

# Eyespot of Wheat

## How to identify and control this common fungal disease

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**E**yespot of wheat, also known as strawbreaker foot rot, is a lower stem-infecting foot rot pathogen that limits winter wheat yields in the dryland Pacific Northwest.

The scientific nomenclature of eyespot has undergone many changes, which can lead to confusion for the common name of the disease.

For the purpose of this publication we will refer to *Oculimacula yallundae* and *Oculimacula acuformis* as the causal agents of eyespot of wheat. Both species can co-exist within the same field, cause identical symptoms on wheat, and follow the same disease cycle. Management recommendations for both species are interchangeable.

### Disease cycle

Eyespot survives on infected wheat straw residue between seasons until autumn rains spread inoculum to autumn-sown seedlings. The infection can begin at the coleoptile and continues through fall, winter, and early spring. Conidia, or asexual spores, germinate from previously infected crop residue and directly penetrate coleoptiles and leaf sheaths at the ground surface. Wind-borne infection has been documented in Australia, Germany, and the United

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**Figure 1.** Eyespot-susceptible winter wheat variety with severe eyespot lesions at the base of the stem near the crown, shown in June 2017 near Adams, Oregon.

### Eyespot, or strawbreaker foot rot: A disease by many other names

#### Previously known as:

FUNGUS NAME	DISEASE NAME
<i>Cercospora herpotrichoides</i>	Cercospora foot rot
<i>Pseudocercospora herpotrichoides</i>	Pseudocercospora foot rot

#### Two causal agents:

CURRENT SCIENTIFIC NAMES	
<i>Oculimacula yallundae</i>	<i>Oculimacula acuformis</i>

OBSOLETE SCIENTIFIC NAMES	
<i>P. herpotrichoides</i> var. <i>herpotrichoides</i>	<i>P. herpotrichoides</i> var. <i>acuformis</i>
<i>Tapesia acuformis</i>	<i>Tapesia yallundae</i>

#### What eyespot is not:

*Rhizoctonia cerealis*, the causal agent of sharp eyespot of wheat

Kingdom. However, infection is more likely to be spread by rain.

Depending on environmental conditions, symptoms may not be visible until spring. Infection results in characteristic elliptical or eye-shaped lesions on the lower stem (Figure 2A). Lesions typically have a dark brown perimeter that distinguishes between healthy and infected tissue (Figure 2B). The center of each lesion is typically yellowish-brown and may have dark brown-blackish pseudoparenchyma fruiting bodies (Figure 2B). In light infections, eyespot symptoms may be limited to leaf sheath layers with no progression into the culm. In severe infections, the lesion will penetrate the stem tissue, and a whitish-gray mass of fungus, called mycelium, may be visible inside the hollow wheat stem (Figure 2C). Eyespot lesions are brittle in contrast to surrounding healthy tissue and thus weaken the stem (Figure 1, page 1), which may result in multidirectional lodging (Figure 3). Lesions can also girdle the flow of water and nutrients to the ripening wheat head, resulting in prematurely ripened white heads that are visible after heading but before the crop dries down prior to harvest (Figure 3).

## Yield loss

Yield loss depends upon the degree and timing of the disease and the extent of lodging. Losses of up to 50 percent in winter wheat fields with uniform eyespot infection have been documented. Superficial eyespot infections that do not fully penetrate the culm typically do not affect the flow of water or nutrients through the plants, or result in lodging or significant yield loss.

Eyespot infection of spring wheat is possible but of minor economic importance.

Once eyespot is established in a field, it can remain indefinitely. Regions with limited rotations of winter wheat with cool, moist, fall seeding conditions, such as the dryland Pacific Northwest, are particularly vulnerable to eyespot infection. Spores can overwinter in the stubble of the previous year's crop, perpetuating the cycle. Factors that may increase risk of eyespot infection include:

- Planting a susceptible variety
- Past history of eyespot in the field leading to inoculum reserves



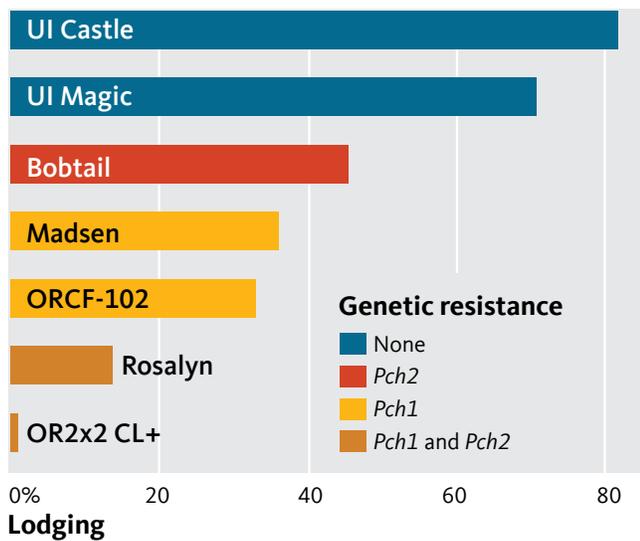
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**Figure 2A.** Winter wheat stem of an eyespot-susceptible cultivar with multiple elliptical eyespot lesions on the lower stem. **Figure 2B.** Closeup of an eyespot lesion on winter wheat to reveal the brown margin of the elliptical lesion separating healthy and diseased tissue. The center of the lesion contains dark brown-blackish pseudoparenchyma fruiting bodies. **Figure 2C.** Bisected eyespot-infected winter wheat stem reveals whitish-gray mycelium.



**Figure 3.** Winter wheat white heads and severe lodging due to an eyespot infection on a highly susceptible variety near Adams in June 2017.

**Figure 4. Percentage of lodging and genetic resistance to eyespot by cultivar**



Graphic © Oregon State University

- Early planted wheat, leading to increased production of fall biomass

Eyespot is mainly a dryland wheat issue, although it can occur in irrigated wheat and in regions that receive a lot of rain. Greater moisture availability contributes to straw decomposition, which reduces inoculum much more quickly. Growing wheat in a short rotation in wet conditions increases the chance of eyespot infection.

### Genetic resistance

Planting genetically resistant varieties is the most effective and economically viable way to prevent yield loss from eyespot.

Two named genes confer a level of resistance to eyespot in wheat: *Pch1* and *Pch2*. *Pch1* was derived from a wild grass species, and *Pch2* was derived from the popular European wheat variety ‘Cappelle Desprez’. Both genes confer resistance to both *O. yallundae* and *O. acufornis*.

The resistance gene *Pch1* provides a greater degree of resistance than *Pch2*. The winter wheat cultivar ‘Madsen’ was the first eyespot-resistant cultivar in the United States containing *Pch1*, though many more have subsequently been released (Table 1).

*Pch2* is less characterized in Pacific Northwest germplasm, but the cultivar ‘Bobtail’ is known to have *Pch2*, but not *Pch1*.

Figure 4 shows the degree of lodging caused by eyespot in inoculated field plots for two highly susceptible cultivars; two cultivars with *Pch1*; ‘Bobtail’ with *Pch2* only; and two cultivars combining *Pch1* and *Pch2*. Combining the two resistance genes appears to provide a significant positive synergism for increased resistance.

**Table 1. Pacific Northwest winter wheat varieties containing the *Pch1* or *Pch2* genes for resistance to wheat eyespot**

Variety	Market class	<i>Pch1</i>	<i>Pch2</i>	Clearfield	Source
Bobtail	Soft white	No	Yes	No	OSU
Nixon*	Soft white	Yes	No	No	OSU
Rosalyn	Soft white	Yes	Yes	No	OSU
Tubbs 06	Soft white	Yes	No	No	OSU
Weatherford	Soft white	Yes	No	No	OSU
ORCF-102	Soft white	Yes	No	1-gene	OSU
OR2x2 CL+**	Soft white	Yes	Yes	2-gene	OSU
ARS-Selbu	Soft white	Yes	ND	No	ARS/WSU
Finch	Soft white	Yes	ND	No	ARS/WSU
Madsen	Soft white	Yes	No	No	ARS/WSU
Masami	Soft white	Yes	ND	No	WSU
Otto	Soft white	Yes	ND	No	WSU
Puma	Soft white	Yes	ND	No	WSU
Purl	Soft white	Yes	ND	No	WSU
LCS Sonic	Soft white	Yes	ND	No	LCS
SY Banks	Soft white	Yes	ND	No	AgriPro
SY Candor	Soft white	Yes	ND	No	Agripro
ARS-Chrystal	Club	Yes	ND	No	ARS/WSU
ARS-Crescent	Club	Yes	ND	No	ARS/WSU
Cara	Club	Yes	No	No	ARS/WSU
Chukar	Club	Yes	ND	No	ARS/WSU
Coda	Club	Yes	No	No	ARS/WSU
Hyak	Club	Yes	ND	No	ARS/WSU
Pritchett	Club	Yes	ND	No	ARS/WSU
LCS Colonia	Hard red	Yes	ND	No	LCS

ND = not determined

OSU = Oregon State University

ARS = USDA Agricultural Research Service

WSU = Washington State University

SY =Syngenta

LCS = Limagrain Cereals Seeds

\* Experimental No. OR2121086

\*\* Experimental No. ORI2150031 CL+

The real world is more complex than the graph in Figure 4, however; many resistance genes of small effect have not yet been well characterized. As a result, cultivars vary significantly in their resistance to eyespot, beyond the resistance conferred by *Pch1* and *Pch2*.

Because of this variation, Oregon State University screens a collection of elite winter wheat varieties annually in inoculated trials in Corvallis. The trial aims to identify resistant cultivars and inform recommendations to growers. Inoculated plots are rated for lodging on a quantitative scale for eyespot resistance. Table 2 shows ratings for current cultivars that have been evaluated in at least two recent growing seasons.

## Fungicides

### TIMING

When an eyespot-susceptible cultivar is planted, fungicides may be used to control infection. Apply fungicide before stem elongation for maximum efficacy. Timing depends on regional factors and is heavily influenced by weather.

In the dryland Pacific Northwest, fungicides are often tank mixed with the late winter herbicide application, commonly referred to as “herbicide timing” application. Unfortunately, in most typical years, an herbicide timing fungicide application to control eyespot will wear off before the jointing phase is complete, leaving the plant unprotected against additional infections.

Applying fungicide after jointing will not be effective, because eyespot damage has already been initiated. A fungicide application after lodging would not prevent yield loss.

Scout and sample to determine whether an eyespot infection is severe enough to warrant fungicide.

To scout for eyespot spray threshold, sample at least 10 plants (totaling at least 50 tillers) from representative areas throughout the field. Wash or soak the samples in water to remove excess soil. Examine the washed tillers for characteristic eyespot lesions—outer leaf sheaths will have a brownish appearance (Figure 1). When leaf sheaths are stripped away, an elliptical lesion will be revealed on the stem (Figures 2A, 2B). If 10 percent of the collected tillers have obvious eyespot lesions in the period from early March to mid-April, consider applying fungicide.

### GLOBAL RESISTANCE ISSUES

Due to the lack of genetic resistance to eyespot available until the 1980s, wheat production in many parts of the world, including the United States, relied on benzimidazole (Group 1, MBC) fungicide sprays such as benomyl, carbendazim, or thiophanate-methyl for eyespot control. Eyespot is classified as a medium risk pathogen for developing fungicide resistance. Eyespot resistance to MBC (Group 1) fungicides is likely because MBC fungicides

## Table 2. Eyespot resistance rating based on lodging in inoculated field plots

For current cultivars that have been evaluated in at least two growing seasons in recent years.  
1= highly resistant, 9 = highly susceptible

Soft white, non-Clearfield	Rating	Soft white, Clearfield	Rating
Bobtail	6	Curiosity CL+	9
Jasper	9	Mela CL+	9
Kaseberg	9	LCS Biancor	8
LCS Art Deco	8	LCS Art Deco	8
LCS Biancor	8	ORCF-101	7
LCS Drive	7	ORCF-102	4
LCS Shark	8	OR2×2 CL+*	2
Mary	7	UI Castle CL+	9
Nixon	6	UI Magic CL+	9
Norwest Duet	9	UI Palouse	4
Norwest Tandem	7	WB1376 CL+	4
Rosalyn*	3	<b>Hard red, non-Clearfield</b>	
Stephens	7	Keldin	8
SY Assure	8	LCS Azimut	5
SY Banks	4	LCS Aymeric	4
SY Dayton	4	LCS Colonia	4
SY Ovation	7	LCS Evina	4
Tubbs 06	5	LCS Jet	4
WB1529	8	Norwest 553	6
WB1529	8	SY Touchstone	4
<b>Hard white, non-Clearfield</b>		WB Arrowhead	8
Irv	6	Whetstone	8
		<b>Hard red, Clearfield</b>	
		SY Clearstone CL2	9
		WB4623 CLP	8

\* Cultivars that carry both *Pch1* and *Pch2*

are high risk for developing fungicide resistance, MBC resistance is common in many other fungal species, and resistance is conferred by several target-site mutations.

The UK’s Agricultural Development and Advisor Service estimated that 52 percent of winter wheat crops were sprayed one or more times with an MBC fungicide in 1982 for eyespot control. Benomyl resistant eyespot was first detected in Germany in 1975, and was detected in the UK in 1981 after two growers in the UK reported severe eyespot infections in fields that had previously received

a timely application of an MBC fungicide. This discovery prompted a larger survey of the UK for MBC-resistant eyespot in 1982. Results of this survey detected MBC resistance in 40 percent of surveyed fields.

### LOCAL RESISTANCE ISSUES

More than 1.2 million acres of winter wheat across Oregon, Washington, and Idaho were affected by eyespot prior to the release of genetically resistant cultivars adapted to the Pacific Northwest. The majority of affected acreage received a benzimidazole fungicide spray annually for eyespot control, which put heavy selection pressure on eyespot to favor the buildup of MBC-resistant eyespot isolates. This caused a fungicide resistance issue similar to that of the UK to unfold in the 1980s in the Pacific Northwest, with the detection of benzimidazole-resistant eyespot isolates in commercial winter wheat fields

There is positive cross-resistance between MBC group members, meaning fungal resistance to a benomyl product confers resistance to all other MBC group members, including carbendazim, fuberidazole, thiabendazole, thiophanate, and thiophanate-methyl.

Benomyl was federally registered for use on wheat for eyespot control in 1977, and a spring benzimidazole fungicide application before stem elongation became standard practice in the dryland Pacific Northwest for eyespot control. At the time of the MBC-resistant eyespot discovery in the Pacific Northwest in 1989, the majority (ranging from 67 to 100 percent) of isolates sampled from nine commercial winter wheat fields were resistant to benzimidazole. Despite the discovery of MBC-resistant eyespot in the Columbia Basin by Murray in 1996, many growers in the region still apply MBC products for eyespot control; however, these applications have little to no effect in controlling eyespot.

### FUNGICIDE ALTERNATIVES

Strobilurin blends registered in Oregon, though effective against stripe rust, are not effective in controlling eyespot. The DMI fungicide prothioconazole, alone or in mixtures, has shown promise in Washington, and is considered a good fungicide for eyespot control in the UK. Priaxor, a blend of the succinate dehydrogenase inhibitor fluxapyroxad and the strobilurin pyraclostrobin, has shown better eyespot control than several other fungicides tested in Washington.

Due to the expense of these fungicides and the risk of fungicide resistance, scout-based spray programs are key to achieve economic returns on spray investment.

### 2017 MBC RESISTANCE CASE STUDY

In June 2017, a producer in Adams, 10 miles north of the Columbia Basin Agricultural Research Center, reported a severe eyespot infection. The 40 foot-by-100 foot winter wheat drill strip variety trial with an eyespot-susceptible cultivar was nearly 100 percent lodged (Figure 4, page 3).

Recommended scouting procedures were followed, and 92 percent of sampled tillers showed characteristic eyespot lesions. The field had a history of eyespot and received a timely application of Topsin-M (thiophanate-methyl, UPI) before stem elongation. Isolations were made from infected stems using a protocol modified from Murray and molecularly confirmed as *O. yallundae*. Evidence showed the addition of thiophanate-methyl did not significantly reduce *O. yallundae* growth after eight days of incubation ( $p=0.73$ ), 12 days of incubation ( $p=0.54$ ), or 35 days of incubation ( $p=0.27$ ). In contrast, strong evidence showed the addition of thiophanate-methyl significantly reduced growth of *O. acuformis* and *O. yallundae* 1990 reference isolates at all concentrations tested, and at all incubation lengths ( $p<0.001$ ).

### Conclusion

Eyespot of wheat has a regional distribution in the dryland Pacific Northwest and can cause significant yield loss if conditions are favorable. The most economical method for avoiding yield loss due to eyespot is host genetic resistance. If not planting a resistant cultivar, deploy a scout-based fungicide spray program to limit economic loss.

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