Pulling genes

Learning the game of disease resistance

Understanding how plants and pathogens interact in the wild can help to improve disease resistance in agricultural crops and conserve native species. Graeme O'Neill reports.

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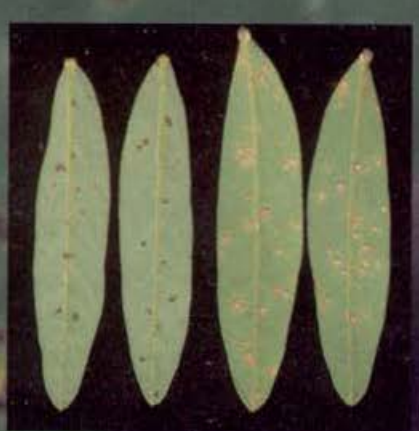

Main picture: Soybean (*Glycine max*) infected with the damaging rust *Phakopsora pachyrhizi*.

Left: Identifying genes for resistance to soybean leaf rust in *Glycine canescens*, an Australian endemic relative of soybean. The two leaves on the left were taken from a susceptible plant; those on the right from a resistant one.

The Australian bush is so peaceful at sunrise that the bushwalker is oblivious to the silent battles going on all around. It's plant versus pathogen, genes at dawn. Gene wars, says Dr Jeremy Burdon, may account for the patchiness of natural plant communities, and the vulnerability of monoculture cereal crops to destructive outbreaks of fungus diseases.

Burdon, is an evolutionary biologist with the Centre for Plant Biodiversity Research, a joint venture between CSIRO Plant Industry and the Australian National Botanic Gardens. He has spent much of his research career delving into the netherworld of plant-pathogen interactions. This year (1996), his peers recognised Burdon's work by electing him to the Australian Academy of Science.

While most researchers at CSIRO Plant Industry work with crop plants, Burdon tends to work with wild relatives of crop species, such as Australian native soybeans (*Glycine* sp.). 'Some of my work has been on the genetics of rust resistance in Australian *Glycine* species, because there is a grave lack of rust resistance in commercial soybeans,' Burdon says.

Originally domesticated in China, the annual soybean has only two or three wild relatives in Asia. In contrast, Australia has 16-plus native *Glycine* species that are a treasure trove of genes for disease resistance and other valuable traits. Soybean breeders at the University of Illinois in the United States have been working with scientists at CSIRO Plant Industry using advanced hybridisation techniques to transfer rust-resistance genes into soybeans.

'Finding disease-resistance genes for use in agricultural species is the most obvious reason for studying wild species,' Burdon says. 'Moreover, it is becoming much easier to import wild genes with recombinant DNA technology.

'But studying host-pathogen interactions in wild species is also important because crops often have weedy or native relatives growing in close proximity. There is a risk that pathogens such as fungus diseases will cross over and infect the crop. For example, wild oats often are found growing near oat crops, and we find that rust fungus strains on wild oats have far more genetic variability than do strains found on cultivated oats.'

Two types of resistance

There are two basic systems whereby plants can resist disease pathogens genetically. The first, called 'gene-for-gene' resistance, involves individual contests between a plant's resistance genes and the corresponding genes for virulence in the pathogen. This is virtually an all-or-none affair in which a plant that is resistant to one pathogen race may be totally susceptible to a different race. The second system of resistance is more of a shotgun approach, in which the plant deploys many genes in its defence, each having only a small effect on the pathogen. While such resistance usually still allows some disease to develop, it tends to be effective against all races of a pathogen.

Burdon says the gene-for-gene resistance system occurs in many crops such as maize, sunflower, oats and wheat and is used widely to combat disease in many agricultural situations. 'In most agricultural crops, especially our major grain crops, disease resistance is largely based on the use of just a few major genes,' he says. 'This is because in a breeding program, it's easier to manipulate single genes which have a large effect, than to move many genes which, individually, have little effect.'

At its most basic level, the gene-for-gene contest may reduce to just two genes: a virulence gene wielded by some anonymous strain of the rust, versus a particular resistance gene in the crop plants. While most rust strains are successfully repulsed by the crop's resistance genes, the resistance genes in turn challenge the pathogen to generate new strains that can overcome them.

This may lead to a much more complex system, such as that occurring in gene-for-gene interactions involving the cereal stem rust *Puccinia graminis*, a serious pathogen of wheat crops. In the northern hemisphere, the rust copes with the seasonal absence of its cereal host by spending part of its life cycle on another plant species, the barberry.

'Many rusts have two hosts, Burdon says. 'They cycle asexually on one host, and sexually recombine on the other. Stem rust has its asexual cycle on wheat. It can go for hundreds of generations without sexual recombination, as it does in Australia.

'Luckily, barberry is extremely rare in this country, and there is no evidence that it has played any role in creating new strains of the pathogen. But there have been rare episodes of asexual recombination between different races of stem rust, and they can be quite devastating.'

Despite this understanding of plant-pathogen interactions, surprisingly little is known about how major resistance genes evolved, and how they interact with pathogen genes in a gene-for-gene system. Agriculture has been around for only about 10 000 years, and extensive monocultures for less than one hundred.

Researchers have tried to simulate crop-pathogen genetic interactions with

Where are the gene-for gene interactions likely to evolve? Dr Jeremy Burdon believes that even within the one host species, different pathogens attacking different tissues may induce different types of resistance. For example, the interaction occurring between a rust attacking the leaves of a deciduous tree such as birch (above) may be based on a gene-for-gene interaction, while that involving a stem canker (left) may involve the action of many genes.

computer-based models. In agriculture, typically, both the host plant and the pathogen occur as monolithic populations. 'To get these models to work, you have to build in cheats, otherwise, either the crop is constantly attacked or the pathogen rapidly goes extinct,' Burdon says.

Wild strategies

The contest between plant and pathogen in the wild is likely to be a far more complex and subtle affair, with the host plant's fate resting upon the activity of a suite of resistance genes operating in a far more variable and unpredictable environment.

Burdon is trying to understand the evolutionary pressures that cause gene-for-gene systems to evolve in some plants, and multi-gene defensive arsenals to arise in others. He says studies of the dynamics of host-pathogen interactions in natural ecosystems may suggest strategies that could be usefully exploited in agriculture.

Plant species and their specialised pathogens are locked in a perennial struggle for existence; when a chance mutation throws up some new variant of a resistance or virulence gene, it threatens the survival of the other. Natural selection winnows the population, leaving the survivors to face new cycles of challenge and counter-challenge.

Agriculture, through bitter experience, has learned that monoculture and genetic uniformity carry a high risk of local damage. For a plant species in the wild, the ideal strategy is not to grow in large, genetically uniform populations. For its part, the fungal pathogen must disperse a variety of strains throughout its host plant's range that constantly probe its defences.

Our bushwalker might come across a once-healthy patch of plants that has been devastated by a pathogen. Some distance away is a healthy patch of the same species. Burdon says these plants may have survived because they possessed a resistance gene that the local pathogen population could not overcome. But the plants may simply have been lucky; they may be just as susceptible as their neighbours, but did not come under attack from the pathogen.

Burdon says plant population biologists now talk of metapopulations: species in the

***Linum marginale* infected with the rust fungus *Melampsora lini*. This is one of the interactions that Dr Jeremy Burdon is using to investigate gene-for-gene systems in natural systems. Plants to the left carry a single gene that make them resistant to the rust attacking the plant on the right.**

Skeleton weed (*Chrondrilla juncea*) infected with the rust fungus *Puccinia chondrillina* illustrates another use of fungi: as biological control agents for use in the control of introduced weeds.

While we must be concerned about the possibility of exotic diseases entering Australia, some of our native diseases have spread overseas. Here a common groundsel plant in the United Kingdom is heavily infected with *Puccinia lagenophorae*, a rust endemic to Australia.

A common sight on Australian acacias: galls resulting from infection by the endemic Australian rust fungus *Uromycladium tepperianum*.

wild which exist as scattered populations with only limited gene flow between them. In time, they may evolve subtly different genetic structures. Pathogen populations evolve similar patterns.

'In a metapopulation, both the host and the pathogen are always present, he says. 'If the pathogen finds a particularly rich patch, the host gets the hell beaten out of it, while at a nearby patch, the plant may dominate, and the pathogen becomes locally extinct.

'This is why it is important to study plant-pathogen interactions in the wild, and at the level of the metapopulation. 'If we look at individual populations, we really can't say much about the evolutionary dynamics of what is happening. We need to study many populations within a larger area.'

At different locations within the geographic range of their metapopulations, the plant species and its pathogen may find themselves duelling with different genetic weapons. The action is local, the consequences global: Burdon believes these games of genetic chance, played out over millenia, can account for much of the seemingly random 'patchiness' typical of natural ecosystems, and the ephemeral nature of many plant populations.

These 'gene wars' involve another dimension: time. Over time, plants have evolved many different strategies to avoid their pathogens. For example, the genetic interactions between a pathogen and a herbaceous plant that dies back to an underground rootstock during winter or summer are likely to be quite different from those involving a tree species that lives for several centuries.

Gene-for-gene interactions may be particularly associated with systems in which the host plants are annuals or herbaceous perennials that periodically 'disappear' and in which the pathogen has no good way of surviving those poor times. In such situations, if the host's population is small (as it tends to be in natural ecosystems) the pathogen may even become locally extinct. This means that the host can recycle the same resistance genes from season to season.

'I can envisage a situation in which the same plant species will evolve a gene-for-gene defence against one pathogen, and a multi-gene system for another,' Burdon says.

For example, foliar pathogens that target the fresh leaves of a deciduous tree face similar problems in surviving the seasonal disappearance of their food resource. It may be this type of selection pressure that leads to gene-for-gene warfare between host and pathogen. Occasionally, the pathogen overcomes the host's resistance with a new virulence gene, with devastating consequences.

'In contrast, the same tree species represents a constantly present food resource to a pathogen like a canker that attacks the trunk,' Burdon says. 'For a tree that lives a long time, disease is probably inevitable. Here it's dangerous to base your defence on a single resistance gene that may be overcome, leaving you totally vulnerable. A multi-gene defence where resistance genes tend to slow the infection, rather than exclude it, may be better.'

Burdon says these strategies are the tip of an iceberg for interactions in nature which help to maintain biodiversity. Indeed, there are some good overseas data supporting the idea that fungal pathogens influence plant distribution.

That is certainly the case with the root-rot fungus *Phytopthora cinnamomi* which has decimated susceptible forests and heathlands in Western Australia. Plant disease experts now believe that *P. cinnamomi* is not native to Australia, which is why so many native plant species are vulnerable.

Burdon fears a repeat of the WA tragedy. 'As global trade and travel increase, we are seeing more weeds coming into Australia, and more and more pathogens,' he says. 'It worries me that other overseas pathogens could get into Australia and attack our native species.

'In South America, a fungus disease that evolved on guavas has spread to *Eucalyptus* plantations causing about 30% defoliation of the trees. Guavas and eucalypts are both members of the same family, the Myrtaceae. Our eucalypts and acacias are now widely grown throughout the world, and we may have built genetic bridges that increase the risk of exotic pathogens entering Australia and attacking our native flora.'

There is a important message here for conservation, Burdon says. 'If we are really interested in biodiversity as a whole, we shouldn't stop with the warm and cuddly species, or pretty plants. We need to look at a much broader range of species.'

More about plant pathogens

Burdon JJ (1996) The Dynamics of Disease in Natural Plant Populations. In *Frontiers of Population Ecology*, eds RB Floyd, AW Sheppard and PJ De Barro: pp 291-300. CSIRO, Melbourne.

An oat crop heavily infected with crown rust (*Puccinia coronata*) which uses weedy wild oat species as an alternative host.