Role of plant pathogens in food insecurity

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Plants are the primary means by which food is produced for living organisms. These include the species *Homo sapiens* – all 7 billion plus of us. But we are far from being the only species that depends on plants. There are many herbivores with which we compete, some of the most devastating being insects. Moreover, plants deemed useful as sources of food may be outcompeted by other plants of less practical use: these are often regarded as weeds. More insidiously, there are many infectious agents ranging from viroids, consisting of a few hundred nucleotides, through viruses, bacteria, mycoplasmas, nematodes and fungi to plants themselves that parasitise those crop plants we use as sources of food.

There is little doubt that plants have always been parasitised as they present a banquet of tempting nutrients to any organism with the necessary equipment to invade and absorb the goods on offer. Once humans started to cultivate plants as crops, about 10,000 years ago, these plant parasites became our enemies. Perhaps the earliest evidence of their recognition was in the Romans' sacrifice to the god, Rubigo, in order to avert attacks by a rust fungus on their wheat (Figure 1). However, it was the great Irish potato famine of the late 1840s that gave the impetus to the study and combating of organisms that cause disease of crop plants (Figure 2).

Many plant pathogens are highly destructive of crop plants and consequently threaten the food security of those who depend on them. The toll of the Irish potato famine is not known with great accuracy but it is estimated that about 1 million people out of a population of 8 million died of starvation and a further 1.5 million emigrated either to England, North America or Australia. Of those who took the longer voyages, about a quarter did not survive owing to their malnourishment and ill health on embarkation. The causal organism was long thought to be a fungus but more recently nucleic acid and protein sequencing have shown that it is a member of the Oomycetes, a group of organisms more closely related to the golden-brown algae[1]. The potato pathogen is known as *Phytophthora infestans* and the global damage it causes to the crop is estimated at $3 billion per annum. The organism is also a destructive pathogen of tomato.

P. infestans is an example of a hemibiotroph. These pathogens have a short period of "peaceful co-existence" with their host before necrosis sets in. Other pathogens are necrotrophs, causing death of the plant and living off the dying and dead cells or biotrophs, which maintain the plant host in a living condition but subvert its metabolism. There are numerous examples of all three types of parasitism so selection of examples is difficult.

Leaving *P. infestans* as our example of a hemibiotroph, an example of a necrotroph is *Ascochyta rabiei*, which causes Ascochyta blight of chickpea. It is a fungus that causes havoc in chickpeas grown in cool and moist climates. When the attack is heavy the plant blackens and dies, a disaster for countries such as Pakistan where people depend on it for sustenance, in particular because of its high protein content. How does the pathogen kill the plant? One possibility is that it produces toxins. Certainly *A. rabiei* produces compounds in culture that kill cells of chickpea. These were isolated and identified as Solanapyrones A, B and C (Figure 3). The question is, how important are they in the disease syndrome? Some evidence was obtained when cuttings of plants were placed in dilute solutions of one of the toxins and developed breakage of petioles, a characteristic symptom of the disease[2]. It seems probable that the cells surrounding the stele of the plant lose their turgor and, therefore, no longer provide sufficient support, the stele tissue alone being insufficient. Better evidence would be to produce knock-out mutants of the fungus lacking toxin production and to demonstrate that such mutants have lost virulence. Better still would be the demonstration that both toxin production and virulence were restored by reintroduction of the appropriate genes to the mutants. From a practical point of view, this would establish the rationale of using the toxins to select toxin-insensitive genotypes of chickpea which would be expected to be resistant to the pathogen. In addition, it might prove possible to produce such genotypes by genetic modification with genes that encode enzymes that destroy the toxins.

Stem rust of wheat, caused by *Puccinia graminis* f. sp. *tritici* is an example of a biotroph (Figure 1). The fungus enters the plant via

Figure 1. Stem rust of wheat caused by *Puccinia graminis* f. sp. *tritici.* Inset showing a close-up of a stem heavily infected with the fungus. The rust-coloured spores seen here are disseminated by wind, giving any surface they alight on *en masse,* such as farm equipment, a rusty appearance.

stomata, producing a substomatal vesicle from which infection hyphae ramify. From these haustoria penetrate the walls of cells but not their plasmalemmas. Concentrations of cytokinins, which are plant hormones, increase in the vicinity of the infection sites causing these areas to act as sinks for nutrients and thus feed the fungus at the expense of the host. Much of these nutrients goes to the production of huge numbers of spores of a rust colour. At harvest, these may cover the harvesting equipment, giving it the appearance of being rusty and also giving the fungus its trivial name.

Plant pathogens have proved to be slippery adversaries as, like all other organisms, they evolve and, ironically, it is our species, *Homo sapiens*, that has promoted this evolution. How can this be? The cause of the sometimes speedy evolution of plant pathogens lies in the domestication of crop plants and the selection of those that are resistant. Once Mendel's laws

Figure 2. A potato crop ravaged by *Phytophthora infestans,* the cause of the great Irish potato famine in the late 1840s Photograph courtesy of Alison Lees, The James Hutton Institute, Invergowrie, Scotland, UK.

of inheritance had been rediscovered and Biffen[3] had shown that resistance was a Mendelian trait, it became feasible to breed specifically for resistance. This has been very successful in some cases when the resistance has proved durable but, in many others, almost as fast as the breeder has produced a new resistant variety, the pathogen has also produced a variant that can overcome the resistance. Part of the reason for this rapid adaptation of the pathogen to the new variety is our predilection for growing genetically uniform crops over wide areas, providing a huge selection pressure for any pathogen able to overcome the plant's defences. Hence, the so-called "boom and bust" cycle of new cultivars – they become popular to grow because of their resistance to a given pathogen (boom) but then succumb to a variant of the same pathogen that can overcome the resistance (bust). At the genetic level, the relationship between those plants and their pathogens which behave in this way is described as "gene-for-gene" and was first established by the pioneering work of Flor[4] with flax (*Linum usitatissimum*) and its rust (*Melampsora lini*). More accurately, the relationship is described as "allele-for-allele".

The simplest expression of the gene-for-gene relationship is that for every gene encoding resistance in the plant there is a corresponding gene encoding avirulence in the pathogen. It follows that if the avirulence gene in the pathogen is eliminated or masked in some way, the pathogen is, once more, virulent. Not surprisingly, the nature of these gene pairs has excited

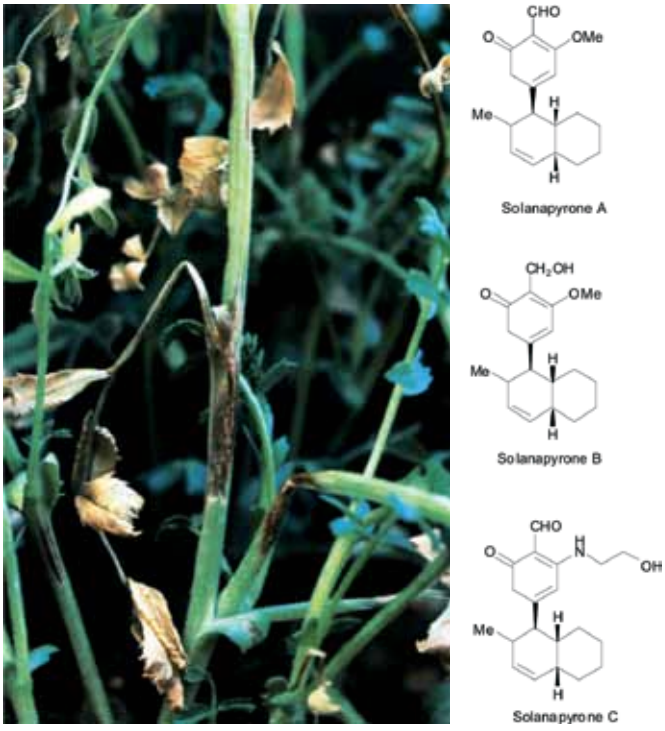

Figure 3. Symptoms of Ascochyta blight of chickpea and the structures of the toxins that may be responsible for them. Note the breakage of stems and petioles, symptoms which develop when cuttings are placed in dilute solutions of the toxin solanapyrone A.

considerable interest. The first report of the cloning of an avirulence gene was in 1984. Staskawicz and co-workers[5] cloned an avirulence gene from *Pseudomonas syringae* pv. *glycinea*, a pathogen of soybean. Sequence data showed that it encoded a single 100 kDa protein. Since these pioneering experiments, many avirulence genes have been cloned, not only from bacteria but also from fungi, nematodes and viruses and the molecules they are responsible for synthesising, termed effectors, have been isolated and identified.

What of the corresponding resistance genes? It took a little longer for the first resistance gene to be cloned because of the difficulty in locating the appropriate gene in the large amount of plant DNA. But in 1993 Martin and co-workers[6] were successful in cloning the *Pto* gene from tomato which confers resistance to *Pseudomonas syringae* pv. *tomato*. Since then a large number of resistance genes have been cloned and their structures determined including those from flax, the original plant for which the gene-for-gene concept was proposed, and its rust (for a review see reference 7). Here 30 resistance genes have been mapped to five loci (K, L, M, N and P) and 19 of these have been cloned. They all encode the same class of intracellular proteins, namely the Toll interleukin 1 receptor-nucleotide binding site-leucine-rich repeat (TIR-NBS-LRR). Most of the variation among these proteins occurs in the LRR domain and this is the region that is important in determining recognition of avirulence in the pathogen. Avirulence gene products of the pathogen are small proteins which are expressed in haustoria and are secreted into host cells.

Two general models have been proposed as to how the products of resistance and avirulence genes interact, directly and indirectly. Three examples of the direct model are the rice – rice blast pathogen, *Arabidopsis thaliana – Ralstonia solanacearum*, and tobacco – Tobacco Mosaic Virus. In the indirect model, resistance proteins detect changes in other plant proteins brought about by avirulence proteins. An example is the interaction of *A. thaliana* and *Pseudomonas syringae*. Here the protein products of resistance genes RPM1, RPS2 and RPS5 recognise changes in the plant proteins RIN4 and PBS1 caused by the presence of bacterial effectors.

There is now abundant evidence that resistance-avirulence gene pairs co-evolve and the reader is referred to the recent review of Brown and Tellier[8] for further information on this point. Moreover, the long-held view that necrotrophic fungi, "blast their way through host tissue with a battery of lytic and degradative enzymes" appears not to be true[9]. It seems that there are examples of these plant pathogens that also produce effectors similar to those of biotrophs but in these instances they are responsible for causing susceptibility rather than resistance.

While not wishing to discomfort the reader unduly, I hope the above short account has brought into focus the very real dangers presented by plant pathogens to the food security of the planet's human population. Many of these organisms are adept at eluding the resistance mechanisms of their hosts and are constantly evolving new biotypes that can successfully attack plants that were previously resistant. We need to be constantly on guard if we are to keep their ravages within bounds and avoid the famines they have caused in the past and have the potential to do so again.

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Biography

Dr Richard Strange is a plant pathologist and is currently Honorary Professor of Biology, University College London (UCL) and Honorary Research Fellow in the School of Biological and Chemical Sciences, Birkbeck College, University of London. He has published over 100 scientific papers and two books: *Plant Disease Control, Towards Environmentally Acceptable Methods* (1992) and *Introduction to Plant Pathology* (2003). He is co-founder (with Peter Scott) and Editor-in-Chief of the journal, *Food Security: the Science, Sociology and Economics of Food Production and Access to Food*, which began publication in the spring of 2009 and already has an impact factor of 1.658. www.springer.com/life+sci/agriculture/journal/12571

He is married to Lilian, a professional pianist, and they have two grown up children and four grandchildren. His main hobby is playing the 'cello and he performs regularly in recitals of the UCL Chamber Music Club.