Septoria tritici, caused by the fungus *Mycosphaerella graminicola*, is the most important wheat disease in the UK. Yield losses of 30-50% have been reported and susceptible varieties in the HGCA Recommended List can average a yield loss of 20% in untreated trials. This equates to a cost of up to £250 per hectare, based on a grain price of £150/tonne and an average treated yield of 8.5 t/ha.

### Fungicide timings to control disease

A typical fungicide programme focusing on managing *Septoria tritici* needs to start in the spring to limit the disease on the lower leaves but the GS39 timing is the most important to keep upper leaves disease free to optimise yield and quality.

#### Action

- **Select wheat varieties with the best available septoria resistance.**
- **Start a fungicide programme focusing on septoria in the spring to limit disease on lower leaves.**
- **Use different modes of action to achieve the best eradicant and protectant control.** See www.hgca.com/diseasecontrol for HGCA’s annually-updated information on fungicide performance.
- **Use anti-resistance strategies, such as alternating and mixing active ingredients and using alternative control methods, to help prolong the useful life of azole and SDHI fungicides.**

*Always consider your local conditions and consult a professional agronomist if necessary.*

#### Septoria tritici risk levels

- High
- Moderate

#### Importance

### Timing | Comments
--- | ---
Autumn-winter | No specific action is required. Some seed treatments applied to protect from rusts may limit infection.
Early Spring T0 (GS25-30) | Early-sown crops and susceptible varieties may start to develop extensive symptoms on the lower leaves. Protectant multi-site fungicides will help reduce infection and spread to developing leaves. An additional systemic fungicide applied for prevention of rusts/eyespot/mildew may also be considered. No specific action is required for resistant varieties.
T1 (GS31-32) | GS32 is an optimum early time to control *Septoria tritici*. Crops treated at T0 under high disease pressure and resistant varieties that have yet to receive a fungicide are best treated now. **The best approach is to use a fungicide mixture that both eradicates disease and also provides protection to emerging leaves (eg azole + multi-site +/- SDHI).** Other diseases, including eyespot, will also require attention and this can be achieved with some azole or SDHI fungicides.
T2 (GS37-39) | **This timing is important for all crops regardless of varietal resistance, timing of earlier treatment and perceived disease risk.** The aim is to eradicate disease already developing in healthy-looking leaves lower down the canopy and to protect the flag leaf, which has just emerged, for as long as possible. **The use of different modes of action is needed to achieve the best eradican and protectant control: use an azole + SDHI fungicide and keep dose rates high for susceptible varieties.** The addition of a multi-site protectant will help protect the leaves from secondary infection but check labels since its addition may reduce eradican properties of some fungicides.
T3 (GS61-65) | **In the north and west**, the septoria tritici risk continues throughout the season and treatment at T3 is required to prolong the protection of the upper leaves. **In the drier east**, there can be less focus on septoria tritici and more on managing fusarium head blight. Ensure the fungicide is effective against both diseases (eg prothioconazole, metconazole or tebuconazole) for greatest flexibility. **In cool and wet years**, a multi-site protectant applied now can help minimise damage associated with reinfection but check the number of applications that have already been applied and the latest time of application.
Overwinters on crop debris
Spore-producing structures develop within lesions
Airborne sexual spores
Rain-splashed asexual spores
Spore-producing structures develop within lesions

High-risk situations
- Early-sown susceptible varieties
- Crops sown where straw debris is common
All crops are likely to be affected by wind-blown spores.

Contact protectant fungicides
Protectant contact multi-site fungicides (e.g. chlorothalonil, mancozeb and folpet) will prevent spores from germinating and infecting the plant if applied prior to spore release. Once infection has occurred and the fungus has penetrated the leaf, these fungicides will no longer control the disease.

Systemicazole fungicides
Systemic curative fungicides, including azole fungicides epoxiconazole (Ignite), prothioconazole (Proline 275) and formulations of multiple azoles such as epoxiconazole + metconazole (Brutus) can control the fungus when it has infected a leaf and is developing inside the plant during the early stages of development. These fungicides inhibit an essential enzyme for the formation of cell membranes, arresting the growth of actively growing fungus inside a leaf.

Systemic SDHI fungicides
Succinate Dehydrogenase Inhibitor fungicides (SDHIs) include the active ingredients boscalid (Tracker), isopyrazam (Seguris), bixafen (Aviator Xpro) and fluxapyroxad (ADEXAR). They can act at early stages of spore germination and also on the actively growing fungus inside the leaf. This group of systemic fungicides blocks the function of succinate dehydrogenase, an important enzyme required for mitochondrial respiration, leading to the death of the fungus.

Seed treatments
Septoria tritici is not considered to be seed-borne but some systemic seed treatments commonly used to protect crops from yellow rust may control early symptoms.

Symptoms
Following the latent phase, where the fungus can develop undetected by eye, an interaction of the fungus with the host leads to cell death, resulting in the production of pale brown lesions with small dark fungal bodies (pycnidia) inside.
Yield loss occurs as a consequence of reduction in green leaf area for photosynthesis.

Fungicide resistance and anti-resistance strategies
Significant shifts in the sensitivity of septoria tritici to azoles since the late 1990s have increased concern that the most commercially important azoles could come under threat from resistance development. Azole fungicides are the primary control method for septoria tritici in UK winter wheat crops: varieties differ in their susceptibility but all commercial varieties are susceptible to some extent. Any further substantial decline in efficacy would have serious commercial implications for disease control in wheat.

Average azole usage is 2.85 azole-based fungicide applications per crop (CropMonitor, 2010 data), with a total of 99% of crops treated with this chemical group.

Research shows that all azoles are affected, to varying degrees, by changes in the sensitivity of the Mycosphaerella graminicola population. The efficacies of older azole products tend to be more affected; many now offer very poor control of septoria tritici. In practice, usage in the UK is largely focused on a few azoles where good efficacy is retained, i.e. epoxiconazole and prothioconazole. It is imperative that the best available anti-resistance strategies are applied to preserve and extend the useful life of these active ingredients.

Anti-resistance strategies are based on three principles:
- To make full use of alternative control methods, such as varietal resistance
- To alternate fungicides with alternative modes of action when treating crops
- To use mixtures of fungicides with alternative modes of action when treating crops

SDHIs are considered to have a medium to high-risk of resistance development because of their single site of action and so should only be used in mixtures with fungicides with a different mode of action.
Septoria tritici in winter wheat

Symptoms

The first common sign of the disease in the autumn and winter is pale brown lesions on the bottom leaves, with small black fungal bodies (pycnidia) easily seen by eye. During leaf production phases of growth in the winter and early spring, it is common for new leaves to appear green and healthy and for older leaves to die back showing typical symptoms.

Stressed crops can show greater levels of infection, where new leaf development is impeded by stress.

High-risk situations

Rain and windy conditions provide the optimum conditions for spores to be released and splash onto healthy leaves.

Leaf wetness can be caused by either rain or dew, so, even during dry weather spells, spore dispersal and infection can still take place.

Once a spore has landed on a new leaf, it can take 12 hours for the spore to germinate; infection of the new leaf usually takes place within 24 hours of the spore being released. Wet conditions are required during this infection process.

Latent period

Symptoms do not appear immediately on a new leaf. The fungus grows undetected inside the leaf for a period of 14-28 days. The speed of visible symptom development is linked to temperature so during the winter it can take a long time for symptoms to appear, while in the summer, symptoms can develop more rapidly.

If the fungus is well into its latent phase, no fungicide will provide effective eradicant activity.

Mixing or alternating differentazole fungicides

Recent research has shown that alternating and mixing different azoles might have some potential to delay or reduce the risk of resistance development.

While this is not as strong an anti-resistance strategy as mixing or alternating azoles with fungicides with different modes of action, such as multi-site inhibitors or SDHIs, it has shown some potential to manage resistance development due to differential control of isolates with particular mutations in the azole target protein, CYP51. The effectiveness of this strategy needs further monitoring, as new CYP51 variants and resistance mechanisms (eg CYP51 overexpression) affecting the sensitivity of multiple azoles can develop and spread in field populations.

Consider integrated crop management practices that reduce disease pressure, for example, selecting wheat varieties with the best available resistance for the desired market. Optimal rates of azoles and azole/SDHI mixtures should always be used to avoid the risk of poor disease control.

Azole fungicides work by binding to the target site protein, sterol 14α-demethylase (CYP51) - a key enzyme in the biosynthesis of a fungal cell membrane component.

Several mechanisms of azole resistance have been identified/reported:

1. Alterations of the target site protein (CYP51) affecting azole binding.

Different changes in the CYP51-encoding gene (mutations and deletions) have led to changes in the protein. Target site mutations are categorised by a series of letters and numbers that identify the position of the substituted amino acid in the protein (eg I381V: isoleucine replaced by valine). Common target site mutations, which are becoming widespread with increases in frequency after treatments and between seasons, include D134G, V136A, I381V, Y461H and S524T.

2. The fungus can also show reduced sensitivity to azole fungicides by producing more (overexpressing) the CYP51 protein.

A low frequency of CYP51 overexpressing strains has been found in the UK since 2009.

3. The fungus can also potentially pump/export azole fungicide out of cells.

This type of resistance is linked with changes in the expression of efflux pumps and can affect fungicides with different modes of action (multi-drug resistance: MDR). Although reported by INRA, MDR strains have not been found in the UK by other research groups despite intensive monitoring since 2009.
Varietal resistance

The HGCA Recommended List (RL) provides information on the resistance of wheat varieties on a 1-9 scale, where a higher number represents better varietal resistance. The ratings are based on results from naturally infected crops grown in field trials throughout the UK.

When choosing a variety, the primary focus is to select a variety that is suitable for the chosen end market and which performs well in the specific region. When these factors have been taken into account, selecting a variety with a better spectrum of disease resistance will make the variety easier to manage and is good practice to reduce the reliance on fungicides to control disease.

There are no RL varieties that are fully resistant to septoria tritici and only a small number with a resistance rating of 7. The treated yield of more resistant varieties does not equal that of the most susceptible varieties but the untreated yield shows that yields will not fall in situations where disease control from fungicides is not effective (Table 1).

Table 1. Average yields of winter wheat varieties on the HGCA Recommended List 2012/13. The resistance to septoria tritici in the first column is given on a 1-9 scale where higher numbers indicate greater resistance.

<table>
<thead>
<tr>
<th>Varietal resistance to septoria tritici (1-9 scale)</th>
<th>Number of winter wheat varieties on the HGCA Recommended List 2012/13</th>
<th>Average treated yield (t/ha)</th>
<th>Average untreated yield (t/ha)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>3</td>
<td>10.4</td>
<td>9.6</td>
</tr>
<tr>
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<td>10</td>
<td>10.6</td>
<td>9.1</td>
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<tr>
<td>5</td>
<td>18</td>
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</tr>
<tr>
<td>4</td>
<td>2</td>
<td>10.8</td>
<td>8.8</td>
</tr>
</tbody>
</table>

A recent research project, part-funded by HGCA, ‘Improved Resistance to Septoria in Superior Wheat Varieties’, aimed to apply knowledge of the genetics of varietal resistance against septoria to improve methods of breeding wheat varieties that combine better disease resistance with high yield and quality in UK conditions. It showed that wheat breeders can improve septoria resistance by combining genes from parental varieties derived from diverse pedigrees and by selecting varieties with genes that increase yield but do not reduce resistance or vice versa. The development of new varieties is a long-term process but the efforts of breeders and researchers have resulted in a steady improvement in the septoria resistance of wheat varieties on the HGCA Recommended List, with the majority now having ratings of 5, 6 or 7 for this trait.

The type of resistance in UK varieties has a partial effect, so it reduces levels of septoria but does not eliminate the disease altogether. A key benefit of partial resistance is that it may be durable so the resistance of UK varieties should remain stable and not be overcome by new strains of the septoria pathogen, *Mycosphaerella graminicola*.

Overview

- The GS39 spray timing is the most important to keep upper leaves disease-free to optimise yield and quality.
- Azole and SDHI fungicides are the primary control method: anti-resistance strategies will help to preserve and extend their useful life.
- Recent research has shown that alternating and mixing different azoles can improve disease control and might have some potential to delay or reduce the risk of resistance development.

Further information

Simon Oxley, HGCA
simon.oxley@hgca.ahdb.org.uk

Bart Fraaije, Rothamsted Research
bart.fraaije@rothamsted.ac.uk

Fiona Burnett, SAC
fiona.burnett@sac.ac.uk

Wheat disease management guide (updated annually)
Wheat disease management guide
www.hgca.com/diseasecontrol

Cereal growth stages – a guide for crop treatments (HGCA, 2009)
Cereal growth stages – a guide for crop treatments (HGCA, 2009)
www.hgca.com/varieties

The encyclopaedia of cereal diseases (HGCA/BASF, 2008)
The encyclopaedia of cereal diseases (HGCA/BASF, 2008)
www.hgca.com/diseasecontrol

Fungicide Resistance Action Group
www.pesticides.gov.uk/guidance/industries/pesticides/advisory-groups/Fungicide-Resistance-Groups/frag

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