17

PLANT DEFENCES AGAINST PATHOGENS

David Guest and John Brown

Contents

17.1 Host–parasite relationships

Earlier chapters have described the diverse and constant threat pathogens pose to plant health. Yet, surprisingly, disease is the exception rather than the rule in natural plant communities. Put another way, most pathogens are unable to attack most plants; they have a restricted host range. Assuming environmental conditions favour pathogen development, the resistance or susceptibility of a plant to a particular pathogen depends on two interrelated factors: (i) the substrate requirements of the pathogen and (ii) the response of the plant to the pathogen.

In the previous chapter two broad groups of pathogens, **necrotrophs** and **biotrophs**, were distinguished by their different substrate requirements (Table 16.1). Necrotrophs are 'thugs' in the sense that they kill plant cells before parasitising them. Host and parasite cells cannot coexist harmoniously. Thus, an incompatible cellular relationship between the parasite and host is essential for disease development. If the toxins used to kill host cells are not released at the right time, place or concentration, or if a particular host genotype is insensitive to the toxin, host cells will not die. The necrotroph will be unable to colonise or reproduce and the plant will be resistant. Two types of necrotrophic pathogens exist: (i) those with a wide host range involving many plant species and (ii) those

with a host range restricted to a few plant species or even to cultivars within a species. The key difference between these two types of necrotroph is the specificity of the toxin(s) produced. Necrotrophs with a broad host range secrete toxins that act on metabolic targets common to many plants. In contrast, the pathogenic ability of necrotrophs that release host-specific toxins is conditioned by the gene that encodes the ability to produce the toxin and by a gene in susceptible cultivars of the host that encodes sensitivity to that toxin. Host-specific necrotrophs usually form a pathogenic race or pathotype structure where some races can attack some cultivars within a species but not others. If the gene that conditions sensitivity to a particular host-specific toxin is absent from a cultivar, that cultivar will be resistant to the disease caused by that pathogen.

Biotrophs on the other hand are obligate parasites that obtain nutrients from living cells. Consequently, they must establish a compatible cellular relationship with their hosts. Biotrophs act as 'sneaks'. They typically infect through natural openings or by directly penetrating their host's surface. They mostly then grow between the cells of their host and only penetrate host cell walls (but not host cell membranes) to form food-absorbing haustoria. The pathogen develops without eliciting the host's defence responses or by spreading in advance of the plant's ability to activate its defence responses. The level of specialisation required to establish this type of relationship usually means that biotrophs have a restricted host range and a well-defined pathogenic race structure. If host cells die in advance of invasion by a biotrophic pathogen, the plant will be resistant because the pathogen is unable to establish a parasitic relationship.

A second factor that influences whether a parasitic relationship will become established is the way that the plant under challenge responds. Some interactions between individual pathogen propagules and plant cells may lead to successful pathogen establishment, while others may not. In this chapter it will become evident that resistance or susceptibility of a whole plant and plant communities is the sum of many individual cellular interactions. Plants that are resistant restrict or retard the development and reproduction of an overwhelming majority of individual pathogen propagules that attack it. In this sense resistance is quantitative—resistant hosts prevent or slow the development and reproduction of a higher proportion of pathogen propagules than susceptible hosts. For the purposes of plant breeding, the response of a plant to pathogen inoculation is often categorised as either 'resistant' or 'susceptible', although from a cellular perspective this distinction is not always so clear. Resistance and susceptibility are more accurately portrayed as the extremes of a continuum upon which most host–parasite interactions sit. Resistance may be expressed in many ways, from the inhibition of propagule germination and penetration, the killing of pathogens before establishment, to the restriction or retardation of colony development and reproduction once the pathogen has established. For example, different genes for stem rust resistance in wheat act at different stages of the host–parasite interaction. Some cause the rapid death of the pathogen following attempted penetration, others allow initial infection, but prevent haustorial development and starve the pathogen, while the 'slow-rusting' genes allow parasitism and pathogen reproduction, but at a much slower rate than in susceptible cultivars. Each type of interaction provides useful resistance for plant breeders because they all delay the onset of epidemics and reduce yield losses.

The early steps involved in the establishment of a host–pathogen relationship are delicate and sensitive to environmental factors, including the presence of other micro-organisms. The host–parasite–environment interaction is mediated by a complex interchange of signals. Plants respond to pathogen attack by erecting a highly coordinated series of molecular, cellular and tissue-based defence barriers.

All plants have the capacity to activate these defences. However if they are activated too little, too late, or in the wrong place, they will fail to restrict the pathogen and the plant will be susceptible. Pathogens respond by escaping or suppressing plant defence responses or by rendering these responses impotent, for example by detoxifying plant antibiotics.

The interaction of pathogen nutrient requirements and host responses leads to five possible outcomes if environmental conditions favour infection (Fig. 17.1).

- **No relationship** is established when the plant and the pathogen ignore each other. For example, a spore of a fungus may germinate, but because the host does not provide essential requirements for pathogen development, the resulting hypha fails to penetrate or establish a parasitic relationship. The fungus dies when its energy reserves are exhausted. The plant does not react in any way and is resistant by default. It is a non-host.

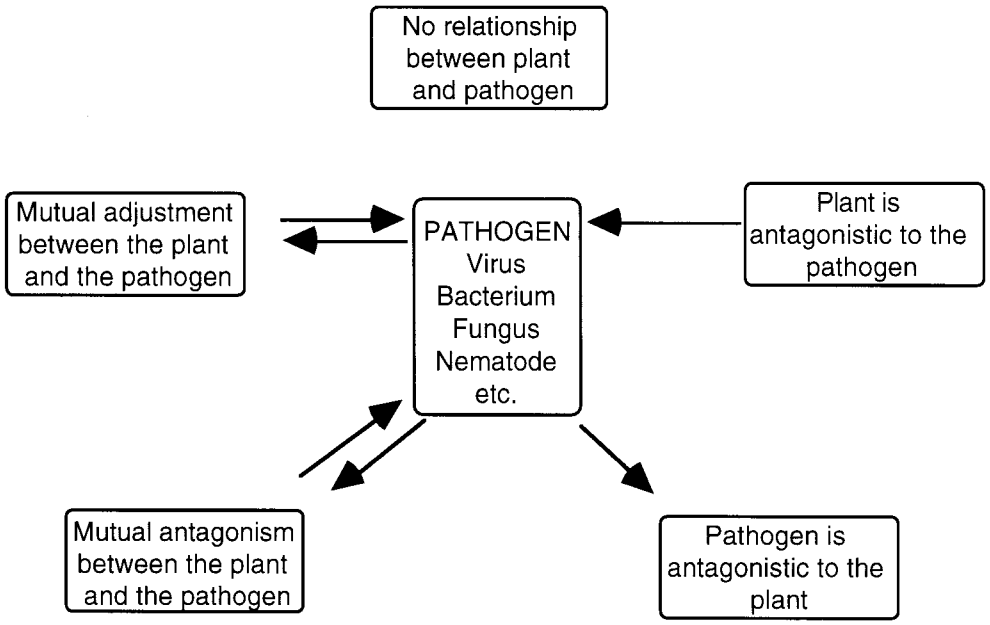

Figure 17.1 Five possible relationships between plants and potential pathogens.

- **A plant is antagonistic to the pathogen** when it secretes inhibitory compounds into its environment that prevents pathogen development. For example, the stubble of some brassicas releases 'biofumigants' into the soil that prevent the hatching of nematode eggs and inhibit the growth of some root-infecting fungi. Asparagus and marigolds (*Tagetes* spp.) secrete substances into the rhizosphere that are toxic to nematodes and provide useful protection against nematodes when interplanted with nematode-susceptible plants like tomato. Many plants secrete phenolic compounds onto their leaf surfaces that not only discourage herbivore feeding, but also inhibit many micro-organisms, including potential pathogens. In this relationship, the pathogen fails to develop and has no observable effect on the metabolism of the host plant. In some cases, such as in the quiescent infection of ripening avocado fruit with *Colletotrichum gloeosporioides*, plant antagonists only temporarily inhibit pathogen development. Spores germinate to form appressoria, but their development is arrested by fungistatic substances in the peel. After harvest, these substances are enzymically degraded and the appressorium germinates to form infection hyphae. Eventually, anthracnose lesions develop. This type of interaction involving a quiescent stage in

pathogen development is common among the stem end rot pathogens of avocado and mango (e.g. *Dothiorella dominicana, Lasiodiplodia theobromae, Phomopsis* spp. and *Colletotrichum gloeosporioides*).

- **The pathogen is antagonistic to the plant** when it secretes compounds that damage the plant. For example, *Periconia circinata*, infects the roots of sorghum, but only those strains of the fungus that produce the host-specific toxin, periconin, induce symptoms of milo disease, but only in cultivars that are sensitive to this toxin. Similarly, some strains of *Alternaria alternata* release host-specific toxins that kill cells of susceptible host species and cultivars. For example, a strain of the fungus that is pathogenic on tomato produces AAL-toxin, to which tomato is uniquely sensitive. Strains producing AAM-toxin attack apples, AAK-toxin producing strains affect Japanese pears, AAC toxin-producing strains affect citrus and so on. The tomato, apple and Japanese pear strains are not pathogenic to citrus because citrus is only sensitive to the AAC-toxin. *Cochliobolus victoriae* produces the toxin victorin that causes severe seedling blight on susceptible cultivars of oats, but has little effect on resistant cultivars or on other plant species. Resistance is the result of insensitivity to the toxin produced by the pathogen. If this insensitivity is common to all cultivars within a plant species, that species is said to be a non-host.
- **Mutual antagonism between plant and pathogen** results in the inhibition or death of both the host tissue and pathogen. For example, an incompatible interaction between the stem rust pathogen, *Puccinia graminis* f. sp. *tritici* and resistant cultivars of wheat causes the death of both host and pathogen cells.
- **Mutual adjustment** leads to a compatible cellular relationship between the host and pathogen. Symbiotic relationships between mycorrhizal fungi and plant roots and between nitrogen-fixing prokaryotes and plant roots, are examples of mutually beneficial interactions. Endophytic fungi and bacteria colonise the intercellular spaces of plant tissue, apparently without damaging their host cells. Many stem end rot pathogens have an endophytic phase in leaves and twigs before they infect fruits. Biotrophic pathogens, like the mildews and rusts, grow and reproduce on living host tissue. However, the diversion of nutrients to the invading pathogen adversely affects the growth of the host, even though host cells are not killed.

In this chapter, plant defence mechanisms will be discussed in the order they are usually confronted by pathogens. Broadly speaking, **passive defence mechanisms** are those that are present before contact with the pathogen, while **active defence mechanisms** are activated only after **pathogen recognition** (Fig. 17.2). In reality this distinction is not always clear, as many pre-existing defences are modified after infection.

17.2 Passive defences

To gain access to the nutrients or replication machinery available within the host cell, pathogens must first breach the natural barriers presented by healthy plants. These barriers may be physical (the cuticle, cell wall, stomatal aperture or lenticel) or chemical (including inhibitory compounds or the absence of stimulatory compounds needed for pathogen development). Saprophytes lack the ability to penetrate these natural barriers.

Physical barriers

The importance of the cuticle as a barrier to penetration has been demonstrated by the dependence of many pathogens on adhesion and the subsequent release of

cutin-degrading enzymes at the time of penetration. Although cutin-degrading enzymes are also secreted by many saprophytic fungi and bacteria, their primary activity is to allow access to cellulose in plant cell walls as a nutritional substrate. Different forms of cutin-degrading enzymes are used by pathogens to puncture the cell wall (Chapter 16). The activity of this type of cutinolytic enzyme in isolates of *Fusarium solani* f. sp. *pisi* is directly related to their aggressiveness on pea stems, indicating that pathogens unable to dissolve the cuticle at the point of penetration are excluded.

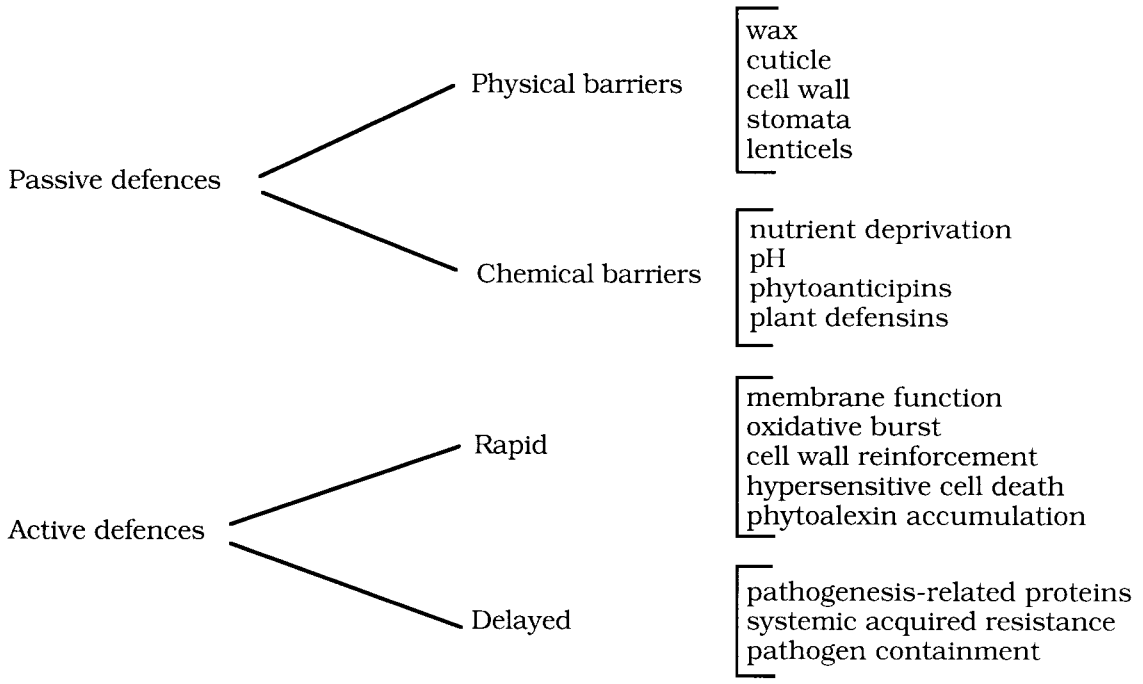

Figure 17.2 Some defence mechanisms in plants.

Cuticle and cell wall thickness may influence resistance to certain pathogens. Some types of 'adult plant resistance' could be associated with a reduced ability of pathogens to enter through thicker, tougher cell walls. Some pathogens such as *Puccinia graminis* only infect young barberry leaves with thin cuticles and the germ tubes emerging from basidiospores do not penetrate thicker cuticles on mature leaves. Similarly, the ability of *Taphrina deformans* to infect only young, newly unfolded leaves has been attributed to the inability of germ tubes to penetrate the thicker cuticles of older leaves. The presence of secondary cell walls in sclerenchyma, xylem or older plant tissue often retards pathogen development, leading, for example, to angular leaf spots where pathogen spread is restricted by leaf veins. Thick cuticles may physically prevent the eruption of sporophores and release of spores. However, most experimental evidence suggests that toughened cuticles and cell walls are just one of the many factors that contribute to resistance.

Waxy cuticles and vertically oriented leaves may prevent the formation of moisture films on leaf surfaces. Dry leaf surfaces inhibit infection by pathogens such as bacteria, nematodes and fungal zoospores that require a film of water for motility. Fungal spores might also be inhibited because most require moisture for germination. This must be balanced with the fact that vertically oriented leaves

are more prone to impaction by wind-borne pathogen propagules and are likely to face higher inoculum levels compared with those that are horizontally oriented.

Many pathogens enter through wounds, natural openings or are introduced by vectors. In these cases it is difficult to see how natural barriers such as the cuticle and cell wall could be involved in resistance. Some researchers have proposed that plants that have stomatal apertures that are the wrong shape or size for pathogen infection structures to enter or that have stomata that close at the time of day that pathogen spores normally germinate, may be more resistant to pathogen attack. The black pod pathogen, *Phytophthora palmivora*, enters cocoa pods through stomata. Cocoa genotypes that produce pods with few, relatively smaller stomata, allow fewer lesions to establish than genotypes with more numerous, larger stomata. Not surprisingly, as the pathogen enters through stomatal pores, there is no correlation between cuticle thickness or pod case hardness and resistance to black pod. The bacterium that causes citrus canker, *Xanthomonas campestris* pv. citri, enters grapefruit through open stomata. Mandarins are resistant because their stomata are too small to allow entry of the bacterium. Similarly, lenticels that suberise rapidly so that their size is reduced may physically exclude pathogens such as *Streptomyces scabies*, the cause of common scab of potato.

Chemical barriers

Exudates on the surfaces of plants or compounds in plant cells may stimulate or inhibit the development of pathogens. Sometimes, plants resist infection because they do not provide the pathogen with its required nutrients. Resting spores of pathogens such as *Spongospora subterranea* (powdery scab of potato), *Urocystis agropyri* (flag or leaf smut of wheat) and *Plasmodiophora brassicae* (club root of crucifers) and eggs of the potato cyst nematode, *Globodera rostochiensis*, require specific substances to stimulate germination or hatching. These are provided in secretions from certain plants, including potential hosts. Plants that fail to secrete these stimulators are resistant by default.

Other plant secretions may simply not support the pre-penetration growth of the pathogen. Experimental depletion of iron availability using binding agents (siderophores) inhibits the growth of certain fruit-rotting bacteria. Host cultivars that secrete lower than normal levels of iron onto their surface may deprive pathogens of essential nutrients, inhibiting their growth. Similarly, micro-organisms that sequester available iron on leaf surfaces have potential as biocontrol agents (Chapter 27).

Plants sometimes produce compounds during normal growth that inhibit the development of pathogens. **Phytoanticipins** may be excreted into the external environment (e.g. rhizosphere or phylloplane), accumulate in dead cells or they may be sequestered in vacuoles in an inactive form. The dead cells of brown onion skins contain the quinones catechol and protocatechuic acid, which inhibit germination of spores of the smudge pathogen, *Colletotrichum circinans*, and the neck rot pathogen, *Botrytis cinerea*. White onions do not produce these compounds and are susceptible to smudge. *Aspergillus niger* is insensitive to these inhibitors and attacks both white and brown onions. Avocado rootstocks resistant to root rot caused by *Phytophthora cinnamomi* secrete borbinol, an antimicrobial phenolic compound, into the rhizosphere. The secretion of nematode-inhibiting substances into the rhizosphere surrounding asparagus and marigold roots has already been mentioned. Symptoms of anthracnose of avocado, caused by *Colletotrichum gloeosporioides*, only develop on ripe fruit. The peel of unripe avocado fruit contains antifungal lipids called dienes that prevent

appressorial germination. As these dienes are gradually metabolised during fruit ripening to less toxic compounds, quiescent appressoria germinate and susceptibility to anthracnose increases. In anthracnose-resistant cultivars, diene breakdown is blocked following infection, so that antifungal levels are sustained for longer periods. The resistance of immature apples and pears to scab, caused by *Venturia inaequalis* and *V. pirina* respectively, correlates with the presence of the phenolic compounds chlorogenic acid, phloridzin, arbutin and iso-chlorogenic acid in the outer layers of the fruit. These compounds also contribute to the bitter taste of unripe apples and pears and, as the fruit ripens and sweetens, it also becomes more susceptible to scab.

One group of phytoanticipins, the **saponins**, are plant glycosides with surfactant (wetting agent) properties. Saponins bind sterols in pathogen cell membranes, destroying membrane integrity and function. In this way saponins are toxic to organisms containing sterols in their membranes (e.g. plants and fungi, but not Oomycota). Inactive saponin precursor molecules appear to be stored in vacuoles of intact plant cells, but hydrolase enzymes released following wounding or infection convert these precursors to active, antimicrobial forms. Several lines of evidence suggest that saponins are involved in disease resistance and host range determination. It appears that the ability of some pathogens to detoxify specific saponins matches their host range. For example, a strain of the take-all pathogen that attacks oats as well as wheat and barley (*Gaeumannomyces graminis* var. *avenae*), releases the enzyme avenacinase. Avenacinase detoxifies the triterpenoid saponin, avenacin, found in epidermal cells of the roots of oat plants. Mutants in which the gene for avenacinase production has been deleted are sensitive to avenacin in vitro and are not pathogenic on oats, but remain pathogenic to wheat and barley. *Gaeumannomyces graminis* var. *tritici* lacks avenacinase and attacks wheat and barley, but not oat species containing avenacin. An oat species that does not produce avenacin, *Avena longiglumis*, is susceptible to *Gaeumannomyces graminis* var. *tritici*. Another saponin, tomatine, contributes to the resistance of tomato leaves to *Botrytis cinerea*.

Some plant peptides also inhibit the development of fungi, bacteria, viruses and insects. They act as proteinase and polygalacturonase-inhibitors, as ribosome inhibitors or lectins. These inhibitors interfere with pathogen nutrition and retard their development, thus contributing to disease resistance. Because of their similarity to peptides called defensins found in insects and mammals, they have been termed **plant defensins**. Secreted defensins provide an important defence against damping-off pathogens. While only 0.5% of the total protein found in ungerminated radish seeds is defensin, it makes up 30% of the proteins released from germinating seeds. It provides an antimicrobial micro-environment around the emerging radicle. Defensins may constitute up to 10% of the total proteins in cereal, legume and solanaceous seeds. Similar studies have shown defensins are also present in the outer cell layers of other plant organs such as flowers, leaves and tubers. While many defensins accumulate during normal plant development, others are induced, or their accumulation is enhanced, after wounding. Defensins, because of their anti-feeding activity against insects, provide a defence against insect-transmitted viruses.

17.3 Pathogen recognition

The ability of plants to respond to challenge by potential pathogens implies that plants recognise these potential pathogens as 'non-self'. While mammals use antigen-antibody interactions to recognise non-self, plants recognise a vast array

of signals originating from micro-organisms and the environment to elicit defence responses.

Non-specific elicitors

Many signals of abiotic and biotic origin induce defence responses in a range of cultivars and host species that bear little relationship to pathogen host ranges. The magnitude of the response depends on the amount of elicitor present. Abiotic elicitors, including heavy metal ions, UV light and some metabolic inhibitors, precipitate physiological stress responses, some of which contribute to resistance. Their effect is generally transitory and non-specific. The significance in host–parasite interactions of abiotic elicitors is not always obvious as they are rarely present at the infection court. However solar UV radiation may elicit stress responses in exposed plant tissues, providing an additional barrier for invading pathogens. On the other hand, environmental stresses usually increase the susceptibility of plants to necrotrophic pathogens.

Cell wall fragments released from fungi and bacteria elicit defence responses in plants. Cell wall fragments from *Phytophthora megasperma* f. sp. *glycinea* are potent elicitors of defence responses in soybeans. The smallest active fragment is a heptabetaglucan (seven glucose units) that is found in cell walls of many pathogenic and non-pathogenic races and species of oomycetes. Recently, a receptor was identified in the plasma membrane of soybean cells. This, together with its potency, suggests a role for heptabetaglucan and related oligosaccharins, in pathogen recognition.

Hydrolytic enzymes of plant or pathogen origin also catalyse the release of plant cell wall fragments (**endogenous elicitors**) that elicit defence responses. For example, polygalacturonase enzymes released by fruit decay fungi and bacteria dissolve the middle lamella of plant tissues. While this facilitates pathogen colonisation, it also causes the release of pectic fragments, oligosaccharides consisting of nine to thirteen polygalacturonate units, that are potent elicitors.

A number of **peptides** and **glycoproteins** that elicit defence responses in plants have been isolated from culture filtrates of bacterial and fungal pathogens. A 46 kD glycoprotein extracted from culture filtrates of the black shank pathogen, *Phytophthora nicotianae* var. *nicotianae* and from tobacco leaves infected with this pathogen, is a potent elicitor. There is some evidence that Ppn 46E, a 46 kD glycoprotein, has endoxylanase activity, suggesting that it may also elicit through the release of cell wall fragments. A 42 kD glycoprotein with glucanase activity has been isolated from *Phytophthora megasperma* f. sp. *glycinea*. The active fragment of this glycoprotein is a thirteen-amino acid peptide that binds to a receptor on the host plasma membrane. These elicitors are found in both avirulent and virulent isolates, suggesting that their activity does not determine resistance.

A family of 10kD peptides called '**elicitins**' has been isolated from culture filtrates of *Phytophthora* spp. and a number of related oomycetes. There are two groups of elicitins (i) the acidic α-elicitins such as parasiticein produced by *P. nicotianae* var. *parasitica*, and capsicein produced by *P. capsici* and (ii) the basic β-elicitins such as cryptogein, produced by *P. cryptogea*, melonin produced by *P. melonis* and cinnamomin produced by *P. cinnamomi*. All elicit systemic necrosis in tobacco. Elicitins are translocated when applied to the plant, but they have yet to be found at the infection court. They are not known to have any metabolic function in the fungi that produce them. Highly aggressive isolates of *P. nicotianae* var. *nicotianae* do not release an elicitin and do not elicit host

defence responses. However, less aggressive isolates and isolates from hosts other than tobacco, release parasiticein. This evidence indicates that elicitin release may limit the host range of certain oomycetes. The black shank pathogen is a biotroph in the early stages of infection and aggressive mutants with low elicitin levels may have been selected during co-evolution with its host, tobacco.

Polyunsaturated fatty acids like arachidonic and eicosapentaenoic acid from cell membranes of *Phytophthora infestans* elicit defence responses in potato slices. Although they have lower elicitor activity in other plants when applied on their own, these fatty acids enhance the elicitor activity of glucans when applied in combinations. This, and other evidence, indicates that the complex responses of some infected plants may depend on the recognition of a combination of elicitors.

Gene-specific elicitors

Gene-specific elicitors are those conditioned by avirulence genes in the pathogen. Their activity precisely matches the gene-for-gene hypothesis. Only recently has the application of molecular techniques allowed the characterisation of a few gene-specific elicitors, although their presence has been inferred for many years. A series of race-specific peptide products of the avirulence genes of *Fulvia fulva*, a biotrophic pathogen of tomato, has been identified. These peptides were first isolated from intercellular fluids of infected leaves and have since been found around the infection site.

A heat labile exudate from germinating basidiospores of incompatible races of cowpea rust (*Uromyces vignae*) elicits defence responses only in cowpeas with the corresponding resistance gene. Similarly, a 6.4 kD peptide from the barley leaf scald pathogen, *Rhynchosporium secalis*, specifically elicits resistance in cultivars with the corresponding resistance gene. Host receptors for these peptides have yet to be identified.

A number of avirulence genes have been identified in plant pathogenic bacteria, although their gene products are yet to be characterised. Avirulence (***avr***) genes determine host range (species/pathovar and cultivar/race interactions) according to the gene-for-gene hypothesis. However, studies with genetically-transformed bacteria show that *avr* genes only appear to function in the presence of another set of genes, the ***hrp*** (***h***ypersensitive ***r***esponse and ***p***athogenicity) gene cluster. *Hrp* genes are found among a wide range of pathogenic and non-pathogenic Gram-negative bacteria. They function as pathogenicity genes in the absence of the *avr* gene and hypersensitive response-eliciting genes in their presence. One of these *hrp* genes encodes a heat stable protein, **harpin**, that is involved in membrane transport. Clusters of harpin subunits apparently line a pore allowing secretion of *avr* gene products. *Hrp* gene products are also involved in the secretion of the extracellular polysaccharides that disguise the pathogen from host recognition, thus functioning in both virulence and avirulence.

Suppressors and compatibility factors

It has been proposed that compatibility factors operate at two levels. All biotrophs must establish basic compatibility with their hosts. Virulent races might also produce specific compatibility factors that delay, avoid or negate recognition by normally resistant cultivars of a host species. Experiments using a range of host–parasite interactions have demonstrated that co-inoculation of a host with compatible and incompatible strains of a pathogen allows the normally avirulent strain to infect, colonise and reproduce (Fig. 17.3). These results suggest that the

virulent isolate somehow suppresses the resistance mechanisms of the host. However, if the virulent strain is inoculated some hours after the avirulent strain, the host is resistant to both, indicating that suppressors are unable to switch off resistance responses once they are activated. Water-soluble molecules found on the surface of virulent, but not avirulent, isolates of *Phytophthora infestans* suppress defence responses in potato tuber slices. Glycopeptides produced by *Ascochyta rabiei* and *Mycosphaerella pisi* suppress defence responses in their respective hosts, chickpea and pea. Such interactions may be common in nature.

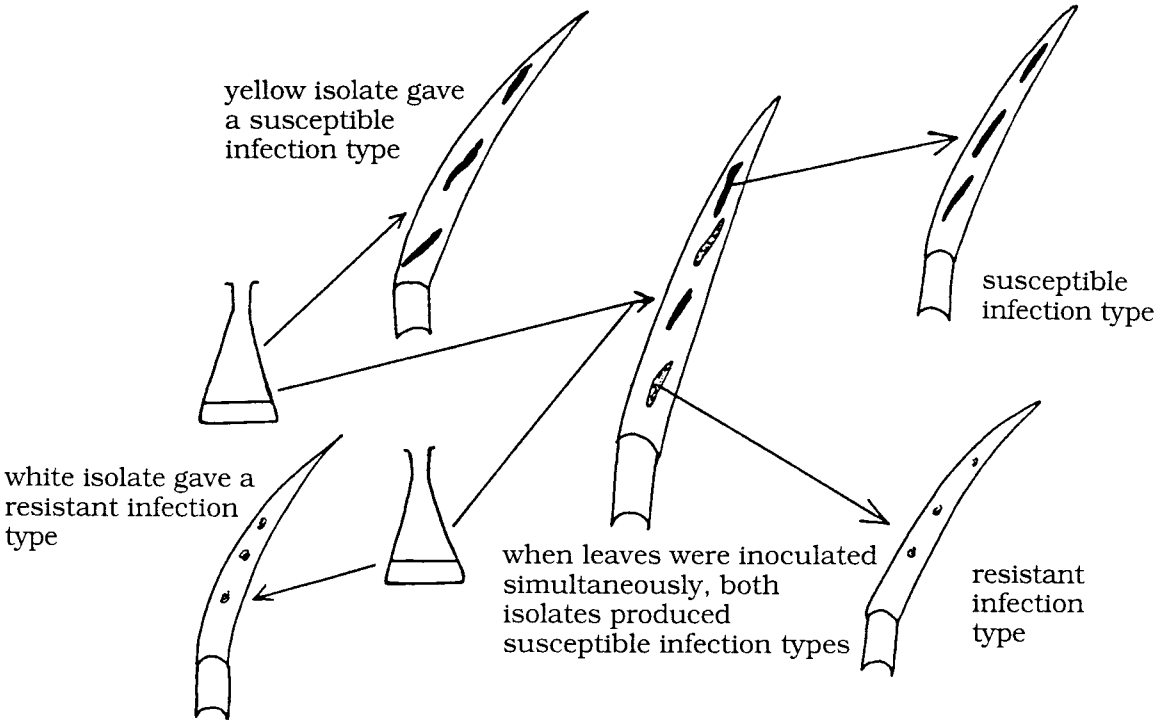

Figure 17.3 Induced susceptibility to stripe rust of wheat induced by simultaneous inoculation of plants with compatible and incompatible isolates of *Puccinia striiformis*. (After Brown and Sharp, 1970.)

Physiological role of elicitors

To understand fully how the discriminatory expression of active defence responses determines resistance or susceptibility, we must understand the basis of specificity. In other words, why do incompatible pathogens trigger plant defence responses, while compatible pathogens do not?

The simplest prediction from the gene-for-gene hypothesis would be that avirulence and resistance gene products recognise each other, triggering a race-specific response. Only recently have molecules been identified that elicit plant defence responses according to the gene-for-gene hypothesis. These molecules are peptides encoded by avirulence genes, and some, perhaps all, bind to receptor peptides encoded by host resistance genes. Of the half-dozen or so resistance genes sequenced, most have some homology to genes encoding proteins involved in protein–protein interactions in cells, such as protein kinases and polygalacturonase-inhibiting proteins. Some are membrane-bound, while others are cytoplasmic. Activation of these proteins following the recognition of avirulence gene products triggers a cellular alarm mechanism, involving signal transduction pathways that lead to a massive shift in gene transcription and