

Pathogens of Autotrophs

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There are currently about 7.0 billion people on planet Earth, and this will increase to 9 billion by 2050, with 80% in the developing world. Feeding this growing population depends on agricultural crops. Plant pests and diseases can cause yield losses of over 50% for major crops, and up to 80% for some crops (e.g. cotton). In the first few years of the twenty-first century, average losses of rice were 37.4%. When you consider that currently, rice feeds about 25 people per hectare, and this will have to increase to about 45 per hectare by 2050, the importance of dramatically reducing losses from pest and disease is clear. Crop losses worldwide cost US\$550 billion each year. While about 60% of losses are caused by invertebrate pests, weeds and weather – frosts, drought, floods, etc., 40% are due to disease, of which fungi cause about two-thirds. Fungal infections currently destroy over 125 million tonnes of rice, wheat, maize, potatoes, and soybeans each year. Rice blast disease, caused by *Magnaporthe oryzae* (pp. 265–267), is at present the most destructive disease of rice worldwide with losses of over 50 million tonnes per annum. Ninety percent of wheat varieties grown worldwide are susceptible to a recently emerged lineage of the stem (black) rust pathogen *Puccinia graminis* f.sp. *tritici*, race Ug99. These examples highlight the importance of mitigating disease spread by fungal pathogens to safeguard world food security. Fungus and fungus-like diseases of plants can have terrible consequences to the human population, as attested to by the Irish Potato famine of the mid 1800s, where the crop was devastated by the fungus-like *Phytophthora infestans* (oomycete) (pp. 277–279). This led to death by starvation, of 1.5 million people, and emigration of over a million, largely to North America. The necessity to understand fungal diseases of plants is evident!

In this chapter we will first consider the variety of fungi that causes diseases of plants and then the differences in susceptibility of plants to pathogens, and how plants defend themselves. We will next examine the main events in the disease cycle of pathogens, from arrival, attachment, and entry into the plant, through the different ways that pathogens establish in and exploit the plants, finally to exit of the pathogen from the plant, and survival until it finds another suitable host. These general concepts will be illustrated with case studies of a range of types of disease, mainly of crop plants. Of course, plants in natural environments also suffer

from fungal diseases, as do other autotrophs such as lichens and seaweeds, and these are mentioned in separate sections. Finally we look at newly emerging diseases and the potential threats they pose to the security of our food supply.

SPECTRUM OF INTERACTIONS OF FUNGI WITH PLANTS

Fungi that attack living plants are called pathogens, or phytopathogens – to distinguish them from fungi that cause disease in other organisms. This is a catchall term that includes fungi that destroy living cells and feed on their contents, as well as fungi that absorb nutrients from living cells without killing them, but as a consequence, considerably reduce plant fitness. Plant pathologists commonly use the terms **necrotrophs** and **biotrophs** to describe these two categories of phytopathogen, respectively. The situation is complicated further by the fact that some fungi can initiate plant infection as biotrophs and switch later to the wholesale destruction of tissues as necrotrophs, a relationship termed **hemi-biotrophic**. Even dividing into three categories is not sufficient to cover the range of different characteristics and the mechanisms operated by plant pathogens, and does not get full support from genomic studies. Nonetheless we will use these categories here for simplicity. ‘Parasite’ is also used to describe pathogenic fungi. Some mycologists reserve this term for biotrophs, regarding all biotrophic pathogens as parasites; others treat parasite and pathogen as synonymous, using parasitism to describe plant infections by biotrophs and necrotrophs. Given the potential for ambiguity, we have avoided the term parasite in this chapter.

Necrotrophic and biotrophic pathogens behave differently in many ways, including the way in which they gain entry into the host, the way in which a host responds defensively to invasion, and in the signalling that occurs (see below; [Table 8.1](#)). Fungi also vary in the degree to which they obtain their nutrition as pathogens. Some are obligate whereas others are facultative. Biotrophic pathogens tend to be obligate, often having specialised structures for nutrient absorption, whereas necrotrophs and some hemibiotrophs are often facultative, being able to survive to some extent saprotrophically. Some fungal pathogens have a broad host range whereas others, especially biotrophic pathogens, have a very narrow host range, attacking only a few plant species. Some pathogen species are divided into specialised groups (*formae speciales*, f.sp.) which only cause disease of a few plant species; some *formae speciales* even have races that only colonise a few varieties of a species (e.g. *Fusarium oxysporum* has many *formae speciales* each of which is pathogenic to a narrow range of plant species [see the section on ‘[Case Studies](#)’, pp. 265–281]).

From the plant’s perspective it is important to realise that though necrotrophic fungi kill host cells that does not necessarily imply that the whole plant will be affected. On the other hand, although biotrophic pathogens feed off living host cells, and often there are few visible signs of early infection, they can considerably reduce host plant fitness/productivity. It is also important to recall that although biotrophic pathogens reduce host fitness, many fungi, though feeding biotrophically, are mutualists that increase plant fitness, such as mycorrhizal, lichen, and many endophytic fungi (Chapter 7). This highlights the difficulty of determining the nature of some mycorrhizal symbioses in which it is unclear whether the fitness of both partners is enhanced by the relationship.

TABLE 8.1 A Comparison of Biotrophic and Necrotrophic Plant Pathogen Characteristics

Characteristic	Biotrophs	Necrotrophs
Obligacy	Specialised, obligate	Relatively unspecialised, facultative
Host range	Narrow	Broad
State of host on entry	All ages; in possession of host defence	Often immature, overmature or damaged
Axenic culturability	Not easy and sometimes not yet possible	Easy
Entry into host	Specialised, e.g. direct penetration of cell walls, e.g. powdery mildews; or via natural openings, e.g. <i>Puccinia hordei</i> (Fig. 8.3)	Unspecialised: via wounds or natural openings
Production of appressoria and haustoria	Appressoria or Appressoria-like structures evident; haustoria generally present (Figs. 8.5, 8.6, 8.7)	Not usually
Damage to host tissues	Little in compatible host	Rapid cell death
Production of lytic enzymes	Localised to hyphae, and limited quantity	Depending on mode of killing: often copious doing massive damage
Production of toxins	Not usually produced	Depending on mode of killing: often produced acting relatively locally or spread extensively in xylem. Some produce host-specific toxins (HSTs)
Survival following host death	Little or no saprotrophic ability; often survives as dormant spores	Can often grow saprotrophically
Control by resistance/susceptibility genes	PRR proteins; R gene products	PRR proteins; host specific toxin (HST) binding susceptibility gene products
Host defence pathways	NPR1; salicylic acid (SA)	COI1/EIN2; jasmonic acid (JA)/ethylene (ET)

DISTRIBUTION OF PATHOGENS AMONGST FUNGAL GROUPS

Most fungal phyla contain species that are plant pathogens, but most pathogens are found in a limited number of taxonomic orders, and within these orders often cause similar types of disease (Table 8.2). Ascomycota contains several orders with many or all species that are plant pathogens (e.g. Taphrinales). The Erysiphales are important biotrophic pathogens causing ‘powdery mildews’; the Helotiales, Pleosporales, and Hypocreales contain many necrotrophic pathogens; several other orders also contain necrotrophic pathogens. In Basidiomycota, the majority of pathogens are the biotrophic Uredinales (smuts) and Ustilaginales (rusts); a few others are important root and butt pathogens of trees. There are many fungus-like oomycete pathogens of plants, within the Peronosporales; the Saprolegniales have few terrestrial pathogenic representatives, though some *Aphanomyces* spp. cause root disease in several crop plants. Other phyla contain relatively few pathogens.

TABLE 8.2 Distribution of Pathogens Among Fungal Groups

Phylum and order	Fungus species	Pathogenic mode	Disease	Symptoms	Host	Comments ^a
Mucoromycotina: mucorales	<i>Rhizopus</i> spp.	Necrotroph	Soft rot	Rotting of fruit and fleshy organs	Many fruits and vegetables	
Chytridiomycota	<i>Synchytrium endobioticum</i>	Obligate	Potato wart disease	Cauliflower-like warts on the tubers	Potato	
Ascomycota: Capnodiales	<i>Mycosphaerella graminicola</i>		Leaf spot	Leaves: water-soaked lesions, black pycnidia later	Cereal grasses, mainly wheat	
Ascomycota: Diaporthales	<i>Cryphonectria parasitica</i>	Necrotroph	Blight	Sunken cankers and split bark	Chestnut (<i>Castanea</i>) trees	Destroyed about 4 billion American chestnut trees
Ascomycota: Erysiphales	<i>Blumeria (Erysiphe) graminis</i>	Biotroph	Powdery mildew	Leaves: chlorotic or necrotic Leaves, stems, heads: covered with spores and mycelium giving a powdery/‘woolly’ appearance	Cereal grasses	
Ascomycota: Eurotiales	<i>Penicillium</i> spp.	Necrotroph	Soft rot	Tissues with high water content mostly affected, including fruit	Many fruits and vegetables, including citrus and apple	
Ascomycota: Glomerellales	<i>Colletotrichum</i>		Anthracnose	Stems and fruits: dark sunken regions Can cause rot of fruit	Beans, fruits, rye, some fruit and forest trees	
Ascomycota: Helotiales	<i>Botrytis</i> spp., including <i>B. cinerea</i>	Necrotroph	Blight and rots	Blossoms; Water-soaked rot blossoms; Leaves, stems, fruit: powdery lesions Fruit: rot	Fruit, ornamental and fruit trees	
Ascomycota: Helotiales	<i>Monilinia</i> spp.		Brown rot	Brown rotting of fruit	Stone fruits	
Ascomycota: Helotiales	<i>Sclerotinia sclerotiorum</i>	Necrotroph	Soft rot and wilt	Tissues with high water content mostly affected. Chlorosis, wilting, leaf drop; colonisation of fruit touching the ground and in storage	Wide range; over 400 species	

Ascomycota: Hypocreales	<i>Claviceps purpurea</i>	Necrotroph		Dark coloured sclerotia in ears	Rye and related grasses	Consumption of the sclerotia causes ergotism in humans and other mammals
Ascomycota: Hypocreales	<i>Fusarium</i> spp., including <i>F. graminearum</i>	Necrotroph	Head blight	Shriveled grain, premature bleaching of spikelets	Cereal grasses	
Ascomycota: Hypocreales	<i>Fusarium oxysporum</i>	Necrotroph	Wilt	Wilting, chlorosis, necrosis, leaf drop, stunted growth, damping off	Many, including most crop plants	
Ascomycota: <i>Incertae sedis</i>	<i>Gaeumannomyces graminis</i> var. <i>tritici</i>	Necrotroph	Take-all disease	Blackening of roots and a black crust around the stem base. Fungus invades phloem and xylem, preventing uptake and movement of water and assimilates	Wheat and barley	The most widespread cereal root disease in the world
Ascomycota: <i>Incertae sedis</i>	<i>Verticillium dahliae</i> , <i>V. albo-atrum</i>	Necrotroph	Wilt	Wilting, stunting and yellowing of older leaves	Many crop plants and trees	
Ascomycota: Magnaporthales	<i>Magnaporthe oryzae</i>	Hemibiotroph	Rice blast	Leaves; lesions whitish/grey when older, which may coalesce to kill entire leaf (Fig. 8.5)		Estimated to destroy annually 10–30% of rice crops, enough to feed 210–740 million people
Ascomycota: Microascales	<i>Ceratocystis fagacearum</i>	Necrotroph	Oak wilt	Wilting, browning of leaves; defoliation Sapwood beneath bark: brown discolouration	Oaks	Caused considerable damage in American mid-west; originated in Russia
Ascomycota: Ophiostomatales	<i>Ophiostoma ulmi</i> , <i>O. novo-ulmi</i>	Necrotroph	Dutch elm disease	Wilting, yellowing/browning of leaves Sapwood beneath bark: brown/green streaks (Fig. 8.10)	Elm	Devastated elms across Europe and North America; identified first in the Netherlands
Ascomycota: Pleosporales	<i>Alternaria</i> spp.	Necrotroph	Leaf spot	Dark blemishes on leaves, stem lesions and on fruit	Wide range of crops including tomato, potato, cucurbits, cotton, brassica	

(Continued)

TABLE 8.2 Distribution of Pathogens Among Fungal Groups—cont'd

Phylum and order	Fungus species	Pathogenic mode	Disease	Symptoms	Host	Comments ^a
Ascomycota: Pleosporales	<i>Cochliobolus sativus</i>	Necrotroph	Blight and root rot	Diseases of root, stem, leaf, and head tissues	Cereals and other grasses	
Ascomycota: Pleosporales	<i>Venturia inaequalis</i>	Necrotroph	Scab	Fruit and leaves: dark lesions	Apple	
Ascomycota: Taphriniales	<i>Taphrina</i> spp.	Biotroph	Leaf curl and blisters	Leaf curls, blisters, fruit deformities and stem dieback, depending on the plant species	Fruit trees	
Basidiomycota: Agaricales	<i>Armillaria mellea</i>	Necrotroph	Root rot	Above ground symptoms are similar to other root rotters: browning of foliage; decline of vigour; stem dieback. In main roots and at trunk base sapwood is water-soaked with creamy fan-shaped sheets of mycelium growing over the surface, and black zone lines within. Rhizomorphs often present (pp. 58–60)	Many species, especially woody	
Basidiomycota: Cantharellales	<i>Rhizoctonia solani</i> (= <i>Thanatephorus cucumeris</i>)	Necrotroph	Damping-off disease	Seeds are colonised and fail to germinate or seedlings are killed	Wide host range	
Basidiomycota: Russulales	<i>Heterobasidion annosum</i>	Necrotroph	Root and butt rot	Above ground symptoms: abnormal needle growth; bark becomes paler; crown thinning; decline in vigour; eventual death. Below ground: white-rotted woody roots (Fig. 8.11)	Conifer and angiosperm trees	Most economically important forest pathogen in the northern hemisphere
Basidiomycota: Tilletiales	<i>Tilletia</i> spp.		Smut/bunt	Grain: replaced by black spore masses	Wheat	
Basidiomycota: Uredinales	<i>Hemileia vastatrix</i>	Biotroph	Coffee rust	Leaves: rust-coloured, oval lesions on lower surface; leaves eventually drop	Coffee	Devastating in coffee plantations

Basidiomycota: Uredinales	<i>Puccinia graminis</i>	Biotroph	Black stem rust	Leaves, stems and heads: rust-coloured, diamond shaped, raised lesions; black teliospores when mature	Cereal grasses	Causes wheat losses of 10–70%, enough to feed 200–1410 people per annum
Basidiomycota: Uredinales	<i>Uromyces viciae-fabae</i>	Biotroph	Rust	Rust-coloured pustules surrounded by a yellow halo	Bean rust	
Basidiomycota: Ustilaginales	<i>Ustilago hordei</i>	Biotroph	Smut	Grain: replaced by black spore masses	Oats	
Basidiomycota: Ustilaginales	<i>Ustilago maydis</i>	Biotroph	Smut	Grain: galls are formed (Fig 8.8)	Maize	Formerly was a major problem. Now causes mean losses worldwide of 2–20%, enough to feed 26–260 million people. Losses in individual fields can approach 100%. Eaten as a delicacy in central America called huitlacoche
Oomycota: Peronosporales	<i>Bremia lactucae</i>	Biotroph	Downy mildew	Pale yellow angular patches on leaves delimited by veins. Downy white masses on underside of leaves beneath these patches, also appearing on upper surface later	Lettuce (<i>Lactuca sativa</i>)	
Oomycota: Peronosporales	<i>Phytophthora</i> spp.	Necrotroph	Root rots	Wilting; rotting roots; death when severe	Trees, shrubs, vegetables	Many emerging diseases
Oomycota: Peronosporales	<i>Phytophthora infestans</i>	Hemibiotroph	Late blight	Leaves: water-soaked lesions, followed by dead brown areas on lower leaves; white woolly growth on lower surface. Tubers: watery, dark rotted tissue	Potato	Cause of the Irish potato famine in nineteenth century. Causes 50–78% crop loss, enough to feed 80–1270 million people
Oomycota: Pythiales	<i>Pythium</i> spp.	Necrotroph	Damping off	Rotting of seeds and roots, and seedling death		
Phytomyxida	<i>Plasmodiophora brassicae</i>	Necrotroph	Clubroot	Wilting Roots: distortion, swelling, rotting when severe	Cabbage and other Brassicas	

^aEstimates of number of people who could be fed if crops were not destroyed were made by Sarah J Gurr.

Examples of some common and economically significant fungal and fungus-like pathogens and diseases of plants.

SUSCEPTIBILITY TO AND DEFENCE AGAINST FUNGAL PATHOGENS

Despite the large scale of some crop losses, most plants are resistant to most pathogens, most of the time. Three factors must interact to enable the development of disease: susceptibility of host, favourability of environment, and virulence/abundance of the pathogen. The likelihood of plants becoming diseased is often visualised by considering a disease triangle, in which these three factors are indicated on its sides (Figure 8.1a). If any of the three factors are zero, disease will not develop. The pathogen may be of a more or less virulent race or the inoculum size of the pathogen may vary (Figure 8.1b). Equally, the pathogen must be in an infective stage of its lifecycle and not in a dormant state for successful disease development to occur. With regard to the host, different plant species and cultivars often vary in their susceptibility to different pathogens, depending on their age, vigour, and their defence mechanisms. Plants may also be genetically uniform (monocultures) which will influence the rate of disease development by a given pathogen. With regard to environmental conditions, temperature, soil moisture, atmospheric humidity, wind, insolation, pH, nutrient status, and other aspects of soil quality, all have a major impact on disease development and severity, via their effects on fungal inoculum production, spread, growth and colonisation, and via their effects on plant vigour.

When a fungus encounters a plant, it is rarely able to be pathogenic, because plants have preformed physical and chemical defence barriers (described in more detail below p. 254), and even if these are overcome, the presence of a fungus will induce additional plant defences (explained in more detail on pp. 254–256). Most plants are, therefore, immune to most fungi (termed **non-host resistance**), and plants have the ability to reduce the severity of any potential fungal infection (termed **basal resistance**). Resistance initially involves the recognition of ‘non-self’ cues derived from the fungus – originally referred to as **elicitors** and more recently termed **microbe-associated molecular patterns** (MAMPs; or PAMPs in the case of pathogens). MAMPs are recognised when they bind to transmembrane proteins called pattern recognition receptors (PRRs), which go on to trigger plant defence responses termed PTI (pattern triggered immunity or plant innate immunity) (p. 254). This response is often swift and transient.

However, successful biotrophic pathogens (and mutualistic fungi), which grow in healthy hosts, can overcome the plants innate immunity by evading detection and/or by interfering with host defence responses. They do this by producing **effector** molecules (p. 255), that are induced during infection and are targeted to the plant cell apoplast, and in some cases to the plant cytoplasm, where they are able to suppress the plants innate immunity response resulting in effector triggered susceptibility (ETS). Of course the plant responds, and recognition of these fungal-derived virulence factors results in a second level of resistance, mediated by phytohormones and gene cascades culminating in effector triggered immunity (ETI), which is generally accompanied by the hypersensitive response (HR, pp. 255–256), programmed cell death, and restriction of pathogen ingress. So at the first level of plant immunity (PTI) recognises and responds to non-self molecules common to many microbes, including non-pathogens, and the second level (ETI) responds to pathogen effectors (virulence factors), either directly or indirectly, via their effects on host targets.

As mentioned above, these aspects of pathogenicity and plant immunity are attributed to fungi whose interests are to keep the invaded host plant cell alive. But what about the immunity elicited by plants in response to the group of fungi which benefit from the induction

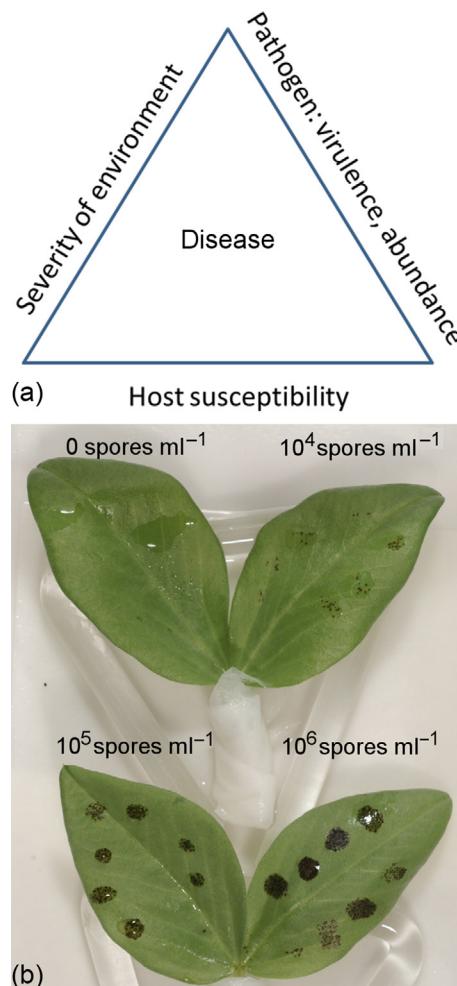


FIGURE 8.1 (a) The disease triangle. Each side of the triangle represents one of the factors involved in disease development. The length of each side can be set as proportional to the total of all aspects of each component that favour disease. The area, therefore, represents the amount of disease. If any of these factors are zero, disease will not develop. For example, if the plants are resistant then the host side and hence amount of disease would be small. In contrast, if the hosts were susceptible and planted densely, the host side would be large and so would the extent of disease, provided that the other sides were not zero. Likewise an extremely abundant, virulent pathogen would make a long pathogen side, and provided that environment was favourable and the host susceptible there would be a large amount of disease, and so on. (b) The inoculum potential (i.e. the amount of pathogen available) can influence success of colonisation, as illustrated here with different spore loads of the necrotrophic pathogen *Botrytis cinerea* added to the surface at eight points on broad bean (*Vicia faba*) leaves. Source: (b) © Peter Spencer-Phillips.

of plant cell death – the necrotrophs? Necrotrophic fungi can be divided into two groups, the broad host range necrotrophs and the host-specific necrotrophs. Notwithstanding that most necrotrophic fungi are unable to overcome the innate immune response of vigorous plants, broad host range necrotrophic infections on susceptible plant hosts can elicit host cell death (HCD) and other defense responses. However, unlike with biotrophic pathogens, HCD does

not confine fungal ingress but rather is an indicator of successful infection, in many cases enhancing pathogen colonisation. There are a small group of host-specific necrotrophic fungal pathogens which produce 'effector-like' host-selective toxins (HSTs), which are toxic only to the specific host of the disease and are ineffective against the vast majority of other plants; these HSTs mediate susceptibility of the host to the host-specific necrotrophic pathogen. When a necrotrophic fungus produces a HST and the specific host plant has the corresponding dominant susceptibility gene, then ETS is established. A growing amount of evidence is now emerging in favour of there being common signalling pathways associated with both necrotroph susceptibility (inducing ETS) and biotroph resistance (inducing ETI). It is feasible that HST pathogens can 'deliberately' activate host ETI responses directed against biotrophic pathogens in order to establish ETS and thereby thrive in environments that would not be conducive with biotrophic lifestyles.

The following three subsections provide more detail about preformed and induced defences, and a simple consideration of the genetic basis for host/pathogen compatibility. Readers who do not want this detail may wish to move directly on to key events in the disease cycle (p. 257).

Preformed (Constitutive) Defence

Plants can defend themselves against fungal attack with physical and chemical barriers. Plant cells have thick walls, and when they are on the outside of plants, they are usually covered with cutin or suberin. Leaves of land plants have surface waxes. Bark is a physical protection, but its chemical properties are also extremely important. Size, shape, and location of stomata can also influence pathogen entry. Plants contain a range of low-molecular-weight antimicrobial compounds (**phytoanticipins**) that inhibit germination and/or growth, including phenols and quinones, long-chain aliphatic and olefinic compounds, aldehydes, cyanogenic glucosides, saponins, terpenoids, stilbenes, glucosinolates, cyclic hydroxamic acids and related benzoxazolinone compounds, tannins, lysozyme, proteinase inhibitors, polygalacturonase-inhibiting proteins (PGIPs), defensins, anti-microbial proteins, peptides, and low-molecular-weight compounds. Low availability of nutrients to the pathogen also contributes to defence. Further, most plants are in some state of basal defence most of the time, as the innate immunity of plants, though rapid and transient, is constantly being activated, since plants are continuously encountering bacteria, fungi, and damage in the rhizosphere and phyllosphere.

Active (Induced) Defence

As well as preformed defence mechanisms, plants have evolved at least two lines of active defence. As already mentioned, the first provides basal defence (akin to innate immunity) against all non-self-organisms and is based on the recognition of structurally conserved MAMPs by PRRs that then activate a pattern triggered immunity (PTI) preventing further colonisation of the host tissue. MAMPs are typically evolutionarily conserved molecules required for growth and/or development of the fungus, and include chitin found in fungal cell walls, and β -glucan found in fungal and oomycete cell walls, both of which have corresponding plant PRRs. However, fungal pathogens continue to evolve mechanisms to overcome preformed defences, to evade MAMP detection and/or produce effectors that can suppress

the MAMP triggered immunity (PTI) thereby allowing the fungus to invade host tissue. Effectors are a group of mainly small secreted proteins (with very little homology to each other or existing proteins within databases). Fungal effector genes are typically induced upon host colonisation. Effector proteins are mainly produced in the ER and are secreted through the Golgi and are delivered to the apoplast where some are then subsequently transported into the host cytoplasm. Most are small secreted proteins with very little homology among them or to other proteins within fungal databases. They function either in the apoplast or within the cytoplasm of the host cell; yet how they are transported has not been established. Fungal apoplastic effectors typically include cell wall-degrading enzymes and necrosis- and ethylene-inducing (NEP1)-like proteins termed NLP's; however, the function of only a very few cytoplasmic effectors has been determined.

Plants have evolved a second active defence system, based on the recognition of such effectors or effector mediated changes in host components, by R proteins (which can be race-specific or race non-specific types) and the subsequent activation of ETI, which leads to a more rapid and enhanced defence response which is more robust than PTI. These responses are mediated by phytohormones such as jasmonic acid (JA), salicylic acid (SA), ethylene (ET), and abscisic acid (ABA). The JA and SA pathways are, in general, mutually antagonistic, with the JA/ET dependent pathway regulated by COI1/EIN2 genes and controlling responses to necrotrophs and chewing insects, and the SA pathway regulated by NPR1-like genes and controlling programmed cell death, which curbs the spread of biotrophs and hemibiotrophs as well as inducing systemic-acquired resistance (SAR). SAR is a resistance response of the whole plant at a distance from a site of localised infection by a pathogen. Effectively, it immunises the plant against future attack. The SAR response is mediated by a salicyclic acid-dependent process. MAP (mitogen-activated protein) kinase signal transduction cascades are involved in regulation of salicyclic acid accumulation. PRPs are formed and accumulate in SAR. Induced systemic resistance (ISR), unlike SAR, is mediated by a jasmonic acid/ethylene-dependent process, at least in some systems, and PRPs do not accumulate. Abscisic acid (ABA) plays a role in abiotic stress responses (drought, salinity, etc.) as well as influencing plant immunity through cross talk between the SA dependent pathway.

Plant defence responses vary in their time of activation, some occurring rapidly and others more slowly. The main rapid responses are an oxidative burst, generation of nitrous oxide (NO), cross-linking of cell wall proteins, and synthesis and deposition of callose concurrent with the HR reaction. HR is genetically programmed suicide of cells in the immediate vicinity of the invading fungus, a response which is particularly effective against biotrophs as they need living plant cells for growth and abstraction of nutrients. O_2^- and H_2O_2 are rapidly generated in many interactions between plants and fungal pathogens, and in conjunction with NO have antimicrobial activity. A weak response occurs in both compatible and incompatible interactions, but lasts longer (3–6 h) in the latter. Cross-linking of cell wall proteins occurs rapidly, making cell walls more resistant to hyphal penetration; its effect has been likened to self-sealing car tyres. Production of callose – a β -1,3-linked glucan – occurs rapidly as it does not require transcription or protein synthesis, just activation of the enzyme which is present in an inactive form. It is deposited as papillae localised in the plant cell wall, which again resists hyphal penetration.

Resistance responses that occur more slowly are: production of phytoalexins; lignification; suberisation; production of hydroxyproline-rich glycoproteins (HGRPs); production

of pathogenesis-related proteins (PRPs); systemic-acquired resistance (SAR); and ISR. These responses all require gene transcription, hence their slower occurrence. However, they can be primed by the presence of some beneficial microbes, including root colonisation by AM fungi (pp. 206–212). **Phytoalexins** are low-molecular weight compounds, with antimicrobial properties against a wide range of fungi and other organisms, which are synthesised by plants and accumulate in them after exposure to a potential pathogen. Pathogens differ widely in their sensitivity, and some have detoxification mechanisms. **Lignin** and **suberin** are both found in the walls of healthy plant cells, but synthesis and deposition increases during attempted infection, increasing resistance to penetration and decomposition. Likewise, HRGPs are present in healthy cell walls, but increase during infection; their function in defence is not entirely clear but they play a role in cross-linking proteins and provide a template for lignin deposition in papillae. PRPs include a range of acidic protease-resistant proteins with different functions, for example, glucanases and chitinases break down components of fungal cell walls, thionins increase permeability of fungal cell membranes, defensins affect fungal membrane transport, and others inactivate fungal ribosomes.

Genetic Compatibility/Incompatibility of Host and Fungus

Whether fungi have the ability to establish within living plant cells is determined at the genetic level (Table 8.3). The gene-for-gene hypothesis proposed by Harold Flor, based on his pioneering work on flax (*Linum usitatissimum*) and the flax rust fungus *Melampsora lini*, states that for every dominant avirulence (*Avr*) gene in the pathogen, there is a cognate resistance (*R*) gene in the host and that the interaction of the *Avr/R* gene products results in the activation of host defence mechanisms, such as the hypersensitive response (HR) that causes localised cell death and halts the ingress of biotrophic fungal pathogens. The first fungal *Avr* gene was cloned in 1991 by van Kan and colleagues, and the first oomycete *Avr* gene in 2004 by Shan and colleagues. Over the last 20 years a multitude of *Avr* and cognate *R* genes have been identified which has significantly enhanced our molecular understanding of host pathogen interactions. Attempts have been made to study binding of *Avr/R* gene products; however, this has proved difficult and resulted in the proposal of an alternative (guard) model, whereby *R* proteins may not directly bind to their corresponding *Avr* gene product, but instead monitor the state of host components that are targeted by these molecules. Consequently, the term

TABLE 8.3 Gene-for-gene interaction resulting in resistance or susceptibility of a plant to a potential fungal pathogen

Fungus genotype	Plant genotype			
	<i>R₁R₂</i>	<i>R₁r₂</i>	<i>r₁R₂</i>	<i>r₁r₂</i>
<i>A₁A₂</i>	Resistant	Resistant	Resistant	Susceptible
<i>A₁a₂</i>	Resistant	Resistant	Susceptible	Susceptible
<i>a₁A₂</i>	Resistant	Susceptible	Resistant	Susceptible
<i>a₁a₂</i>	Susceptible	Susceptible	Susceptible	Susceptible

A symbolises avirulence and *a* virulence genes in the fungus, as avirulence is dominant. *R* symbolises resistance and *r* susceptibility genes in the host plant.

'effectors' was adopted to describe pathogen-derived gene products that alter (either positively or negatively) the interaction between plant and pathogen. These include the previously termed avirulence genes of the biotrophs and the host-specific toxin (HST) genes of the necrotrophs. Effectors are induced by transcriptional regulators when the host is colonised, some pathogens tailoring expression of these in individual host tissues. Effectors are targeted to the apoplast or cytoplasm and are delivered to the host through invading hyphal tips or across specialised biotrophic structures called haustoria (Figures 8.6, 8.9).

The mode of nutrition of necrotrophs does not require them to maintain a healthy viable host and it was, therefore, thought that necrotrophic fungal pathogens do not follow the gene for gene model; however, a small group of necrotrophic pathogens, including *Alternaria*, *Stagonospora*, and *Cochliobolus*, produce HST's that can be categorised as effectors. These are small peptides that are recognised by plant susceptibility genes. These HST effectors promote disease in a particular host species (and sometimes only within specific genotypes of the host) when the host expresses a specific dominant susceptibility gene. In effect, the pathogen produces an effector capable of promoting disease and the host plant produces a receptor that is required for susceptibility. This is a mirror image of the gene for gene hypothesis of biotrophic fungi where dominant host resistance and pathogen avirulence gene products are required for resistance.

KEY EVENTS IN THE DISEASE CYCLE OF PATHOGENS

A series of reasonably distinct events occurs during the development and spread of a disease in a plant and plant population (Figure 8.2). These events are termed the disease cycle and usually correspond closely to the lifecycle of the pathogen, though some pathogens (e.g. rust fungi) have very complex lifecycles. The different phases of the disease cycle are considered below.

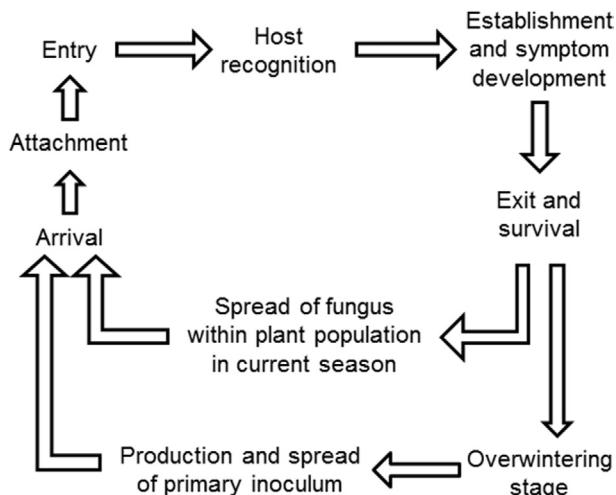


FIGURE 8.2 Events in the development of a disease cycle.

Arrival, Attachment, and Entry

The first key event in the infection cycle is for the fungus to come into contact with a potential host plant. For some fungi this is in soil, for others in the aerial environment, and yet others encounter plants in both. In soil, most plant pathogenic fungi are present as resting spores or sclerotia (pp. 60–61). Even when the microclimatic environment is suitable, most only germinate when mycostasis (p. 356) is overcome by the presence of nutrients exuded from living roots. Fungi which survive as resting spores have to await the arrival of roots in their vicinity, while others have motile zoospores that are attracted to their host roots but not to those of other species. For example, the fungus-like *Phytophthora cinnamomi* and *Phytophthora citrophthora* are attracted respectively to avocado (*Persea americana*) and citrus, but not vice versa.

Some fungi can reach roots as mycelium, particularly when an infected root contacts an uninfected one. This is a major means of spread of the tree root pathogen *Heterobasidion annosum* (Figure 8.11; pp. 276–277). The necrotroph *Rhizoctonia solani* (one cause of ‘damping-off’ disease of seedlings) grows freely through soil and utilises cellulose-rich substrata. It is as combative (pp. 349–351) as many obligate saprotrophs, aided by production of luxuriant mycelium and the ability to mycoparasitise (pp. 341–349) some fungi. Necrotrophic *Armillaria* spp. can grow between hosts as rhizomorphs (pp. 58–60). Biotrophs, hemibiotrophs and most necrotrophs are, however, unable to grow through soil.

Aerial plant surfaces are reached by spores transported by wind, rain splash or animal vectors (Chapter 3). There are many different types of spore (Chapter 3); some species produce several types and rust fungi produce up to five types during their life cycle (Table 8.6). Spores of some species germinate immediately in water or a humid atmosphere, rather than lying dormant. Others do not germinate in the absence of stimulants from the host, have endogenous self-inhibitors which prevent germination at high spore densities, and germination synchronises with dark periods, resulting in less chance of desiccation of hyphae. Spores of many facultative pathogens germinate more rapidly and extensively in the presence of nutrients derived by exudation, wounding or decay of the host.

Before fungi can parasitise plants they must breach their physical and chemical defences (see above, pp. 254–256). Entry occurs either through wounds, natural openings (e.g. stomata), or through an intact surface. Many obligate pathogens enter via stomata, though the powdery mildews (obligate) and many facultative pathogens enter directly through the intact plant surface. The site and mechanism of entry varies not only between species but can also vary between different types of spores of the same species.

Prior to penetration, germ tubes grow across plant surfaces and, in the case of many rusts, grow at right angles to veins, maximising the chance of encountering stomatal pores. Rust germ tubes respond to topographical and other stimuli (Figure 8.3). For the next stage of infection, adhesion to the plant surface must occur. An appressorium (Figures 8.5c–8.7, 8.9) forms on the plant surface or over openings, depending on the point where a particular species enters. These appressoria range from simple swollen cells, through lobed structures to complex infection cushions, that anchor the fungus prior to and during penetration. A thin penetration hypha (peg), which usually emanates from the centre of the appressorium adjacent to the plant, then effects entry into underlying tissues by wall-bound enzymes, including

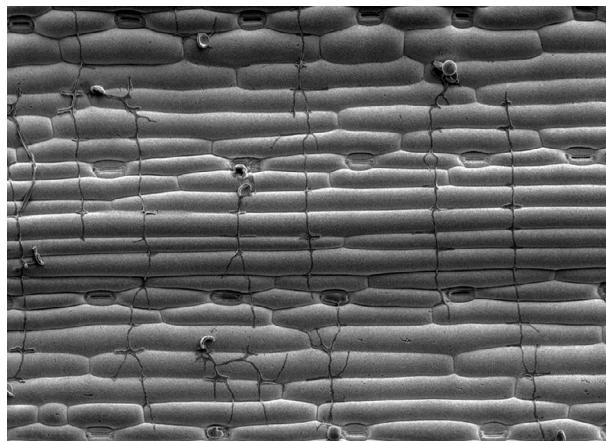


FIGURE 8.3 Hyphae of *Puccinia hordei*, cause of barley (*Hordeum vulgare*) brown rust, growing across the surface of a leaf. The hyphae are thigmotropic, and use the topography of the epidermal cells to orient their growth direction. Source: © Nick Read.

cutinases, and physical force. The physical force generated by turgor pressure in the penetration peg, which is usually insulated with melanin, can in some species reach $17\text{ }\mu\text{N }\mu\text{m}^{-2}$; if a force of this magnitude were exerted over the palm of a single hand, it would be sufficient to lift an 8000 kg bus! Changes in fungal cell biology that occur during these early stages are detailed in some of the case studies.

Following penetration of the outer cuticle, subsequent growth and exploitation differs considerably depending on the host–fungus combination, but the vast majority grow between and/or into host cells; the powdery mildews only grow over the external surface and penetrate epidermal cells with haustoria (p. 272). Different fungi acquire nutrients from plants in different ways, and have different effects on them, leading to different symptoms (Table 8.4). At the two extremes, necrotrophs and biotrophs operate very differently and have different effects on hosts (Table 8.1).

Establishment and Exploitation: Necrotrophic Pathogens

In susceptible plants, necrotrophs typically produce extracellular hydrolytic enzymes, necrosis-related proteins and/or toxins which kill host cells and make them available as nutrient resources. Hyphae penetrate between cells but cell death often occurs in advance of hyphae. When necrotrophs kill with extracellular enzymes, copious quantities of a wide variety of pectolytic enzymes are secreted from hyphal tips. Though plants only contain a small quantity of pectic substances relative to cellulose and hemicelluloses, they are vital to the middle lamella, and when broken down, cells leak and lose turgor, and tissues become soft and ‘watery’. This occurs, for example, in ‘damping-off’ (hence the name) diseases of seedlings and soft rots of fruit (Table 8.2). After using cell contents and pectic substances, cell wall components are broken down by cellulases and hemicellulases.

TABLE 8.4 Plant Disease Symptoms Caused by Fungal and Fungus-Like Pathogens

Name of symptom	Description of symptom
Abnormal growth	Including enlarged gall-like or wart-like swellings on above ground tissues, leaf curls, profuse branching to form witches brooms
Anthracnose	Sunken, dead blemishes on above ground plant parts
Blast	Destructive lesions on leaves and flowers
Blight	Browning of above ground plant parts
Canker	Localised wounds on woody stems; often sunken tissue
Damping off	Rapid death and collapse of seedlings, especially in warm, wet conditions
Decline	Loss of vigour
Dieback	Death of twigs starting at tips and advancing distally
Mildew	Chlorotic or necrotic tissue covered with mycelium and spores, giving a woolly or powdery appearance
Rots	Disintegration of plant tissues
Rusts	Small rust-coloured lesions on stems or leaves
Scab	Localised, scab-like lesions
Spots	Localised, dark-coloured lesions on leaves
Wilts	Wilting, discolouration and death of leaves, shoots and sometimes whole plants

Fungi produce a wide range of toxins with a variety of modes of action (Table 8.5). Some are host-selective/specific (i.e. toxic to host plants but not to non-host plants), whereas others are non-selective/non-specific (i.e. not related to the host range of the fungus). Plant resistance to a certain necrotrophic pathogen often results from insensitivity to the toxin that the fungus produces or from the ability to degrade the toxin rapidly. When toxins kill host cells, tissues often remain dry, but discoloured, and lesions remain discrete and spread slowly (e.g. *Alternaria* leaf spot diseases), though in vascular wilts they are spread over large distances in xylem (see below). Many toxins can, however, affect membrane permeability resulting in leakage of solutes, as with pectinases. Following killing by toxins, the fungi produce extracellular enzymes and utilise host tissues for their nutrition.

Vascular wilt fungi grow and proliferate in the vascular elements of plants, where they utilise nutrients in xylem sap. They produce both low and high molecular weight toxins in the xylem, the former being transported to leaves where they impair functioning of stomata, causing loss of transpirational control and leaf death. High-molecular-weight toxins increase the viscosity of sap, which also contributes to leaf death. In addition, vascular wilt fungi form extremely small spores which are transported in the xylem and often become lodged on and block the perforated end walls of the xylem vessels. Here they germinate, the germ tube grows through to the next vessel, spores are immediately produced and the process continues (see the section on 'Vascular Wilts; pp. 272–274). The plant's resistance mechanisms may also exacerbate the problem by forming gums and tyloses that block xylem vessels. Plants die rapidly as a result of these activities, herbaceous plants often being killed within a few days and large trees within a few weeks or months.

TABLE 8.5 Examples of Host-Specific and Non-Host-Specific Toxins Produced by Necrotrophic Pathogens of Plants

Toxin	Fungus	Disease	Effects and other properties
<i>Host specific</i>			
ACT-toxin, ACR-toxin, AF-toxin, AK-toxin, AM-toxin,	<i>Alternaria alternate fornae</i> <i>specialis</i>	Respectively leaf spots of tangerine, rough lemon, strawberry Japanese pear, apple,	Some (AF-, ACT-) act at the plasma membrane; others (ACR-) affect mitochondria
HC-toxin	<i>Cochliobolus carbonum</i> race 1	Northern leaf spot and ear rot of maize	Deacetylates chromatin, hence affects gene expression
HS-toxin	<i>Bipolaris sacchari</i>	Eye spot disease of sugar cane	The toxin is a terpenoid
Peritoxins (PC-toxin)	<i>Periconia circinata</i>	Sorghum root rot	Inhibition of mitosis and of growth of primary roots; electrolyte leakage
Ptr Tox A, Ptr Tox B	<i>Pyrenophora tritici-repentis</i>	Tan spot of wheat	Tan necrosis and extensive chlorosis
T-toxin	<i>Cochliobolus heterostrophus</i> race T	Southern corn (maize) leaf blight	Male sterility
Victorin	<i>Cochliobolus victoriae</i>	Victoria blight of oats	Leaves become a bronze colour. Membrane depolarisation; ion leakage; inhibition of protein synthesis
<i>Nonhost specific</i>			
Cercosporin	<i>Cercospora</i> spp.	Various	Generates active oxygen species; it is photactivated
Cerato-ulmin	<i>Ophiostoma novo-ulmi</i>	Dutch elm disease	A hydrophobin
Enniatins	<i>Fusarium</i> spp.	Dry rot of potato	Ionophores
Fomannoxin	<i>Heterobasidion annosum</i>	Root and butt rot of conifers and also some broadleaves	Inhibits plant cell growth and protein synthesis
Fumonisins	<i>Fusarium moniliforme</i> (= <i>Giberella fujikuroi</i>)	Ear rot of maize	Inhibits sphingolipd biosynthesis
Fusicoccin	<i>Fusicoccum amygdali</i>	Canker of almonds and peaches and wilt	Irreversible opening of stomata
Solanopyrones	<i>Alternaria solani</i> , <i>Ascochyta</i> <i>rabiei</i> (= <i>Didmella rabeiae</i>)	Chickpea blight	Inhibitor of DNA repair
Tentoxin	<i>Alternaria</i> spp.	Various	Chlorosis
Trichothecenes	<i>Fusarium</i> species	Head blight, root rot, and seedling blight of wheat	Inhibit protein synthesis

Information largely from Strange (2003).

Even at early stages of disease, or when pathogens remain localised, physiological processes are influenced throughout the plant. Host metabolism and biosynthesis increase, leading to rapid use of energy, increased respiration, and rapid depletion of carbohydrate reserves. Photosynthesis can be affected by damage to chloroplasts, rapid ageing and premature senescence of leaf tissues. Changes in plant water relations result from decreased uptake from soil if roots are damaged, changes in rate of transpiration, changes in water potential gradients, and resistance to flow through the plant, as well as from wilt diseases. Carbohydrate metabolism and movement around the plant are altered indirectly by disruption of water transport and directly by reducing the amount of carbohydrate leaving infected leaves. Plant-wide symptoms often result from hormone changes as a result of production by the fungus or modification of those produced by the plant. For example, epinasty (downward curving of leaves) is a symptom of over production or increased sensitivity to ethylene. Ethylene also has a role in chlorosis and necrosis. Decrease in indole acetic acid (IAA) can cause premature abscission resulting in leaf drop.

Establishment and Exploitation: Biotrophic Pathogens

In contrast to necrotrophs, there is a more balanced relationship between plant and pathogen, at least for much of the time of the association. Biotrophic pathogens abstract nutrients from host cells, but rather than producing vast quantities of extracellular enzymes or toxins, their production is controlled and probably only 'switched on' when required. Some enzymes are probably wall-bound having only localised effects. Further, it has been suggested that host enzymes may work for the fungus; invertases may cleave sucrose into glucose and fructose, (e.g. in broadbean, *Vicia faba*, infected with *Uromyces fabae*), when carbohydrate source tissues become sinks. As well as producing extensive intercellular hyphae, biotrophic fungi penetrate host cells with a narrow hyphal branch which then expands to form variously shaped structures – termed haustoria – which provide a large surface ([Figures 8.6, 8.9](#)). Haustoria, do not, however, penetrate the plasmalemma (see Broad bean rust case study for further details). Nutrient acquisition from the host can occur both via intercellular hyphae and haustoria. In the case of *Uromyces fabae*, amino acid uptake is via both, but sugar uptake is solely by haustoria. Haustoria also perform some biosynthetic activities, including synthesis of vitamin B1.

Whilst biotrophic pathogens do not kill host cells, they affect host functioning considerably. Their success depends largely on maintaining a balance between extracting nutrients for growth plus reproduction and impairing the ability of the host plant to produce further nutrients. The fungus acts as a sink for nutrients. Metabolites commonly accumulate at infection sites, both as a result of increased rate of transport to these regions and a reduction in nutrient movement away from them (e.g. rusted primary leaves of broadbean import up to forty times as much assimilate as uninfected leaves). Movement of nutrients towards infected regions occurs partly because the fungus acts as a sink, causing diversion of nutrients from other plant parts, and as a result of the fungus producing cytokinins – plant hormones. Localised increase in cytokinin concentration gives rise to 'green islands' typical of powdery mildew and rust infections of many plants. These result from chlorophyll retention in the vicinity of infections for longer than in other parts of leaves, and in retention of the capacity for protein synthesis. Effectively, the plant cells are maintained in a juvenile condition.

Biotrophs also have other effects on plant development via effects on hormones, either by synthesising or degrading plant hormones, or by altering the plant's metabolism of hormones and by interfering with the plant tissue response to hormones. Alterations in the quantity and type of auxins, cytokinins and gibberellins can result in hypertrophy (increase in the size of tissues/organs), hyperplasia (cell proliferation), and abnormal differentiation of organs. The witch's brooms caused by *Moniliophthora perniciosa* on cocoa, for example, form when lateral buds are released from apical dominance.

Exit and Survival

A successful pathogen produces dispersal structures once it has become established. These can be asexual or sexual spores, or mycelium, depending on the type of pathogenesis, fungus species and time of year (see the section on '[Case Studies](#)'). During the growing season of the host, dispersal structures aid spread between potential hosts, whereas following death of whole plants or parts of plants, pressure on the pathogen is for survival. These different requirements are reflected in many leaf and stem pathogens by the production of asexual spores which spread rapidly during the growing season, and sexual spores that lie dormant throughout the adverse season for host growth. Interestingly, in black stem rust of wheat caused by *Puccinia graminis* f.sp. *tritici*, pustules which had been forming uredospores switch to produce thick-walled teliospores (p. 18) as autumn approaches. Switching from vegetative growth to spore formation, and between formation of different spore types relates to changes in weather, light, and the host. Unspecialised, necrotrophs often kill their hosts rapidly and produce resting stages after a short period of growth: the fungus-like *Pythium* and *Phytophthora* species form oospores on young colonies, while *Fusarium* spp. and *Rhizoctonia* spp., respectively, produce chlamydospores and sclerotia in response to nutrient depletion. Obligate biotrophs (e.g. those species that cause downy mildew, powdery mildew, and rusts) are dependent on spores for dispersal as they are unable to grow in dead tissues.

Spores can be dispersed by wind over long distance – across continents and even between continents ([Figure 8.4](#), see also Chapter 3). This is particularly important for establishing emerging diseases in new areas and for re-establishing diseases in areas where host plants have been absent for several seasons. Single-step invasions of disease from one continent to another by airborne spores are rare. However, examples include sugarcane rust (*Puccinia melanocephala*) which was introduced into the Dominican Republic and thence more widely in America from Cameroon (West Africa) as uredospores moved across the Atlantic Ocean in June 1978 by cyclonic winds. Coffee leaf rust (*Hemileia vastatrix*) probably moved in the same way from Angola to Brazil in 1970. Other diseases have been moved between continents first in infected material and then by wind-blown spores across the continent to which they have been introduced. Potato late blight (see the section on '[Case Studies](#)') was introduced to Europe in infected tubers and then spread as spores across Europe. Yellow rust (=stripe rust) of wheat moved to Australia in 1979 as spores of *Puccinia striiformis* f.sp. *tritici* on clothing and then across the continent and to New Zealand as wind-blown uredospores. Many biotrophs have cycles of extinction after host death followed by recolonisation of the new crop. This can occur over large distances (500–2000 km) and is in some ways analogous to the annual migration of some insects and birds, for example, the downy mildew *Peronospora tabacina* on tobacco in the eastern

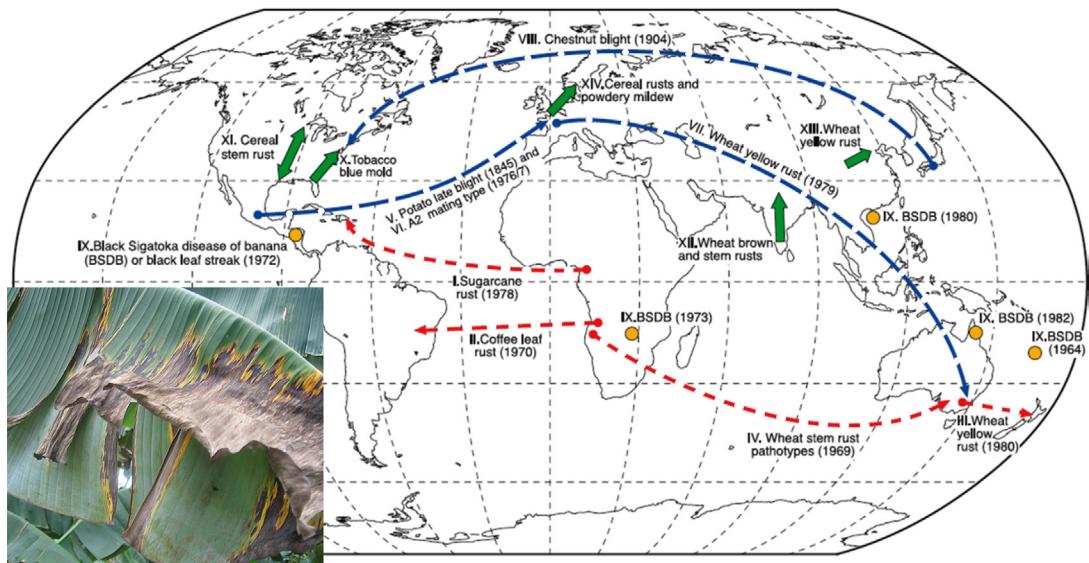


FIGURE 8.4 Plant pathogenic fungi can be transported extremely long distances by wind dispersal of spores. Though travel between continents is a rare event, unusual atmospheric conditions do sometimes occur, leading to dispersal and establishment of disease far from its long established origins. Red (black in the print version) arrows (on lines with short dashes) indicate disease spread by direct movement of airborne spores while blue (dark grey in the print version) arrows (on lines with long dashes) show where pathogens were first spread to new regions in infected plant material or by man and then onwards as airborne spores. Orange circles (grey in the print version) show the global spread of black Sigatoka disease of banana (inset) caused by *Mycosphaerella fijiensis*, the first recorded outbreak on each continent is indicated by IX. Green arrows show five examples of diseases which periodically migrate via airborne spores from one region to another in extinction (due to absence of crops over one or more seasons)- recolonisation cycles (X, XI, XII, XIII, and XIV). The inset shows the symptoms of black Sigatoka disease of banana caused by *Mycosphaerella fijiensis* © John Lucas. Source: *Brown and Hovmöller (2002)*.

United States, rust pathogens of wheat in North America, and wheat yellow rust which reestablishes each autumn in northern China as there are no wheat plants during summer (Figure 8.4).

Exit and/or survival as somatic structures is largely confined to necrotrophs, since most biotrophic pathogens have highly specialised physiological relationships with their hosts, and little or no saprotrophic ability. However, some species of Taphriniales and Ustilaginales can grow saprotrophically in a yeast form on the surface of leaves, and this has a survival role.

Initially, necrotrophs utilise tissues which they have killed, unhindered by other fungi. However, competition from antagonistic obligate saprotrophs (Chapter 10) will increase with time and the initial advantage lost. Four different types of behaviour occur amongst necrotrophs in response to competition. Firstly, some necrotrophs simply survive in a dormant vegetative form or grow and utilise/decompose organic matter very slowly. This is typical of some stem pathogens. *Oculimacula yallundae* (= *Cercospora herpotrichoides*), the cause of eye-spot lodging in wheat and barley, only has limited ability to decompose cellulose and hence grows slowly, but it is able to form pseudoparenchyma (p. 63) within dead host tissue and can remain viable for up to 3 years in this form despite the presence of competitive saprotrophs.

Secondly, some necrotrophs actively colonise tissues that were uninhabited prior to host death (e.g. *Cochliobolus sativus* in cereal roots and *Gaeumannomyces graminis* [cause of take-all disease] in cereals). If sufficient resources have been secured before combative saprotrophs invade, then survival is ensured, although resistant spores may also assist. Thirdly, some necrotrophs actively grow into soil. *Rhizoctonia solani* (one cause of 'damping-off' of seedlings) grows freely through soil producing luxuriant mycelium, utilising cellulose-rich substrata, and is as combative as many obligate saprotrophs, being mycoparasitic on some fungi. It also produces resistant sclerotia (pp. 60–61). Lastly, some necrotrophs extend into soil but are not combative whilst migrating to new resources. For example, *Armillaria* species pathogenic on trees grow as rhizomorphs, insulated from their surroundings and supplied with nutrients from host residues.

CASE STUDIES

Rice Blast Disease

Rice blast disease, caused by *Magnaporthe oryzae* (Ascomycota), occurs in about 80 countries on all continents where rice is grown, in both paddy fields and upland cultivation. The extent of damage caused depends on environmental factors, but worldwide it is one of the most devastating cereal diseases, resulting in losses of 10–30% of the global yield of rice. In rice seedlings, small necrotic regions appear initially, which become larger and coalesce, and have chlorotic margins (Figure 8.5). In older rice plants, disease symptoms can occur in leaves, collar – junction of the leaf blade and leaf sheath, nodes, neck, and panicle (Figure 8.5). Neck rot and panicle blast are particularly devastating causing up to 80% yield losses in severe epidemics. Triangular, purple-coloured lesions form on the neck node which elongate on both sides, seriously impairing grain development. The panicles become white when young neck nodes are invaded; infection later in plant growth results in incomplete grain filling.

The disease cycle begins when a conidium lands on a rice plant and becomes attached to the host surface through production of spore tip mucilage. This is followed by a series of developmental steps: germination, germ tube growth, formation of an appressorium, emergence of a penetration peg from the appressorium, and subsequently invasive growth in the host (described in Figure 8.5). Appropriate development of infection structures on the leaf surface involves sensing plant-derived cues, transduction of signals through G-protein-coupled receptors; activation of signalling cascades mediated by cAMP, mitogen-activated protein kinase (MAPK; proteins that are evolutionarily conserved and function as key signal transduction components in fungi, and also plants and mammals), phospholipase- and calmodulin-dependent pathways and resultant up-regulation and down-regulation of genes. The genome sequence for strain 7-15 was published in 2005 and today there are over 30 genomes available from strains around the globe. There are up to 1500 secreted proteins in the genome, the function of many being unknown, but some are certainly effector proteins. Many novel genes/metabolic clusters are being found in different isolates of the fungus, so large scale sequencing will be revealing. Over 20% (2154) genes are differentially expressed, mostly up-regulated during the processes prior to invasion. Temporal analysis of the transcriptome during infection, together with gene replacement experiments, identifies virulence-related genes required for successful invasion. For example, avirulence in some strains of

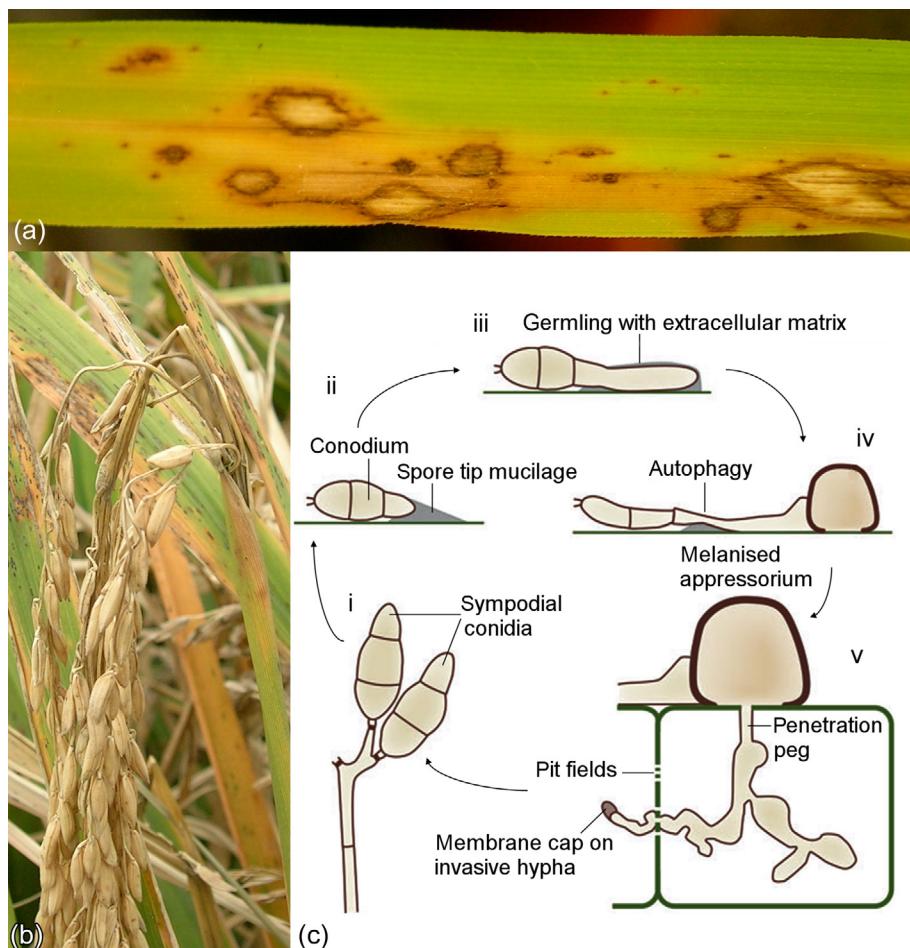


FIGURE 8.5 Rice blast disease caused by *Magnaporthe oryzae*. (a) necrotic lesions on rice seedlings. (b) Neck and panicle blast. (c) The disease cycle: (i) The teardrop shaped conidia have three cells each with a single nucleus. (ii) Germination proceeds commonly from the tapering end and the nucleus passes into the germ tube, where mitosis occurs. One daughter nucleus moves back into the spore and the other moves into the swollen tip of the germ tube. Appressorium initiation is controlled by progression of the latter nucleus into the S-phase of the cell cycle, which is within 2–4 h on a hydrophobic surface. Appressorium differentiation is controlled at the G2-M transition. At an environmental level, appressorium formation depends on plant-based and non-plant cues, including physical cues, such as hydrophobicity and hardness (appressoria will form on artificial surfaces), plant cutin monomers and nitrogen depletion. (iii) Autophagy of the spore and germ tube occurs and glycerol synthesised from the lipid and glycogen moves into the appressorium. Autophagy is essential for virulence, since the appressorium forms at the expense of the conidial contents, as shown by targeted mutation of the autophagy gene *MgATG8* which resulted in an avirulent strain. (iv) Up to 8 MPa turgor pressure (i.e. 40 times higher than a car tyre) is generated, by accumulation of glycerol, within the single-celled, dome-shaped appressorium, which is insulated by a layer of melanin in the wall. This turgor pressure, supplied in a very controlled manner, allows the penetration peg to force its way through the cuticle and cell wall, but the plasma membrane remains intact and becomes invaginated around the penetrating hypha. This is a pivotal recognition point for fungus and host. The plant responds by production of reactive oxygen species (ROS) around the infection site, but the fungus has evolved a rapid ROS detoxification mechanism. Growth into the next cell is through clusters of plasmodesmata, termed pit fields. The hyphae that penetrate into adjacent cells have a membrane cap comprising membrane lamellae, which probably secrete protein into the cytoplasm of the living host cell. (v) Emergence of conidiophores 4–5 days after infection. Source: (a, b) From Galhano and Talbot (2011); (c) Ebbole (2007).

Magnaporthe oryzae has been traced to loss of function in several specific genes: two of these encode proteins in signalling pathways linked to G-protein-coupled receptors, both of which are required for appressorium formation.

In susceptible hosts, the fungus ramifies between and within cells, probably spreading from one plant cell to another via plasmodesmata. The fungus is hemibiotrophic, initially feeding on living cells but subsequently causing death. Lesions on the plant surface start to form when host cell death begins 4–5 days after infection. Melanised macroconidia are then produced on conidiophores that protrude from lesions, with up to about 20,000 spores produced from one lesion on a leaf, and 60,000 on a single spikelet in one night. Spore release is triggered by a 1–2 h period of darkness. The fungus can sporulate repeatedly for around 20 days. The disease is polycyclic (i.e. multiple infection cycles in a growing season) with 7 days between spore germination and production of conidia.

Because rice provides almost 25% of the calories consumed by humans, understanding the biology of this pathogen is particularly important for developing disease control strategies. Moreover, the pathogen has now jumped hosts to wheat, and is now an urgent problem in South America. Due to its agricultural impact and tractability, *Magnaporthe oryzae* has become a model fungus for studying host pathogen interactions, at both the cell biology and molecular level. Rice blast populations comprise sets of discrete clonal lineages with a limited spectrum of virulence. The most cost-effective, and hence preferred, way of managing rice blast disease is by growing high yielding cultivars with single, dominant resistance genes; over 70 blast resistance (*R*) genes have been identified. However, the problem with single-locus resistance is that it is often short-lived, only lasting for a few years. To lessen the chance of *R* gene breakdown broad-spectrum *R* genes can be deployed, which provide resistance to different strains of the fungus (e.g. resistance gene *Pi2* confers resistance to over 450 isolates). Another proposed strategy is to stack several *R* genes, having different resistance spectra, into a single rice cultivar; this has also been proposed for control of cereal rusts. The use of antagonistic phylloplane bacteria and fungi is also being tested. Fungicides are available but these have attendant problems of cost, development of fungal resistance and damage to ecosystems.

Rust

Black stem rust of wheat, caused by *Puccinia graminis* f.sp. *tritici* (Basidiomycota), is a major concern for the world's food security, causing crop losses of up to 70%, enough to feed several hundred million people (Table 8.2). Not surprisingly, this rust fungus has been extensively studied. Its lifecycle has already been described in Chapter 1 (pp. 18–19), so here we consider broad bean rust which, though less important overall for global food security, has also been studied in detail and has long been used as a model for studying cytology, physiology, biochemistry, and molecular aspects of rust fungus biology. *Uromyces fabae* causes disease of many legumes, including broad bean (*Vicia faba*), with up to 50% losses of yield, and over 50 other *Vicia* spp., 20 *Lathyrus* spp., lentil (*Lens culinaris*), and pea (*Pisum sativum*). Like other rusts, it is an obligate biotroph.

Uromyces fabae is macrocyclic (i.e. multiple disease cycles during each growing season), and produces all five spore forms found in the Uredinales. Its lifecycle and the role of the different spore types are shown in Figure 8.6 and Table 8.6. **Telia** form on stems and leaves

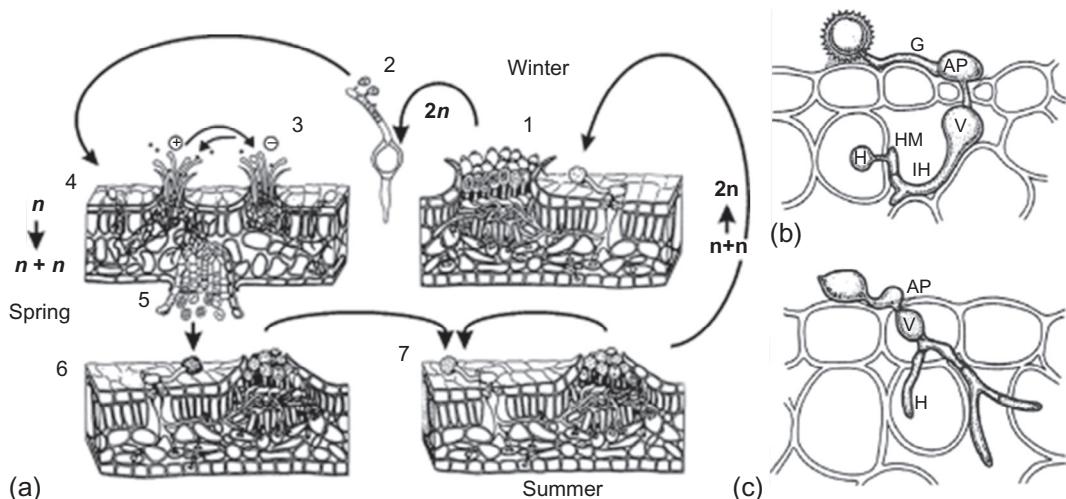


FIGURE 8.6 The life cycle of *Uromyces fabae*, cause of broad bean (*Vicia faba*) rust, is extremely complex. (a) (1) The fungus overwinters as telia, which produce teliospores ($2n$), surviving in crop residues in the field and also on seed. The teliospores are spread by wind. (2) They germinate on a plant in spring to form a metabasidium with four haploid basidiospores (with two mating types [p. 115]). (3) These germinate and infect the plant (b) – see below for detail. (4) Pycnia are formed, on the upper surface of leaves, which contain haploid pycniospores. The latter are exchanged between pycnia of different mating type (+, -). (5) Spermiation (p. 112) occurs, and then aeica, containing dikaryotic ($n+n$) aeciospores, are produced on the lower surface of the leaf. The aeciospores spread the infection within the crop and to other nearby bean crops. (6) Uredinia are then produced on the stems and leaves; they contain dikaryotic uredospores, which (7) also spread the fungus throughout the crop canopy. Late in the season, the uredinia differentiate into telia and remain in crop debris ready for the infection cycle to start again on next season's crop. (b) When uredospores land on a plant surface they have an irregular shape, because they are completely dehydrated. They rehydrate rapidly and become ellipsoidal. Their surface is hydrophobic, which facilitates initial adhesion to plant surfaces. An extracellular matrix of carbohydrates and glycosylated polypeptides form, followed by an adhesion pad. Spores only germinate when fully hydrated, at temperatures between 5 and 26°C (optimum 20°C), and after at least 40 min of darkness, but will do so on almost any surface. When the germ tube (G) grows, cytoplasm moves into it from the spore. An appressorium (AP) forms when appropriate signals concerning leaf surface topography are received. Cytoplasm moves from the germ tube into the appressorium and a septum separates the two structures. A penetration peg is forced, by turgor pressure, into the stomatal cavity. A vesicle (V) forms and from this an infection hypha (IH) develops. On contact with a mesophyll cell a haustorial mother cell (HM), with a thick multi-layered wall, is formed and attaches to the plant cell wall. All of the cytoplasm moves into this, and is walled off by a septum. A penetration hypha breaches the mesophyll cell wall by localised production of enzymes, including pectin esterases, pectin methylesterases, and cellulases, and a haustorium (H) is formed causing the host plasma membrane to invaginate – so the haustorium is not truly intracellular. A matrix forms around the haustorium, and this is the site of nutrient uptake and information exchange. (c) Unlike uredospores, teliospores have smooth, thin walls. The infection structures that develop from them – appressoria (AP), vesicles (V) and haustoria (H) – are much less differentiated. Further, penetration pegs enter directly into plant cells rather than via stomata. Source: Modified from Voegeli (2006).

late in the season, remaining in crop debris after harvest, the fungus over-wintering as **teliospores**. In spring teliospores germinate to form a **metabasidium** that produces **basidiospores**, that themselves germinate to form monokaryotic (p. 113) infection structures. During the rest of the growing season infection mainly occurs following germination of **uredospores**, which with **aeciospores** spread the fungus throughout the canopy and between plants.

TABLE 8.6 *Uromyces fabae* spore types

Spore type	Ploidy	Produced by/in	Infect plant tissues	Position in lifecycle
Teliospores	Diploid	Telia	No	Nuclei fuse during teliospore production. Overwintering stage in plant remains in the field and on seed. Germinate to produce a metabasidium
Basidiospores	Haploid	Four produced by each metabasidium following meiosis. Two mating types	Yes	Smooth, thin-walled. Germinate on host in spring and produce infection structures
Pycniospores (spermatia)	Haploid	Pycnia	No	They act as sperm and enter a receptive hypha of a pycnium of a different mating type. Dikaryotisation occurs in aecial primordial
Aeciospores	Dikaryotic	Aecia	Yes	Aecial spores germinate and produce an infection structure from which uredia are produced
Uredospores	Dikaryotic	Uredia	Yes	Thick-walled with spines and darkly pigmented. The main asexual spore produced in vast quantities and dispersed aerially. Repeatedly infect host plants during the summer. Uredia differentiate into telia during the fall

The events following germination are well characterised for basidiospores and uredospores. The infection processes are rather different. Both produce appressoria with a penetration peg beneath, followed by a vesicle when entry into the plant has been effected, and subsequently a haustorium within a plant mesophyll cell, but these structures are less differentiated when they are produced following germination of basidiospores (Figure 8.6). Also, the penetration peg produced following basidiospore germination enters an epidermal cell directly and then forms a vesicle, whereas that from an uredospore enters via a stoma and produces the vesicle in the stomatal cavity. As mentioned previously (p. 258, Figure 8.3), the germ tubes produced by rust spores can follow topographical features of leaves to locate natural openings. *Uromyces appendiculatus* can recognise the height of the lip of the guard cells (0.5 µm) on broad bean leaves. Rust fungi also detect chemical signals (e.g. leaf alcohols).

The haustorium is the site of uptake of carbohydrate and amino acids, though the latter can also be taken up via intercellular hyphae, and also synthesises other nutrients *de novo*. The haustorium plays a major role in primary metabolism. *Uromyces fabae* has evolved several mechanisms to avoid host recognition, including masking the chitin in infection structures by acidic cellulases and protease action, conversion of chitin to chitosan, release of mannitol that suppresses host defence responses involving reactive oxygen species (ROS), production of protein effectors that take control of host metabolism. *Vicia faba* gene expression patterns not only alter in the vicinity of infection, but also in remote organs (e.g. stems and roots).

Control is by use of resistant races, fungicide application and/or horticultural practices such as appropriate rotation with non-host crops and removal of plant debris containing the fungus.

Colletotrichum Anthracnose and Blights

Almost all crops worldwide are susceptible to one or more *Colletotrichum* spp. (Ascomycota), causing anthracnose (sunken dead spots) and blights (tissue browning) of aerial tissues. *Colletotrichum* can also be latently present causing post-harvest rots, infecting tissues pre-harvest but not developing overtly until after harvest. They cause major economic loss of fruits and vegetable crops, including staples in developing countries (e.g. banana, cassava, and sorghum). Spores germinate and enter the plant via a fine penetration peg produced beneath an appressorium (Figure 8.7). *Colletotrichum* is hemibiotrophic, initially establishing itself biotrophically within the plant. First an intracellular vesicle is formed, and from this a few large intracellular primary hyphae develop and extend into only a few cells (Figure 8.7). These hyphae and the vesicle are surrounded by a matrix which is the interface with the plant apoplast. Specific genes are expressed during the biotrophic phase, including *C1H1* which encodes a glycoprotein, and *CgDN3* which is thought to maintain the biotrophic phase of development. Subsequently, the fungus switches to a necrotrophic phase in which narrower hyphae ramify through host tissue. These hyphae secrete endopolygalacturonases and other cell wall-degrading enzymes. *Colletotrichum gloeosporioides* secretes a *pelB*-encoded

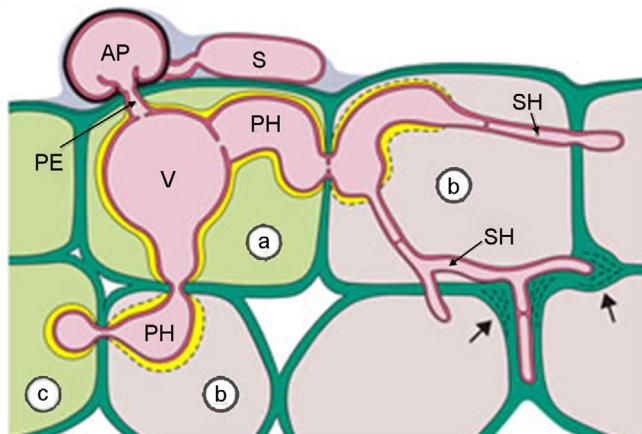


FIGURE 8.7 *Colletotrichum* anthracnose. Most species are hemibiotrophic as seen in this diagram of infection by *Colletotrichum lindemuthianum*. A spore (S) attaches to the surface of the host. When it germinates, it produces a short germ tube, which differentiates into an appressorium (A), from the underside of which develops a penetration peg (PE) which pierces the cuticle and wall of the epidermal cell. The hypha swells to form a vesicle (V) from which develop broad primary hyphae (PH) surrounded by plant plasma membrane. This is the biotrophic stage (a); the plant cell remains alive, and the host and fungal protoplasts remain separated by an interfacial matrix (indicated by yellow (light grey in the print version) colouring). After 1 or 2 days the plant plasma membrane begins to disintegrate and the host cell dies (b). A sequence of colonisation of plant cells by new primary hyphae occurs (c) with subsequent death after a few days. The biotrophic phase ends when narrow secondary hyphae (SH) develop from the primary hyphae. The secondary hyphae are not surrounded by host membrane/interfacial matrix, and secrete plant cell wall-degrading enzymes (indicated by arrows) in this necrotrophic phase. Source: Mendgen and Hahn (2002).

pectate lyase which not only breaks down cell wall components but also reduces host defence responses which are triggered by released oligogalacturonides.

The fungus survives between cropping seasons within crop residues where it can grow saprotrophically. The disease is spread by asexual spores via water splash, wind, and invertebrates. Germination and infection require high (near 100%) humidity, and pre-harvest disease is most serious at warm (25–20 °C) temperatures. Post-harvest disease, however, can occur in much drier conditions, when tissues are damaged or through ageing, as the fungus is already latently present.

Corn Smut

Smuts are caused by members of the Ustilaginales (Basidiomycota) and occur worldwide, with over 1200 species. Most attack the ovaries of Gramineae, and develop in them and the grain. Until the twentieth century, smuts, along with rusts, were the major cause of grain loss. They are now controlled using resistant varieties and seed treatment to kill teliospores and mycelium.

Corn smut, caused by *Ustilago maydis*, affects corn wherever it is grown. Its complex life-cycle has been introduced earlier (pp. 16–18). It overwinters as teliospores (diploid) in soil and on crop debris, and these resistant spores can remain viable for several years (Figure 8.8).

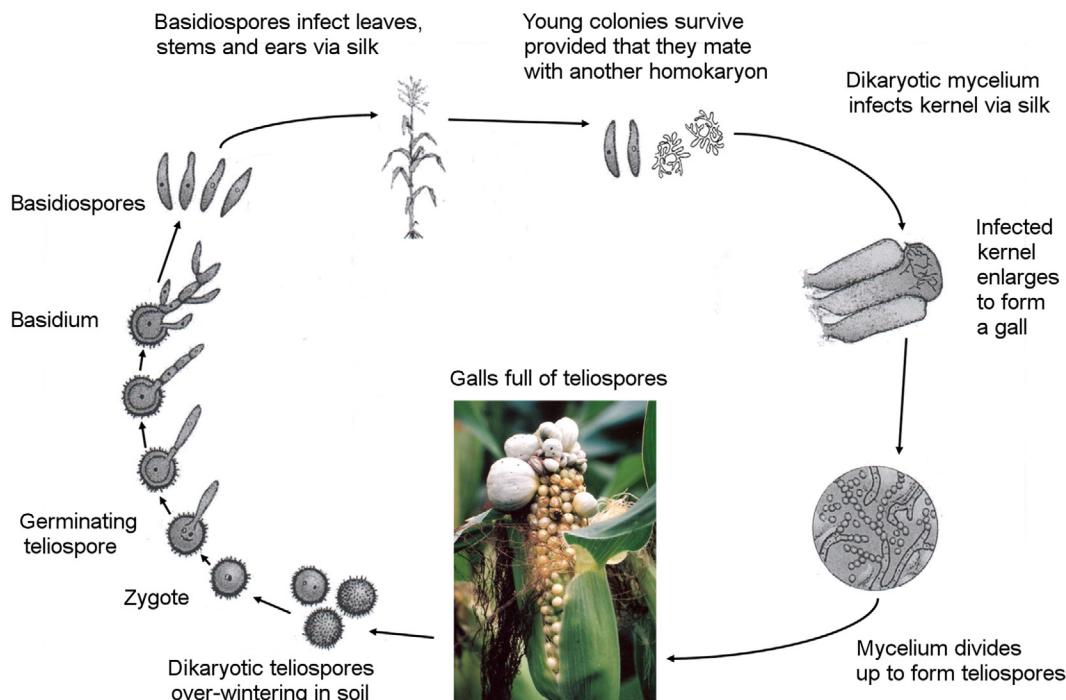


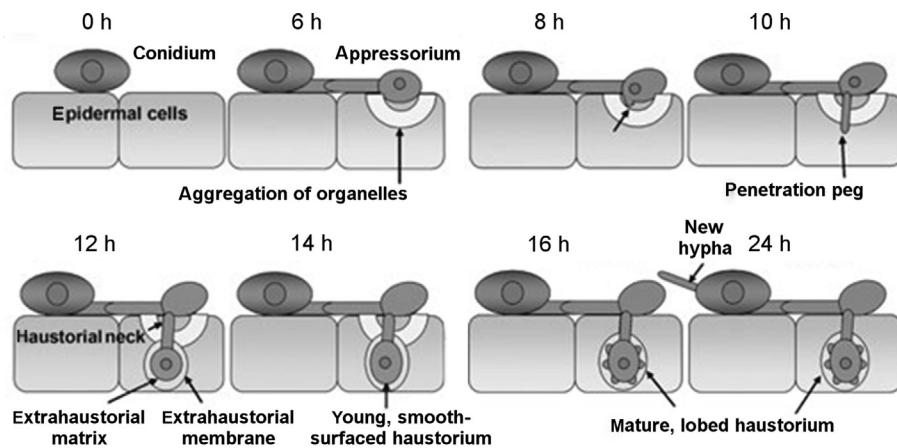
FIGURE 8.8 The disease cycle of Corn (*Zea mays*) smut, caused by *Ustilago maydis*. Source: Modified from Agrios (2005); image © Albert Brand.

The teliospores germinate to form a **promycelium**, on which a basidium and basidiospores (sporidia) form, following meiosis. The basidiospores, spread onto plants by rain splash and air currents. They bud in a yeast-like way, and are a saprotrophic phase. Under nutrient limiting conditions they form fine hyphae. If fusion with a compatible homokaryotic mycelium occurs, the dikaryotic hyphae penetrate directly into epidermal cells and spread intercellularly within plant tissues, but if successful mating does not occur the hyphae often die. Cells in the vicinity of living hyphae enlarge and divide forming galls, with the hyphae remaining intercellular for most of gall formation. In seedlings, systemic infections occasionally occur; in older plants infections are local, many of the galls remaining too small to be visible, only a few forming the characteristic large galls (Figure 8.8). The fungus invades galls prior to sporulation, uses plant cell contents and then converts the dikaryotic mycelium to teliospores. Released teliospores that land on corn meristematic tissue may cause new infections, but the majority are survival structures until the next season.

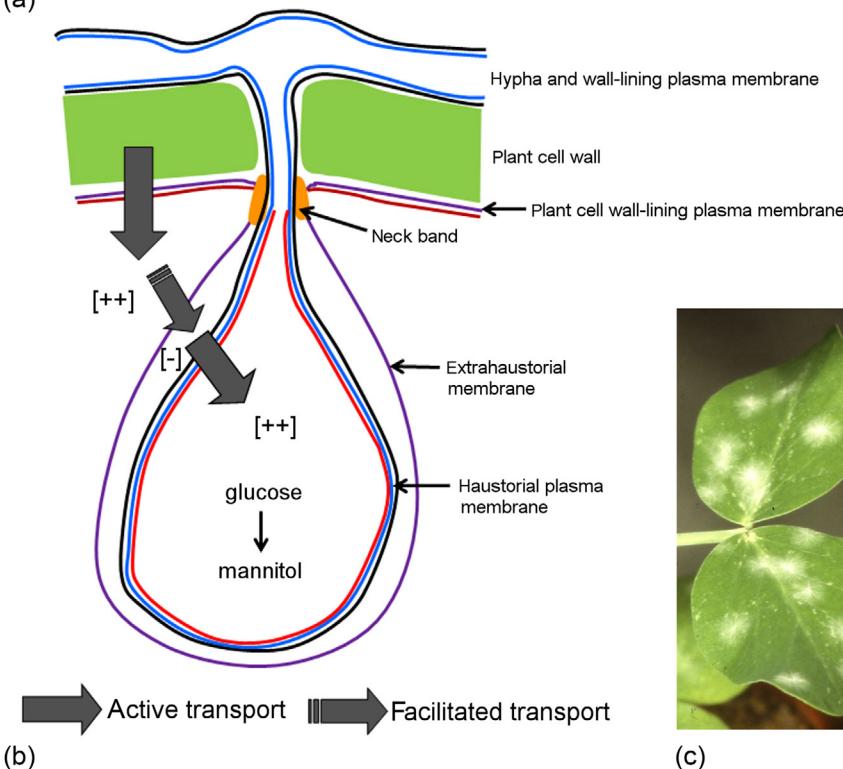
Powdery Mildews

Powdery mildews are extremely common and widespread, and economically one of the most important groups of diseases infecting many plant taxa, though not gymnosperms, the most severely infected crops including cereals and cucurbits. They are caused by many species within the family Erysiphaceae (Ascomycota) (e.g. *Blumeria graminis* on cereals and other grasses, and *Erisiphe cichoracearum* on *Arabidopsis*). They are obligate biotrophic pathogens and are mostly unculturable, though artificial growth media have now been developed for *Blumeria graminis*. Powdery mildew is most common on the upper surfaces of leaves, and to a lesser extent on lower surfaces of leaves and other organs. The mycelium grows only on the surface of the plant and does not invade tissues, nutrients being obtained via haustoria in the plant epidermal cells. Chains of round, ovoid, or rectangular conidia (p. 67) are formed on conidiophores on the surface, giving the powdery appearance. These spores are dispersed by air. Sexual **cleistothecia** (p. 20), containing one or a few asci are produced when conditions become unfavourable. Powdery mildews rarely kill their hosts but their pathogenesis causes considerable loss of productivity (as much as 20–40%) due to nutrient removal, reduced photosynthesis, increased respiration and transpiration and impaired growth.

The infection process has largely been studied in *Erysiphe pisi* on pea (*Pisum sativum*), *Blumeria graminis* on barley (*Hordeum vulgare*) and *Erysiphe cichoracearum* on *Arabidopsis*. Conidia are released, germinate and infect epidermal cells in the absence of a water film provided that air relative humidity is high. Once infection has begun, surface colonisation continues irrespective of atmospheric humidity, causing severe disease in warm, dry climates as well as cooler and more humid regions. When conidia land on a susceptible host they germinate and within 24 h produce an appressorium from which a penetration peg develops and penetrates the epidermal cuticle of the host, forming a haustorium within an epidermal cell, as described in Figure 8.9. The haustorium is separated from the host cytoplasm by an extrahaustorial membrane and gel-like amorphous matrix derived from the host – though the pathogen may contribute components. The haustorium obtains water and nutrients from the host. As soon as the haustorium is completely formed, another hypha emerges from the spore, usually on the opposite side to the germ tube, fed by nutrients from the plant. By 5 days conidiophores start to form.



(a)



(b)



(c)

FIGURE 8.9 Powdery mildew. (a) The infection processes is illustrated diagrammatically for colonisation of susceptible *Arabidopsis* by *Erysiphe cichoracearum*. Within about 6 h following inoculation, conidia germinate and form an appressorium at the tip of the germ tube. The host cell (whether susceptible or not), and even the adjacent one if the appressorium is close to it, responds by accumulation of numerous organelles and vesicles in the cytoplasm beneath the appressorium (stage 1). They are probably responsible for production of papillae–callose-rich deposits in the plant cell wall, which is a defence response. During the next 6–10 h (stage 2) a penetration peg develops beneath the appressorium and penetrates the plant epidermal cell. The haustorium then begins to develop (stage 3; 10–14 h) as a swollen, smooth-surfaced, elongated sac at the tip of the penetration peg, the nucleus migrates from the appressorium into the haustorium, and a septum separates them. By 24 h (stage 4) the lobed haustorium, with its extrahaustorial membrane and matrix, is fully formed. (b) Uptake of sugars from the host plant cell, illustrated here for powdery mildew of pea, is via the haustorium. Glucose from the host is converted to mannitol in the haustorium, and then moves out to allow further growth of mycelium. (c) Powdery mildew on pea (*Pisum sativum*). Source: (a) Modified from Koh et al. (2005); (b, c) © Peter Spencer-Phillips.

Control of powdery mildews is by application of fungicides or defence activating compounds. The use of antagonistic microbes is promising for the future.

Vascular Wilts: *Fusarium oxysporum*

Vascular wilts are widespread, extremely destructive diseases which can cause rapid wilting and death of entire plants within a few weeks, though death of perennials can take months or even years. Wilts result from the presence and activities of the specialised necrotrophic fungi in plant xylem. Four genera cause wilts: *Ceratocystis*, *Fusarium*, *Ophiostoma*, and *Verticillium* (all Ascomycota). Here we will focus on *Fusarium oxysporum* disease of tomatoes, and then Dutch elm disease (*Ophiostoma ulmi* and *Ophiostoma novo-ulmi*).

Fusarium oxysporum is common in soil and rhizosphere communities throughout the world. All strains are able to grow saprotrophically and survive in dead organic matter. Many strains can penetrate roots but not all cause disease. Those which do cause disease can result in severe losses of most vegetables, flowers and several field crops, e.g. banana (*Musa* spp.), coffee (*Coffea* spp.), plantain (*Musa paradisiaca*) and sugarcane (*Saccharum officinarum*). Pathogenic fusaria have a high level of host specificity for plant species and cultivars, with over 120 *formae speciales* and races. Most *Fusarium* wilts have similar disease cycles to *Fusarium oxysporum* f.sp. *lycopersici* on tomato (*Solanum lycopersicum*). The fungus has an asexual life cycle. It produces three types of asexual spores: **microconidia**, **macroconidia**, and thick-walled **chlamydospores** on older mycelium or within macroconidia. Hyphae from established mycelia, and the germ tube developing from spores, perceive signals from root exudates. The hyphae secrete a battery of cell wall-degrading enzymes, including pectate lyases, polygalacturonases, proteases, and xylanases, which assist entry into roots through wounds, at branching points or directly through root tips. It has multiple mechanisms for overcoming host defence. The mycelium spreads between root cortex cells until it reaches xylem vessels, which it enters through pits. As described earlier (p. 260), from there the fungus travels through the plant, mostly upwards by microconidia in the sapstream. Later spread into adjacent vessels can occur by penetrating pits. The xylem becomes clogged by mycelium and spores, and by plant-produced gels, gums and tyloses. Water transport to the leaves fails, and the plant wilts and dies. The fungus then invades all plant tissues and obtains nutrition by decomposing them. When the fungus reaches the plant surface it sporulates profusely. Control of the disease is difficult, because the fungus can survive as a saprotroph for a long time in the absence of the host. Greenhouse soil can be sterilised, but this is impracticable in the field. Use of resistant varieties is the main approach, combined with fungicides. Biological control is a future possibility, with promising results from use of antagonistic bacteria (e.g. *Burkholderia cepacia* strains) and fungi (e.g. *Gliocladium* spp., *Trichoderma* spp.) and non-pathogenic *Fusarium oxysporum* strains.

Vascular Wilts: Dutch elm Disease

Dutch elm disease (DED) is a devastating wilt disease of elm (*Ulmus*) trees. In the last century there were two extremely destructive pandemics of DED, which spread across Europe and North America ([Figure 8.10a and b](#)). The first, caused by *Ophiostoma ulmi* (Ascomycota), started in about 1910 and had died down by the 1940s after killing 10–40% of elms. The second epidemic, which appeared around the 1940s was caused by

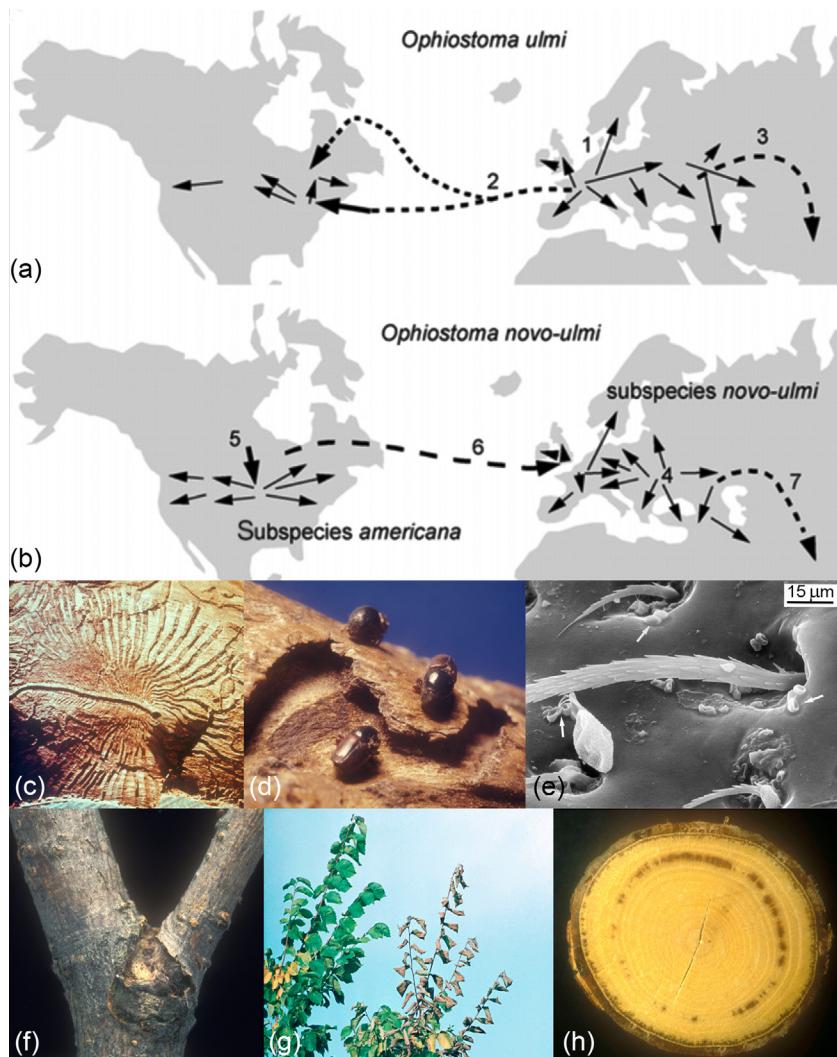


FIGURE 8.10 Dutch elm disease. (a) The first outbreak of *Ophiostoma ulmi* occurred in northwest Europe (1), from where it spread westwards to the UK and North America (2), and eastwards into central Asia (3). (b) The *Ophiostoma novo-ulmi* epidemics began simultaneously in (5) the southern Great Lakes region of North America (subspecies *americana*), from where it spread across the continent and to Europe in the 1960s (6), and in (4) Moldova-Romania (subspecies *novo-ulmi*) from where it spread westward across Europe, and eastward into the Tashkent area (7) in the 1970s. Where the distribution of subspecies *americana* and subspecies *novo-ulmi* overlaps, they are hybridising, and a new form may emerge in the future. The solid arrows indicate natural migrations from likely sites of initial introduction. The dashed arrows, show subsequent spread following additional importation events. Natural vectoring of spores is on the bodies of adult bark beetles while in the galleries that the larvae create beneath elm bark (c and d). Conidia of the pathogen are carried to new host trees directly on the exoskeleton surface of the beetles, not in a protective mycangial cavity (p. 333), seen here (arrowed) lodged in setal pits of *Scolytus scolytus* (e). Unsurprisingly, after flight, often less than half of the beetles still carry a viable spore load. The beetles feed in twig crotches (f), providing a route of entry for the fungus into the tree. The pathogen causes trees to wilt, typically with yellowing of the leaves (g). Cross sections of diseased branches have characteristic dark spots (h). Source: (a, b) From Brasier and Buck (2001); (c, d, f, g, h) © Forestry Commission Picture Library; (e) courtesy of Joan Webber.

Ophiostoma novo-ulmi, a much more aggressive pathogen. These two species evolved independently in different parts of the world. As *Ophiostoma novo-ulmi* spreads, it is replacing the *Ophiostoma ulmi*, and may well have picked up 'useful' genes from the latter during this process. *Ophiostoma novo-ulmi* exists as two subspecies – Eurasian (*novo-ulmi*) and North American (*americana*). There is another DED pathogen – *Ophiostoma himal-ulmi* – found in the Himalaya; this is in a natural balance with the elms and bark beetles in the area, and does not cause epidemics.

The disease is spread by carriage of spores on the bodies of elm bark beetles. The spores enter the xylem when the beetles feed in twig crotches (Figure 8.10f). The vessels become blocked by gums and tyloses and fungal material (Figure 8.10h), and foliage wilts, ultimately resulting in tree death. The fungus can also spread between trees via root grafts. The beetles breed in galleries under the bark (Figure 8.10c and d), where the fungus can also grow, and it is from there that the spores lodge on their bodies (Figure 8.10e). Most species of elm are affected, although the beetles do not favour European white elm (*Ulmus laevis*). Though the disease will not die out in the near future, some Wych elm (*Ulmus glabra*) in Scotland has been unaffected, and relatively resistant cultivars of other species have been bred.

Botrytis Grey Mould

Botrytis cinerea (Ascomycota) infects over 200 plant species, causing grey mould, evident on the surface as grey fluffy mycelium. Worldwide, it causes annual losses of \$10 billion to \$100 billion. It is able to counteract a broad range of plant defence chemicals. It is one of the most extensively studied necrotrophic plant pathogens.

Botrytis cinerea produces vast quantities of asexual spores which, when they land on a plant surface, germinate and form an appressorium and penetration peg that breaches the plant cuticle. Since the appressorium is not separated from the germ tube by a septum it is unlikely that sufficient turgor can be generated to effect entry by physical pressure alone. Enzymes, including cutinases and lipases, are secreted, and the tip of the penetration peg produces H₂O₂. When the cuticle has been breached, the penetration peg reaches an epidermal cell and often grows into the pectin-rich cell wall that is perpendicular to the plant surface. Plant species with low pectin content in cell walls are poor hosts for *Botrytis cinerea*, which has effective pectinolytic machinery.

Botrytis cinerea produces a wide arsenal of chemicals that cause host death, including a spectrum of low-molecular weight metabolites (e.g. botrydial, oxalic acid, and HSTs). During cuticle penetration and formation of primary lesions, *Botrytis cinerea* triggers an oxidative burst from the plant, accumulation of free radicals and hypersensitive cell death in the plant cells. While this confers resistance against biotrophic pathogens (p. 252), programmed plant cell death is beneficial to necrotrophs, including *Botrytis cinerea*, since they feed on dead cells. The fungus is also able to suppress host immunity by producing small RNA (sRNA) molecules which cause gene silencing (p. 134). As well as the aforementioned pectinases, *Botrytis cinerea* produces cellulases and hemicellulases to decompose plant cell walls to obtain nutrition.

Grey mould can be partially controlled in the field by combinations of fungicides. Biological control of grey mould on flowers and fruits using antagonistic microbes has future potential. Removal of infected plant material and reduction of humidity during storage of fruits and bulbs, and reduction of humidity in glasshouses are important control measures.

Annosum Root and Butt Rot

Root and butt rot caused by *Herobasidion annosum* (Basidiomycota) and other *Heterobasidion* species (Figure 8.11) are one of the most economically important diseases of conifers in the northern hemisphere, especially Europe, causing annual losses of over US\$1030 million. The fungus was, for a long time, thought to be a single species – *Heterobasidion annosum*. Mating experiments, however, revealed host-specialised intersterile groups, with three species in Europe, two in North America and others elsewhere, though not necessarily pathogenic (Table 8.7).

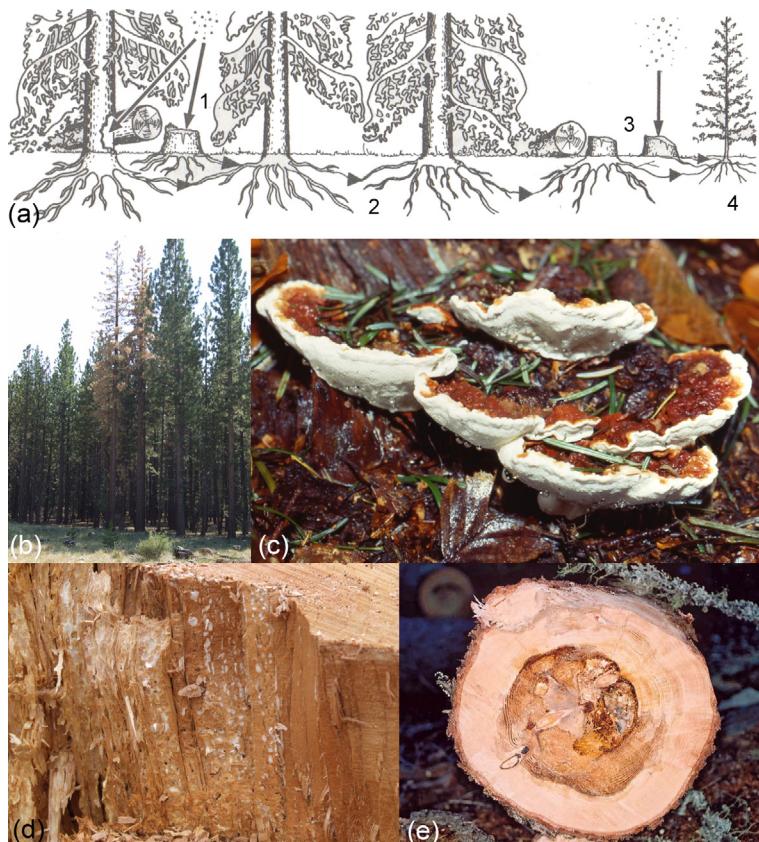


FIGURE 8.11 Annosum root and butt rot is caused by several species of *Heterobasidion* (Table 8.7). (a) Basidiospores infect freshly felled stumps at thinning and final harvest (1 and 3). Mycelium colonises the stumps and spreads into the woody roots from where it spreads as mycelium to roots of mature trees (1) and seedlings (4) by grafts and root contact. Spread from colonised trees (2) can occur in the same ways. (b) Jeffrey pine at the edge of a large and still expanding disease centre caused by *Heterobasidion irregularare* in the Sierra Nevada. (c) Fruit body of *Heterobasidion annosum*. (d) Severe white pocket rot decay in sitka spruce (*Picea sitchensis*) caused by *Heterobasidion annosum*. (e) Felled Norway spruce (*Picea abies*) showing heart rot due to *Heterobasidion annosum*. Source: (a, b) Modified from Asiegbu *et al.* (2005); (c) © Alan Outen; (b, d, e) © Stephen Woodward.

TABLE 8.7 Species of *Heterobasidion* and Their Hosts

Species	Inter-sterility group	Host	Location
<i>H. annosum</i>	P	Scots pine (<i>Pinus sylvestris</i>), but also other conifers and broadleaved trees	Europe up to 66°N
<i>H. abietinum</i>	F	Silver fir (<i>Abies alba</i>), but also other hosts	Europe, where <i>A. Alba</i> grows naturally
<i>H. parviporum</i>	S	Relatively strictly on Norway spruce (<i>Picea abies</i>)	Europe, where <i>P. abies</i> grows naturally. In China two separate populations exist
<i>H. irregulare</i>	P	<i>Pinus</i>	North America, in eastern (Quebec to Florida) and western (British Columbia to Mexico) forests, but less common in central parts
<i>H. occidentale</i>	S	<i>Abies</i> , <i>Tsuga</i> , <i>Picea</i> , <i>Pseudotsuga</i> , and <i>Sequoia</i>	Western North America, from Alaska to California
<i>H. insulare</i>			Eastern and southern Asia, from Russia and Japan in the north to Borneo and New Guinea in the south, and India and Nepal in the west
<i>H. araucariae</i>		A saprotroph on dead wood of <i>Auracaria</i> , <i>Cunninghamia</i> , and <i>Pinus</i>	Australia, New Zealand, New Guinea, and the Fiji islands

Basidiospores infect freshly felled stump surfaces or stem and root wounds, between 5 and 35 °C. Conidia are also produced but their role is unclear. Basidiospores germinate and colonise stumps including the roots. The mycelium produced can live in stumps for a long time without causing disease in a living tree. The fungus spreads from stumps to healthy trees by mycelial growth through root grafts or contacts (Figure 8.11). Only occasionally does colonisation occur through fine roots. The fungus spreads necrotrophically in the sapwood in living trees, but later grows in the heartwood of most tree species despite the presence of fungistatic compounds. It produces a wide range of wood decay enzymes and several toxins: fommanoxin, fommanosin, fommanoxin acid, oosponol, and oospoglycol. Several morphological and chemical responses are activated in the host at late stages of infection, including the production of a range of phenolic compounds which may damage fungal membranes, lignification which prevents diffusion of toxins and enzymes, suberisation and papillae formation also occurs, and volatile and non-volatile terpenes are produced, and resins are mechanical barriers. Depending on the time of year, moisture content of the sapwood, and the age and vitality of the tree, *Heterobasidion annosum* can extend at rates of up to 1 to 2 m year⁻¹ in stems and roots, respectively. It is not known how long an individual *Heterobasidion* genet (clone) can remain alive on the same site, but they are capable of occupying disease centres of 50 m diameter which would probably be over 100 years old; individuals have been found in stumps 62 years after felling, and in root systems of diseased trees for several decades.

Strategies for control of annosum root rot include use of chemicals, biological control agents, and silvicultural practices. Chemical and biological control methods involve

applications to the stumps after felling. Urea and borates are chemicals used commercially for control, and the saprotrophic *Phlebiopsis gigantea*, is available in several commercial formulations for biocontrol. Planting species with low susceptibility can reduce root rot problems, and disease in mixed stands is less than in monocultures. Delaying thinning until trees are older can reduce the problem, as can thinning at times when basidiospores are not being dispersed.

Oomycete Diseases: Potato Blight, Downy Mildews, Pythium Damping-Off, and Rot

The oomycetes, though not fungi (p. 35), operate in many similar ways, cause a range of diseases of plants (Table 8.2), and are studied by mycologists. Pathogens include many *Phytophthora* species (Peronosporales), the downy mildews (Peronosporales) and *Pythium* species (Pythiales) (Table 8.2), mentioned below. Late blight, caused by *Phytophthora infestans*, is the most devastating disease of potatoes worldwide, especially in regions which often experience cool, damp weather. *Phytophthora infestans* is a specialised necrotroph, and also causes major problems with other members of the Solonaceae (e.g. tomato). It kills stems and foliage at any time during the growing season, and can kill whole fields of plants in less than 2 weeks under optimal cool, wet conditions. Potato tubers and tomato fruits are also attacked, rotting in the field or during storage.

In the past, the pathogen over-wintered solely as mycelium within infected potato tubers, growing through aerial parts producing sporangiophores that project through stomata. The sporangia are released into the air or can be dispersed by rain. At 12–15 °C, sporangia germinate releasing three to eight motile zoospores, but above 15 °C sporangia can germinate directly to form a germ tube. Until the 1980s, only the A1 mating type was present in most areas of the world, and in the absence of a compatible mating type sexual reproduction could not occur (pp. 114–115). The compatible mating type A2 has now spread from Mexico to the rest of the world, allowing sexual reproduction resulting in the formation of resistant oospores in infected tissues both above and below ground. Oospores can over winter and survive for 3 or 4 years in soil, and recombination allows emergence of more virulent strains.

On wet leaves or stems, spores germinate and the germ tube penetrates directly through the epidermis or enters through a stoma. The hyphae grow extensively between cells and penetrate cells forming long, curled haustoria. Infected cells die, but the mycelium continues to spread into living tissue, lesions enlarge and new ones develop, foliage is killed and the tuber yield considerably reduced. The pathogen is multicyclic with many asexual generations each growing season; in optimal conditions sporangia can form within 4 days from initial infection. In wet weather, when sporangia are washed from leaves into soil, the second phase of the disease develops. Zoospores emerge and enter the tubers via wounds and lenticels. Mycelium develops mostly intercellularly, haustoria again being formed within cells.

The development of epidemics depends very much on climatic conditions. Optimal conditions for production of sporangia are close to 100% humidity with temperatures between 15 and 25 °C. At over 30 °C growth ceases, though the oomycete is not killed and it can sporulate again when conditions become favourable. The disease is controlled by sanitary measures (destroying infected material and planting with disease-free tubers), planting with resistant

varieties (though each variety is only resistant to some races of *Phytophthora infestans*), and appropriately timed application of chemical fungicides. The arrival of the A2 mating type, with consequent formation of resistant oospores, and the emergence of new pathogenic races (see Chapter 4) may considerably change man's ability to control this disease. One promising approach with tomatoes is the induction of systemic-acquired resistance (SAR) by infecting with the tobacco necrosis virus or application of DL-3-amino-butyric acid.

Downy mildews, another type of oomycete disease, are all caused by obligate biotrophic pathogens. For example, *Bremia lactucae* is the most important cause of disease of lettuce (*Lactuca*) worldwide; *Hyaloperonospora parasitica* causes downy mildew of *Arabidopsis*, and though not hugely destructive nor of economic significance, it has been used extensively as a model organism in molecular studies. *Hyaloperonospora parasitica* downy mildew of *Arabidopsis*, like other downy mildews of crucifers, typically occurs in cool (10–15 °C), moist conditions. The life-cycle is relatively simple (Figure 8.12); a conidium germinates and forms an appressorium either directly or on a short germ tube, within about 6 h after landing on a leaf. A penetration hypha forms beneath the appressorium and penetrates the leaf where two epidermal cells meet, or occasionally through a stoma. As the coenocytic penetration hypha grows between cells, haustoria are often inserted into adjacent epidermal and then mesophyll cells. In compatible interactions there is minimal macroscopic disruption to the host until sporulation, when the conidiophores protrude from stomata as a downy growth (hence the disease name).

Incompatible host/pathogen combinations can result in various resistance phenotypes. Usually there is an oxidative burst and a salicylic acid (SA) dependent hypersensitive response (HR) in the epidermal cells and a few adjacent mesophyll cells, with a shift from housekeeping to defence metabolism, and about a 10% change in the transcriptome. HR involves at least one phytoalexin – camalexin. Systemic-acquired resistance (SAR) is also induced, and is associated with increased SA and systemic accumulation of PR-proteins.

A third major type of oomycete disease is caused by *Pythium* species. They are necrotrophic pathogens, causing damping-off diseases of seedlings, and seed, root and fruit soft-rot worldwide, though the species responsible vary according to abiotic environment. *Pythium ultimum*, for example, is common in soils of cool regions, while *Pythium panieratum* and *Pythium irregularare* are common where soil temperatures are higher. Species of *Fusarium* (Ascomycota), *Rhizoctonia* (Basidiomycota), and many *Phytophthora* species (oomycete) also cause similar damping-off diseases. The diseases kill young and over-mature plants and tissues, but mature plants are rarely killed, though lesions can develop on stems (at the soil line) and roots; roots rot and plant growth and yield can be severely reduced. In infected soils, seeds can fail to germinate, and seedlings can be attacked before or after emergence; invaded tissues become water-soaked, discoloured and soon collapse, the fungus-like organism continuing to colonise the fallen seedling. Broadleaf plants and Gramineae are especially susceptible. The disease is spread in infected plant material and via soil water.

Pythium survives in soil as thick-walled, sexual oospores and asexual sporangia. At 10–18 °C, germination of both oospores and sporangia tends to be by means of zoospores, whereas above 18 °C germ tubes tend to be produced. Germination, hyphal growth and tissue penetration can be induced by plant exudates. *Pythium* penetrates the plant directly by physical force and enzymic activity. Pectinases break down pectins in the middle lamella causing cells to part and tissues to break up. Cellulases result in plant cell wall disintergration. The oomycete is unable to advance into lignified tissue.

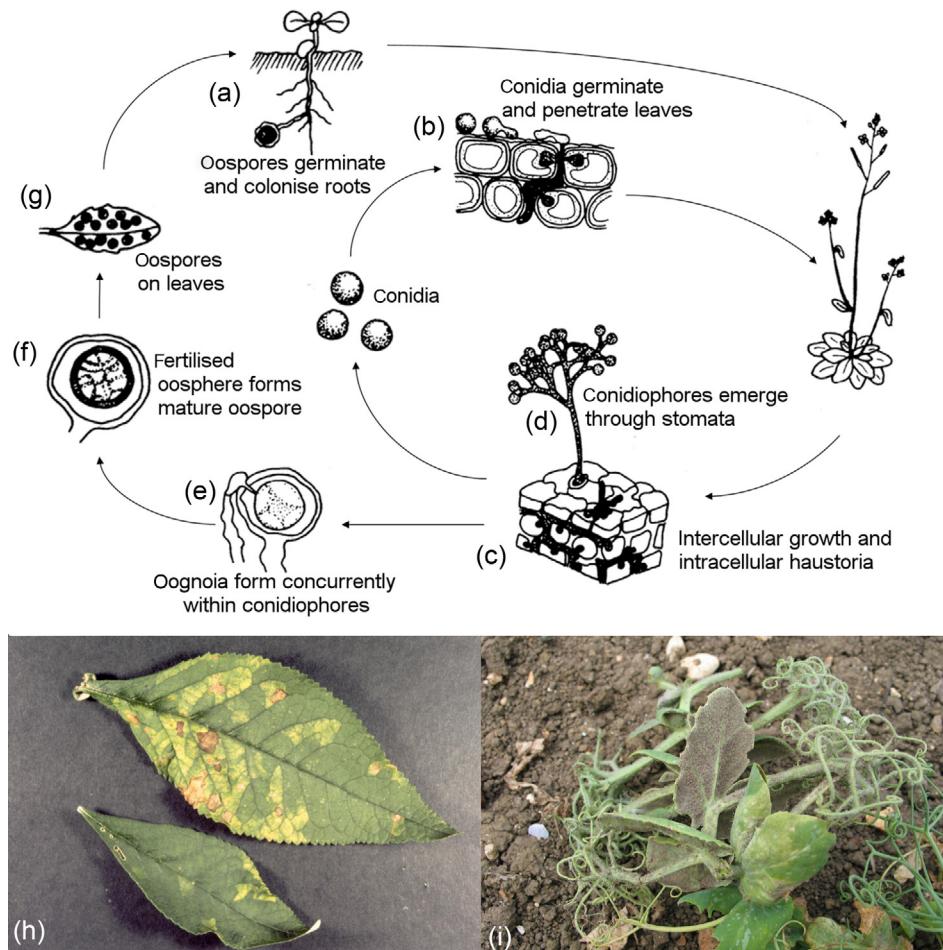


FIGURE 8.12 Downy mildews. (a–g) The life cycle of *Hyaloperonospora parasitica* causing downy mildew on *Arabidopsis thaliana*. Plants can be colonised from oospores that germinate in soil (a) and by conidia on leaves that germinate and penetrate between epidermal cells (b). (c) Coenocytic mycelium grows between cells and swells to fit spaces. Pear-shaped haustoria penetrate cells and absorb nutrients. After 1–2 weeks conidiophores, bearing asexual conidia, protrude through stomata (d) and at the same time oospores are formed (e–g). Oogonia (female sex organs) contain an oosphere which is fertilised via a fertilisation tube that grows from the male antheridium through the oosphere wall. The fertilised oosphere matures into a thick-walled oospore (f). Different components of the lifecycle are drawn at different scales (a–g). (h) *Buddleja globosa* leaves, showing characteristic downy mildew (*Peronospora hariotii*) lesions where hyphae are restricted to colonising islands delimited by larger veins. (i) Pea (*Pisum sativum*) leaves and tendrils with *Peronospora viciae* infection. Source: (a–g) Modified from: *Slusarenko and Schlaich (2003)*; (h, i) © Peter Spencer-Phillips.

Plant varieties resistant to *Pythium* are virtually unknown, and control is by good horticultural practice, including sanitation, drainage, and shallow planting; soil in glasshouses can be sterilised. Variable success has been obtained with biological control agents, using bacteria, including species of *Bacillus*, *Burkholderia*, and *Pseudomonas*, fungi including *Gliocladium*, *Trichoderma*, and non-pathogenic *Fusarium oxysporum*, and non-pathogenic *Pythium*.

DEVELOPMENT OF DISEASE IN NATURAL ECOSYSTEMS AND CROPS

Domestication of crop plants started 10,000–12,000 years BP in the Fertile Crescent in the Middle East, and is the origin of many of our currently most important crop species. The development of new agricultural practices and plant breeding resulted in significant changes to plant populations, and the simultaneous emergence of new pathogens. Genetic mechanisms behind evolution of new fungal species are reviewed in Chapter 4, and specific examples of evolutionary mechanisms resulting in emergence of new strains/species of plant pathogenic fungi are presented in the following section. In current, modern agro-ecosystems, populations of fungal pathogens are frequently challenged by new plant genes for resistance, fungicides, and a range of management practices aimed at reducing infection of crop plants. Genotypes that allow fungi to overcome these will increase rapidly and spread through the population. Spread from one plant to another is often rapid as plants are grown at very high density.

Man's modern cultivation practices leave us wide open to crop loss on a massive scale. Most crops have limited genetic diversity and are grown in vast monocultures. Since the nineteenth century plants have been bred for desired characteristics such as large yield and resistance to pathogens. Germplasm is shared worldwide, so several resistance genes are now used globally. Just a single gene mutation may cause a pathogen to become virulent, and spread worldwide is then just a matter of time. In the tropics, for example, banana (*Musa* spp.) and coffee (*Coffea* spp.) are planted as single clones and are, respectively, prone to black Sigatoka disease (Figure 8.4 inset) caused by *Mycosphaerella fijiensis*, and leaf rust. When a pathogen infects a crop with little or no genetic diversity, spread throughout the crop is often devastating. Monocultures inevitably lead to high disease incidence because of high rates of host-pathogen encounter, and disease is positively density dependent. Competition can stress plants and reduce their resistance to infection.

Though the majority of research effort has focussed on crops of economic importance, pathogens also affect individual plants in nature. However, the spatial and temporal scales at which plant-pathogen interactions occur is very different, because of the spatially and genetically heterogeneous nature of natural populations. Fungal pathogens cause death and reduced fitness of individual plants (Table 8.8), which can result in declines of populations of some host species and shifts in plant community composition. These effects can help maintain plant species diversity and genetic diversity, and affect plant community succession. Plant population dynamics are affected directly as a result of pathogen effects on survival, growth, and fecundity. Plants killed by pathogens before reproduction occurs do not contribute to the next generation, and since disease affects growth, this influences fecundity, and decreases contribution to the next generation. Competition between plants can be affected by differential susceptibility to pathogens. For example, infection of groundsel (*Senecio vulgaris*) with the rust *Puccinia lagenophorae* reduces its growth and competitiveness against lettuce (*Lactuca sativa*) and petty spurge (*Euphorbia peplus*). Succession of plant communities on sand dunes in Europe is influenced by pathogens; Marram grass (*Ammophila arenaria*), which dominates wind-blown coastal foredunes, is debilitated by pathogenic soil fungi and nematodes, and is replaced by the resistant fescue grass (*Festuca rubra*), which dominates

TABLE 8.8 Examples of Direct Effects of Fungal Diseases on Individual Plants in Nature

Plant stage	Effects on		
	Survival	Growth	Fecundity
Seed decay	Rates of disease related death are high, e.g. in the tropics ranging from 10% in the invasive <i>Mimosa pigra</i> in Australia to 47% in pioneer trees in Panama. In Wyoming shrub-steppe, ranging from 0–90% mortality of 5 plant species	NA	NA
Seedling diseases	Rates of death due to damping-off disease are high. In Barro Colarado Island, Panama, it was the primary cause of death of 80% of plant species tested killing 74% of a parent tree's seedlings	?	NA
Foliar diseases	Plants are sometimes killed, especially if seedlings	Foliar diseases reduce leaf area, hence photosynthetic activity and growth. Small plants are competitively disadvantaged. In Mexican tropical rain forest, mean leaf area damage was <1% and always <20%. However, in Costa Rica, growth of the tree <i>Erythrociton gymnanthus</i> , infected by the petiole pathogen <i>Phylloporia chrysita</i> , was reduced by 52%	Reproduction can be reduced because of reduced growth
Systemic infections	Some fungi and oomycetes can have major effects. The systemic smut <i>Urocystis trientalis</i> caused 50% mortality of <i>Trientalis europaea</i> (Primulaceae). The oomycetes <i>Albugo candida</i> and <i>Peronospora parasitica</i> caused death of up to 88% of shepherd's purse, <i>Capsella bursa-pastoris</i> , seedlings	?	?
Cankers, wilts, and dieback	There have been several widespread, dramatic epidemics resulting in rapid death of trees when cankers girdle stems or block vascular transport causing wilts. These include: <i>Ophiostoma ulmi</i> and <i>O. novo-ulmi</i> on elms (p. 274); chestnut blight caused by <i>Cryphonectria parasitica</i> ; sudden oak death caused by the oomycete <i>Phytophthora ramorum</i>	With some canker diseases, if cankers remain small and localised, then death may not ensue, but growth will be impaired	?

(Continued)

TABLE 8.8 Examples of Direct Effects of Fungal Diseases on Individual Plants in Nature cont'd

Plant stage	Effects on		
	Survival	Growth	Fecundity
Root diseases and butt rots	In native North American conifer forests, the basidiomycetes <i>Heterobasidion annosum</i> (pp. 276–277) and <i>Phellinus weiri</i> cause high mortality. Death of large dominant trees alters forest structure	Root rots do not always cause death of whole trees, but growth and reproduction can be dramatically reduced. The basidiomycete <i>Armillaria ostoyae</i> reduced Douglas fir trunk radial increase by up to 60%. <i>H. annosum</i> reduced <i>Pinus taeda</i> trunk radial increase by 36%	?
Floral infections	NA	NA	Attack of flowers and developing fruits can considerably reduce fecundity. <i>Exobasidium vaccinii</i> caused a 50% reduction in flowers of <i>Rhododendron calendulaceum</i> in the Appalachian Mountains in the eastern United States. Anther smut of <i>Silene</i> spp., vectored by pollinators, and caused by <i>Microbotryum violaceum</i> , replaces stamens and staminoids with spore-bearing structures, and hence has a major effect on plant reproductive capacity

NA – not applicable; ? – effects are likely but examples are not available.

Information from [Gilbert \(2002\)](#).

stabilised dunes. The root rotter of trees, *Phellinus weiri*, drives forest structure and succession in conifer forests of the Western USA; *Phellinus weiri* removes overstory trees of the extremely susceptible Douglas fir (*Pseudotsuga menziesii*) and mountain hemlock (*Tsuga mertensiana*), resistant plants taking their place.

DISEASES OF OTHER AUTOTROPHS: LICHENS AND SEAWEEDS

Fungi are not only pathogenic to plants, but also to other photoautotrophs, including algae and lichens (Chapter 7). Some fungi – termed lichenicolous fungi – live exclusively on lichens. Though some are saprotrophs, most are either host-specific or broad-spectrum pathogens.

There are over 1800 described species currently, 95% of which are Ascomycota (in 19 orders) and 5% Basidiomycota (in 8 orders). *Athelia arachnoidea* is an extremely common, widespread perennial, destructive lichenicolous basidiomycete of numerous lichen-forming Ascomycota and their photobionts. Little is known about the modes of pathogenesis/parasitism, virulence or nutrient exchange. There are also lichenicolous lichens that colonise another lichen host. For example, *Fulglesia bracteata* colonises the lobulate *Toninia caeruleonigricans*. The infection process begins with ascospores of the *Fulglesia bracteata* fungus; small scales form and eventually its yellow thallus covers the grey thallus of *Toninia caeruleonigricans*.

Sometimes, the lichenicolous fungus or lichen acquires photobionts from the host and becomes a lichen with that photobiont species. For example, the mycobiont that eventually forms the lichen *Diploschistes muscorum*, is initially lichenicolous, parasitising squamules of *Cladonia* species. Eventually, it acquires photobiont cells from the host and forms the independent lichen *Diploschistes muscorum*.

Aquatic autotrophs are also affected by fungus and fungus-like pathogens. There are three main categories of association with algae: (1) biotrophic with few macroscopic symptoms; (2) biotrophic with severe disease symptoms in which host organelles are destroyed and the entire cell is occupied by the fungus; and (3) necrotrophic on a partially senescent host with further tissue destruction. Examples include chytrids on green algae (Chlorophyta) (e.g. *Olpidium* spp. on *Cladophora*). The chloroplasts turn brown and surround the sporangium. *Lindra* spp. (Ascomycota) grow on air vesicles of *Sargassum*, turning them into soft, dark brown, wrinkled structures giving the name 'raisin disease'. *Phycomelaina laminariae* is common on brown algae, causing 'stipe blotch' of *Laminaria*. The pathogen forms black oblong or circular patches on the host's stipe, but although infected areas are severely damaged the host is not killed.

EMERGING DISEASES AND THE BIOSECURITY THREAT

As plant communities evolve in different parts of the world, pathogenic fungi evolve in association with them locally. They often cause little damage in the regions where they co-evolved, being in natural balance. However, when they arrive in different parts of the world where native plants have little resistance and/or their natural enemies are absent and unable to control them, damaging disease episodes can occur. Many new diseases are emerging (e.g. diseases caused by *Puccinia graminis* f.sp. *tritici*, *Magnaporthe oryzae* and *Phytophthora* species, [Figure 8.13](#)). Man has been responsible for many of these emerging infectious diseases (EIDs) by moving plant material, or soil harbouring the fungus, around the world ([Table 8.9](#)). Potato late blight (pp. 277–279), for example, emerged when *Phytophthora infestans*, that co-evolved with wild potato (*Solanum tuberosum*) in the Andes, was transported to Mexico, and then to Europe in the mid-nineteenth century. Movement by man or natural agents is, however, not the only cause of EIDs. New infectious diseases emerge when pathogens have:

- (1) increased in incidence, host range or geographical range;
- (2) altered pathogenesis;
- (3) newly evolved;
- (4) been newly discovered/recognised.

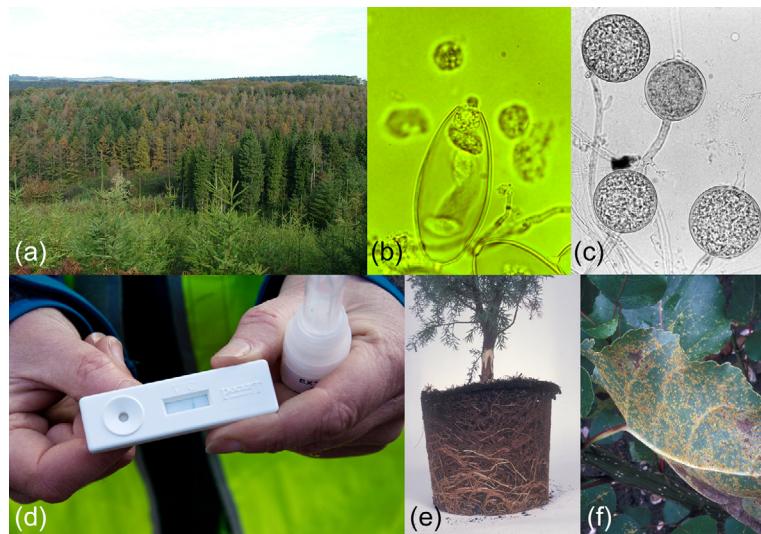


FIGURE 8.13 Emerging diseases. Many new *Phytophthora* (oomycete) species are emerging to cause disease. *Phytophthora ramorum* kills oak (sudden oak death in California, USA) and infects many other tree species, significantly rhododendron, causing non-fatal foliar dieback, which can be a source of infection for other plant species. Spores are spread by rainwater and wind. *Phytophthora ramorum* is also now killing larch (*Larix*) over large areas in the UK. (a) *Phytophthora ramorum* affecting larch in south west England. (b) *Phytophthora ramorum* chlamydospores on a leaf. (c) *Phytophthora ramorum* chlamydospores on a leaf. (d) Test kit for *Phytophthora ramorum*. (e) Root and stem base killing of container grown *Juniperus communis suecica* by *Phytophthora* sp. (f) *Populus* rust caused by *Melampsora* sp. Uredosori and orange spore masses can be seen on the underside of a leaf. Source: All images © Forestry Commission Picture Library.

Some crop diseases might now be considered persistent, having caused man problems for hundreds or even thousands of years, but new races of these emerge which may cause major epidemics (e.g. *Puccinia graminis* f.sp. *tritici*; Table 8.10). The large amount of repetitive DNA and the high frequency of single-nucleotide polymorphisms (SNPs) in the genomes of some major crop pathogens, such as *Puccinia graminis* and *Phytophthora infestans* (Table 8.10), indicate that more virulent strains and resistance to fungicides are likely to evolve. A recent study of a few major ascomycete plant pathogens revealed that evolution of pathogenicity in this group is not only ancient but is both rapid and on-going. Fungal pathogens have undergone and continue to undergo extensive 'genomic tillage' where chromosome duplications, genomic rearrangements, horizontal gene acquisitions (pp. 134, 201), and large scale gene evolution caused by proliferation of transposons (pp. 132–134) and repeat-induced point mutations (p. 134), drive the evolution and expression of 'effectors' (see Chapter 4 for a discussion of these genetic mechanisms). In the dynamic 'evolutionary arms race' that exists between plant and pathogen, these mechanisms allow the pathogen to evolve novel effectors which exploit plant metabolism and evade host defence mechanisms allowing pathogens to take the upper hand and successfully infect the host plant.

Rapid evolution of new pathogens and adaptation to new environments and new hosts can arise as a result of hybridisation between resident pathogens, horizontal gene transfer (as seen with HSTs of *Stagnospora nodorum*) and host jumps or shifts (as seen in *Magnaporthe*

TABLE 8.9 Examples of Emerging Infectious Diseases (EIDs) of Plants, Including Staple Crops, Other Crops, and Plants in Natural Ecosystems

Pathogen	Disease	Hosts	Region and host origin	Time and place of emergence	Factors driving emergence
<i>EIDs of world staples</i>					
<i>Phytophthora infestans</i> (oomycete)	Late blight (pp. 277–279)	Potato (<i>Solanum tuberosum</i>)	Mexico; wild <i>Solanum</i> spp.	Mid-nineteenth century in Europe; 1990s in North America	Repeated introductions by man into non-native regions
<i>EID of other crops</i>					
<i>Puccinia kuehnii</i>	Sugarcane orange rust	Sugarcane (<i>Saccharum officinarum</i>)	Australia; sugarcane	Australia	Evolution of a new strain that broke resistance
<i>EIDs in natural ecosystems</i>					
<i>Ophiostoma ulmi</i> and <i>Ophiostoma novo-ulmi</i> (p. 274)	Dutch elm disease	Elm (<i>Ulmus</i> spp.)	Elm; various places worldwide	Twentieth century; repeated pandemics in Europe, Asia, and North America	Introductions in imported timber. Hybridisation resulting in increased virulence
<i>Cryphonectria parasitica</i>	Chestnut blight	Chestnut (<i>Castanea dentata</i> , <i>C. sativa</i>)	Japanese chestnut <i>Castanea crenata</i> ; Southeast Asia	Early twentieth century; Eastern USA	Imported chestnut plants
<i>Phytophthora ramorum</i> (oomycete)	Sudden oak death	Many woody plant species	California bay laurel- Oregon myrtle (<i>Umbellularia californica</i>) and <i>Rhododendron</i> spp.	1990s; Europe	Imported nursery stock
<i>Phytophthora cinnamomi</i> (oomycete)	Jarrah dieback/ Cinnamomi root disease	>3000 trees, woody ornamentals and herbs	Wide range of plants; Southwest Pacific area	1800s–1900s; Europe, North America, Australia	Imported plants

Extracted from [Anderson et al. \(2004\)](#) and [Brasier \(2008\)](#).

oryzae), often associated with newly introduced exotic pathogens (Table 8.11). *Ophiostoma novo-ulmi* (p. 274) has acquired ‘useful’ major genes by hybridising with *Ophiostoma ulmi*, the resident species, as it has spread across Europe and North America. *Phytophthora alni* subsp. *alni* (oomycete) has recently emerged by hybridisation, and is killing alder (*Alnus*) across Europe. Other examples include *Botrytis allii* an onion pathogen, *Melampsora columbiana* the

TABLE 8.10 Characteristics of Three Persistent Diseases of World Staple Crops, with Emerging Races

	Disease	Host range	Epidemics since	Growth/ survival temperature (°C)	Asexual and sexual life-cycle	Spore forms	Clonal	Genome size	Gene number	Repetitive DNA	SNP ^a frequency
<i>Magnaporthe oryzae</i> (Ascomycota)	Rice blast	50 grass species	Seventeenth century	18–24 4–35	Yes	2	Yes	41.7 Mb	12 841	9.7%	1 per 2.3 Kb
<i>Puccinia graminis</i> (Basidiomycota)	Wheat stem rust	365 cereal grass species; barberry, <i>Mahonia</i> spp.	690 BC	15–24 –10 to 35	Yes	5	Yes	88.6 Mb	20 567	80%?	>1 per Kb
<i>Phytophthora infestans</i> (oomycete)	Late blight of potato	Solanaceae	1845	8–20 –5 to 28	Yes	3	Yes	228.5 Mb	18 179	74%	1 per 426 bp

^aSNP, single nucleotide polymorphism.

Compiled from Gurr et al. (2011).

poplar rust, and *Verticillium longisporum* a pathogen of crucifers. Not only does man's propensity for moving plant and soil material about bring pathogens into contact with new hosts and other closely-related pathogens with potential for hybridisation, but also plant nurseries in Europe and elsewhere are infested with many species in the same genus (e.g. *Phytophthora*), and are potential breeding grounds for more aggressive hybrid pathogens.

New pathogens can emerge by adaptation to a new, closely-related plant (e.g. wild to domesticated barley (*Hordeum vulgare*); host shift) or to one that is genetically distant (e.g. different genus; host jump) (Table 8.11). Common scenarios are from: (1) wild hosts (e.g. weeds close by); (2) planting new crops in natural ecosystems (e.g. introduction of soybean, *Glycine max*) into cleared areas of Amazonian rainforest); and (3) by transporting infected plant material into new areas with naive populations of related species with a lower level of resistance

TABLE 8.11 Examples of Evolutionary Mechanisms Resulting in Emergence of Plant Pathogens in Agro-ecosystems

Evolutionary mechanism	Plant pathogen	Time scale
<i>Domestic/host-tracking</i>		
Co-evolution of a pathogen with its host	<i>Mycosphaerella graminicola</i> on wheat	10–12,000 BP
	<i>Magnaporthe oryzae</i> on rice	7000 BP
	<i>Phytophthora infestans</i> on potato	7000 BP
	<i>Ustilago maydis</i> on maize	8000 BP
<i>Host jump/host shift</i>		
Adaptation allows a pathogen to shift to a new closely-related host or jump to a more distantly related host species	<i>Magnaporthe oryzae</i> from Setaria millet to rice	Abrupt evolutionary change, ~7000 BP
	<i>Rhynchosporium secalis</i> from wild grasses to barley and rye	Abrupt evolutionary change, ~2000 BP
	<i>Phytophthora infestans</i> from wild <i>Solanum</i> species to potato	Abrupt evolutionary change, <500 BP
<i>Horizontal gene transfer</i>		
HGT: Part of a genome is transferred to another organism (species or vegetatively incompatible individual), (e.g. by a transposon)	<i>ToxA</i> from <i>Phaeospaeria nodorum</i> into <i>Pyrenophora tritici-repentis</i>	Abrupt evolutionary change, ~60 BP
	PEP cluster in <i>Nectria haematococca</i>	Unknown
	Host-specific toxins in <i>Alternaria alternate</i>	Unknown
<i>Hybridisation</i>		
Mating between two closely-related species	Hybrid of <i>Phytophthora cambivora</i> and relative of <i>Phytophthora fragariae</i> on alder (<i>Alnus</i>)	Abrupt evolutionary change within the last century
	Hybrids of <i>Ophiostoma ulmi</i> and <i>Ophiostoma novo-ulmi</i> on elm (<i>Ulmus</i>) trees	Abrupt evolutionary change within the last century

From [Stukkenbroek and McDonald \(2008\)](#).

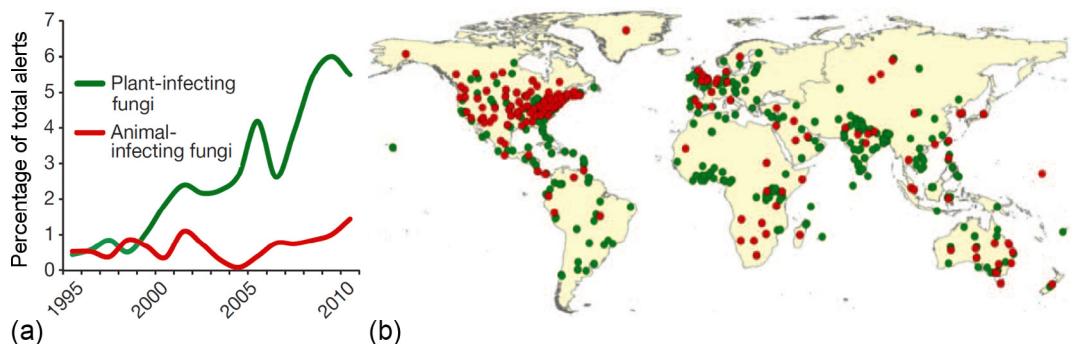


FIGURE 8.14 Increase in emerging infectious diseases (EIDs). New plant pathogenic diseases are increasing as revealed by the alerts in the ProMED (Program for Monitoring Emerging Diseases; <http://www.promedmail.org>) database. Alerts rose from 0.4% in 1995 to 6% in 2010. (a) Disease alerts and (b) the location of the reports. *Source:* Modified from Fisher et al. (2012).

(e.g. chestnut blight moving from imported Asian chestnuts, *Castanea crenata* to North American chestnuts, *Castanea dentata*). Over millions of years jumps can even occur between kingdoms. For example, *Claviceps purpurea* (Clavicipitaceae) is a pathogen causing ergot of rye (*Secale cereale*), but the common ancestor of the Clavicipitaceae was an animal pathogen.

EIDs are increasing (Figure 8.14). The huge impact that EIDs can have on the natural environment, and on crop losses, is illustrated by the death of 100 million elm (*Ulmus*) trees in the UK alone, due to Dutch elm disease, and 3.5 billion sweet chestnut (*Castanea sativa*) trees killed by chestnut blight in the United States. The cost in terms of human life has already been mentioned at the start of this chapter, with regard to the Irish potato famine caused by *Phytophthora infestans*. A worst case food loss scenario would be if the five main staple food crops (rice (*Oryza* spp.), wheat (*Triticum* spp.), maize (*Zea mays*), potatoes (*Solanum tuberosum*), and soya (*Glycine max*)) simultaneously succumbed to severe epidemics (of *Magnaporthe oryzae*, *Puccinia graminis*, *Ustilago maydis*, *Phytophthora infestans* and *Phakospora pachyrhizi*, respectively) leaving sufficient food for less than 40% of the world's population, though this is an unlikely scenario. The impact of EIDs goes beyond plant death: 270 megatonnes of CO₂ is predicted to be released, during 2000–2020, due to loss of western Canadian pine (*Pinus* spp.) trees due to the pathogenic association of the mountain pine beetle (*Dendroctonus ponderosae*) with blue stain fungi (*Grosmannia clavigera*).

Further Reading

General

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