The encyclopaedia of cereal diseases
How to use this encyclopaedia

This publication provides easy reference to the diseases of UK cereals and the pathogens responsible (the causal organisms). The diseases are ordered alphabetically by common name (including variants), from ascochyta leaf scorch to yellow rust. The pathogens responsible are also listed alphabetically at the back of the encyclopaedia (alongside the common names). Both the teleomorph (sexual stage) and anamorph (asexual stage) are listed, when appropriate.

A simple glossary of technical terms can also be found at the end of the encyclopaedia.

For each disease, the pages are structured in a similar way. The common name features at the top of each page. A blue box includes the pathogen responsible and the hosts affected. Where fungal pathogens have both a teleomorph and an anamorph, the teleomorph is listed first, with the anamorph in brackets. There then follows a more detailed description of the disease under the following headings; Hosts, Symptoms, Life cycle, Importance. This is complemented by photographs, as appropriate.
The need to deliver consistent, high yields of high quality grain makes control of cereal diseases an important component of successful crop management. Recognition of the disease, and an understanding of the pathogen(s) responsible, is the first step in successful disease control.

This encyclopaedia was produced to help the grower, adviser and others involved in cereal production recognise diseases and learn something about them.

Symptoms of both common and less frequently found diseases are illustrated and described, together with an outline of the disease cycle of the pathogen and an indication of the importance of the disease. This encyclopaedia is not intended to be a guide to disease control. Such information is readily available elsewhere, for example the principles of wheat disease management can be found in the Wheat disease management guide and current information on fungicide performance is published annually on the AHDB website.

The production of this encyclopaedia was very much a team effort, bringing together Bill Clark of Broom’s Barn Research Centre (now at NIAB), who wrote the text, with Rosie Bryson and Lindy Tonguç of BASF and Clare Kelly and Graham Jellis from AHDB, who edited and formatted it. The publication was funded by BASF and AHDB Cereals & Oilseeds. We hope you find it interesting and informative.
Ascochyta leaf scorch (spot)

Hosts
The disease affects wheat, barley, oats, rye, triticale and many grass species.

Symptoms
Disease symptoms are found on lower leaves early in the season and on upper leaves later on. The lesions are usually elliptical and although chlorotic at first, soon become buff to brown in colour, often splitting longitudinally. Initially lesions have a dark brown margin with a papery white centre. The fungus often invades damaged leaf tissue such as that caused by liquid urea or nitrogen. Symptoms become less distinct with time and become very similar to those caused by S. nodorum. Pycnidia within the lesions are generally black, distinguishing the disease from S. nodorum which tends to have light coloured pycnidia.

Life cycle
Pycnidia and mycelium within leaf tissue are thought to survive on crop debris, much like the septoria pathogens.

Importance
The disease is of relatively minor importance although in individual crops it is likely to add to leaf death in the same way as the septoria diseases.

Symptoms are often seen later in the season towards the end of grain filling when they are unlikely to cause any yield loss. The teleomorph stage of the fungus (Didymella exitialis) is common in Europe and the air-borne ascospores of the fungus are commonly found in late summer in the UK, where they have been implicated in late summer asthma.

Typical ascochyta lesion showing dark margin and black pycnidia
Ascochyta leaf scorch (spot)

Leaves become infected by rain splash or as air-borne ascospores

Brown lesions occur on older leaves

Pycnidiospores are released from pycnidia

Pycnidia (rain splash)

Ascopores

Perithecia (wind blown)

Overwinters as mycelium and pycnidia in host debris

Ascochyta leaf scorch (spot) life cycle

Typical ascochyta symptoms on wheat leaves
Barley yellow dwarf virus (BYDV)

Hosts
The disease affects all cereals and grasses. Barley and oats are usually more severely affected than wheat.

Symptoms
The initial symptoms of BYDV infection are normally seen as individual plants with bright yellow upper leaves scattered through the crop. Later, as infection spreads, larger areas of the crop become infected, appearing as patches of bright yellow and severely stunted plants. BYDV is most damaging to plants infected at early growth stages; plants can be killed by very early infections. The effects of BYDV may be exacerbated by other stress factors such as adverse weather conditions, soil acidity and other pests and diseases.

Life cycle
BYDV became much more important and widespread with the increase in early-drilling of winter cereals. The virus exists as several strains and is transmitted by various species of cereal aphid. The bird-cherry aphid (*Rhopalosiphum padi*) is the principal vector in the south of England. In the north of England and in the Midlands the grain aphid (*Sitobion avenae*) is usually more important.

In the autumn, BYDV can be introduced into cereal crops in two ways:

1. Direct transfer by wingless aphids living on grass or on volunteer cereals which survive cultivation and move through the soil colonising the following cereal crop. This is much more common in coastal areas of the south west where cereals may follow grass and winters can be mild.
2. Indirect transfer by winged aphids migrating into newly emerged crops from grass or volunteer cereals elsewhere. BYDV introduced by winged aphids flying into crops is generally much more common and important than BYDV resulting from direct transfer.

**Importance**

With the now common practice of sowing winter barley very early, BYDV has increased in importance in many areas of the UK. The frequency of very mild winters has also meant that, for many farms, BYDV is now a regular problem. Early infections can kill patches of plants potentially resulting in large yield losses.
**Black point**

Hosts
The disease can affect all cereal species although wheat and barley are most commonly affected. The same fungi can cause discolouration of oats.

Symptoms
Symptoms are only visible after harvest. Affected grain shows a darkening of the outer coat particularly at the embryo end of the grain.

Life cycle
The disease is associated with a number of airborne fungi including *Alternaria* spp. and *Cladosporium* spp. although the evidence for these fungi actually causing the disease is still limited and is mainly circumstantial. High humidity or frequent rainfall from the milky ripe to soft dough stage and lodging can often trigger infection by these fungi.

Importance
The disease has no significant effect on yield but can have serious implications for the quality of milling wheat, barley and oats for processing. The discolouration of the grain can lead to poor flour and bran colour, and rejection on the basis of discoloured grains. Durum wheat seems to be particularly susceptible.

The disease is commonly reported to be more severe on larger grains, so high specific weight grain can have a higher incidence. This is thought to be due to the larger grains producing a more open floret, allowing fungal spores greater access to the germ end of the grain.

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Pathogen: *Alternaria* spp. and *Cladosporium* spp.

- Wheat
- Barley
- Oats
- Rye
- Triticale

Darkening of grain at the germ end
Black point

Conidia infect ears post GS 75

Air-borne conidia

Cladosporium

Alternaria

Dark brown discolouration at embryo end of grain

Seed infection can cause poor germination

Overwinters on crop debris and vegetation

Black point life cycle
Black (stem) rust

Pathogen: *Puccinia graminis* f.sp. *tritici*

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Hosts**

*Puccinia graminis* f.sp. *tritici* is specific to wheat.

**Symptoms**

Despite its name, the characteristic symptoms are brown sporulating pustules which occur in stripes on leaves and stems. Later in the season, black elongate pustules containing the teliospores develop, mainly on the stems.

**Life cycle**

The fungus develops teliospores on the wheat plant which produce a secondary spore, the basidiospore. This spore infects a completely different host – the barberry (*Berberis* spp.). A further spore stage, the aeciospore, is produced on the barberry which can spread and reinfect the cereal host. This infection gives rise to the uredospore stage which produces the normal symptoms on wheat. Outbreaks in Britain are caused by air-borne spores originating in South West Europe and North Africa so infection
Black (stem) rust generally occurs when appropriate air movements occur. The optimum temperature is in excess of 20°C so suitable air movements must be associated with high temperatures. Temperatures below 15°C inhibit development of the disease so it rarely occurs under UK conditions.

Importance
The disease is very rare in the UK but may occur late in the season. Losses due to the disease in the UK are presently very small.
Hosts

*Puccinia triticina* is specific to wheat, other *Puccinia* spp. and pathotypes can affect barley, rye and triticale but do not cross-infect.

Symptoms

Symptoms of brown rust infection are often seen in the autumn on early-sown crops as individual orange to brown pustules. With early autumn infection individual pustules can be confused with yellow rust, being orange to brown in colour and about 0.5–1.0mm in diameter. Later in the season, diagnosis is much easier as the brown pustules tend to be scattered at random compared with the more striped symptoms of yellow rust (*Puccinia striiformis*). Although symptoms are most common on leaves, in severe attacks pustules can also occur on the stem and glumes. Brown rust infection of the glumes can result in a reduction in specific weight. When leaves begin to senesce, a ‘green island’ develops around individual pustules. Towards the end of the season dark teliospores are sometimes produced.
**Life cycle**

The fungus over winters primarily on volunteers and early drilled crops. The alternate hosts for *P. triticina* include species of thalictrum, isopyrum and clematis, although their role in the life cycle in the UK is not thought to be significant.

Until recently the disease was rarely important in the spring as temperatures between 15°C and 22°C, accompanied by 100% relative humidity, are needed for sporulation and spore germination. Consequently, brown rust epidemics have normally occurred during mid to late summer in the UK with dry windy days which disperse spores, and cool nights with dew, favouring the build-up of the disease.

However, with mild winters, brown rust can often be found at high levels in the spring. With climate change mild winters and warm springs are likely to become more common which could lead to brown rust becoming a much more common problem earlier in the season.
Brown (leaf) rust

Importance
Until recently brown rust was not considered to be a major problem despite early-sown crops generally carrying high levels of brown rust through the winter. However, the occurrence of new virulent strains overcoming varietal resistance in a few key wheat varieties has moved brown rust up the league table of importance. Severe attacks result in a significant loss of green leaf area and hence yield, infection of the ears will also result in loss of grain quality.
Bunt or stinking smut

Pathogen: *Tilletia tritici*

<table>
<thead>
<tr>
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<th>Barley</th>
<th>Oats</th>
<th>Rye</th>
<th>Triticale</th>
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Hosts

The disease is specific to wheat.

Symptoms

No symptoms can be observed prior to ear emergence. The flag leaves of infected plants show yellow streaks and plants can be stunted, with ‘squat’, dark grey-green ears and slightly gaping glumes. In infected ears the grain is replaced by seedlike ‘bunt balls’ each containing millions of greasy, black, foul smelling spores. In severe cases, the whole field may smell of rotting fish. In wet weather the ears may appear to be covered in a black ink like substance as the spores ooze out of the protective glumes onto the ear and stem.

Life cycle

The spores on the seed surface germinate along with the seed. Each produces a short fungal thread terminating in a cluster of elongated cells. These then produce secondary spores which infect the coleoptiles of the young seedlings before the emergence of the first true leaves. The mycelium grows internally within the shoot, infecting the developing ear. Infected plants develop apparently normally until the ear emerges and the grain sites can be seen to have been replaced by bunt balls.
In damp soil, spores usually germinate and then, in the absence of the host plant, die. However, in dry seasons, they may survive in the soil (especially if they are protected within the glumes of shed ears) from the harvesting of one crop to the sowing of the next. Wind blown spores, particularly from late-harvested crops, can contaminate neighbouring fields which may present bare soil ready for planting the next crop.

**Importance**

As each bunt ball contains millions of spores, the capacity for contamination of healthy grain in the same field is enormous. Thus, if seed is continually saved and re-sown without treatment the disease can build up very rapidly. Dry spores can survive for several years. Harvesting or handling equipment contaminated by spores from an infected crop can introduce the pathogen into seed lots harvested in the following season. The disease is rare in the UK as the vast majority of seed is treated with a fungicidal seed treatment.

However, the disease is potentially very damaging and can lead to complete crop loss due to the grain being unsaleable because of discoloration and smell. Cases usually arise from sowing untreated farm-saved seed although soilborne infections also occur.

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**Bunt or stinking smut life cycle**

- **Germinating wheat kernel infected by teliospores**
- **Mycelium from germinating teliospores reaches and follows growing point of wheat plant**
- **Wheat kernel replaced by teliospores in ‘bunt balls’**
- **Intact bunt balls survive in soil overwinter**
- **Bunted ear**
- **Infection results in stunted plant and bunted ear**
- **Bunt balls broken open during harvesting releasing teliospores which overwinter on seed**
Cephalosporium leaf stripe

Pathogen: *Hymenella cerealis* (*Cephalosporium gramineum*)

Hosts
The disease affects wheat, barley, oats, rye and triticale.

Symptoms
Affected plants are usually randomly scattered throughout the crop. Affected tillers have a single distinct bright yellow stripe on each leaf which extends onto the leaf sheath. All leaves on a tiller usually show symptoms but not necessarily all tillers on a plant. The vascular tissue close to the nodes is frequently discoloured. Tillers can ripen prematurely and produce white-heads.

Life cycle
The fungus causing the disease is soil-borne and enters the roots of plants via physical damage. In the UK this disease used to be common in wheat following grass where high levels of wireworm (*Agriotes* spp.) were found – causing root damage. Soil-borne conidia normally enter roots through damage in the winter months and the fungus grows in the xylem vessels, blocking vascular tissues, particularly at the nodes.

The fungus survives in crop debris returned to the soil after harvest.

Importance
The disease is common at very low levels in the UK and does not cause economic losses.
Cephalosporium leaf stripe

During winter and early spring, conidia enter roots from soil through damaged areas.

Conidia carried by xylem lodge and multiply in nodes and leaves.

Sporangia produce conidia which enter soil.

Causes leaf stripe symptoms – usually single yellow stripe on each leaf.

Infected debris remains after harvest.

Fungus survives as saprophyte on straw and other crop debris.

Cephalosporium leaf stripe life cycle
**Covered smut**

**Hosts**
The species of the pathogen are crop specific, mainly occurring on barley and oats.

**Symptoms**
There are no symptoms of the disease before ear emergence. At ear emergence the ears seem to be normal except that the grains appear to be covered in a thin membrane. If this is broken open it can be seen that the grains have been replaced by masses of black spores held in place by the transparent membrane. The membrane is relatively easily ruptured and as spores are released the symptoms become similar to those of loose smut.

**Life cycle**
After ear emergence some spores may be released on to the rest of the crop and carried by the wind to neighbouring plants (as in loose smut, caused by *U. avenae* and *U. nuda*). However, many are retained within their membranous envelope until the crop is harvested when, during the threshing process, they are released to contaminate the surrounding seeds. In either case, the spores remain dormant on the outside of the seed until it is sown when they germinate and infect the developing seedling. The fungus then develops with the growing point of the plant until it once again colonises the developing ear.

Covered smut affected barley ears (healthy one on right)
**Covered smut**

**Importance**
The disease is very rare in the UK and is usually only found in crops grown repeatedly from home-saved, untreated seed.
**Crown rust**

Pathogen: *Puccinia coronata*

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<th>Oats</th>
<th>Rye</th>
<th>Triticale</th>
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**Hosts**

The crown rust fungus affects only oats: it does not attack other cereals. It can infect a number of grasses, particularly ryegrass but the forms on grass do not cross infect to oats.

**Symptoms**

The first symptoms of crown rust are very similar to brown rust of wheat and barley. Orange-brown pustules appear scattered over the leaf surface. Leaf sheaths, and later the oat panicle, can also become infected. The disease is favoured by high temperatures (20–25°C) so epidemics usually occur in June–July. Late in the season black pustules appear within the existing crown rust lesions.

**Life cycle**

The orange spores (uredospores) produced on leaves are air-borne and spread the disease long distances to other plants and adjacent crops. Later in the season black pustules containing teliospores are produced. These remain dormant on crop debris until the spring when they germinate to produce basidiospores.

The basidiospores of this fungus infect the alternate hosts, the buckthorn (*Rhamnus catharticus*) and alder buckthorn (*Frangula alnus*) on which are produced a third spore type, the aeciospores, which can then infect oats once again.

![Crown rust pustules](image)
Crown rust

Uredospores produced in the oat crop also infect volunteer oat plants and then emerging winter oats.

**Importance**
The disease is favoured by warm and humid weather and mild winters. Severe attacks have been more common in recent years. Such epidemics can reduce yield by 10–20%.
**Dwarf bunt**

Pathogen: *Tilletia controversa*

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Hosts**

The disease affects wheat, winter barley, rye and triticale.

**Symptoms**

This disease is not yet known in the UK but is of interest because of evidence of the long-term survival of bunt (*T. tritici*) in soil. As dwarf bunt can also survive for long periods in the soil, the possibility of hybrids between *T. controversa* and *T. tritici* has been widely debated. Dwarf bunt occurs in Canada, North and South America and many parts of Europe and Asia.

Microscopic examination of teliospore morphology is the primary means of distinguishing species of *Tilletia*. However, there is a wide range of teliospore characteristics in *Tilletia* spp. so spore morphology alone is not a suitable method of identification. The symptoms of dwarf bunt are very similar to bunt except that affected plants are stunted to between half and quarter of their normal height. Infected ears tend to have a more ragged appearance than with bunt. Yellowing and flecking of leaves of affected plants is also occasionally found.
**Dwarf bunt**

**Life cycle**
The disease cycle of *T. controversa* matches that of *T. tritici* very closely. Infected plants appear unaffected until ear emergence. When the ears emerge the seeds are seen to have been replaced by 'bunt balls' which break open during harvest, contaminating healthy grain. When contaminated grain is sown the following season the spores on the outside of the grain germinate, eventually reaching the growing point of the plant. It is at this stage that *T. controversa* differs from *T. tritici*. With bunt, the fungus normally infects the plant via the coleoptile as the seedling is emerging. However, with *T. controversa*, there is a long incubation period and a requirement for cool temperatures before the spores will germinate. As a result, the fungus frequently infects plants much later in development.

At harvest, 'bunt balls' contaminate the soil as well as healthy seed but with dwarf bunt this is very significant as the fungus can survive in soil for many years. Free spores are reported to remain viable in soil for at least three years and 'bunt balls' can survive for up to 10 years.

**Importance**
Presently, the disease is not known in the UK. However, like bunt, the disease could be very damaging although seed treatments commonly used in the UK would prevent seed-borne infection.
**Ergot**

**Pathogen:** *Claviceps purpurea*

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Hosts**
The disease affects all cereal crops and a wide range of grasses, particularly black-grass (*Alopecurus myosuroides*).

**Symptoms**
The causal fungus only attacks the ear at flowering, replacing the grain in a few spikelets by a hard, purple-black sclerotium, known as an ergot. Such ergots can be very large, up to 2cm in length, and are very obvious in the standing crop and in contaminated grain samples.

**Life cycle**
Ergot is not truly a seed-borne disease as it is not carried on or in seed. However, it can be spread by ergots in contaminated seed. It is also one of only two diseases which are included in the UK Seed Certification Scheme for Cereals (the other being loose smut).

At or near to harvest, ergots fall to the ground where they remain until the following summer, when they germinate to produce club-shaped spore-bearing structures (stroma). These ascospores are spread by the wind to nearby open flowers of grasses and cereals. The spores germinate in the flower, infecting the ovaries. This infection leads to the production of secondary spores (conidia) encased in a sticky secretion commonly referred to as honeydew. This attracts insects which carry the spores to other flowers where further infection can occur.

There are a number of strains of the fungus, some of which can infect grasses and cereals, others which are restricted to certain hosts. Wheat and other cereals are less
Ergots in infected wheat ears

Ergot

Ergots in a sample of treated seed

Club-shaped, spore bearing structures (stroma) from germinated ergots

severely affected than rye although occasionally more open-flowered wheat varieties can be badly affected. The disease is favoured by cool, wet conditions during flowering which facilitate spore production and prolong the flowering period, making infection more likely.

Importance

The disease has very little direct effect on yield but the ergots contain large amounts of toxic alkaloids. Consequently, if grain contaminated with ergots is fed to stock or used to make flour then there are risks to animal and human health. As a result, contaminated grain may be rejected.

Ergots in a sample of treated seed
Ergot

Perithecia release ascospores which infect flowering plant. Mycelium penetrates ovary tissue

Grains replaced by sclerotia

Secondary spread from grasses in honeydew

Sclerotia overwinter in soil

Ergot life cycle

Germinating sclerotia produce stroma containing perithecia

Ascospores infect grasses – especially black-grass

Perithecia
Eyespot

Pathogen: *Oculimacula yallundae*, (*Helgardia herpotrichoides*) *O. acuformis* (*H. acuformis*)

☒ Wheat
☒ Barley
☒ Oats
☒ Rye
☒ Triticale

Hosts

*O. yallundae* (W-type) is more pathogenic on wheat and barley than on rye, *O. acuformis* (R-type) is pathogenic on wheat, barley, rye and triticale.

Symptoms

Early symptoms can be confused with sharp eyespot and *Fusarium* spp. Frequently, all that is visible is a brown smudge on the leaf sheath at the stem-base. In early-sown crops eyespot lesions may penetrate one or two leaf sheaths, making identification more conclusive. Lesions caused by *Fusarium* spp. and sharp eyespot are generally confined to the outer leaf sheath. Later in the season eyespot symptoms become more distinct and appear as an eye-shaped lesion with a dark margin, usually below the first node. Later still, the margin of the eyespot lesion is often dark and diffuse with a central black ‘pupil’ occasionally visible. In severe attacks of eyespot, white-heads (‘bleached’ ears) are commonly seen scattered through the crop, later in the season these may become colonised by sooty moulds.

White-head symptom in wheat crop
Eyespot tends to be more severe if plants are also suffering from take-all.

**Life cycle**

The fungus over-winters on infected stubble, volunteers and grass weeds acting as sources of inoculum. It can survive on stubble for up to three years, so a break from cereals will not necessarily reduce eyespot risk in following crops. Spores are produced throughout autumn and winter, posing a threat to early sown crops. Infection occurs at temperatures above 5°C and during wet periods. Spores are rain splashed short distances from infected stubble. The development of symptoms following infection takes 6–8 weeks, depending upon environmental conditions. Eyespot can be a serious problem in continuous cereals, where inoculum may build up from year to year.

The sexual stage of both eyespot fungi may play an important part in the pathogen life cycle. This stage of the fungus is produced on stubble at the end of the season and after harvest, ascospores may travel long distances and infect emerging or young plants.
Eyespot

Importance
Eyespot is often underestimated in importance because few farmers ever look at the stem bases of crops at the milky ripe stage or later when severe eyespot can often be seen. Moderate or severe eyespot infections can cause yield loss in the order of 10–30%, even in the absence of lodging. Where eyespot is severe, lodging can occur – causing problems in harvesting and frequently a reduction in Hagberg Falling Number.
Eyespot

Conidia splash from debris to young plants. Ascospore infection of autumn sown crops.

‘Eyespot’ lesions on stem base

Sexual stage on straw debris may result in long distance spread of ascospores.

Lesions cause white-heads and sometimes lodging.

Eyespot life cycle
Flag smut

Pathogen: *Urocystis agropyri*

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Hosts**
The disease affects wheat and many grass species; the strain(s) that affect wheat are specific to that crop.

**Symptoms**
Affected plants are severely stunted. Excessive tillering is common and often the ears fail to emerge, remaining within the boot. Plants show long dark grey to black streaks on the leaf blades and leaf sheaths. The streaks eventually erupt, giving the leaves a ragged appearance and exposing the black teliospores which are then dispersed, giving the plants the appearance of being covered in soot.

**Life cycle**
The teliospores released from the leaves can either be blown onto grain of healthy plants, contaminating the grain, or they can drop to the soil where they are very persistent, surviving up to four years. When contaminated grain is sown, or if healthy grain is sown into contaminated soil, the teliospores germinate, producing a secondary spore type – the sporidia. These spores infect the germinating wheat seedlings coleoptile. The fungus, having penetrated the seedling, then grows inside the plant, eventually producing the typical striping on the upper leaves late in the season, giving rise to a new generation of teliospores. The teliospores can survive in soil for several years, so even where a break from cereals occurs, subsequent wheat crops may become infected.

**Importance**
Flag smut of wheat occurs in some European countries and in Australia, Canada and the USA. However, it was not known in the UK until 1998 when an outbreak was confirmed in Essex. The disease is not particularly damaging unless present at high levels but it can have serious consequences with regard to exporting grain or wheat products. Many countries have quarantine restrictions which prohibit the import of wheat products from countries where the disease is established.
Flag smut

Fungus grows systemically within plant

Sori erupt releasing spore balls containing teliospores

Teliospores germinate forming promycelium and sporidia

In autumn and spring sporidia germinate. Mycelium infects seedlings

Sori develop between veins on leaves and glumes

Spore balls

Overwinters as spore balls on seed in the soil

Flag smut life cycle
## Foot rot

**Pathogen: Cochliobolus sativus (Drechslera sorokiniana)**

- Wheat
- Barley
- Oats
- Rye
- Triticale

### Hosts
The disease affects all cereals.

### Symptoms
The fungus causes disease symptoms similar to *Fusarium* spp. Seedborne infection can result in seedling death although infected plants usually grow to maturity. Affected plants show brown spotting on the lower leaves and, if severely affected, can show stem-base rotting and poorly filled ears. This severe symptom is very rare in the UK.

### Life cycle
The fungus behaves very much like *Fusarium* spp. in its survival and life history. It is both soil and seed-borne. It infects seedlings as they emerge, occasionally producing a seedling blight. More usually it infects roots of seedlings, allowing the plant to survive. Leaf spotting and stem-base infections produce splash-borne spores which can be carried to emerging ears resulting in seed infection.

### Importance
Foot rot due to *C. sativus* is traditionally a disease of hotter climates than that of the UK.
Foot rot

Foot rot browning at the base of young barley plants

Diseased plants appear stunted, mature early and have shrivelled grain

In autumn and spring ascospores and conidia from conidiophores infect roots and stem bases

Pseudothecia occur on the stem-base at the end of the season

Overwinters on crop debris as pseudothecia and mycelium

Foot rot life cycle
Fusarium (foot rot, seedling blight, ear/head blight)

Pathogen: *Fusarium* spp. and *Microdochium nivale*

- Wheat
- Barley
- Oats
- Rye
- Triticale

Hosts
Wheat, barley, oats, rye, triticale and grasses.

Symptoms
There are many species of *Fusarium* that affect cereals. These fungi form a complex of diseases on seeds, seedlings and adult plants. The seed-borne pathogen *Microdochium nivale* (formerly known as *Fusarium nivale*) is also usually included in this group of fungi.

*M. nivale* is the primary pathogen in the group which causes seedling blight resulting in seedling death and thinning of the plant stand. Other species cause a range of symptoms including brown lesions on stem bases, often restricted to the outer leaf sheath. *Fusarium* lesions often begin in the leaf sheath at the stem base where crown roots split the leaf sheath when emerging. This infection can then spread up the leaf sheath causing long dark brown streaks at the stem base. The most commonly seen symptom in the UK is the dark brown staining of the lower nodes.

Fusarium staining on the lower nodes

Fusarium on the stem-base
Fusarium (foot rot, seedling blight, ear/head blight)

On older plants Fusarium infection can produce a true foot rot, where the stem base becomes brown and rotten, resulting in lodging and white-heads.

This symptom is less common in the UK, although it can be found in very dry seasons.

Many of the Fusarium species cause a range of symptoms – often termed ear blights. F. culmorum and F. graminearum are the two most commonly found species in the UK. Other species include F. avenaceum, F. poae and F. langsethiae. Infection frequently results in the whole or part of the ear becoming bleached. This symptom is seen when ears become infected during the early flowering stages. Later infections may result in infection of the grain but without obvious bleaching of the ears. The ear blight phase of the disease can cause yield loss but is most important as it can result in mycotoxin production in the grain. Mycotoxins are substances toxic to animals and humans. Levels in grain, flour and flour products for human and animal consumption are limited under EU legislation. See Guidelines to minimise risk of fusarium mycotoxins in cereals (AHDB Cereals & Oilseeds) for more details.
Fusarium *(foot rot, seedling blight, ear/head blight)*

Ear blight symptoms on wheat (*Fusarium poae*)
**Fusarium** *(foot rot, seedling blight, ear/head blight)*

**Life cycle**
The most important source of *Fusarium* on wheat crops is the seed but the fungus can also survive on debris in the soil. In seasons where weather conditions are wet during flowering and grain formation, spores are splashed from lower in the canopy causing ear blights and seed-borne infection. In such seasons seed-borne infection can pose a serious threat to crop establishment unless seed is treated to control *Fusarium*. All of the cereal *Fusarium* species are common in soil. Most have competitive saprophytic abilities which allow them to colonise debris and stubble in soil. Volunteers may also act as a source of inoculum.

**Importance**
Symptoms of *Fusarium* infection are common in wheat crops in the UK and most cereal crops will have some symptoms of one or other of these diseases. When weather conditions are wet during flowering, high levels of ear blight can occur but their incidence is frequently over-estimated and losses are only rarely serious. Severe foot rotting is very rare in the UK and losses are generally very small. The seedborne phase of the disease is potentially very damaging. Seed treatment plays a major role in preventing seedling losses in wheat. Seedling blight is rare in barley.

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**Fusarium (foot rot, seedling blight, ear/head) life cycle**
**Halo spot**

Pathogen: *Selenophoma donacis*

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Hosts**
The disease only affects barley.

**Symptoms**
Halo spot is found mainly in western coastal areas where outbreaks occur in wet summers after flag leaf emergence. The disease appears as small leaf spots (1–3mm long) often square or rectangular in shape, pale brown in the centre with dark purple/brown well defined margins. Pycnidia occur in lines along the veins within the central area of a lesion. Spots generally occur towards the tips and along the edges of leaves. They also affect the leaf sheath and ear (especially the awns). This disease often occurs with rhynchosporium but can be distinguished from the latter by the smaller size of the spots and the presence of pycnidia within the lesions. Also, halo spot tends to occur most frequently on the upper leaves whilst rhynchosporium is often more common on the older foliage.

**Life cycle**
The disease originates from infected seed, stubble and volunteer barley plants. Symptoms on lower leaves which arise from seed infection or from stubble contact or splash are indistinct but spread up the plant in rain-splash, usually in warm conditions later in the season. The disease rarely becomes important until after flag leaf emergence when it can develop rapidly in wet weather.

**Importance**
The disease occurs sporadically, usually in wet seasons. Traditionally the disease occurs mainly in the south west of England where rainfall is high but is rare elsewhere in the UK – it generally does not cause significant yield loss.
Halo spot symptoms on a barley leaf

In autumn and spring crops are infected by pycnidiospores

Splash dispersal of conidia up plant

Halo spot symptoms occur on leaf tissue

Pycnidia form in lesions

Overwinters as pycnidia on crop debris

Halo spot life cycle
Leaf and glume blotch

Pathogen: *Phaeosphaeria nodorum* (Stagonospora nodorum formerly *Septoria nodorum*)

- ✔ Wheat
- ✔ Barley
- 🚫 Oats
- ✔ Rye
- 🚫 Triticale

**Hosts**
Mainly wheat, but occasionally barley and rye.

**Symptoms**
*S. nodorum* can be seed-borne and infect seedlings, resulting in water-soaked, dark green areas on the coleoptile, later becoming necrotic. Twisted, distorted and stunted seedlings may also occur. On mature leaf tissue the first symptoms of infection are small necrotic lesions. Later these develop into brown oval lesions surrounded by a chlorotic halo. These lesions frequently coalesce to produce large areas of dead, dry and sometimes split tissue. Pycnidia form within infected tissue, but these are a pale pinkish brown colour and difficult to see in the field, even with a hand lens. They are best seen by viewing the lesions in transmitted light with a hand lens.

*S. nodorum* can also infect the ears, particularly of wheat, causing glume blotch. Dark brown patches like burn-marks develop on the glumes, which later become purple-brown. Glume blotch symptoms are easiest to see on green ears.

Glume blotch symptoms on a wheat ear
Leaf and glume blotch

Although more usually associated with necrotic blotching of leaves and glumes, *S. nodorum* can cause post-emergence seedling blight in cool wet soils.

**Life cycle**

*S. nodorum* survives as dormant mycelium, and as pycnidia and pseudothecia on seed, stubble, debris, autumn-sown crops and volunteers. In the absence of crop debris, initial infections in the autumn or spring may result from wind-borne ascospores released from pseudothecia long distances away. As temperatures rise and humidity increases pycnidiospores are produced from the pycnidia. These are splash-dispersed up the infected plant and from plant to plant. Temperatures of 20–27°C, together with 100% relative humidity, are optimal for spore production and germination and a period of rain is essential for spore dispersal. The disease cycle can be completed in 10–14 days during such conditions. Spores produced from pseudothecia and pycnidia, which develop on the flag leaf and ear at the end of the season, can initiate infection in early autumn-sown crops and volunteers and may also remain dormant for the winter. Glume blotch infection of the ear can lead to infection of the seed. Like the *Fusarium* spp., *S. nodorum* can survive between crops either on seed or on plant debris.

While trash-borne inoculum is usually more important in initiating the later phases of the disease (leaf and glume blotch), fungus carried on the seed is more likely to be responsible for septoria seedling blight.

**Importance**

*S. nodorum* was once the most serious pathogen on cereals in the UK, although it now rarely causes significant losses except in wet seasons in the south west of England. Yield losses up to 50% have been reported in trials although average annual losses in the UK probably do not exceed 3%. Losses caused by septoria seedling blight are generally not significant.
Leaf and glume blotch

In spring, crops also infected by pycnospores and ascospores

Seed infection causes damping off and early infection of plant

Overwinters on crop debris, grass weeds, and volunteers

Leaves and ear infected by contact and rain splash

Seed infection

Pycnospores

Ascospores

Leaf and glume blotch life cycle
Leaf spot

Pathogen: *Pyrenophora avenae (Drechslera avenae)*

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Hosts**
The disease is specific to oats.

**Symptoms**
The primary phase of the disease (from seed-borne infection) appears as short brown stripes with purple edges on the emerging leaves. These stripes appear on the first three or four leaves of emerging seedlings. The secondary phase of the disease (splash-borne spores) appears as red-brown spots with purple margins on leaves.

**Life cycle**
The primary phase of the disease arises from seed-borne infection. Spores from the early leaf stripes then splash up the plant, producing the secondary leaf spot phase of the disease. Eventually spores splash up onto the ear where the grain becomes infected. Infected grain can cause seedling death during or soon after emergence. Surviving seedlings give rise to the primary phase of the disease. Infected debris is not thought to be a significant part of the disease cycle.

**Importance**
The disease is no longer a serious pathogen of oats. Although it is not uncommon, severe outbreaks are very rare and probably associated with repeated home-saving of untreated seed.
Leaf spot

Close-up of symptoms showing short brown stripes with purple edges

Leaf spot life cycle

Upper leaves with spotting symptoms

Conidia are produced on infected leaves

Spread to upper leaves

Seedling infection

Emerging leaves Seedling striped

Conidia are blown onto adjacent ears infecting seeds

Perithecia may occur but are rare in nature
**Leaf stripe**

Pathogen: *Pyrenophora graminea* (*Drechslera graminea*)

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Hosts**
The disease is specific to barley.

**Symptoms**
The disease is seed-borne and causes long brown stripes on the leaves. The stripes are often pale green at first, becoming yellow and then finally dark brown. Usually all of the leaves of affected plants show these symptoms and some leaves split along the stripes giving the leaf a shredded appearance. Symptoms are usually most prominent at ear emergence. The disease is generally most severe on crops which have been grown from untreated seed.

Leaf stripe can affect the plant in three ways; first it can kill seedlings as they emerge. This is unusual but can occur if soil conditions are very poor. Secondly, it can reduce the efficiency of the plant by reducing green leaf area and thirdly, it can result in complete blindness of the ear resulting in no harvestable grain from affected tillers.

**Life cycle**
The fungus is present on the seed surface and as mycelium in the seed coat. As the coleoptile emerges, the fungus invades the tissue and penetrates through to the emerging first leaf. The fungus grows through successive leaf sheaths, producing the characteristic symptoms on each leaf until it infects the ear which often remains in the leaf sheath. Although the fungus produces spores on the stripes these are not thought to be very important in the UK as a means of spreading the disease.

**Importance**
This is potentially the most serious seed-borne disease of barley. If seed from affected crops is re-sown without an effective fungicidal seed treatment being applied, the disease can multiply very significantly and produce large yield losses. If seed is saved and re-sown repeatedly, complete crop loss is possible within a few generations of seed multiplication.
Leaf stripe

Leaf stripe symptoms on barley

At ear emergence conidia are produced on infected leaves.

Conidia are blown onto adjacent ears infecting seeds.

Perithecia may occur but are rare in nature.

All leaves show leaf stripe.

Seedling infection.

Overwinters as seed borne mycelium.

Leaf stripe life cycle.
**Loose smut**

*Ustilago nuda f.sp. tritici (U. tritici)* – Wheat; *Ustilago nuda f.sp. hordei* – Barley; *Ustilago avenae* – Oats

### Hosts
There are distinct forms of the pathogen which are crop specific.

### Symptoms
Loose smut is easily recognised at ear emergence as individual grains are completely replaced by a mass of black fungal spores. Partly affected ears are sometimes seen. The spores are released as soon as the ear emerges, leaving only the bare remains of the ear rachis. Because the blackened ears are so obvious in the crop at ear emergence the disease appears to be very severe, even at very low incidence.

### Life cycle
Spores are released from infected ears and are carried by the wind to the open flowers of surrounding healthy plants. There they germinate and the fungus grows into the developing grain site. Weather conditions during flowering affect the length of time that the florets remain open and hence the time that the plant is susceptible to infection. Thus, the likely level of infection varies considerably from season to season. The fungus lies dormant within the embryo of the seed until the seeds are sown and germinate.
**Loose smut**

When the infected seed germinates the fungus grows within the developing shoot, eventually reaching the ear primordia. The fungus develops within the young ear, eventually replacing spikelets with masses of fungal spores which are released once again as the ear emerges.

**Importance**

The UK Seed Certification Scheme is undoubtedly successful in ensuring that loose smut remains at very low levels in UK seed stocks and is of low importance. Seed crops grown under the scheme are inspected for loose smut and because the disease is so easily seen at low levels it can be detected by visual examination.

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**Loose smut infected wheat ears**

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- Mycelium follows growing point of wheat plant
- Mycelium invades young seedlings
- Mycelium invades part of embryo in seed
- Mycelium invades the grain sites
- Grain sites replaced by masses of teliospores
- Teliospores land on flowers of healthy plants and infect developing grain

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**Loose smut life cycle**
**Mosaic viruses**

**Viruses: See below**

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Mosaic Viruses**

- Barley Yellow Mosaic Virus (BaYMV)
- Barley Mild Mosaic Virus (BaMMV)
- Oat Mosaic Virus (OMV)
- Oat Golden Stripe Virus (OGSV)
- Soil-borne Cereal Mosaic Virus (SBCMV)
- Soil-borne Wheat Mosaic Virus (SBWMV)

Each of these mosaic diseases is caused by a virus, transmitted by the soil-borne vector *Polymyxa graminis*. The viruses causing these diseases are closely related single stranded RNA (ribonucleic acid) rod or filamentous viruses belonging to the genus *Furovirus* or *Bymovirus*.

**Hosts**

The diseases affect only winter-sown crops. All cereals are affected by their own form of the virus which does not cross-infect to other cereals.

**Symptoms**

Affected plants can be very stunted and pale in colour. Typical symptoms are pale yellow streaks in the leaves which are most pronounced during the early spring, particularly following a prolonged cold spell.

The yellow streaks may become brown or purple at the leaf tip and dark brown flecking may replace the yellow streaking.

![Typical yellow streaks and brown flecking of barley yellow mosaic](image-url)
**Mosaic viruses**

**Life cycle**
The disease often occurs in distinct patches which increase in size from one year to the next. The virus is carried in the soil by the root-infecting organism *Polymyxa graminis*. Movement of the soil during cultivations will spread the disease within the field and to other areas. The virus survives within the spores of the vector so that once it is present in soil it persists even in the absence of cereal crops for many (more than 25) years.

**Importance**
Yield losses of 50% have been recorded in the patches on susceptible varieties. BaYMV and BaMMV are very common in the UK. SBWMV and SBCMV are present in the UK but are not yet commonly found. OMV is commonly found OGSV is usually found in association with OMV.

Close-up of yellow streak symptoms on oats

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**Mosaic life cycle**

*Polymyxa graminis* forms intercellular plasmodia and resting spores

*Polymyxa graminis* zoospores carrying virus particles, infect root hairs

Virus particles multiply causing leaf chlorosis and stunting

The vector, *Polymyxa graminis* overwinters in soil

Polymyxa resting spores in root tissue
**Net blotch**

**Pathogen:** *Pyrenophora teres f. teres (Drechslera teres) Pyrenophora teres f. maculata* (spot form)

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Hosts**
The disease affects a wide range of grasses but the forms on barley are specific to that crop and do not affect other cereals or grasses.

**Symptoms**
Infection of young seedlings with net blotch can look very similar to leaf stripe infection – the first leaf often has a single brown stripe extending the whole length of the leaf. However, later leaves do not usually show striped symptoms. Leaves infected by splash-borne spores typically show short brown stripes or blotches with a network of darker lines at random on the leaves. The disease tends to produce ‘stripe’ symptoms or ‘netting’ symptoms which are distinctly different in appearance. There is also another, less common symptom which is termed ‘spot blotch’ where lesions are more oval in appearance. Leaves frequently have yellowing associated with all of these types of lesion, particularly when the symptoms are severe. The glumes and awns can also be affected, producing dark brown flecking and striping.

**Life cycle**
Seed-borne mycelium infects the coleoptile and the first leaf becomes infected as it emerges. Spores produced on this first leaf serve to spread the disease to other leaves and to surrounding plants. Seed-borne inoculum is usually much less important than infected stubble and debris which allows the pathogen to over-winter.

Close-up of typical symptoms of net blotch
**Net blotch**

Trash and crop debris provides much higher levels of inoculum which is splash borne up the plant. Although there are suggestions of long distance spread of ascospores from overwintering pseudothecia the role of these is not thought to be as important as trash-borne inoculum.

**Importance**

Because the seed-borne phase is relatively unimportant compared with trash-borne inoculum the seed-borne phase does not often threaten yield. Net blotch is now a very important disease of barley and can cause large losses where the disease is not well controlled. The disease can be particularly damaging when symptoms continue to develop through the winter and into the early spring, producing an early epidemic as the crop develops.

Net blotch symptoms on barley

**Net blotch life cycle**

- **Primary infection results from conidia and ascospores**
- **Overwinters as seed-borne mycelium and as pseudothecia on crop debris**
- **At end of season pseudothecia develop**
- **Typical net-like symptoms occur**
- **Splash dispersal of conidia up plant**
- **Strong air currents release conidia causing re-infection**
Omphalina patch

Pathogen: Omphalina pyxidata

- [✓] Wheat
- [✓] Barley
- [ ] Oats
- [ ] Rye
- [ ] Triticale

Hosts
The disease affects wheat, barley and grasses.

Symptoms
The disease is normally first seen as stunted areas of poor growth. Affected crops are frequently found on light land. Affected patches can be visible as early as December as sharply delineated but irregular areas of stunted growth although the crop remains a normal green colour. Roots and the soil adjacent to affected plants show characteristic white hyphal masses (like small pieces of cotton wool about 1mm across). Fruiting bodies (basidiocarps) like tiny mushrooms can sometimes be found within affected patches between January and March. They are approximately 2cm high with a 1cm diameter convex cap.

Life cycle
Little is known about the disease cycle although this is a soil-borne fungus which survives between susceptible hosts as sclerotia or as mycelium on root debris. It infects crops soon after emergence, colonises roots, produces sclerotia and then produces air-borne basidiospores. These spores may play a part in long distance spread of the disease. The role of basidiospores is unclear.

Fruiting bodies in an infected patch of barley
**Omphalina patch**

**Importance**
Until recently, the fungus had not been recognised as a pathogen of cereals in the UK. It is now believed to be sporadic in nature, affecting a small number of crops each year, mainly winter barley. Winter wheat is less frequently affected than barley and losses each year will be very small in comparison with other diseases. Limited trials on winter barley suggest infection can reduce fertile tiller number by 40% and yields by 25–50%.

Close-up of the fruiting bodies (basidiocarps)

Omphalina patch life cycle

1. **Basidiocarps seen in crop January to March**
2. **Overwinters as mycelium on crop debris and grass hosts**
3. **White hyphal masses on roots (resemble small pieces of cotton wool)**
4. **Long distance spread by air borne spores**
5. **Infected plants appear stunted and may develop white-heads**
6. **Healthy**
7. **Diseased (stunted)**

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**Basidiocarps seen in crop January to March**

**Infected plants appear stunted and may develop white-heads**

**Long distance spread by air borne spores**

**White hyphal masses on roots (resemble small pieces of cotton wool)**

**Healthy**

**Diseased (stunted)**

**Overwinters as mycelium on crop debris and grass hosts**
**Powdery mildew**

Pathogen: *Blumeria graminis* f. sp. *tritici* – Wheat, Triticale; *Blumeria graminis* f. sp. *hordii* – Barley; *Blumeria graminis* f. sp. *avenae* – Oats; *Blumeria graminis* f. sp. *secalis* – Rye

| ☑️  | Wheat |
| ☑️  | Barley |
| ☑️  | Oats |
| ☑️  | Rye |
| ☑️  | Triticale |

**Hosts**

All cereals can be affected by mildew although there are several forms of the disease which are specific to individual crops and do not cross-infect.

**Symptoms**

Symptoms of powdery mildew can be found on leaves, stems and ears, but leaves are most commonly infected. Typically, white pustules appear which produce a mass of spores with a powdery appearance. As the mildew pustules become older, they darken to a grey or brown colour. Eventually, black spore cases (cleistothecia) can be found embedded in the mildew pustules – usually towards the end of the season.

**Life cycle**

Mildew overwinters primarily as mycelium on volunteers and autumn-sown crops. The cleistothecia produced during late summer are resistant to low temperatures and drying which allows the fungus to survive in the absence of a host.
Powdery mildew

In humid weather, cleistothecia release the sexually produced ascospores which can initiate autumn infections. However, in the UK, green plant material is nearly always available and cleistothecia are thought to be of secondary importance to mycelium. As temperatures rise in the spring, dormant mycelium starts to grow and spores are quickly produced. These germinate over a wide range of temperatures, from 5–30°C, although 15°C is optimal with relative humidity above 95%. Free water inhibits spore germination. Under dry conditions, fresh spores can be formed in about seven days. At the end of the season, volunteers and early autumn-sown crops may become infected, providing inoculum for the following crop.
**Powdery mildew**

**Importance**
Late-sown winter wheat crops are often particularly prone to attack, especially when growing rapidly in the spring. High levels of nitrogen fertiliser encourage the disease and mildew can be particularly severe in dense crops. The visual appearance of the disease usually outweighs its damage potential especially during the autumn and winter. In susceptible varieties, yield losses can be high (up to 20%) and early control can be very important. However, the disease generally causes much smaller yield losses and late attacks (after flowering) on the flag-leaf and ear rarely cause significant losses.

[Image: Powdery mildew with cleistothecia on wheat]
Powdery mildew

Young barley plant infected with powdery mildew

Mycelium develops on young plants

Conidiophores release conidia

In spring, conidia and ascospores start early infections

Re-infection of leaf layers by air-borne conidia

Cleistothecia develop on lower leaves

Overwinters as mycelium and cleistothecia on crop debris, autumn-sown crops and volunteers

Powdery mildew life cycle
Ramularia leaf spot

Pathogen: *Ramularia collo-cygni*

<table>
<thead>
<tr>
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<th>Wheat</th>
<th>Barley</th>
<th>Oats</th>
<th>Rye</th>
<th>Triticale</th>
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### Hosts

The disease affects only winter and spring barley.

### Symptoms

Typical symptoms of ramularia comprise small brown rectangular lesions, often surrounded by a yellow halo. They resemble the spot-form of net blotch. Following high levels of infection, the leaves may senesce rapidly. Lesions are often obvious on dead leaves as black spots. The spores of the fungus are visible on the surface of dead leaves. Ramularia is frequently found in association with other leaf spots such as abiotic sun scorch, physiological leaf spot and spotting caused by damage to the leaf wax following the application of some fungicides.

### Life cycle

Ramularia can be detected on the seed and within symptomless leaves. The disease can also be dispersed via air-borne spores. Symptoms can develop on dead lower leaves but symptoms are rarely seen on healthy green leaves until after flowering. There may be a stress or physiological trigger for symptoms to develop.
The toxin rubellin D is also thought to be produced by the fungus when the barley host is stressed. Under certain light conditions, this toxin causes oxidative stress, leading to plant cell damage and causing typical leaf symptoms.

**Importance**
Ramularia leaf spot can cause extensive damage to the upper leaves in spring and winter barley once crops have finished flowering. This can cause extensive losses in yield and quality. Yield losses in spring barley can be up to 0.6 tonnes per hectare.
**Rhizoctonia stunt**

**Pathogen:** *Thanatephorus cucumeris (Rhizoctonia solani)*

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Hosts**
The disease is known to affect all the major cereal crops and probably most grasses. Barley is much more susceptible to the disease than other cereals.

**Symptoms**
The disease is usually apparent as patches of thin stunted plants, which often show yellowing or purpling with die-back of seedlings in the autumn. Patches may also become visible in early spring as areas of stunted purpled plants. The root systems of plants within these patches are usually poor and branched. Points of brown rotten tissue may be seen at intervals along the length of the root where they appear as constrictions, often giving the root the appearance of a string of sausages. Affected plants remain thin and stunted throughout the season and their maturity is often delayed. Affected crops are usually confined to sandy loam or loamy sands, in areas such as the Brecklands of Norfolk or Suffolk.

**Life cycle**
The disease is soil-borne. The fungus is very common in soils and can survive between susceptible crops as mycelium on dead tissues and other hosts.

**Importance**
The disease is erratic in occurrence and frequently more severe where crops have been established by direct drilling or minimum cultivation. Individual crops may suffer significant losses but in general wheat is not seriously affected. Losses in barley can be much more significant.
Rhizoctonia stunt

Roots of a stunted plant

Root infection weakens seedlings causing patches of poor growth

In autumn and spring sclerotia germinate producing infectious hyphae

Overwinters in soil and on host debris as sclerotia and mycelium

Affected plants often remain stunted

Rhizoctonia stunt life cycle
Rhynchosporium (leaf scald)

Pathogen: *Rhynchosporium commune*

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Hosts**
The disease affects barley, rye, triticale and a number of grasses, particularly ryegrasses. There are specialised forms of the pathogen which are generally restricted in their host range.

**Symptoms**
The fungus causes scald-like lesions on leaves, leaf sheaths and ears. Early symptoms are generally oval lesions which are pale green. As the lesions age they acquire a dark brown margin, the centre of the lesion remaining pale green or pale brown. Lesions often coalesce forming large areas around which leaf yellowing is very common. Infection often occurs in the leaf axil which can cause chlorosis and eventual death of the rest of the leaf.

**Life cycle**
The fungus is seed-borne but the importance of this phase of the disease is not fully understood. The most important source of the disease is probably crop debris from previous crops and volunteers which become infected from the stubble from previous crops.

Scald-like lesions showing dark brown margins and pale centres
Autumn-sown crops can become infected very soon after sowing. The disease spreads mainly by rainsplash although long-distance spread by air-borne spores is also possible.

**Importance**

The disease can be very severe, particularly in the south west and west of the UK where conditions are generally mild and wet. The most serious effect on yield in both winter and spring barley results from attacks that develop between first node detectable and boot-swollen growth stages.

**Rhynchosporium (leaf scald)**

Rain splash causes spread of conidia up plant

Conidia from fungal stromatic tissue is primary source of inoculum

Seed infection

Overwinters as mycelium in seed and on crop debris

Infection causes blotch symptoms

Seedling infection

Rhynchosporium (leaf scald) life cycle
**Hosts**
Mainly wheat, but also occasionally on rye, triticale and some grass species.

**Symptoms**
Symptoms of septoria can be seen very early in the growing season in most years. On young autumn-sown wheat, water-soaked patches which quickly turn brown and necrotic may be evident by early December and throughout the winter on the lowest leaves. These contain the visible black pycnidia which are the most characteristic feature of *Z. tritici*. Pycnidia are particularly common on dead over-wintering leaves of winter wheat. Lesions on the mature plant are brown and are sometimes restricted by veins giving a rectangular appearance. The black pycnidia become more visible in the lesions as the symptoms develop. Lesions may coalesce leading to large areas of necrotic brown tissue.

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*Septoria tritici (septoria leaf blotch)*

Brown, necrotic lesions on young wheat plant showing black pycnidia
**Septoria tritici (septoria leaf blotch)**

**Life cycle**

The disease cycle of *Z. tritici* is similar to that of *S. nodorum*, although *Z. tritici* can go through its life cycle at slightly lower temperatures with 15–20°C as the optimum and requires longer periods of high humidity to initiate infection. The lower leaves of winter-sown crops are normally infected by long distance spread of air-borne ascospores throughout the winter and early spring. In the spring the lower leaves of the most susceptible varieties are infected and have actively sporulating lesions. Most disease spread to upper leaves occurs by rain-splash from the lower-leaves during heavy rainfall. Physical spread can occur without heavy rainfall, particularly when leaves 3 and 4 overlap the upper leaves as they emerge.

**Importance**

This is the most important foliar disease on winter wheat in the UK. Losses of 50% have been reported in severely affected crops. This is largely because of the predominance of susceptible varieties.

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Coalesced lesions giving large areas of necrotic brown tissue

In autumn and spring, crops infected by airborne ascospores

Spread of pycnidiospores up plants by contact and rain splash

Pseudothecia and pycnidia develop within lesions

Overwinters as mycelium, pycnidia and pseudothecia on crop debris, autumn sown crops and volunteers

Ascospores

Perithecia (wind blown)

Pycnidia (rain splash)
Sharp eyespot

Pathogen: Ceratobasidium cereale (Rhizoctonia cerealis)

Hosts
The disease affects wheat, barley, oats, rye and triticale.

Symptoms
Symptoms occur as sharply defined lesions on the outer leaf sheaths. Young lesions have a sharply defined dark margin and frequently have shredding of the epidermis within the lesion. Multiple lesions up the stem can be found up to 30cm from the stem base. Later in the season, lesions on the stem have a pale cream centre with a dark brown, sharply defined edge. Sharp eyespot lesions are often superficial, but severe sharp eyespot is not uncommon and can cause white-heads or lodging.

Life cycle
The fungus over-winters primarily as mycelium on infected stubble with volunteers and some grass weeds also acting as sources of inoculum. The fungus can produce sclerotia which may act as overwintering structures. Infection may occur at any time during the growing season, but the disease is favoured by temperatures of around 9°C. Acid, dry and sandy soils and early sowing favour the disease.

Cool autumn or spring temperatures may result in early infection by the fungus which can lead to severe disease.

Importance
Sharp eyespot is common in the UK although, nationally, it does not usually cause significant yield loss. However, individual crops may suffer significant losses, particularly if the disease is present with take-all. Severe sharp eyespot has been shown to reduce yield by up to 25% but this is very unusual. Annual losses in the UK on average are probably less than 0.5%.
**Sharp eyespot**

Distinct sharp eyespot lesions with sharply defined edge

Mycelial growth infects young plants

Sharply defined lesions high up the stem

Lesions can cause lodging and sometimes white-heads

Overwinters on stubble, autumn-sown crops, volunteers, grass weeds and as a soil saprophyte

Sharp eyespot life cycle
Snow mould (pink snow mould)

Pathogen: Monographella nivalis (Microdochium nivale)

Hosts
Snow mould is mainly a disease of winter barley, although other winter cereals are also occasionally affected.

Symptoms
Symptoms are typically seen after snow melts in the spring. Infected plants often have an extensive covering of white mycelium which spreads on overlapping leaves, causing a matting of leaf tissue. Later, as spores are produced on the mycelial mats, the affected patches assume a pink colouration. The fungus often infects the oldest leaves directly from the soil but eventually the whole plant can be affected. Plants die-off in patches, but good growing conditions in the spring can allow crop recovery where plants have survived infection. In years with prolonged snow cover, the disease can be severe. Large areas of the crop may be killed and re-drilling with spring barley may then be necessary.

Life cycle
Winter-sown crops become infected during the winter months, often under snow cover. The lower leaves of plants touching the soil surface become infected by hyphae growing from perithecia or directly from plant debris in the soil. Affected plants or dead plant material carrying perithecia or mycelial growth are returned to the soil after harvest. Infected seed may also contribute to initial infection of seedlings in the autumn. Spring sown crops are rarely affected.

Importance
Snow mould is commonly recorded but, except in isolated cases, damage is rarely severe. The disease is generally more damaging in parts of Scotland where snow cover is more common.
Snow mould (pink snow mould)

Damaged patch of young seedlings due to snow mould

Primary infections occur on lower leaves and stem base
Conidia and mycelium spread from debris

Perithecia develop in late spring and summer
Seedlings infected in autumn by hyphae from perithecia, ascospores and conidia

Overwinters on crop debris

Snow mould life cycle
Snow rot (grey or speckled snow mould)

Pathogen: Typhula incarnata

- Wheat
- Barley
- Oats
- Rye
- Triticale

Hosts
Snow rot affects only winter cereals, especially winter barley, although infections have been noted in winter wheat.

Symptoms
The fungus infects the oldest leaves first but eventually the whole plant can be affected causing yellowing and wilting. Affected plants usually have abundant red-brown resting structures (sclerotia) 2–3mm in diameter embedded in the lower leaf sheaths and on dead leaf tissue. Plants can be killed, but often good growing conditions in the spring allow crop recovery. Surviving tillers compensate for dead shoots so that yield loss is usually small. In years with prolonged snow cover, the disease can be more severe. Large areas of the crop may be killed and re-drilling with spring barley may then be necessary.

Life cycle
Affected plants produce large numbers of sclerotia which can survive between crops over-winter. The sclerotia germinate and produce spores and/or mycelium which infect emerging crops. Winter-sown crops become infected during the winter months, often under snow cover. Spring-sown crops are rarely affected.

Importance
Snow rot is commonly recorded but, except in isolated cases, damage is rarely severe. The disease is generally more damaging in parts of Scotland where snow cover is more common.
Snow rot (grey or speckled snow mould)

Resting structures (sclerotia) of snow rot on lower leaf sheaths

Snow rot life cycle
**Sooty moulds**

Pathogen: *Alternaria* spp. and *Cladosporium* spp.

- Wheat
- Barley
- Oats
- Rye
- Triticale

### Hosts

These fungi are saprophytes, are not host specific and so can affect all cereals.

### Symptoms

The usual symptoms of sooty moulds are a darkening of the ears before harvest. This is commonly seen when weather conditions are wet, but severe symptoms are often associated with root or stem base diseases which cause premature ripening of the crop. Delays in harvesting in wet weather can lead to severe blackening of ears which can lead to discolouration of the grain.

### Life cycle

The fungi that can cause sooty moulds are very common in the atmosphere and can survive adverse conditions as spores or as mycelium on a wide range of materials. They do not require living host material to survive.

### Importance

The discolouration of ears and grain rarely have any effect on grain yield but the mixing of spores with the grain at harvest can lead to discolouration which will affect marketability, particularly if the grain is planned for milling for flour production.
Sooty moulds

Comparison of untreated and treated plots showing the difference in sooty mould infection

Conidia infect ears post GS 75

Colonisation of prematurely ripe ears or where aphid honeydew present
White-heads often become severely affected by sooty moulds

Airborne conidia post GS 75

Cladosporium

Alternaria

Overwinters on crop debris and vegetation

Sooty moulds life cycle
**Stripe smut**

Pathogen: *Urocystis occulta*

<table>
<thead>
<tr>
<th>Wheat</th>
<th>Barley</th>
<th>Oats</th>
<th>Rye</th>
<th>Triticale</th>
</tr>
</thead>
</table>

**Hosts**
The disease is specific to rye.

**Symptoms**
This disease differs from the other smuts of UK cereals in that it affects not only the ears but also the stems and leaves. It is specific to rye, producing long dark blisters in stripes parallel to the veins which eventually rupture to expose the spores.

**Life cycle**
The disease is both soil-borne and seed-borne. The developing grains in ears are contaminated by wind-blown spores but the spores remain on the seed surface. Infection of the seed occurs at germination as with bunt. Soil-borne inoculum is more important with this disease than with the smuts affecting other cereals in the UK.

**Importance**
The disease is sporadic in occurrence but rarely causes significant loss.
**Stripe smut**

Sori develop between veins on leaves and glumes.

Fungus grows systemically within plant.

In autumn and spring sporidia germinate. Mycelium infects seedlings.

Sori erupt releasing spore balls containing teliospores.

Overwinters as spore balls in soil and on seed.

Teliospores germinate forming promycelium and sporidia.

Infected plants appear yellowish and stunted.

Spore balls germinating teliospore.

**Stripe smut life cycle**
Take-all

Pathogen: *Gaeumannomyces graminis* var. *tritici*
*Gaeumannomyces graminis* var. *avenae*

- Wheat
- Barley
- Oats
- Rye
- Triticale

**Hosts**

*Gaeumannomyces graminis* var. *tritici* attacks wheat, barley, rye and may attack some grass species, particularly couch grass (*Elytrigia repens*). Oats are immune. *Gaeumannomyces graminis* var. *avenae* attacks oats, wheat, barley, rye and many grass species.

**Symptoms**

The take-all fungus attacks the roots of plants as it is soil-borne. If diseased plants are pulled up, the roots can be seen to be blackened and rotten and have a 'rat-tail' appearance. In severe outbreaks the base of infected plants may also show blackening. Above ground symptoms are seen as patches of stunted plants and white-heads ('bleached' ears) in mature plants. White-heads generally contain small grains or, occasionally, no grain at all.

**Life cycle**

The take-all fungus survives the winter as mycelium primarily on roots or stubble debris but also volunteer cereals, early autumn-sown crops and some grass weeds. Primary infection occurs in autumn from inoculum in the soil.

Secondary (root-to-root) infection occurs mostly in spring and summer. The disease spreads from infected seedling roots to developing crown roots. As the disease progresses, the root area lost increases and the ability of the plant to absorb water and nutrients declines. When root rotting is severe plants are unable to absorb water and nutrients. As a result the plants ripen prematurely, resulting in white-heads and often poor grain filling.

**Importance**

Take-all is one of the most important diseases of wheat in the UK, partly because it is not easily controlled chemically or by varietal resistance and relies mainly on rotational strategies for control. Even on chalky boulder clay soil, losses of 10–20% are common in second and third wheat crops. On less well-bodied soils, yield losses can be much higher or indeed it may be impossible to grow second or subsequent wheat. Grain from plants showing white-heads are usually small and shrivelled.
Take-all causes most damage on light soils, particularly if they are alkaline in nature. Severe attacks can also occur in acid patches. Poor drainage and nutrient status also encourage the disease. Take-all is particularly encouraged by early sowing and light, puffy seedbeds.

The disease is usually most severe in second, third or fourth successive cereal crops, but generally declines in importance in continuous cereals (take-all decline).

Typical 'rat-tail' appearance of roots due to take-all

Severe infection results in stunted patches, 'rat tail' appearance and whiteheads

Runner hyphae on roots

Fungus overwinters as mycelium on roots and stem bases of infected plants, spreading to volunteers and autumn-sown crops

White-head

Take-all life cycle
Tan spot (DTR)

Pathogen: *Pyrenophora tritici-repentis* (*Drechslera tritici-repentis*)

- Wheat
- Barley
- Oats
- Rye
- Triticale

Hosts
The disease affects wheat but can also attack barley, rye and some grasses.

Symptoms
*P. tritici-repentis* can be seed-borne and infect seedlings, resulting in small tan to light brown flecks on young leaves. However, symptoms are generally seen later in the season on the middle and upper canopy. Early symptoms appear as small tan to light brown flecks, with a chlorotic halo, often with a dark spot at the centre. Later these develop into light brown oval lesions with slightly darker margins with a light coloured spot at the centre. Under wet conditions the lesions produce spores which can make lesions darker in colour. Under ideal conditions these lesions coalesce to produce large areas of dead tissue.

Life cycle
*P. tritici-repentis* survives mainly as dormant mycelium on stubble and crop debris. This produces pseudothecia on stubble which in turn produce ascospores for long distance spread. Under warm, wet conditions, leaf spots produce dark conidia which are spread up the plant. The disease can infect the ear and cause discolouration of the glumes and the grain.
**Tan spot (DTR)**

Symptoms on the head are indistinct but can cause brownish glumes. Infected grains can have a reddish appearance, similar to *Fusarium* spp. infection. The disease develops over a wide range of temperatures but has quite a high optimum (20–28°C) and is favoured by long periods (18 hours or more) of dew or rain.

**Importance**

Tan spot is very common in Scandinavia and parts of France but is still rare in the UK. Although it is occasionally recorded in disease surveys it rarely causes serious losses. However, in a few isolated cases the disease has caused serious losses on individual crops grown under minimum tillage systems.

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Rain splash moves conidia up plant causing re-infection

Primary infection results from conidia and ascospores

Typical eye-shaped leaf spotting symptoms occur

At end of season pseudothecia develop

Overwinters as seed-borne mycelium and as pseudothecia on crop debris

Tan spot (DTR) life cycle
Yellow (stripe) rust

Pathogen: *Puccinia striiformis*

- Wheat
- Barley
- Oats
- Rye
- Triticale

### Hosts

There are distinct forms of the fungus that are specific to different cereal crops, i.e., *P. striiformis* f.sp. *tritici* attacks wheat whereas *P. striiformis* f.sp. *hordei* can only attack barley. Within the forms of *P. striiformis* there are a number of different races that can only attack certain varieties.

### Symptoms

Yellow rust symptoms appear as parallel rows of yellowish orange coloured pustules on the leaves of adult plants. Epidemics often start on individual plants, usually in the autumn. Symptoms develop slowly over winter and are often missed until the early spring when small patches or foci of infected plants can be seen in fields. Early on, the yellow to orange coloured yellow rust pustules are difficult to distinguish from brown rust. However, yellow rust lesions tend to spread as a yellow band on young leaves moving ahead of the sporulating lesion. On older leaves pustules occur in obvious stripes hence it sometimes being referred to as stripe rust. Severe infections quickly give rise to chlorosis, and later necrosis, of leaves resulting in desiccation in May/June if the weather conditions are warm and dry. In severe attacks yellow rust infection of the ears can occur with the formation of masses of spores between the grain and the glumes. At the end of the season, secondary black spores (teliospores) are sometimes produced amongst the stripes of pustules.

Typical striped symptoms of yellow rust
Yellow (stripe) rust

Life cycle

*P. striiformis* requires living green plant material in order to survive, over-wintering as dormant mycelium or active sporulating lesions on volunteers or early autumn-sown crops. Within plant tissue the fungus can survive at very low temperatures and will usually survive the winter in infected plants. In the spring, particularly in cool moist weather, the fungus starts to grow and produces active sporulating lesions. Temperatures of 10–15°C and a relative humidity of 100% are optimal for spore germination, penetration and production of new, wind-dispersed spores. The fungus is generally inhibited by temperatures over 20°C although strains tolerant of high temperatures do exist. The complete cycle from infection to the production of new spores can take as little as seven days with ideal conditions and may be repeated many times in one season. During late summer, the dark teliospores may be produced. These can germinate to produce basidiospores, however, no alternate host has been found. Although the teliospores seem to have no function in the disease cycle they may contribute to the development of new races through sexual recombination.

Importance

The disease is generally sporadic in the UK occurring more often in the east of the country and in coastal areas which may have cool summer weather accompanied by regular mists. Severe epidemics are usually associated with susceptible varieties, mild winters and cool moist summers. The development of new races of *P. striiformis* can result in varietal resistance being overcome within a short period of time. Yield losses of 40–50% have often been recorded in susceptible varieties.
Yellow (stripe) rust

Disease spread by wind dispersal

Pustules erupt releasing uredospores

Early infection of plants by wind-borne uredospores

Overwinters on volunteer plants

Late in the season black telia form on leaves

Spring

Summer

Autumn

Teliospores produce basidia and basidiospores

Alternate host (Berberis spp.)

Overwinters on volunteer plants

Disease spread by wind dispersal

Pustules erupt releasing uredospores

Early infection of plants by wind-borne uredospores

Overwinters on volunteer plants

Late in the season black telia form on leaves

Teliospores produce basidia and basidiospores

Alternate host (Berberis spp.)
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<td><strong>Ascus (pl. asci)</strong></td>
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<td><strong>Basidiocarp</strong></td>
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<td><strong>Damping-off</strong></td>
<td>Disease of plant seedlings caused by seed- or soil-borne fungi</td>
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<td><strong>Dicotyledon</strong></td>
<td>A flowering plant where the embryo has two cotyledons (seed leaves)</td>
</tr>
<tr>
<td><strong>Dieback</strong></td>
<td>Necrosis of a shoot beginning at the apex and spreading towards the older tissue, stem death may occur</td>
</tr>
<tr>
<td><strong>Direct drilling</strong></td>
<td>The drilling of seed into ground which has received minimal cultivation</td>
</tr>
<tr>
<td><strong>Ear blight</strong></td>
<td>Infection of cereal ears resulting in bleaching of parts of the ear or discolouration of the glumes and grains</td>
</tr>
<tr>
<td><strong>Endosperm</strong></td>
<td>Nutritive tissue in a seed</td>
</tr>
<tr>
<td><strong>Epidemic</strong></td>
<td>A widespread increase in the incidence of an infectious disease</td>
</tr>
<tr>
<td><strong>Epidermis</strong></td>
<td>The outermost layer of cells of an organ, usually only one cell thick</td>
</tr>
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<td><strong>Ergot</strong></td>
<td>The fruiting structure (sclerotium) of Claviceps spp.</td>
</tr>
<tr>
<td><strong>Flag leaf</strong></td>
<td>The final leaf to emerge in a cereal plant</td>
</tr>
<tr>
<td><strong>Focus</strong></td>
<td>A site of local concentration of diseased plants, usually about a primary source of infection or coinciding with an area originally favourable to disease establishment</td>
</tr>
<tr>
<td><strong>Forma specialis</strong></td>
<td>A group within a pathogen species that can only infect particular hosts</td>
</tr>
<tr>
<td><strong>Glume</strong></td>
<td>An outer and lowermost bract of a grass (including cereals) spikelet (inflorescence)</td>
</tr>
<tr>
<td><strong>Honeydew (fungal)</strong></td>
<td>A sticky secretion containing conidia produced during the lifecycle of Claviceps purpurea (Ergot)</td>
</tr>
<tr>
<td><strong>Host</strong></td>
<td>A living organism harbouring a pathogen</td>
</tr>
<tr>
<td><strong>Host specific</strong></td>
<td>Pertaining to a particular host, generally species specific</td>
</tr>
<tr>
<td><strong>Hypha</strong></td>
<td>One of the filaments of a mycelium</td>
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<th>Definition</th>
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<td><strong>Immune</strong></td>
<td>Cannot be infected by a given pathogen</td>
</tr>
<tr>
<td><strong>Inoculum</strong></td>
<td>Micro-organisms or virus particles which act as a source of infection</td>
</tr>
<tr>
<td><strong>Inflorescence</strong></td>
<td>The group or arrangement in which flowers are borne on a plant</td>
</tr>
<tr>
<td><strong>Internode</strong></td>
<td>Part of a plant stem between two successive nodes</td>
</tr>
<tr>
<td><strong>Leaf sheath</strong></td>
<td>The lower part of the leaf which surrounds the stem more or less completely</td>
</tr>
<tr>
<td><strong>Lesion</strong></td>
<td>A localised area of diseased tissue</td>
</tr>
<tr>
<td><strong>Lodging</strong></td>
<td>When a standing crop is caused to lean or bend due to adverse weather or soil conditions</td>
</tr>
<tr>
<td><strong>Minimal cultivation</strong></td>
<td>A reduced form of cultivation</td>
</tr>
<tr>
<td><strong>Morphology</strong></td>
<td>The form and structure of an organism</td>
</tr>
<tr>
<td><strong>Mosaic</strong></td>
<td>A pattern of disease symptoms on a plant apparent as green/yellow or dark/light areas, usually referring to virus infections</td>
</tr>
<tr>
<td><strong>Mycotoxin</strong></td>
<td>A toxin produced by a fungus</td>
</tr>
<tr>
<td><strong>Mycelium</strong></td>
<td>The mass of hyphae forming the body of a fungus</td>
</tr>
<tr>
<td><strong>Necrotroph</strong></td>
<td>Micro-organism feeding only on dead organic tissue</td>
</tr>
<tr>
<td><strong>Node</strong></td>
<td>The level of a stem at which one or more leaves arise</td>
</tr>
<tr>
<td><strong>Pathogen</strong></td>
<td>An organism which causes disease</td>
</tr>
<tr>
<td><strong>Perithecium</strong></td>
<td>An ascocarp shaped like a flask containing asci</td>
</tr>
<tr>
<td><strong>Primary inoculum</strong></td>
<td>Spores or fragments of mycelium capable of initiating disease</td>
</tr>
<tr>
<td><strong>Pseudothecia</strong></td>
<td>A perithecium-like structure with a single cavity containing ascospores</td>
</tr>
<tr>
<td><strong>Pustule</strong></td>
<td>A spore mass developing below the epidermis and then breaking through at maturity</td>
</tr>
<tr>
<td><strong>Pycnidium</strong></td>
<td>Flask shaped fruiting body with an apical pore lined internally with pycnidiospores</td>
</tr>
<tr>
<td><strong>Pycnidiospores</strong></td>
<td>Spores from within a pycnidium</td>
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<td>-----------------------</td>
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<tr>
<td>Rachis</td>
<td>The main axis of the inflorescence, or spike, of wheat and other cereals, to which the spikelets are attached</td>
</tr>
<tr>
<td>Resistance</td>
<td>The inherent capacity of a host plant to prevent or reduce the development of a disease</td>
</tr>
<tr>
<td>Saprophyte</td>
<td>A nan organism deriving its nutrients from dead or decaying tissue of another organism</td>
</tr>
<tr>
<td>Sclerotia</td>
<td>Compact mass of fungal hyphae e.g. ergot, capable of being dormant for long periods, and giving rise to fruiting bodies or mycelium</td>
</tr>
<tr>
<td>Seminal roots</td>
<td>The roots developing directly from the seed</td>
</tr>
<tr>
<td>Senescence</td>
<td>The dying process of a plant or plant part</td>
</tr>
<tr>
<td>Sporangiophore</td>
<td>A hypha or fruiting structure bearing spores</td>
</tr>
<tr>
<td>Sporangium</td>
<td>A container or case of asexual spores. In some cases it functions as a single spore</td>
</tr>
<tr>
<td>Spore</td>
<td>A reproductive unit in fungi</td>
</tr>
<tr>
<td>Sporulation</td>
<td>The period of active spore production</td>
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<tr>
<td>Susceptible</td>
<td>A group within a pathogen species that can only infect particular hosts</td>
</tr>
<tr>
<td>Telium</td>
<td>Structure containing teliospores</td>
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<tr>
<td>Teliospores</td>
<td>Sexual spores produced within a telium</td>
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<tr>
<td>Teleomorph</td>
<td>The sexual or so-called perfect growth stage or phase in fungi</td>
</tr>
<tr>
<td>Tolerance</td>
<td>The ability of a plant host to sustain the effects of a disease without dying or suffering serious injury or crop loss</td>
</tr>
<tr>
<td>Uredium</td>
<td>The fruiting structure of a rust fungi in which uredospores are produced</td>
</tr>
<tr>
<td>Uredospore</td>
<td>The asexual spore of the rust fungus</td>
</tr>
<tr>
<td>Vector</td>
<td>An organism capable of transmitting inoculum</td>
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<tr>
<td>Virulence</td>
<td>The ability of a pathogen to produce disease</td>
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<tr>
<td>White-head</td>
<td>Prematurely ripened ears of cereals often caused by pathogens attacking the roots or stem base</td>
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