PLANT DISEASE: A Threat to Global Food Security

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■ **Abstract** A vast number of plant pathogens from viroids of a few hundred nucleotides to higher plants cause diseases in our crops. Their effects range from mild symptoms to catastrophes in which large areas planted to food crops are destroyed. Catastrophic plant disease exacerbates the current deficit of food supply in which at least 800 million people are inadequately fed. Plant pathogens are difficult to control because their populations are variable in time, space, and genotype. Most insidiously, they evolve, often overcoming the resistance that may have been the hard-won achievement of the plant breeder. In order to combat the losses they cause, it is necessary to define the problem and seek remedies. At the biological level, the requirements are for the speedy and accurate identification of the causal organism, accurate estimates of the severity of disease and its effect on yield, and identification of its virulence mechanisms. Disease may then be minimized by the reduction of the pathogen's inoculum, inhibition of its virulence mechanisms, and promotion of genetic diversity in the crop. Conventional plant breeding for resistance has an important role to play that can now be facilitated by marker-assisted selection. There is also a role for transgenic modification with genes that confer resistance. At the political level, there is a need to acknowledge that plant diseases threaten our food supplies and to devote adequate resources to their control.

INTRODUCTION

More than 800 million people do not have adequate food; 1.3 billion live on less than \$1 a day and at least 10% of global food production is lost to plant disease (20, 34, 63). Plant pathologists cannot ignore the juxtaposition of these figures for food shortage and the damage to food production caused by plant pathogens.

Fourteen crop plants provide the bulk of food for human consumption (Figure 1). In addition, there are numerous plants that are less intensively grown yet fulfil important nutritional requirements in restricted areas of the globe. All are subject to disease both in the field and post-harvest, the major groups of pathogens being

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viruses, bacteria, Oomycetes, fungi, nematodes, and parasitic plants. These biotic constraints can, at times, seriously compromise food security. For example, potato blight, caused by *Phytophthora infestans*, struck Europe like "a bolt from the blue" in the 1840s. In Ireland, about a million people died of starvation and more than a million attempted to emigrate (80, 117). The reasons for this calamity were the arrival in Europe of a virulent strain of the pathogen, the high dependence of much of the Irish population on potato for sustenance, the lack of resistance in the plant to the pathogen, and weather conditions favorable to epidemic development. There have been other disasters caused by plant diseases such as the Great Bengal Famine of 1943 (98) and the southern corn leaf blight epidemic of 1970–1971 in the USA (130), to name but two. In the former, an estimated 2 million people died owing to the high dependence of most of the population on a single crop, rice, which was attacked by the fungus *Cochliobolus miyabeanus*. In the USA, by contrast, although in some areas the corn (maize) crop was completely destroyed by another fungus from the same genus, *Cochliobolus heterostrophus*, alternative sources of nutrition were plentiful so no one died, although the effect on the agricultural economy was severe.

The first two of these painful examples demonstrate with brutal clarity that in areas of the world where a large proportion of the population is dependent on a single crop or a few crops, they are at risk should that crop fail owing to one or more devastating diseases. At the present time, the threat is particularly great in developing countries, where populations are growing fastest, poverty is endemic, the population depends on locally produced staples, and the infrastructure of extension and R & D is often poorly resourced.

Globalization of agriculture has meant that crop plants, often with a narrow genetic base, are now grown far from their centers of origin and therefore far from the pathogens that have coevolved with them. As a result, they are unlikely to have evolved resistance to new strains of the pathogen that may have subsequently arisen in the center of origin. Should these new strains migrate to the new area of cultivation, as may have happened with *P. infestans* in the 1840s, the crop would be susceptible. Moreover, crops introduced to a new area may be poorly equipped to resist pathogenic organisms that may be resident there.

The problem of plant disease, particularly in developing countries, is exacerbated by the paucity of resources devoted to their study. In part, this may be a result of poor governance but it also arises from the difficulty of quantifying plant disease and relating this to the failure of crops to reach achievable yields. The inability to supply such hard data to administrators and politicians has meant that plant pathology, in relation to its importance, continues to be grossly underfunded. We hope that this review will help plant pathologists, particularly those in developing countries, to focus on the acquisition of such data. Moreover, once armed with hard statistics, it is hoped they will be more successful in securing adequate and appropriate funding from governments and other agencies in the public and private sectors in order to tackle effectively the plant diseases that destroy their most important crops.

Other difficulties faced by plant pathologists are the reliable identification of the causal agents of a disease to levels—species, formae speciales, pathovars, biovars and races—that are appropriate to their properties as plant pathogens and therefore relevant to their control; identification of sources of inoculum; the wide host range of some pathogens; lack of availability of sources of durable genetic resistance; and the slow acceptance in some cultures of the value of transgenic traits in plant improvement. Tropical climates, prevalent in most developing countries, cause particular difficulties in that they allow continuous cropping with the consequent build-up of inoculum. In contrast, the winters of temperate climates reduce inoculum levels owing to the reduced availability of hosts and inclement weather.

Solutions to the problems of plant disease come from many quarters. Exclusion of the pathogen through plant quarantine is the first line of defense and deserves more resources in relation to food security in developing countries. Other solutions include the exclusion, elimination or reduction of the pathogen's inoculum and development, for example by good cultural practices; intercropping and rotation; the judicious use of pesticides; exploiting the gene pool of the plant and its relatives in breeding programs (often the preferred approach where it is practical); understanding and combating virulence mechanisms of the pathogen; biological control (often difficult for plant pathogens); post-harvest protection; and improving plant performance through biotechnology. Current cultural reservations in public opinion about biotechnology should not inhibit its imaginative application to the challenging demands of food security, especially in developing countries. Maintenance of genetic diversity in crop plants has an overriding importance.

In this review, some examples of organisms that pose the greatest hazard to the successful cultivation of the world's most important edible crop plants are considered. Areas covered are pathogen identification, disease severity, epidemiology, and the evolution of virulence. The discussion then moves to options for control. In the first instance, these center on the exclusion and elimination of pathogens and, where these have failed, crop husbandry practices that limit the impact of disease. An important factor in control is the availability of resistant plants with favorable agronomic characteristics, and therefore the means by which they may be generated will be treated, including marker-assisted selection (MAS) and exploitation of knowledge of the biochemistry and molecular biology of plant-pathogen interaction. Finally, consideration is given to the feasibility and desirability of using biotechnology to ensure that sufficient food of high quality is available to all in the twenty-first century. Because this is a review of plant disease, we have given little attention to biologically caused post-harvest losses—though these have a major additional effect on global food security, and the problems they cause deserve more research and development than they receive. At the political level, the International Society for Plant Pathology has established a Task Force on Global Food Security. The challenges that it addresses attract less attention than those of global terrorism in the current climate of world opinion, but they present threats of a different

degree, which need to be addressed through science-based co-operation on a global scale.

WHAT ARE THE THREATS?

The Crop Plants that are the Principal Sources of Human Nutrition and their Most Important Pathogens

These are given in Table 1. Although this is not the place to give a detailed account as to how each pathogen threatens food security, some examples of diseases caused by the major types of pathogens have been chosen to demonstrate the vulnerability of some crop plants that are of global importance or of critical importance to communities living in more restricted areas.

Virus Diseases

Many of the more than 700 known plant viruses cause devastating diseases and often have wide host ranges. Barley yellow dwarf viruses (BYDV), for example, are distributed worldwide and infect over 150 species of the Poaceae, including most of the staple cereals—wheat, barley, oats, rye, rice, and maize (Table 1). A number of studies have demonstrated the debilitating effect these viruses have on cereals. For example, grain yield of maize infected with the PAV serotype of the virus was 15%–20% less than controls because infected plants have fewer kernels per ear (12), whereas in barley, infected with the same serotype, reductions of up to 38% were recorded (29).

Cassava is a very important food security crop for smallholder farmers, particularly in low-income, food-deficit countries, owing to its reputation for reliability (8). World production from 2000 to 2003 averaged 185 million tons/year with just over 100 million tons harvested in Sub-Saharan Africa (35). However, the plant is subject to diseases caused by mosaic viruses. Originally there were thought to be two strains, African cassava mosaic virus (ACMV) and East African cassava mosaic virus (EACMV), but in the late 1980s an unusually severe form of the disease was reported from Uganda. This was caused by double infection with a recombinant virus derived from ACMV and EACMV, termed UgV, and one of the parental strains, ACMV (139). In 1999, Legg reported that the area affected by the severe form of the disease had expanded to cover virtually all Uganda and large parts of Kenya, Tanzania, Sudan, and the Democratic Republic of Congo (85). Also, such was the sensitivity of local cultivars and the consequent losses that many farmers had abandoned cassava cultivation, consequently destabilizing food security in East Africa (85). More recently, the disease has spread to Rwanda (86), Burundi (14), and Gabon (87).

Diseases of cassava caused by mosaic viruses are not known in South America, the center of origin of the crop (36). The African experience is therefore a good illustration of the hazard of exporting a crop to another country where it may fall victim to a pathogen acquired from the local flora.

TABLE 1 The world's staple[a] crops and their principal diseases

Crop	Principal diseases				
	Fungal	**Viral**	**Nematode**	**Bacterial**	**Oomycete**
Barley (*Hordeum vulgare*)	Mildew: *Erysiphe graminis* (*Blumeria graminis*) Spot blotch: *Cochliobolus sativus* Scald: *Rhynchosporium secalis* Scab: *Gibberella zeae* Rusts: *Puccinia* spp. Net blotch: *Pyrenophora teres* Barley stripe: *Pyrenophora graminea* Smuts: *Ustilago* spp.	Yellow dwarf: barley yellow dwarf luteovirus Stripe mosaic (barley stripe mosaic hordeivirus)	Root-knot nematode (*Meloidogyne* sp.) Cyst nematode (*Heterodera* sp.) Root-lesion nematode (*Pratylenchus* sp.)		
Cassava (*Manihot esculenta*)	Anthracnose: *Colletotrichum gloeosporioides* (*Glomerella cingulata*)	Cassava mosaic: African cassava mosaic geminivirus, East African cassava mosaic geminivirus, Indian cassava mosaic geminivirus		Bacterial blight: *Xanthomonas axonopodis* pv. *manihotis*	
Lentil (*Lens culinaris* ssp. *culinaris*)	Wilt: *Fusarium oxysporum* f.sp. *lentis* Blight: *Ascochyta lentis* (*Didymella lentis*) Rust: *Uromyces viciae-fabae* Vascular wilt: *Fusarium oxysporum* f.sp. *lentis* Anthracnose: *Colletotrichum truncatum*				
Maize (*Zea mays*)	Northern corn leaf blight: *Helminthosporium turcicum* (*Setosphaeria turcica*)	Chlorotic dwarf: maize chlorotic dwarf machlovirus		Stewart's wilt: *Erwinia stewartii*	Downy mildew: *Sclerospora* spp. and others

(Continued)

TABLE 1 (*Continued*)

Crop	Principal diseases				
	Fungal	**Viral**	**Nematode**	**Bacterial**	**Oomycete**
	Southern corn leaf blight: *H. maydis* (*Cochliobolus heterostrophus*) Rust: *Puccinia* spp. Smut: *Ustilago zeae* Stalk and ear rots: *Gibberella zeae*, *Diplodia* spp. and others	Streak: maize streak geminivirus Yellow dwarf: barley yellow dwarf luteovirus		Corn stunt disease: *Spiroplasma kunkelii*	
Millet					
Common millet (*Panicum miliaceum*)					Downy mildew: *Sclerospora graminicola*
Finger millet (*Eleusine coracana*)	Blast: *Pyricularia setariae* Leaf blight: *Cochliobolus nodulosus*				
Foxtail millet (*Setaria italica*)	Blast: *Pyricularia setariae* Rust: *Uromyces setariae-italicae* Smut: *Ustilago crameri*				Downy mildew: *Sclerospora graminicola*
Pearl millet (*Pennisetum glaucum*)	Ergot: *Claviceps fusiformis*				Downy mildew: *Sclerospora graminicola*
Teff (*Eragrostis tef*)	Rust: *Uromyces eragrostidis* Head smudge: *Helminthosporium miyakei*				
Oats (*Avena sativa*)	Crown rust: *Puccinia coronata* Stem rust: *Puccinia graminis*	Yellow dwarf: barley yellow dwarf luteovirus Mosaic: oat mosaic potyvirus		Halo blight: *Pseudomonas syringae* pv. *coronafaciens*	

Crop	Fungal diseases	Viral diseases	Bacterial diseases	Oomycete diseases
	Powdery mildew: *Erysiphe graminis* (*Blumeria graminis*)	Golden stripe: oat golden stripe furovirus		
	Smut diseases: *Ustilago avenae* and *U. hordei*			
	Leaf blight: *Phaeosphaeria avenaria*			
	Various diseases caused by *Fusarium* spp., including root rot and crown rot			
	Seedling blight: *Glomerella graminicola*			
	Snow mold: *Monographella nivalis*			
	Leaf blotch: *Pyrenophora avenae* (*P. chaetomioides*)			
	'Groat-blackening' mainly caused by *Alternaria alternata*, *A. tenuissima*, and *Cladosporium* spp.			
Potato (*Solanum tuberosum*)	Early blight: *Alternaria solani*	Leafroll: potato leafroll luteovirus	Bacterial wilt: *Ralstonia solanacearum*	Late blight: *Phytophthora infestans*
	Black scurf: *Rhizoctonia solani*	Mosaic viruses: notably potato X potexvirus, potato Y potyvirus	Bacterial soft rot: *Erwinia carotovora*	Pink rot: *Phytophthora erythroseptica*
			Common scab: *Streptomyces scabies*	
			Bacterial ring rot: *Clavibacter michiganensis* subsp. *sepedonicus*	
Rice (*Oryza sativa*)	Blast: *Magnaporthe grisea*	Rice tungro disease: rice tungro spherical machlovirus, rice tungro bacilliform badnavirus	Bacterial leaf blight: *Xanthomonas oryzae* pv. *oryzae*	
	Brown spot: *Cochliobolus miyabeanus*	Yellow dwarf: barley yellow dwarf luteovirus		
	Sheath blight: *Rhizoctonia solani*			

(Continued)

TABLE 1 (*Continued*)

Crop	Principal diseases				
	Fungal	**Viral**	**Nematode**	**Bacterial**	**Oomycete**
Rye (*Secale cereale*)	Snow mold: *Monographella nivalis* Brown rust: *Puccinia recondita* Ergot: *Claviceps purpurea* Eyespot: *Tapesia yallundae* Sharp eyespot: *Rhizoctonia solani* Powdery mildew: *Erysiphe graminis* (*Blumeria graminis*) Stem rust: *Puccinia graminis* Glume blotch: *Phaeosphaeria nodorum* Leaf blotch: *Rynchosporium secalis*	Yellow dwarf: barley yellow dwarf luteovirus	Eelworm: *Ditylenchus dipsaci*		
Sorghum (*Sorghum bicolor*)	Grain molds: complex of fungal pathogens, predominantly *Cochliobolus lunatus*, *Fusarium* spp. and *Mycosphaerella holci* Anthracnose: *Glomerella graminicola* Leaf blight: *Setosphaeria turcica* Zonate leaf spot: *Gloeocercospora sorghi* Tar spot: *Phyllachora sorghi* Charcoal rot: *Macrophomina phaseolina* Rust: *Puccinia purpurea* Ergot: *Claviceps sorghi*	Streak disease: maize streak geminivirus			Downy mildew: *Peronosclerospora sorghi*
Soybean (*Glycine max*)	Rust: *Phakopsora pachyrhizi* Anthracnose: *Colletotrichum truncatum* and *Glomerella glycines* Purple seed stain: *Cercospora kikuchii*	Mosaic: soybean mosaic potyvirus Yellow mosaic: bean yellow mosaic potyvirus		Bacterial pustule: *Xanthomonas axonopodis* pv. *phaseoli*	Downy mildew: *Peronospora manshurica*

Crop	Fungal diseases	Viral diseases	Nematodes	Bacterial/other
	Pod and stem blight: *Diaporthe phaseolorum* var. *sojae*			
Sweet potato (*Ipomoea batatas*)	Scab: *Sphaceloma batatas* (*Elsino batatas*)	Feathery mottle: sweet potato feathery mottle potyvirus	Root-knot nematode: *Meloidogyne* spp.	Soil rot: *Streptomyces ipomoea*
	Fusarium wilt: *Fusarium oxysporum*			Little leaf: sweet potato little leaf phytoplasma
	Black rot: *Ceratocystis fimbriata* Java black rot: *Botryodiplodia theobromae* Scurf: *Monilochaetes infuscans*			
Wheat (*Triticum aestivum*, bread wheat; *Triticum turgidum*, durum wheat)	Stem rust: *Puccinia graminis* f.sp. *tritici*	Yellow dwarf: barley yellow dwarf luteovirus		Bacterial leaf streak or black chaff: *Xanthomonas translucens* pv. *undulosa*
	Leaf rust: *Puccinia recondita* f.sp. *tritici* Stripe or yellow rust: *Puccinia striiformis* Spot blotch: *Cochliobolus sativus* Head scab and foot/root rot: *Fusarium* spp. Sclerotium foot rot: *Corticium rolfsii* Tan spot: *Pyrenophora tritici-repentis* Powdery mildew: *Erysiphe graminis* (*Blumeria graminis*) Speckled leaf blotch: *Mycosphaerella graminicola* Glume blotch: *Phaeosphaeria nodorum* Alternaria leaf blight: *Alternaria* spp. Loose smut: *Ustilago nuda* f.sp. *tritici* Rhizoctonia root rot: *Rhizoctonia* spp.			
Yam (*Dioscorea alata*)	Anthracnose: *Colletotrichum gloeosporioides* (*Glomerella cingulata*)	Yam virus complex: including yam mosaic potyvirus, yam mild mosaic potyvirus		
	Tuber rots: *Fusarium* spp., *Penicillium* spp., *Rosellinia* spp.)			

aCrops identified as staples in FAO (33).

Bacterial Diseases

Species of bacteria belonging to several genera are devastating plant pathogens. For example, at least 350 different plant diseases are caused by species of *Xanthomonas* (88). One of these is bacterial blight caused by *Xanthomonas oryzae* pv. *oryzae* (Table 1). It is a major disease of rice in tropical Asia and all varieties of basmati rice are highly susceptible. Unfortunately, the pathogen is present in the entire basmati rice-growing area in India and, in consequence, production is severely constrained (70).

Recently, a severe disease of banana has been reported from Uganda (129). The symptoms were rapid yellowing and wilting of younger leaves, discoloration of the internal vascular vessels, and occasional dieback and internal rotting of banana fruits. These symptoms are very similar to moko disease caused by *Ralstonia solanacearum*, but this pathogen had not previously been recorded on banana in Africa. A bacterium was isolated from diseased bananas and proved to be the causal organism by the application of Koch's Postulates. Identification was made by comparing DNA fingerprints of the bacterium obtained from rep-PCR, using ERIC and BOX primers (90), and by showing that these matched fingerprints obtained from authentic cultures of *X. campestris* pv. *musacearum*, originally isolated from *Ensete* and *Musa* in Ethiopia (129).

Ralstonia (*Pseudomonas*) *solanacearum*, a plant pathogen with a worldwide distribution, causes diseases in more than 200 host species belonging to 50 families including potato (112) (Table 1). It exists as five races with different hosts and geographic distributions. Race 1 has a very wide host range and is endemic in the southern United States. Bananas are the principal host of race 2, in which, as noted above, it causes moko disease. This race is present in the Caribbean, Brazil, and the Philippines. Race 3 is distributed worldwide with the exception of the United States and Canada and infects many members of the Solanaceae such as potatoes, tomatoes, eggplant, and peppers, as well as geraniums. Ginger is affected by race 4 in much of Asia and mulberries in China by race 5. The organism is further differentiated into biovars on the basis of biochemical tests, e.g., race 3 biovar 2, which is present in Europe, Asia, South and Central America, and Australia (22; http://www.aphis.usda.gov/ppq/ep/ralstonia/background.html). Recently, this organism was detected in the United States in some greenhouses that had imported geranium plants from Kenya. The identification was confirmed in February 2003 and the organism eradicated. However, it was detected again in late December 2003, and the facility in Guatemala where the infected geraniums originated has stopped shipping to the United States (http://www.aphis.usda.gov/ppq/ep/ralstonia/background.html).

Fungal Diseases

There are at least five reasons why fungi may cause catastrophic plant disease:

1. Many sporulate prolifically, the spores providing copious inoculum, which may infect further plants.

2. Their latent period, i.e., the time between infection and the production of further infectious propagules, usually spores, may be only a few days.

3. The spores, if they are wettable, may be spread as high-density inoculum in surface water or in droplets by rain-splash. Alternatively, nonwettable spores may be carried long distances by the wind.

4. They may produce compounds that are phytotoxic and/or a battery of enzymes that destroy the plant's structure.

5. Pathogens may draw nutrients away from the economically valuable part of the plant by the production or induction of growth regulators, such as cytokinins, and consequently depress yields (117).

Colletotrichum gloeosporioides (anamorph) or *Glomerella cingulata* (teleomorph) are names given to an organism that causes diseases, often referred to as anthracnoses, in many tropical and subtropical crops. Considerable variation occurs in culture and host range, with some strains able to attack many host species whereas others, such as those infecting mango, are confined to a single species (52). This variation has cast doubt on whether *C. gloeosporioides* can really be considered as a single species, and it has been suggested that it is a species complex (119). Here, molecular techniques are invaluable in defining strains. For example, molecular approaches demonstrated that *C. gloeosporioides* infecting *Stylosanthes* in Australia consisted of two clonal populations that do not combine readily in the field and that probably resulted from two separate introductions into the country (94). Similarly, isolates from yam (*Dioscorea alata*), a weed (*Mikania micrantha*), kava (*Piper methysticum*), and pigeonpea (*Cajanus cajan*) obtained from islands in the South Pacific were identical when compared by Amplified Fragment Length Polymorphism (AFLP), suggesting that they were clonal. Also, isolates from cassava and coffee obtained from Hainan Island, China, produced identical AFLP profiles, but these were different from the South Pacific profile and the profile obtained from a strain isolated from infected cassava in Ghana (2).

The wide host range of some strains of *C. gloeosporioides* presents a considerable threat to crops growing in countries where there is no fallow period corresponding to the winter of temperate climates. Even if time is allowed to elapse between the plantings of susceptible crops, the fungus may survive on weed species. Moreover, care has to be taken, if intercropping with other species is practiced, that these are not also susceptible to the prevalent strain of *C. gloeosporioides*.

Rice is second only to maize in global production (Figure 1) and is more important since it is a staple for about half the world's population, among whom are many of the poorest. It is attacked by the Ascomycete fungus *Pyricularia oryzae* [teleomorph *Magnaporthe grisea*], causing rice blast, resulting in losses of 10%–30% of the crop every year (121). To give one example, more than 700 ha of rice of diverse genotypes with varying levels of resistance in Bhutan were affected in 1995, resulting in losses of 1090 tonnes (126). Other cereals are also affected by *P. oryzae* or the similar species *P. setariae*; these include finger millet, *Eleusine coracana*, which, when attacked before grain formation, can suffer complete loss

of yield (30) (Table 1). Such an attack has serious consequences in India and southern and east African countries where finger millet is an important food security crop. A species of *Pyricularia* has also been reported as an increasingly important problem on wheat, and serious outbreaks have occurred in Paraná, Brazil (61).

Several fungi that infect staple foods before and/or after harvest produce powerful mycotoxins. For example, the fumonisin toxins were discovered as a result of an investigation into the high level of esophogeal cancer in the Transkei region of South Africa. They were isolated from cultures of *Gibberella fujikuroi* (anamorph *Fusarium moniliforme*) grown on maize, and the most active compound was designated fumonisin B_1 (FB_1) (42). FB_1 is a sphinganine analogue that, in both plant and animal cells, competitively inhibits sphingolipid biosynthesis, causing sphingoid bases to accumulate (95). High levels of virulence for maize were always associated with strains of the fungus that produced fumonisins (27).

Oomycete Diseases

Although the Oomycetes share the first four characteristics of fungi enumerated above that make them such dangerous pathogens of plants, they differ from them in several important features. These include the absence of chitin from their cell walls, the negligible sterol content of their membranes, their predominantly diploid karyotype, and their biflagellate zoospores. Nucleic acid and protein sequence data have now shown that they are members of the Kingdom Stramenopila and are more closely related to the golden brown algae than to true fungi (5). An important consequence of their independent evolution of the plant parasitic lifestyle from that of fungi is that some plant defenses such as the production of saponins and chitinases, which specifically target the sterols of fungal membranes and chitin, respectively, are ineffective against the Oomycetes.

The genus *Phytophthora* (meaning plant destroyer) is an Oomycete, and *P. infestans*, mentioned in the Introduction, is perhaps its most notorious species, costing annually on a global basis in excess of $5 billion in terms of losses of the potato crop and control measures (15). *Pythium* is another genus that can cause serious disease problems. For example, *P. aphanidermatum* and *P. myriotylum* cause severe root rot in bell pepper with 42% and 62% plant mortality, respectively (19). Similarly, members of the downy mildews such as *Peronosclerospora, Peronospora, Pseudoperonospora, Plasmopara, Sclerophthora*, and *Sclerospora* regularly cause severe diseases on a variety of crops that include maize, sorghum, pearl millet, onion, soybean, cucurbits, and grapes (125) (Table 1).

Nematode Diseases

Of the 17 orders of nematodes, only two, the Tylenchida and the Dorylaimida, contain members that are plant parasites (135). However, those that are parasitic may cause serious crop losses. For example, *Ditylenchus dipsaci* attacks over 450 different plant species, including weeds, and is one of the most devastating nematode species (45). Similarly, species of *Meloidogyne* such as *M. hapla* also

attack many important crop plants, including groundnut, potato, carrot, sugarbeet, strawberry, and onion, and on occasions, may cause total crop loss (32, 91). Other species such as the soybean cyst nematode (*Heterodera glycines*) are more specific. Often its direct effects on crop losses are not noticed in the field but yields may be severely depressed. For example, losses in the southeastern United States were estimated at $88.4 million in 1990 (113). Nematodes that are members of the Dorylaimida may also act as vectors for viruses. For example, *Tobacco rattle virus*, which causes considerable economic damage to vegetable and ornamental flower-bulb crops, is efficiently transmitted by *Paratrichodorus anemones* (72).

Diseases Caused by Parasitic Plants

There are over 3000 species of parasitic plants among which *Striga* and *Orobanche* are, perhaps, the most notorious (116). Species of *Striga* infect more than two thirds of the 73 million ha of cereals and legumes grown on the African continent, affecting the lives of over 100 million people in 25 countries. Losses may be total, and infestation of some areas, owing to the enormous seed production by the pathogen, may be so great that the cultivation of some crops has to be abandoned (31). Broomrapes (*Orobanche* species) are parasitic weeds that are widespread in many countries of Central Asia and the Mediterranean region (65). They infect the roots of dicotyledonous crops and, because they do not contain chlorophyll, derive their entire nutrient requirements from their hosts. An aerial shoot is produced that may support several hundred flowers, each containing around 500 seeds, but by this time, the health and productivity of the host plant has been severely compromised. Like *Striga*, these pathogens may remain dormant in the soil for >10 years but still cause serious economic losses if susceptible crops are planted (24).

HOW SERIOUS ARE THE THREATS?

To evaluate the seriousness of a plant disease it is essential to identify the pathogen, determine the effect it is causing, identify the sources of inoculum, and gain some knowledge of the means by which it is spread. Although these stipulations appear elementary, they are frequently ignored because of lack of personnel and facilities for such work. In consequence, the disease develops and spreads unchecked. Such problems are most severe in developing countries, sometimes reflecting poor governance but also the lack of availability of resources to identify and quantify disease outbreaks and to relate them to the failure of crops to reach achievable yields. In the absence of a clear presentation of this relationship, plant pathology is likely to remain severely underfunded in relation to its importance for food security—a problem in both industrialized and developing countries.

Knowing the Enemy—Pathogen Identification

The correct identification of plant pathogens can hardly be overemphasized since it is fundamental to control. Initially, pathogens are often identified by the symptoms

they cause. Although such identifications can be reliable when staff are experienced, they can frequently be wrong when such experience is not available. For example, it is difficult to avoid the conclusion that some formae speciales of *Fusarium oxysporum* are diagnosed purely on the basis of the vascular discoloration and wilts they cause, coupled with the name of the plant from which they were isolated. Indeed, recently a highly toxigenic isolate of a *Fusarium* obtained from chickpea was demonstrated by its morphology and rDNA sequences to be *F. acutatum* rather than *F. oxysporum* f. sp. *ciceri* (S. Gopalakrishnan & R.N. Strange, submitted).

Fortunately, a number of aids are available to plant pathologists with minimal facilities. At the field level there is the CABI Crop Protection Compendium (24), which provides a simple basis for preliminary identification through narrowing down the possibilities in a relational database of potential causes. The dimensions in the relational database allow the user to specify (*a*) the crop (say rice: of nearly 4000 plant pathogens listed in the Compendium, some 114 are recorded as affecting rice as a major host); (*b*) the country (say India: this reduces the number of possibilities to some 72); (*c*) the part of the plant affected (say the leaves: this reduces the number of possibilities to some 43); and (*d*) the symptom (say yellowed or dead leaves: this reduces the number of possibilities to some 8). A conclusion is reached in a few seconds. Each item in the final list is accompanied by a thumbnail illustration, and then leads to a full description of the condition, its cause, how damaging it is, and what can be done about it, together with illustrations and distribution maps.

At the laboratory level, many techniques are now available for definitive identification of plant pathogens, ranging from those that are simple and do not require expensive apparatus to the complex, which require costly equipment (reviewed in 107). Enzyme Linked Immunosorbent Assay (ELISA) is the preferred technique of diagnostic laboratories for bacteria and viruses because of its high throughput capacity (50, 105, 109), but PCR (polymerase chain reaction) techniques are rapidly becoming established. In the *Laboratory Guide for Identification of Plant Pathogenic Bacteria* (109) many primers for important plant pathogenic bacteria are listed. Real-time PCR is increasingly being used but the cost is prohibitive for many countries where it could be of most benefit. However, some lower-cost portable thermocyclers have been developed that can give real-time results in the field. For example, both watermelon fruit blotch, caused by *Acidovorax avenae* subsp. *citrulli,* and Pierce's disease of grape, caused by *Xylella fastidiosa*, were diagnosed in 1 h or less on-site (108, 110, 111).

PhytID (99) offers a molecular key to 50 species of *Phytophthora*, for which accurate identification is crucial to plant health legislation and plant quarantine issues. The system is based on the Internal Transcribed Spacer (ITS) regions of ribosomal RNA. Digest patterns are entered into an analysis package or simply compared with samples for different species. If a match can be found, unknown isolates may be identified in a matter of hours rather than the several days required for traditional morphological analysis.

If a known organism has been reliably identified as the cause of disease, then referral to the literature will often give invaluable help in evaluating the risk it poses. The *Crop Protection Compendium* (24) includes an Economic Impact module, and its descriptions of each of the major diseases include an expert assessment of the damage they do.

Rapid detection is of the essence and may be the key to success in containment and control (107). However, any doubt about the identity of the causal agent should be removed by the application of Koch's postulates, suitably modified in the case of obligate pathogens.

Measurement of Disease and Its Effect on Yield

The measurement of disease continues to be an area fraught with difficulty. In the first instance, plants are irregularly shaped three-dimensional objects. Second, the symptoms caused may range from a few isolated lesions to wilting and collapse of the whole plant. Third, there is usually no simple relationship between measures of symptoms and the failure to reach achievable yields (117). Although image analysis may be of help in obtaining an objective measure in some instances, e.g., with detached leaves bearing discrete lesions, it is generally of little help in evaluating disease in the field, particularly when this involves hundreds of lines in a trial. Here the time-honored practice of scoring on an ordinal scale, often of 1–9, is still the most common approach. However, little credence can be placed in such scores when they are not made in comparison with agreed standards. Generally, these should take the form of a hand-held key.

The measurement of yield is less controversial and is usually scored in terms of weight or number (117). However, the relation of disease severity to yield may not be obvious and requires empirical measurement. Various models—critical-point, multiple-point, and Area Under the Disease Progress Curve (AUDPC)—have been described (117). However, yield may be more directly related to the duration and amount of tissue that remains healthy (10). Integrated estimates of the relative damage done by diseases in different regions and environments, to arrive at comparative values for economic impact, are especially challenging (reviewed in 41, 92, 97, 106).

Identifying Sources of Inoculum and Its Means of Dissemination

Once the causal agent of a disease has been identified and its potential effects ascertained, either by reference to the literature or by experimentation, to be sufficiently significant to warrant control, the identification of sources of inoculum and its dissemination become immediate priorities. Infected seed, propagative material, or intact plants are often the means by which a pathogen may traverse long distances and establish itself in a new country. For example, race 3 biovar 2 of *Ralstonia solanacearum* was imported into the United States in infected geranium plants from Kenya and Guatemala, as noted above (http://www.aphis.usda.gov/ppq/ep/

ralstonia/background.html). Similarly, blight of chickpea caused by *Ascochyta rabiei* was imported into the U.S. Pacific states and Australia on infected seed (40, 71, 76). Unfortunately, deliberate release of plant pathogens by terrorists cannot be ruled out (93).

Virulence Characteristics of Pathogens, Their Nature, Dissemination, and Evolution

Plant pathogens are in a constant state of flux. Not only do they move in space and time, but they also evolve. In particular, they evolve variants capable of overcoming the resistance of genotypes of crop plants that are widely grown and that may have been the results of years of work by plant breeders. It is essential, therefore, if we are to control these pathogens, that we understand the nature of pathogenicity, how it may be transferred between organisms, and how it may evolve.

For an organism to be a plant pathogen, it has to have a number of virulence functions such as the production of degradative enzymes (53, 59, 104, 136), toxins (9, 77, 134), and "effector" molecules (1, 78, 131). The last are the products of *avr/pth* genes. These are genes that are necessary for pathogenicity in host plants but confer avirulence in resistant plants. For example, in *Xanthomonas citri* the *pthA* gene was originally identified as a pathogenicity factor because it controls the ability of the bacterium to multiply in citrus plants and cause cankers. However, the same gene in *X. campestris* pvs. *phaseoli* and *malvacearum* confers avirulence on bean and cotton, respectively (84).

Interference with induced resistance is the primary target of some virulence factors. For example, a number of pathogens can convert the phytoalexins produced by their hosts to less toxic compounds. Genetic studies of populations of the pea pathogen *Nectria haematococca* have shown that metabolism of the phytoalexin pisatin is an important virulence mechanism of the fungus and that the genes required to cause disease in pea are clustered on a chromosome that is dispensable for normal growth (23).

Interference with the induction of defense may be an important virulence mechanism of bacterial pathogens (25). The *hopPtoM* and *avrE* genes of *Pseudomonas syringae* encode suppressors of salicylic acid-dependent callose deposition and promote cell death. Moreover, these genes are widespread in bacterial pathogens of plants, suggesting that they are important general virulence components (25). The attack on the signaling pathways of plant defense is likely to prove a fertile field of discovery in the future for those interested in virulence mechanisms of plant pathogens. For example, an isolate of *Colletotrichum gloeosporioides* metabolized salicylic acid in culture (2). Since this compound is fundamental to both genetic and systemic acquired resistance (26, 39), its destruction may go some way to explaining the wide host range of some strains of this pathogen.

Other genes necessary for pathogenicity or virulence have been uncovered by gene disruption experiments. For example, when genes encoding two proteins, ECP1 and ECP2, that are expressed during the infection of tomato by *Cladosporium*

fulvum were disrupted, virulence was attenuated (81). ECP2 was the more important of the two since strains deficient in the production of this protein colonized tomato leaf tissue poorly and produced little emerging mycelium and few conidia.

Pathogenicity genes may be disseminated among potential plant pathogens by horizontal gene transfer. In bacterial plant pathogens, virulence genes and regulatory genes required for pathogenicity are often found in blocks, called pathogenicity islands, on the bacterial chromosome (78). These differ in G + C content from that of the bacterial species containing them, implying their acquisition from another organism. For example, the horizontal transfer of a pathogenicity island carrying the *nec1* virulence gene and flanking sequences is thought to have occurred among *Streptomyces* strains in the Diastatochromogenes cluster (18). Moreover, it was postulated that the peptide synthase gene *txtAB,* which is required for the synthesis of a toxin, thaxtomin A, is located on the pathogenicity island and that this has conferred plant pathogenicity on saprophytic strains (18).

Horizontal gene transfer is also thought to occur in fungi (103) and may be an important mechanism for the acquisition of pathogenicity. For example, the pathogenicity cluster of *N. haematococca* (see above) is thought to have been acquired by horizontal gene transfer because its genes differ in G + C content and codon usage from genes in other chromosomes. Also, homologs have been found in another pea pathogen, *Fusarium oxysporum* f.sp. *pisi*, but are uncommon among other fungi that are closely related phylogenetically. Moreover, homologs have also been found in *Neocosmospora boniensis*, which is also able to cause disease in pea (124).

Genes required for the synthesis of host-selective toxins of strains of *Alternaria alternata* provide another example of possible horizontal gene transfer in fungi. Two genes, *ATK1* and *ATK2*, from the Japanese pear pathotype of *A. alternata* were necessary for both production of the host-selective AK-toxin and pathogenicity for certain genotypes of pear (122). Homologs of these genes were found in strawberry and tangerine pathotypes of *A. alternata*, which produce toxins that are similar in structure to AK-toxin, but not in other pathotypes nor in nonpathogenic strains. *AKT* homologs have been found in multiple copies on a 1.05-Mb chromosome of a strawberry pathotype (51). The chromosome, like the one from *N. haematococca* that is responsible for the metabolism of pisatin (23), appeared to be conditionally dispensable since it was not essential for saprophytic growth.

So far virulence attributes of pathogens that are positive have been discussed. However, when considering gene-for-gene interactions, the absence or alteration of avirulence genes are important causes of virulence. For example, those races of the tomato pathogen *Cladosporium fulvum* that are virulent for tomatoes with the *Cf9* gene lack the corresponding avirulence gene *avr9* (68). In contrast, three races of the pathogen that were virulent for tomatoes with the *Cf4* gene had *Avr4* genes that were modified by a single base change, A to G, which occurred at a different position in each race. The result of the changes was that the proteins contained a tyrosine residue (codon TAT) rather than a cysteine residue (codon

TGT). The modified proteins were unstable in the plant and therefore failed to trigger a resistance response (68).

Although loss or point mutations could explain the evolution of races of *C. fulvum* that are virulent to tomatoes with the *Cf9* and the *Cf4* genes, respectively, other causes of mutation may also be at work with other plant-pathogen systems. The *avrBs1* gene of the bell pepper pathogen *X. campestris* pv. *vesicatoria*, which causes bacterial spot of the plant, is one example. Here the gene is frequently disrupted by an insertion sequence, IS476, rendering the corresponding resistance gene, *Bs1*, ineffective. Ironically, selection for the insertion sequence was promoted by the use of copper sprays that were used to protect the plant against the disease, the reason being that they selected for bacteria containing a plasmid with a copper resistance gene that also contained three copies of the insertion sequence (74).

No doubt other virulence factors will be discovered in the future. Exactly what these are will become plainer in the next few years with the increasing use of microarrays to determine which genes are expressed during pathogenesis and gene disruption experiments (79, 89, 101, 114).

HOW CAN THE THREATS BE MINIMIZED?

Exclusion, Elimination, or Reduction of Inoculum

The most fundamental approach to the management of a disease is to ensure that it is not present through exclusion (quarantine) or eradication (28). National Plant Protection Services assume responsibility for protecting their countries from the unwanted entry of new pathogens and pests and for coordinating programs to eradicate those that have recently arrived and are still sufficiently confined for their elimination to be realistic. Regional plant protection organizations, for example, the European and Mediterranean Plant Protection Organization (EPPO), play a role in coordinating such "phytosanitary" activities across a region (115), and the Secretariat of the International Plant Protection Convention has a global responsibility in this field. While developed countries are sometimes concerned with the rare event of malicious introduction of pathogens as a form of bioterrorism, there is enormous scope for simply strengthening the resources and capacity of plant protection services in developing countries, with the goal of improving food security (3).

Plant material may be tested for specific pathogens, and a number of rapid and sensitive techniques are available (reviewed in 107, 109). Material that is found to be infected can then be destroyed. Once a pathogen is endemic in a region, much can be done to limit the amount of inoculum produced and its effectiveness. Seed and propagative material may be treated with biocides, and infected plants in the field may be rogued or the infected parts pruned (36, 128).

There are many other ways in which the farmer can take action to reduce disease in the field. Inoculum from crop residues may be reduced by crop rotation (in practice, this is one of the most important approaches to disease control), by

plowing (118) or by adjusting planting time, and from the soil by solarization (73), the addition of amendments (83), and in some instances, by flooding (127). Some pathogens require specific wavelengths of light in order to sporulate (102). Their prevalence in greenhouses may be reduced by coating the glass with materials that do not allow the penetration of such wavelengths while still allowing through the light of wavelengths important for photosynthesis (102).

In the field, however, when an attack is imminent, there is little alternative to the application of pesticides, although by forecasting the occurrence of conditions favorable to the pathogen, the number of applications and the amount of pesticide used may be reduced (55). Many crops do benefit from routine or managed application of pesticides and this remains one of the principal control methods available for pathogens, especially fungi and nematodes, and their vectors, especially insects and nematodes (53a). In developing countries the use of pesticides is critically important for seed health. Pesticide use is particularly appropriate in systems of integrated pest management (94a), in which the aim is to create a sustainable balance of complementary approaches to disease control, avoiding excessive use of pesticides whose side-effects may be unwanted.

Some control of windborne pathogens may be achieved by planting mixtures of varieties or multilines. For example, an experiment on the control of rice blast caused by *Magnaporthe grisea* was carried out over a large area in Yunnan Province, China. Susceptible sticky rice was planted as single rows interspersed between four or sometimes six rows of resistant hybrid rice. Grain production of sticky rice was 89% greater and blast 94% less when planted in mixtures compared with monocultures (140). This principle may be extended to intercropping whereby different species of plants are grown in close proximity. However, care must be taken to ensure that no two or more members of the mixture are susceptible to the same organism, a condition that is not easy to fulfil with pathogens that have a wide host range. Diseases caused by pathogens such as mycoplasmas, viruses, and viroids are transmitted by vectors. It follows that control of vector mobility, for example by means of reflective mulches or interference with their feeding habits on plants by the use of genotypes with sufficient concentrations of antifeedants, would reduce infection by these agents (43, 47).

While there are many examples of successful use of biological control agents to control insect pests of crops, there are far fewer cases of successful use of such methods for field management of plant diseases (18a). Nevertheless, benefit is probably often achieved from unrecognized effects of biological control as when, for example, incorporation of crop residues into the soil affects the soil microflora to the disadvantage of a soilborne pathogen.

Rendering Crop Plants Genetically Resistant to Disease

One of the most effective approaches to disease management is the use of genetically resistant plants. Although the genetic basis for resistance to a plant disease was first elucidated only in 1905 (13), selection for disease resistance has probably

been practiced by farmers and later by plant breeders since the beginning of agriculture. Spontaneous variation in disease resistance can be selected, and genes for resistance can be introduced by hybridization.

Genetic resistance to disease, once secured, is a low-cost method of control. Like other approaches to disease management, it need not be complete to be useful. Rather, it is more important that it should have the characteristic of durability, a concept proposed by Johnson (66). Durable resistance, i.e., resistance that remains effective over many years, can only be recognized retrospectively. It may not be readily available since pathogens have a facility, as described above, to evolve virulent variants, a phenomenon that has been much studied in terms of mechanism, genetics, and epidemiology. However, knowledge of the biochemistry and molecular biology of disease has identified some instances where durability may be expected (see below). Furthermore, examples of resistance that have been shown by experience to be durable can be used in breeding programs with the expectation that their effect will again be durable.

Advances in genomics have demonstrated that a considerable proportion, 1%–2%, of a plant's genome is devoted to resistance genes or genes with similar properties that could conceivably confer resistance to a pathogen possessing a complementary avirulence gene. Such genes are often clustered or occur in tandem repeats, suggesting that resistance genes with different specificities arise by gene duplication followed by intragenic and intergenic recombination, gene conversion, and diversifying selection (96). Maintenance of this genetic flux is crucial to the survival of the plant and therefore for maintenance in the gene pool from which the plant breeder can draw.

In gene-for-gene systems, resistance is triggered by the direct or indirect recognition of an avirulence gene product of the pathogen by a resistance gene product of the plant (16, 132). Breeding plants with resistance against a specific disease in such interactions requires the identification of resistant plants, which are then crossed with agronomically acceptable but susceptible plants. A program of backcrossing to the susceptible parent and selection of resistant phenotypes leads to the production of plants that are similar to the susceptible parent but have the required resistance. Typically, this process takes 10 or more years, and by this time, in some instances, the pathogen has already evolved a variant that is not recognized by the hybrid, leading to susceptibility.

Johnson (67) has described the challenge of breeding for resistance to two contrasting diseases of wheat: yellow (stripe) rust (caused by *Puccinia striiformis*) and eyespot (caused by *Pseudocercosporella herpotrichoides*). *P. striiformis* occurs as many races, differing genetically in their virulence to wheat varieties with different resistance genes, in a gene-for-gene relationship. The development of a new race can suddenly negate years of effort by a plant breeder, as happened in the United Kingdom to the variety Joss Cambier in 1971, causing fears of a crisis like the one due to southern corn leaf blight in the United States at the same time (see below), even though its slow-rusting quality had raised hopes of durability. Resistance to *P. herpotrichoides* is normally durable and not race-specific, but only

moderate in degree. Introduction of a higher degree of resistance required cytogenetic manipulation to allow recombination between wheat and a wild relative from North Africa, *Aegilops ventricosa*, which possessed resistance. A block of genes was introduced into wheat that was undefined (as is normal in plant breeding) but contained one or more genes for resistance. Coming from an alien plant species, this might now be regarded with suspicion by some as being transgenic, although the procedure then appeared benign and a valuable application of novel genetic technology.

Marker-assisted selection (MAS) can help the breeder since it not only reduces the reliance on laborious and fallible screening procedures but also allows the pyramiding of resistance genes (60). Moreover, MAS may be used in the selection of genotypes with quantitative resistance to pathogens (17). The essential feature of any marker is that it should be tightly linked to the gene specifying the required character—in this case, resistance. For example, two microsatellite markers were found to cosegregate with two resistance genes for rice blast (38). Similarly, MAS is now routine in bean breeding programs for resistance to several diseases such as bean rust, anthracnose, common bacterial blight, and white mold (75).

Maintaining the Genetic Diversity of Crop Plants

Why is genetic diversity desirable in crops? An example will show how uniformity can lead to genetic vulnerability.

An unexpected and devastating epidemic of southern corn leaf blight (caused by *Cochliobolus heterostrophus*) occurred on the maize crop in the United States in 1970, resulting in an estimated loss of 1 billion bushels of the crop with a value of \$1 billion (123, 130); adjusted for inflation, this would have a value of more than \$4 billion in 2005. Apart from its use as human food, maize was the principal component of livestock feed, and it was a major export commodity. The impact was such that the U.S. Department of Agriculture and indeed U.S. President Nixon were drawn into reacting to what was seen as a potential agricultural crisis. Farmers' organizations in the U.S. corn belt brought legal actions against companies that had supplied their seed, for reasons that will become clear.

The epidemic proved to be due to the rapid evolution of a new race of the pathogen, Race T. This race is specific for maize that has a particular cytoplasm containing the gene *T-cms* and renders such crops highly susceptible to attack. The so-called Texas cytoplasm (T-cytoplasm) was widespread in 1970 as it contained a valuable mitochondrial gene that conferred male sterility and was consequently used in the production of hybrid seed.

Maize seed is normally produced as an F1 hybrid between different parents, because the hybrid vigor released in the F1 generation results in significantly enhanced performance. Maize is naturally self-pollinating, so some intervention is needed to produce F1 hybrid seed: for example, the pollen-producing tassels can be removed by hand. The male-sterilizing effect of T-cytoplasm was universally attractive as a means of reducing the cost of hybrid seed production. The plants were

self-sterile so they did not need to be manually detasseled. This cytoplasmically based technology was indeed a revolution in plant breeding. It suited the seed companies, and it suited the farmers. These were the factors that led to at least 80% of the US maize crop being sown with cultivars based on the Texas cytoplasm by 1970.

The critical characteristic of Race T of *C. heterostrophus* is that it produces a toxin specific for the mitochondria of the T-cytoplasm, causing necrotic lesions on the leaves, stalks, and ears, which in turn produce inoculum that may lead to rapid spread of the disease. Symptoms can be severe, and as a result the plant may die and produce no useful yield.

The nearly universal use of cultivars with T-cytoplasm by 1970 selected strongly for Race T in the population of *C. heterostrophus*. It is unusual for cytoplasmic genes to affect resistance to disease, and it would not easily have been predicted that the widespread adoption of a particular cytoplasm would result in a new and damaging epidemic.

The scientific and public impact of the Race T episode provoked widespread concern about what was called genetic vulnerability (56). This valuable concept is meant to carry a warning that genetic uniformity leads to the potential for problems, if they occur, to be widespread and intense. Granted that it was difficult to predict the emergence of Race T, we might now think that it should have been easier to be forewarned that the very widespread use of T-cytoplasm rendered the maize crop vulnerable to something.

The term genetic vulnerability is less familiar now, and some related concepts that currently command greater attention, such as biodiversity and perhaps factory farming, do not carry the same warning that uniformity itself brings potential danger—especially from plant pathogens. It has been convincingly argued that genetic mixtures possess greater stability of performance and that their inherent resistance to disease is rendered more effective and more durable by their diversity (133). Yet uniform crops are convenient and financially efficient in the short term, and in practice they remain the norm. In most environments, a degree of genetic diversity is more readily achieved across the farm, or across a region, whether by accident or design.

Exploiting the Knowledge of the Biochemistry and Molecular Biology of Disease in Order to Increase Resistance

Plant pathogens are often at their most vulnerable at the pre-penetration stage of infection and at their most responsive to environmental influence. For example, *Striga* may be induced to germinate in the absence of the host by the introduction of ethylene into the soil, although this is a hazardous procedure. However, a strain of *Pseudomonas syringae* pv. *glycinea* has been found that produces ethylene and that consistently stimulated better germination of seed of the parasite than the gas (11). This discovery holds out the prospect that amendment of soil with this organism might lead to control of *Striga* owing to its ability to stimulate germination of seed of the parasite in the absence of a suitable host.

Other procedures for enhancing resistance include exploiting a pathogen's dependence on certain virulence mechanisms. For example, *X. albilineans*, causal agent of sugarcane leaf scald, produces a family of toxins and antibiotics known as the albicidins, which selectively block DNA replication in bacteria and chloroplasts. In consequence, sugarcane infected by the bacterium is chlorotic. During a screening program, an albicidin-resistant isolate of *Pantoea dispersa* was found that efficiently detoxified albicidin and was effective as a biocontrol agent (137).

As mentioned above, the secretion of two proteins, ECPI and ECP2, by *Cladosporium fulvum* was required for full expression of virulence of this tomato pathogen. When a construct of the gene encoding ECP2 was made with potato virus X (PVX::*ECP2*) and used to infect a range of tomato genotypes, four were found that responded with a systemic hypersensitive response. This reaction was encoded by a single dominant gene, designated *Cf-ECP2*, that recognizes the protein ECP2. Since this factor is required for virulence, the resistance conferred by *Cf-ECP2* is expected to be durable (82).

The GM Option

With the recognition that tumor induction in plants by *Agrobacterium tumefaciens* is caused by the transformation of its hosts with T-DNA present on a plasmid in the bacterium, the use of this vector to transform plants has become almost routine, although other techniques such as electroporation and biolistics are also used. The question then arises as to what genes should be considered for introduction into crop plants. Given the severe limitations of agricultural technology, especially in developing countries, to provide secure food supplies for a rapidly growing global population, the opportunity to introduce entirely new characteristics into plants has been seized upon energetically by plant breeders, through transgenic techniques—often described as genetic modification (GM). Disease resistance is among the most promising applications of GM. However, the growth of transgenic technologies in agricultural practice has been limited by public opinion in some countries. Whatever precautions are taken in their development and testing, transgenic plants are regarded by some as unnatural and potentially dangerous. This climate of opinion, especially in developed countries, has unfortunately limited the adoption of novel and potentially valuable opportunities for crop improvement—especially in developing countries, where new developments are most needed to enhance food security.

This attitude could change as the benefits of transgenic technologies become more apparent in practice. Indeed, during the period 1996 to 2003, the global area of transgenic crops increased 40-fold, from 1.7 to 67.7 million ha. Furthermore, 30% of this area is grown in developing countries, where the recent rate of increase has been higher than in industrialized countries (64).

An interesting discussion of the potential of biotechnology for solving intractable constraints to food security in Africa is provided by Gressel et al. (48) and, in a more general approach, by Christou & Twyman (20). Candidate genes

include those that inhibit or destroy virulence attributes of the pathogen, such as degradative enzymes and toxins, and those that enhance defense. The latter include genes that increase the concentrations of saponins, antimicrobial peptides, and reactive oxygen species or modify the phytoalexin response, and those that switch on systemic acquired resistance. Paradoxically, transformation of plants with pathogen genes such as coat protein and replicase genes of viruses confers resistance, a phenomenon known as pathogen-derived resistance. Finally, the ability to transform plants allows the pyramiding of resistance genes, including those that are normally found as alleles at the same locus (117). Space does not permit the extensive discussion of all these possibilities, so mention will only be made of some of the more recent and most promising new developments.

Apple has been transformed with a stilbene synthase gene from grapevine and a polygalacturonase-inhibiting protein (PGIP) from kiwi (120). The stilbene synthase catalyzes the synthesis of resveratrol, a phytoalexin found in both grapevine and peanuts but not apples. It is therefore probable that apple pathogens would be sensitive to it since they would be unlikely to have evolved a mechanism for detoxifying the compound. Chemical analysis demonstrated the presence of a resveratrol glycoside in transformed plants, and the presence of the PGIP gene was demonstrated by PCR and Southern blot analysis. Although studies of the disease resistance of the transgenic apples are still awaited, precedents in the literature suggest that they will be more resistant to fungal pathogens than the untransformed types (49, 100).

Transformation of rice with the *afp* gene of *Aspergillus giganteus*, which encodes the antifungal AFP protein, has been achieved and the gene demonstrated to be stably integrated and inherited. Protein extracts of the transgenic plants were antifungal, and detached leaves from the plants showed varying levels of resistance to rice blast caused by *Magnaporthe grisea* (21). In other experiments with rice, overexpression of a thionin gene from oats had resistance to *Burkholderia plantarii* that was virtually complete (62).

As already mentioned, an isolate of *Pantoea dispersa* has been found that was an effective biocontrol agent for sugarcane leaf scald caused by *X. albilineans*, probably owing to its ability to degrade the albicidin toxins produced by the pathogen (137). Sugarcane was transformed with the gene responsible for toxin degradation. Transgenics, expressing the gene at levels of 1–10 ng of enzyme per mg leaf protein, did not develop the chlorotic symptoms that are characteristic of the disease when challenged with the pathogen, whereas the controls were severely affected (138).

Peanut cultivars transformed by microprojectile bombardment of embryogenic callus derived from mature seeds with the coat protein gene of peanut stripe potyvirus gave rise to plants that were resistant or immune to the virus. The transgenics are now an important germplasm resource for countries where peanut stripe virus is endemic (54). Farming of papaya (*Carica papaya*) in Hawaii was threatened by papaya ringspot virus. Initially, control was obtained by cross protection with a mild strain of the virus (44). However, more recently a transgenic approach has been adopted in which plants have been transformed with the coat protein of the virus (6, 44). Although some coat protein genes tended to give high levels of

protection only to homologous strains of the virus, others gave broad-spectrum resistance (6, 7, 37).

There is a vigorous global debate about the scientific and ethical acceptability of transgenic crops—those that are genetically modified through biotechnology. It will probably seem surprising in the future that this caused so great a cultural divide at the turn of the twenty-first century. But what is beyond doubt is that, in these early stages of a revolution in the technology of plant improvement, the number genes being deployed is small.

Transgenic crops in commercial production (currently maize, soybean, cotton, and canola) occupied 68 Mha in 18 countries in 2003, and this rose by 15% from 2002 (64). At present, only two traits are the subject of the commercialized transgenic biotechnology: herbicide (glyphosate) tolerance, and insect resistance conferred by the *Bt* gene from *Bacillus thuringiensis*. This means that new technologies to fight weeds and insects are in place, in itself a remarkable and positive step. Technologies have to start somewhere, and this has started with just two traits. The time will soon come when there are many genes for insect and disease resistance in play, and then we may feel more secure from the genetic vulnerability that may be presented at the current stage of development of the technology.

THE CHALLENGE OF THE FUTURE

The challenge of the future may be viewed at two levels, the biological and the political. At the biological level there is clearly much to be done to expand our knowledge of plant pathogens and our ability to deal with them. At the political level, plant pathologists should persistently draw these opportunities to the attention of politicians and public and private fund holders until they make appropriate provision for the subject, which is vital to the support of human life in industrialized and especially in developing countries.

The Biological Challenge

Plant pathogens pose an enormous problem for global food security. In the first instance we are still ignorant of the identity of many damaging pathogens that currently exist. Second, our means of measuring the severity of the symptoms they cause is often subjective and qualitative rather than objective and quantitative. Third, there are few examples in which a definite and dependable coefficient has been established that links the severity of symptoms to failure to reach obtainable yields. Fourth, plant pathogen populations are variable in time and space. Although there have been many epidemiological studies, much remains to be done in the field of limiting inoculum, its multiplication, effectiveness, and spread. Finally, and potentially most dangerously, plant pathogens are genetically variable. Nobody predicted the occurrence of the recombination of African Cassava mosaic virus (ACMV) with East African Cassava mosaic virus (EACMV) to produce a strain, UgV. Nor was it predicted that the UgV strain, in combination with ACMV in doubly infected plants, would give rise to an unusually severe form of the disease

causing numerous farmers in Africa to abandon growing this vital staple crop. Also, nobody predicted that the near-exclusive reliance on the *T-cms* gene for male sterility in maize in the United States would presage the epidemic of southern corn leaf blight in 1970, one of the worst plant disease epidemics ever recorded. This stands as a warning that genetic uniformity of even a small part of the genome of a widely grown crop risks the hazard of a major epidemic of a plant pathogen with the ability to exploit the trait(s) that it governs.

It is difficult to predict the origin of the next plant disease catastrophe that will affect one or other of our crops vital to food security in some part of the globe. However, of two things we can be certain: There is inadequate control of known diseases in many parts of the world today, and in the future there will be some unpleasant shocks from pathogens that have evolved new virulence characteristics.

The Political Challenge

In many countries, politicians and their constituents are preoccupied by the threat to peace and prosperity posed by terrorism. It is time that they were also concerned with the terrorism perpetrated by plant pathogens and pests on our sources of food, which threaten our very security and survival.

There are no easy solutions to poverty. Scientists can, however, contribute in important ways to the more tangible issue of food security. For plant pathologists, the loss of food to plant disease presents a challenge. Experience shows that there is always some new threat to food security, so the challenge is ongoing. The people most strategically placed to meet that challenge on behalf of the undernourished are the staff of the national programs in the affected countries. But these are precisely the institutions that are under-resourced to meet the challenge.

This dilemma is somewhat relieved by the organizations with resources from the developed world that have a concern for food security in developing countries, and by their support for national programs there. The Consultative Group for International Agricultural Research is of primary importance, with its mission to bring the benefits of modern science to poor farmers "living on the fringes of the global economy" (22a). Eleven of its globally distributed International Agricultural Research Centers have programs in the management of plant disease, each Center being mandated to handle certain crops: Africa Rice Center (WARDA), Centro Internacional de Agricultura Tropical (CIAT), Center for International Forestry Research (CIFOR), Centro Internacional de Mejoramiento de Maiz y Trigo (CIM-MYT), Centro Internacional de la Papa (CIP), International Center for Agricultural Research in the Dry Areas (ICARDA), International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), International Institute of Tropical Agriculture (IITA), International Plant Genetic Resources Institute (IPGRI), International Rice Research Institute (IRRI), World Agroforestry Centre (ICRAF).

Internationally, the Food and Agriculture Organization (FAO) of the United Nations plays a key role in addressing the challenge of food security, including the depravations of plant disease (34). And throughout the developed world, national

governments have development-assistance agencies that support agricultural development in developing with countries, sometimes with specific attention to plant disease issues.

At a voluntary level, the International Society for Plant Pathology (ISPP) is among many scientific organizations that are deeply concerned for global food security and the contribution that science can make to it. Being involved in ISPP's programs, we welcome participation in the Society's small effort to address these issues through a Task Force on Global Food Security (http://www.isppweb.org/foodsecurity_background.asp). We present this as a small example of an action-oriented project designed to address pressing local issues. The initiative was stimulated by a public meeting on "Global Food Security: the Role for Plant Pathology" during the seventh International Congress of Plant Pathology in Edinburgh in 1998. It was recognized then that more than 800 million people do not have adequate food, that 1.3 billion live on less than $1 a day, and that at least 10% of global food production is lost to plant disease. The Task Force has initiated activities in (a) changing public policy and opinions on global food security; (b) postgraduate training for plant pathologists in developing countries; (c) quantification of the economic impact of some major diseases; (d) farmer training in simple disease management: pilot project in Ghana; and (e) development of the ISPP website.

The project in Ghana is on development of appropriate strategies to control cassava diseases. Its aims are to (a) develop appropriate measures to control root rot disease of cassava; (b) create and increase farmers' awareness of diseases of cassava, particularly the root rot caused by *Polyporus sulphureus*; (c) train and equip farmers with simple skills that will enable them to identify diseases and control them on their farms; and (d) develop simple educational materials on diseases and their control to improve extension delivery. Those expected to benefit include over 80% of 18 million people in Ghana who depend on cassava as a major staple food, as well as industries that depend on cassava as a raw material.

The efforts of an international society with voluntary officers and limited funding can only be sadly inadequate in relation to the enormity of the problem of providing food for the world and securing its availability into an uncertain future.

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LITERATURE CITED

1. Abramovitch RB, Martin GB. 2004. Strategies used by bacterial pathogens to suppress plant defenses. *Curr. Opin. Plant Biol.* 7:356–64

2. Aiyere T. 2004. *Molecular characterization of isolates of* Colletotrichum gloeosporioides *from tropical crops and virulence factors of the fungus*. M. Phil. thesis. London Univ., U.K.

3. Asher R, Ben-Ze'ev IS, Black R. 1996. Plant clinics in developing countries. *Annu. Rev. Phytopathol.* 34:51–66

4. Deleted in Proof

5. Baldauf SL, Roger AJ, Wenk-Siefert I, Doolittle WF. 2000. A kingdom-level phylogeny of eukaryotes based on combined protein data. *Science* 290:972–77

6. Bau HJ, Cheng YIH, Yu TA, Yang JS, Yeh SD. 2003. Broad-spectrum resistance to different geographic strains of *Papaya ringspot virus* in coat protein gene transgenic papaya. *Phytopathology* 93:112–20

7. Bau HJ, Cheng YH, Yu TA, Yang JS, Hsiao CH, et al. 2004. Field evaluation of transgenic papaya lines carrying the coat protein gene of *Papaya ringspot virus* in Taiwan. *Plant Dis.* 88:594–99

8. Bellotti AC, Smith L, Lapointe SL. 1999. Recent advances in cassava pest management. *Annu. Rev. Entomol.* 44:343–70

9. Bender CL. 1999. Chlorosis-inducing phytotoxins produced by *Pseudomonas syringae*. *Eur. J. Plant Pathol.* 105:1–12

10. Bergamin A, Carneiro SMTP, Godoy CV, Amorim L, Berger RD, Hau B. 1997. Angular leaf spot of *Phaseolus* beans: relationships between disease, healthy leaf area, and yield. *Phytopathology* 87:506–15

11. Berner DK, Schaad NW, Volksch B. 1999. Use of ethylene-producing bacteria for stimulation of *Striga* spp. seed germination. *Biol. Control* 15:274–82

12. Beuve M, Naibo B, Foulgocq L, Lapierre H. 1999. Irrigated hybrid maize crop yield losses due to *Barley yellow dwarf virus—*PAV luteovirus. *Crop Sci.* 39:1830–34

13. Biffen RH. 1905. Mendel's laws of inheritance and wheat breeding. *J. Agric. Sci.* 1:4–48

14. Bigirimana S, Barumbanze P, Obonyo R, Legg JP. 2004. First evidence for the spread of East African *Cassava mosaic virus*-Uganda (EACMV-UG) and the pandemic of severe cassava mosaic disease to Burundi. *Plant Pathol.* 53:231

15. Birch PRJ, Whisson SC. 2001. *Phytophthora infestans* enters the genomic era. *Mol. Plant Pathol.* 2:257–63

16. Bonas U, Lahaye T. 2002. Plant disease resistance triggered by pathogen-derived molecules: refined models of specific recognition. *Curr. Opin. Microbiol.* 5:44–50

17. Bormann CA, Rickert AM, Ruiz RAC, Paal J, Lubeck J, et al. 2004. Tagging quantitative trait loci for maturity-corrected late blight resistance in tetraploid potato with PCR-based candidate gene markers. *Mol. Plant Microbe Interact.* 17:1126–38

18. Bukhalid RA, Takeuchi T, Labeda D, Loria R. 2002. Horizontal transfer of the plant virulence gene, *nec1*, and flanking sequences among genetically distinct *Streptomyces* strains in the Diastatochromogenes cluster. *Appl. Environ. Microbiol.* 68:738–44

18a. Butt T, Jackson C, Magan N, eds. 2001. *Fungi as Biocontrol Agents: Progress, Problems and Potential.* Wallingford, UK: CAB Int.

19. Chellemi DO, Mitchell DJ, Kannwischer-Mitchell ME, Rayside PA, Rosskopf EN. 2000. *Pythium* spp. associated with bell pepper production in Florida. *Plant Dis.* 84:1271–74

20. Christou P, Twyman RM. 2004. The potential of genetically enhanced plants to address food insecurity. *Nutr. Res. Rev.* 17:23–42

21. Coca M, Bortolotti C, Rufat M, Penas G, Eritja R, et al. 2004. Transgenic rice plants expressing the antifungal AFP protein from *Aspergillus giganteus* show enhanced resistance to the rice blast fungus *Magnaporthe grisea*. *Plant Mol. Biol.* 54:245–59

22. Collonnier C, Mulya K, Fock I, Mariska I, Servaes A, et al. 2001. Source of resistance against *Ralstonia solanacearum* in fertile somatic hybrids of eggplant (*Solanum melongena* L.) with *Solanum aethiopicum* L. *Plant Sci.* 160:301–13

22a. CGIAR Secretariat. 2004. *Consultative Group on International Agricultural*

Research: Annual Report 2003. Washington, DC: CGIAR Secretariat.

23. Covert SF, Enkerli J, Miao VPW, VanEtten HD. 1996. A gene for maackiain detoxification from a dispensable chromosome of *Nectria haematococca*. *Mol. Gen. Genet.* 251:397–406

24. *Crop Protection Compendium*. 2004. CD-Rom. Wallingford, UK: CAB Int. http://www.cabi.org/compendia

25. DebRoy S, Thilmony R, Kwack Y-B, Nomura K, He SY. 2004. A family of conserved bacterial effectors inhibits salicylic acid-mediated basal immunity and promotes disease necrosis in plants. *Proc. Natl. Acad. Sci. USA* 101:9927–32

26. Delaney TP, Uknes S, Vernooij B, Friedrich L, et al. 1994. A central role of salicylic-acid in plant-disease resistance. *Science* 266:1247–50

27. Desjardins AE, Plattner RD, Nelsen TC, Leslie JF. 1995. Genetic-analysis of fumonisin production and virulence of *Gibberella fujikuroi* mating population A (*Fusarium-Moniliforme*) on maize (*Zea-Mays*) seedlings. *Appl. Environ. Microbiol.* 61:79–86

28. Ebbels DL. 2003. *Principles of Plant Health and Quarantine*. Wallingford, UK: CAB Int.

29. Edwards MC, Fetch TG, Schwarz PB, Steffenson BJ. 2001. Effect of *Barley yellow dwarf virus* infection on yield and malting quality of barley. *Plant Dis.* 85:202–7

30. Ekwamu A. 1991. Influence of head blast infection on seed-germination and yield components of finger millet (*Eleusine coracana* L Gaertn). *Trop. Pest Manag.* 37:122–23

31. Estabrook EM, Yoder JI. 1998. Plant-plant communications: rhizosphere signaling between parasitic angiosperms and their hosts. *Plant Physiol.* 116:1–7

32. Evans K, Trudgill DL, Webster JM. 1993. *Plant Parasitic Nematodes in Temperate Agriculture*. Wallingford, UK: CAB Int.

33. FAO. 1995. *Dimensions of Need: An Atlas of Food and Agriculture*. Rome, Italy: FAO, UN. ISBN 92-5-103737-X

34. FAO. 2000. *The state of food insecurity in the world* (*SOFI*). Rome, Italy: FAO, UN. www.fao.org/FOCUS/E/SOFI00/sofi001-e.htm

35. FAO. 2004. *FAOSTAT: FAO statistical databases*. Rome, Italy: FAO, UN. http://faostat.fao.org/

36. Fauquet C, Fargette D. 1990. African *Cassava mosaic virus*—etiology, epidemiology, and control. *Plant Dis.* 74:404–11

37. Fermin G, Inglessis V, Garboza C, Rangel S, Dagert M, Gonsalves D. 2004. Engineered resistance against *Papaya ringspot virus* in Venezuelan transgenic Papayas. *Plant Dis.* 88:516–22

38. Fjellstrom R, Conaway-Bormans CA, McClung AM, Marchetti MA, Shank AR, Park WD. 2004. Development of DNA markers suitable for marker assisted selection of three Pi genes conferring resistance to multiple *Pyricularia grisea* pathotypes. *Crop Sci.* 44:1790–98

39. Gaffney T, Friedrich L, Vernooij B, Negrotto D, Nye G, et al. 1993. Requirement of salicylic-acid for the induction of systemic acquired-resistance. *Science* 261:754–56

40. Galloway J, MacLeod WJ. 2003. *Didymella rabiei*, the teleomorph of *Ascochyta rabiei*, found on chickpea stubble in Western Australia. *Aust. Plant Pathol.* 32:127–28

41. Gaunt RE, 1995. The relationship between plant disease severity and yield. *Annu. Rev. Phytopathol.* 33:119–44

42. Gelderblom WCA, Jaskiewicz K, Marasas WFO, Thiel PG, Vleggaar R, Kriek NPJ. 1988. Fumonisins—novel mycotoxins with cancer promoting activity produced by *Fusarium moniliforme. Appl. Environ. Microbiol.* 54:1806–11

43. Gibson RW, Pickett JA. 1983. Wild potato repels aphids by release of aphid alarm pheromone. *Nature* 302:608–9

44. Gonsalves D. 1998. Control of *Papaya ringspot virus* in papaya: a case study. *Annu. Rev. Phytopathol.* 36:415–37

45. Goodey JB, Franklin MT, Hooper DJ. 1965. *The Nematode Parasites of Plants Catalogued Under Their Hosts*. Wallingford, UK: CAB Int.

46. Deleted in proof

47. Greenough DR, Black LL, Bond WP. 1990. Aluminum-surfaced mulch—an approach to the control of *Tomato spotted wilt virus* in solanaceous crops. *Plant Dis.* 74:805–8

48. Gressel J, Hanafi A, Head G, Marasas W, Obilana B, et al. 2004. Major heretofore intractable biotic constraints to African food security that may be amenable to novel biotechnological solutions. *Crop Prot.* 23:661–89

49. Hain R, Reif HJ, Krause E, Langebartels R, Kindl H, et al. 1993. Disease resistance results from foreign phytoalexin expression in a novel plant. *Nature* 361:153–56

50. Hampton RO, Ball EM, De Boer SH, eds. 1990. *Serological Tests for Detection of Viral and Bacterial Pathogens*. St Paul. MN: APS Press

51. Hatta R, Ito K Hosaki Y, et al. 2002. A dispensable chromosome controls host-specific pathogenicity in the fungal pathogen *Alternaria alternata. Genetics* 161:59–70

52. Hayden HL, Pegg KG, Aitken EAB, Irwin JAG. 1994. Genetic relationships as assessed by molecular markers and cross-infection among strains of *Colletotrichum-gloeosporioides. Aust. J. Bot.* 42:9–18

53. Herron SR, Benen JAE, Scavetta RD, Visser J, Jurnak F. 2000. Structure and function of pectic enzymes: virulence factors of plant pathogens. *Proc. Natl. Acad. Sci. USA* 97:8762–69

53a. Hewitt HG. 1998. *Fungicides in Crop Protection*. Wallingford, UK: CAB International.

54. Higgins CM, Hall RM, Mitter N, Cruickshank A, Dietsgen RG. 2004. Peanut stripe potyvirus resistance in peanut (*Arachis hypgaea* L.) plants carrying viral coat protein gene sequences. *Trans. Res.* 13:59–67

55. Hijmans RJ, Forbes GA, Walker TS. 2000. Estimating the global severity of potato late blight with GIS-linked disease forecast models. *Plant Pathol.* 49:697–705

56. Horsfall TG. 1972. *Genetic Vulnerability of Major Crops*. Washington DC: Natl. Acad. Sci. ISBN 0-309-02030-1

57. Deleted in proof

58. Deleted in proof

59. Huang Q, Allen C. 1997. An exo-poly-alpha-D-galacturonosidase, PehB, is required for wild-type virulence of *Ralstonia solanacearum. J. Bacteriol.* 179:7369–78

60. Huang XQ, Roder MS. 2004. Molecular mapping of powdery mildew resistance genes in wheat: a review. *Euphytica* 137:203–23

61. Igarashi S, Utiamada CM, Igarashi LC, Kazuma AH, Lopes RS. 2004. *Pyricularia* in wheat.1. Occurrence of *Pyricularia* sp. in Paraná state. *Fitopathol. Bras.* 11:351–52

62. Iwai T, Kaku H, Honkura R, Nakamura S, Ochiai H, et al. 2002. Enhanced resistance to seed-transmitted bacterial diseases in transgenic rice plants overporducing an oat cell-wall-bound thionin. *Mol. Plant Microbe Interact.* 15:515–21

63. James C. 1998. Global food security. *Abstr. Int. Congr. Plant Pathol., 7th Edinburgh, UK, Aug.* No. 4.1GF. http://www.bspp.org.uk/icpp98/4/1GF.html

64. James C. 2003. *Global Status of Commercialized Transgenic Crops:* 2003. Manila: Int. Serv. Acquis. Agri-Biotech Appl. (ISAAA)

65. Joel DM. 2000. The long-term approach to parasitic weeds control: manipulation of specific developmental mechanisms of the parasite. *Crop Prot.* 19:753–58

66. Johnson R. 1984. A critical analysis of durable resistance. *Annu. Rev. Phytopathol.* 22:309–30

67. Johnson R. 1992. Reflections of a plant pathologist on breeding for disease resistance, with emphasis on yellow rust and eyespot of wheat. *Plant Pathol.* 41:239–54

68. Joosten MHAJ, De Wit PJGM. 1999. The tomato–*Cladosporium fulvum* interaction: a versatile experimental system to study plant-pathogen interactions. *Annu. Rev. Phytopathol.* 37:335–67

69. Joseph M, Gopalakrishnan S, Sharma RK, Singh VP, Singh AK, et al. 2004. Combining bacterial blight resistance and Basmati quality characteristics by phenotypic and molecular marker-assisted selection in rice. *Mol. Breed.* 13:377–87

70. Deleted in proof

71. Kaiser WJ, Hannan RM. 1987. First report of *Mycosphaerella rabiei* on chickpeas in the Western Hemisphere. *Plant Dis.* 71:192

72. Karanastasi E, Wyss U, Brown DJF. 2003. An in vitro examination of the feeding behaviour of *Paratrichodorus anemones* (Nematoda : Trichodoridae), with comments on the ability of the nematode to acquire and transmit Tobravirus particles. *Nematology* 5:421–34

73. Katan J. 1981. Solar heating (solarization) of soil for control of soilborne pests. *Annu. Rev. Phytopathol.* 19:211–36

74. Kearney B, Ronald PC, Dahlbeck D, Staskawicz BJ. 1988. Molecular basis for evasion of plant host defense in bacterial spot disease of pepper. *Nature* 332:541–43

75. Kelly JD, Gepts P, Miklas PN, Coyne DP. 2003. Tagging and mapping of genes and QTL and molecular marker-assisted selection for traits of economic importance in bean and cowpea. *Field Crops Res.* 82:135–54

76. Khan MSA, Ramsey MD, Corbiere R, Infantino A, Porta-Puglia A, et al. 1999. Ascochyta blight of chickpea in Australia: identification, pathogenicity and mating type. *Plant Pathol.* 48:230–34

77. Kimura M, Anzai H, Yamaguchi I. 2001. Microbial toxins in plant-pathogen interactions: biosynthesis, resistance mechanisms, and significance. *J. Gen. Appl. Microbiol.* 47:149–60

78. Kjemtrup S, Nimchuk Z, Dangl JL. 2000. Effector proteins of phytopathogenic bacteria: bifunctional signals in virulence and host recognition. *Curr. Opin. Microbiol.* 3:73–78

79. Koide T, Zaini PA, Moreira LM, Vencio RZN, Matsukuma AY, et al. 2004. DNA microarray-based genome comparison of a pathogenic and a nonpathogenic strain of *Xylella fastidiosa* delineates genes important for bacterial virulence. *J. Bacteriol.* 186:5442–49

80. Large EC. 1940. *The Advance of the Fungi*. London, UK: Jonathan Cape

81. Laugé R, Joosten MHAJ, Van den Ackerveken GFJM, et al. 1997. The *in planta*-produced extracellular proteins ECP1 and ECP2 of *Cladosporium fulvum* are virulence factors. *Mol. Plant Microbe Interact*. 10:725–34

82. Laugé R, Joosten MHAJ, Haanstra JPW, Goodwin PH, Lindhout P, De Wit PJGM. 1998. Successful search for a resistance gene in tomato targeted against a virulence factor of a fungal pathogen. *Proc. Natl. Acad. Sci. USA* 95:9014–18

83. Lazarovits G. 2001. Management of soilborne plant pathogens with organic soil amendments: a disease control strategy salvaged from the past. *Can. J. Plant Pathol.-Rev. Can. Phytopathol.* 23:1–7

84. Leach JE, Cruz CMV, Bai JF, Leung H. 2001. Pathogen fitness penalty as a predictor of durability of disease resistance genes. *Annu. Rev. Phytopathol.* 39:187–224

85. Legg JP. 1999. Emergence, spread and strategies for controlling the pandemic of cassava mosaic virus disease in east and central Africa. *Crop Prot.* 18:627–37

86. Legg JP, Okao-Okuja G, Mayala R, Muhinyuza JB. 2001. Spread into Rwanda of the severe cassava mosaic virus disease pandemic and the associated Uganda

variant of East African *Cassava mosaic virus* (EACMV-Ug). *Plant Pathol.* 50:796

87. Legg JP, Ndjelassili F, Okao-Okuja G. 2004. First report of cassava mosaic disease and cassava mosaic geminiviruses in Gabon. *Plant Pathol.* 53:232

88. Leyns F, Decleene M, Swings JG, Deley J. 1984. The host range of the genus *Xanthomonas. Bot. Rev.* 50:308–56

89. Lorenz MC. 2002. Genomic approaches to fungal pathogenicity. *Curr. Opin. Microbiol.* 5:372–78

90. Louws FJ, Fulbright DW, Stephens CT, Debruijn FJ. 1994. Specific genomic fingerprints of phytopathogenic *Xanthomonas* and *Pseudomonas* pathovars and strains generated with repetitive sequences and PCR. *Appl. Environ. Microbiol.* 60:2286–95

91. Luc M, Sikora RA, Bridge J, eds. 1990. *Plant Parasitic Nematodes in Subtropical and Tropical Agriculture*. Wallingford, UK: CAB Int.

92. Madden LV, Nutter FW. 1995. Modeling crop losses at the field scale. *Can. J. Plant Pathol.* 17:124–35

93. Madden LV, Wheelis M. 2003. The threat of plant pathogens as weapons against US crops. *Annu. Rev. Phytopathol.* 41:155–76

94. Manners JM, He C. 1997. Recent advances in studies of anthracnose of *Stylosanthes*. IV. Molecular approaches to studies of *Colletotrichum gloeosporioides* causing anthracnose of *Stylosanthes* in Australia. *Trop. Grassl.* 31:435–44

94a. Maredia KM, Dakouo D, Mota-Sanchez D, eds. 2003. *Integrated Pest Management in the Global Arena*. Wallingford, UK: CAB International.

95. Merrill AH, Liotta DC, Riley RT. 1996. Fumonisins: fungal toxins that shed light on sphingolipid function. *Trends Cell Biol.* 6:218–23

96. Michelmore RW, Meyers BC. 1998. Clusters of resistance genes in plants evolve by divergent selection and a birth-and-death process. *Genome Res.* 8:1113–30

97. Oerke E-C, Dehne D-W, Schonbeck F, Weber A. 1994. *Crop Production and Crop Protection: Estimated Losses in Major Food and Cash Crops*. Amsterdam: Elsevier

98. Padmanabhan SY. 1973. The great Bengal famine. *Annu. Rev. Phytopathol.* 11:11–26

99. PhytID. 2004. *Identification of plant pathogenic* Phytophthora *species by ITS Fingerprinting*. http://www.phytid.org/

100. Powell ALT, van Kan J, ten Have A, Visser J, Greve LC, et al. 2000. Transgenic expression of pear PGIP in tomato limits fungal colonization. *Mol. Plant Microbe Interact.* 13:942–50

101. Quirino BF, Bent AF. 2003. Deciphering host resistance and pathogen virulence: the *Arabidopsis/Pseudomonas* interaction as a model. *Mol. Plant Pathol.* 4:517–30

102. Reuveni R, Raviv M, Bar R. 1989. Sporulation of *Botrytis cinerea* as affected by photoselective polyethylene sheets and filters. *Ann. Appl. Biol.* 115:417–24

103. Rosewich UL, Kistler HC. 2000. Role of horizontal gene transfer in the evolution of fungi. *Annu. Rev. Phytopathol.* 38:325–63

104. Rouanet C, Reverchon S, Rodionov DA, Nasser W. 2004. Definition of a consensus DNA-binding site for PecS, a global regulator of virulence gene expression in *Erwinia chrysanthemi* and identification of new members of the PecS regulon. *J. Biol. Chem.* 279:30158–67

105. Saettler AW, Schaad NW, Roth DA, eds. 1989. *Detection of Bacteria in Seed and Other Planting Material*. St Paul, MN: APS Press

106. Savary S, Willocquet L, Elazegui FA, Castilla NP, Teng PS. 2000. Rice pest constraints in tropical Asia: Quantification of yield losses due to rice pests in a range of production situations. *Plant Dis.* 84:357–69

107. Schaad NW, Frederick RD, Shaw J, Schneider WL, Hickson R, et al. 2003.

Advances in molecular-based diagnostics in meeting crop biosecurity and phytosanitary issues. *Annu. Rev. Phytopathol.* 41:305–24

108. Schaad NW, Gaush P, Postnikova E, Frederick R. 2001. On-site one hour PCR diagnosis of bacterial diseases. *Phytopathology* 91:S79–89

109. Schaad NW, Jones JB, Chun W. 2001. *Laboratory Guide for Identification of Plant Pathogenic Bacteria*. St Paul, MN: APS

110. Schaad NW, Opgenorth D, Gaush P. 2002. Real-time polymerase chain reaction for one-hour on-site diagnosis of Pierce's disease of grape in early season asymptomatic vines. *Phytopathology* 92:721–28

111. Schaad NW, Song WY, Hatziloukas E. 2000. *U.S. Patent No. 6146834*

112. Schell MA. 2000. Control of virulence and pathogenicity genes of *Ralstonia solanacearum* by an elaborate sensory network. *Annu. Rev. Phytopathol.* 38:263–92

113. Sciumbato GL. 1993. Soybean disease loss estimate for the southern United States during 1988–1991. *Plant Dis.* 77:954–56

114. Shelburne SA, Musser JM. 2004. Virulence gene expression in vivo. *Curr. Opin. Microbiol.* 7:283–89

115. Smith IM, McNamara DG, Scott PR, Holderness M. 1996. *Quarantine Pests for Europe*. Wallingford, UK: CAB Int.

116. Stewart GR, Press MC. 1990. The physiology and biochemistry of parasitic angiosperms. *Annu. Rev. Plant Physiol. Plant Mol. Biol.* 41:127–51

117. Strange RN. 2003. *Introduction to Plant Pathology*. Chichester, UK: Wiley. xvi + 464 pp.

118. Summerell BA, Burgess LW. 1989. Factors influencing survival of *Pyrenophora tritici repentis*—stubble management. *Mycol. Res.* 93:38–40

119. Sutton BC. 1992. The genus *Glomerella* and its anamorph *Colletotrichum*. In *Colletotrichum: Biology, Pathology and Control*, ed. JA Bailey, MJ Jeger, pp. 1–26. Wallingford, UK: CAB Int.

120. Szankowski I, Briviba K, Fleschhut J, Schonherr J, Jacobsen HJ, Kiesecker H. 2003. Transformation of apple (*Malus domestica* Borkh.) with the stilbene synthase gene from grapevine (*Vitis vinifera* L.) and a PGIP gene from kiwi (*Actinidia deliciosa*). *Plant Cell Rep.* 22:141–49

121. Talbot NJ. 2003. On the trail of a cereal killer: exploring the biology of *Magnaporthe grisea. Annu. Rev. Microbiol.* 57:177–202

122. Tanaka A, Shiotani H, Yamamoto M, Tsuge T. 1999. Insertional mutagenesis and cloning of the genes required for biosynthesis of the host-specific AK-toxin in the Japanese pear pathotype of *Alternaria alternata. Mol. Plant Microbe Interact.* 12:691–702

123. Tatum LA. 1971. The Southern corn leaf blight epidemic. *Science* 171:1113–1116

124. Temporini ED, VanEtten HD. 2004. An analysis of the phylogenetic distribution of the pea pathogenicity genes of *Nectria haematococca* MPVI supports the hypothesis of their origin by horizontal gene transfer and uncovers a potentially new pathogen of garden pea *Neocosmospora boniensis. Curr. Genet.* 46:29–36

125. Thakur RP, Mathur K. 2002. Downy mildews of India. *Crop Prot.* 21:333–45

126. Thinlay X, Finckh MR, Bordeos AC, Zeigler RS. 2000. Effects and possible causes of an unprecedented rice blast epidemic on the traditional farming system of Bhutan. *Agric. Ecosyst. Environ.* 78:237–48

127. Thurston HD. 1990. Plant-disease management-practices of traditional farmers. *Plant Dis.* 74:96–102

128. Trutmann P, Paul KB, Cishabayo D. 1992. Seed treatments increase yield of farmer varietal field bean mixtures in the Central African Highlands through multiple disease and beanfly control. *Crop Prot.* 11:458–64

129. Tushemereirwe W, Kangire A, Sseki-woko F, Offord LC, Crozier J, et al. 2004. First report of *Xanthomonas campestris* pv. *musacearum* on banana in Uganda. *Plant Pathol.* 53:802

130. Ullstrup AJ. 1972. The impact of the southern corn leaf blight epidemics of 1970–71. *Annu. Rev. Phytopathol.* 10:37–50

131. van't Slot KAE, Knogge W. 2002. A dual role for microbial pathogen-derived effector proteins in plant disease and resistance. *Crit. Rev. Plant Sci.* 21:229–71

132. Van der Hoorn RAL, De Wit PJGM, Joosten MHAJ. 2002. Balancing selection favors guarding resistance proteins. *Trends Plant Sci.* 7:67–71

133. Wolfe MS. 1993. Can the strategic use of disease resistant hosts protect their inherent durability? In *Durability of Disease Resistance*, ed. T Jacobs, JE Parleviet. Wageningen, Neth.: Kluwer

134. Wolpert TJ, Dunkle LD, Ciuffetti LM. 2002. Host-selective toxins and avirulence determinants: What's in a name? *Annu. Rev. Phytopathol.* 40:251–85

135. Wyss U. 1982. Virus-transmitting nematodes—feeding behavior and effect on root cells. *Plant Dis.* 66:639–44

136. Yakoby N, Beno-Moualem D, Keen NT, Dinoor A, Pines O, Prusky D. 2001. *Colletotrichum gloeosporioides* pelB is an important virulence factor in avocado fruit–fungus interaction. *Mol. Plant Microbe Interact.* 14:988–95

137. Zhang LH, Birch RG. 1997. The gene for albicidin detoxification from *Pantoea dispersa* encodes an esterase and attenuates pathogenicity of *Xanthomonas albilineans* to sugarcane. *Proc. Natl. Acad. Sci. USA* 94:9984–89

138. Zhang LH, Xu JL, Birch RG. 1999. Engineered detoxification confers resistance against a pathogenic bacterium. *Nat. Biotechnol.* 17:1021–24

139. Zhou XP, Liu YL, Calvert L, Munoz C, OtimNape GW, et al. 1997. Evidence that DNA-A of a geminivirus associated with severe cassava mosaic disease in Uganda has arisen by interspecific recombination. *J. Gen. Virol.* 78:2101–11

140. Zhu YY, Chen HR, Fan JH, Wang YY, et al. 2000. Genetic diversity and disease control in rice. *Nature* 406:718–22
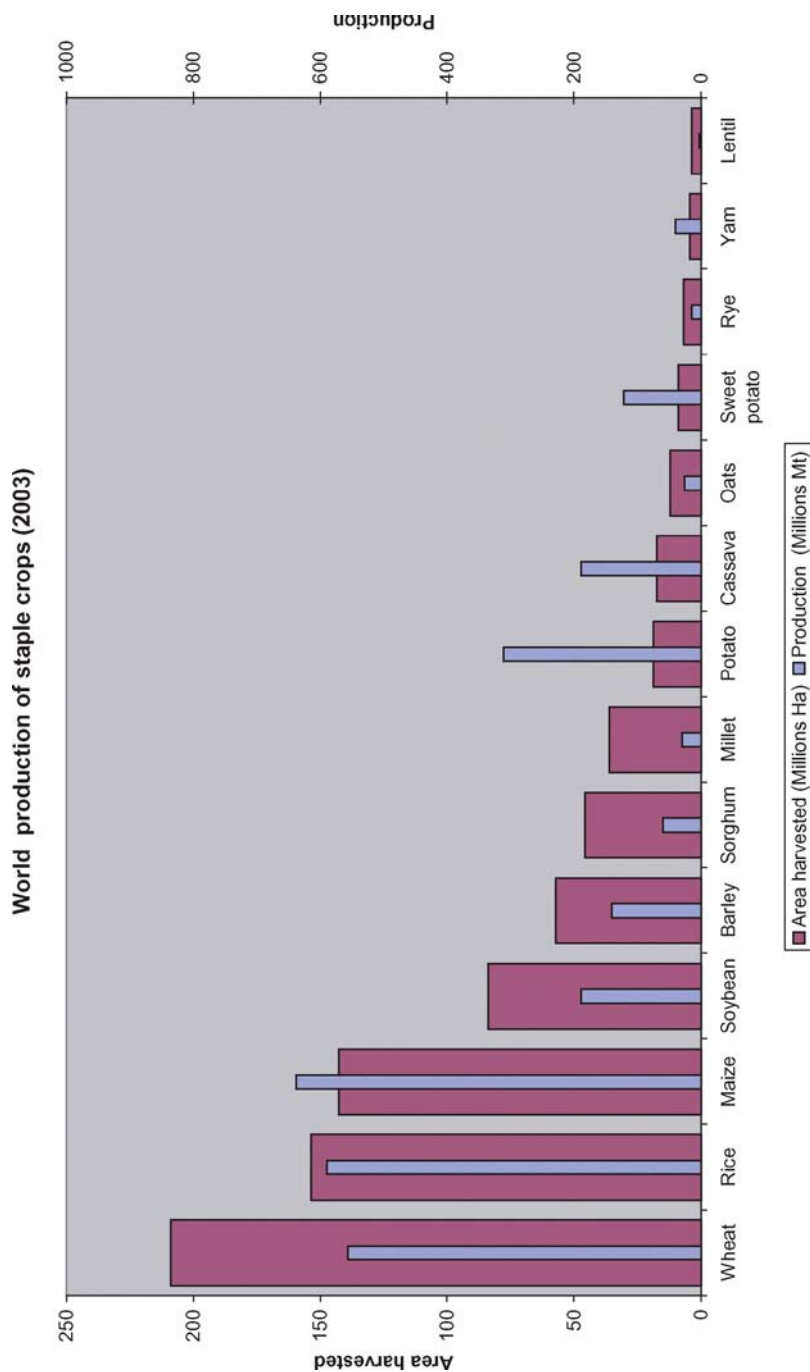
Figure 1 World production of staple[a] crops.
[a]Crops identified as staples in FAO (33). Data from FAO (35).

CONTENTS

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