

traditional biologists would recognize as a species. Species concepts are difficult to apply to bacteria and protozoa, where sexual isolation (the cornerstone of the original biological species concept) is not relevant. A bewildering abundance of species concepts have been proposed and, practically, there are many cases that fall between different sets of criteria^{18,19}; molecular taxon concepts may add to the confusion. This problem is properly the domain of systematists, but a useful compromise will be achieved by extensive sampling from within recognized species and across known species 'flocks'; and developing rules of thumb that can be used to associate molecular differences with useful biological categories¹².

Hebert *et al.* approach this problem by showing that anonymous specimens were robustly associated with their correct morphological species, but it is likely that different rules may have to be implemented for different animal groups with different evolutionary rates. Until a comprehensive database is assembled, it is not clear how helpful the system using COI will be: for example, the nematode samples in Hebert and colleagues' data set were not well resolved, and there were significant problems with speciose taxa such as beetles.

Nonetheless, a taxonomic genomics programme should yield huge benefits for biodiversity science and ecology, much as the sequencing of genomes has benefited other areas of biology. As was the case with the initial work of Hebert and colleagues, the first stages of such a programme will need identified specimens to represent known diversity and link barcodes to biology. Subsequently, barcoding of environmental samples or collections of unidentified specimens will reveal novelties: flocks of taxa where one

species was expected, or divergent taxa with possible novel biology. One of the advantages of this approach is that the raw data — the DNA sequence — can be stored in databases and accessed through the Internet for comparison and reanalysis. Another is that debate about what usefully defines a taxon, how many taxa there are and what taxon diversity means will become a data-rich science, rather than resembling theological speculation as to how many angels can dance on the head of a pin. ■

Mark Blaxter is at the Institute of Cell, Animal and Population Biology, University of Edinburgh, Edinburgh EH9 3JT, UK.
email: mark.blaxter@ed.ac.uk

- Roca, A. L., Georgiadis, N., Pecon-Slattery, J. & O'Brien, S. J. *Science* **293**, 1473–1477 (2001).
- Eggert, L. S., Rasner, C. A. & Woodruff, D. S. *Proc. R. Soc. Lond. B* **269**, 1993–2006 (2002).
- Hebert, P. D. N., Cywinska, A., Ball, S. L. & deWaard, J. R. *Proc. R. Soc. Lond. B* (in the press); www.pubs.royalsoc.ac.uk
- May, R. M. *Science* **241**, 1441–1449 (1988).
- Lambshhead, J. *Oceanis* **19**, 5–24 (1993).
- Woese, C. R. *Curr. Biol.* **6**, 1060–1063 (1996).
- Lawton, J. H. *et al.* *Nature* **391**, 72–75 (1998).
- Godfray, H. J. C. *Nature* **417**, 17–19 (2002).
- Pace, N. R. *Science* **276**, 734–740 (1997).
- Moreira, D. & Lopez-Garcia, P. *Trends Microbiol.* **10**, 31–38 (2002).
- Lopez-Garcia, P., Rodriguez-Valera, F., Pedros-Alio, C. & Moreira, D. *Nature* **409**, 603–607 (2001).
- Moon-van der Staay, S. Y., De Wachter, R. & Vaulot, D. *Nature* **409**, 607–610 (2001).
- Theron, J. & Cloete, T. E. *Crit. Rev. Microbiol.* **26**, 37–57 (2000).
- Floyd, R., Abebe, E., Papert, A. & Blaxter, M. *Mol. Ecol.* **11**, 839–850 (2002).
- Wuyts, J., van der Peer, Y., Winkelmans, T. & De Wachter, R. *Nucleic Acids Res.* **30**, 183–185 (2002).
- Chase, M. W. & Fay, M. F. *Genome Biol.* **2**(4): reviews1012 (2001).
- Markmann, M. *Entwicklung und Anwendung einer 28S rDNA Sequenzdatenbank zur Ausschlusselung der Artenvielfalt limnischer Meiobenthosfauna im Hinblick auf den Einsatz moderner Chipstechnologie*. Thesis, Ludwig Maximilians Univ., Munich (2000).
- Noor, M. A. F. *Trends Ecol. Evol.* **17**, 153–154 (2002).
- Adams, B. J. J. *Nematol.* **33**, 153–160 (2001).

example of a fluid so resistant to flow that it 'remembers' its past history and produces forces within it that are proportional to the spatial inhomogeneities in its stretching. This resistance tends to make the incursions into these materials narrow and crack-like, similar to the hairline fractures in a brittle material. Experimentally, this kind of behaviour has been seen in wet clays² and in dilute solutions containing long-chain polymers³.

Levermann and Procaccia's technique builds on the work of Hastings and Levitov⁴, who developed methods based on the theory of analytic functions of complex variables to describe situations in which the retreating fluid was an ordinary, 'newtonian' viscous fluid. The pressure in this kind of fluid obeys the partial differential equation called Laplace's equation. Mathematicians would say that the pressure is a harmonic function. Laplace's equation provides a precise statement of the condition that the pressure within a region varies as smoothly as it can, given its values at the boundaries of the region. Complex-variable methods provide a natural framework and convenient language to describe any situation possessing planar geometry, and particularly harmonic functions. From the coordinates x and y in a plane, the complex variable $z = x + iy$ (where i is the square root of -1) can be defined. As if by magic, the maths tells us that any analytical function of z obeys Laplace's equation. These functions can then be used to build a computer model to calculate the pressure in a system.

For an elastic, rather than a viscous, material, the equation to be solved is more difficult. Levermann and Procaccia have used complex-variable analysis to solve the equations for the elastic material. They obtain the displacement of the material and the forces within it, given only the shape of the crack.

But how can the shape of the crack be determined? Here is the novelty of Levermann and Procaccia's work. In the past, it has mostly been assumed that the motion at the boundary between the two materials is smooth. Instead, Levermann and Procaccia divide the motion into little bursts of activity, with each burst localized in a small region of the interface between the two materials. The probability that a burst will happen depends on the forces in that region of the material boundary. Bursting could be explained by the material being so dirty that the motion takes the form of successive events of sticking and slipping, a chaotic jittering motion. This same motion was assumed by Witten and Sander⁵. Their idea was later applied to viscous flow, and then extended⁶ to problems of fracture in amorphous solids.

To make their calculation predictive, Levermann and Procaccia choose a particular model for calculating the probability of a burst at each point on the interface. They roll the computer's dice to figure out where the

Materials science

Bursting apart

Leo P. Kadanoff

When a low-viscosity fluid is injected into an elastic material, it forces its way through by making slender cracks, in a random, fractal pattern. The spreading of the cracks can be modelled through a series of 'bursts'.

Nature abounds in branched objects possessing random, fractal structures: the pattern of air vessels in our lungs, or the branched structure of a river and of the streams and rivulets that feed it. Such patterns arise when the branching process occurs easily and is sensitive to small, random features in the environment. A laboratory example is the pressure-driven expansion of one low-viscosity fluid into another, more viscous fluid. As the fingers of low-viscosity material push outwards, they create high pressure-gradients, and their motion becomes unstable. As a result, little bits of

'dirt', or some other randomness, can considerably affect the flow. The dirt effects show up both in the details of the pattern and also in its gross appearance.

Writing in *Physical Review Letters*, Anders Levermann and Itamar Procaccia¹ describe a calculational procedure that can predict the shape of these patterns. The authors have extended the theories and simulations developed for two-dimensional viscous fluids to the extreme case in which the retreating medium flows elastically. Elastic materials such as rubber resist stretching and deformation. They may be considered to be the extreme

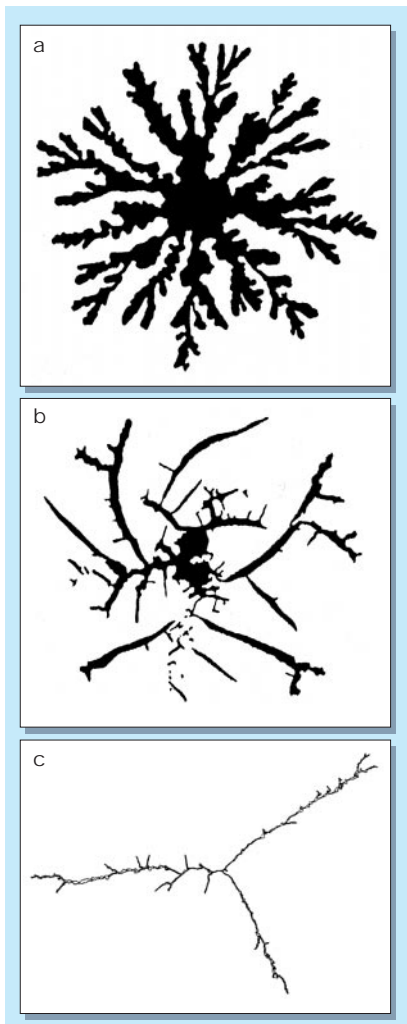


Figure 1 Cracking up. For a fluid injected into viscous low-density mud (a), the measured pattern² of its spread looks very different from the pattern in higher-density, elastic clay (b): in the elastic medium, the pattern is much less full and less branched. Levermann and Procaccia¹ have calculated a theoretical profile (c) for a low-viscosity liquid injected into an elastic medium. Their simulation is closer to the measured elastic pattern than the viscous one, but shows still less branching. (Graphics derived from refs 1, 2.)

next burst will occur, move the interface with that burst, recalculate the stresses and strains and then calculate the probability of burst activity in this new configuration. A complete simulation is shown in Fig. 1, and, although not exactly right, the pattern is indeed reminiscent of those seen in experiments² with elastic materials. For now, the pictures are all we have; more quantitative tests of the model should be developed.

As Levermann and Procaccia point out with commendable candour, the applicability of their method to any particular example of fluid flow depends on several assumptions that are not obviously correct and which should be tested in future work. For example,

the retreating material must be truly elastic for Levermann and Procaccia's method to work, whereas almost all materials in fact show a mixture of elastic and viscous behaviour. Moreover, for any given fluid it is not clear that the particular algorithm used to calculate the bursts is the right one — other workers have disagreed over which equations describe the motion of the interface. The bursting nature of the model might, in any case, not be a correct representation of the situation. Nevertheless, this work represents a promising starting point, and

deserves to be checked and extended. ■

Leo P. Kadanoff is at The James Franck Institute, University of Chicago, 5640 South Ellis Avenue, Chicago, Illinois 60637, USA.
e-mail: leop@uchicago.edu

1. Levermann, A. & Procaccia, I. *Phys. Rev. Lett.* **89**, 234501 (2002).
2. Lemaire, E., Levitz, P., Daccord, G. & van Damme, H. *Phys. Rev. Lett.* **67**, 2009–2012 (1991).
3. Zhao, H. & Maher, J. V. *Phys. Rev. E* **47**, 4278–4283 (1993).
4. Hastings, M. B. & Levitov, L. S. *Physica D* **116**, 244–252 (1998).
5. Witten, T. A. Jr & Sander, L. M. *Phys. Rev. Lett.* **47**, 1400–1403 (1981).
6. Barra, F., Hentschel, H. G. E., Levermann, A. & Procaccia, I. *Phys. Rev. E* **65**, 045101 (2002).

Physiology

Cost-free longevity in mice?

Gordon J. Lithgow and Matthew S. Gill

Studies of worms have revealed hundreds of proteins that, when mutated, extend lifespan. Can this work tell us anything about mammalian ageing? A look at the effects of one such protein on lab mice suggests that it can.

Just over five years ago, molecular geneticists working with the microscopic worm *Caenorhabditis elegans* made an intriguing discovery¹ about a protein that, when its activity is reduced, doubles the animal's lifespan². They found that the protein — DAF-2 — is structurally similar to the human receptor proteins that allow cells to respond to insulin or insulin-like growth factors (IGF). Moreover, a similar receptor influences the lifespan of the worm's distant cousin, the fruitfly *Drosophila melanogaster*³. So could the insulin or IGF receptors also influence human longevity? If so, this would have significant implications for the study and treatment of age-related diseases. On page 182 of this issue, Holzenberger and colleagues report that reducing the levels of the receptor for a subtype of IGF (IGF-I) significantly lengthens the lifespan of laboratory mice⁴. This exciting finding hints that very different sorts of animals, from worms to mammals, age in similar ways. Moreover, it seems that IGF and its receptor are prime candidates for determinants of human longevity.

Ageing is the single largest problem facing biomedicine in developed countries. So it is not surprising that any discovery in the field leads to endless cogitation — none more so than the identification over the past decade of hundreds of *C. elegans* genes that, when mutated, greatly increase lifespan. Each new finding has prompted the same question: could studies of these worm genes reveal general features of ageing that might be applied in combating human age-related diseases?

Hopes have been high ever since the nature of the DAF-2 protein was identified. In *C. elegans*, DAF-2 detects the presence of both insulin-like and IGF-like peptides at the surface of cells, and causes a wave of

intracellular signalling activity that can affect development, fertility, metabolism and the response to stress, as well as lifespan. In mammals, however, the situation is complicated by the existence of distinct insulin and IGF receptors, with the insulin receptor being generally associated with metabolic regulation, and the IGF-I receptor being responsible for growth (Fig. 1, overleaf). Given this additional complexity, it has been important to find out which signalling pathway — insulin-activated or IGF-activated — is involved in regulating mammalian lifespan.

Clues have been emerging from other genetic and environmental interventions that extend the lifespan of mice⁵ (Fig. 1). For instance, some dwarf mouse strains that show abnormal development of the pituitary gland have low levels of growth hormone (a positive regulator of IGF-I), and are long-lived. Similarly, inactivating the growth-hormone receptor lowers IGF-I levels, causing dwarfism, and also extends mouse lifespan. Furthermore, restricting the calorie intake of mice from birth produces low IGF-I levels, dwarf animals, and an increase in lifespan. In all of these cases, longevity is associated with a reduction in IGF-I levels (and hence decreased signalling through the IGF-I receptor) throughout life, with a corresponding deficit in body size. This contrasts, however, with reports that restoring normal IGF-I levels in elderly people, by means of growth-hormone-replacement therapy, may result in significant health benefits⁶.

Genetically altered mice have proved to be essential tools for unravelling the complex and interacting roles of insulin and IGF-I⁷. Using this approach, Holzenberger *et al.*⁴ now confirm the link between IGF-I and ageing in mammals, and show that it is