

Serdc GROWNOTES™



SUNFLOWER SECTION 9 DISEASES

PRINCIPLES OF DISEASE MANAGEMENT | INTEGRATED DISEASE MANAGEMENT AT THE FARM OR CROP LEVEL | STEPS TO EFFECTIVE DISEASE MANAGEMENT | PROVIDING AN ACCURATE DIAGNOSIS | TOOLS OF THE TRADE | SUNFLOWER DISEASES: BIOLOGY, SYMPTOMS, MANAGEMENT | BIOSECURITY AWARENESS: POTENTIAL DISEASE THREATS FOR AUSTRALIAN SUNFLOWER | REFERENCES AND FURTHER READING



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http://www.grdc.com.au/ uploads/documents/GRDC_FS_ Sunflowerdisease.FINAL.pdf

http://regional.org.au/au/gcirc/3/430. htm

Diseases

9.1 Principles of disease management

9.1.1 IPM and biological control

Integrated pest management is sometimes confused with classical biological control. Classical biological control involves the importation and release of exotic control agents (predators and parasites) to control (usually) exotic pests. This practice is used because there are no native control agents, or because the native ones are (or are thought to be) ineffective.

Most plants are immune or resistant to almost all plant pathogens. This immunity is normal. However, occasionally a pathogen develops the ability to either bypass or overcome the inbuilt resistance mechanisms of a plant. When this occurs, the **host** is considered **susceptible** to the pathogen and the **pathogen** is described as being **virulent** on that host.

When a virulent pathogen comes across a susceptible host and the environmental conditions are suitable, a disease develops in the host and characteristic symptoms are produced.

The disease triangle of interactions between the host, pathogen and environment determines the initial severity of the disease outbreak (Figure 1).

Integrated disease management (IDM) involves the selection and application of a range of control measures that minimise crops losses, maximise returns, and ideally have minimal impact on the environment, flora and fauna.¹

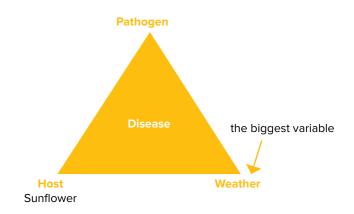


Figure 1: Interactions between host, pathogen and weather determines disease severity.

Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), https://bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3







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GRDC Tips and Tactics: <u>Sunflower</u> disease management

9.2 Integrated disease management at the farm or crop

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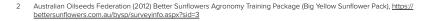
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Effective IDM should be integrated with that of the whole farm. Basis strategies should be implemented regardless of whether or not a significant disease problem exists. Prevention or minimisation of disease risk is the key to effective IDM.

Best practice strategies include:

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- **Disease awareness.** Ensure you have up-to-date information on the incidence and biology of current disease outbreaks, and on whether incidence/severity of outbreaks is increasing each season.
- **Staff training.** Educate staff on possible disease incursions and encourage proactive feedback on unusual symptoms and plant growth habits.
- **Farm hygiene.** Minimise movement of pathogens between paddocks and between farms. Remember many pathogens survive in the soil and plant debris and can easily be transferred on tyres, machinery, boots or plant matter such as hay.
- Resistant varieties. Use whenever available. Source information on hybrids of different genetic backgrounds to assist with minimising the build-up of specific pathogens; for example, rust races have specific virulence genes which will overcome some hybrids and not others.
- **Crop nutrition.** A healthy crop is more able to express its resistance/tolerance than a crop under stress. Be aware that some pathogens such as powdery mildew are more virulent on crops that have been grown under conditions of excess nitrogen.
- **Management of crop residues and weeds.** This minimises carryover and buildup of pathogens; for example, nearly all weeds host *Sclerotinia*.
- **Developing a sound crop rotation strategy.** Repeated plantings of sunflowers will lead to the build-up of soilborne pathogens including *Sclerotinia, Phoma, Phomopsis*, and *Verticillium*.
- **Regular crop monitoring.** Regularly check your crops for anything out of the ordinary. Do not hesitate to get a second opinion. Walk through the crop in a 'W' pattern; this will minimise the risk of missing disease hotspots and allow you to gauge any possible edge effects or disease gradients.
- **Knowledge update.** Ensure you understand the biology of the pathogens that will potentially infect your crop. Monitor according to risk analysis based on infection timing; for example, *Phoma* and *Phomopsis* species symptoms rarely become obvious before budding or flowering. Monitor regularly for powdery mildew, as in ideal conditions the short life-cycle can result in an epidemic, which could severely affect the crop within 2–3 weeks (Figure 2).²







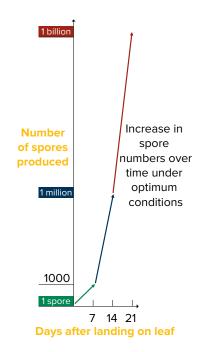


Figure 2: Potential exponential increase in powdery mildew spore numbers in a 2-3-week timeframe.

9.3 Steps to effective disease management

9.3.1 Risk assessment

Assessment of disease risk relies on the knowledge base of the end user (grower or advisor) with respect to their paddock history, their confidence in forecasts of both weather and possible price, their willingness to educate themselves about pathogen biology, and their tolerance for accepting risk.

Some disease management decisions can be made pre-planting; for example, a paddock with a history of heavy *Sclerotinia* infection in chickpeas would not be a good choice for sunflowers in the next rotation since both crops are susceptible to this pathogen. A cereal crop would be the best choice.

Other disease management risk factors involve both pre- and post-planting decisions. For example, pre-planting knowledge of the powdery mildew pathogen's preference for cooler weather means the grower understands that crops growing in the cooler ends of the planting season are more likely to suffer powdery mildew infection than crops growing in the middle of summer. Soil moisture levels permitting, planting times can be adjusted accordingly if a cool season is forecast.

Post-planting decisions would involve a risk assessment of allowing a powdery mildew outbreak to progress further up the plant than the bottom third, without chemical control measures. This is currently a difficult decision as yield loss threshold data are not available and the use of fungicides is the least desired control option.

Each crop/disease risk analysis will:

1. Identify the factors that determine risk.





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- **Pathogen:** pathogenicity, survival, transmission and infection mechanisms, availability of control measures, is it widespread or sporadic?
- Host: vulnerability, varietal reactions, availability of resistant lines, multiple hosts, seed dressings.

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- **Agronomy:** row spacing, soil conditions, cultural practices, plant residues, nutritional interactions, irrigation, dryland, herbicides, time of planting.
- Weather: weather forecasts including possible temperature variation, rainfall, relative humidity. Climatic conditions affect both plant growth and pathogen's biological responses. A stressed plant is vulnerable to disease outbreaks.
- **Risk management:** assess level of risk, contemplate ease of implementing management plan, assess costs of implementation, assess value of possible returns over known risk factors
- 2. Analyse specific known risk factors
- Pathogen: virulence level against particular hybrid, level of inoculum in air/soil/ seed, known paddock history, alternative weed hosts for either the pathogen or its vector, e.g. parthenium weed and the *Tobacco streak virus* (TSV) thrips vector.
- Host: susceptibility to pathogen, stress reactions to herbicides, nutritional disorders.
- Agronomy: weather outlook, time of planting, effectiveness of cultural control methods prior to planting, airborne inoculum levels built up during season.
- *Weather:* water storage in profile, long term forecast for rain or abnormal conditions, potential for water stress during growing season.
- Risk management: ensure strategy is flexible and adjust as necessary.
- 3. Acknowledge your own acceptable risk level
- **Low:** crop failure would seriously affect the farm's economic situation, not necessarily a good time to try new and untried cropping options.
- **High:** a risk of substantial losses if potential returns/financial rewards are high, a failure in the rotation would not unduly affect the potential earning capacity of the farm. ³

9.4 Providing an accurate diagnosis

9.4.1 Observations

Diagnosing plant diseases is not always quick or easy. Unlike insect pests, which are relatively easy to identify, the accurate diagnosis of plant diseases requires patience and, at times, a microscope.

A number of sunflower pathogens produce similar and confusing symptoms, so the diagnostician needs to keep an open mind until all aspects of the host– pathogen interaction are considered. It is possible that multiple causal organisms are involved, as was the case with a recent Phoma black stem and Phomopsis stem canker outbreak.

However, some pathogens do show characteristic symptoms, and with experience, a network of other specialists and suitable reference material, a reasonably quick diagnosis can be achieved. There is no substitute for having a hands-on approach to sunflower disease management—each season will bring its own unique mysteries to solve.

- **Be observant.** Analysing plant symptoms is just the first step in the diagnostic process. Attention to detail is essential.
- **Make a list.** List all possible culprits as you observe symptoms. Most will be discounted as you progress through your diagnostic analysis.



³ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3</u>



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Recognise a healthy plant. This seems obvious, but some varieties display idiosyncrasies which can be mistaken for early stages of a disease; for example, varieties with certain genetic backgrounds may display apparent swelling at the nodes, raising questions of possible viral infection or herbicide injury. Leaf colour variations may be confused with symptoms of viruses; long, leafy bracts or tubular-shaped petals may be genetic but excessive bract development may also be caused by herbicide injury or a phytoplasma. Ask your seed company representative or breeder about specific traits, if concerned.

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- **Check all parts of the plant.** Leaf symptoms can be the result of leaf pathogens, vascular tissue infection or a pathogen invading the roots.
- Note extent of the symptoms. Is the issue widespread, only an individual plant or clumps. Soilborne pathogens will often affect plants individually or smaller clumps (Figure 3).



Figure 3: Soilborne pathogens such as Sclerotinia minor, Sclerotinia sclerotiorum and Sclerotium rolfsii *infect individual plants or clumps if infection is severe. Basal lesions on these plants are typical of* S. minor.

Photo: S. Thompson, QDAF

- Are the symptoms uniform throughout the paddock? If so, are there any abiotic (non-living) reasons why the crop would be uniformly affected: for example, was a particular herbicide used in the previous rotation that may not have broken down as quickly as expected if weather conditions were particularly dry; was the appropriate rate of fertiliser applied or could the symptoms indicate a nutritional disorder; was a chemical applied off-label or in a mix that caused phytotoxicity? Could drift have been a factor?
- Widespread and even symptoms indicate abiotic factors. These might include soil conditions (deficiencies, toxicities, pH, excess salt in irrigation water), adverse climatic conditions (hail, drought, floodwaters, cold or heatwaves), toxic chemicals (inappropriate chemical usage, experimental products, growth regulators), human error.
- Pathogens take time to build up in a crop. Regular monitoring for insects and diseases, and weed hosts, will decrease the chances of unpleasant surprises later in the season. For example, damage caused by a high population of *Helicoverpa* at budding or flowering can lead to a high incidence of head and stem rots later in the season.
- Check for distinctive visual or smell symptoms. Is there ooze or an unpleasant odour? Bacterial infections such as *Erwinia* are most likely to be secondary and infect after damage by insects. However, under extremely wet conditions, bacterial infection may be the culprit (Figure 4).





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Figure 4: Sucking insects such as green vegetable bug can lead to secondary bacterial infections.

Photo: S. Thompson, QDAF

- Do the affected plants follow the row or are they randomly distributed across the paddock? Wilted plants along a row can be the result of mechanical damage where the scarifier has nicked the roots or stem at soil level allowing pathogens to colonise. Plants dotted through the paddock are more likely to indicate a soilborne pathogen such as *Sclerotinia, Verticillium, Macrophomina or Phomopsis.*
- Is there a disease gradient into the paddock? If so, has a neighbouring
 paddock or laneway had chemical applied that may be phytotoxic to your crop;
 or is a weed growing alongside your crop that may be host for a vector such as
 the thrips species, which transfer TSV.
- Is the problem restricted to one paddock? Is it across varieties, are plants at a certain growth stage affected, and did the previous crop show any similar symptoms? Some pathogens, e.g. *Sclerotinia, Macrophomina,* TSV, have a broad host range.
- Geographical distribution. Occasionally, pathogens are restricted to certain cropping areas. For example, to date TSV has only been recorded in sunflower in central Queensland (CQ) because the thrips vectors requires particular infected weed hosts which have not yet been found in the southern Qld and NSW cropping areas.
- Vigilance is essential. Do not assume that TSV will not be found outside CQ; as research on weed hosts continues, more hosts are being identified and it is possible TSV will eventually be identified elsewhere. Inter-farm hygiene is essential to limit the possibility of infected weed seeds/insects being transferred from CQ—clean contract headers, muddy ute tyres, boots, machinery.
- Know your pathogens. Understanding the life-cycles of the pathogens can help enormously with getting an accurate diagnosis. *Example 1.* Some pathogens do not show symptoms until budding–flowering, e.g. *Verticillium, Phoma* and *Phomopsis.* If a young plant is displaying a stem lesion prior to late budding–flowering, it is unlikely to be *Phoma* or *Phomopsis* infection—look for Sclerotinia base rot, insect damage and subsequent secondary rots, or TSV.

Example 2. TSV is commonly found in CQ and less likely to be found in southern



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Qld and NSW due to the lack of weed hosts for the vector, but keep an open mind. TSV, its thrips vectors and weed hosts could easily be found outside CQ; stem streaks are usually black, rather than brown. Do not hesitate to send any suspected TSV samples for testing to either the Plant Pathology team in Toowoomba or the virologists at Indooroopilly.

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Example 3. Macrophomina phaseolina (charcoal rot) may have infected the roots and lower stem early in the season but will not cause the plant to wilt or lodge until the plant is under stress during the head filling stage or during hot, dry weather conditions. Check pith in the lower stem for peppery, dark-coloured microsclerotia; stems often have a bleached appearance. If light brown or orange basal lesions are present, check for *Sclerotinia minor* or *Sclerotium rolfsii. Example 4:* Patches of poor emergence or damaged seedlings? Disease, poor vigour or lack of moisture may not be the cause. Mice will dig up freshly planted seeds retrieving seeds along many metres of row per night; rabbits, birds, cutworms and cockroaches will chew off newly emerged seedlings at ground level.

• Use your hand lens and knife/secateurs. Sacrifice an infected plant. Cut through stems. Discoloration of the pith–vascular tissue usually means a pathogen is present somewhere in the plant (Figure 5). Sclerotia or microsclerotia may be observed with a hand lens or with the naked eye if the pathogen is *Sclerotinia*. Check roots for discoloration, pruning and 'right-angle root syndrome'.



Figure 5: Black sclerotia of Sclerotinia minor inside stem base.

Photo: S. Thompson, QDAF

 Check new growth (Figure 6). Does the new growth have symptoms, or are the symptoms progressing as the plant ages, e.g. plants affected by low rates of herbicides such as SpraySeed[®] may produce very evenly spaced lesions and symptom-free growth as the plant ages.





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Figure 6: New growth growing away from symptoms of early herbicide drift. Note top leaves are free of symptoms.

Photo: N. Stevenson, Spackman & Associates

Know your herbicides. Chemical injury can easily be confused with disease symptoms; e.g. the twisting and stunting caused by 2,4-D damage is similar to the distortion of stems and leaves caused by TSV infection. Look for black streaks to assist with TSV field diagnosis (Figure 7).



Figure 7: Twisting of upper stem caused by TSV can be confused with herbicide damage. Photo: M. Sharman, QDAF



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Check watering schedules. Herbicides can accumulate in tail ditches of paddocks. Is there a gradient of symptoms up from the end of the paddock? Could these plants be waterlogged? Waterlogging can lead to nutritional deficiencies.

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Look for signs of fungal growth. Cut through the lower stems to look for sclerotia; the pith will have a peppery appearance if Macrophomina is present, large black sclerotia will point to Sclerotinia stalk rot, and caramel-coloured sclerotia at soil level will indicate an infection of Sclerotium base rot (Figure 8). Mycelium (fungal threads) growing on the heads may indicate Rhizopus head rot-check for insect damage. If roots are soft or dark coloured, pruned or poorly developed, a fungus is probably present although waterlogging can also be a culprit.



Figure 8: Mycelium (fungal threads) and protruding black sclerotia at the base of the plant. Sclerotinia base rot caused by Sclerotinia sclerotiorum. Photo: M. Ryley, QDAF

- Symptom variability can lead to an improper diagnosis. Environmental conditions, varietal differences and multiple pathogens infecting the one plant can cause symptom variability. Inspect a number of plants and note common irregularities. If in doubt, get a second opinion.
- Check soil compaction. Compacted soils and plough pans will often lead to 'right-angle root syndrome' (Figure 9). Roots are unable to penetrate through the impacted layers, and therefore grow horizontally. With poor root development, plants can develop symptoms of water stress, nutritional deficiencies and herbicide damage. Poor root development means plants are unable to access water and nutrients adequately and may show symptoms of a deficiency even though fertiliser may have been applied. In compacted soil areas, herbicides applied to prior crops may be unable to leach away. Products applied to the current crop may be concentrated in a small area of the root-zone and result in unexpected herbicide injury.





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Figure 9: Compaction leads to 'right-angle root' syndrome. Photo: K. McCosker, QDAF

 Ask questions (Figure 10). The more information you can gather about a site and affected crop, the better—crop rotational history, variety, herbicide, insecticide and fungicide applications for both this crop and previous crops, fertiliser applications, chemical applications in nearby paddocks. Wind and inversion layers can cause serious herbicide damage to non-target crops.



Figure 10: *Diagnostics is the result of a two-way flow of information.* Photo: S. Thompson, QDAF

- Listen, be aware. Many disease outbreaks occur in tandem with outbreaks in other cropping areas; for example, powdery mildew outbreaks in sunflower in 2008–09 were mirrored by unusually severe powdery mildew infection in tomatoes in the Burdekin, vegetables in the Lockyer and mungbeans in CQ. Although multiple powdery mildew species were involved in these outbreaks, all were favoured by the same environmental conditions. Being aware of changes in environmental conditions alerts the diagnostician to potential disease outbreaks.
 - **Ask for help.** Don't hesitate to contact other specialists. Working together will enhance the chances of an accurate diagnosis.



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So, what would you diagnose for the sample in Figure 11?

- Is this pith damage the result of intense *Phoma* infection or early stage *Phomopsis* infection?
- Or is the black stem discoloration early TSV infection?
- Is the lesion roundish, black and shiny, and therefore possibly Phoma?
- Or is it more brown than black, irregular or oval at the nodes and possibly *Phomopsis* infection?
- Then there is Sclerotinia stem rot—is the lesion the right colour and are sclerotia present? If so, what colour?

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• Or could it be insect damage with secondary *Erwinia* bacterial infection?



Figure 11: What diagnosis would you give?

Photo: S. Thompson, QDAF

A field diagnosis can be difficult. Symptoms may be confusing. If unsure of a diagnosis, send samples to a diagnostician (Figure 12). Sometimes, only laboratory isolations will provide the answer. (Regarding Figure 11, this stem lesion was identified as Phomopsis stem canker even though the lesion was very dark in colour—typical *Phomopsis* lesions are brown.) ⁴

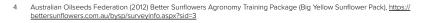






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Figure 12: Laboratory analysis may be necessary to confirm identification. Photo: M. Ryley, GDAF

9.4.2 Samples

- **Provide more than one sample if possible.** Discuss with the diagnostician the type of sample required, i.e. whole plants or plant parts. For small plants, send whole plants. If practical, send an unaffected plant for comparison (not always necessary or practical).
- Send information sheet with the samples. The more information given on the crop/location/environmental conditions/chemicals, fertilisers, insects, rotations etc., the easier it is for the diagnostician to make an accurate diagnosis. Send in a Disease Enquiry Sheet with each sample.
- **Provide samples in good condition.** Laboratory testing relies on good quality samples. A piece of stem or leaf that has been in the back of the ute all week is unlikely to retain viable fungal material.
- Phone or email your diagnostician. Samples sent can then be tracked if lost in transit. Samples inadvertently 'lost' in reception areas can be rendered non-viable if not refrigerated as soon as possible after arrival.

Sending infected plant tissue

Rust spores rapidly loose viability and die if samples are left in plastic, or if leaves sit in direct sunlight on the seat of a car in any temperatures particularly over 25°C. Send rusty leaves in paper bags only—never store in plastic.

Phoma and **Phomopsis** are relatively resilient in old dry stems, hence the length of time they remain a source of inoculum in the field. Cut lengths of infected stem and post/courier in paper envelope or box. Send head or seed sample with infected plant samples if mature. Both *Phoma* and *Phomopsis* species can be seedborne.

TSV, like all viruses, is difficult to detect, so good quality samples are essential for an accurate diagnosis. Place cut samples into a paper bag or newspaper, dampen the paper and then place the sample into a plastic bag before posting or sending by courier.



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Keep samples cool and send as soon as possible. Send more than one sample. For seedling death, include a healthy specimen. Email photos if possible.

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Caution: store samples in a fridge or Esky until posted or sent by courier. Try to avoid a situation where a sample may sit in the post office or courier's shed over the weekend. Wet or damp samples rot rapidly while in transit—if possible avoid collecting in the rain; paper towel is useful for drying samples if necessary. ⁵

9.5 Tools of the trade

Ute guides. Even specialists who work with plant diseases on a daily basis need reference material; symptoms can be variable and atypical under some environmental conditions and reference material is invaluable for comparison of symptoms. ⁶

Hand lens, magnifiers. These are essential when looking for fruiting bodies in lesions such as *Phoma*, *Phomopsis*, *Sclerotinia*, *Macrophomina*.

Knife or secateurs. These are invaluable for checking for damage to the pith (*Phomopsis, Macrophomina*) or vascular system infection (*Verticillium, Erwinia*).

Paper and plastic bags. Generally, placing a sample in a plastic bag and keeping it cool will be adequate to keep the pathogen alive until lab testing can be completed. If the sample has to be posted, ensure overnight delivery or the sample may rot in a plastic bag. If in doubt, use both plastic and paper bags. Note: for rusty leaves, always use paper bags or the moisture in the leaf will cause the rust spores to be non-viable if stored in plastic.

Esky and cooler bricks. Even if it is impractical to carry cooler bricks around, having a small cheap Esky in the car greatly increases the chances of the samples arriving at their eventual destination with the pathogen still viable. At the very least, keep all samples out of the sun.

GPS. Provides accurate location data. Samples sent on to the Queensland Herbarium provide important records of Australia's biodiversity and are a useful tool for monitoring disease spread and the potential locations for biosecurity outbreaks.

Felt pens. If more than one sample is collected from the site or multiple locations, having effective writing tools for the sample bags helps avoid later confusion. Biros and sunscreen infused paper bags cause undue frustration and biros invariably punch holes in plastic when trying to write a label. Keep a pencil handy.

Clipboard and sample information sheets. Collect the grower's details, crop variety and paddock history as soon as possible. Observe weeds in the vicinity for symptoms, e.g. wild sunflowers will often carry *Phoma/Phomopsis* lesions and rust; parthenium weed growing near a crop will indicate that TSV infection is possible.

Remember: fill in a Plant Disease Enquiry Sheet and send it with your sample. ⁷

9.6 Sunflower diseases: biology, symptoms, management

9.6.1 Rust (Puccinia helianthi)

Economic importance

Current hybrids generally have good levels of resistance so in recent years, serious losses due to rust have been uncommon (Figure 13). However, the pathogen is continually changing and resistance can be overcome. Without adequate resistance in hybrids, rust levels can quickly build causing severe epidemics and high yield



⁵ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://</u> bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3

⁶ GRDC (2011) Sunflower disorders: the ute guide. Ground Cover Direct, GRDC

⁷ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3</u>



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losses. Therefore, changes in the rust spectrum are constantly monitored by the QDAF and seed companies, with researchers at QDAF having identified over 115 rust races (pathotypes) to date. Breeding rust resistant hybrids is an ongoing process greatly assisted by gene pyramiding techniques.

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Figure 13: Selected rust-resistant hybrids. Photo: S. Thompson, QDAF

Survival and spread

In Australia, rust survives on volunteer sunflower plants and in the wild sunflower populations. Wild sunflowers are not only the source of inoculum at the start of each cropping season but also host a huge range of races (pathotypes) which survive at low frequency until a susceptible hybrid provides them with the ideal host and rust infection levels escalate.

Rust spores are air-borne and can be transported many hundreds of kilometres.

Significantly, although wild sunflowers are a source of new rust races, they are also a valuable source of disease resistance and utilised by plant breeders in Australia and internationally (Figure 14).



Figure 14: Wild sunflowers such as Helianthus debilis are a source of rust and resistance genes.

Photo: G. Kong, QDAF

Conditions for infection

Temperatures ~18–25°C favour development of the disease. Free water or dew on the surface of leaves is essential for infection. With adequate moisture and favourable temperatures, infection can occur within 12 hours but pustules containing the reddish brown spores do not develop for 7–12 days, depending on host and weather conditions. Successive cycles of wet weather or dews cause rapid development of the disease.





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Rust-resistant hybrids and recent drier climatic conditions can give the illusion that rust is no longer a disease issue in sunflowers. However, monitoring of the rust races present in wild sunflower populations, seed company nurseries and on susceptible hybrids such as confectionary lines indicates that pathotypes virulent on current hybrids are ticking over and could quickly increase to epidemic proportions given the right conditions.

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The re-releasing of some older hybrids which have little or no rust resistance may lead to an increase in rust incidence—monitor these crops carefully because, given the right conditions, their susceptibility means they are vulnerable to infection by multiple races which, like in past years, could result in severe yield losses (Figure 15).

Caution: planting rust susceptible varieties will lead to increased rust infection under favourable conditions.



Figure 15: Severe rust infection leads to yield losses. Photo: S. Thompson, QDAF

Symptoms

Small reddish-brown pustules will be seen on both the upper and lower leaf surfaces and sometimes on leaf petioles and flower bracts (Figure 16). Very high levels of infection can cause eventual death of leaf tissue. In some varieties, resistance declines at or following flowering and low levels of infection can occur. Unless severe infection occurs early, yield loss generally results from smaller, rather than fewer, seeds.





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Figure 16: *Small reddish brown rust pustules; heavy infection can blight the leaf.* Photo: S. Thompson, QDAF

Control

If practical, plant a mix of rust-resistant hybrids. Select varieties according to the planting time and the region. Avoid successive plantings of the same or susceptible varieties. ⁸

9.6.2 Powdery mildew (Golovinomyces cichoracearum)

Economic importance

The incidence of powdery mildew infection in sunflower has increased significantly in the past 5 years (Figure 17).

Sunflower is the only crop known to be attacked by *G. cichoracearum*. All other hosts are other members of the Asteraceae family such as wild sunflowers and ornamentals, e.g. *Zinnia*, *Dahlia*. Two other species of powdery mildew infect sunflowers overseas.

No data are available for economic yield loss thresholds caused by sunflower powdery mildew under Australian conditions. However, overseas literature suggests that under ideal disease conditions where infection levels are high, yield losses do occur. Plant death can also result if early infection is left untreated in young plants.



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Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), https://bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3



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Figure 17: Grey-white fungal colonies on leaf surface. Photo: S. Thompson, QDAF

Survival and spread

All hosts of sunflower powdery mildew are members of the Asteraceae family particularly the wild sunflower. Spores are wind-borne and will remain viable after spreading long distances under cool conditions. Spread up the plant can be rapid due to the short life-cycle and high rate of sporulation. Powdery mildew infection levels can develop from mild to epidemic proportions within three weeks under ideal conditions of cool weather and high humidity.

Conditions for infection

- Infection is favoured by high humidity, low light and temperatures of 20–25°C.
- Spores geminate within 2–4 h under ideal conditions.
- Short life-cycle of 5–7 days under ideal conditions.
- Spores disperse by wind leading to rapid movement between leaves and crops.
- Free water (rain or irrigation) on the leaf inhibits spore germination but the resulting humidity favours infection once leaves dry.

Symptoms

Fungal colonies first appear on the lowest leaves as powdery, greyish white spots on the upper leaf surface. Much of the fungal growth remains on the upper surface, the powdery appearance being the result of the production of conidia (spores). Although primarily a leaf pathogen, colonies can be found on stems and heads and bracts if infection is severe (Figure 18).



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Figure 18: Powdery mildew infection on stems. Photo: S. Thompson, QDAF

Strategies to minimise powdery mildew buildup

- 1. Cultural control
- Time of planting may influence the speed of disease build-up within the crop cooler temperatures at the beginning and end of the growing season can lead to increased infection.
- Avoid growing crops under conditions of high humidity—not often a practical option.
- Irrigate in the mornings to limit the buildup of humidity in the crop overnight (Figure 19)
- Overhead irrigation where the entire crop canopy becomes wet will lead to higher humidity within the crop—use droppers if possible.
- Overseas recommendations are that good air circulation within the crop can minimise disease development and heavy planting rate and narrow row spacing can lead to heavier disease pressure.
- But in Australia, conversely, experience gained while working on a number of fungicide trials in Queensland since 2009 has indicated that heavier plantings can be advantageous as dense plant stands ensure the leaves remain wet longer, thus helping minimise powdery build-up. It is often also noticeable that plants on the crop edges where air movement is highest carry more infection than those deeper in the crop. These findings are the opposite of those recorded overseas.
- These observations on the role of plant density on powdery mildew build-up under Australian environmental conditions need further investigation.



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Figure 19: Morning watering helps to limit humidity build-up overnight. Photo: S Thompson, QDAF

- Chemical control. The APVMA has granted an Emergency Use Permit (PER12045) for the fungicide TILT[®] 250EC (propiconazole) until 30 June 2014. Rate: 250– 500 mL product/ha.
- Maximum two (2) sprays per crop.
- Application timing: 21–28 days between applications.
- Last application: no later than at 5% ray floret emergence.
- Recommended timing and rate: under conditions of low to moderate infection, if
 powdery mildew is present in the bottom third of the canopy and moving into the
 middle third, then a single application of **500 mL/ha applied at the 5% ray floret**emergence stage will protect the crop until physiological maturity.
- When deciding whether to apply TILT®, consider the inoculum load in your own crop and surrounding crops, future weather conditions plus the crop stage cutoff for applications of 5% ray floret emergence. ⁹

9.6.3 Stem canker (Diaporthe/Phomopsis spp.)

Economic importance

Phomopsis helianthi is not recorded in Australia.

The first serious outbreak of Phomopsis stem canker in Australian sunflowers occurred in NSW and Queensland during the 2009 growing season. Current research has revealed that a number of previously undescribed *Phomopsis* species are responsible. Three newly described species have been named as: *Diaporthe (Phomopsis) gulyae* (highly virulent), *D. kongii* and *D. kochmanii* (low to moderate virulence) (refer Thompson et al. 2011). ¹⁰

Phomopsis helianthi is an exotic pathogen, not recorded in Australia but known to cause substantial yield losses overseas. To date, the isolates tested have NOT been identified as *Phomopsis helianthi*; however, our virulent Australian species appear to display almost identical symptoms (Figure 20). Mid-stem lodging and the associated loss of yield is the most significant impact of *Phomopsis*, with losses of 40–60% recorded overseas for *P. helianthi* outbreaks. Oil content can also be affected.

Vigilance is encouraged in all situations where *Phomopsis* outbreaks occur—the aim is to limit the spread of these pathogens, determine the extent of infected cropping

i MORE INFORMATION

www.apvma.gov.au



GRDC Update Paper: <u>Disease control</u> <u>in summer crops and management</u> strategies to minimise financial losses



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⁹ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://</u> <u>bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3</u>

¹⁰ Thompson SM, Tan YP, Young AJ, Neate SM, Aitken E, Shivas RJ (2011) Stem cankers on sunflower (Helianthus annuus) in Australia reveal a complex of pathogenic Diaporthe (Phomopsis) species. Persoonia 27, 80–89.



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area and continue to identify the complex of pathogens responsible. Biosecurity Awareness is essential.

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Figure 20: Phomopsis infection leads to mid-stem lodging, lesions at multiple nodes, yield loss.

Photo: L. Serafin, NSW DPI

Dispersal and spread

Phomopsis survives in plant debris with small black/brown pycnidia (fruiting bodies) developing in the dry stalks as conditions become favourable. Spores can be dispersed by wind over short distances with raindrop splash and irrigation enhancing spread (Figure 21).

Phomopsis can be seedborne, so seed production nurseries should be monitored for any signs of this disease.

Queensland researchers have found that, depending on the *Diaporthe* species, living volunteer plants of crop hosts and living plants of weeds in paddocks and adjacent areas can act as the 'green bridge' between highly susceptible crops, while colonised dead plants and stubble of crop and weed hosts can act as the dead or 'brown bridge' between major crops. Almost 30 months after the severe *Diaporthe* lodging event in sunflower crops on the Liverpool Plains, *D. gulyae* was isolated from the 'brown bridge' of sunflower and Noogoora burr stubble lying on the soil surface after zero till farming practices and two cereal crops planted into the sunflower stubble.¹¹

The study revealed that *Diaporthe/Phomopsis* species have wider host ranges than previously thought and it is considered likely that the same will be found for other groups of pathogens such as the *Fusarium* species which are also opportunistic colonisers of both live and dead plant tissues.

It is apparent that these fungi form a group of pathogens/saprophytes that are potentially capable of surviving on both 'brown' and 'green' bridges between growing seasons in the northern region and that the role of weed stubble in aiding survival has been largely unrecognised. Since the introduction of zero and minimum-tillage systems, crop and weed stubble is commonly found across the various cropping



¹ S Thompson, S Neate, YP Tan, R Shivas, E Aitken (2016) Weeds and crop stubble as a pathogen 'host' or reservoir. What species are involved with what crop impact—implications for management. GRDC Update Paper 1 March 2016, <u>https://grdc.com.au/Research-and-Development/GRDC-Update-Papers/2016/03/Weeds-and-crop-stubble-as-a-pathogen-host-or-reservoir</u>



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systems of the northern region. An inoculum reservoir can be found in these residues regardless of the presence of the primary crop host.

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The impact of strategic tillage on the survival of these groups of pathogens in crop and weed residues under Australian conditions is largely unstudied. Crown rot researchers, (Simpendorfer et al. NSW DPI) have looked at the role of tillage for the *Fusarium* crown rot pathogen in Australia and multiple tillage investigations have been completed by overseas researchers.

A GRDC funded project has been initiated with the aim of looking more intensively at alternative hosts and survival of the *Fusarium* species on sorghum as well as early studies on the impact of burial on infected sorghum. ¹²

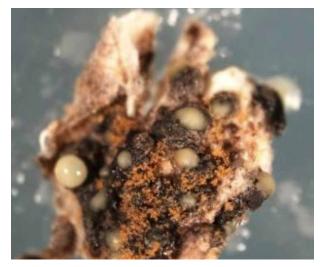


Figure 21: Spores oozing from Phomopsis-infected pith. Photo: S. Thompson, QDAF

Symptoms

The first symptoms appear on the lower or middle leaves around the leaf margins usually around the time of budding or flowering. Small necrotic areas, sometimes showing a chlorotic border, quickly merge and infect the leaf veins. Leaves die off rapidly as the infection moves down the leaf petiole to the node where the characteristic light brown lesion develops.

Lesions are always centred on the axils and start as small brown sunken spots, which can rapidly develop into an elongated, light tan to dark brown lesion, up to 20 cm in length. Lesions may appear water-soaked and vary in colour from olive greenish brown (often water-soaked) to pale caramel brown to dark brown with a black edge (Figures 22, 23).

Small black fruiting bodies (pycnidia) may be visible in the lesion if infection is advanced.

The fungus rots the pith behind the lesion eventually leaving the stem hollow. This results in stem weakness, sometimes wilting and leaf necrosis if infection is severe, and subsequent lodging as the head fills if pith damage is advanced.



¹² S Thompson, S Neate, YP Tan, R Shivas, E Aitken (2016) Weeds and crop stubble as a pathogen 'host' or reservoir. What species are involved with what crop impact—implications for management. GRDC Update Paper 1 March 2016, <u>https://grdc.com.au/Research-and-Development/GRDC-Update-Papers/2016/03/Weeds-and-crop-stubble-as-a-pathogen-host-or-reservoir</u>



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Figure 22: Phomopsis pale brown stem lesions. Photo: S. Thompson, QDAF



Figure 23: Phomopsis brown black stem lesions. Photo: S. Thompson, QDAF



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(i) MORE INFORMATION

http://www.grdc.com.au/uploads/ documents/2010ASG CEditedPapersPDF/ Thompson_Phomopsis StemCancer_edited_paper.pdf

http://www.dpi.nsw.gov.au/__data/ assets/pdf_file/0011/249779/ Sunflower-production-guidelines-forthe-northern-grains-region.pdf

http://www.grdc.com.au/Media-Centre/Ground-Cover/Ground-Cover-Issue-106-Sept-Oct-2013/Fungusposes-new-sunflower-disease-threat



Infection conditions

Phomopsis infection is favoured by wet conditions from late budding through to flowering. Disease severity depends primarily on climatic conditions and plant growth stage. Optimal temperatures are 23–25°C but the fungus will grow at temperatures 14–32°C. Characteristically, plants display brownish lesions dotted regularly up the stems at the nodes. Symptoms usually occur from budding through flowering to maturity. Depending on the severity of infection, mid-stem lodging may occur, not necessarily at a node or the site of infection.

Control

- *Phomopsis* survives on plant debris so effective cultural control methods are essential.
- Burying crop residues minimises inoculum build-up.
- Avoid consecutive plantings of sunflowers.
- Use non-susceptible crop rotations.
- Ensure no infection is present in seed production blocks.
- Excess nitrogen encourages *Phomopsis* incidence.
- In Australia, no products are registered for the chemical control of *Phomopsis* on sunflower.
- Some tolerance has been identified in screening trials overseas.

Important note. Infected seed will appear normal until after favourable weather conditions occur—fruiting bodies (pycnidia) will then develop on the seed coat (Figure 24). Laboratory testing is necessary to determine whether *Phomopsis* or *Phoma* infection is present. Mixed infections of *Phoma* and *Phomopsis* can occur - symptoms can be confusing. Send samples for laboratory identification. Send suspect samples to the QDAF Plant Pathology Laboratory, Toowoomba, Queensland (203 Tor St, Toowoomba, Qld).

Remember: *Phomopsis* can be seedborne. Ensure seed production blocks are disease-free.



Figure 24: Pycnidia on infected seed may spread infection to new sites. Photo: S. Thompson, QDAF





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Caution: *Phoma* and *Phomopsis* lesions can be difficult to tell apart. Field diagnostics can be confusing with lesion colours and sizes varying according to lesion age and crop susceptibility.

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As a general rule: *Phoma* lesions are black, shield-shaped and shiny; limited, if any pith discoloration. *Phomopsis* lesions are more oblong, brown or brown/black, sometimes have a water-soaked appearance; may have pith damage behind the lesion (Figure 25).¹³



Figure 25: *Pith damage behind a* Phomopsis *lesion.* Photo: S. Thompson, QDAF

9.6.4 Phoma black stem (Phoma spp.)

Economic importance

Although previously recorded as a minor pathogen of sunflower crops in Australia, an outbreak of a mix of *Phoma* and *Phomopsis* species (Figure 26) occurred in both NSW and Qld sunflower crops in the 2009 growing season.

Generally, *Phoma* produces a surface lesion that causes minimal damage to the crop. Researchers overseas report that this pathogen has been known to cause some damage to the pith and subsequent lodging but this is not usually seen under Australian conditions.

The economic importance of this pathogen is considered low unless infection occurs in conjunction with other pathogens such as *Phomopsis* spp., *Fusarium* spp. or bacterial pathogens. When pith damage occurs due to secondary infection, plants may lodge.

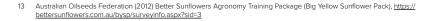






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Figure 26: Black shield-shaped lesions dot the stems at the nodes, mixed infections of Phoma and Phomopsis can occur.

Photo: S. Thompson, QDAF

Conditions for infection

Disease severity depends primarily on climatic conditions and plant growth stage. Most sunflowers are susceptible but a degree of tolerance can be observed in some lines.

Phoma infection is favoured by moist conditions from late budding through to flowering. Raindrop splash and irrigation enhance spread. Insects may also spread the pathogen particularly if they drill into the stem or leaf petiole.

Many *Phoma* spp. have been reported as seedborne but the pathogen will also survive for long periods on infected stubble or trash.

Dispersal and spread

Phoma overwinters as small black pycnidia and/or mycelium in infected stubble, and can be transmitted by seed. Optimum temperature for spore germination is 25°C with conditions of high moisture for 24 h being optimal. At lower temperatures, longer periods of free water are required. Spores will germinate at temperatures of 5–30°C as long as free water is consistently present.

Phoma spores travel for short distances on the wind and are more easily transported by rain splash. Overseas, leaf-feeding insects have been recorded overseas as transmitting the pathogen.

Symptoms

Phoma infects via the leaves as wind-blown or rain-splashed spores. Leaf spots merge until they meet the veins, which then turn black. The infection travels down the petiole to the stem where a shiny, jet-black, round to oval shaped lesion develops centred on the node. Lesion size ranges from 1 to 5 cm, usually only affecting the epidermal layer and does not penetrate the pith. Recent studies from Russia indicate that the pith may become damaged in some circumstances.

Small black pycnidia, tiny fungal fruiting bodies, may be visible in the lesion using a hand lens but often laboratory isolations may be necessary to make a diagnosis.



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If field inoculum levels are high, *Phoma* may infect the roots of young seedlings leading to a girdling lesion at the soil line. Girdling may result in stunted plants, smaller heads, blackened pith and poor seed set if the plant does not succumb to lodging.

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Caution: *Phoma* infection may be confused with *Phomopsis* symptoms. *Phoma* produces smaller, darker (black) lesions than *Phomopsis* (Figure 27, 28), causing minimal damage to the pith and does not usually lead to lodging. *Phoma* infection may also be confused with early TSV infection—both can form black lesions at the node (Figures 27, 29 and 30).

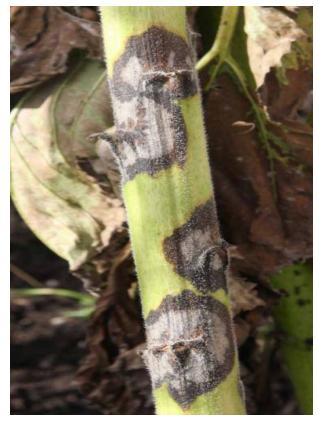


Figure 27: BLACK shield-shaped Phoma lesion. Photo: S. Thompson, QDAF





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Figure 28: Early-stage BROWN Phomopsis lesion, often elongated with a darker edge.

Photo: S. Thompson, QDAF



Figure 29: Phoma surface lesion. Photo: S. Thompson, QDAF



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Figure 30: TSV petiole and node infection—note blackening of pith and streak forming below node.

Photo: S. Thompson, QDAF

Control

- Phoma survive on plant debris so effective cultural control methods • are essential.
- Burying crop residues minimises inoculum build-up.
- Avoid consecutive plantings of sunflowers.
- Use non-susceptible crop rotations. •
- Ensure no infection is present in seed production blocks.
- Excess nitrogen encourages Phoma incidence. •
- No totally resistant sunflower lines are available, to date, but some resistance/ • tolerance has been observed in some wild Helianthus spp. and sunflower cultivars (Figure 31).
- In Australia, no products are registered for the chemical control of Phoma on sunflower.¹⁴



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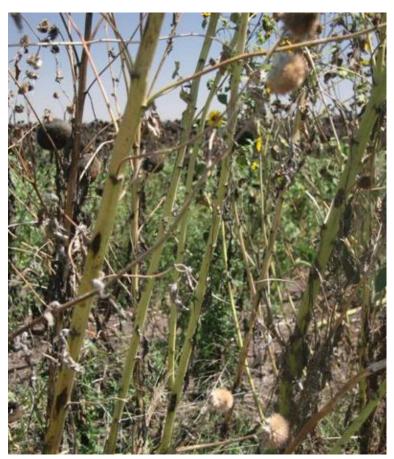


Figure 31: Wild sunflower host Phoma and Phomopsis species. Photo: S. Thompson, QDAF

9.6.5 Tobacco streak virus

Economic importance

Prior to 2009, a severe sunflower decline disease, now known to be caused by Tobacco streak virus (TSV), caused significant losses across the sunflower industry in CQ. ¹⁵ GRDC research has found with careful management of potential disease sources around crops and the use of tolerant cultivars, the risk of TSV in CQ can be largely minimised. However, TSV remains a significant concern for CQ sunflower growers with the potential for infection levels of 20–60% in susceptible cultivars if environmental conditions are conducive to disease outbreaks. TSV has not been found in sunflowers outside of CQ and as such should not currently be considered a risk in other regions.

Conditions for infection

TSV is transmitted only in pollen and seed of some hosts. It infects healthy plants via the feeding wounds of thrips, which allow TSV-infected pollen to enter. TSV disease is favoured by climatic conditions that enable high thrips populations to develop, and large amounts of infective pollen to be produced by host plants such as parthenium. These conditions generally occur during warmer months, and are highly dependent on rainfall and weed growth patterns.

The life-cycle of thrips is shorter during summer months, which allows population numbers to increase rapidly. Thrips populations will generally increase as weed growth increases following intermittent rainfall, while prolonged periods of rain over



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¹⁵ QDAF (2006) Central Queensland sunflower disorder—Tobacco streak virus. Fact Sheet 2006. Department of Agriculture, Fisheries and Forestry, Queensland.



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many days may reduce thrips populations. The highest rates of virus transmission will occur when thrips numbers are high and there are large areas of a flowering, virus host plant, such as parthenium. These conditions can result in high TSV disease rates in susceptible crops as large quantities of virus infected pollen is moved rapidly into crops by high numbers of thrips. Plants are most susceptible at the seedling stage.

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TSV disease incidence is generally much lower during the dryer, cooler months of the year as thrips numbers are lower, their reproduction slows significantly and there is less virus-infected pollen produced by alternative weed hosts. TSV disease incidence has also been much lower during the very wet summers of 2010–11 and 2011–12 when grasses have been dominant over parthenium in many regions. Thrips populations also appear to have been reduced by regular, large rain events. Summers of intermittent rain following dry winters appear to be most favourable for development of large populations of both parthenium and thrips, which results in more TSV disease.

Dispersal

TSV-infected pollen may be dispersed significant distances by wind or thrips (Figure 32). TSV is seed-transmitted in some alternative hosts such as parthenium, crownbeard, cobbler's pegs and fleabane. In particular, TSV is seed transmitted in parthenium at high rates and may remain viable for several years in ungerminated seed in the soil. There is significant risk of long distance dispersal if infected parthenium seed is moved with machinery or harvested goods. Research to date indicates that TSV is not seed-transmitted in sunflower.

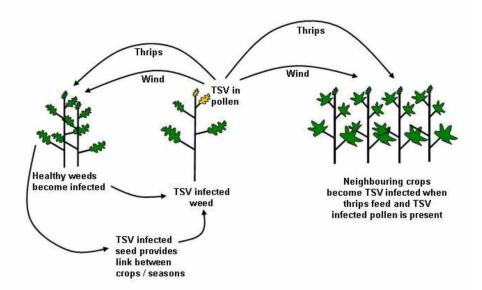


Figure 32: Disease cycle of TSV.

Survival

TSV can survive only in living plant hosts or in seed of some of those hosts (e.g. parthenium). It can survive in pollen grains but the length of time is unknown and probably varies depending on the host plant and climatic conditions. It does not survive in soil or on dead plant material.

Symptoms

The symptoms of TSV on sunflowers include: black streaks on the stem and leaf stalks, stunted growth, deformed growing tip, yellow and/or necrotic blotches on leaves, shortened internodes, plant death (especially in plants that become infected in early stages of development) and lodging of older plants due to weakened stems and blackened pith (Figures 33, 34, 35, 36).





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Figure 33: TSV leaf symptoms. Photo: M. Sharman, QDAF



Figure 34: Characteristic black streak caused by TSV. The dark necrotic streaks running down the stem and the curling over and necrosis of terminal growth is distinctive.

Photo: M. Sharman, QDAF



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Figure 35: TSV causes twisted stems and deformed heads. Photo: M. Sharman, QDAF



Figure 36: Early stages of stem necrosis caused by TSV with streaks starting from the leaf nodes. TSV usually causes dark streaks below and above the node. Can be confused with Phoma infections, which cause dark shield-shaped lesions at the node (see Figure 23).

Photo: M. Sharman, QDAF



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MORE INFORMATION

GRDC & QDAF Crop Note: Central

Sunflower disorder (Tobacco streak

Queensland sunflower disorder.

QDAF Sunflower Production:

virus or TSV).

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Control

Planning:

All research indicates that TSV is currently only a problem for sunflowers grown in CQ where the key alternative host, parthenium weed, is common. However, there is a real risk that TSV can be moved to new regions via infected seed of alternative hosts such as parthenium or fleabane, so vigilance in all regions is recommended for suspect symptoms in sunflowers and the appearance of parthenium.

 Determine and where possible, avoid areas of high risk such as locations either with a history of TSV or next to areas with a high density of parthenium (e.g. neighbouring grazing paddocks). Planting upwind of high risk areas may help to reduce transmission of TSV into crops; higher rates of disease are commonly seen downwind of these high-risk areas.

Hybrid tolerance:

GRDC funded research has demonstrated that there can be significant differences between some sunflower hybrids in their tolerance to TSV infection. Rating for susceptibility in these trials was based on whether plants could produce harvestable seed; hence, only severely affected plants were rated as susceptible. Trials are continuing. For further information please refer to the September 2009 issue of Cropping Central Magazine or the article 'Sunflowers—a real option for 2012', Cropping Central, Issue 52, p. 6–8 or contact the QDAF Virology unit.

Ground preparation:

• Maintain crop hygiene; particularly control of parthenium in or upwind of the crop both prior to planting and during the early stages of the crop.

In-crop:

It is unlikely that in-crop applications of insecticides for thrips control will to provide effective control of TSV transmission into the crop and may disrupt effectiveness of integrated insect management systems. QDAF trial results on various seed treatments showed no significant difference to TSV transmission by thrips. ¹⁶

9.6.6 Alternaria blight (*Alternaria helianthi*, *A. alternata*)

Economic importance

Under favourable conditions, Alternaria blight can defoliate plants within a week and cause yield losses as high as 70%. Fortunately, conditions favouring epidemics of Alternaria blight occur infrequently and tend to be more common in subtropical regions such as CQ.

Dispersal and spread

Alternaria spores are airborne. The pathogen survives in wild sunflower populations, volunteer plants or on infected plant debris. Epidemics of *Alternaria* infection will rapidly blight and defoliate crops under suitable wet, warm conditions.

Conditions for infection

Development of the disease is favoured by warm (26–30°C) wet weather. Infection is highly dependent on long periods of leaf wetness. Rain periods lasting for several days cause the disease to develop rapidly. Airborne spores produced on necrotic (dead) tissue initiate new infections. Seedling and flowering plants are highly susceptible. Older (lower) leaves are more susceptible.



¹⁶ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3</u>



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Symptoms

Roughly circular, dark brown to black necrotic lesions develop on leaves, petioles, stems, flower bracts and petals (Figures 37, 38, 39). Lesions on leaves may be surrounded by a yellow halo (Figure 37). Under favourable conditions, lesions expand rapidly and coalesce to form large dead areas, commonly described as blighting (Figure 38). Petiole infections can result in rapid leaf death. Infections generally cause premature leaf senescence.

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Figure 37: Alternaria leaf lesions—a yellow halo may surround lesions. Photo: S. Thompson, QDAF



Figure 38: Severe infection will blight the leaf. Photo: G. Kong, QDAF





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Figure 39: Alternaria stem lesions. Photo: S. Thompson, QDAF

Control

- Select those hybrids which show some level of tolerance. (Currently, there is no immunity in hybrids).
- Avoid successive plantings.
- Ensure infected, older standing stubble is removed quickly if younger crops are growing nearby.
- Practice effective cultural control methods where infected stubble is buried.
- Be aware that periods of sustained leaf wetness—warm, drizzly weather—are ideal for rapid spread of this disease. ¹⁷

9.6.7 Sclerotinia rot (Sclerotinia sclerotiorum, S. minor)

Economic importance

Losses due to this disease can be quite high, but its occurrence is generally restricted to late plantings because the disease develops at low temperatures.

(i) MORE INFORMATION

GRDC Update Paper: <u>Disease control</u> in summer crops and management strategies to minimise financial losses



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¹⁷ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://</u> bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3



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Survival and spread

Both species of *Sclerotinia* produce hard black sclerotia composed of compacted fungal strands and can survive many years in the soil.

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Sclerotia can be physically transported in plant debris, boots, machinery and floodwaters.

Sclerotia can germinate in two ways: (1) by producing mycelium (fungal threads) which infect roots and lower stem leading to base rot (Figures 40, 41); or (2) by producing a fruiting body (apothecium, dish-like) which fires spores out into the crop when triggered by relative humidity and temperature (Figure 42). These airborne spores are responsible for stem and head rot.

Sclerotia formed in the heads can be transported with seed and be difficult to sieve out from a seed sample if clumped together.



Figure 40: Sclerotinia minor *basal lesion and stalk shredding*. Photo: S. Thompson, QDAF





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Figure 41: White mycelium growing on stem lesion caused by Sclerotinia minor. Photo: M. Ryley, QDAF



Figure 42: Apothecium produced by a sclerote to fire spores out into the crop. Photo: M. Ryley, QDAF





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Conditions for infection

Cool (<18°C) moist conditions favour the germination of sclerotia in the soil, which can either directly infect roots or produce airborne spores that infect flowers and sometimes stems and petioles.

Symptoms

Plants with infected roots wilt and die. Fluffy white mycelium and hard black sclerotia are formed at the base and inside the stems of infected plants (Figure 43, 44).

Infected heads show a light brown rot on the back of the head, which may extend down the stalk. Rotted heads eventually fall apart, leaving only the fibrous strands of the stalk (Figure 45). Sclerotia form in the rotted tissue.



Figure 43: Sclerotia in infected stem. Photo: S. Thompson, QDAF





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Figure 44: Hard black sclerotia of Sclerotinia sclerotiorum will sometimes be accompanied by white mycelial growth. Photo: G. Kong, QDAF



Figure 45: Shredding of head after infection by Sclerotinia sclerotiorum; sclerotia fall to the ground. Photo: G. Kong, QDAF









(i) MORE INFORMATION

Better Sunflowers – Disease Management Library.

Better Sunflowers - Disease Management Library. Population structure of Sclerotinia sclerotiorum on sunflower

Better Sunflowers – Disease Management Library. Genotypeisolate interaction for resistance to Sclerotinia sclerotiorum in sunflower.



Control

Adopt a planting strategy that avoids paddocks with a history of *Sclerotinia* and ensure the crop is not flowering during cool weather.

Sclerotinia is a pathogen of many broadleaf crops and weeds—soybeans, french beans, cowpea, broccoli cauliflower and other vegetables and legumes . If a crop becomes infected, plant cereal crops in that area for at least 4–5 years.

Ensure seed production nurseries are kept free of Sclerotinia to avoid transferring the disease to all cropping areas. $^{\rm 18}$

9.6.8 Sclerotium base rot, crown rot (Sclerotium rolfsii)

Economic importance

Sclerotium rolfsii is currently considered a pathogen of low importance as usually only scattered plants in a field are affected. Hot, dry weather and lighter soil types can predispose plants to infection particularly if the crop is planted into undecomposed residues. Irrigated crops can also be severely affected as sclerotia are easily spread by water.

Dispersal and spread

Sclerotia survive in the soil for many years. Infected crop residues are the most significant source of infection. Repeated plantings of sunflower in the one paddock lead to an increase in inoculum in the soil. Good farm hygiene and rotations helps limit the spread of this disease.

Symptoms

A rot develops at ground level and it is usually covered by whitish fungal threads in which small caramel—brown-coloured sclerotia form (Figures 46, 47). The white mycelium can spread over the soil at the base of the infected plant (Figure 48). Plants eventually wilt and die.



Figure 46: Distinctive orange–brown lesion and white mycelium at soil level and on the roots.

Photo: M. Ryley, QDAF



⁸ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://</u> bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3



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Figure 47: Caramel–brown-coloured sclerotia in infected tissue. Photo: S. Thompson, QDAF



Figure 48: *Mycelium (fungal threads) may grow across the top of the soil.* Photo: T. Gulya, USDA

Infection conditions

Sclerotium rolfsii is a common soilborne pathogen and is most serious in warm humid weather with optimal temperatures of 27–30°C. Planting sunflowers into undecomposed residues presents the highest risk. Root damage also leads to increased infection levels. High temperatures and high relative humidity favour infection.

Control

Control is difficult as *S. rolfsii* has a host range of >500 species across 100 plant families, both mono and dicots. It effectively colonises non-host plant debris and can live as a saprophyte until a more favourable host is planted.

- Rotate with less susceptible crops such as cereals.
- Bury stubble/residues.
- Control highly susceptible weeds. ¹⁹



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¹⁹ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3</u>



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9.6.9 White blister (Albugo tragopogonis)

Economic importance

Sunflower appears to be able to sustain high levels of leaf infection (up to 50%) without significant yield loss. Epidemics of white blister are infrequent, due to the specific environmental conditions required for infection. However, in recent years, there has been a trend to cooler conditions both early and late in the growing season so *Albugo* is a pathogen that may become more significant in Australian crops. Overseas, crop losses occur due to both lodging and head infection. The 2005–06 season was the most recent season where white blister was significant in northern NSW.

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Survival and spread

Albugo survives in infections in wild sunflower populations, volunteer plants or resistant oospores in infected plant debris. The oospores germinate and produce zoospores, which then infect young seedlings. White spores are produced in large numbers on the leaves and are spread by wind and rain.

Conditions for infection

Cool (<20°C) moist conditions, either from rainfall or dew, are required for infection. These factors have usually limited the occurrence of white blister in Australia, when cool wet conditions do not often occur during the growing season.

Symptoms

Raised pustules containing the whitish spores develop on the underside of leaves, causing the leaf surface above the pustules to have a blistered yellowish-green appearance (Figure 49). Stems and petioles can also be infected, but pustules do not develop. Instead, infected areas have a grey bruise-like appearance due to the presence of oospores, the overwintering phase of the fungus (Figures 50, 51). Severe stem infections have not been recorded in Australia, but in South Africa, lodging is not uncommon (Figure 52).

Because *Albugo* is so sensitive to cool weather conditions, bands of infection may be found in layers up through the crop as infection develops only when cool wet conditions prevail.



Figure 49: Upraised blisters on upper leaf surface. Photo: R. Kloppers, Pannar Seeds, Republic South Africa





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Figure 50: Head infection. Photo: G. Kong, QDAF



Figure 51: Greyish coloured Albugo stem infection (blisters) can lead to lodging. Photo: G. Kong, QDAF





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Figure 52: Severe infection of Albugo causing lodging in susceptible line, Republic of South Africa.

Photo: G. Kong, QDAF

Control

Many Australian hybrids have high levels of resistance to *Albugo*. Avoid early planting, although infections that occur early in the life of a crop will not progress as temperatures increase through the growing season. 20

9.6.10 Charcoal stem rot (Macrophomina phaseolina)

Economic importance

This pathogen is found worldwide, has a wide host range, and is present in most sunflower-growing areas. Yield losses in sunflowers are usually due to a reduction in head diameter and/or seed weight, premature plant death and occasionally lodging. Oil composition can also be affected.

Survival and spread

Macrophomina produces microsclerotia, which will survive for many years in the soil. Sclerotia in the soil or on infected plant debris can be easily spread by poor farm hygiene practices.

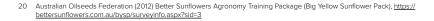
Conditions for infection

Infection levels may become serious when excessive heat or drought follow periods of good growth, i.e. a stressed crop with high soil temperatures (>35°C).

Sclerotia in the soil or on infected plant debris are the primary source of inoculum. Soil temperatures of $30-35^{\circ}$ favour disease development with mycelium colonising the roots after the sunflower plants reach anthesis and invades both xylem and phloem of the lower stem, growing upwards if conditions are ideal.

Symptoms

Symptoms usually appear after flowering when plants can die rapidly particularly is the plant is stressed. High soil temperatures and low soil moisture leads to more severe disease symptoms.





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Small black sclerotia form on the inside of the stem resulting in the distinctive peppery appearance to the pith (Figure 53). An ashy grey to silvery grey stem discoloration may be present although some lesions may be darker (Figure 54). Stalk fibres become shredded and covered with very small black sclerotia and pycnidia, which form on the outside of the stem. If infection is particularly severe heads may be infected and display the characteristic peppery appearance throughout the entire head (Figure 55, 56).

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Sclerotia and pycnidia can also be found on seed of both infected and healthy plants, the seed from healthy plants being infected from airborne pycnidiospores. Infected seed in soil at 35°C will result in severe damping off of the seedlings. If the soil is cool, healthy seedlings will be produced. Infected plants die prematurely with discoloured, bleached or ash grey stalks.



Figure 53: *Microsclerotia producing the distinctive 'salt and pepper' appearance of the pith.*

Photo: S. Thompson, QPIF





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Figure 54: Bleached stems: severe Macrophomina infection in a waterstressed crop.

Photo: S. Thompson, QDAF



Figure 55: Charcoal rot infection in the back of a sunflower head. Photo: G. Kong, QDAF





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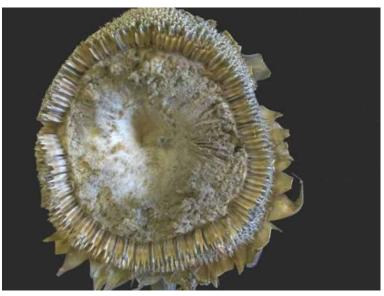


Figure 56: Severe charcoal rot infection of the head.

Photo: G. Kong, QDAF

Control

- Avoid any cultural practices that stress the crop. Any stress will predispose sunflowers to attack by *Macrophomina*—herbicide damage, water stress, leaf loss, excess nitrogen, low potassium.
- Sources of resistance/tolerance have been identified overseas including a number of drought-resistant hybrids.
- Fungicides can act as effective seed treatments.
- Because Macrophomina has an extensive host range of >300 genera (both monocots and dicots), eradication of this pathogen is not practical. However, host preference has been recorded, indicating it is possible that, for example, corn, sorghum following a sunflower crop would assist with limiting the build-up of the pathogen.
- Avoid very susceptible crops in your rotation if you have had a problem with Macrophomina. Soybean, navy bean, mungbeans are very susceptible and could cause more inoculum build-up.²¹

9.6.11 Rhizopus head rot (Rhizopus spp.)

Economic impact

This disease rarely causes economic losses but some infected heads can be observed in most crops following rain periods. Seed from infected heads are often lighter, are lower in oil content and higher in free fatty acids. Hulls and embryos can be discoloured.

Survival and spread

Rhizopus spores are airborne and readily infect any damaged tissue. *Rhizopus* is a common and widespread fungus that can attack many hosts under most summer crop growing conditions.

Conditions for infection

Rhizopus infects the head through damage caused by insects such as *Helicoverpa* and Rutherglen bugs as well as by birds and hail (Figures 57, 58). Humid wet weather favours infection and large numbers of spores are spread on the wind.



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²¹ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3</u>



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Brown, sunken, water-soaked lesions develop on the back of the head around wounds (Figure 59). As the mycelium invades the tissue, the interior of the head becomes soft, rots and turns brown.

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Black sporangia will give the inside of the head a peppery appearance and is a diagnostic feature that distinguishes *Rhizopus* infection from that of *Botrytis* and *Sclerotinia* (Figure 60). To add to the confusion, head infections of *Macrophomina* (charcoal rot) appear similar when sclerotia are embedded in the head.

Infected heads dry prematurely, may shrivel and appear to shred. Fungal threads may form on the shredded material. If rot is severe, the head may fall off. In extremely hot conditions, the head may mummify.



Figure 57: Insect damage can lead to Rhizopus infection after wet conditions. Photo: S. Thompson, QDAF





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Figure 58: Severe Helicoverpa damage followed by bird damage caused while searching for Helicoverpa, and then Rhizopus infection. Photo: S. Thompson, QDAF



Figure 59: Rhizopus head rot. Photo: S. Thompson, QDAF





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(i) MORE INFORMATION

<u>Better Sunflowers – Disease</u> Management Library.

Figure 60: *Rhizopus infection showing black sporangia.* Photo: T. Gulya, USDA

Control

- Controlling insect pests at or before flowering is the most effective way to minimise disease incidence.
- Varieties with more upright heads are more prone to infection.
- Sources of genetic resistance have been identified but incorporating the resistance into commercial hybrids has not been considered a priority.²²

9.6.12 Botrytis head rot or grey mould (Botrytis cinerea)

Economic importance

Botrytis usually does not cause significant losses although long periods of cool wet weather will increase the incidence.

Survival and spread

Botrytis can survive as infection in many hosts, as sclerotia in soil or infected seed. Sclerotia and seed can easily be spread by farm equipment, boots and irrigation.

Conditions for infection

Cool wet conditions favour the pathogen with 17–27°C being the optimal temperature range. Free water is required and germination is stimulated by exudates from pollen. If sunflowers are infected pre-bloom, an infection can remain latent for up to 9 weeks.

Symptoms

Head infection is the most characteristic symptom of *Botrytis* on sunflower (Figure 61). Soft brown spots form on the back of the head or bracts usually during maturation. In conditions of high humidity, a fuzzy, grey mycelial growth may cover the back of the



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²² Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3</u>



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head giving the disease its common name of grey mould (Figure 62). Heads develop into a spongy rot. Characteristically, no sporulation occurs within the head.

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Severe *Botrytis* infection can cause petiole lesions, stalk lesions and wilted leaves. Infected seeds will result in pre- and post-emergence damping off with characteristic grey mycelium on the dead seedlings as a diagnostic feature.

This disease is easily confused with early head infection caused by Sclerotinia head rot and Rhizopus head rot. *Botrytis*-infected heads do not disintegrate during the advanced stages of the disease but the hulls and infected seeds are highly flammable when driers are used.



Figure 61: *Botrytis symptoms occur at flowering.* Photo: T. Gulya, USDA



Figure 62: Grey mycelial growth—grey mould. Photo: T. Gulya, USDA





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- Late infections closer to harvest have little effect on yield due to the fungal infection remaining superficial.
- If levels of *Botrytis* infection in the crop are initiated at flowering and are excessively high, early desiccation at ~30% moisture is recommended if suitable driers are available. Flammability of infected seed can be an issue.

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- Chemical control is unfeasible.
- Some research groups have reported differences in susceptibilities between hybrids and wild sunflower species but no resistant hybrids are available.²³

9.6.13 Verticillium wilt (Verticillium dahliae)

Economic importance

Losses from *Verticillium* in Australia are usually not significant although many confectionary lines lack resistance and could show more susceptibility.

Survival and spread

Verticillium is both seedborne and soilborne and can survive for many years in the soil without a host. Small microsclerotia in plant residue will remain dormant until root exudates break dormancy as the roots advance. Microsclerotia will also contaminate the seed coat and mycelium can be found inside the seeds therefore ensuring seed transmission is a significant source of infection.

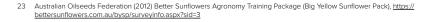
Conditions for infection

Verticillium is a soilborne pathogen of low importance but can infect at temperatures up to 30°C and cause early plant death. Root systems can become infected by secondary invaders—the disease is usually only found in isolated plants or small clumps.

Symptoms

Characteristic yellow-brown mottling between the veins appears around the time of flowering (Figure 63). Leaves wilt and rapidly dry out as the pathogen infects the vascular tissue in the stem and releases toxins. Earliest symptoms appear on the lower leaves as pale yellow, chlorotic spots, which enlarge, turn brown and become necrotic, often with halos surrounding the necrotic tissue. Caution: may be confused with leaf symptoms of boron deficiency.

Severely diseased plants will be stunted and have a smaller head diameter. The vascular tissue of the stems will show a brownish discoloration (Figure 64) and roots may be poor. The plant may remain upright with only the leaves wilting but the leaf mottle will become more severe and move up the plant.









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Figure 63: Characteristic Verticillium leaf symptoms. Photo: G. Kong, QDAF



Figure 64: *Discoloration and infection of the vascular tissue.* Photo: T. Gulya, USDA

Control

- Crop rotations may not control Verticillium as the microsclerotia are so longlived. Immune crops such as corn, grain sorghum, barley and lucerne (alfalfa) have little impact on Verticillium levels in the soil as the pathogen will often survive superficially on the roots of non-hosts as well as surviving in the soil for long periods.
 - Soil fumigation is effective but not practical for sunflowers in a broadacre situation.



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Seed treatments offer effective control of seedborne infection but do not protect the plant from soilborne infection. $^{\rm 24}$

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9.6.14 Septoria leaf spot (Septoria helianthi)

Economic importance

This pathogen rarely causes economic damage in Australia. Infection may become severe in warm moist conditions.

Survival and spread

Septoria survives on crop debris, volunteer plants and wild sunflowers. Spores produced in small black fruiting bodies on the leaf are spread during wet and windy weather.

Infection conditions

Warm moist weather favours this pathogen.

Symptoms

Characteristic angular, diamond or angular necrotic lesions (Figure 65) appear brown on the upper leaf surface (Figures 66, 67) and a lighter grey-brown on the lower leaf surface. Lesions may develop dark margins and light centres, which are often surrounded by a yellow halo. With use of a hand lens, small black fruiting bodies may be visible in the necrotic tissue.

Lowest leaves are infected first, then infection spreads to the upper leaves. Spots may coalesce, producing large, irregularly shaped dead areas of leaf, which can wither and dry.



Figure 65: Grey-brown angular lesions on underside of leaf. Photo: G. Kong, QDAF



²⁴ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://</u> bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3



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Figure 66: Brown-coloured Septoria leaf symptoms with darker edge and halos. Photo: G. Kong, QDAF



Figure 67: Distinctive brown lesions: Septoria can be confused with Alternaria infection if halos are present (as in Figure 66), but Alternaria lesions are black. Photo: S. Thompson, QDAF





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Control

 Destroy volunteer plants and ensure plant debris is incorporated. Crop rotations help limit disease build-up.

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Can be seedborne—use clean seed. ²⁵

9.7 Biosecurity awareness: potential disease threats for Australian sunflower

9.7.1 Downy mildew (Plasmopara halstedii)

Economic importance

Australia is the only sunflower-growing continent that has not recorded downy mildew as a sunflower pathogen. Losses vary according to the percentage of infected plants in the crop and are due to plant death, smaller heads, lighter seed weights and lower oil content. Downy mildew is seedborne and soilborne, with its oospores (survival structures) capable of surviving for as long as 8–10 years in the soil. Although seedborne infection is uncommon and results in a very low percentage of systemically infected plants, vigilance is essential when importing sunflower seed into Australia from overseas. The disease would be extremely difficult or impossible to eradicate once it is established.

The impact on native species has not been determined.

Survival and spread

Plasmopara halstedii is mainly a soilborne pathogen that survives in seed, soil and plant debris. Oospores in the soil or plant debris germinate to produce sporebearing structures called sporangia. Zoospores that are released from the sporangia germinate and serve as primary inoculum infecting young sunflower seedlings via the roots.

Infection is possible via seed but this is uncommon; most of the seed from an infected plant is non-viable and not all seeds are infected.

Conditions for infection

Cool moist conditions and poorly drained soils, especially clay soils, favour the development of this disease. Moisture and temperature are the most important environmental factors affecting infection and spread. Zoospores require free water to retain viability and actively move towards infection sites with the aid of a flagella or tail. Consequently, rainfall or intensive irrigation is a prerequisite for the initiation of primary infection.



²⁵ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://</u> bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3



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Figure 68: Downy mildew leaf infection. Photo: G. Kong, QDAF

Symptoms

The type and severity of symptoms will depend on the inoculum load, plant part attacked (Figure 68), age of host at the time of infection and environmental conditions. Symptoms are usually classified as either systemic or localised.

Root and seedling infection is usually the result of systemic infection (Figure 69). Damping off (pre-emergent seedling death) and seedling blight (post emergence death) result from soil infection and cool waterlogged soils.

Systemic root infection leads to stunted plants and chlorotic puckered leaves (Figure 70). If systemically infected plants reach maturity, the head bears few, if any viable seeds and the heads face vertically upwards (Figure 71).

A white layer of downy growth may be produced on the underside of leaves. Sporangia are produced on the downy mycelial growth and are dislodged by rain and wind leading to localised or secondary infection within the crop. Lesions are angular, delimited by veins, chlorotic, and with age turn necrotic and may coalesce.

Soilborne inoculum is the major source of infection; infection can be carried from wild sunflower populations near crops, irrigation water runoff or by wind.



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Figure 69: Downy mildew infection on seedlings. Photo: G. Kong, QDAF



Figure 70: Downy mildew symptoms—chlorotic twisted leaves and deformed head. Photo: G. Kong, QDAF





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Figure 71: Stunted head, twisted and discoloured leaves. Photo: G. Kong, QDAF

Control

- Roguing of infected plants helps to reduce inoculum build-up.
- Seed treatments such as fungicides containing metalaxyl are used effectively overseas.²⁶

Plant breeding for resistance is ongoing overseas as a number of races of this pathogen have been identified ²⁷. Multiple sources of resistance have been identified including *Helianthus argophyllus* recently collected from the only known Australian site at Yeppoon, Queensland by QDAF researchers. Even with the use of resistant cultivars, seed dressing with fungicides is highly recommended to prevent underground infection of the seedlings.

Overseas, chemical control of downy mildew involves a combination of seed dressings and systemic foliar fungicides containing metalaxyl and related compounds. ²⁸

9.7.2 Broom rape (Orobanche spp.)

Prohibited import: Orobanche cumana not recorded in Australia.

Economic importance

- If introduced to Australia, the economic impact would be significant.
- Australian sunflower cultivars lack resistance.
- There are limited in-crop herbicides options available for Orobanche control.
- Contaminated land has limited cropping options.
- The potential host range of Australian native species is unknown.
- Host plants are deprived of nutrients and water and will become stunted or die. Yield losses would occur.



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²⁶ Virányi F, Oros G (1991) Developmental stage response to fungicides of *Plasmopara halstedii* (sunflower downy mildew). Mycological Research 95, 199–205. <u>http://www.sciencedirect.com/science/article/pii/S0953756209810125</u>

²⁷ Gulya TJ, Sackston WE, Virányi F, Masirevic S, Rashid KY (1991) New races of the sunflower downy mildew pathogen (*Plasmopara halstedii*) in Europe and North and South America. Journal of Phytopathology 132, 303–311. <u>http://onlinelibrary.wiley.com/doi/10.1111/j.1439–04341991.tb00125.x/abstract</u>

²⁸ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3</u>



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Survival and spread

Enormous numbers of small seeds are produced by each parasite. Wild sunflower populations and volunteer plants would assist migration of the parasite through the sunflower growing areas. The seed is small and light—easily dispersed by wind, water and livestock.

Conditions for Infection

Orobanche is a parasitic weed that obtains all its nutrients through contact with the root systems of its host plant. *Orobanche cumana* germinates at 15–20°C. *Orobanche* have evolved to germinate in response to host plant root exudates. Huge numbers of seed are produced by each parasitic plant; survival time is estimated to be up to 10 years.

Symptoms

A single stem grows upwards to ~38 cm in height, has no chlorophyll, yellow alternate leaves and off-white to yellow flowers borne in a spike (Figure 72). Multiple stems may develop (Figure 73). Flowers continue to produce seed after the plant has been removed from its host.



Figure 72: Orobanche species parasitise their host. Not recorded in Australia. Photo: G. Kong, QDAF





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Figure 73: *Multiple stems of* Orobanche *may parasitise a plant.* Photo: T. Gulya, USDA

Control

- Dig up parasite and host plant—the host will help identify the species of *Orobanche*.
- Mark the site and inspect regularly for regrowth.
- Fumigate the soil.
- Clean any machinery and ensure boots and clothing are free of seeds.
- Herbicides may be effective but *Orobanche* has developed resistance.
- Orobanche has also overcome resistance bred into crops.
- Physical and cultural control measure should be strictly enforced. ²⁹

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²⁹ Australian Oilseeds Federation (2012) Better Sunflowers Agronomy Training Package (Big Yellow Sunflower Pack), <u>https://bettersunflowers.com.au/bysp/surveyinfo.aspx?sid=3</u>



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