This updated Wheat disease management guide brings together the latest information on controlling economically important wheat diseases. Foliar, stem-base, root and ear diseases are covered.

Reliance on repeated fungicide sprays has increased the pressure for development of fungicide resistance. Some important mode of action groups have lost activity against major diseases. The guide describes how to account for varietal resistance when making spray decisions.

Most energy, greenhouse gas and economic costs are invested in crop establishment and nitrogen application, in order to grow green canopy. It is important to prevent diseases destroying green area before it can repay the investment during grain filling.

Seed production and certification

Certified seed

All seed bought and sold in the UK must be certified. Wheat quality standards (including varietal and species purity, germination, loose smut and ergot) are prescribed in Cereal Seed Regulations issued by the UK within the EU-wide framework.

EU member countries can prescribe stricter standards than the EU minimum. The UK sets a Higher Voluntary Standard (HVS) with higher standards for varietal and species purity, ergot and loose smut. HVS seed is sold at a premium.

Seed can be certified at various stages as a variety is commercialised. Second generation certified seed (C2) is the category normally bought for commercial production.

Certification and seed-borne disease

The Cereal Seed Regulations state: “Harmful organisms which reduce the usefulness of the seed shall be at the lowest possible level.” Standards exist for loose smut, currently rare in UK wheat, and ergot but there are no standards for bunt or fungal seedling blights. Although not a requirement, most certified seed is treated. The diseases controlled depend on the treatment.

Other related HGCA information

HGCA disease management guides are updated annually with the latest fungicide information and are available online at www.hgca.com/publications


CropMonitor – Get in-season information on emerging disease threats from www.cropmonitor.co.uk

For HGCA mycotoxin risk assessment tools and information, visit www.hgca.com/mycotoxins

For the Recommended List and the latest HGCA variety information, visit www.hgca.com/varieties

RL Plus – This web-based tool allows you to interrogate HGCA variety data to suit your specific cropping requirements. www.hgca.com/varieties/rl-plus

Naming of fungal diseases

Latin names of pathogenic fungi are agreed by international convention and these can change over time as new scientific evidence emerges (e.g. Septoria tritici is now Mycosphaerella graminicola). However, in some cases the original scientific names have become widely used to describe the diseases they cause (e.g. septoria tritici or septoria leaf blotch) – distinguished from Latin names by not being in italics. In general the most widely used common names are given in this guide.
Farm-saved seed
Quality seed can be grown and processed on farm. The aim should be to meet at least the minimum certified seed standards.

Note: by law farm-saved seed cannot be sold, shared or bartered.

Any use of farm-saved seed must be declared to the British Society of Plant Breeders (BSPB). Most varieties are eligible for farm-saved seed payment; the list is available at www.bspb.co.uk. This must be paid via a registered processor or directly to BSPB. Payments for previously zero-rated varieties will be refunded immediately but declarations of these varieties are subject to verification by BSPB.

Organic seed production
Organic certified seed must meet the same quality standards as conventionally produced seed. No conventional seed treatments should be used on organic certified or farm-saved seed. All seed considered for organic production should be tested for germination and seed-borne diseases.

<table>
<thead>
<tr>
<th>Most recent name</th>
<th>Previous name</th>
<th>Commonly called</th>
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<tbody>
<tr>
<td>Mycosphaerella graminicola</td>
<td>Septoria tritici</td>
<td>Septoria tritici</td>
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<tr>
<td>Stagonospora nodorum or</td>
<td>Septoria nodorum</td>
<td>Septoria nodorum</td>
</tr>
<tr>
<td>Phaeosphaeria nodorum</td>
<td></td>
<td></td>
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<td>Blumeria graminis</td>
<td>Erysiphe graminis</td>
<td>Powdery mildew</td>
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<tr>
<td>Puccinia triticina</td>
<td>Puccinia recondita</td>
<td>Brown rust</td>
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<td>Stem-base diseases</td>
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Seed sampling and testing

By law, seed must be officially sampled and tested before it can be certified. Sampling and testing are also important for grain intended for farm-saved seed. The value of any seed test is limited by the sampling methodology used. It is vital to collect a representative sample of grain.

Sampling
- Sample grain before cleaning or drying, ideally with a single or multi-chamber stick sampler
- Wash equipment with water and detergent, before and between lots
- Keep grain intended for sowing separate from larger grain bulks
- Only use seed from one field to reduce variability within a seed lot
- Subdivide seed lots over 30 tonnes into smaller lots
- Sample across the bulk or trailer at different depths (each sample taken is a primary sample)
- Thoroughly mix all primary samples in a clean bucket to create a ‘composite sample’ and divide for testing

Primary samples required for given lot sizes

<table>
<thead>
<tr>
<th>Lot size (tonnes)</th>
<th>Primary samples required</th>
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</thead>
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<tr>
<td>&lt;5*</td>
<td>10</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>20–30</td>
<td>40</td>
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</tbody>
</table>

* Seed treatment should only be used where necessary but with small seed lots it may be cheaper to treat than to sample and test for seed-borne diseases.

Figure 1. Preparing samples for testing

Step 1. Check sample size required for tests. 300-500g is normally sufficient for germination and disease.

Step 2. Thoroughly mix all primary samples in a clean bucket to create a ‘composite sample’.

Step 3. Pour onto a clean plastic sheet, divide into two equal portions.

Step 4. Halve each portion and halve again to create eight equal portions. Arrange these in two rows of four.

Step 5. Take first and third sample in one row and second and fourth in other row and combine.

Step 6. Weigh combined sample. If more seed is required, mix remaining samples. Repeat steps 3 to 5 until required sample weight is obtained.
Cleanliness and hygiene
Bunt spores can contaminate equipment and storage areas and diseased seed lots can contaminate healthy lots. Wash all sampling equipment prior to starting sampling and between lots using water, detergent and a brush. Dry equipment or allow it to dry before use.

Equipment
The single chamber sampler (or “deep bin probe” or “Neate sampler”) collects one primary sample at a time. Screw-on extensions can be used if the depth of grain in the bulk is greater than the length of the sampler.

The multi-chamber sampler usually has three or more chambers; all seed collected in this one sampling action equals one primary sample. It can be used to sample grain up to two metres deep and is suitable for most trailers. A piece of plastic guttering is useful for collecting samples from this type of sampler.

If you do not have access to appropriate equipment, have your grain sampled by a trained agronomist.

Germination testing
Low germination, due to disease, sprouting, drying, mechanical or chemical damage, is a major cause of poor quality in UK seed. Where time is limited, the tetrazolium (TZ) test is recommended. This does not detect low germination caused by disease damage but gives a good indication of potential germination after treatment for seedling blights.

Seed health testing
- Never sow untreated seed without testing for seed-borne diseases, particularly bunt and microdochium seedling blight.
- Test for ergot, loose smut, septoria and fusarium seedling blight if a problem is suspected.

Regulatory standards and advisory thresholds

<table>
<thead>
<tr>
<th>Disease</th>
<th>Method</th>
<th>Duration</th>
<th>Results given as</th>
<th>Regulatory S standard Advisory threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bunt</td>
<td>Wash</td>
<td>48 hours</td>
<td>Spores per seed</td>
<td>A Treat if 1 spore/seed or more</td>
</tr>
<tr>
<td></td>
<td>Molecular test</td>
<td>48 hours</td>
<td>Either over or under 1 spore/seed</td>
<td></td>
</tr>
<tr>
<td>Microdochium seedling blight</td>
<td>Agar plate</td>
<td>7-10 days</td>
<td>% infection</td>
<td>A Treat if over 10% infection</td>
</tr>
<tr>
<td></td>
<td>Molecular</td>
<td>48-72 hours</td>
<td>Either over or under 10% infection</td>
<td></td>
</tr>
<tr>
<td>Septoria and fusarium seedling blights</td>
<td>Agar plate</td>
<td>7-10 days</td>
<td>% infection</td>
<td>A Treat if over 10% infection</td>
</tr>
<tr>
<td>Ergot</td>
<td>Visual 500g or 1000g search</td>
<td>24 hours</td>
<td>Number of pieces in 500g or 1000g</td>
<td>Maximum pieces: A S 3 pieces/500g – minimum standard S 1 piece/1000g – HVS</td>
</tr>
<tr>
<td>Loose smut</td>
<td>Embryo extraction</td>
<td>48 hours</td>
<td>% infection in 1000 embryos (advisory) or 2000 embryos (certification)</td>
<td>Maximum infection: A S 0.5% – minimum standard S 0.2% – HVS</td>
</tr>
</tbody>
</table>

Treat for seedling blights when sum of infection levels exceeds 10%.

At present seedling blight caused by *Cochliobolus sativus* is a low risk in UK wheat.
Seed-borne diseases

Bunt  
– *Tilletia tritici*

Symptoms
Symptoms appear after ears emerge. Plants are often stunted and sometimes have yellow streaks along the flag leaf. Infected ears are dark grey-green with slightly gaping glumes. Bunt balls replace all grains and, if broken, release millions of black spores smelling of rotten fish.

Importance
Bunt occurs at low levels in some seed stocks. Contaminated grain may cause rejection.

Life cycle
Spores on the seed surface germinate with seeds. After invading shoots and growing points, the fungus grows within the plant until ear emergence when bunt balls replace grain. The spores contaminate healthy grain during harvest, transport and storage. Spores can land on soil or spread by wind to neighbouring fields. Soil-borne spores can invade seedlings very early in germination.

Risk factors
Seed-borne infection:
– Seed repeatedly sown without a fungicide treatment
– Seedbed conditions leading to slow emergence

Soil-borne infection:
– Very short time between harvesting first wheat and sowing second wheat
– Dry soil conditions between harvesting and sowing

Septoria seedling blight  
– *Phaeosphaeria nodorum*

Symptoms
The most common effect is poor plant establishment. Symptoms are so similar to those of microdochium seedling blight that only laboratory analysis can distinguish them. Septoria nodorum is more commonly associated with necrotic blotching of leaves and glumes.

Importance
Effects of septoria seedling blight are usually less severe than microdochium seedling blight. However, at high levels crop establishment can be badly affected.

Life cycle
While the disease can survive on plant debris, most infections result from seed-borne inoculum.

Risk factors
– High seed infection levels
– Untreated seed sown into poor seedbeds
– Cool, wet soils

Microdochium seedling blight  
– *Microdochium nivale* and *M. majus*

Symptoms
The most common symptom of a serious attack is poor establishment. The fungus can also cause root rotting, brown foot rot, leaf blotch and, in combination with fusarium species, ear blight.

Importance
In most years microdochium seedling blight occurs on wheat seed and is the most important cause of seedling blight in the UK. Sowing untreated seed with high levels of infection causes very poor crop establishment leading to yield loss.

Life cycle
Inoculum (spores) are found in soil and on infected seed. Spores, released when seedling blight or stem-base browning occurs, are splashed up the plant and ultimately infect the ear.

Risk factors
– Wet weather during flowering
– High level of seed infection
– Untreated seed sown into poor seedbeds
– Late-sown crops
Fusarium seedling blight
– Fusarium graminearum

Symptoms
Poor plant establishment is the most common effect together with root rotting, brown foot rot and ear blight.

Importance
At present Fusarium graminearum is the only fusarium species that causes significant seedling losses in the UK.

Life cycle
Inoculum occurs mainly on crop debris, but can be seed-borne. Spores are splashed up the plant to infect ears.

Risk factors
– High levels of seed infection
– Sowing untreated seed into poor seedbeds
– Maize in the rotation

Ergot
– Claviceps purpurea

Symptoms
A hard, purple-black sclerotium, up to 2cm long, replaces some grains in the ear.

Importance
Yield is hardly affected but ergot is highly poisonous to humans and animals, so contaminated grain will be rejected or require cleaning. Standards for number of ergot pieces exist for certified seed.

Life cycle
At or near harvest, ergots fall to the ground or are spread with contaminated seed. They remain dormant until the following summer, when they germinate and produce spores, encased in sticky ‘honeydew’. Spores spread by wind to open grass and cereal flowers nearby. Rain splash or insects carry spores to other flowers, leading to further infection.

Control
No fungicide is effective against ergot. In the absence of host crops, ergots decay over 12 months. Check weed grasses and field margins for ergot. Consider ploughing between host crops and break crops. Control cereal volunteers and grass weeds.

Risk factors
– Cool, moist conditions during flowering
– Infected neighbouring crops
– Seed repeatedly sown without treatment

Loose smut
– Ustilago nuda f. sp. tritici

Symptoms
The ear is usually completely replaced by black fungal spores. Sometimes ears are partly affected. Spores are released as soon as the ear emerges leaving a bare ear rachis with total grain loss. Blackened ears are so obvious that very low incidence appears severe.

Importance
Seed certification and resistant varieties have minimised seed-borne infection.

Life cycle
The fungus is present inside the embryo. As seed germinates the fungus grows within the plant and infects the ear at an early stage. Eventually, spikelets are replaced with masses of fungal spores which are released at ear emergence. Spores spread by wind to nearby open flowers and infect developing grain sites on healthy plants.

Risk factors
– Cool, moist conditions during flowering
– Infected neighbouring crops
– Seed repeatedly sown without treatment

Seedling blight, foot rot and leaf spot
– Cochliobolus sativus

Symptoms
Early symptoms include brown roots and coleoptiles. Infected plants with brown spotting on lower leaves usually grow to maturity. Severe infections cause stem-base rotting and poorly filled ears.

Importance
Cochliobolus sativus is traditionally a disease of hotter climates. Symptons only occasionally occur in the UK.

Life cycle
The soil and seed-borne fungus survives on crop debris and grass weeds. It sometimes causes seedling blight. More usually it infects roots but the plant survives. Splash-borne spores infect seed in ears.

Risk factors
– Any factor that slows germination and emergence, eg poor seedbeds
– Extended periods of warm, moist weather
Seed treatment

Certified seed

Test seed for bunt and microdochium seedling blight
Test for septoria seedling blight if septoria nodorum is present in the growing crop

Does bunt exceed one spore/seed?

NO

Do combined seedling diseases exceed 10%?

YES

Very low risk of seed-borne disease, consider sowing seed untreated

Farm-saved seed

Germination test

85% plus

Considering sowing untreated?

YES

NO

78–84%

Is heat damage present?

NO

YES

Under 78%

Do not use for seed

Treat seed with an appropriate product (see page 9)

85% plus

Considering sowing untreated?

YES

NO

78–84%

Is heat damage present?

NO

YES

Under 78%

Do not use for seed

Treat seed with an appropriate product (see page 9)
## Seed treatment options

<table>
<thead>
<tr>
<th>Active ingredient, ingredient</th>
<th>Product</th>
<th>Seed-borne diseases</th>
<th>Other diseases</th>
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<tbody>
<tr>
<td></td>
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<td>Bunt</td>
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<td>carboxin, thiram</td>
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<tr>
<td>silthiofam</td>
<td>Latitude</td>
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**Key to company:**  
Bs=BASF  By=Bayer CropScience  Ch=Chemtura  Mo=Monsanto  MA=Makhteshim-Agan  Sy=Syngenta

- ✓ label recommendation  
- some known activity, but no label recommendation

Updated annually, see [www.hgca.com/diseasecontrol](http://www.hgca.com/diseasecontrol)
Foliar diseases

Impact on yield formation
Most foliar diseases accelerate senescence of the top three leaves and so reduce yield. Fungicide sprays during canopy growth prevent green leaf area loss during grain filling.

Construction phase
Canopy growth: Canopy expansion accelerates in April/May as temperatures rise and large upper leaves emerge. The maximum green area of leaves and stems (measured as green area index – GAI) is reached as ears emerge, just before grain filling begins.

Stem reserve accumulation: During stem extension, stored soluble carbohydrates accumulate in the stem.

Applying sprays during this critical phase – at T1 (GS32) and T2 (GS39) – limits disease progress and protects emerging upper leaves, so maximising photosynthesis later.

Production phase
Grain filling: In this six to seven week period, up to 80% of yield comes from photosynthesis. On bright days, yield typically increases by 0.2t/ha/day. In this phase, stem reserve relocation accounts for 20% of grain filling.

A fungicide at full ear emergence may help prevent premature leaf loss.

Flag leaf and ear contribute 65% of total yield

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Infection and development

Initial infection
Infection usually results from spores moving into the crop. When this occurs depends on the disease. For example, by spring, septoria tritici is present on the lower leaves of most crops.

Disease development
Infection is followed by a ‘latent period’ when the fungus grows within the leaf but the leaf exhibits no symptoms.

The cycle of leaf emergence, infection, latent period and symptom expression applies to all foliar diseases. The latent period varies considerably between pathogens and is affected by temperature. At higher temperatures, latent periods are shorter.

Septoria tritici has a very long latent period (14–28 days). Many modern fungicides can control disease after a leaf becomes infected but only for about half of the latent period. In the summer, septoria tritici may have a latent period of 14 days, but fungicides provide eradicant control for only about seven days. Although there may be no symptoms on a leaf, infection may be so far into the latent period that no fungicide at any dose will control the fungus.

Infection from within crops
As stems extend and upper leaves emerge, the crop tends to grow away from the disease. Newly emerged leaves always appear free from septoria tritici between GS32 and GS39.

However, the crucial final three leaves are at risk as soon as they emerge. By this stage, most inoculum comes from within the crop and spore movement from other fields is much less important. Rusts and powdery mildew have very short latent periods and can be found before leaves have fully expanded.

In the absence of fungicide use, the final severity of disease is determined by variety and weather.

Importance of spray timing and latent period

Latent periods, fungicide activity and spray timing
Example based on septoria tritici

GS32
Leaf 3 just emerged
First spores arrive
Leaf 3

GS33
Leaf 2 just emerged
First spores arrive
Leaf 2

GS39
Flag leaf emerged
Latent infection (no symptoms)
Leaf 1

GS59
Ear emerged
First lesion
Leaf 1

Leaf 2
First lesion
10–14 days

Leaf 3
Latent infection (no symptoms)

Leaf 4 and below already showing symptoms. Disease on these leaves cannot now be controlled.

Leaf 3 infected but disease still in latent period and controllable.

Leaf 2 just infected.

Leaf 3 can be protected.

Leaf 2 may now be showing symptoms – disease not now controllable on this leaf layer.

Symptoms now visible on leaves 2 and 3, not controllable by ear sprays. Ear sprays top up protectant activity on the flag leaf.
Foliar diseases

Spray timing

Foliar treatments
To ensure adequate protection of the key yield-forming leaves, fungicide treatments should be targeted to leaf emergence, rather than growth stage. Full emergence of leaf 3 usually coincides with GS32 but this can vary between crops. Very early sowing can lead to leaf 3 emerging as late as GS33 but leaf 3 may emerge at GS31 in late-sown crops. Leaf emergence is, therefore, the best guide for decisions on spray timings. With practice, this can be assessed quickly in the field. Growth stages provide a second best option.

Spray window
The ‘spray window’ for effective disease control on a particular leaf layer is relatively narrow. The optimum spray timing is when a leaf has just fully emerged.
- Spraying too early, when the leaf is not fully emerged, results in insufficient spray on the leaf and poor control
- Spraying too late means the disease is already established and results in poor control, especially with protectant fungicides

Main timings
T1 timing – Leaf 3 emerged (usually coincides with GS32, but can be as early as GS31 or as late as GS33).
T2 timing – Flag leaf emerged (GS39).
Applying both T1 and T2 sprays at optimal timings gives effective disease control on the top three leaves – those most important in grain filling.

Additional timings
T0 timing – Usually two to four weeks earlier than T1.
Instances where a T0 spray may be considered include:
- To delay septoria tritici development
- Where mildew, yellow or brown rusts are active
- When eyespot requires earlier treatment
T3 timing – Ear spray
May be used to control ear diseases and ‘top up’ foliar disease control on the flag leaf on susceptible varieties under high disease pressure.

Effects of spray timing on disease control
The optimum T1 spray gives maximum disease control on leaf 3, and provides some protection for leaf 2.

The optimum T2 spray gives maximum disease control on the flag leaf and eradicates any latent infections on leaf 2 that have escaped earlier sprays.

These graphs were produced from trials. Sprays were applied at frequent intervals across a range of growth stages. Each curve shows the level of control achieved on each of the top three leaves for the spray timings from GS31 to GS59. The interval between T1 and T2 is important. Disease control on upper leaves will be lost if the interval extends beyond three weeks, especially when diseases with short life cycles, eg rusts or powdery mildew, are active.
Foliar diseases – Septoria tritici

**Septoria tritici**
*Mycosphaerella graminicola*
sometimes known as leaf blotch

**Symptoms**
In autumn and winter, brown oval leaf spots (lesions) which contain the diagnostic small black fruiting bodies (pycnidia) occur on older leaves. Several lesions may turn large areas of leaf brown. In spring and summer, lesions are usually rectangular and confined by leaf veins. Leaf lesions are often surrounded by areas of leaf yellowing or death. During rapid development, water-soaked lesions gradually turning brown may be present.

**Importance**
Septoria tritici is the most damaging foliar disease of UK wheat, causing significant yield loss every year. Infection occurs in all crops. Unusually dry weather throughout May and June may reduce losses. Higher rainfall areas in the south and west are most at risk.

CropMonitor national survey data showed that septoria tritici was the most common foliar disease in 2010/2011, affecting 56% of crops. Crops had an average of 1% of the area of leaf 2 affected at GS75, continuing a downward trend in severity of the disease.

**Life cycle**
Airborne spores disperse in autumn/winter from previous wheat stubbles. These ascospores infect leaves to produce leaf spots from mid-autumn onwards and then spread by rain splash and physical contact between leaves.

Heavy rainfall encourages rapid spore movement from lower to upper leaves during stem extension. Optimum temperatures are 15–20°C.

Symptoms appear after a latent period (14–28 days after infection); this period reduces as temperatures rise.

**Risk factors**
- Susceptible varieties
- Early sowing
- Rainfall: high-risk septoria periods occur during ‘splashy’ or prolonged rain, especially in May and early June

Mild winters and wet, windy conditions in early spring increase risk.

**Control**

**Varieties**
The HGCA Recommended List 2012/13 contains varieties with resistance ratings from 4 to 7 so a fungicide treatment is likely to be necessary in most instances. However, a lower dose is required for effective control on more resistant varieties.

**Cultural**
Avoid very early sowing of susceptible varieties.

**Fungicides**
Control relies on using robust rates ofazole fungicides at T1 and T2, in mixture with a fungicide of a different mode of action; usually an SDHI (bixafen, isopyrazam, fluxapyroxad or boscalid) and/or chlorothalonil.

Resistance to strobilurin products is widespread and they are unlikely to be effective.

Some systemic seed treatments (eg fluquinconazole) may give limited early control.

For more information, see Topic Sheet 113.
www.hgca.com/publications
**Septoria nodorum**  
*Phaeosphaeria (Stagonospora) nodorum*  
Sometimes known as leaf and glume blotch

**Life cycle**

The pathogen survives in crop residues, volunteers and wild grasses. It can be seed-borne. Airborne ascospores from wheat stubbles spread infection to newly-emerged crops. Secondary spread occurs when pycnidiospores, produced within leaf spots, are dispersed by rain splash.

Symptoms appear within 7–14 days. The disease can develop very rapidly in warm temperatures (20–27°C) with long periods (6–16 hours) of high humidity.

**Symptoms**

On leaves, symptoms are mainly oval brown lesions with a small yellowish halo. Pale brown, rather than black, pycnidia distinguish septoria nodorum from septoria tritici. The indistinct brown pycnidia may be only visible when lesions are held up to the light. Under high disease pressure, leaf symptoms can include small purplish-brown spots.

On ears, symptoms are typically purplish-brown blotches on glumes.

**Importance**

Septoria nodorum was not recorded in the 2010/2011 CropMonitor national survey. When severe attacks occur, it is usually in association with high rainfall at ear emergence (eg in the south west). Here yield losses in untreated crops may exceed 50%.

**Risk factors**

- Susceptible varieties
- High rainfall during and after ear emergence
- South-west and coastal locations

**Control**

**Varieties**

The HGCA Recommended List 2012/13 contains varieties with resistance ratings from 5 to 8. Resistant varieties are preferred in high-risk areas.

**Cultural**

Ploughing or cultivation to bury crop residues after harvest may provide some control.

**Fungicides**

T1 and T2 sprays applied to control other diseases usually control septoria nodorum on the leaves. When risk is high, a T3 protectant ear spray is important.
### Yellow rust

*Puccinia striiformis*

**Symptoms**
Groups of yellow pustules forming stripes between veins are the main symptoms. Under hot, dry conditions – or after fungicide use – pustules may be difficult to detect.

In spring, small patches (foci) of leaves with scattered yellow pustules precede rapid spread throughout the field.

Ears can also be affected.

**Importance**
New races developed in 2009 and 2011. Full implications for varietal resistance are still being determined.

Severe epidemics can occur where large areas of susceptible varieties are grown. In untreated susceptible varieties, yellow rust can reduce yields by over 50%. However, well-timed fungicide sprays are usually very effective so annual losses are small. Outbreaks often occur in coastal areas from Essex to the Borders and central England.

**Life cycle**
Epidemics are associated with mild winters that enable the pathogen to overwinter in crops and volunteers. Cold winters with severe frosts restrict its survival.

In early spring, distinct foci may occur; secondary spread is through airborne spores as well as leaf-to-leaf contact. Cool (10–15°C), damp weather, with overnight dew or rain, provides optimum conditions for disease development.

UK weather conditions are unlikely to limit development in spring or early summer. Symptoms appear 7–14 days after infection so leaf tips may show symptoms before leaves fully emerge. Hot, dry weather with temperatures over 25°C limit development.

**Risk factors**
- Large variation between years in epidemic risk
- Overwinter survival is critical
- Problems occur after mild winters and on susceptible varieties
- Cold winters with several frosts below -5°C reduce survival
- New races continue to develop that overcome the major gene resistance of some new varieties

**Control**

**Varieties**
Varieties with resistance ratings of 7 and 8 on the HGCA Recommended List give effective control and may be less prone to sudden loss of resistance than varieties with a rating of 9. The wheat yellow rust diversification scheme is designed to help growers choose combinations of varieties in such a way as to reduce the risk of crop loss due to disease spreading from one variety to another (see Topic Sheet 112).

**Cultural**
Control volunteers that provide a ‘green bridge’ between harvest and emergence of new crops.

**Fungicides**
Azole and most strobilurin products are very effective; some SHDIs also have good activity. Systemic seed treatments (e.g. fluquinconazole or triadimenol) may delay epidemic development where risk is high.

For more information, see Topic Sheet 112. www.hgca.com/publications
Brown rust

Symptoms
Most commonly seen as tiny orange-brown pustules scattered over leaves. During autumn and winter, a few pustules, confined to older leaves, may be seen. While the pustules can be a similar colour to those of yellow rust, they usually have a chlorotic halo.

Late in the season, brown rust can become very severe and cause leaf death. Leaf sheaths and ears are also affected. Black spots occur on maturing crops when pustules produce a second, teliospore stage.

Importance
There is large seasonal and geographic variation in brown rust severity. The disease is more common in southern and eastern England on susceptible varieties.

Brown rust had been absent in the 2009/2010 CropMonitor national survey but was seen in 2011 at its greatest incidence since 2008, with nearly 8% of crops affected. In 2007, a season when brown rust was prevalent, the estimated cost of yield loss was £11.2 million.

Life cycle
Brown rust overwinters in crops and on volunteers. It spreads by airborne spores. Cold winters may reduce its survival.

Optimum conditions are days with high temperatures (15–22°C) followed by overnight dews. Surface moisture on leaves is essential for spore germination.

Symptoms can appear in 5–6 days at optimum temperatures. The disease is active over a wider range of temperature (7–25°C) than yellow rust.

Risk factors
- Seasonal weather – the disease is normally most active when June and July temperatures are high
- In 2007, above average temperatures led to unusually early epidemics; with higher than average temperatures in March 2012, a similar situation may arise
- High humidity is necessary for epidemic progress
- Susceptible varieties
- Early sowing
- New races continue to develop that overcome the major gene resistance of some new varieties

Control

Varieties
Resistant varieties provide good control. Varieties on the HGCA Recommended List 2012/13 have ratings from 3-9. In 2011, new races of brown rust appeared. Full implications for varietal resistance are being determined. Most current UK wheat varieties are very, or moderately, susceptible, but some have good resistance.

Cultural
Control volunteers that provide a ‘green bridge’ between harvest and emergence of new crops. Susceptible varieties should not be sown early in September.

Fungicides
Products containing azoles, strobilurins and SDHIs are very effective. In a programme, treatment intervals should not exceed three to four weeks; they should be substantially shorter under high disease pressure.

Systemic seed treatments (eg fluquinconazole) may help delay epidemics developing where risk is high. However, seed treatments are likely to provide less control of brown rust than yellow rust.

HGCA Wheat disease management guide 2012
Tan spot
*Pyrenophora (Drechslera) tritici-repentis*

Symptoms
Tan spot symptoms are variable. Small tan, or brown, flecks develop into pale-brown oval spots with dark centres. These lesions sometimes have a chlorotic halo. Numerous lesions can coalesce into large necrotic areas. Symptoms are very similar to those of septoria nodorum and diagnosis relies on spore identification.

Importance
Tan spot is still a minor UK disease but appears to be becoming more common. The 2010/2011 CropMonitor national survey showed tan spot to be the third most commonly occurring disease for the third successive year, with over 20% of crops affected. Some severe cases have occurred in recent years and it is a major problem in Sweden, Denmark, Germany and France.

Life cycle
Like septoria nodorum, tan spot is trash-borne and favoured by minimum tillage. Ascospores produced on stubble, probably in spring, introduce the disease into crops. Leaf lesions appear in 7–14 days and produce splash-dispersed asexual spores.

High temperatures (20–28°C) and rain causing long periods of leaf wetness are ideal for tan spot development.

Risk factors
- Minimum and non-inversion tillage
- Long periods of wet weather from GS32 onwards

Control
Varieties
Resistance ratings are not currently available on the HGCA Recommended List.

Cultural
Ploughing or cultivation to bury infected crop residues.

Fungicides
Fungicidal control is difficult due to the short latent period but older azoles used in the spring to protect crops from rust (eg tebuconazole, cyproconazole and propiconazole) will help to protect crops from early infection.

Strobilurin resistance in tan spot has been confirmed elsewhere in Europe and is likely to be present in the UK.
Powdery mildew
*Blumeria graminis*

**Symptoms**
White fluffy colonies of pustules often occur on leaves from autumn onwards.

On yellowing leaves, pustules retain a distinctive ‘green island’. Severe mildew can cover almost the entire leaf surface and develop on ears and stems.

Later in the season, pinhead-sized, black fruiting bodies (cleistothecia) that produce sexual spores (ascospores) may be found on the white colonies.

**Importance**
Mildew can develop over a wide range of conditions but is sporadic, often affecting crops under stress. Although very visible, it generally reduces yield much less than other foliar diseases. Damaging attacks can occur anywhere in the UK but yield losses rarely exceed 10%.

The 2010/2011 CropMonitor national survey showed that mildew levels remained similar to those recorded in previous years and the long-term mean, with an average of 0.1% mildew on leaf 2 at GS75 and more than 30% of crops affected.

**Life cycle**
Airborne conidia, produced on crops or volunteers, enable mildew to spread widely. Warm (15–22°C), breezy conditions with short periods of high humidity favour infection. New pustules are produced in 5–14 days.

Temperatures over 25°C and rain can inhibit development. Sexually-produced spores provide a mechanism for summer survival when leaf growth has slowed.

**Risk factors**
- Susceptible variety
- Sheltered fertile sites
- High nitrogen
- Warm dry, but humid, weather

**Control**
**Varieties**
The HGCA Recommended List 2012/13 contains varieties with resistance ratings from 3 to 9.

**Cultural**
Avoid excessive nitrogen fertilisation.

**Fungicides**
Fungicides with specific activity against mildew are required where powdery mildew is a particular threat. Some treatments, applied at T0 or T1, provide long-term protection. Fungicide resistance is known to affect the performance of various fungicide groups (eg strobilurins, azoles, morpholines).
**Take-all**

*Gaeumannomyces graminis var. tritici*

### Symptoms
Take-all can infect plants at a low level without causing obvious symptoms. However, moderate or severe infection reduces the number of active roots over winter, which restricts canopy growth. Infection of the crown (adventitious) roots in the spring and early summer restricts water and nutrient uptake, resulting in patches of whiteheads (bleached ears) as grain fills.

### Importance
Take-all is the major cause of ‘second wheat syndrome’ when yields of second wheat crops are frequently 10–15% less than those of first wheats.

Take-all is usually most severe in the second to fourth successive cereal crop but yields generally recover to some extent in continuous cereals – ‘take-all decline’.

Take-all causes most damage on light soils where loss of active roots has a large effect on water and nutrient uptake.

Even on chalky boulder clay soils with high water-holding capacity, losses of 10% are common in second and third wheat crops. On less well-bodied soils, yield losses can be much higher, so it may be uneconomic to grow second or subsequent wheat crops.

### Risk factors
- High pH increases disease risk but severe attacks can also occur in acidic patches
- Poor drainage, low nutrient status and particularly early sowing and light, puffy seedbeds, encourage the disease
- Cereal volunteers and grass weeds, especially couch, in break crops will carry the disease through to following cereals

### Control

**Cultural**
Control relies largely on rotation and good soil management and husbandry. Reducing the severity in second and subsequent wheats is achieved by delaying drilling compared with first wheats and maintaining good soil structure and nutrient levels.

**Varieties**
All varieties are susceptible to take-all but some are more tolerant in the presence of disease. The HGCA Recommended List yields for second wheats give some guidance to choosing the most suitable varieties for second and subsequent sowings. Yield responses reflect take-all tolerance and eyespot resistance. Varieties known to perform well as second wheats include Duxford, Grafton and Ketchum.

**Fungicides**
Seed treatments based on silthiofam and fluquinconazole can help to reduce the effects of take-all, particularly when used in conjunction with cultural control measures. Seed treatments do not alter the optimum sowing date but can delay the take-all epidemic and reduce the yield penalty from sowing second wheats early.

Azoxystrробin or fluoxastrobin applied at T1 timing can help suppress take-all.

### Seed treatment does not alter optimum sowing date

HGCA-funded trials, ADAS Rosemaund

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For further information

See *Take-all in winter wheat – management guidelines, HGCA (2006).*

[www.hgca.com/publications](http://www.hgca.com/publications)
Stem-base diseases can be difficult to distinguish, particularly early in the season when treatment decisions are made. Identification is necessary because eyespot is much more important than sharp eyespot (*Rhizoctonia cerealis*) and fusarium foot rot. During stem extension, lesions caused by fusarium and sharp eyespot are generally confined to the outer leaf sheath. Some fungicides give incidental sharp eyespot control but specific treatment is difficult to justify.

**Eyespot**

*Oculimacula acuformis* (R-type)

*Oculimacula yallundae* (W-type)

**Symptoms**

Eyespot affects the stem base of winter wheat from autumn onwards. Lesions appear as red-brown blotches. Mixed R and W-type eyespot infections occur in most UK crops. Symptoms may disappear as leaf sheaths die off during spring growth but can reappear later.

**Importance**

Eyespot reduces yield and quality by restricting water and nutrient uptake. Yield losses may be large, particularly in early-sown crops. Severe eyespot can cause lodging from weakened stems. Even without lodging, severe eyespot can reduce yield by 10–30%.

**Risk factors**

Traditionally, eyespot assessment in wheat crops was carried out in the spring (GS30-31) to judge the need for chemical control. A new risk assessment method has been developed that includes an early, pre-sowing, assessment of agronomic risks. This enables fields at greater disease risk to be identified, allowing an integrated management approach.

Risk is highest:

- In the west
- On heavy soil
- Where the previous crop was wheat or another cereal
- With ploughing
- Where crops are sown early

**Control**

**Varieties**

Eyespot-resistant varieties on the HGCA Recommended List 2012/13 include Battalion, Beluga, Cocoon, Grafton, Humber and Scout.

**Fungicides**

Good control is difficult to achieve and may be affected by the type of eyespot present. Many treatments only reduce severity.

Prothioconazole or boscalid (the latter in mix with epoxiconazole), control both eyespot and septoria tritici well – a useful option at T1. Higher doses are usually required for eyespot control. Specific eyespot treatments should be applied at GS30-31 if high risk justifies early treatment.

Yield response to foliar disease control usually exceeds response from eyespot treatment. However, delaying treatment until GS32 – for better disease control on leaf 3 – may compromise eyespot control, especially if disease levels are high on susceptible varieties.

Some control may be achieved at GS37 if eyespot has not penetrated the stem base.

For the eyespot risk assessment and more information, see Topic Sheet 111.

www.hgca.com/publications
Ear diseases

Diseases can affect grain quality by reducing grain filling, leading to low specific weights and shrivelled grain. Infections of fusarium and microdochium species may result in mycotoxins. Sooty moulds, and similar diseases, may affect grain appearance causing rejection for milling.

Foliar diseases that affect the ear

**Septoria nodorum** is potentially the most damaging to yield. In south-west England, foliar and ear infections can cause yield losses of up to 70%.

**Yellow rust** infects ears in severe cases.

**Brown rust** can also affect ears.

**Powdery mildew** although very obvious on ears, does not cause large yield losses.

Specific ear diseases

**Sooty moulds** are caused by a mixture of fungi, mainly *Cladosporium* and *Alternaria* species, which grow on glume surfaces in wet weather or on prematurely ripened ears. They cause little yield loss but can discolour grain, which affects marketability, particularly for milling.

**Fusarium ear (or head) blights** are caused by a range of fusarium species and *Microdochium nivale* and *Microdochium majus*.

In UK wheat, the main mycotoxin-producing species are *Fusarium culmorum*, *Fusarium graminearum* and *Fusarium avenaceum* which all produce similar symptoms. The presence of ear blight is not a good indicator of likely mycotoxin risk in UK crops.

Legislation imposes a limit for deoxynivalenol (DON) in grain for human consumption of 1250 parts per billion. In most years few UK grain samples have exceeded this limit.

Risk of mycotoxin formation can increase if:

- Maize preceded the wheat crop
  - NB: ploughing maize residues reduces risk
- Wet weather occurred during flowering

Fungicide treatment can help reduce ear blight and mycotoxin risk provided the recommended rate is used as near to infection time as possible.

Seed-borne ear diseases

Details of diseases such as bunt, ergot and smut can be found at pages 6 and 7.

Control of ear diseases

Sprays applied after ear emergence:

- Top-up protection for flag leaf and leaf 2 against foliar disease
- Protect the newly-emerged ear against foliar diseases
- Protect against specific ear diseases
- Limit mycotoxin accumulation

Sprays applied around GS59 can help maintain canopy size and prolong its duration by protecting leaf and ear green area against disease. For ear blight control, spray during anthesis (GS61-65).

The T3 ear spray should be considered where it is necessary to ‘top-up’ disease control on the flag leaf and/or to protect the ear from disease. If no T3 spray is planned, it is important not to delay the T2 spray. Delaying the T2 spray to allow part of the ear to emerge will lead to poorer foliar disease control on the critical flag leaf and leaf 2.

For the latest information on fusarium mycotoxins and the risk assessment, see: [www.hgca.com/mycotoxins](http://www.hgca.com/mycotoxins)
Barley yellow dwarf virus (BYDV)

**Symptoms**
In wheat, infections cause leaf yellowing and stunting, initially confined to single plants scattered randomly in a field. Distinct circular patches develop later as secondary spread occurs.

**Importance**
BYDV is most damaging when young plants are infected in autumn. Economic loss from a severe infection can make the crop unprofitable. Substantial yield loss is rare in spring-sown crops.

**Life cycle**
BYDV is transmitted by the grain aphid and the bird cherry aphid. Grain aphids fly into crops during late summer and autumn, spreading disease. The LT50 (lethal temperature for 50% mortality) for grain aphid is -8°C. The bird cherry-oat aphid is more frost-susceptible, with an LT50 of 0.5°C.

**Control**
Control measures aim to prevent infection and reduce spread.

**Cultural**
Good stubble hygiene and an interval of five weeks between ploughing and sowing help prevent transmission via the ‘green bridge’ (ie aphids on grass weeds and volunteers).

**Chemical**
Insecticidal seed treatments can provide four to six weeks’ protection for early-sown crops in high-risk areas. An aphicide spray can prevent wingless second and third generation aphids spreading disease within the crop.

Development time for each generation depends on temperature. An accumulated sum of 170 day-degrees above 3°C is necessary to produce a generation (the ‘T-sum 170’). Treatments are timed to coincide with the production of second generation aphids in a crop, at T-sum 170. The T-sum 170 is calculated either following emergence or the end of seed treatment protection.

Treatments at T-sum 340 may be justified where aphids continue to fly after the T-sum 170 spray.

Decisions on whether to treat crops emerging in September must be made by the start of October. The treatment window is wider for later-emerging crops.

Soil-borne cereal mosaic virus (SBCMV)

Soil-borne wheat mosaic virus (SBWMV)

The viruses are transmitted by the soil-borne organism *Polymyxa graminis* and can remain viable in soil for at least 15 years. The viruses are spread by any movement of soil infested with *Polymyxa graminis* containing SBCMV or SBWMV particles.

**Symptoms**
Symptoms vary from pale green to prominent yellow streaks on leaves and leaf sheaths, accompanied by moderate to severe stunting. Infections usually occur in distinct patches that increase in size in successive years.

**Importance**
Mosaic virus is present in the UK but symptoms are not commonly found.

**Control**
Once land is infected by SBCMV or SBWMV, the only practicable means of control is to grow resistant varieties.
Assessing disease risk

Disease pressure must be balanced against field resistance

Crop protection decisions need to be taken as upper leaves emerge, well before symptoms develop on yield-forming leaves.

To estimate the chance of disease development, the likelihood of infection – 'disease pressure' – has to be balanced against the ability of the crop to resist or avoid infection – 'field resistance'.

Disease inoculum

Disease lesions on lower leaves are the most common source of infection of upper leaves emerging during stem extension. Crops should be inspected regularly. If even a small amount of disease is visible on lower leaves, the potential risk is high. Whether disease develops subsequently on upper leaves depends on varietal resistance, crop management and weather.

Crop management

High nitrogen uptake encourages rapid development of rusts and powdery mildew. In such crops even moderately resistant varieties can suffer high levels of disease.

Early drilling (early September) puts crops at higher risk to most diseases and moderately resistant varieties can suffer high levels of disease.

Varieties

HGCA Recommended List trials assess disease susceptibility and yield across the UK, helping growers select varieties with resistance to diseases prevalent in specific regions. Robust fungicide programmes are used in RL trials to maximise varietal potential. Comparing treated and untreated yields provides an indication of total yield response to fungicides. Varieties with larger differences between treated and untreated yields will usually need higher fungicide inputs to achieve their yield potential.

The susceptibility of varieties to each disease is partly reflected in the yield response to fungicides. Other factors may affect varietal yield response:

- Differing yield sensitivity to disease (tolerance)
- Diseases have differing effects on yield
- Fungicide effects not entirely linked to control of visible disease, eg canopy ‘greening’ or growth regulatory effects

Disease resistance

Some varieties are highly resistant to disease and may not require fungicide treatment if disease pressure is low. Even under high disease pressure a moderate fungicide input may be sufficient. Other varieties are very susceptible, requiring high fungicide input to reach their yield potential.

It is important to take disease resistance into account before deciding on an appropriate fungicide dose.

Choice of a more disease-resistant variety can significantly reduce the total fungicide input needed.

Types of resistance

Race non-specific resistance is effective against all fungal races and is not subject to sudden failure. Therefore, the Recommended List ratings provide a guide to the relative resistance of this type of variety that is unlikely to change quickly. Although control is not complete, it can be as effective as fungicides. This is the main type of resistance available against septoria tritici.

Race-specific resistance can provide complete protection (RL rating 9) against rusts or powdery mildew. But new ‘races’ of the fungus may develop which can overcome this form of resistance, leading to sudden loss of control.

Race-specific resistance to rusts and powdery mildew will no longer be effective if a virulent race of the fungus emerges. When such a change occurs, the level of varietal susceptibility then depends on its underlying race non-specific resistance. New races of brown rust and yellow rust were detected in 2011 – all varieties should be monitored closely in 2012 season, particularly in regions with a high risk of infection.
### Active ingredients for wheat disease control

**Active ingredients**

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<th>Active ingredient</th>
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Updated annually, see [www.hgca.com/diseasecontrol](http://www.hgca.com/diseasecontrol)
New fungicides and resistance management

Resistance
It can arise rapidly so that control is lost in a single step or develop gradually. In this case, the pathogen population becomes progressively less sensitive and field performance may initially only be affected under very high disease pressure and with susceptible varieties.

Septoria tritici
Sensitivity to triazoles has declined slowly since the mid-90s but efficacy of epoxiconazole and prothioconazole remains good at full label doses. Azole resistance is due to various mutations and their frequency has changed quite rapidly in recent years. Resistance is widespread to both MBC fungicides and strobilurin fungicides.

Yellow and brown rust
Although shifts in sensitivity to triazoles were reported in the 1990s, field performance has been maintained. No resistance has been found to morpholine or strobilurin fungicides.

Powdery mildew
With rapid growth and many disease cycles each season, there is an inherently high resistance risk. High levels of resistance to strobilurins in mildew mean these fungicides are ineffective. After an initial shift, the sensitivity to morpholines and triazoles has stabilised and they still provide partial control. Quinoloxefen-resistant isolates are now widespread in the UK. In 2009 and 2010, low levels of isolates resistant to metfenafenone were detected in parts of Europe.

Septoria nodorum
Strobilurin resistance has been reported in mainland Europe and may be affecting fungicide performance in the UK.

Eyespot
Reduced sensitivity to prochloraz and cyprodinil has been known in parts of Europe for several years. There is no evidence of any sensitivity shift to cyprodinil in the UK. Some triazoles and boscalid achieve good control.

Succinate Dehydrogenase Inhibitors (SDHIs)
Since 2010, a number of new SDHIs have been approved for use on wheat: bixafen (in Aviator Xpro and other products), isopyrazam (in Seguris) and fluxapyroxad (in Adexar). SDHIs inhibit a different enzyme in the mitochondria of fungi to azoles. These new generation SDHIs have excellent efficacy on septoria tritici and a broad spectrum of foliar disease control. They are at medium/high risk of fungicide resistance so should always be used in mixtures (see below).

Reducing resistance risk: the role of mixtures
Azole and SDHI-based fungicides, are at risk of resistance development. These fungicides should be used in a way which slows resistance, to avoid future loss of efficacy.

Using mixtures of fungicides with different modes of action and good efficacy, is key to reducing resistance risk and achieving good control.

For this reason, SDHI fungicides are only available in mixtures with fungicides of a different mode of action. Azole fungicides are available as single active substance products, but should not be used alone.

For the major diseases, basing the programme on azole + SDHI mixtures should provide some mutual protection from the risk of resistance development against both the components.

For septoria tritici control, in most circumstances, adding chlorothalonil to azole or azole + SDHI mixtures aids efficacy, reduces the risk of resistance development, and should prolong their usefulness. However, if T2 sprays are delayed, the addition of chlorothalonil may slightly reduce triazole or SDHI / triazole eradicant activity. Therefore, where infections are already established and full eradicant activity is needed, chlorothalonil should be omitted.

For rust control, adding a strobilurin fungicide to an azole or azole + SDHI mixture may improve control and reduce the risk of resistance development. Only two strobilurin sprays can be applied in any season.

Good resistance management is based on limiting the level of exposure of the target pathogen to the fungicide

– Fungicide input is only one aspect of crop management and other control measures should always be used, such as good hygiene through disposal of crop debris and control of volunteers which may harbour disease
– Always aim to select varieties exhibiting a high degree of resistance to diseases known to be prevalent in your area, in addition to the main agronomic factors you desire
– Avoid growing large areas of any one variety, particularly in areas of high disease risk where the variety is known to be susceptible
– Only use fungicides in situations where the risk or presence of disease warrants treatment
– Use a dose that will give effective disease control and which is appropriate for the cultivar and disease pressure
– Make full use of effective fungicides with different modes of action in mixtures or as alternative sprays
– Ensure that mixing partners are used at doses that give similar efficacy and persistence
– Monitor crops regularly for disease and treat before the infection becomes well established
– Avoid repeated applications of the same product or mode of action and never exceed the maximum recommended number of applications

For more information on resistance management, see the Fungicide Resistance Action Group - UK (http://www.pesticides.gov.uk/guidance/industries/pesticides/advisory-groups/Resistance-Action-Groups/frag)
Foliar diseases – Fungicide dose

Determining appropriate dose
Fungicides are rigorously tested in HGCA-funded trials. Each year, a single spray is applied at a range of doses on varieties which are highly susceptible to each major disease, and at sites where disease pressure is high. Disease levels are observed a few weeks later.

Performance of individual active ingredients can be assessed by comparing dose-response curves. These show average performance measured across a range of sites, seasons and leaf layers.

Disease severity in untreated crops depends on local disease pressure and varietal resistance. In treated crops, severity also depends on fungicide dose applied.

The dose-response curve

Fungicide dose and margin
Fungicide spray cost increases with dose applied, while yield loss, to some degree, is proportional to the amount of disease present. The figure below plots fungicide dose against margin and identifies when the return from a higher dose would not be economically justified.

The appropriate dose depends on disease risk and predicted yield loss and is defined as that point where margin is maximised.

Below the appropriate dose, profit is seriously reduced by ineffective disease control.

Maximising profit may mean accepting a small amount of disease in the crop despite treatment.

How disease and variety affect dose
Differing disease pressure is a major reason for varying appropriate doses between different crops. Clearly, higher disease pressure and disease susceptibility justify higher inputs.

However, crop tolerance to disease (i.e., yield loss from a given level of disease) and fungicide effectiveness also modify the appropriate dose.

To help select an appropriate dose, see the HGCA website: www.hgca.com/diseasecontrol
**Fungicide performance curves**

**Protection v eradication**

‘Protection’ curves show the activity of fungicides when they are applied soon after the emergence of a leaf layer before much infection has occurred.

‘Eradication’ curves indicate fungicidal activity against septoria tritici after infection has occurred, but before symptoms become visible.

Performance of products on each leaf layer, and at each site, was classified as protectant or eradicant based on timing of leaf emergence relative to spray application.

Updated annually, see [www.hgca.com/diseasecontrol](http://www.hgca.com/diseasecontrol)

### Septoria tritici

#### Protection

![Graph showing septoria tritici protection curves for different fungicides.](image)

**2009-2011 aggregate data**
- Adexar
- Aviator Xpro
- Brutus
- Opus*
- Proline
- Seguris

*(1.0 = label recommended dose)*

### Septoria tritici

#### Eradication

![Graph showing septoria tritici eradication curves for different fungicides.](image)

**2009-2011 aggregate data**

*Opus has been succeeded by Ignite in trials but performance is similar*
Yellow rust

Brown rust

Mildew

Updated annually, see www.hgca.com/diseasecontrol
<table>
<thead>
<tr>
<th>Spray timing</th>
<th>Rationale</th>
<th>Product choice and dose</th>
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<tbody>
<tr>
<td><strong>T0</strong></td>
<td>Treatment combined with PGR before T1 may be appropriate in high disease years on susceptible varieties. Any yellow rust found should be controlled immediately. Controlling brown rust, mildew and eyespot may be economic where disease risk is high and on susceptible varieties. Treatment may slow early rust epidemic development and reduce disease pressure at T1 and T2. Check crops in early spring for yellow rust, brown rust, mildew and eyespot. Low doses – quarter to half label recommended dose – are appropriate. Timing for eyespot and brown rust control – 2–3 weeks before T1 applications. For septoria tritici control, sprays are largely for insurance. Chlorothalonil applied three weeks before T1 applications would be adequate.</td>
<td>Rusts – triazoles, strobilurins  &lt;br&gt; Mildew – cyflufenamid, fenpropidin, metrafenone, proquinazid, quinoxyfen, spiroxamine  &lt;br&gt; Eyespot – boscalid + epoxiconazole, cyprodinil, flusilazole, metrafenone, prochloraz, prothioconazole  &lt;br&gt; Septoria tritici – chlorothalonil</td>
</tr>
<tr>
<td><strong>T1</strong> Leaf 3 emerged</td>
<td>Primarily aimed at controlling septoria tritici on recently-emerged final leaf 3 and sometimes diseases on leaf 4. Varieties susceptible to septoria tritici (rated 5 or less on the HGCA Recommended List) should be targeted as high priority. Check growth stage and leaf emergence carefully at this time. Spraying too early or too late will give poorer disease control. Sprays applied for septoria tritici will normally also control rusts. Eyespot risk should be assessed. Strobilurins add to disease control and increase yield due to greening effects.</td>
<td>Base spray on a triazole/chlorothalonil mixture, possibly with the addition of SDHIs. Use doses between half and three-quarters of label recommended dose to ensure eradicant control of septoria tritici. The triazole choice and dose is particularly important. Septoria tritici – chlorothalonil, triazoles, SDHIs  &lt;br&gt; Rusts – morpholines, spiroxamine, strobilurins, triazoles, SDHIs  &lt;br&gt; Mildew – cyflufenamid, cyprodinil, fenpropidin, metrafenone, metrafenone, proquinazid, quinoxyfen, spiroxamine, SDHIs  &lt;br&gt; Eyespot – boscalid + epoxiconazole, cyprodinil, flusilazole, metrafenone, prochloraz, prothioconazole</td>
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<tr>
<td><strong>T2</strong> Flag leaf emerged</td>
<td>This is the most important spray as yield responses to flag leaf sprays are consistently profitable. This spray is aimed at controlling disease on the top two leaves, which contribute approximately 65% of yield. Apply when most flag leaves on main tillers have emerged. Varieties prone to septoria tritici (rated 5 or less on the HGCA Recommended List) should be targeted for treatment first as delaying flag leaf sprays will be costly. Spray timing is less critical on more resistant varieties.</td>
<td>Doses between half and full label recommended dose are usually appropriate. Higher triazole doses are needed on septoria tritici susceptible varieties under high disease pressure. Lower doses are appropriate on disease-resistant varieties under low disease pressure. Use triazole/SDHI mixture to ensure good eradicant control of septoria tritici and prolong green leaf area of the top two leaves. Add chlorothalonil on septoria tritici susceptible varieties in protectant situations. Septoria tritici – chlorothalonil, triazoles, SDHIs  &lt;br&gt; Rusts – morpholines, spiroxamine, strobilurins, triazoles, SDHIs  &lt;br&gt; Mildew – cyflufenamid, fenpropidin, metrafenone, metrafenone, morpholines, proquinazid, quinoxyfen, spiroxamine, SDHIs</td>
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<td><strong>T3</strong> Ear spray</td>
<td>The ‘ear’ spray targets ear diseases, but also gives additional control of disease on the top two leaves – important in high disease seasons and on disease-prone varieties. On septoria tritici susceptible varieties, ensure triazole applied for ear diseases is also active against septoria tritici. In disease-resistant varieties, an ear spray may not be necessary. Brown rust, yellow rust and septoria nodorum can be damaging if ears are affected. Wet weather during flowering can lead to fusarium ear blight and possibly mycotoxins and discoloured grain. Control of ear blight is difficult and costly as high doses must be applied close to the infection period. Avoiding mycotoxins is more important for wheat intended for human consumption. Growers should use the HGCA fusarium risk assessment tool and treat accordingly. Sooty moulds, which result in discoloured grain, have little effect on yield but can be important in milling varieties.</td>
<td>Choose a triazole-based product or mixture with specific activity against ear diseases. This also provides broad-spectrum eradicant control on upper leaves. Consider adding a strobilurin where grain filling is likely to be prolonged or where brown rust is a risk. A quarter to a half label dose is appropriate for additional disease control. A minimum of half dose is necessary for fusarium control.  &lt;br&gt; <strong>Preferred active ingredients specifically for ear disease control:</strong>  &lt;br&gt; Septoria nodorum – azoxystrobin, dimoxystrobin, epoxiconazole, prothioconazole, pyraclostrobin, trifloxystrobin  &lt;br&gt; Fusarium ear blight – epoxiconazole plus dimoxystrobin, metconazole, prothioconazole, tebuconazole  &lt;br&gt; Sooty moulds – azoxystrobin, dimoxystrobin, pyraclostrobin, tebuconazole</td>
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Further information

HGCA publications and details of HGCA-funded projects are all available on the HGCA website – www.hgca.com

HGCA Guides
HGCA Recommended Lists for cereals and oilseeds, HGCA (annual)
G34 Guidelines to minimise the risk of fusarium mycotoxins in cereals, HGCA (2010)
P05 Nozzle selection chart, HGCA (2010)
G41 The encyclopaedia of cereal diseases, HGCA/BASF (2008)
G39 The wheat growth guide, HGCA (2008)
G31 Take-all in winter wheat – management guidelines, HGCA (2006)
G14 Pest management in cereals and oilseeds, HGCA (2003)

HGCA Topic Sheets, Information Sheets and Project Progress
TS113 Septoria tritici in winter wheat, HGCA (2012)
TS112 Wheat yellow rust management, HGCA (2012)
TS111 Managing eyespot in winter wheat, HGCA (2012)
TS108 HGCA risk assessment for fusarium mycotoxins in wheat, HGCA (2011)
TS96 Options for low volume spraying of winter wheat, HGCA (2007)
TS56 Managing ergot in crops, HGCA (2002)

HGCA Project Reports
PR477 Improving risk assessment to minimise fusarium mycotoxins in harvested wheat grain (2011)
PR475 Understanding evolution and selection of azole resistance mechanisms in UK populations of Mycosphaerella graminicola (2011)
PR462 EURO-wheat: A European collaboration on resistance characteristics of wheat cultivars, wheat pathogen virulence, disease management tools and fungicide efficacy (2011)
PR459 Monitoring risks of mycotoxin contamination caused by fusarium head blight pathogens in winter wheat (2009)
PR456 Towards a sustainable whole-farm approach to the control of ergot (2009)
PR444 Appropriate Doses Network: up-to-date information on fungicide performance for wheat growers (2008)
PR439 Fungicide doses in sequences and mixtures for winter wheat (2008)
PR432 Understanding the basis of resistance to fusarium head blight in UK winter wheat (2008)

Current projects
3187 United Kingdom Cereal Pathogen Virulence Survey (UKCPVS)
3453 Integrated strategy to prevent mycotoxin risks
3479 Study of Fusarium langsethiae infection of cereals
3517 Improved tools to rationalise and support stewardship programmes for SDHI fungicides to control cereal diseases in the UK
3570 Cephalosporium leaf stripe – an emerging threat to wheat crops in short rotations
3573 Improved modelling of fusarium to aid mycotoxin prediction in UK wheat
3625 Identification, prevalence and impacts of viral diseases in UK winter wheat crops (PhD)
3670 Screening for costs of disease resistance caused by stomatal dysfunction
3713 Identification and characterisation of azole sensitivity shifts in Irish and UK populations of Mycosphaerella graminicola sampled from HGCA fungicide performance winter wheat trials
3729 Development of novel methods for detecting and quantifying viable inoculums of Oculimacula yallundae and O. acuformis (PhD)
3730 Value of resistance genes for controlling septoria tritici in high-yielding wheat varieties (PhD)
3734 New fungicide performance in winter wheat

Websites
Information on the efficacy of individual products will be updated annually with a range of online resources. Always consult the HGCA website for the latest versions.
HGCA – www.hgca.com
- Fungicide performance tool (www.hgca.com/diseasecontrol)
- Fusarium mycotoxin risk assessment (www.hgca.com/mycotoxins)
- RL Plus (www.hgca.com/varieties/rl-plus+)
- The Encyclopaedia of Cereal Diseases (www.hgca.com/cde)
- The UK Cereal Pathogen Virulence Survey

Other websites
British Society of Plant Breeders – www.bspb.co.uk
Chemicals Regulation Directorate – www.pesticides.gov.uk
CropMonitor – www.cropmonitor.co.uk
Crop Protection Association – www.cropprotection.org.uk
Food Standards Agency – www.food.gov.uk
Liaison – https://secure.fera.defra.gov.uk/ liaison
Fungicide Resistance Action Committee (FRAC) – www.frac.info
NIAB TAG – www.niab.com
SAC – www.sac.ac.uk/crops
Science and Advice for Scottish Agriculture – www.sasa.gov.uk
The Voluntary Initiative – www.voluntaryinitiative.org.uk
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