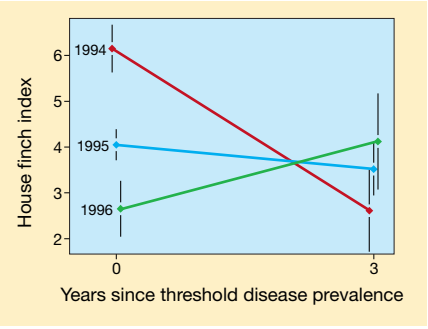

Figure 2 **House finches and *Mycoplasma gallisepticum* infection. The plot shows changes in the birds' abundance in three areas where the threshold disease prevalence (20%) was reached in 1994, and the first halves of 1995 and 1996. There were significant differences in finch abundance before the epidemic, but no significant differences three years later. The study[5] provides convincing evidence that a parasite can regulate host populations in a density-dependent manner.**

pathogenicity in captive house finches[7]. Since 1994, an army of volunteers has tracked the spread of the disease month by month across the finch's eastern range[5,8,9]. To date, the disease has not been reported in the western population.

Armed with information on the proportion of hosts infected and the abundance of the hosts before and after infection, Hochachka and Dhondt showed that there had been a rapid decline in finch numbers at a series of sites with the onset of disease. This effect was most pronounced over the first three years of infection, after which bird numbers stabilized at a lower level. Crucially, bird numbers declined more quickly in those areas infected first, which were also those where the birds were more numerous (Fig. 2) — this is the first demonstration of density-dependent regulation of an animal host by a parasite under natural conditions.

There are many, often spectacular, examples of invading pathogens reducing host abundance (rinderpest in bovine species and phocine distemper virus in seals, for instance[10]). Providing evidence of population regulation is far more difficult, however. The strength of the new study[5] is that the authors had numerous replicate sites where they recorded host abundance before the wave of infection, the time infection arrived and the subsequent host population size.

But there are alternative explanations for the patterns described. For example, the reduced effects of the disease in areas with low bird numbers could be due to declining pathogen virulence. Equally, the course of the disease could have been driven by differences in host susceptibility across the host range. Nevertheless, parasite-induced regulation by a virulent pathogen remains the parsimonious hypothesis. It finds support both from molecular studies that have shown no change in the *M. gallisepticum* strain through time, and from the observation that chronic disease persists for several years after the initial outbreak[11].

Hochachka and Dhondt[5] also assumed, not unreasonably, that parasite transmission depends on bird density. If infection occurs at feeders, however, then the frequency of feeder use might be a better predictor of transmission than density alone — a possibility not tested by the authors. In reality, the spread of most directly transmitted diseases will be fuelled by a subtle combination of both density- and frequency-dependent processes.

Overall, the story is unusual in several respects. Rarely has a disease been so readily and reliably recognized. Both parasite and host find themselves in the eastern states thanks to respective accidental and deliberate release by human agencies (it is possible that 'founder effects', stemming from a limited gene pool in the initial eastern population, have predisposed these birds to disease).

Moreover, the landscape has been heavily modified by human activity, but house finches are well adapted to such environments, from rural dwellings to suburbia, and take readily to garden feeders. This characteristic, combined with a gregarious nature, has been part of their undoing. Unwittingly, people may have been 'killing with kindness' as feeders increase transmission by bringing birds into close contact, and perhaps by keeping the infectious birds alive longer. More positively, the paper illustrates the potential of 'citizen science'[8] in harnessing the enthusiasm of volunteers.

The authors now plan to examine the mechanisms of host–parasite interaction inferred from their study. This is a matter of urgency, because the disease is not only still spreading in finches, but has passed into other songbirds such as *Passer domesticus*, *Carpodacus purpureus* and *Carduelis tristis*[5] — the house sparrow, purple finch and American goldfinch, respectively. ■

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Daedalus

Green smelters

Polluted land can be slowly reclaimed by bioremediation — using microorganisms or plants to detoxify the pollution. Certain 'hyperaccumulator' plants can accumulate amazing concentrations of metal in their tissues, often as insoluble or biologically unavailable compounds such as histidine complexes. Plants have no way of excreting toxic metals, and have evolved this defence against them. Metal hyperaccumulators often occur naturally on the metal-rich terrain of mining districts. Daedalus is now taking the biochemistry a step further.

He notes that a bacterial enzyme, mercury reductase, can reduce mercury ions in solution to mercury metal. The gene for this enzyme has recently been transferred to a water-weed. The weed can then reduce deadly mercury ions in the water to the insoluble metal, which simply sits inertly in its tissues. The same enzyme can also reduce silver and cadmium ions. DREADCO biochemists are therefore looking for plant and bacterial enzymes that can work the trick on other metals. They will then insert them into plants which are hyperaccumulators for that metal. The resulting varieties will extract metallic residues from contaminated land, concentrate them, and smelt them internally to pure metal.

Daedalus is not sure what form the metal will take inside the plant. He would like it to occur as one or more big chunks, ideally in the nuts or fruit of the plant, so that it could be picked at harvest time. But it will probably be distributed widely in the cells as tiny single crystals. The plants will have to be reaped in their entirety, and comminuted in water. The suspension could then be centrifuged or sedimented to extract the metal.

This elegant and highly 'green' metal-smelting process will be widely applauded. Unlike conventional smelting, it uses no fuel and puts no CO_2 into the atmosphere. It should be ideal for reclaiming regions despoiled by conventional mining, as well as tracts of low-grade ore too dilute to dig out in bulk. Easily reduced, high-value metals such as silver, copper and nickel are the most obvious targets. Iron looks feasible too — it certainly has a rich biochemistry. Titanium is a tantalizing hope. But the big target is undoubtedly aluminium. It occurs plentifully in almost all soils, and its conventional smelting needs vast amounts of costly electricity. Yet its biochemistry seems forbidding. Only around Chernobyl may plants have mutated enough to have invented the necessary enzymes. **David Jones**