

solidification in the west and melting in the east will lead in a general way to east–west asymmetry, and provides an alternative idea to long-term control by the mantle for understanding inner-core asymmetry<sup>11</sup>, much work needs to be done to understand the origin of the inferred seismic properties of the inner core.

In a start on that task, Monnereau *et al.*<sup>12</sup> suggest that the east–west asymmetry in direction-averaged seismic velocity and attenuation is due to growth of already solidified grains as inner-core material moves eastwards (although they are unclear as to why this would lead to

east–west variations in elastic anisotropy). This serves as an example of how, by offering an ingenious proposal, the study by Alboussière *et al.* opens up new avenues to investigate the strange centre of our planet. ■

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## ECOLOGY

# Close relatives are bad news

Owen T. Lewis

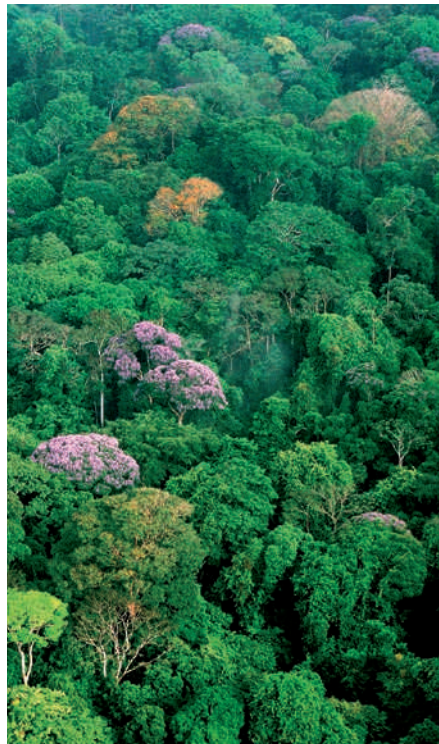
**In tropical rainforests, tree seedlings growing close to their parent are more likely to die. This mortality, caused by soil organisms, helps to explain the coexistence and relative abundance of species.**

Simple models of competition among species suggest that a few tree species, those that are best at exploiting limiting resources such as light and nutrients, should dominate ecosystems such as tropical rainforests<sup>1</sup>. However, rainforests support hundreds of apparently very similar tree species — typically a small number of abundant species and many rare ones. How do these species coexist? Why are some of them rare and others common? Complementary studies in Panama by Comita *et al.*<sup>2</sup> (published in *Science*) and Mangan *et al.*<sup>3</sup> (page 752 of this issue) show that a form of negative feedback driven by soil organisms can explain the relative abundance of tropical tree species, as well as promoting their coexistence.

The theory that pests and diseases can be good for diversity was formalized independently by Dan Janzen and Joseph Connell 40 years ago<sup>4,5</sup>. Under the Janzen–Connell hypothesis, seeds and seedlings close to members of the same species will suffer particularly high mortality from specialized enemies such as herbivores and pathogens, a pattern referred to as ‘negative density-dependence’. The Janzen–Connell mechanism of negative feedback can enhance tree diversity because it prevents any one species from becoming locally dominant. Plants form the foundations of ecological communities, so if we can explain the high diversity of tropical forest trees then we may also explain the high diversity of species further up the food chain<sup>6</sup>.

Negative density-dependent seedling and sapling survivorship consistent with the Janzen–Connell mechanism has been widely documented<sup>7,8</sup>. However, several uncertainties have remained about the biological mechanisms driving these patterns. In particular,

variation among species in terms of the strength of negative density-dependence has been overlooked. Many ecologists have assumed that the most abundant tree species in a local community suffer most from the proximity of neighbours of the same species. Such a pattern could arise if, for example, abundant species support



**Figure 1 | Tree diversity in Panama.** Seedlings of rare species suffer more than those of common species from the presence of same-species neighbours, soil-dwelling organisms being implicated in the process.

large populations of specialized pathogens or herbivores. But both Comita *et al.*<sup>2</sup> and Mangan *et al.*<sup>3</sup> show that the reverse is true: it is locally rare species that suffer most from the proximity of relatives, suggesting that variations in abundance are a consequence rather than a cause of negative density-dependence.

The two investigations took place in the well-studied forests of central Panama (Fig. 1), but applied contrasting approaches. Comita *et al.*<sup>2</sup> analysed an exceptionally complete data set on 31,000 seedlings representing 180 tree species, identified and tagged in 2001 and re-surveyed five years later. They investigated seedling survival as a function of the abundance of trees of the same (conspecific) or different (heterospecific) species growing within a 30-metre radius, and the number of conspecific or heterospecific seedlings growing in the same 1-m<sup>2</sup> plot. Neighbouring trees and seedlings of different species had little effect on seedling survivorship. However, seedlings were much more likely to die close to neighbours of the same species. Strikingly, the extent to which conspecific neighbours affected seedling mortality correlates with species' abundances at the community level: rare species suffer more than common species from the presence of same-species neighbours.

Mangan and colleagues<sup>3</sup> isolated the feedback mechanism underlying these patterns, firmly implicating soil-dwelling organisms. In an elegant reciprocal experiment, Mangan *et al.* grew seedlings of six tree species in pots filled with ‘home’ soil (collected under trees of the same species) or ‘away’ soil (collected under other tree species). Relative to other species, seedling growth and survival were significantly lower on ‘home’ soil, which is more likely to harbour species-specific pests and diseases. Seedlings transplanted into the field near conspecific or heterospecific trees showed similar effects, with little evidence that above-ground pests such as leaf-feeding insects contributed to the results. Again, species experiencing stronger negative feedback were rarer in the community.

Understanding the factors that determine the commonness and rarity of species is a major preoccupation for ecologists, and is also

relevant to conservation because rare species are most at risk of extinction. The results will encourage researchers working in a variety of ecosystems to look more closely at the 'self-limiting' effects of species interactions. Correlations between the strength of feedback effects and relative abundance have also been documented for plants invading temperate grasslands<sup>9</sup>, and it seems likely that similar processes operate in other habitats, including temperate forests.

In the future, one challenge will be to pinpoint more precisely the organisms causing feedback effects, and to assess their specificity<sup>10</sup>. Fungi and bacteria seem the most likely culprits, and Mangan and colleagues plan to use the latest genomics approaches to compare soil microbial communities associated with different tree species. All else being equal, feedback caused by highly specific pathogens will generate the strongest patterns of negative density-dependence. However, pests or diseases that affect several tree species could alter the probability that individuals of closely related species (which are more likely to share natural enemies) will survive in close proximity. If this happens, its signal should be apparent in the seedling data sets studied by Comita and colleagues.

Further experimental research — for

example, through the application of pesticides — may help to unravel the detailed ecological consequences of altering the soil biota. The 'unseen majority' of soil organisms are not a conventional target of conservation efforts, but the new results show that they may be essential to maintaining diverse tropical forest ecosystems. By altering the composition and activity of these communities, human actions such as forest exploitation and anthropogenic climate change may have long-term repercussions for the processes structuring and maintaining rainforest biodiversity. ■

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ATG16L1 variant associated with increased disease risk — show similar changes<sup>5</sup>.

As part of their new study<sup>1</sup>, Cadwell *et al.* find that ATG16L1<sup>HM</sup> mice raised in an enhanced barrier facility (one that is free from pathogens) did not show the inflammatory trait. So, while considering which environmental factors might trigger Crohn's disease, Cadwell *et al.* investigated whether murine norovirus (MNV) — which is often found in conventional, but not in enhanced, barrier facilities — could be a contributor. To do this, they infected normal and ATG16L1<sup>HM</sup> mice (both raised in enhanced barrier conditions) with an MNV strain that causes a persistent infection<sup>6</sup>.

A week after infection, Paneth-cell abnormalities were seen in the ATG16L1<sup>HM</sup> mice, whereas the normal mice showed a normal Paneth-cell response to infection. Furthermore, only Paneth cells from infected ATG16L1<sup>HM</sup> mice showed the pro-inflammatory gene-expression profile and developed intestinal ulceration in response to the toxic substance dextran sodium sulphate. This mucosal injury depended on the presence of the mouse microbiome and was mediated by the pro-inflammatory cytokines interferon- $\gamma$  and tumour-necrosis factor- $\alpha$ .

These results<sup>1</sup> demonstrate that, in mice, several factors — variations in the host's genetic make-up, exposure to a specific virus, toxin-mediated mucosal-barrier injury and the microbiome — can act together to trigger inflammation with similarities to Crohn's disease in humans. Such models will be of growing importance in understanding both inflammatory and complex diseases generally and how the immune system communicates with the environment.

Immune responses to viruses rely on sensors of infection in the shape of 'pattern-recognition receptors', which recognize evolutionarily conserved motifs present in microorganisms; in the case of viruses, these are often viral nucleic acids. Signalling through these receptors affects the induction of autophagy, cytokine secretion and timely antigen presentation to immune cells. Of the known susceptibility genes associated with Crohn's disease, the strongest candidate encodes NOD2, a possible intracellular sensor of nucleic acid<sup>7</sup> that acts upstream of ATG16L1 in the induction of autophagy<sup>8,9</sup>. NOD2 is expressed exclusively in monocyte-derived cells, in which MNV replicates<sup>10</sup>, and in Paneth cells. ATG16L1<sup>HM</sup> mice infected with MNV would therefore be an attractive model for studying the combined effects of genetic risk factors and specific autophagy-mediated immune processes such as antigen presentation in response to inflammation.

A notable difference between Cadwell and co-workers' mouse model and Crohn's disease in humans is that, in the mouse, the genetic risk is mimicked by reducing ATG16L1 expression rather than by expressing the actual human-gene variant. In the human disease, ATG16L1

## CROHN'S DISEASE

# Genes, viruses and microbes

Alison Simmons

**Variations in several genes can increase an individual's susceptibility to complex disorders. But what tips the balance to cause the full-blown disease? For Crohn's disease, viruses could provide part of the answer.**

Crohn's disease, a common inflammatory bowel disorder, is debilitating. Yet, other than the fact that variations in several genes as well as unknown environmental factors contribute to it, little is known about its cause. Writing in *Cell*, Cadwell *et al.*<sup>1</sup> show that, against the right genetic background, a viral infection can make the difference between health and inflammation in mice with a condition that mimics Crohn's disease.

That the host's genetic make-up can account for around 50% of the risk of Crohn's disease<sup>2</sup> is well known. Genome-wide association studies<sup>3</sup> have pinpointed more than 30 genomic regions (loci) variations in which are associated with an increased risk of developing the disease. The question is what contributes to the remaining 50% of risk. Environmental factors, and in particular the host's resident microorganisms (the microbiome), are strong contenders, although direct evidence for an environmental contribution in either humans with Crohn's disease or animal models has not been forthcoming.

Cadwell *et al.*<sup>1</sup> focus on *Atg16L1*, a Crohn's

disease susceptibility gene. The authors had previously generated mice expressing low levels of the ATG16L1 protein (ATG16L1<sup>HM</sup>) and observed two abnormalities in these animals<sup>4</sup>. First, their cells showed reduced levels of autophagy, the homeostatic process by which cells break down their own components. This finding was consistent with a study from another group<sup>5</sup> in which mice expressing a mutant version of ATG16L1, lacking a domain required for starvation-induced autophagy, showed enhanced inflammatory immune responses on exposure to bacterial components.

Second, Cadwell and co-workers found abnormalities in the Paneth cells, a subset of intestinal epithelial cells that secrete antibacterial peptides. Indeed, Paneth cells of ATG16L1<sup>HM</sup> mice show defects in the packaging and extrusion of antimicrobial granules and express higher levels of genes that influence the response to intestinal injury<sup>4</sup>. Consistent with this, Paneth cells of patients with Crohn's disease who express ATG16L1 T300A — an