sup>.

long as normal. They stay younger longer, as judged by their movement and appearance<sup>4,8</sup>. The extended longevity requires the activity of DAF-16, a transcription factor in the forkhead family<sup>9,10</sup>, and also involves DAF-12 (ref. 5), a nuclear hormone receptor<sup>6</sup>. The *daf-2* mutants overexpress CTL-1, a cytosolic catalase that may prevent oxidative damage, and CTL-1 activity is required for increased longevity<sup>11</sup>. A *ctl-1* mutant has a 'progeric' (precocious ageing) phenotype. The overall conclusion in this context<sup>11</sup> is that the catalase protects the nematode from oxidative damage during dormancy, and increased protection extends adult life.

Hsin and Kenyon<sup>2</sup> have discovered that DAF-16 and DAF-12 activities are required for germline ablation to extend life. These proteins both appear to be transcription factors regulated by germline signalling. To reveal the hypothesized signals from the somatic gonad, they carried out a series of surgical experiments on animals lacking *daf* gene functions. Germline ablation did not extend the life of *daf-16* mutants, but whole-gonad ablation shortened life relative to that of intact *daf-16* controls, supporting the idea that the somatic gonad promotes longevity. Furthermore, ablation of either the germline precursors alone, or both the germline and somatic-gonadal precursors, extended *daf-2* mutant longevity, indicating that DAF-2 activity is required for the life-shortening response to destruction of the somatic gonad.

Given these results, the authors propose that signals from the normal somatic gonad promote longevity by inhibiting DAF-2 receptor activity. The idea is relatively simple. The germline signal shortens lifespan by downregulating DAF-16 and DAF-12 activities, whereas the somatic gonad produces a signal that lengthens life by inhibiting DAF-2 (Fig. 1). The activities of germ line and soma are opposite, because one acts on DAF-16,

which enhances longevity, and the other acts on DAF-2, which normally acts to shorten life.

One can imagine how gonadal signalling for longevity might be adaptive. Prolonged survival of the adult in response to limited food might improve its chances of reproducing. This is a strategy analogous to dauer arrest, and is apparently controlled by some of the same genes. On the other hand, a healthy germ line associated with maximal reproduction might also be adaptive by limiting a parent's competition with its progeny for food.

The dauer or non-dauer developmental decision, and the trade-offs between reproductive pattern and longevity, are major aspects of *C. elegans* life history. Investigations of how they have co-evolved, and the molecular controls that are responsible for their integration, are addressing one of the most complex issues in evolutionary biology. Perhaps it is not surprising that insights on ageing arise from developmental genetics. It is interesting to consider whether similar gonadal signals affect longevity in mammals, especially in species that hibernate. □  
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#### 100 YEARS AGO

Thirty years ago, a French entomologist, named Leopold Trouvelot, was living at Medford, in Massachusetts. He was engaged in carrying on a series of experiments on rearing moths. ... He imported the Gypsy Moth, and by some accident, some of the insects escaped from his custody into his own or the neighbours' gardens. ... Had prompt measures been taken, the insect might possibly have been exterminated; but it does not seem to have attracted any attention till about 1880, when the people then living in or near M. Trouvelot's former residence began to be troubled with swarms of caterpillars, though what they were, and whence they came, was then unknown. For several years the neighbouring houses suffered severely, apple- and pear-trees and shade-trees being stripped of their leaves and killed, and the caterpillars creeping all over and into the houses. Nevertheless, they spread very slowly along the street, and into surrounding woods till 1889, when the insects multiplied so much that the caterpillars stripped all the trees in the immediate neighbourhood of M. Trouvelot's old house, and then marched forth in armies sufficient to blacken the streets, in search of fresh provender. A terrible account of the ravages of the caterpillars is given by those who witnessed them. From *Nature* 25 May 1899.

#### 50 YEARS AGO

The numbers of university undergraduates, in the technical faculties especially, are to-day greater than ever before, and yet apprehension as to the true worth of university training was never so widespread as at the present time; for modern scholarship has made such strides, particularly in the field of science, that no one subject can be adequately studied without undue specialization and consequent neglect of other points of view. This danger of specialization was the theme of Mr. Oliver Stanley's recent address, on being installed as chancellor of the University of Liverpool, when he said that too many men and women to-day leave a university complete masters of a subject but still incomplete individuals, unable to act as evangelists of that broader culture, that more general philosophy which should be the university's gift to the people. From *Nature* 28 May 1949.