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# **Plant diseases** impacting **oaten hay production** in Australia – a review

National Hay Agronomy project

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# **Plant diseases** impacting **oaten hay production** in Australia – a review

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## Contents

In Australia, there are a range of fungal, bacterial and viral pathogens that infect oats, impacting on yield and quality of grain and hay crops. The overall impact of these diseases on oaten hay production is not well researched or understood, especially compared to that of other cereal crops. Export hay is evaluated on physical qualities such as stem thickness and greenness and nutritional qualities including water-soluble carbohydrates, neutral detergent fibre, acid detergent fibre and dry matter digestibility. The colour/ greenness of oaten hay can be reduced by disease lesions, chlorosis and saphrophytic fungi growing on dead tissue all of which can cause export hay to be downgraded. How disease generally, and individual diseases specifically impact on nutritional quality are less well understood. For hay production to be profitable, inputs, yield and quality need to be managed. The ability to manage diseases impacting oaten hay production economically has the potential to increase grower returns. This review outlines the major diseases of oaten hay crops in Australia. For the purpose of this review they have been separated into categories based on where they infect the plant (leaf/ foliar, grain or roots). This review also summaries the disease symptoms, epidemiology, current control strategies and suggested future work on these diseases that could increase export hay profitability or address potential future issues in export market access. A list of currently registered seed dressings, in furrow treatments and foliar fungicides has been included containing any potential maximum residue limit (MRL) issues in the export industry.

# Foliar diseases

## Bacterial blight

There are at least two bacterial diseases capable of infecting oats, stripe blight (*Pseudomonas syringae* pv. *striafaciens*) and halo blight (*Pseudomonas syringae* pv. *coronafaciens*). Both diseases are capable of reducing oaten hay quality through damage to leaves. Recent paddock surveys suggest that stripe blight is more common than halo blight in western and southern production regions and was found in up to 40% of paddocks but often at low incidence in individual paddocks. The disease was found predominantly in higher rainfall regions.

### Stripe blight

**Causative agent:** Stripe blight is caused by the bacterium *Pseudomonas syringae* pv. *striafaciens*

**Distribution:** Found across the grain growing regions of Australia.

**Symptoms:** Stripe blight is the predominant bacterial disease of oats. Symptoms, found predominantly on leaves, include water-soaked spots, without the presence of a halo. Spots lengthen and form patches that can then form red-brown stripes, which can distort leaf growth in young leaves. Stripes can develop yellow and red margins. The lesions can merge, forming irregular blotches that can cause the leaf to senesce prematurely.

Stripe blight symptoms on oats. Stripe blight causes water soaked spots to occur on oat leaves which then lengthen to form red-brown coloured stripes with yellow margins. Images courtesy of DPIRD.

The stripes dry and bleach to off-white. The leaf disease symptoms and subsequent senescence can reduce hay colour parameters. Symptoms can be exacerbated by frost events. Stripe blight symptoms can appear similar to halo blight especially at the early infection stages.

**Disease Epidemiology:** The bacteria survive between seasons on seed and plant residues. Factors influencing pathogen survival are not well documented. Rain splash and leaf contact under moist conditions are primary modes of transmission in-crop. I Insects (mainly aphids) can have a small role in spread of the bacteria. As such, bacterial blight is more predominant in high rainfall conditions and in dense crops which are conducive to its spread and infection of the bacteria. Bacterial blight symptoms can be exacerbated by frost. Many Pseudomonas bacteria are ice nucleating meaning they can produce ice crystals which can damage plant cells and allow bacteria to infect the damage cells and multiply. The development of disease is slowed and stopped by warm, dry conditions.

**Management**: Current management strategies include rotating oats with non-host crops, growing resistant varieties, using seed from uninfected crops and burning oat stubble. Chemical control of bacterial blight has been attempted using copper-based products; however, no products are currently registered for this use and their efficacy is not well documented.

Halo blight symptoms on oats. Halo blight causes yellow- pale green water soaked spots on the oat leaf, which then turn brown and join together to form blotches. Images courtesy of DPIRD.

### Bacterial halo blight

**Causative agent:** Halo blight is caused by the bacterium *Pseudomonas syringae* pv. *coronafaciens*.

**Distribution:** Found across the grain growing regions of Australia

**Symptoms:** Halo blight causes oval water soaked spots pale green or yellow in colour. The centre of the spots become yellow-brown in colour, surrounded by a yellow-green halo. As the disease develops the lesions turn brown in colour and join together and form irregular blotches. Severe infection can lead to premature senescence of leaves.

**Disease epidemiology:** Epidemiology of halo blight is similar to that of bacterial stripe blight. Recent paddock surveys suggest that at the current time stripe blight is more common in oat paddocks than halo blight. This may be due to epidemiological differences or variety responses of current dominant varieties.

**Management:** Management strategies are the same as stripe blight.

**Future work:** The impact of bacterial blight (both stripe and halo blight) on hay quality should be assessed. The best approach for reduction of disease incidence currently appears to be the use of genetic resistance,

to facilitate selection of appropriate varieties better understanding of any pathovar virulence within populations and improved variety screening nurseries is a priority (in the first instance stripe blight will be the priority). Understanding the relationship for resistance to the two bacterial blight diseases would provide greater certainty of response. Research is being done through the COGGO funded project "The role of ice nucleating bacteria in frost sensitivity of cereals in Western Australia" to investigate the source of ice nucleating bacteria inoculum. Investigation into bactericidal and bacteriostatic products that can be applied in-crop to provide disease control would help management of severe infection events.

## Red leather leaf (RLL)

Recent surveys of oat paddocks have identified RLL as the most common disease occurring in oat paddocks in the southern production zones of Victoria. It has not been detected in Western Australia.

**Causative agent:** RLL is caused by the fungus *Spermospora avenae*.

**Distribution:** RLL is a significant disease in South Australia and Victoria and has been also found in New South Wales and Tasmania. RLL has not been found in the Northern or Western Regions of Australia.

**Symptoms:** RLL is a major constraint in oat crops reducing hay and grain yield, reducing grain quality and significantly impacting hay appearance and colour. RLL produces small grey-green lesions with red-brown coloured borders. The lesions are surrounded by a

An oats paddock infected with red leather leaf. Image courtesy of Agriculture Victoria.

Red leather leaf (RLL) symptoms on oats. RLL produces small grey-green lesions with red-brown coloured borders. The leaf can turn red-brown in colour (similar to the colour of leather) and become stiff, giving the leaves a leather-like appearance. Images courtesy of Agriculture Victoria.

chlorotic zone on the leaf. As the disease develops, the central area of the lesion becomes necrotic and can disintegrate forming a hole. The leaf can turn red-brown in colour (similar to the colour of leather) and become stiff, giving the leaves a leather-like appearance. Leaf margins and tips can die prematurely. Symptoms of RLL have been confused with BYDV, stripe blight and nutrient deficiencies.

**Disease epidemiology:** The detailed epidemiology of this disease is not well understood and no detailed study of the pathogen life cycle currently exists. RLL survives on crop residue and infection is likely to arise from spores produced in the infested residue and secondary spread from infected plants. RLL is commonly found in areas with high rainfall, mild weather and high humidity.

**Management:** Limited research has been conducted into the IDM for this disease; however, as a trash borne disease, crop rotation will be an important tool in reducing disease pressure. Stubble management is also likely to contribute to reducing inoculum pressure. Varieties differ in disease susceptibility. The planting of more resistant types will reduce losses, however recent research suggests virulence may be affected by disease pathotypes so monitoring for disease is important in all varieties. No fungicides are currently registered for RLL in oats, however fungicides registered in oats for other diseases have been shown to reduce disease severity and provide grain and hay yield benefits. Recent research suggests applications need to be timed to prevent infection and applications may be effective at or before stem extension when disease is first evident.

**Gaps:** Several research and knowledge gaps exist for this disease, some are being addressed in part by different programs but an integrated approach to understanding and managing this disease would be beneficial for the oat industry, particularly in eastern Australia.

Surveillance and diagnostics - The status of RLL in WA is currently unknown. Disease surveys are being conducted as part of the Agrifutures “National Hay Agronomy project” and the GRDC “WA Disease Surveillance” projects. The Albany and Esperance regions of WA will be targeted in the 2020 surveys as these regions have climatic conditions conducive to RLL development most similar to eastern Australian affected regions. The symptoms of this disease can be hard to diagnose, particularly for those without experience. The development of a molecular diagnostic tool would increase the speed and accuracy of RLL identification to facilitate more targeted management in affected crops and to improve surveillance in unaffected areas. AgVictoria are currently working towards developing a molecular diagnostic test.

Pathogen populations - Understanding how the pathogen population varies with regard to virulence and how this influences variety response is vital for management of this disease. The GRDC funded SARDI project “Improved Resistance to Oat Pathogens and Abiotic Stress Management” is currently collecting RLL isolates to determine and characterise pathotype variation. They are also identifying isolates representative of virulence groups to be used for future RLL screening of oat breeding lines. The GRDC project “Improving Grower Surveillance, Management, Epidemiology Knowledge and Tools to Manage Crop Disease in South Australia” is currently creating single spore isolates of RLL. Identification of sources of resistance and targeted breeding for resistance should be longer term goals for oat breeding.

Impact and management - The relationship between severity of RLL and impact on production is currently unknown, although yield losses and hay quality reductions in appearance and feed quality are have been measured where disease levels are high. Work is being done as part of the AgriFutures “National Hay Agronomy” project to address this gap. There are currently, no products registered for RLL control. The registration of products for RLL would be of great benefit to the industry. Research needs to continue to determine the best application timings and chemistries for RLL control.

Epidemiology - The disease lifecycle is not well understood. Epidemiological research could increase understanding of inoculum development, secondary spread within the crop and specific factors driving disease risk.

## Rusts

### Leaf rust

Leaf rust can be a major constraint to oaten hay production as it can reduce both hay appearance and feed quality. Oats are infected by an oat specific leaf rust, it will not infect wheat or barley and the leaf rusts of those crops do not infect oats.

**Causative agent:** Leaf rust also known as crown rust is caused by the fungus *Puccinia coronata* var. *avenae*.

**Distribution:** Found in all grain growing regions of Australia.

**Symptoms:** Leaf rust can affect the leaves, leaf sheaths and panicles of oats. Leaf rust produces small round to oblong shaped pustules, predominantly on the upper surface of the leaf but also leaf sheath and head. These pustules contain masses of yellow-orange powdery spores. The leaf area surrounding the pustule is usually pale green in colour. As the season develops, the rust pustules may turn black and eventually heavily infected leaves can senesce and die. Leaf rust symptoms can be confused with those of stem rust.

Leaf rust symptoms on oats. Leaf rust symptoms include yellow-orange pustules on the oat leaf. Image curtesy of DPIRD.

**Disease epidemiology:** Rusts require living plants to grow and reproduce; the fungus is carried over between seasons on volunteer oats and wild oats. The abundance of wild oats in oat production regions provides a reservoir of green host material to support rust survival and spread. There is an increased risk of a rust epidemic if rust was present at high levels in the previous season and a green bridge has built up rust levels over summer. Urediniospores (yellow-orange powdery spores in pustules) are produced in huge numbers and disseminated by wind over long distances. A spore landing on the surface of an oat leaf requires the presence of moisture to germinate. It can take up to 6 hours for infection to occur and symptoms (pustules) can be observed as soon as 8–10 days after infection. This period is determined primarily by temperature and the optimum temperature for leaf rust development being between 15–22°C. The spore produces a germ tube that grows over the plant surface to a stoma (opening on leaf for transpiration) which it penetrates with an appressorium (infection structure). The rust then grows between the plant's cells, penetrating live plant cells with

haustoria to obtain nutrients. After 1–2 weeks, the rust starts to sporulate producing pustules filled with spores. The pustules then rupture releasing the next generation of spores. As the leaf rust fungus is favoured by temperatures of 15-22°C, rate of epidemic development can often accelerate as temperatures begin to warm in spring.

**Management**: A range of IDM approaches are recommended for management of leaf rust in oats. Green bridge management including grazing or herbicide application to remove oat regrowth or wild oats, prior to sowing should help to reduce carryover of inoculum between seasons. Other strategies include growing resistant varieties, sowing date selection (early sown oats have a higher risk of developing rust) and maintaining good crop nutrition.

Rust populations have pathotypes with varying degrees of virulence to resistance genes within oat varieties. Choosing varieties with resistance profiles suited to regional rust risk is an important rust management tool. Avoiding susceptible and very susceptible varieties in all environments significantly reduces the risk of damaging levels of disease developing in crop, reduces epidemic development and reduces green bridge carryover between seasons. Requirement for fungicide intervention is more likely in susceptible types.

Evolution of new rust pathotypes, or incursion of exotic virulences, can rapidly alter variety resistance responses. Monitoring of rust virulence through contribution of samples to the Australian Cereal Rust Control Program (ACRCP) National Pathotype Survey is vital to having up to date knowledge of expected variety responses.

Fungicides can be applied for rust control, either at seeding as a seed treatment or in-furrow fertiliser application, or in-crop as a foliar application. It is important to apply fungicides to control rusts early in the disease epidemic, before severe infection occurs. Fungicide sprays should aim to protect the top three leaves (including the flag leaf) for as long as possible. Because leaf rust is favoured by warm moist conditions in early spring and has a rapid lifecycle, applications may be required when rust severity is low to protect against rapid disease development. Crop monitoring, particularly in susceptible varieties is essential to optimise response to disease outbreaks.

Biosecurity measures are important in the prevention of rust outbreaks, particularly introduction of new pathotypes from interstate or overseas. Rust spores can be carried on clothing, personal effects or machinery, cleaning and disinfection are important biosecurity measures.

**Future work:** The epidemiology and management of this disease are well understood. The ongoing need with this disease is for monitoring of regional pathotype virulence and screening of varieties for resistance response to dominant pathotypes. Management of rusts in cereals, including oats for grain production is relatively well understood; demonstration of how this relates to oaten hay production and whether control strategies are similar needs further investigation. Integration of foliar disease management strategies for rusts, septoria blotch, RLL and other foliar diseases for oaten hay is an evolving area needing further research.

## Stem Rust

Stem rust is one of the most damaging diseases that can infect an oat crop. Under favourable conditions in susceptible varieties, stem rust can destroy a crop. Oat stem rust only infects oats and oats are not infected by wheat stem rust.

Stem rust pustules in oats on the stems, leaves and leaf sheaths. Image courtesy of DPIRD.

**Causative agent:** Oat stem rust is caused by the fungus *Puccinia graminis* var. *avenae.*

**Distribution:** Found across the grain growing regions of Australia.

**Symptoms:** Stem rust can cause major losses in both hay and grain yields (50-100% of yield) and dramatically impact hay appearance and feed quality. Stem rust can affect oat leaves, leaf sheaths, stem and panicles. Stem rust produces elongated pustules which erupt with tattered edges on both sides of the leaf. The pustules contain masses of reddish-brown powdery spores, which are easily dislodged and distributed by wind. As the season develops, the pustules may turn black with another spore stage. In severe cases, stem rust may weaken or damage the stem enough to cause lodging. The disease is more prevalent in high rainfall areas and in large biomass crops. The severity of disease is driven by inoculum pressure, variety resistance and environmental conditions.

**Disease epidemiology:** Similar to leaf rust, oat stem rust grows and reproduces only on living plants and must continually infect new hosts. It survives over summer by infecting volunteer or wild oats. Destruction of green bridge carryover is an important component of disease management. The disease cycle is similar to that of leaf rust with potential for long distance spread of spores by wind and a relatively short period between infection and sporulation. The optimum temperature for stem rust infection is higher than leaf rust (18–30°C), consequently outbreaks of stem rust may occur later in the growing season in wet and warm spring conditions.

**Management:** Control measures for stem rust are similar to those in leaf rust. It should be noted that varietal resistance for rust diseases can be independent and therefore a variety may be susceptible to one rust disease and resistant to the other. Given the capacity for rapid development and the severe impact of this disease, the threshold for fungicide application can be very low in susceptible varieties. Detection of stem rust pustules in a susceptible type can be sufficient to trigger the need for fungicide intervention.

**Future:** Similar to leaf rust, the epidemiology and management of this disease are well understood. The ongoing need with this disease is monitoring of regional pathotype virulence and screening of varieties for resistance response to dominant pathotypes. Screening for new sources of durable resistance continues to be of importance.

## Septoria avenae blotch

Septoria avenae blotch is a stubble borne disease, which is found throughout Australia but is most damaging in Western Australia where it is the most common oat disease. Recent paddock surveys have found this disease in the majority of paddocks surveyed, with severity being greatest in higher rainfall areas and seasons.

*Septoria avenae* blotch symptoms on oats. *Septoria avenae* blotch causes dark brown-purple lesions on oat leaves, which then develop to form brown blotches. Image courtesy of DPIRD.

**Causative agent:** Septoria blotch of oats is caused by the fungus *Phaeosphaeria avenaria* f.sp. *avenaria* (*Parastagonospora avenae*)

**Distribution:** Found across the grain growing region of Australia. Septoria blotch is most common in Western Australia.

**Symptoms:** Septoria is a major constraint to hay production reducing hay yield and quality (both appearance and feed quality). Septoria symptoms start as small oval to elongated lesions on the leaves. The lesions are dark brown- purple in colour. As the disease develops, the lesions increase in size to form brown blotches up to 2cm in size. Blotches can coalesce causing large areas of necrosis, which in severe cases can lead to premature senescence of the entire leaf. Lesions can also form on the leaf sheath and stem which can be grey-brown to black in colour. Very severe infections can cause lodging. Blotches can also form on the florets and glumes leading to black staining of seed in some varieties. Small black/ brown fruiting bodies (pycnidia) develop within the lesions. If the dead tissue becomes wet, it can become black with mould which could downgrade hay quality.

**Disease epidemiology:** Septoria blotch survives on crop residue from previous seasons. The fungus produces sexual fruiting bodies (perithecia) embedded in infected stubble in autumn. Following rain or heavy dew in late autumn and early winter, they will produce ascospores, which can be disseminated by wind over large distances to infect oat seedlings. These ascospores are thought to be the primary source of inoculum. Ascospores germinate on oat leaves and cause lesion development. A second type of fruiting body (pycnidia) are produced in the necrotic lesion area. The pycnidia produce asexual spores (pycnidiospores) which ooze out of the pycnidia in response to wet conditions and are spread within the surrounding plant canopy by rain splash. Pycnidiospores are the secondary source of inoculum. The severity of the disease in season is related to the number of generations produced in season. Disease severity is higher in early sown crops in high rainfall areas, where rain can readily disperse Pycnidiospores and multiple generations of disease can occur. Cooler winter temperatures can lengthen the disease lifecycle, and warm damp spring conditions can result in rapid disease development. Short (dwarf) or fast-maturing varieties where the upper canopy is more rapidly infected are more likely to be affected than tall or slow-maturing varieties.

**Management:** *Septoria avenae* blotch is a stubble borne disease, consequently a key plank of integrated disease management (IDM) involves avoidance of infested stubble through rotation of oats with non-host crops or stubble management, such as burning, to reduce disease carryover. Deployment of resistant varieties is an effective approach to reduce disease severity, however the current suite of hay and milling varieties is predominantly susceptible and selection of agronomically suitable resistant types is limited. Foliar fungicides can be applied for in season control of Septoria. Where disease occurs at early growth stages, applications around stem extension can reduce development of disease in the lower canopy and reduce development of secondary inoculum. Application following flag leaf emergence is recommended to protect the upper canopy from disease development during head emergence and flowering in environments where winter and spring rainfall will support disease development. Protection of the upper canopy from infection will retain green leaf area for optimum hay quality.

**Future work:** The general epidemiology of the fungus is understood, however the timing of ascospore release under Australian conditions and utilisation of this information to drive disease intervention is an important gap. As new more resistant varieties are released an understanding of any pathotype and virulence differences in the population is important. The impact of Septoria on oaten hay yields and quality above just the impact on green leaf area retention needs to be understood. Fungicide applications can control the development of disease but more work is required to determine the most economical spray timings, disease thresholds and number of fungicide applications to give best return on investment for hay production. An integrated disease management strategy that encompasses all foliar disease control would be ideal.

## Yellow dwarf virus (YDV)

Yellow dwarf virus (YDV) symptoms on oats. Infected plants show typical reddening of oat leaves. Image courtesy of DPIRD.

**Causative agent:** Yellow dwarf is caused by a group of aphid-borne viruses belonging to the family *Luteoviridae*. There are at least five strains. Three BYDV strains (PAV, MAV and RMV) and cereal yellow dwarf virus (CYDV) have been identified in Australia. As symptoms of the BYDV strains are similar, diagnosis requires serology or PCR analysis.

**Distribution:** Found across the grain growing regions of Australia.

**Symptoms:** BYDV is capable of reducing yield and quality of oaten hay. Affected plants can occur as a single plant, as a patch or in severe cases, the entire crop can be affected. BYDV causes leaf discolouration starting from the tip of the plant ranging from red, orange to yellow in colour depending on the oat variety. Areas of leaf and whole plants can die prematurely. If the dead tissue becomes wet, it can become black with mould which would degrade hay quality. Other plant symptoms include stunting, sterile tillers and aborted florets. Symptom severity depends on the time of infection, plant growth stage, environmental conditions, virus strain and oat variety. Early sown crops and long season crops sown in higher rainfall areas are particularly vulnerable to BYDV. Greatest yield impact of this disease occurs in plants infected at early growth stages, which can result in severe stunting of plants. Symptoms of BYDV can be similar to that of nutrient deficiencies, waterlogging, herbicide damage, plant stresses and some other plant diseases such as red leather leaf.

**Disease epidemiology:** YDV's survive year round in living grasses. The virus is transmitted predominantly by the oat aphid (*Rhopalosiphum padi*) and to a lesser extent the corn aphid (*R. maidis*). YDV's are persistently transmitted virus, meaning that aphids acquire them while feeding on infected plants and then transmit them when moving to and feeding on healthy plants. Winged aphid migrations occur primarily in autumn/ spring under warmer temperatures and these initiate primary infection in the crop from summer weeds and volunteers. Wingless and winged aphids moving through the crop are then responsible for secondary spread of YDV's.

**Management:** A suite of well-established IDM approaches exists for managing YDV's in cereals. Current strategies include managing alternate grass weed and volunteer cereal hosts adjacent to cropping paddocks to diminish virus reservoir and reduce aphid population. Choosing the appropriate sowing window to avoid exposing young plants to aphid vectors, sowing resistant varieties where disease risk is high and application of preventative insecticide treatments to protect crops against early season infection in the first 8-10 weeks after emergence (including systemic seed treatments and in season foliar treatments to control aphids).

**Future work:** Virus epidemiology and relevant management options are well understood. Further research work is not a high priority, however continued variety screening remains important.

# Grain diseases

## Smuts

Smut symptoms in oats. Oat florets are replaced with black powdery spores. Image courtesy of DPIRD.

There are two types of smuts able to infect oats, loose smut (*Ustilago avenae*) and covered smut (*Ustilago hordei*). Both are seed-borne diseases with similar symptoms, and are difficult to distinguish between in the field. Both diseases are managed in the same way. Smuts can severely affect the yield and appearance of oaten hay. The spores produced by the smuts have been shown to cause breathing difficulties in stock.

**Causative agents:** Loose smut is caused by the fungus *Ustilago avenae*, covered smut is caused by *U. hordei*. Covered and loose smuts can be distinguished by spore morphology but are difficult to distinguish in the field.

**Distribution:** Found throughout the grain growing regions of Australia.

**Symptoms:** Stunted plants with oat florets replaced with black powdery spores. The heads of affected plants may emerge earlier than the surrounding crop and are conspicuous amongst other normally developed heads. Symptoms of both smuts are similar; however, covered smut spores are enclosed by the lemma and palea in the oat floret. In susceptible varieties with repeated seasons without control, smuts can exceed 50% incidence.

**Disease Epidemiology:** Airborne spores, dislodged from infected plants, are distributed onto healthy plants and remain dormant on harvested seed until seeding. Mild and moist conditions during flowering favour smut infection. The spores germinate at seeding and infect the coleoptile of emerging oat seedling. The fungus grows systemically in the plant, which remains asymptomatic until head emergence where the oat florets have been replaced by masses of black smut spores. Smut incidence is higher when infected seed is sown early into a warm moist seedbed.

**Management:** Seed harvested from smutted crops is likely to have some level of infection. Where infection levels are high replace contaminated seed with seed from a clean source. Effective application of label rates of registered seed dressing products should reduce smut transmission levels to almost zero. The use of in-furrow fertiliser treatments are not effective for these diseases. Regular use of registered seed dressing fungicides should eliminate anything but trace levels of these diseases. Varieties with different levels of genetic resistance are available.

**Future work:** Disease including management options are well understood. Further work is not a high priority.

# Root diseases

## Crown rot

**Causative agent:** Crown rot is predominantly caused by the fungi *Fusarium pseudograminearum,* and *F. culmorum*. *Fusarium* species can be distinguished using molecular techniques, *in vitro* growth characteristics and conidia morphology.

**Distribution:** *Fusarium pseudograminearum* is found across the grain growing regions of Australia. *Fusarium culmorum* is more common in cooler higher rainfall zones.

**Epidemiology:** The crown rot fungus survives between seasons on infested crop residues. The same fungus causes crown rot in all cereals, including oats, barley and wheat. Infection occurs by direct root or sub-crown internode contact with infected plant residues. The fungus then colonises roots in moist soil but causes little to no damage at this early infection stage. As the fungus grows through the developing crown, the fungal hyphae blocks the xylem preventing translocation of water throughout the plant. The growth of the fungus and its impact are favoured by moisture stress.

**Symptoms:** Cereal oats are colonised by the crown rot fungi but rarely develop symptoms, which are apparent in other cereals. Oats can maintain or increase the inoculum levels. In moist soil, after senescence of affected plants, pinkish fungal growth may form at the crowns. Crown rot infection can impact growth and reduce yield in milling oats but to a far lesser degree than in wheat.

**Management:** Levels of *F. pseudograminearum* and *F. culmorum* in the soil can be determined using PREDICTA B soil testing which measures the amount of the fungal DNA present in the soil and provides a risk analysis for disease development. As a stubble / trash borne disease, the most effective approach is reducing exposure of the plant to the infected crowns from the previous year. Rotation with non-host (non-cereal) crops or pastures is recommended. Inter-row sowing to provide greatest separation of seed from infested crowns can provide some reduction in disease incidence. Sowing rate and nutrition strategies to reduce drought stress can help minimise disease expression. Oat varieties do not differ in response to crown rot. At the time of writing there are no registered fungicide options that eradicate crown rot or significantly yield loss from the disease in cereal crops, although there is one seed dressing registered for crown rot suppression in oats.

**Future work:** The impact of crown rot on hay physical and nutritional quality is currently unknown. It is possible that even in oats which has less obvious symptoms of infection than other cereals, that crown rot could impact on nutritional hay quality parameters due to the restriction of water throughout the plant. The implications of fusarium stem base infection for development of mycotoxins (eg. DONs) is not understood. The detection of DONs in hay samples suggests that crown rot could pose a risk for export markets. Further research is needed to explore the causes and implications of this.

Availability, registration and efficacy of fungicide seed dressings for crown rot suppression or control are a gap for oat production.

## Nematodes

### Cereal cyst nematode (CCN)

**Causative agent:** Cereal Cyst Nematode (CCN; *Heterodera avenae*)

**Distribution:** CCN is found across the grain growing regions of Australia but is most widely distributed in sandy and clay soils in South Australia and Victoria. It is not regarded as a pest in Queensland or most of New South Wales but is a problem in some parts of southern NSW and Western Australia.

**Symptoms:** CCN feed on the roots of oat plants, restricting nutrient and water availability to the host plant. Plants are generally affected in uneven patches of poor emergence or establishment throughout the paddock. Plants may be stunted with reduced tillering, and the leaves turning yellow and red in colour. Root symptoms in oats differ from other cereals; they appear ‘ropey’ and swollen. Severely affected plants will have stunted short, thickened roots. CCN can reduce oaten hay yields. Above ground symptoms can resemble root lesion nematodes, viruses, herbicide damage and nutrient deficiencies, however examination of root symptoms are distinctive. As weather warms in Spring, white pin head sized cysts full of eggs will appear on the roots, browning as they mature and eventually fall off into the soil in summer.

**Disease epidemiology:** CCN is a cyst nematode with one generation per year. The eggs of CCN hatch in autumn when stimulated by soil moisture, and root exudates and soil temperatures below 15°C. The juveniles travel through the soil and invade roots of susceptible plants. Once matured, males remain mobile and females develop a fixed feeding cell and become sedentary for the rest of their lifecycle. After mating the females continue to feed on nutrients and water from the plant roots; swelling as eggs develop internally. In spring the female gets large enough to break through the plant root wall, she then becomes visible to the naked eye as white cysts. After death, the female body hardens into robust hardened brown cyst, each containing approx. 200-300 eggs. The next season up to 85% of the eggs hatch while the remaining eggs remain dormant until the following year. As CCN is a sedentary nematode spread through soil is slow in natural circumstances, but can be moved widely in contaminated soil or plant material.

**Management:** CCN negatively impacts crop emergence and growth and is therefore detrimental to hay production. Levels of CCN in the soil can be determined by reputable diagnostic services or using PREDICTA B testing which can determine the number of nematodes present per 100g of soil. Crop rotation with non-host crops and pastures, growing resistant varieties, maintaining adequate nutrition and sowing early to improve plant establishment prior to eggs hatching are all methodologies suitable for reducing risk and impact of CCN on oat production.

No nematicides are currently registered for broadacre cereal crop production.

**Future:** Registered nematicides for CCN control.

## Root lesion nematodes (RLN)

**Causative agent:** Root lesion nematodes belong to the genus Pratylenchus. In Australia, there are three predominant RLN species affecting broadacre crops; *P. thornei*, *P. neglectus* and *P .quasitereoides.* Damage from RLN results in reduced yield and possibly quality of oaten hay.

**Distribution:** *Pratylenchus neglectus* and *P. thornei* are widespread across Australia while *P. quasitereoides* are only of importance in Western Australia (WA). *P. penetrans* is rarer but also damaging in WA.

**Symptoms:** Distribution in paddocks is uneven, so symptomatic plants appear in patches, or give the crop an undulating uneven growth pattern. .The nematodes are feeding on the crop root system so above group symptoms look much like nutrient and water deficiency; delayed establishment, reduced plant growth, reduced tillering, yellowing of leaves, wilting and reduced yields. Symptoms in roots include shallow and retarded with the loss of lateral roots , brown lesions. Sometimes the reduced branching giving roots spaghetti noodle like appearance. All Pratylenchus species cause similar symptoms.

**Disease epidemiology:** *Pratylenchus* spp. are migratory endoparasites of plant roots. This means that they enter the root, feed and either remain in the root system or exit and travel to another host plant. RLN cause damage to the host plants in two ways. Firstly, physical

Root lesion nematode (RLN) symptoms in oats. RLN can cause patchy uneven growth in oat crops. RLN root symptoms include reduced branching, which gives the roots a spaghetti noodle like appearance.  Images courtesy of DPIRD.

damage to the host root system when the nematodes break through the outer cortex to enter the root and move within the root. This also leaves the root exposed to entry by other pathogens. Most damaging when nematode numbers are high is the impact of the nematodes feeding from the plant roots; sucking water and nutrients that are usually reserved for plant growth and health. RLN survive over summer in a dehydrated state in the soil and roots. Once there is adequate moisture RLN can become active and invade plant roots. RLN number can increase very quickly in infested soils. They complete a lifecycle approximately every 30 days when soils are warm and moisture. It's estimated that three reproduction cycles are common in the southern regions of Australia. Four lifecycles may be possible in the warmer climates of northern NSW and Queensland.

**Management**: Levels of each of the RLN species in the soil can be determined by reputable diagnostic services or using PREDICTA B testing. Oats are resistant to *P. thornei*, moderately susceptible to *P. neglectus*, and susceptible to *P. quasitereoides* and *P. penetrans.* Control relies on crop rotation with resistant or non-host crops to reduce soil concentration of nematodes. In crop, good plant nutrition, particularly nitrogen can help plants to withstand some impacts of nematode attack. No nematicides are currently registered.

**Future work:** To determine if oat varieties vary in susceptibility to RLN species. The impact of RLN on hay appearance and feed quality is unknown. Registered nematicides for RLN control.

## Stem nematode

Stem nematode symptoms on oats. The two plants on the right are infested with stem nematode and are stunted with swelling at the base of the stem. Image courtesy of S. Taylor (PHA).

**Causative agent:** Stem nematode (*Ditylenchus dipsaci*).

**Distribution:** Found in the Southern grain growing regions of Australia (Victoria, New South Wales and South Australia). Stem nematode is not found in Western Australia, and is listed as a quarantine pest in the state.

**Symptoms:** Stem nematode can cause reduced emergence, stunted plants, swelling at the base of the stem, twisted tillers or rotting of the crown. Severe cases can result in plant death. Symptoms can be confused with herbicide damage, water-logging or nutrient deficiencies. Symptoms in oats will persist throughout the season.

**Disease epidemiology:** Stem nematode can be introduced on farm through infested seed contamination, hay straw, weeds, soil and other plant material. Stem nematodes can tolerate desiccation and remain inactive for many years. Once adequate moisture is available the nematodes become active again and can infect young stem tissue especially regions of the seedling that are below the soil surface. In moist conditions, the nematodes move up plant stems and distort regular plant growth. As plants reach maturity the nematodes dry out. Stem nematodes can reproduce 4-5 times per season. Stem nematodes are more prevalent on heavy soils and prefer cool, moist conditions.

**Management:** Identification of stem nematode can be determined by a reputable Plant Pathology diagnostic Service or using PREDICTA B testing. Crop rotation, controlling volunteer hosts and preventing the spread of infested plant material, seeds and soil can help with stem nematode control. Farmers in areas free of the oat race of stem nematode should adopt biosecurity practices to reduce the risk of introducing it onto their properties. Stem

nematode is not present in broadacre growing areas of Western Australia. If crops exhibit symptoms or soil results suggest a detection contact PaDIS (Pest and Disease Information Service) or the Senior Nematologist at DPIRD for help.

**Future work:** Registered nematicides for stem nematode control. Ongoing surveillance and biosecurity measures to retain area freedom for regions where stem nematode is not present.

## Pythium (minor disease)

**Causative agent:** *Pythium* species are pathogenic oomycetes.

**Distribution:** Found across the grain growing regions of Australia.

**Symptoms:** Affected plants can occur in patches or at paddock level. Patches predominantly occur in wet areas of the paddock. Seedling symptoms include stunting and pale colouration. Older plants can exhibit reduced tillering and in severe cases plant death. Roots symptoms include stunting, reduced lateral roots, and discolouration of root tips to yellow-pale brown colour. Symptoms can be confused with those of *Rhizoctonia* although *Pythium* is often associated with wetter area of paddocks unlike *Rhizoctonia*.

**Disease epidemiology:** *Pythium* spp. can survive in soils as oospores, which remain dormant until there is adequate moisture, and fresh plant tissue is detected. *Pythium* is more prevalent in cool wet seasons and in heavy acidic soils with high organic carbon levels.

**Management:** Best management strategies include crop rotation, improved soil drainage, weed control, adequate crop nutrition, and registered seed treatments.

**Future work:** Disease including management options well understood. Further work is not required.

## Rhizoctonia root rot

**Causative agent:** Rhizoctonia root rot (bare patch) is caused by the fungus *Rhizoctonia solani* AG8.

**Distribution:** *Rhizoctonia solani* is present across the grain growing regions of Australia.

**Symptoms:** *Rhizoctonia* causes crop damage by pruning newly emerged roots, ‘spear-tipped roots’, which results in water and nutrient stress to the plant, as the roots have been compromised in their ability to translocate both moisture and nutrients. Where disease is severe and infects seedlings after emergence, patches of severe stunting and plant death occur (rhizoctonia bare patch). If the disease develops later in the season uneven growth can occur. Oats are the most tolerant cereal crop, although they still exhibit crop damage and yield loss.

Rhizoctonia symptoms in oats. Rhizoctonia root rot can cause patches of severely stunted plants in the oat crop. Rhizoctonia can also cause root pruning also known as “spear tipping”.

**Disease epidemiology:** *Rhizoctonia solani* AG8 survives on crop residues just below the soil surface. The fungus is adapted to dry conditions and lower fertility soils. In autumn with opening rains, it forms a hyphal network in the top 5cm of soil, which can then infect roots of germinating seedlings. Disturbance of this hyphal network can help manage rhizoctonia. The fungus infects the plant roots preventing water and nutrient translocation throughout the plant causing water and nutrient stress.

**Management:** Levels of rhizoctonia pathogen in the soil can be determined using PREDICTA B testing which can help determine need for disease management in coming seasons. Rhizoctonia has a wide host range, although generally cereals are most affected. Rotation with oilseed or legume crops or weed free fallow can help reduce soilborne inoculum. Tillage or soil disturbance below the seed at sowing can promote rapid root growth and disrupt the fungal hyphal networks. Registered seed dressings and in-furrow fungicides are available to suppress rhizoctonia. Ensuring good root growth through good plant nutrition particularly nitrogen and avoiding herbicides that slow or damage root growth can help minimise losses to rhizoctonia.

**Future work:** The impact of rhizoctonia bare patch on plant growth is obvious; however, the impact of less severe infection on plant growth and on oaten hay yield and quality is not known. There is the potential that rhizoctonia could impact nutritional hay quality parameters due to the restriction of water and nutrients throughout the plant. Research in this area will help to determine return on investment of using seed dressings and in-furrow fungicides for rhizoctonia suppression.

## Take-all (minor disease)

**Causative agent:** Take-all is caused by the fungus *Gaeumannomyces graminis*. There are three variants of *G. graminis*, but only one, *G. graminis var. avenae* can infect oats.

**Distribution:** Present across the grain growing regions of Australia.

**Symptoms:** Take-all can cause blackened roots which can extend into the crown and lower stem. In some cases, the entire root system can be rotted. Early infection of plants can cause stunting, reduced tillering and in severe cases even plant death. Symptoms in adult plants can include whiteheads which often occur in patches, or in severe cases, can affect large areas of a paddock. Symptoms of take-all can be confused with crown rot and frost.

**Disease epidemiology:** The fungus survives on infected plant residues. The fungi then grow short distances, colonising the young roots of the host plant which includes all cereals and grass weeds. The fungal hyphae grow along the root, and infect growing into the centre of the root, causing it to blacken. The short growing season and early cutting of oaten hay means take-all generally a minor disease of oaten hay.

**Management:** Levels of the take-all pathogens present in the soil can be determined using PREDICTA B testing. Crop rotation with non-cereal crops is the main control for take-all and ensuring good grass weed control. Some fungicides applied as seed treatments or with fertiliser can give good control of take-all. However, their use on oaten hay crops is unlikely to be economic because take-all does not cause sufficient damage to this crop.

**Future work:** Disease including management options well understood. Further work not a high priority.

## Seed dressing and in furrow fungicides registered for use on oats– 2019-20

Updated 5th December 2019. This information is a guide only. Chemical labels should be read and rates should be checked before using fungicides. Mention of trade names does not imply endorsement of any company's products. Symbols used refer to footnotes. Crown rot suppression has been added to the table.

| Seed Dressing Active ingredient (fungicide/insecticide). Rate of product/100 kg/seed | Smut (covered and loose) | Pythium | Rhizoctonia | Crown rot suppression | Product examples by Trade Names |
|---|---|---|---|---|---|
| Carboxin + cypermethrin[a] | 60/70/125-250g or mL | – | – | – | Vitaflo®C, |
| Carboxin + thiram | 250-500mL | – | – | – | Vitavax® 200FF |
| Difenoconazole + Metalaxyl-M + Sedaxane | 180mL | 180mL | 360mL[3] | – | Vibrance™ |
| Difenoconazole + Metalaxyl-M + Sedaxane + Thiamethoxam | 325mL | 325 mL | 650mL[3] | – | Vibrance® Extreme |
| Flutriafol + Cypermethrin[a] (Flutriafol + Imidacloprid) | 100mL *(400 mL) | – | – | – | Vincit® C, Veteran® C, Vibrant® 25C, Apparent Flutriafol 25®, (Veteran® Plus) |
| Flutriafol + Metalaxyl-M + Imidacloprid | 400mL/(400mL) | – | – | – | Tri Power®, (Pontiac®) |
| Flutriafol with zinc | 400mL | – | – | – | Vincit® Zinc |
| Ipconazole + cypermethrin[a] | 100mL | – | – | – | Rancona® C |
| Ipconazole + metalaxyl | 80mL | 200mL[3] | 360mL[3] | – | Rancona® Dimension |
| Penflufen | 40 – 80mL | – | 40 – 80mL | – | EverGol® Prime[4] |
| Penflufen + Prothioconazole+ Metalaxyl | 130mL | 100mL | 130-260mL | 260mL | EverGol® Energy |
| Tebuconazole + imidacloprid[a] | 200mL | – | – | – | Hombre® Ultra, Proguard® Ultra |
| Tebuconazole+ cypermethrin[a] or (triflumuron[a]) | 100g *or* mL | – | – | – | Proguard® T, Tebuconazole 25C or T, Blaster® 25C, TebbyC®, Veto® 25C or T, Tebu- C® 25, (Raxil® T) |
| Triadimenol+ imidacloprid[a] | 400mL | – | – | – | Imid-Triadimenol *4*Farmers |
| Triadimenol + cypermethrin[a] or (triflumuron[a]) | 100mL or (100mL) | – | – | – | Phoenix® C, Vanguard® 150C, Seedpik 150 C , Baytan® T, Triadimenol 150C (Syngenta)/+ (4 Farmers); (Proleaf® T); Foliarflo® C |
| Triticonazole+ cypermethrin[a] | 100mL | – | – | – | Premis™ Protect, Premis® Pro C, Triticonazole 200 *4*Farmers |

**Note: 1)** A blank cell (-) indicates that product is not registered for use in the disease specified; **2)** ®Registered trademark; **3)** [3]Disease suppression only; **4)***Rates in brackets correspond to relevant product in brackets in the same row

**Insecticides: 1)** [a] Cypermethrin, imidacloprid and triflumuron compounds (insecticides) are effective against insect pests of stored grain. **2)** [b] Hombre® Ultra, Tri-Power®, Veteran Plus, Arrow Plus, Imid-Tridimenol,, Gaucho 600, senator 600 and Cruiser Opti are also registered for aphid control and the prevention of the spread of "barley yellow dwarf virus" (BYDV) and cereal yellow dwarf virus (CYDV) in cereal crops. Cruiser® 350FS contains thiamethoxam which, like imidacloprid, is also a neonicotinoid insecticide which registered to protect stored grain pests.

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## Foliar fungicides registered for use on Oats– 2020-21

This information is a guide only. Rates of registered products in mL/ha or g/ha. Chemical labels should be read and rates should be checked before using fungicides. Mention of trade names does not imply endorsement of any company's products. Symbols used refer to example product trade names. Please inform Ciara Beard of any errors or omissions, ciara.beard@dpird.wa.gov.au.

| Active Ingredient | Stem rust | Leaf (crown) rust | Septoria leaf blotch (suppression) | Example Product Trade Names<br>® = registered trademark | Withholding Period<br>(Harvesting or Grazing, in weeks) |
|---|---|---|---|---|---|
| Epoxiconazole 62.5g/L + Pyraclostrobin 85g/L | No registration | No registration | 500 | Opera®, Pirastar | Harvest- 0 if not applied after Z59,Grazing- 3 |
| Epoxiconazole 500g/L | No registration | No registration | 65 | Epoxiconazole® 500, Soprano® 500 | Harvest- not required when used as directed. Grazing-3 |
| Propiconazole 250g/L | 500 | 250–500 | 250–500 | Aurora® 250EC, Bumper®, Cracker® 250, Detour® 250EC , Procon® 250EC, Propicol® 250EC , Tilt® 250EC , Prestige®, Pace® 250EC, Restore® 250EC, Propiconazole® 250 & 250EC, Petulant® 250EC, Propeller®, Colossus® 250 | Harvest- 4, Grazing-1 |
| Propiconazole 435g/L | 285 | 145-285 | 145-285 | Propimax®, Fitness® | Harvest- 4, Grazing-1 |
| Propiconazole 500g/L | 250 | 125-250 | 125-250 | Throttle® 500, Prop® 500, Propiconazole 500EC, Tilt® 500EC, Propi Super® 500 | Harvest- 4, Grazing-1 |
| Propiconazole 550g/L | 230 | 115-230 | 115-230 | Cracker Jack® 550EC, Propiconazole® 550EC, Pacer® 550EC, Picaro Hi-Load® 550EC, Prestige® 550, Proforce Regiment® 550 | Harvest- 4, Grazing-1 |
| Propiconazole 625g/L | 200 | 100-200 | 100-200 | Bumper® 625EC, Propiconazole® 625EC | Harvest- 4, Grazing-1 |
| Propiconazole 750g/L | 170 | 85-170 | 85-170 | Cracker Jack 750EC | Harvest- 4, Grazing-1 |
| Propiconazole 250g/L and Tebuconazole 250g/L | 250 | 125-250 | 125-250 | Cogito® | Harvest-5, Grazing-2 |
| Prothioconazole 210g/L and Tebuconazole 210g/L | 300 | 300 | 150-300 | Prosaro® 420SC, Mantaray®420SC, Mightyzole® 420, Pro Grow® 420SC, Proteb® 420SC, Prothio T®, Prothio T® 420, Prothioconazole® and Tebuconazole®, Teb Pro® 420, Tebupro® 420SC | Harvest-5, Grazing-2 |
| Tebuconazole 430g/L | 145-2901<br>Or<br>1452 | 145-2901, 2 | No registration | 1: Laguna® 430, Orius® 430SC, Rebuke® 430SC, Zolo® 430SC, Stingray®, Tebazal® 430, Tebucol® 430, Tebucon® , Whirlwind® 430, Tebuconazole 430 & 430SC, Hornet® , Tebcon® 430SC, Toledo® 430EC, Cure®430, Teboo® 430SC, Tebby® 430, Tebuconazole®, Tripod®, Mayatebu® 430<br>2: Tusk® | Harvest-5, Grazing-2 |
| Tebuconazole 750g/kg | 83-166 | 83-166 | No registration | Tebuconazole 750WDG, Buzz Ultra 750 WG, Ultrateb® 750 WG | Harvest-5, Grazing-2 |
| Tebuconazole 800g/kg | 78-156 | 78-156 | No registration | Tebuconazole 800WG, Turbulence® 800WG, Laguna Xtreme® 800 WG | Harvest-5, Grazing-2 |

Continued overleaf

## Foliar fungicides registered for use on oats – 2020 (cont.)

| Active Ingredient | Stem rust | Leaf (crown) rust | Septoria leaf blotch (suppression) | Example Product Trade Names<br>® = registered trademark | Withholding Period<br>(Harvesting or Grazing, in weeks) |
|---|---|---|---|---|---|
| Sulphur 700g/Kg and Tebuconazole 45g/kg | 1370-2750 | 1370-2750 | No registration | Unicorn® 745WG | Harvest-5, Grazing-2 |
| Azoxystrobin 80g/L and Epoxiconazole 31.25g/L | 500-1000 | No registration | 500-1000 | Tazer® Xpert | Harvest-0 if not applied after Z59, Grazing-3 |
| Azoxystrobin 200g/ L and cypronazole 80g/L | 400-800 | 400-800 | 400-800 | AmiStar® Xtra | Harvest-0 if not applied after Z59, Grazing-3 |

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Note: Isis® 430SC is only registered for leaf (crown) rust (unlike other tebuconazole 430g/L products, it is not registered for stem rust)

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