

I Introduction

The economic impact of plant diseases probably first became apparent when plants were grown together as crops. The majority of plant pathogens have a limited host range and many are species-specific. A mixed flora, where hosts of plant diseases are intermingled with other species, acts as a buffering system with respect to pathogens. The chances of pathogens finding fresh hosts in a species-rich habitat are much lower than in a crop consisting of a single plant species. Pathogens evolved over millions of years in species-rich habitats, and to succeed in finding new hosts they developed highly efficient mechanisms of spread.

With the aggregation of plants together as crops, almost ideal conditions were created for the establishment and spread of pathogens. The continuous processes of selection and propagation of crop species have resulted in the situation today where the planting of large areas of land with crops of near-identical genotype is common. It is therefore not surprising that microorganisms that have an efficient dispersal mechanism, and that can infect crops, have on occasion caused devastating losses in yield.

The historical importance of plant diseases and their economic and social effects have been well documented. Currently, within the agricultural systems of developed countries, plant diseases are unlikely to cause crop losses to the extent of those associated with, for example, late blight of potato (Plate 1) in Eire in the 1840s and in Germany in 1917–18. The development and use of crop protection chemicals from the late nineteenth century onwards, and the establishment of scientifically based plant breeding programmes in the early twentieth century, have led to reductions in losses due to disease. Despite increases in the yield and quality of crops over the past

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century, it is estimated that about 20–30% of potential crop yield is lost annually through attack by plant pathogens and pests, and competition from weeds. Losses to this extent or even greater may occur in less developed countries, but loss of potential yield due to attack by pathogens may be much less in crops of countries within the European Union. The EU is now self sufficient in many foodstuffs, and yields in excess of national and EU requirements occur with some crops, notably cereals, for which a considerable export trade has developed in some countries such as the UK.

Improvements in the quality of crops and crop produce have also taken place. The quality of horticultural crops has always been important. The production of sound blemish-free fruit and vegetables has required consistent control of disease in the field and in store. The appearance of such produce is frequently of primary importance to supermarkets and other retail outlets. Quality is also important with arable crops such as cereals and is associated with the potential of wheat and barley cultivars for, respectively, bread making and malting. For example, in the United Kingdom, wheat suitable for bread making has often attracted premiums of 10% above the price for feed wheat, and barley for malting 20% or even higher premiums above the price obtained for animal feed. Good control of disease on wheats of bread-making potential and malting barleys is clearly desirable since attack by pathogens may reduce grain quality to the extent that it may only be suitable for feed purposes.

This text is primarily concerned with disease control measures currently employed by farmers and growers in the EU, and more particularly within the UK. Nevertheless, most of the principles considered are relevant to the control of crop diseases in other parts of the world. Particular consideration is given to those technologies of disease control and their attendant problems that have developed within the intensive agricultural and horticultural systems of the EU.

2 Major crop diseases in the UK

2.1 INTRODUCTION

Surveys of disease incidence and severity have been carried out for important crops in many parts of the world. For example, the comprehensive surveys undertaken under the auspices of the Home Grown Cereals Authority (HGCA) record the incidence of diseases in the principal arable crops in the UK. However, it has often proved difficult to determine accurately in strict monetary terms losses due to attack by individual pathogens. Disease levels often vary greatly from season to season. Crop losses may result from attacks by more than one pathogen. Furthermore, crop growth is affected by other factors such as climate, soil type, and environmental and cultural conditions, and these factors frequently interact.

Consideration is given in this chapter to the aetiology and current importance of diseases of the principal arable crops and selected diseases of top fruit and protected crops grown in the UK. Features of the life cycle of pathogens that are particularly relevant to the development of control measures are emphasised.

2.2 CEREAL DISEASES

Cereals are by far the most extensively sown of the arable crops in the UK, with about 2 million hectares (ha) of wheat and 970,000 ha of barley sown in 2011. Nearly all wheat and about half of the barley crop is now sown in autumn. Continuous cultivation of cereals is common practice in some areas of the UK, with perhaps a break into oilseed rape, and in some areas sugar beet, potatoes and pulse crops, every three to four years. Autumn sowing and monoculture cropping systems favour the development of cereal pathogens that

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Table 2.1 *Principal diseases of cereals in the UK.*

Causal agent	Disease	Symptoms
Foliar pathogens		
<i>Mycosphaerella graminicola</i>	Septoria leaf spot W	Elongated yellow/brown lesions containing pinhead-size black spore cases (pycnidia)
<i>Septoria nodorum</i> *	Glume blotch W	Irregular brown lesions on leaves and particularly glumes
<i>Blumeria graminis</i>	Powdery mildew W,B	Buff-coloured raised pustules
<i>Rhynchosporium secalis</i>	Leaf spot/scald B	Grey lesions with dark brown margins
<i>Pyrenophora teres</i> *	Net blotch B	Net-like lesions and irregular dark/black blotches
<i>Ramularia collo-cygni</i> *	Leaf spot B	Small brown rectangular lesions surrounded by a yellow halo.
<i>Puccinia striiformis</i>	Yellow rust W	Yellow/orange pustules, often in stripes
<i>Puccinia hordei</i>	Brown rust B	Brown pustules with pale haloes
Barley yellow dwarf virus	W,B	Stunting and yellow leaves
Stem base diseases and root rots		
<i>Oculimacula yallundae</i>	Eyespot W	Dark brown, often eye-shaped lesions below the first node
<i>Gaeumannomyces graminis</i>	Take-all W	Blackened stem base and root system: poorly filled ears

Table 2.1 (cont.)

Causal agent	Disease	Symptoms
<i>Microdochium nivale</i> * and <i>Fusarium culmorum</i> *	Brown foot rot W,B	Brown lesions with blackened nodes
Ear diseases		
<i>Ustilago nuda</i>	Loose smut W	Grain replaced by a mass of black spores
<i>Tilletia caries</i>	Covered smut: bunt W	Seed coat containing a mass of black spores

W indicates that a pathogen primarily attacks wheat, B that it primarily attacks barley and W,B that it attacks both.

* These pathogens are also seed-borne.

may spread from crop debris to emerging crops. The major diseases affecting cereals in the UK are outlined in Table 2.1.

2.2.1 Foliar pathogens

The move to autumn sowing of cereals and adoption of monoculture practices led to an increase in septoria leaf spot diseases during the latter part of the twentieth century, and they are now recognised as the most important foliar pathogens of wheat in the UK and many parts of Europe. Infection of susceptible cultivars may result in a halving of crop yield. Septoria diseases of wheat survive between crops on stubble and straw debris, and readily spread to merging seedlings of wheat. Infection may also result from spread of sexual spores – ascospores – produced on crop debris. The disease has a long latent period and can survive in young wheat plants during the winter months. *Mycosphaerella graminicola* (formerly *Septoria tritici*) is the most damaging species and is first apparent as olive-brown lesions (Fig. 2.1) that often coalesce to form extensive senescent areas on

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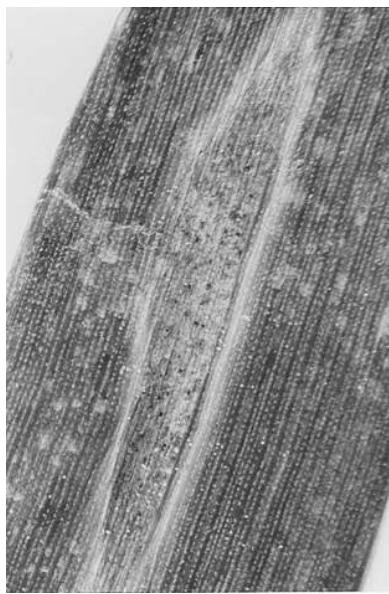


FIGURE 2.1 Lesion of septoria disease of cereals.

leaves (Plate 2). *Septoria nodorum* causes similar foliar symptoms but also attacks the glumes which surround the developing grain, and in fact this pathogen is seed-borne. During the growing season of wheat, both fungi are dispersed by conidia produced in black, pinhead-like pycnidia. The pycnidia are larger and more prominent with *M. graminicola* (Plate 2). In wet conditions conidia ooze from the pycnidia in spore tendrils (Fig. 2.2) and are dispersed by rain splash. The diseases are thus prevalent in wetter areas and in wet seasons.

Another frequent and often severe disease of cereals is powdery mildew caused by *Blumeria* (formerly *Erysiphe*) *graminis*. Cereal powdery mildew fungi are biotrophic pathogens and as such are closely adapted to their host plants. Biotrophs obtain their nutrients solely from living host tissue, and most cannot be grown in artificial culture. Biotrophic pathogens generally have a restricted host range and many exhibit the phenomenon of physiological specialisation, where morphologically similar isolates have different host ranges. For example, isolates of *B. graminis* from barley are specific to this cereal, and these isolates are assigned to *Blumeria graminis* f.sp.

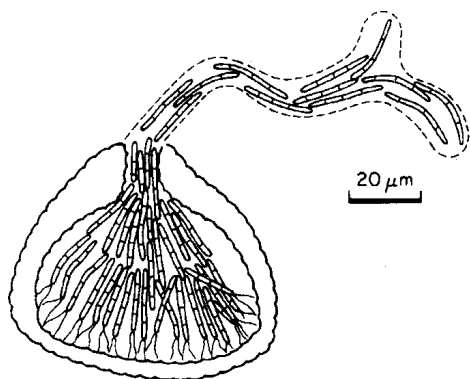


FIGURE 2.2 Section through a pycnidium of *Mycosphaerella graminicola* showing a spore tendril.

(forma specialis – literally ‘special form’) *hordei*. Similarly, isolates of *B. graminis* that attack wheat are denoted as *Blumeria graminis* f.sp. *tritici*. This pathogen exhibits further physiological specialisation in that some isolates have become adapted to individual cultivars of wheat or barley, and these are referred to as races.

Powdery mildews are efficiently dispersed by wind-borne asexual conidia which arise from mycelia on the surface of the host (Fig. 2.3). Conidia are produced in vast numbers in warm, dry weather, and the pathogen can spread very rapidly. Cereal powdery mildews survive between crops mainly on volunteer plants emerging from seed left in fields after harvest, but also in the sexual stage as ascospores within a protective spore case, the cleistothecium (Plate 3). Ascospore release from asci within cleistothecia frequently occurs at the time of emergence of autumn-sown crops.

Puccinia striiformis causes yellow rust of wheat (Plate 4). Two other species of *Puccinia*, *P. triticina* and *P. hordei* are the causal agents of brown rust in cereals, the former on wheat and the latter on barley. *Puccinia striiformis*, like *Blumeria graminis*, exhibits physiological specialisation with a number of races. The pathogen rarely causes severe crop losses, but on occasions severe epidemics have arisen on susceptible cultivars. For example, in the UK, a virulent race of yellow rust became prominent in 1988 and 1989 on the wheat cultivar Sleipner (Plate 5). The pathogen is dispersed by wind-blown

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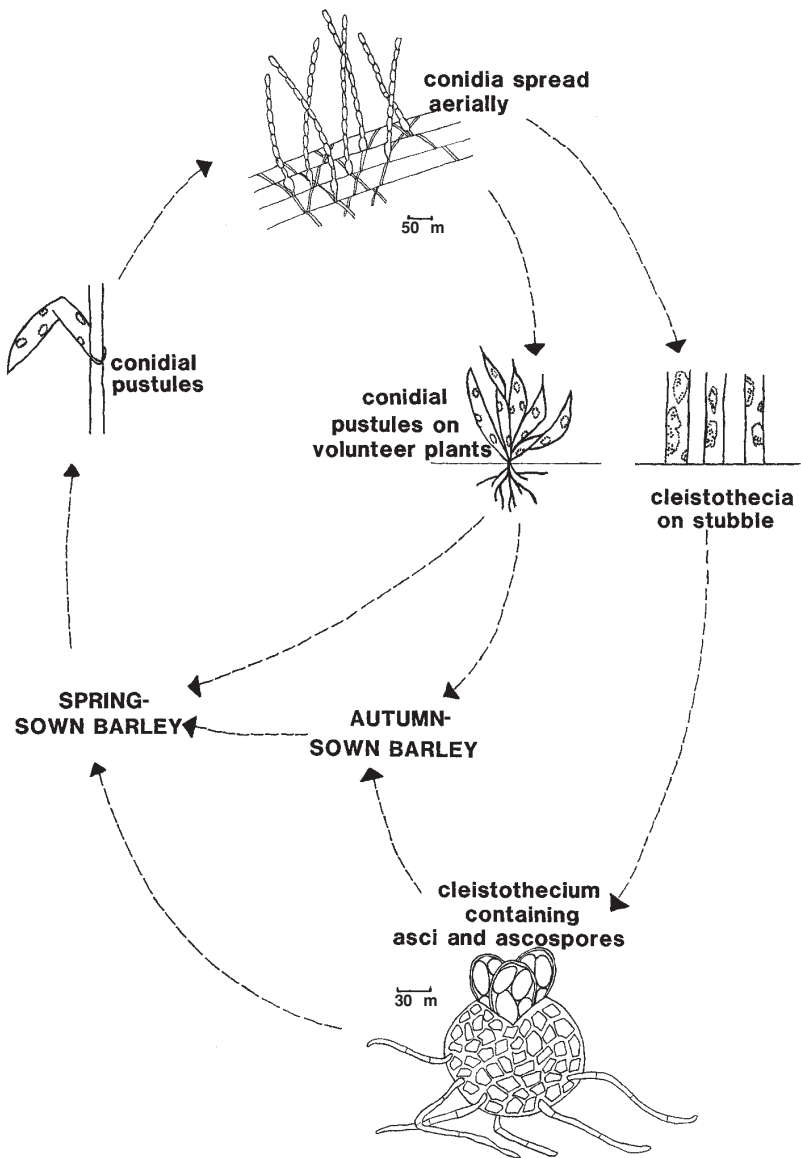


FIGURE 2.3 The life cycle of *Blumeria graminis* f.sp. *hordei* (powdery mildew of barley).



FIGURE 2.4 Symptoms of scald (*Rhynchosporium secalis*) on barley.

urediospores produced in copious quantities from orange-yellow uredial pustules. Crops which are badly affected by *P. striiformis* have a distinct yellow appearance due to leaf chlorosis and the colour of the urediospores. Rapid spread is favoured by warm weather with frequent rainfall, but disease development is inhibited by temperatures above 21–22 °C. The fungus primarily survives between crops on volunteer plants. Brown rusts of both wheat and barley both occur as discrete pustules, often surrounded by a pale halo (Plate 6). Brown rusts were formerly diseases of sporadic importance, but the introduction of wheat and barley cultivars susceptible to these diseases in the early 2000s has led to an increase in severity of both *P. triticina* and *P. hordei*.

Rhynchosporium secalis is the causal agent of leaf blotch or scald of barley (Fig. 2.4), and is the most important foliar disease of barley in some areas of the UK, particularly the west and south-west

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of the country. In severe infections the leaf blade may be covered with lesions which have a dark brown, occasionally purple, border. The fungus survives between crops on stubble and straw debris, and because of this, autumn-sown barley is more prone to attack than spring-sown. It spreads by means of splash-borne conidia and, like the septoria diseases of wheat, *R. secalis* is a wet-weather pathogen. Net blotch, caused by the fungus *Pyrenophora teres* (Plate 7), is another trash-borne disease of barley which is prevalent in wet conditions, and which, like *R. secalis*, may cause severe crop loss in moist summers. The disease often appears as reticulate, net-like lesions on leaves and furthermore is seed-borne. Another leaf spot, *Ramularia collo-cygni*, was recognised as an important disease of barley during the 1990s and early 2000s: prior to that, symptoms may have been erroneously diagnosed as physiological damage due to stress. The disease is apparent on the leaves and awns as rectangular black lesions, often surrounded by a lighter yellow tissue. *Ramularia collo-cygni* may become prominent on the upper leaves of barley after wet conditions in early summer. Net blotch, *R. collo-cygni* and *Rhynchosporium* generally cause little damage in dry summers.

Since the 1980s a steady increase has occurred in the incidence and severity of barley yellow dwarf virus. The virus causes a stunting and yellowing of autumn-sown cereals (Plate 8) and is transmitted by cereal aphids, which have survived in considerable numbers during the predominantly mild winters of many recent years in the UK. Early sowing of winter barley crops has further increased the risk of infection by this aphid-borne virus.

Foliar pathogens of cereals are of particular importance when they attack the flag leaf of wheat, and the flag leaf and awns of barley. The products of photosynthesis in the flag leaves of cereals and in the awns of barley contribute between 50% and 80% of grain weight. If the photosynthetic capacity of flag leaves and awns is reduced, or the products of photosynthesis are utilised by the pathogen acting as a rival 'sink' to the phloem, then a loss in grain number and weight will ensue.