CHICKPEA

SECTION 9

DISEASES

KEY DISEASE MANAGEMENT STRATEGIES FOR CHICKPEAS | FUNGAL DISEASE MANAGEMENT STRATEGIES | INTEGRATED DISEASE MANAGEMENT | RISK ASSESSMENT | ASCOCHYTA BLIGHT | PHYTOPHTHORA ROOT ROT | SCLEROTINIA | PHOMA STEM ROT | ROOT ROTS INCLUDING DAMPING-OFF (FUSARIUM, RHIZOCTONIA AND PYTHIUM SPP.) | COLLAR ROT (SCLEROTIUM ROLFSII) | FUNGAL DISEASE CONTROL | VIRUSES
Diseases

Key messages

• Several foliar fungal diseases, some seedling root diseases, viruses and root-lesion nematode can affect chickpea (Table 1).
• The most significant fungal disease of chickpea is Ascochyta blight. Disease management of chickpea should primarily focus on Ascochyta blight.
• Chickpea crops in southern Australia are being hit by a more virulent strain of the damaging ascochyta blight.
• The diseases Botrytis grey mould, Botrytis cinerea, and Sclerotinia white mould (Sclerotinia sclerotiorum and S. minor) were major diseases of chickpea prior to the incursion of Ascochyta blight, and may again become significant diseases in chickpea varieties resistant to Ascochyta blight. 1
• Integrated disease management in chickpeas involves paddock selection, variety choice, seed dressing, strategic fungicide use and hygiene.
• Implement an appropriate Ascochyta blight strategy based on rain forecasts and the level of varietal resistance.
• Using a number of integrated disease management (IDM) techniques is more likely to control diseases.
• Stay up to date with local Crop diseases forecasts for your region.

In northern Victoria and southern NSW the important disease constraints to chickpea production are Ascochyta blight (caused by Ascochyta rabiei) and grey mould (caused by Botrytis cinerea) (Tables 1 and 2).

Unlike northern NSW, Phytophthora root rot is not a widespread production issue and has not been detected in southern chickpea producing areas. These fungal pathogens have the potential to reduce crop yield and seed quality.

Disease management strategies for both diseases have been developed that utilise a range of chemical and non-chemical approaches, such as paddock selection, crop rotation, selection of seed for sowing, variety selection, sowing date and rate, and the strategic use of fungicides (both fungicidal seed dressings and foliar fungicides).

Producers still rely heavily on fungicides and success is dependent on correct disease identification, timing of product application and fungicide choice. These strategies are available from the Pulse Australia website. 2

Virus management aims at prevention through integrated management practice that involves controlling the virus source, aphid populations and virus transmission into pulse crops.

Rotate legume crops with cereals to reduce virus and vector sources. Where possible avoid close proximity to perennial pastures (eg lucerne) or other crops that host viruses and aphid vectors.

Eliminate summer weeds and self sown pulses that are a ‘green bridge’ as a host for viruses and a refuge for aphids.

<table>
<thead>
<tr>
<th>Disorder and cause</th>
<th>Seed-borne?</th>
<th>Symptoms</th>
<th>Distribution and occurrence</th>
<th>Survival and spread</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seed-borne root rot: Botrytis cinerea</td>
<td>Yes</td>
<td>Seedlings wilt and die, epicotyl rots</td>
<td>Random individual plants (not patches)</td>
<td>Seed</td>
<td>Quality seed; seed treatment</td>
</tr>
<tr>
<td>Seed-borne root rot: Ascochyta rabiei</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phytophthora root rot (PRR): Phytophthora medicaginis</td>
<td>No</td>
<td>Rapid wilting and yellowing; defoliation from lower leaves; rotted roots; plants hard to pull up</td>
<td>Patches; poorly drained areas; heavy rainfall; can occur at any time; history of medics, lucerne or PRR</td>
<td>Oospores in soil and residue persist for many years; survives saprophytically; spread by water and soil</td>
<td>Varietal selection; avoid paddocks with history of PRR; rotation; seed treatment</td>
</tr>
<tr>
<td>Waterlogging: root anoxia</td>
<td>No</td>
<td>Very rapid death; little defoliation; roots not rotted but may be dark; plants hard to pull up</td>
<td>Patches; poorly drained areas; heavy rainfall; can occur at any time; history of medics, lucerne or PRR</td>
<td>Caused by insufficient supply of oxygen to roots</td>
<td>Avoid low lying or poorly drained paddocks or areas within paddocks</td>
</tr>
<tr>
<td>Sclerotinia root and stem rot: Sclerotinia spp.</td>
<td>Yes (ad-mixed)</td>
<td>Wilting and death; bleached root, collar and stem tissue; white cottony mould at site of lesion; sclerotia at lesions or inside stems</td>
<td>Root and collar lesions result from direct infection from sclerotia; stem lesions result from airborne ascospores released from sclerotial apothecia, scattered or patches; favoured by denser canopies; wet events</td>
<td>Sclerotia persist in soil for many years; wide host range including pulses, canola, sunflowers and broadleaf weeds but not cereals or grasses</td>
<td>Avoid paddocks with history of sclerotinia of its hosts; rotate with cereals; some varieties more susceptible</td>
</tr>
<tr>
<td>Rhizoctonia rot: Rhizoctonia solani</td>
<td>?</td>
<td>Death of seedlings, stunting of survivors due to root damage, re-shooting after dampening-off of epicotyl</td>
<td>Can be a problem in irrigated crops grown immediately after cotton. Often occurs in 1–5 m stretches of row</td>
<td>Survives as sclerotia and on decomposing trash. Probably present in most soils</td>
<td>Allow time for decomposition of (preceding) crop debris. Tillage should help</td>
</tr>
<tr>
<td>Ascochyta blight: Ascochyta (Phoma) rabiei</td>
<td>Yes</td>
<td>Ghosting of tissues; lesions with concentric rings of pycnidia; stem stumps; plant death</td>
<td>Small patches enlarge rapidly in wet weather to kill large areas of crop</td>
<td>Chickpea residue very important in spread especially header dust and surface water flow; infected seed; volunteers</td>
<td>Follow chickpea Ascochyta blight management package published annually; includes foliar fungicides</td>
</tr>
<tr>
<td>Botrytis grey mould (BGM): Botrytis cinerea</td>
<td>Yes/no</td>
<td>Stem, flower pod and leaf lesions covered in grey mould</td>
<td>Occurs later in season when canopy closes and warm humid conditions persist; individual plants or patches</td>
<td>Can flow-on from seed-borne root rot but pathogen has wide host range and airborne spores can blow around; sclerotia can survive in soil</td>
<td>Avoid highly susceptible varieties; plant on wider rows; follow chickpea Ascochyta blight management package</td>
</tr>
<tr>
<td>Root-lesion nematodes: Pratylenchus spp.</td>
<td>No</td>
<td>General poor growth; small black lesions on lateral roots sometimes visible</td>
<td>Often affects large parts of crop; P. thornei more prevalent on high clay soils</td>
<td>Wide host range; survives and spreads in soil; anhydrobiosis allows nematodes to persist for prolonged dry periods</td>
<td>Farm hygiene; rotate with resistant species; grow tolerant varieties</td>
</tr>
</tbody>
</table>
### Table 2: Key facts about the biology of major chickpea diseases.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Survival</th>
<th>Spread</th>
<th>Infection by</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ascochyta blight</td>
<td>Stubble, seed, volunteers</td>
<td>Stubble, seed water-splashed spores</td>
<td>Water-splashed spores</td>
</tr>
<tr>
<td>Botrytis grey mould</td>
<td>Stubble, seed, sclerotia, alternative hosts</td>
<td>Stubble, seed, soil, airborne spores</td>
<td>Airborne spores</td>
</tr>
<tr>
<td>Phytophthora root rot</td>
<td>Oospores, alternative hosts</td>
<td>Soil and surface water</td>
<td>Waterborne spores</td>
</tr>
<tr>
<td>Sclerotinia rot</td>
<td>Sclerotia in soil and seed, alternative hosts</td>
<td>Soil and water, airborne spores</td>
<td>Airborne spores or directly into crowns</td>
</tr>
</tbody>
</table>

Source: Pulse Australia
Table 3: Disease rating for current chickpea varieties.

<table>
<thead>
<tr>
<th>Variety</th>
<th>Botrytis grey mould</th>
<th>Ascochyta blight* (Foliage/Stem)</th>
<th>Ascochyta blight (Pod)</th>
<th>P. Thornei (provisional)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desi</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Howzat\textsuperscript{b}</td>
<td>MS</td>
<td>S</td>
<td>S</td>
<td>MSp</td>
</tr>
<tr>
<td>PBA Slasher\textsuperscript{b}</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>MRMS</td>
</tr>
<tr>
<td>PBA Striker\textsuperscript{b}</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td></td>
</tr>
<tr>
<td>Ambar\textsuperscript{b}</td>
<td>S</td>
<td>MS</td>
<td>S</td>
<td></td>
</tr>
<tr>
<td>Neelam\textsuperscript{b}</td>
<td>S</td>
<td>MS</td>
<td>S</td>
<td></td>
</tr>
<tr>
<td>PBA Maiden\textsuperscript{b}</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td></td>
</tr>
<tr>
<td>Kabuli</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genesis\textsuperscript{™} 090</td>
<td>S</td>
<td>MS</td>
<td>S</td>
<td>S</td>
</tr>
<tr>
<td>Almaz\textsuperscript{b}</td>
<td>S</td>
<td>MS</td>
<td>S</td>
<td>VS</td>
</tr>
<tr>
<td>Genesis\textsuperscript{™} 079</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>MR</td>
</tr>
<tr>
<td>Genesis\textsuperscript{™} Kalkee</td>
<td>S</td>
<td>MS</td>
<td>S</td>
<td>MS</td>
</tr>
<tr>
<td>PBA Monarch\textsuperscript{b}</td>
<td>S</td>
<td>S</td>
<td>S</td>
<td>MS</td>
</tr>
</tbody>
</table>

Source: GRDC

Figure 1: Pulse disease diagnosis terms.
Source: Grain Legume Handbook
9.1 Key disease management strategies for chickpeas

- Variety selection is critical. Ideally grow an ascochyta-resistant variety.
- Paddock isolation from chickpea stubble is a high priority (greater than 500 m).
- Paddock history; aim for a break of at least four years between chickpea crops.
- Seed source; use seed from a paddock where disease was not detected.
- Fungicide seed dressing is effective and should be used, especially in high disease risk situations.
- Sowing date; do not sow too early, even with an ascochyta-resistant variety.
- Sowing depth; if using an ascochyta-susceptible variety, sow deeper than normal.
- Sowing rate; aim for 35–50 plants per square metre, depending on the situation and crop type (kabuli or desi).
- Hygiene; reduce disease sources and prevent spread of disease.
- Foliar fungicides; ascochyta-resistant varieties still require foliar fungicide at podding. Success is dependent on monitoring, timeliness of spraying and correct fungicide choice. Early detection and correct disease identification are essential.
- Manage aphids and virus; ground surface cover, healthy plants and crop canopy are important. Control aphids at their source (host) crop.
- Harvest management; harvest early to minimise disease infection of seed.
- Crop desiccation enables even earlier harvest. 3

9.2 Fungal disease management strategies

Disease management in pulses is critical, and relies on an integrated management approach involving variety choice, crop hygiene and strategic use of fungicides. The initial source of the disease can be from the seed, the soil, the pulse stubble and self-sown seedlings, or in some cases, other plant species. Once the disease is present, the source is then from within the crop itself.

Note that the impact of disease on grain quality in pulses can be far greater than yield loss. This must be accounted for in thresholds because the visual quality of pulses has a huge impact on price for food products. Examples are Ascocyahta blight in most pulses and Pea seed-borne mosaic virus in field peas.

A plant disease may be devastating at certain times, and yet under other conditions, it may have little impact. The interactions of host, pathogen and environment are all critical points in disease development, and all can be represented by the disease triangle (Figures 2 and 3). Diseases such as Ascocyahta blight and PRR can cause total crop failures very quickly. The effects of BGM and root-lesion nematodes on crop performance and yield usually unfold more slowly, however, they can cause damage quickly when conditions are suitable.

Disease management should be a consideration when planning any rotation, particularly at the beginning of the season. This is especially important for chickpeas where the first defence against diseases begins with paddock selection. Other criteria such as seed quality and treatment are also vitally important. Determine which diseases have the highest priorities to control in the pulse crop being grown, and sow a variety that is resistant to those diseases if possible. Paddock selection and strategic fungicide use are part of the overall program to minimise disease impact. Fungicide disease control strategies alone may not be economic in high-risk situations, particularly if susceptible varieties are grown.

**Key strategies:**

- **Variety selection.** Growing a resistant variety reduces the need for foliar fungicides.
- **Distance.** Distance from any of last year’s stubble of the pulse will affect the amount of infection for some diseases. Aim for a separation of at least 500 m.
- **Paddock history and rotation.** Aim for a break of at least four years between sowing of the same pulse crop. Having a high frequency of crops such as lentil, faba bean, vetch, field pea, chickpea, lathyrus or clover pasture puts pulses at greater risk of diseases such as Phoma blight, Sclerotinia rot and BGM. Ascochyta blight species are more specific to each pulse crop, but 3–4-year rotations are still important. Canola can also increase the risk of Sclerotinia rot.
- **Hygiene.** Take all necessary precautions to prevent the spread of disease. Reduce last year’s pulse stubble if erosion is not a risk and remove self-sown pulses before the new crop emerges.
• **Seed source.** Use seed from crops where there were low levels of disease, or preferably no disease, especially at podding. Avoid using seed with known disease infection, particularly with susceptible varieties. Have seed tested for disease status.

• **Fungicide seed dressings.** Dressings are partially effective early in situations of high disease risk, particularly for diseases such as BGM, Phoma blight and Ascochyta blight. They are also effective for seed-borne disease control but not effective on viruses and bacterial diseases.

• **Sowing date.** To minimise foliar disease risk do not sow too early, so avoiding excessive vegetative growth and early canopy closure. Early crop emergence also coincides with greater inoculum pressure from old crop residues nearby. Aim for the optimum sowing window for the pulse and the district.

• **Sowing rate.** Aim for the optimum plant population (depending on region, sowing time, crop type, variety), as denser canopies can lead to greater disease incidence. Adjust seeding rate according to seed size and germination. Avoid double sowing headlands, as the denser crop can be more prone to disease establishment and lodging. Seeding rates below the minimum recommended plant populations will have minimal impact on disease incidence, but reduce potential yield and increase harvest losses.

• **Sowing depth.** Sow deeper than normal any seed lot that is infected with disease to help reduce emergence of infected seedlings. The seeding rate must be adjusted upwards to account for the lower emergence and establishment percentage.

• **Foliar fungicide applications.** Disease-resistant varieties do not require the same regular foliar fungicide program that susceptible varieties need to control foliar diseases. Some pulses may require fungicide treatment for BGM if a dense canopy exists. Successful disease control with fungicides depends on timeliness of spraying, the weather conditions that follow, and the susceptibility of the variety grown. Monitoring for early detection and correct disease identification is essential. Correct fungicide choice is also critical.

• **Controlling aphids.** This may reduce the spread of viruses, but not eliminate them. Strategic or regular insecticide treatments are unlikely to be successful or economic. A virus can be spread by aphids before the aphids are detected.

• **Harvest management.** Early harvest will help to reduce disease infection of seed, and is also important for grain quality and to minimise harvest losses. Crop desiccation enables even earlier harvest. Moisture contents of up to 14% are allowable at delivery. Do not prematurely desiccate as this can affect grain quality. 4

### 9.3 Integrated Disease Management

Disease management in chickpeas is critical and relies heavily on an integrated management package involving paddock selection, variety choice, strategic fungicide use and crop hygiene.

The appropriate Ascochyta blight control strategy is then adopted by determining the level of risk in combination with climatic conditions and the level of resistance afforded by the variety chosen.

Disease control strategies may not be economic in high-risk situations if varieties susceptible to Ascochyta blight are grown. 5

IDM (Integrated Disease Management) is an integrated approach of crop management to reduce chemical inputs and resolve ecological problems. Although originally developed for insect pest management, IPM programs now encompass diseases, weeds, and other pests.


IDM is performed in three stages: prevention, observation and intervention. It is aimed at significantly reducing or eliminating use of pesticides while managing pest populations at an acceptable level.

An IDM system is designed around six basic components:

1. Acceptable disease levels:
   • Emphasis is on economical control, not eradication.
   • Elimination of the disease is often impossible, and can be economically expensive, environmentally unsafe, and frequently not achievable. IDM programs work to establish acceptable disease levels (action thresholds) and then apply controls if those thresholds are about to be exceeded. Thresholds are specific for disease and site. What is acceptable at one site may not be acceptable at another site or for another crop. Allowing some disease to be present at a reasonable threshold means that selection pressure for resistance pathogens is reduced.

2. Preventive cultural practices:
   • Use varieties best suited to local growing conditions and with adequate disease resistance.
   • Maintaining healthy crops is the first line of defence, together with plant hygiene and crop sanitation. Crop canopy management is also very important in pulses; hence, time of sowing, row spacing and plant density and variety attributes become important.

3. Monitoring:
   • Regular observation is the key to IDM.
   • Observation is broken into inspection and then identification. Visual inspection, spore traps, and other measuring tools are used to monitor disease levels. Accurate disease identification is critical to a successful program. Record keeping is essential, as is a thorough knowledge of the behaviour and reproductive cycles of target pests.
   • Diseases are dependent on specific temperature and moisture regimes to develop (e.g. rust requires warm temperatures, Ascochyta blight often requires colder temperatures). Monitor the climatic conditions and rain likelihood to determine when a specific disease outbreak is likely.

4. Mechanical controls:
   • Should a disease reach unacceptable levels, mechanical methods may be needed for crop hygiene (e.g. burning or ploughing in pulse stubble, removing hay, cultivating self-sown seedlings).

5. Biological controls:
   • Crop rotation and paddock selection is a form of biological control.
   • Using crops and varieties with resistance to the specific disease is also important. Other biological products are not necessarily available for disease control.

6. Responsible fungicide use:
   • Synthetic pesticides are generally used only as required and often only at specific times in a disease lifecycle.
   • Fungicides applied as protection ahead of conditions that are conducive to disease (e.g. sustained rainfall) may reduce total fungicide usage. Timing is critical with foliar fungicides, and may be more important than rate used. Protection is better than cure, because once the disease is established in the canopy, there is an internal source of infection that is difficult, or even impossible, to control with later fungicide applications. 6

9.4 Risk assessment

Prediction of likely damage from a chickpea disease can be used at the paddock, whole farm, regional, state or national level. The choices of variety and disease management options are some of the factors determining risk.

Knowledge of your paddock, its layout (topography), soil parameters, and cropping history will help you to assess the level of risk.

9.4.1 Steps in risk assessment

1. Identify factors that determine risk:
   - **Pathogen.** Exotic v. endemic; biotypes, pathogenicity, survival and transmission, amenable to chemical management.
   - **Host.** Host range; varietal reactions, vulnerability. Does susceptibility change with growth stage?
   - **Environment.** Weather dependency, interactions with nutrition, herbicides, other diseases, agronomic factors, e.g. planting depth, row spacing, no-tillage, soil conditions.
   - **Risk management.** Access to components of management plan; ease of implementing plan; how many options; cost of implementation.

2. Assess level of factors:
   - **Pathogen.** Level of inoculum, dirty seed, aggressiveness of isolate, weed hosts prevalent in paddock or nearby, paddock history.
   - **Host.** How susceptible, nutritional status, frost susceptibility, herbicide susceptibility.
   - **Environment.** Length of season; likelihood of rain, drought, waterlogging, irrigation; availability of spray gear; paddock characteristics; herbicide history.
   - **Risk management.** Not yet considered; plan being developed; plan in place?

3. What risk level is acceptable?
   - **High.** Grower is prepared to accept substantial yield loss because potential returns are high and financial situation sound; crop failure will not affect rotation or other components of farming system.
   - **Low.** Grower needs cash flow and cannot afford to spend much or lose the crop; failure seriously affects farming system.

9.4.2 Paddock selection

The selection of the most appropriate paddock for growing chickpeas involves consideration of several important factors, some of which are related to the modes of survival and transmission of pathogens such as *Ascochyta rabiei*.

1. Rotation:
   - Develop a rotation of no more than one year of chickpea in four years.
   - Plant chickpea into standing stubble of previous cereal to enhance crop height and reduce attractiveness of the crop to aphids (aphids may vector viruses).
   - Consideration also needs to be given to previous crops that may host pathogens such as *Sclerotinia*, *Rhizoctonia*.
   - *Ascochyta rabiei* is chickpea-specific, whereas *Botrytis cinerea* has a wide host range including sunflower, bean, pea, lentils and weeds (e.g. *Euphorbia* spp., groundsel and emufoot).

2. History of chickpea diseases:
   - Previous occurrence of soil-borne diseases (*Sclerotinia* stem rot or *Pratylenchus* nematodes) constitutes a risk for subsequent chickpea crops for up to ten years.
   - At least 500 m from the previous year’s chickpea crop.

3. Weeds:
   - Nearly all weeds host *Sclerotinia* spp.
• Some of the viruses affecting chickpea also have wide host ranges. Weeds, particularly perennial legumes, host viruses and their aphid and leafhopper vectors (e.g. Cucumber mosaic virus).

4. Herbicide history:
• Have triazine, sulfonylurea or other residual herbicides been applied in the last 12 months?
• The development of some diseases is favoured in herbicide-weakened plants. The presence of these herbicide residues in soil may cause crop damage and thus confusion over in-field disease diagnosis.

9.4.3 Regular crop monitoring
The two main diseases for which monitoring is necessary are Ascochyta blight and BGM. Following the monitoring process recommended for these diseases will provide the opportunity to assess the impact or presence of other diseases or plant disorders. To be effective, crop monitoring needs to include a range of locations in the paddock, preferably following a ‘V’ or ‘W’ pattern.

For Ascochyta blight
The initial symptoms will be wilting of individual or small groups of seedlings, or lesions on the leaves and stems of young plants, often in patches. Monitoring should commence 2–3 weeks after emergence, or 10–14 days after a rain event. This is because the initially infected seedlings soon die and symptoms are difficult to separate from other causes. Plant parts above the lesion may also break off, making symptoms difficult to detect. Timing is critical! After the initial inspection, subsequent inspections should occur every 10–14 days after a rain or heavy dew event. During dry periods, inspections should occur every two weeks. When monitoring, look for signs of wilting in upper foliage (the ‘ghosting’ phenomenon) or small areas of dead or dying plants, and if present, examine individual affected plants for symptoms of infection. This method will allow more of the crop to be inspected than a plant-by-plant check.

For Botrytis grey mould
Botrytis grey mould is more likely to occur in well-grown crops where there is canopy closure. The critical stage for the first inspection will be at the commencement of flowering and then regularly through the flowering period. Lesions occur on stems, leaves and pods, and flower abortion and drop can occur; a fluffy grey fungal ‘bunch of grapes’ growth develops on affected tissue. Normal pod-set will occur when daily temperature exceeds 15°C; BGM ceases to affect the plant once the maximum daily temperature exceeds ~28°C.

More regular crop monitoring may also be required if:
• high-risk situations exist such as non-optimal paddock selection
• shortened rotation
• immediately adjacent to last year’s crop
• high disease pressure experienced last year
• a more susceptible variety is planted.

For more information, see Chickpea disease management – Southern region.

9.4.4 Crop disease forecast
Crop disease forecast is a weekly, location- and season-specific estimate of risk for certain crop diseases during the cropping season and, for some diseases, offers management practices to avoid potential yield loss.

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Who this tool is for?
Grain growers in the southern and western grain-producing regions of Australia.

Questions this tool answers:
• What is the risk of crop loss due to this disease?
• What steps can I take to manage this disease?

Diseases and crops this tool covers:
Viral diseases:
• Barley yellow dwarf virus
• canola Beet western yellows virus
• lupin Bean yellow mosaic virus
• lupin Cucumber mosaic virus

Crops included in the risk forecasts:
• cereals (wheat, barley, oats and triticale)
• pulses (field pea, chickpea, faba bean, lentil)
• canola
• lupins

What this tool does
Crop disease forecast estimates the risk of certain crop diseases during the cropping season for specific locations. For some diseases, it offers management practices to avoid potential yield losses.

Each weekly forecast, where relevant, accounts for varietal resistance, chemical options, agronomic yield potentials and losses, agronomic constraints (frost and terminal drought), risks of spore showers, disease severity and disease-related yield losses.

Inputs
No inputs from growers are required.

Outputs
A disease forecast report may include, for each location:
• forecast risk in tables or maps
• estimated severity or spore maturity
• sowing guide
• rainfall to date and stubble moisture
• suggested management practices

9.5 Ascochyta blight

Key points:
• Chickpea crops in southern Australia are being hit by a more virulent strain of the damaging ascochyta blight.
• Pulse pathologists in Victoria and South Australia have noted a marked decline in the resistance of several varieties of chickpeas, with varieties previously rated as moderately resistant performing like susceptible lines.
• It is imperative all chickpea seed should be treated with a thiram based fungicide to prevent seed transmission of AB on to the emerging seedlings in 2017.

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Ascochyta blight, caused by the fungus *Phoma rabiei* (formerly *Ascochyta rabiei*), is a serious disease of chickpeas in Australia. The fungus is different from the species of ascochyta that infects faba beans, lentils and field peas. The fungus can infect all above-ground parts of the plant and is most prevalent when cool, cloudy and humid weather occurs during the crop season. 

Chickpeas were an important part of southern farming systems crop rotations until the late 1990s. They provided both economic and sustainability benefits to growers throughout southern Australia until widespread outbreak of Ascochyta blight in 1998. All cultivars grown at this time were susceptible to the disease and as a result of the epidemic, the area sown reduced from 150,000 hectares to less than 10,000 hectares within two years. 

Ascochyta blight is now considered to be endemic in most growing regions. Unlike some insect control strategies, there is no economic threshold for ascochyta. Management strategies are aimed at preventing the occurrence of disease and limiting its spread.

Management and control of Ascochyta blight is the important factor in determining the viability of chickpea production. The first variety with improved Ascochyta blight resistance, Genesis™ 508, became available in 2005, Genesis™ 090 followed it in 2006, and others like Genesis™ 509 follow in 2008. The large kabuli varieties Almaz and Nafice became available in 2006. They along with all older varieties will require strategic fungicide management through the application of well-timed protective foliar fungicides. Disease levels will vary according to season, rotation history of the paddock and its surrounds, stubble management, seed hygiene, sowing time and timing of fungicide applications.

Ascochyta blight is managed through crop rotation, hygiene, seed treatment, prophylactic fungicide application and growing varieties with improved resistance. All growers and advisers need to regularly inspect their crops from emergence, through flowering, right up to plant maturity. Inspections should be undertaken 10–14 days after rain events, when new infections will be evident as lesions on plant parts.

**Changes in Ascochyta blight—Southern region**

During 2015 at Curyo (southern Mallee), despite the relatively dry season, in early August a significant outbreak of Ascochyta blight was observed in a kabuli chickpea trial (Figure 4). Symptom assessment indicated that this isolate of ascochyta was different from those observed previously in Victorian trials, having virulence on resistant lines such as Genesis™ 090 and PBA Slasher. From the results in this trial, there appears to be some differences in resistance to this isolate with CICA1454 showing fewer symptoms and PBA Striker being significantly affected (Figure 4). Unfortunately, due to the dry finish to the season, and low yield potential it was impossible to assess the impact of the disease on grain yield. Despite being affected by the disease Genesis™ 090 was the highest yielding (0.5 t/ha) line at Curyo (yield data not shown).

The isolate from this trial was provided to pathologists and has been compared with other new isolates from SA. Results show that Genesis™ 090 has a susceptible

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9 Jenny Davidson (2016). Personal communication.
reaction to the Victorian isolate but not to the other two isolates. Ambar®, Neelam® and Genesis™ 079 demonstrated resistance to all three isolates; Almaz® had a moderate reaction to the new SA isolate but a good level of resistance to the other two isolates; PBA Maiden® had a moderate reaction to all three isolates; Kalkee, PBA Monarch®, and PBA Slasher® have a moderate to susceptible reaction to all three isolates while PBA Striker® had a susceptible reaction to all three isolates (Figure 4). These results still need to be confirmed with field data (Table 4). 14

Figure 4: Ascochyta blight score in the medium sized kabuli chickpea variety trial at Curyo 2015, recorded August 13 and September 9, 2015.
Source: Brand et al via GRDC

Economic importance
The widespread occurrence of this disease in 1998 had a negative impact on the chickpea growing industry. To successfully grow varieties with an ascochyta disease rating less than moderately resistant, foliar fungicides need to be applied throughout the growing season to avoid serious yield losses. Varieties rated as moderately resistant (such as PBA Slasher® and Genesis™ 509) still require at least one fungicide at early pod-set, but the risk of yield loss is minimal. When selecting varieties, the added cost of fungicide applications needs to be considered before selecting and growing susceptible to moderately resistant varieties. 15

9.5.1 Varietal resistance or tolerance
With the spread of the new strain of ascochyta blight, the resistance ratings of all chickpea varieties have been updated (Table 4).
Table 4: Varietal resistance of chickpeas to the new strain of ascochyta blight.

<table>
<thead>
<tr>
<th>Chickpea variety</th>
<th>Ascochyta blight resistance rating rating (old rating in brackets)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desi</td>
<td></td>
</tr>
<tr>
<td>Ambar</td>
<td>MS (R)</td>
</tr>
<tr>
<td>Genisis 509</td>
<td>MS (R)</td>
</tr>
<tr>
<td>Howzat</td>
<td>S (S)</td>
</tr>
<tr>
<td>Neelam</td>
<td>MS (R)</td>
</tr>
<tr>
<td>PBA HatTrick</td>
<td>S (MR/R)</td>
</tr>
<tr>
<td>PBA Maiden</td>
<td>S (MR)</td>
</tr>
<tr>
<td>PBA Slasher</td>
<td>MS (R)</td>
</tr>
<tr>
<td>PBA Striker</td>
<td>S (MR)</td>
</tr>
<tr>
<td>SONALI</td>
<td>S (MR)</td>
</tr>
<tr>
<td>Kabuli</td>
<td></td>
</tr>
<tr>
<td>Almaz</td>
<td>MS (MS)</td>
</tr>
<tr>
<td>Genisis 079</td>
<td>S (R)</td>
</tr>
<tr>
<td>Genisis 090</td>
<td>MS (R)</td>
</tr>
<tr>
<td>Genisis 114</td>
<td>S (S)</td>
</tr>
<tr>
<td>Kalkee</td>
<td>MS (MS)</td>
</tr>
<tr>
<td>PBA Monarch</td>
<td>S (MS)</td>
</tr>
</tbody>
</table>


Before the outbreak of the new strain of ascochyta blight, there were resistant varieties available. Varieties differed in their resistance and/or tolerance to Ascochyta blight (Table 5 and Figure 5).

Table 5 estimates gross margins for chickpeas with ascochyta-susceptible versus ascochyta-resistant varieties. Fungicide costs are based on eight applications at $20/ha per application for the susceptible variety versus one for the resistant variety. Assuming variety yields are the same, desi gross margin of $130 versus $270/ha may be achieved from a 1.5 t/ha grain yield. A $180 versus $420/ha return could be obtained from a kabuli yield of 1.0 t/ha. If choosing a variety susceptible to Ascochyta blight, growers should consider kabuli production in preference to desi where conditions are suitable. 16

Figure 5: Varietal response to Ascochyta blight in 2003 Horsham trials. Trials had no treatment with fungicide.

Source: Hobson et al via University of Sydney

### Table 5: Estimated desi and kabuli returns. NOTE: these calculations are NOT based on varietal tolerance to the new Ascochyta blight strain.

<table>
<thead>
<tr>
<th>Grain yield (t/ha)</th>
<th>Grain price ($/t)</th>
<th>Fungicide cost Susceptible variety ($/ha)</th>
<th>Fungicide cost Resistant variety ($/ha)</th>
<th>Other costs All varieties ($/ha)</th>
<th>Gross margin Susceptible variety ($/ha)</th>
<th>Gross margin Resistant variety ($/ha)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Desi</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.5</td>
<td>300</td>
<td>160</td>
<td>20</td>
<td>160</td>
<td>-170</td>
<td>-30</td>
</tr>
<tr>
<td>1.0</td>
<td>300</td>
<td>160</td>
<td>20</td>
<td>160</td>
<td>-20</td>
<td>120</td>
</tr>
<tr>
<td>1.5</td>
<td>300</td>
<td>160</td>
<td>20</td>
<td>160</td>
<td>130</td>
<td>270</td>
</tr>
<tr>
<td>2.0</td>
<td>300</td>
<td>160</td>
<td>20</td>
<td>160</td>
<td>280</td>
<td>420</td>
</tr>
<tr>
<td><strong>Kabuli</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.5</td>
<td>500</td>
<td>160</td>
<td>20</td>
<td>160</td>
<td>-70</td>
<td>70</td>
</tr>
<tr>
<td>1.0</td>
<td>500</td>
<td>160</td>
<td>20</td>
<td>160</td>
<td>180</td>
<td>420</td>
</tr>
<tr>
<td>1.5</td>
<td>500</td>
<td>160</td>
<td>20</td>
<td>160</td>
<td>430</td>
<td>570</td>
</tr>
<tr>
<td>2.0</td>
<td>500</td>
<td>160</td>
<td>20</td>
<td>160</td>
<td>680</td>
<td>820</td>
</tr>
</tbody>
</table>

Source: Pulse Australia

The national chickpea program continues its commitment to re-establishing the chickpea industry in south-east Australia. A number of international lines with excellent resistance to Ascochyta blight have been released and were the first resistant varieties available. All releases require less fungicide support than current options, but still require one spray at podding to produce good quality seed. Resistant varieties offer growers greater yield security. However, fungicides are still likely to be required to minimise yield losses through pod and seed infection. Seed infected by Ascochyta blight can be small and blemished. It may also attract lower returns through downgrading. Sowing diseased seed sets back crop prospects from the outset. 17

9.5.2 Damage caused by disease

The high-risk and increased cost of controlling Ascochyta blight in a susceptible variety often make desi chickpea production unprofitable but higher value kabuli types remain profitable. 18 Unlike some insect control strategies, there is no economic threshold for Ascochyta. Management strategies are aimed at preventing the occurrence of disease and limiting its spread. 19

9.5.3 Symptoms

*Phoma rabiei* infects the leaves, stems and pods of chickpea plants, causing tan/brown, rounded lesions on affected plant parts. This disease is usually first noticed in late winter when small patches of blighted plants appear throughout the paddock (Photo 1). Usually the first symptoms are the withering of individual or small groups of seedlings. Plants appear as if premature haying off has occurred. Initially Ascochyta blight appears on the younger leaves as small water-soaked pale spots. These spots rapidly enlarge under cool and wet conditions, joining with other spots on the leaves and blighting the leaves and buds. 20

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Photo 1: Wilting of individual or small groups of seedling.
Source: Pulse Australia

Ascochyta leaf ghosting symptoms may appear 4–7 days after rainfall or heavy dew (Photo 2).

Photo 2: Ghosting symptoms of chickpea.
Source: Pulse Australia

Lesions usually begin as a pale green-yellow discolouration on leaves and stems and progress into small round lesions with dark-brown margins and pale grey to tan sunken centres (Photo 3). Note the concentric circles of brown-black dots in the centre of the lesions. These small black spots (pycnidia), or fruiting bodies are unique to Ascochyta blight. Pycnidia, less than 1 mm in diameter, can be seen in the affected areas. Pycnidia are also present in stem lesions. In severe cases of infection the entire plant dries up suddenly.

Lesions on stems at first tend to be oval shaped, with brown centres and a darker margin. Elongated lesions can often form and girdle the stem (Photo 4). The stem may die and break off. Regrowth may occur from the broken stem. Affected areas on the pods tend to be round, sunken, with pale centres and dark margins.

- Leaf lesions: Lesions usually begin as a pale green-yellow discoloration on leaves and stems and progress into small round lesions with dark-brown margins and pale grey to tan sunken centres. Towards the centre of the lesion, fruiting bodies called pycnidia develop (appearing as black specks), often in concentric rings. These pycnidia produce spores, which spread on wind-borne stubble and/ or water (rain splash) to infect other plants. Note the concentric circles of brown-black dots in the centre of the lesions. These are the pycnidia or fruiting bodies that are unique to Ascochyta blight. Ascochyta leaf ghosting may appear 4–7 days after infection following rainfall or heavy dew.
• Stem lesions: Lesions on stems at first tend to be oval shaped, with brown centres and a darker margin. Lesions often girdle the stems of the plant, causing them to weaken and subsequently break off.

• Pod lesions: Pod lesions are similar in appearance to leaf lesions. They lead to infection of the seed. DO NOT keep planting seed from any crop that has been identified as having Ascochyta blight (Photo 5).

![]([image-url])

**Photo 5:** Pod lesions look similar to leaf lesions and lead to infection of the seed (left). Infected pod with a large lesion containing fungal fruiting bodies (right).

Source: Pulse Australia and CropPro

The fungus can penetrate the pod and infect the seed. Pod lesions are similar in appearance to leaf lesions. Severe pod infection usually results in reduced seed set and infected seed. When infected seeds are sown, the emerging seedlings will develop dark-brown lesions at the base of the stem. Affected seedlings may collapse and die.

### 9.5.4 Conditions favouring development

Initial crop infection is due to the introduction of either infected planting seed or from movement of infected trash by wind, machinery or animals. Spores of the fungus can survive for a short time on skin, clothing and machinery. Subsequent in-crop infection and spread occurs when inoculum is moved higher in the canopy or to surrounding plants by wind or rain splash during wet weather. The disease spreads during cool, wet weather from infected plants to surrounding plants by rain splash of spores. This creates large blighted patches within crops. Pycnidia produce spores, which infect other plants through wind-borne stubble and/or water (rain splash). There are no other known hosts of *Phoma rabiei* in Australia.

Ascochyta blight-infected stubble blown about during and after harvest is a major cause of short–medium-distance dispersal (metres to kilometres) along with movement of infected trash by water, machinery or animals. Spores of the fungus can survive a short time on skin, clothing and machinery.

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Ascochyta blight can increase rapidly on volunteer chickpeas if wet weather occurs during spring–summer–autumn. Paddocks with chickpea stubble should be regarded as a source of inoculum even if Ascochyta blight was not observed in last season’s chickpea crop. The pathogen can survive at least three years in the paddock.

Ascochyta blight can develop over a wide range of temperatures (5–30°C) and needs only 3 hours of leaf wetness to infect (Figure 6). However, the disease develops fastest when temperatures are 15–25°C and relative humidity is high (the longer relative humidity remains high, the more severe will be the infection).

Subsequent in-crop infection occurs when spores are moved higher in the canopy or to surrounding plants by rain splash during wet weather. Multiple cycles of infection will occur during the growing season whenever environmental conditions are favourable.

**Figure 6:** Life cycle of Ascochyta blight pathogen. Note: Only the asexual phase is known to occur in Australia at this time.


### 9.5.5 Management of disease

**Monitoring:**
- When inspecting crops, look for signs of wilting in upper foliage and small areas of dead or dying plants.
- Check in a range of locations across the field following a ‘V’ or ‘W’ pattern.
- Spend at least 1 to 2 hours inspecting each crop for Ascochyta blight.
- Ensure good hygiene when moving between crops and farms.

Take extra care when inspecting crops that are growing:
- under centre pivot or lateral-move irrigators
- from seed whose ascochyta status is unknown
- from seed that was not treated with a registered fungicide seed dressing.  

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If ascochyta is suspected

If ascochyta is suspected mark the spot and take samples for diagnosis. DO NOT enter other chickpea paddocks wearing the same clothing. All other chickpea crops on the property need to be inspected for Ascochyta blight. Be sure to follow the hygiene practices outlined below:

- Place samples of suspected ascochyta-infected plants into a plastic bag then seal the bag and keep the samples cool.
- Suspect samples should be referred to a plant pathologist or agronomist familiar with the disease for identification.
- Unnecessary movement within a suspected ascochyta-infected crop should be avoided until the sample has been fully assessed.
- Most importantly, do not visit other chickpea crops until all clothing has been disinfected or changed and machinery has been washed of all plant material and dirt.

Hygiene

The spores of ascochyta can adhere to clothing, machinery, vehicles, people and animals when moving through infected paddocks, so hygiene is a vital component of IDM when ascochyta is found in a crop. Wear waterproof pants, overboots or rubber gum boots when entering a suspected infected paddock, then decontaminate immediately after exiting.

- Farmers and advisers should take precautions to prevent spreading Ascochyta blight via clothing, footwear and vehicles.
- The recommended protocol is for clothing to be washed, changed or disinfected when moving between chickpea paddocks.
- Wash boots in a mixture of 10% bleach and 90% water solution or methylated spirits upon leaving an infected chickpea crop.
- Clothing must be machine-washed in hot water before being worn when entering another chickpea crop.
- Extra care should be taken to remove soil and plant material from boots and vehicles.
- Hands and arms should be washed in warm soapy water or a suitable disinfectant.
- The use of heavy-duty plastic bags to cover boots and legs is a common practice when checking crops. After inspecting the crop, remove these plastic covers and place them in another bag and seal. Use another set of covers if you need to enter another chickpea crop.
- Farmcleanse® can be used to clean equipment.

During harvest

Harvest ascochyta-free paddocks before infected paddocks and preferably use your own harvester. Do not run the straw spreaders when harvesting, which will reduce the spread of small pieces of ascochyta-infected stem and pods.

Thoroughly clean and decontaminate all machinery associated with harvesting in a well-defined and identifiable area before moving to another paddock or property.

Post-harvest

All grain harvested from an ascochyta-infected paddock should be transported off farm to receival sites in well-sealed trucks. If kept for a period on-farm it should be stored in well-sealed and labelled silos which must be thoroughly cleaned after the grain has been removed. Grain harvested from an ascochyta-infected crop must not be retained as planting seed for other crops. Consideration may be given to

incorporation of infected crop residues by the use of off-set discs immediately after harvest to enhance the rate of breakdown. Chickpea volunteers in the infected paddock, along fence lines and near sheds must be controlled. Chickpeas should not be grown in or adjacent to an ascochyta-infected paddock for at least three years.  

Control

Follow the principles of IDM, which include:

- crop rotation and paddock selection
- clean seed and fungicide seed dressings
- regular crop monitoring
- strict hygiene on and off farm
- strategic use of foliar fungicides.

Note: Chickpea seed dressings only protect the emerging seedling from seed-borne ascochyta and seed-borne botrytis. Seed dressings will not protect the emerged seedling from rain-drop splashed ascochyta or wind-borne botrytis. See Section 3 Planting, 3.2 Seed treatments for more information.

Sowing date:

- With Ascochyta blight resistant varieties, sow at traditional sowing dates. Delayed sowing is not necessary.
- With Ascochyta blight susceptible varieties, delayed sowing has been a most important strategy for Ascochyta blight management. It reduces the duration of exposure of chickpea seedlings to Ascochyta blight spores. It will not help though if self-sown chickpeas are nearby. Be aware that delayed sowings can result in lower yields due to increased risks of dry finishes and high temperatures during podding.
- In all varieties, sowing too early can produce poor early pod-set if flowering is in a colder period.

Harvest timing

Harvest as early as possible to minimise Ascochyta blight infection on seed and potential seed downgrading. The damage from the disease is usually more severe when crops are harvested late. Harvest losses, seed splitting and downgrading quality can be substantial if chickpea is harvest is below 12%.

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Management options for minimising the damage by Ascochyta blight (Ascochyta rabiei) in chickpea (Cicer arietinum L.)

Ascochyta blight, a fungal disease caused by Ascochyta rabiei, is the major constraint for chickpea production worldwide. Current cultivars only possess partial resistance to the pathogen, and this level of resistance can breakdown easily because the pathogen is highly variable due to potential for sexual recombination. The development of IDM is the key for successful chickpea production. The use of Ascochyta blight-free seed and seed dressing with effective fungicides reduces the probability of transmitting seed-borne disease to the seedlings. Deep-burying or burning of chickpea stubble minimises stubble-borne inoculum. One to two years of non-host crops for warm and wet areas and 3–4-year crop rotation for cold and dry areas are required to reduce the levels of stubble-borne inoculum. The use of field isolation and sowing chickpea at a distance from previous chickpea crops will reduce the density of airborne ascospores released from infected debris. Optimum sowing date, deep sowing, optimizing plant density, balanced nutrition, and alternative sowing patterns should be considered as a means of reducing Ascochyta blight pressure wherever possible. Sprays at seedling stage or before the occurrence of infection are crucial in short-season areas or where ascospores are the major sources of inoculum. Chickpea growers are strongly encouraged to adopt an integrated approach that combines all agronomic options, including cultivar selection, if they are to manage this disease economically and effectively. 32

9.5.6 Ascochyta blight management in kabuli

Paddock selection

Keep at least a three-year break between chickpea crops in the same paddock. Equally importantly, sow new chickpea crops at least 500 m from any paddock (yours or your neighbours) in which chickpea was grown in the previous season. Ascochyta spores from infected chickpea stubble from the previous season are released in mid-winter and can be blown hundreds of metres, or even kilometres. Small pieces of infected chickpea trash (leaf, pod and stem) may be blown considerable distances during harvest and may also be moved about by winds throughout the summer and autumn. It is important to consider the risks from wind-blown trash prior to the break of the season and wind-borne spores after crop emergence when selecting paddocks to sow to chickpea. 33

Seed

Test your seed for germination and Ascochyta blight infection. Do not sow seed if the ascochyta infection level is above 0.25%. All kabuli seed should be treated with a fungicide seed dressing; this will reduce the transmission of seed-borne fungal infections and also help to protect the emerging seedling from soil-borne pathogens and seedling rots. Seed testing and seed dressing are complementary: seed testing ensures that seed with an unacceptably high level of infection is not being sown.

while seed dressing reduces, but does not eliminate, seed-borne infection. Seed dressing highly infected seed reduces the level of transmission, but may still result in high levels of initial infection of the emerged crop. \footnote{K Regan (2016) Production packages for kabuli chickpea in Western Australia—post planting guide. DAFWA, https://www.agric.wa.gov.au/chickpeas/production-packages-kabuli-chickpea-western-australia-post-planting-guide}

**Fungicide timing**

Where crops of Almaz\textsuperscript{a} and KALKEE\textsuperscript{b} have been established following the above recommendations, growers should budget for two or three strategic fungicide sprays (chlorothalonil 720 g/L applied at 1.0–2.0 L/ha). This is a significant improvement over the regular spray schedule (every three to four weeks) previously recommended.

The fungicide spray is required four weeks after emergence (chlorothalonil 720 g/L applied at 1.5 L/ha). This early prophyllactic spray is required to contain the spread from any Ascochyta blight infections resulting from wind-blown spores from last year’s stubble, seed-borne infections or infected trash that has been carried into the paddock. The level of infection that requires application of a fungicide spray this early in the crop’s life is very low and is below the level that can be reliably identified, even by a person who has considerable experience in identifying this disease in field crops. Additionally, application of an early spray will protect the crop against wind-borne spores released from chickpea stubble during the two to three weeks following the spray application.

A second spray (chlorothalonil 720 g/L applied at 1.0–2.0 L/ha) is recommended at full flowering to protect the developing pods and minimise the risk of reduced quality. The rate of fungicide application depends on the level of Ascochyta blight infection detected in the crop prior to spraying. The high rate (2.0 L/ha) would be appropriate where Ascochyta blight can be easily identified in the crop and the low rate (1.0 L/ha) where only minor disease infection is evident after close inspection. If Ascochyta blight is not identified, even after close inspection of more than ten locations throughout the crop, a fungicide application may not be required at this time. A fungicide spray (chlorothalonil 720 g/L applied at 1.5–2.0 L/ha) may be required during pod-filling if Ascochyta blight becomes evident in the canopy during late flowering or podding. \footnote{K Regan (2016) Production packages for kabuli chickpea in Western Australia—post planting guide. DAFWA, https://www.agric.wa.gov.au/chickpeas/production-packages-kabuli-chickpea-western-australia-post-planting-guide}

### 9.5.7 Foliar fungicide programs

Ascochyta blight has constrained chickpea production in Australia. Therefore, control strategies are required to prevent major crop losses. Field experiments in 1998 and 1999 showed that all the chickpea varieties grown commercially in Australia at that time were very susceptible to the disease. Fortnightly sprays with the fungicide chlorothalonil could effectively control epidemics but the additional cost significantly reduced profitability. The kabuli variety Kaniva was still profitable to grow but desi varieties were less profitable than alternative crops.

Further experiments were conducted throughout Australia in 1999, 2000 and 2001 to compare a range of fungicides and to determine the optimum rates and frequency of fungicide sprays. Chlorothalonil was superior to mancozeb and carbendazim. Fortnightly sprays of chlorothalonil controlled Ascochyta blight in all varieties; sprays every three weeks did not eliminate yield losses due to Ascochyta blight in susceptible varieties under high disease pressure. Low fungicide rates were less effective than maximum recommended rates when conditions favoured a severe epidemic.

Field experiments were established in 2002 and 2005 to compare new varieties with the older, susceptible varieties. The new varieties had significantly less disease than the older varieties and did not require fortnightly sprays. The best new varieties required fungicide sprays only at the podding stage in order to prevent pod and seed infection.

As more varieties with greater resistance become available, growers will need to apply fewer fungicides and the consequences of missing a fungicide spray will be less serious. However, variety specific management strategies still need to be developed to enable growers to tailor their control strategy to each variety susceptibility in order to minimise fungicide usage and maximise profits. 36

**IN FOCUS**

**Economic chickpea production for southern Australia through improved cultivars and strategic management to control Ascochyta blight.**

Cultivars with improved resistance to Ascochyta blight became available in 2005, but the successful economic management of the disease needed to be demonstrated to growers. Experiments were sown at four locations over two seasons in southern Australia to assess fungicide (chlorothalonil or mancozeb) application timing and efficacy in controlling Ascochyta blight in cultivars varying in Ascochyta blight resistance. Resistant (R) cultivars were successfully grown with two or less fungicide applications during podding. Moderately resistant (MR), moderately susceptible (MS) and susceptible (S) cultivars always required at least three and up to nine fungicide applications to prevent yield loss. In all experiments the podding treatment of chlorothalonil had equivalent or greater grain yields than the mancozeb podding treatment. The use of resistant cultivars with one or two strategic foliar fungicide applications ensures chickpeas are a low risk, profitable option in medium rainfall (350–450 mm) cropping areas of southern Australia.

**Experimental design**

Field experiments were sown in 2004 and 2005 across south-eastern Australia to compare the effect of different fungicide regimes on Ascochyta blight foliar symptoms and grain yield of cultivars varying in levels of Ascochyta blight resistance. Trials were located at Turretfield (light clay over medium clay; annual rainfall (AR): 456 mm) and Hart (clay loam over heavy clay; AR: 399 mm), in the Mid North district of SA in 2004 and 2005. In 2005, trials were also located at Kalkee in the Wimmera (black cracking clay, AR: 450 mm) and at Beulah in the southern Mallee (calcareous sandy loam over medium clay, AR: 375 mm) of Victoria (Vic). Five fungicide regimes were compared in each experiment: Nil - no foliar fungicide sprays, Fortnightly application of chlorothalonil (Fn, 1440 gai/ha at SA sites and 750 gai/ha at Vic sites) applied every fourteen days from eight weeks after sowing till end of podding (six to nine sprays), Strategic application of chlorothalonil (St, three sprays at SA sites and four at Vic sites from eight weeks after sowing through to podding, single application at early podding of chlorothalonil (P°C), and mancozeb (PoM, 1650 gai/ha at SA sites and 750 gai/ha at Vic sites). The podding treatments at Turretfield in 2005 included a second application of the same chemical three weeks after the first. Cultivars varied across experiments depending upon seed availability and suitability for each district (Table 6). Nevertheless, all experiments included an Ascochyta blight S cultivar (Howzat® - desi type) and R cultivars (Genesis™ 090 – kabuli type and Genesis™ 509 – desi type). Ascochyta blight ratings of other cultivars evaluated were Genesis™ 508 (desi type R), SONAL® (desi type, MS) and Almaz® (kabuli type MR). All treatments were replicated four times utilising either split plot (SA sites in 2004 and Vic sites) or randomised complete block designs (SA sites in 2005).

Desi chickpea cultivars were sown to achieve a plant density of 50 plants/m² and kabuli cultivars 35 plants/m². Seed was inoculated with Group N rhizobium and sown with suitable rates of fertiliser at appropriate sowing dates for chickpeas in each cropping region. All experiments were sown in paddocks not containing or not close to chickpeas or chickpea stubbles, and were artificially inoculated with infected stubble approximately between five and six weeks after emergence to induce significant Ascochyta blight disease pressure.

The effect of foliar fungicide application timing

A significant interaction between cultivar and fungicide timing for foliar Ascochyta blight infection occurred at all sites. In all experiments, the R cultivars Genesis™ 090, Genesis™ 508 and Genesis™ 509 showed lower levels of foliar symptoms in all treatments (score <2.5 or <22%) (Table 6). In contrast, Ascochyta blight severity in nil treatments of the S cultivar Howzat® ranged from a score of 6.8–7.0 (Hart and Turretfield) in 2004 to greater than 9.0 and more than 90% infection in 2005 at Beulah, Turretfield, Hart and Kalkee. Disease ratings in nil treatments of MS (SONALI®) and MR (Almaz®) cultivars were intermediate with scores of 5.3 or 30–45% infection, respectively. However, these cultivars were only evaluated at sites with higher levels of disease intensity. Unlike the resistant cultivars, foliar symptoms in Howzat®, SONALI® and Almaz® were reduced substantially with Fn treatments. At sites with lower disease severity, the foliar disease level of Howzat® was reduced with St treatments compared to the nil treatment but not to the level of the Fn treatment. The St treatment had no effect at sites where disease levels were high. This partial reduction in foliar disease also occurred in SONALI® and Almaz® with St, PoM and P/C treatments at most sites. Gan et al. (2006) suggested application of foliar fungicides at seedling stages for MR cultivars to minimise the impact of intense showers of ascospores. Our data indicates that while S and partial resistant (MS and MR) cultivars may require this early spray to minimise disease intensity, R cultivars did not.

The interaction between cultivar and fungicide timing for grain yield was significant in all experiments, except Hart in 2004 where dry and hot seasonal conditions during podding resulted in low grain yields. Grain yield was reduced at all other sites in nil treatments of Howzat® compared to Fn treatments, with yield reductions ranging from 66 to 99% (Table 6). The grain yield of R cultivars was not always reduced in nil treatments when compared with Fn treatments. Where grain yield reductions in these cultivars did occur it was substantially lower than in all other cultivars, ranging from 0 to 37% in Genesis™ 090, 0 to 43% in Genesis™ 508 and 0 to 37% in Genesis™ 509. Nil treatments of SONALI® and Almaz® incurred yield loss in all experiments, ranging from 84 to 96% and 51 to 94%, respectively. The P/C and St treatments in Howzat®, SONALI® and Almaz® had substantially lower grain yields than those of the fortnightly treatments in all experiments. At sites with lower disease intensities such as Turretfield in 2004 and Beulah the St treatment reduced yield loss in Howzat® by only 12 and 17% respectively, compared to the Fn treatment. However, higher disease intensity grain yield losses increased to 50 and 96% at Hart in 2005 and Turretfield in 2005, respectively. A similar pattern also occurred in SONALI® for these treatments; however yield losses were generally less than for Howzat®. Grain yield loss of Almaz® when compared with the Fn treatment ranged from 20 to 64% in the P/C treatment and 13 to 76% in the St treatment, significantly less than that incurred for Howzat® and SONALI®. Grain yields were similar in the P/C and St treatment for Almaz®, whereas SONALI® and Howzat® generally had greater losses in the P/C treatment. This clearly indicates that Almaz® has a higher level of Ascochyta blight resistance than Howzat® and SONALI® and would require fewer foliar fungicides sprays for successful production.
<table>
<thead>
<tr>
<th>Site</th>
<th>Treatment</th>
<th>Howzat</th>
<th>Sonali</th>
<th>Almaz</th>
<th>Gen 090</th>
<th>Gen 508</th>
<th>Gen 509</th>
<th>Howzat</th>
<th>Sonali</th>
<th>Almaz</th>
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<th>Gen 508</th>
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</table>

Treatments P+C and St in Genesis™ 509 and Genesis™ 508 and the St treatment in Genesis™ 090 yielded the same as the Fn treatment in all experiments except for Turretfield in 2005. There was also no difference in grain yields of the P+C and Fn treatments of Genesis™ 090 at Turretfield in 2004 and Hart in 2005. However, grain yields were 17%, 13% and 19% below the PoM treatment.
lower in the PoC treatment at Beulah, Kalkee and Turretfield in 2005, respectively. In Turretfield 2005 a second spray of chlorothalonil was used in the PoC spray regime due to an extended period of wet weather and an abnormally long flowering and podding period. Ascochyta blight pressure was particularly severe in this experiment and even MR cultivars were likely to have incurred a yield penalty even with the Fn treatment (Table 8). Botrytis grey mould (Botrytis cinerea) disease was also present at Turretfield in 2005 and pod infection and abortion across all cultivars was observed, potentially confounding results, despite two applications of procymidine (250 gai/ha). Results indicated that if cultivars, unlike all other cultivars evaluated, could be successfully grown with fungicide applications only during podding, apart from in situations of extreme disease severity such as those at Turretfield in 2005.

**Efficacy of mancozeb and chlorothalonil fungicides as a podding spray**

Chlorothalonil had greater efficacy than mancozeb when applied as a podding spray at some sites, particularly on cultivars rated MR or less to Ascochyta blight. At Hart in 2005, pod infection was greater in all cultivars (Table 7) in the PoM compared to the PoC treatment. Grain yields and grain weight were less in all cultivars except Genesis™ 509 (R) and Genesis™ 508 (R). Grain yields at Turretfield in 2005 for SONALI® (MS), Almaz® (MR) and Genesis™ 090 (R) and Beulah in 2005 for SONALI® were significantly less in the PoM compared to PoC treatment. For all varieties at all sites grain yield in the PoM treatment were never significantly greater than PoC.

**Table 7: Comparison of chlorothalonil (PoC) and mancozeb (PoM) foliar fungicide treatments at podding on pod Ascochyta blight severity (0-100% assessed at maturity) and grain weight (g/100 seeds) of chickpea cultivars varying in resistance to Ascochyta blight at Hart in South Australia, 2005.**

<table>
<thead>
<tr>
<th>Site</th>
<th>Treatment</th>
<th>Howzat</th>
<th>Sonali</th>
<th>Almaz</th>
<th>Gen. 090</th>
<th>Gen. 508</th>
<th>Gen. 509</th>
<th>Howzat</th>
<th>Sonali</th>
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<th>Gen. 090</th>
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<td>58.8</td>
<td>11.3</td>
<td>9.3</td>
<td>4.0</td>
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<td>17.5</td>
<td>44.5</td>
<td>33.9</td>
<td>15.7</td>
<td>15.7</td>
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<tr>
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<td>PoM</td>
<td>96.3</td>
<td>97.5</td>
<td>80.0</td>
<td>75.0</td>
<td>40.0</td>
<td>38.8</td>
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<td>29.6</td>
<td>14.5</td>
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**Conclusion**

Cultivars with R to Ascochyta blight can be successfully grown under high disease pressure in southern Australia with only one or two fungicide applications during podding. This will allow growers to economically produce chickpeas again. Under the same conditions MS cultivars will require between three and nine fungicides to avoid severe yield loss. Cultivars with MR will require fungicide sprays prior to flowering in addition to podding sprays to prevent significant yield loss. This is likely to limit production of MR cultivars in southern Australia to the higher valued large seeded kabuli types, which are best adapted to the medium to high-rainfall growing areas. 37

Differing spray programs have been developed based on each variety’s ascochyta rating. Chickpea ascochyta fungicides are protectants only—unlike wheat stripe rust fungicides—they have no systemic or kick-back action, and they will not eradicate an existing infection. To be effective they must be applied before infection i.e. before rain. The key to a successful ascochyta spray program is regular monitoring combined with timely application of registered fungicides (Table 8 and 9). 38

---


### Table 8: Foliar fungicides for the control of Ascochyta and Botrytis grey mould.

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<th>Example trade name</th>
<th>Rate</th>
<th>Ascochyta blight</th>
<th>Botrytis grey mould</th>
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</thead>
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<td>1.0–2.0 L/ha</td>
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</tr>
<tr>
<td></td>
<td>Barrack</td>
<td></td>
<td></td>
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<td></td>
<td>Betterstick® #</td>
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<tr>
<td></td>
<td>Nufarm Unite® 720#</td>
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<tr>
<td>Mancozeb (750 g/kg)</td>
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<td>1.0–2.2 kg/ha</td>
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<td>Mancozeb (420 g/L)</td>
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<td>Not registered</td>
<td></td>
</tr>
<tr>
<td>Carbendazim (500 g/L)</td>
<td>Spin Flo®</td>
<td>Not registered</td>
<td>500 mL/ha</td>
<td></td>
</tr>
</tbody>
</table>

*# These are the only registered chlorothalonil products. It is an offence to use any other product. Refer to current product label for complete ‘Direction for use’ prior to application.*

Source: Pulse Australia

### Table 9: Chickpea foliar fungicides for the southern region.

<table>
<thead>
<tr>
<th>Company</th>
<th>Active Ingredient</th>
<th>Rate</th>
<th>Av.Cost($/ha)</th>
<th>Chickpeas</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Barrack®</td>
<td>Crop Care</td>
<td>Chlorothalonil</td>
<td>1.0-2.0 L/ha</td>
<td>$16.70-$33.42</td>
</tr>
<tr>
<td>2. Unite 720®</td>
<td>2. Nufarm</td>
<td>720 g/L</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>3. Bravo®</td>
<td>3.4 Syngenta</td>
<td></td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>4. Bravo Weather Stik®</td>
<td></td>
<td></td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>1. Bavistin®</td>
<td>1. Crop Care</td>
<td>Carbendazim</td>
<td>500 mL/ha</td>
<td>$10.00-$13.50</td>
</tr>
<tr>
<td>2. Carbend®</td>
<td>2.3 Nufarm</td>
<td>500 g/L</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>3. Spinflo®</td>
<td>4. Farmoz</td>
<td></td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>4. Howzat®</td>
<td></td>
<td></td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>1. Penncozeb 420SC®</td>
<td>1. Nufarm</td>
<td>Mancozeb 420 g/L</td>
<td>1.8-3.5 L/ha*</td>
<td>$11.10-$26.60</td>
</tr>
<tr>
<td>1. Penncozeb 750 DF®</td>
<td>1. Nufarm</td>
<td>Mancozeb 750 g/kg</td>
<td>1.0-2.2 kg/ha*</td>
<td>$7.60-$16.70</td>
</tr>
<tr>
<td>2. Dithane Rainshield®</td>
<td>2. Dow</td>
<td></td>
<td></td>
<td>✓</td>
</tr>
<tr>
<td>1. Polyram®</td>
<td>1. Nufarm 700 g/</td>
<td>Metiram</td>
<td>2.0 kg/ha</td>
<td>✓</td>
</tr>
<tr>
<td></td>
<td>kg</td>
<td></td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>1. Sumisclex®</td>
<td>1. Sumitomo</td>
<td>Procymidine 500 g/L</td>
<td>500 ml/ha</td>
<td>$16.40-$33.80</td>
</tr>
</tbody>
</table>

Note: Observations in 2010 Tamworth trials indicated that the natural resistance all plants have to pathogens and pests is compromised when plants are stressed from waterlogging and that this reduced the ability to manage ascochyta with a fungicide strategy that worked in less stressed plots. In a season when repeated cycles of infection occur, even MR varieties can have yield-reducing levels of disease. 39

Source: NDSU

Fungicide information

Chlorothalonil

Pulse Australia’s application has been denied by the Australian Pesticides and Veterinary Medicine Authority (APVMA) for an emergency use permit to allow for the use of non-registered chlorothalonil formulations. There are a small number of registered chlorothalonil products that have label instructions that do allow for grazing. PIRSA’s Rural Chemicals Operations group of Biosecurity SA provided advice for growers regarding the restrictions on grazing instructions when using chlorothalonil applied to pulse crops. Under SA’s Agricultural and Veterinary Chemical (Control of Use) legislation, chemical users are required to follow all mandatory instructions and withholding period advice on the label specific to the chemical product and label they are using. For most chlorothalonil products, the label instructions for grazing states ‘Do not graze livestock on treated crops’. This advice not to graze livestock must be followed when using a product that has this statement.

Chemical users should be aware that there are a small number of registered chlorothalonil products that have label instructions that do allow for grazing providing the relevant withholding period and export slaughter interval information on the label is followed. These labels state ‘Do not graze for 14 days after application’, and have additional export slaughter interval statements for livestock going for export that states, ‘Livestock that have been grazed or fed treated forage, fodder or stubble should be placed on clean feed for 63 days (nine weeks) prior to export slaughter’. These label statements are the result of the chemical companies that manufactured those specific products providing data when registering the product with the APVMA that supports these grazing claims. Grazing is only allowed when the specific products with label statements that allow for grazing are used. Producers should check with their chemical reseller or consultant which chlorothalonil products allow for grazing.

APVMA pulse permits

Pulse Australia has arranged for the following off-label permits to be issued by the apvma for use this season (2017). These permits re in addition to those already issued and current.

<table>
<thead>
<tr>
<th>Permit</th>
<th>Product Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>PER84309</td>
<td>Azoxystrobin / Cyproconazole Ascochyta in Chickpeas / Lentils</td>
</tr>
<tr>
<td>PER84336</td>
<td>Procymidone Botrytis Grey Mould in Chickpeas</td>
</tr>
<tr>
<td>PER84407</td>
<td>Prosaro (TM) Ascochyta in Chickpeas / Lentils</td>
</tr>
<tr>
<td>PER84408</td>
<td>Boscalid Botrytis Grey Mould in Chickpeas / Lentils</td>
</tr>
<tr>
<td>PER84461</td>
<td>Cypredinil Ascochyta in Chickpeas / Faba Beans</td>
</tr>
</tbody>
</table>


Botrytis grey mould

Botrytis grey mould (BGM) in chickpea is caused by the fungus *Botrytis cinerea*. *B. cinerea* is a significant pathogen of pulse crops particularly lentils, ornamental plants grown under glasshouse conditions, and fruit, including grapes, strawberries and apples. Flowers are especially vulnerable to BGM infection. *B. cinerea* does not infect cereals or grasses.

*B. cinerea* has been recorded on over 138 genera of plants in 70 families. Legumes and asteraceous plants comprise approximately 20% of these records. As well as being a serious pathogen, *B. cinerea* can infect and invade dying and dead plant tissue. This wide host range and saprophytic capacity means inoculum of *B. cinerea* is rarely limiting. If conditions favour infection and disease development, BGM will occur. This makes management of BGM different from chickpea ascochyta, which is more dependent on inoculum, at least in the early phases of an epidemic.
B. cinerea also causes pre and post-emergent seedling death. This happens when chickpea seed, infected during a BGM outbreak, is used for sowing. Seedling disease does not need the wet conditions that are usually required for infection and spread of BGM later in the crop cycle.

**Economic importance**

BGM is a serious disease of chickpeas in southern Australia and can cause total crop failure. Discoloured seed may be rejected or heavily discounted when offered for sale. If seed infection levels are >5% then it may be worth grading the seed. Crop losses are worst in wet seasons, particularly when crops develop very dense canopies.

### 9.5.8 Varietal resistance or tolerance

See Table 3.

### 9.5.9 Damage caused by disease

It can be seed-borne, attacking the seedling during emergence and causing rot on the upper taproot and collar. Affected areas develop a soft rot and a fluffy grey mould. Significant losses can occur in wet springs in crops with dense canopies. As well as being a serious pathogen, *B. cinerea* can infect and invade dying and dead plant tissue. This wide host range and saprophytic capacity means inoculum of *B. cinerea* is rarely limiting. If conditions favour infection and disease development, BGM will occur.

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### 9.5.10 Symptoms

The first symptom of BGM infection in a crop is often drooping of the terminal branches. If groups of plants are infected, these may appear as yellow patches in the crop (Photo 6). The diagnostic feature is a grey ‘fuzz’ which, under high humidity, develops on flowers, pods, stems and on dead leaves and petioles.

**Photo 6:** If groups of plants are infected, these may appear as yellow patches in the crop.

Photo: Phil Davies, Pulse Australia

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Lesions can develop anywhere along the stem but are usually first found on the lower part of the stems often starting in leaf axils (Photo 7). Infected seeds are usually smaller than normal and are often covered with white to grey fungal growth.

Photo 7: Lesions are usually first found on the lower part of the stems often starting in leaf axils.
Source: Pulse Australia

Infected seeds are usually smaller than normal and are often covered with white to grey fungal growth (Photo 8).

Photo 8: Botrytis grey mould on seed.
Source: (left) G. Cumming, Pulse Australia and (right) Pulse Australia

When a severely BGM-infected canopy is opened, clouds of spores are evident (avoid inhaling these). During dry weather the ‘fuzz’ is not obvious, but it develops again when wet weather returns (Photo 9). Small, dark-brown/black resting bodies (sclerotes) of *B. cinerea* may develop on infected dead tissue, and are capable of producing spores on their surface.

Photo 9: BGM on a chickpea flower.
Source: Phil Davies, Pulse Australia
The stem lesions caused by BGM can be confused with those caused by *Sclerotinia sclerotiorum* (at and above ground level) and by *Sclerotinia minor* (at ground level), but neither of these pathogens produce the grey 'fuzz' typical of BGM. Also, sclerotinia lesions tend to remain white, and are covered by a dense cottony fungal growth, in which irregular shaped black sclerotes develop.

In contrast, the sclerotes of *B. cinerea* are more rounded and usually develop after the stems die. They are smaller than the sclerotes of *S. sclerotiorum*, but larger than the angular sclerotes of *S. minor*.  

### 9.5.11 Conditions favouring development

Factors that favour infection and spread of BGM in favourable seasons include:

- early sowing (mid-April to early May) and narrow rows
- frequent overcast, showery weather
- limited supply of effective fungicides
- lack of BGM tolerant/resistant varieties.

High biomass crops and early canopy closure often results in high in-crop humidity and poor penetration of fungicides. If the crop becomes lodged the situation is exacerbated.

Rainy weather not only favours the disease but wet paddocks also limit the spray opportunities for ground rigs.

Following a season where widespread BGM infection has occurred in a district there is often a shortage of disease-free seed for planting and there is a high quantity of infected crop residue across a large area. Both of these factors will increase the disease risk for the following year. Whether BGM becomes a problem the following year will depend on seasonal conditions.

Over 10 million spores can be produced on a single 2 cm long lesion on a chickpea stem. Consequently, *B. cinerea* has the capacity to rapidly develop during conducive weather conditions. The spores can be blown many kilometres, and if deposited on chickpea plants they can remain dormant until conditions favour spore germination.

Free moisture is necessary for germination and infection. Lesions and the grey 'fuzz' are evident 5–7 days after infection under ideal conditions.

*B. cinerea* is favoured by moderate temperatures (20–25°C) and frequent rainfall events. It does not become a risk until the average daily temperature (ADT) is 15°C or higher. The combination of early canopy closure, prolonged plant wetness and overcast weather results in high relative humidity and rapid leaf death in the canopy, conditions which are ideal for *B. cinerea*.

*B. cinerea* can survive on and in infected seeds, in infected stubble, on alternative hosts, in dead plant tissue and as sclerotes. The relative importance of these in Australia is unknown, but recent research in Victoria demonstrated that *B. cinerea* can survive for up to 18 months on infected stubble under field conditions. Other research from WA suggests that sclerotes of *B. cinerea* cannot survive over summer because they lose their viability during hot weather.

Higher seeding rates lead to greater canopy vigour, increased lodging and under ideal growing conditions can increase the risk of BGM.

The fungus survives on infected seed, as a saprophyte on decaying plant debris and as soil-borne sclerotia. The disease is often established in new areas by sowing infected seeds. Masses of spores are produced on infected plants. These fungal spores can be carried from plant to plant by air currents and spread the disease rapidly, (Figure 7). Once a crop has become established, the warm, humid

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conditions under the crop canopy provide ideal conditions for infection and spread of the disease.

**Figure 7: Disease cycle of *Botrytis* grey mould in chickpeas.**

Illustration by Kylie Fowler Source: CropPro

### 9.5.12 Management of disease

#### Stubble management

It is likely that the pathogen can remain viable and capable of survival for as long as infected stubble remains on the soil surface. Burial of stubble removes the ability of *B. cinerea* to produce spores that can be blown around, and increases the rate of stubble breakdown by soil microbes.

Although burning of infected residues will also significantly reduce the amount of infected residues on the soil surface, it will not guarantee freedom from BGM in the following season.

Burying or burning stubble can significantly increase the risk of soil erosion and reduce water infiltration. 43

#### Volunteer control (the green bridge)

Volunteer chickpea plants growing in or near paddocks where BGM was a significant problem are a likely method of carryover and must be managed by application of herbicide or cultivation.

This will also reduce carryover of ascocryta. 44

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Seed source and treatment

Obtain seed from a commercial supplier, or from a source known to have negligible levels of BGM. Irrespective of the source, all seed must be thoroughly treated with a registered fungicide seed dressing. Thiram-based fungicide seed dressings are effective in significantly reducing, but not entirely eliminating, BGM from infected seed. 45

See Section 3 Planting, 3.2 Seed treatments for more information.

Seedling emergence

Research on harvested seed has shown a germination test does not accurately predict emergence. Accordingly, growers are advised to conduct their own emergence test, as follows:

- After grading and treatment, sow 100 seeds at least 5 cm deep in the paddock that you intend for chickpeas and water if necessary.
- Count the number of seedlings that have emerged after one, two and three weeks and note their appearance. Do they look healthy or are they stunted and distorted?
- If you want to get an idea of variability in emergence and the paddock, replicate the test i.e. sow 100 seeds in 3–4 different locations in the paddock. This will also help identify potential herbicide residue problems. 46

Paddock selection

Paddocks in which chickpeas were affected by BGM should not be re-sown to chickpea, faba bean or lentil the following season. Nor should chickpea be sown beside paddocks where BGM was an issue the previous season.

As for Ascochyta blight, chickpeas should be grown as far away from paddocks in which BGM was a problem as is practically possible.

However, under conducive conditions, this practice will not guarantee that crops will remain BGM free, because of the pathogen’s wide host range, ability to colonise dead plant tissue, and the airborne nature of its spores. 47

Sowing time and row spacing

If long-term weather forecasts suggest a wetter-than-normal year (La Nina), consider sowing in the later part of the suggested sowing window for your district and on wider rows (e.g. 100 cm). Planting on wider rows results in increased air movement through the crop and reduced humidity within the canopy. Higher seeding rates lead to greater canopy vigour, increased lodging and under ideal growing conditions can increase the risk of BGM.

Varietal resistance

All current commercial varieties suitable for the northern region are susceptible to BGM, although Howzat46 is reported to have slightly better resistance than other varieties.

Fungicide treatment

In areas outside central Queensland, spraying for BGM is not needed in most years.

However, in seasons and situations favourable to the disease, a preventative spray of a registered fungicide immediately prior to canopy closure, followed by another application two weeks later, will assist in minimising BGM development in most years.

If BGM is detected in a district or in an individual crop, particularly during flowering or pod-fill, a fungicide spray should be applied before the next rain event (Table 10).

None of the fungicides currently registered or under permit for the management of BGM on chickpea have eradicant activity, so their application will not eradicate established infections. Consequently, timely and thorough application is critical. 48

Sumitomo Sumisclex® 500 fungicide plus other registered products containing: 500 g/L PROCYMIDONE as their only active constituent APVMA Permit PER82976

This permit is in force from 19 July to 30 November 2016 for use in chickpea for the control of Botrytis grey mould.

Table 10: Foliar fungicides for the control of Ascochyta and Botrytis grey mould.

<table>
<thead>
<tr>
<th>Active ingredient</th>
<th>Example trade name</th>
<th>Rate</th>
<th>Ascochyta blight</th>
<th>Botrytis grey mould</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorothalonil (720 g/L)</td>
<td>Crop Care Barrack® 720# Barrack Betterstick® # Nufarm Unite® 720#</td>
<td>1.0–2.0 L/ha</td>
<td>Not registered</td>
<td></td>
</tr>
<tr>
<td>Mancozeb (750 g/kg)</td>
<td>Dithane™ Rainshield™</td>
<td>1.0–2.2 kg/ha</td>
<td>1.0–2.2 kg/ha</td>
<td></td>
</tr>
<tr>
<td>Mancozeb (420 g/L)</td>
<td>Penncozeb® SC</td>
<td>1.8–3.95 L/ha</td>
<td>Not registered</td>
<td></td>
</tr>
<tr>
<td>Carbendazim (500 g/L)</td>
<td>Spin Flo®</td>
<td>Not registered</td>
<td>500 mL/ha</td>
<td></td>
</tr>
</tbody>
</table>

# These are the only registered chlorothalonil products. It is an offence to use any other product. Refer to current product label for complete ‘Direction for use’ prior to application.

Source: Pulse Australia

9.6 Phytophthora root rot

PRR is a disease of chickpea caused by the fungus-like oomycete Phytophthora medicaginis. It can cause significant yield losses in wetter-than-normal seasons or following periods of soil saturation in normal seasons. Lucerne, perennial and annual medics (Medicago species) and other leguminous plants including sulla (Hedysarum species) and sesbania (Sesbania species) can also host P. medicaginis. 49

PRR is not usually an issue in the southern region.

9.6.1 Varietal resistance or tolerance

See Table 3.

9.6.2 Symptoms

Infection by P. medicaginis can occur at any growth stage, causing seed decay, pre-and post-emergence damping-off, loss of lower leaves, and yellowing, wilting and death of older plants (Photo 10). The disease is usually observed late in the season but may also affect young plants. Badly affected seedlings suddenly wither and die with no obvious disease symptoms.

Infected plants are often stunted with obvious yellowing and drying of the foliage. They have few lateral roots and the lower portion of the tap root is often decayed (Photo 11). The remaining tap root is usually discoloured dark-brown to black. Sometimes the discolouration can extend to the base of the plant. The advancing margins of the lesions may also have a reddish-brown discolouration.

Photo 10: Cultivated areas killed by phytophthora. Only plants on tops of contours survived (left) and phytophthora in water course (right).

Photos: Mark Schwinghamer, Source: Pulse Australia

Photo 11: Severely affected plants have no lateral roots (right) and defoliation below tips of stems.

Photo: Joe Wessels, CropPro
Photo 12: New roots forming from the top of the taproot.
Photo: Mike Fuhlbohm, Source: Pulse Australia

Symptoms are sometimes delayed if temperatures are cool and the soil is moist. Lateral roots and tap root die, or dark-brown/black lesions often girdle the taproots (Photo 12). On young plants the lesions may extend up the stem for 10 mm or more above ground level (Photo 13).

Photo 13: PRR basal lesions extending up the plant stem.
Photo: Mal Ryley, Source: Pulse Australia

Plants with phytophthora can be easily pulled from the soil. If conditions are mild, affected plants may partially recover by producing new roots from the upper part of the tap root. 50

9.6.3 Phytophthora and waterlogging

PRR and waterlogging have similar symptoms (Table 11) and are both induced by transient or prolonged soil saturation and surface water. They usually occur in low lying areas of paddocks, or where water accumulates such as on the low side of contour banks or in watercourses, or where the soil has been compacted or has hard pans. However, under very wet conditions, entire paddocks can be affected.

Table 11: Symptoms of PRR and waterlogging.

<table>
<thead>
<tr>
<th>Phytophthora root rot</th>
<th>Waterlogging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organism kills roots</td>
<td>Low oxygen kills roots</td>
</tr>
<tr>
<td>Chickpea, medicos, lucerne are hosts</td>
<td>No link with cropping history or weed control</td>
</tr>
<tr>
<td>Occurs any time of year</td>
<td>Usually occurs later in the year</td>
</tr>
<tr>
<td>Symptoms onset occurs after a week or more</td>
<td>Symptoms onset occurs quite rapidly</td>
</tr>
<tr>
<td>Lower leaves often yellow and fall off</td>
<td>Plants die too fast for leaves to yellow or fall</td>
</tr>
<tr>
<td>Roots always rotted and discoloured</td>
<td>Initially roots not rotted or discoloured (tips black)</td>
</tr>
<tr>
<td>Plants easily pulled up and out</td>
<td>Plants not easily pulled up initially</td>
</tr>
<tr>
<td>Manage through paddock rotation varietal choice</td>
<td>Manage through paddock selection, no irrigation in reproductive phase</td>
</tr>
</tbody>
</table>

Source: Pulse Australia

Symptoms of waterlogging can be confused with those of phytophthora but differ in that:

- plants are most susceptible to waterlogging at flowering and early pod-fill
- symptoms develop within two days of flooding compared to at least seven days for phytophthora
- roots are not rotted and are not easily pulled from the soil at first
- plants often die too quickly for the lower leaves to drop off.

9.6.4 Conditions favouring development

*Phytophthora medicaginis* survives in soil mainly as thick-walled oospores, but some strains also survive as chlamydospores. Oospores can survive in soil for at least 10 years. In saturated soil the exudates from the roots of chickpea and other hosts stimulate the oospores to germinate and produce lemon-shaped sporangia. Inside these sporangia, zoospores develop and are released into the soil and surface water, where they are carried by moving water and ‘swim’ towards the roots and collars of chickpea plants.

Zoospores encyst on the root surfaces and germinate to produce hyphae that invade the roots. New sporangia develop from infected roots enabling further cycles of infection to occur. Later, oospores are formed in the infected roots.

Zoospores are only capable of ‘swimming’ for a few millimetres, so long distance dispersal of *P. medicaginis* is by physical movement of soil and water infested with oospores, sporangia, zoospores and/or chlamydospores during floods and irrigation or by machinery. 51

9.6.5 Management of PRR

Though, PRR is not usually an issue in the southern region, the disease can be reduced by seed treatments containing metalaxyl (Apron XL®, Rampart® or Mantle®). 52

Once a plant or crop is infected with phytophthora, there is nothing a grower can do.

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There are no effective chemical sprays as there are for ascochyta and botrytis. Therefore, phytophthora can only be managed by pre-sowing decisions and assessing risks for individual paddocks.

Development of the disease requires both the pathogen in the soil, and a period of soil saturation with water. Losses in a phytophthora-infested paddock may be minor if soil saturation does not occur.

The most effective control strategy is to not sow chickpeas in high-risk paddocks, which are those with a history of:

- phytophthora noted in previous chickpea or lucerne crops
- lucerne or annual or perennial medics
- waterlogging or prone to flooding
- metalaxyl-based seed dressings
- poor drainage.

Do not flood irrigate after podding has commenced especially if the crop has been stressed.

However, if you choose to sow chickpeas in high-risk paddocks, the following measure will reduce losses from phytophthora:

- Growing a chickpea variety with the highest level of resistance. Particularly in medium-risk situations, where medic, chickpea or lucerne crops have been grown in the past 5–6 years. 53

### 9.6.6 New tool to determine risk of chickpea PRR

**Key points:**

- Increasing level of inoculum (oospores/plant) of *Phytophthora medicaginis* (Pm) was strongly correlated with decreasing yield of the moderately resistant variety YORKER®.
- An inoculum level of 660 oospores/plant (PreDicta B™ > 5000 Pm copies/g soil) at sowing significantly reduced yields compared with lower inoculum levels under both dryland and irrigated conditions.
- Testing soil samples from growers’ 2015 paddocks confirmed the results of testing 2014 samples that the PreDicta B™ soil Pm test can identify Pm in growers’ paddocks.
- These findings provide further evidence that the PreDicta B™ Pm test will be a useful tool for growers to determine their risk of PRR.

**Phytophthora medicaginis detection in soil**

*Phytophthora medicaginis* (Pm), the cause of chickpea PRR, is endemic and widespread in northern to central NSW. Under conducive conditions, PRR can cause 100% loss. The pathogen survives from season to season on chickpea volunteers, lucerne, native medics, sulla and as resistant structures (oospores) in roots and soil.

A PreDicta B™ soil DNA test has been developed by the South Australian Research and Development Institute (SARDI) to quantify the amount of Pm DNA in soil samples and so provide a measure of the amount of Pm inoculum (infected root tissue and oospores) in paddocks. SARDI reports on the second season of studies to assess the capability of this test to:

1. Detect Pm inoculum in soil from commercial paddocks
2. Predict the risk of PRR disease and potential yield losses in chickpea

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Pm DNA sampling in paddocks and disease risk determination

The DNA result for a soil sample from a paddock can only provide an indication of inoculum concentration and disease risk for the areas of the paddock which were sampled. Therefore, the spread and locations of sampling across a paddock will affect how representative DNA results are for a paddock. Because of the risk of rapid PRR disease build-up following wet conditions it may be appropriate to treat a negative PreDicta B™ test result as indicating a low risk rather than a nil risk, as the pathogen could still be in areas of the paddock that were not sampled and so still cause PRR and reduce yield.

To maximise the probability of determining the PRR risk of a paddock, target those areas of the paddock where Pm is more likely to occur. The pathogen thrives in high soil moisture contents and so often occurs in low lying regions of paddocks where pooling following rain may occur. The pathogen also carries over from season to season on infected chickpea volunteers, lucerne and, native medics. Including low lying areas and weedy areas of paddocks during PreDicta B™ soil sampling may provide the best strategy to identifying a paddocks disease risk of PRR in chickpea.

9.7 Sclerotinia

Sclerotinia, caused by Sclerotinia sclerotiorum and trifoliorum, is an occasional disease of chickpeas but has caused significant crop losses in eastern Australia. Sclerotinia can cause serious crop losses where a substantial number of plants within a crop are affected. Kabuli chickpeas appear more susceptible to this disease than desi chickpeas but both types can be seriously damaged under favourable conditions. Dense crops are likely to be the most severely affected, particularly under moist conditions. Grain quality can be decreased when infected with sclerotinia, which causes poor colour and shrivelled seed.

9.7.1 Damage caused by disease

Sclerotinia can cause severe damage in chickpeas. This has occurred in kabuli chickpeas in Victoria. Sclerotinia has caused significant crop losses where a substantial amount of the crop is infected. This disease has caused total crop failure where chickpeas were sown in the same paddock in successive years. However, in many situations it only affects a small proportion of plants within the crop.

Kabuli chickpeas are most susceptible to this disease though desi chickpea can also be badly affected under conditions favourable for the disease. Dense crops are likely to be affected, particularly under moist conditions. Grain quality can be decreased when infected with sclerotinia. It causes poor colour and shrivelled seed.

9.7.2 Symptoms

There are two Sclerotinia spp that attack chickpeas and they can be distinguished by the size of their sclerotes (survival structures):

- S. sclerotiorum produces large irregular shaped sclerotes 5–10 mm in diameter as high up as 20–30 cm on the stem
- S. minor produces sclerotes that are angular and much smaller, rarely larger than 2–3 mm in diameter

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**What to look for:**

A small number of dead plants scattered throughout a paddock. Affected plants first wilt and rapidly die, often without turning yellow. Later, as the plant dries out the leaves turn a straw colour (Photo 14).

![Photo 14: Plants killed by S. sclerotiorum (left). Fungal weft of sclerotinia in the lower canopy of a chickpea crop (right).](source)

On the surface of the root, just below ground level, small black fungal bodies called sclerotia (which are irregular in size and shape), can sometimes be seen mingled with white cottony fungal mycelium.

In spring, water-soaked spots may appear on the stems and leaves. Affected tissues develop a slimy soft rot from which droplets of a brown liquid may exude. Infected tissues then dry out and may become covered with a web of white mycelium of the fungus (Photos 15 and 16).

![Photo 15: Early symptoms of stem infection by sclerotinia. White mycelial growth starting to develop (left). RIGHT: Comparison of stem infections caused by sclerotinia (top stem) and botrytis (lower stem)—note the different colour of fungal growth.](source)
Photo 16: Sclerotinia stem infection of chickpeas. White fluffy mycelium and sclerotia formation evident.

Source: CropPro

9.7.3 Conditions favouring development

The disease is usually established from sclerotia (survival bodies of the fungus) present in the soil or introduced with contaminated seed. Outbreaks are more common when very wet conditions occur in July.

The sclerotia germinate in moist soil and either directly infect roots or produce airborne spores (Photo 17) which attack the above-ground parts of the plant. Once established, the fungus rapidly moves to adjacent healthy tissue. Within a few days of infection, plants start to wither then die.
Sclerotia formed on infected plants enable the fungus to survive to the following year. Individual seeds can be contaminated with the fungus and/or sclerotia may be present in the seed sample. Sclerotia can remain viable in the soil for up to eight years (Figure 8).

Soil-borne sclerotia are the most important disease source for establishing disease in following crops. Seeds infected with sclerotinia can be the cause of disease establishment in otherwise sclerotinia-free areas.  

9.7.4 Management of sclerotinia

Before sowing

Use clean seed

Use of disease-free seed minimises the risk of disease and prevents establishment into a new area. It is important to avoid sowing chickpea in areas where the disease is known to be present. The seed harvested from infected crops should not be used for sowing.

Crop rotation

Crop rotation is the best method of control once the disease has become established. Cereal crops are not affected by sclerotinia and provide a good disease break. Pulse crops, oilseeds, legume-based pastures and capeweed are all good hosts to this disease.

If a severe sclerotinia problem does occur, a four-year break from susceptible crops is required to substantially reduce the number of sclerotia in the soil. The most practical option is to use cereals and legumes such as field peas or vetch which have some resistance to sclerotinia. In addition, burning of the disease-infected stubble should be considered. Deep ploughing (5 cm) will also reduce the number of sclerotia, and so minimise disease carry over. Where a minor sclerotinia problem occurs, a two-year break from susceptible crops is advisable.
No commercial seed treatments or fungicides are known to manage this disease in crop.  

**IN FOCUS**

Use of non-conventional chemicals as an alternative approach to protect against sclerotinia

Four non-conventional chemicals, viz., zinc sulphate (ZS), oxalic acid (OA), sodium malonate (SM) and sodium selenite (SS), were applied as foliar sprays to chickpea and the plants were subsequently challenged against *Sclerotinia sclerotiorum*, the causal agent of stem rot in chickpea. All the chemicals reduced mortality of chickpea from *S. sclerotiorum* infection. Among them, ZS at 10-3 mmol gave the best result as only 13.6% mortality was recorded after 28 days compared to 100% in the control. High performance liquid chromatographic analysis of treated chickpea leaves revealed activation of shikimic acid as well as phenyl propanoid pathways and synthesis of several phenolic compounds increased specially after application of OA, ZS and SM. Individual treatment of the chemicals showed better results than their combinations as plant mortality was reduced and accumulation of phenolics increased in their individual treatments. A positive correlation was observed between induction of phenolic compounds and survival of the plants. In vitro assay of the four chemicals showed only SS to be antifungal. The protection of plants by ZS, OA and SM is possibly because of induction of resistance in the host against *S. sclerotiorum*.  

Consult your local agronomist about potential treatment strategies for your area.

**9.8 Phoma stem rot**

Phoma caused by the fungus *Phoma medicaginis* var. *pinodella*, has the potential to be a serious disease of chickpea. relatively few serious outbreaks have occurred; however, the disease is common in southern Australia. The disease can cause serious crop losses in seasons with above average winter rainfall. Careful paddock selection and use of fungicide seed dressings can minimise the impact of this disease.

**9.8.1 Symptoms**

*What to look for*

Seed-borne infection often results in black-brown discolouration of the root near where the seed is attached (Photo 18). Blackening may spread up the root and cause lesions at the base of the stem.

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Initial above-ground symptoms are small, dark tan coloured, irregular flecks on leaves, stems, and pods. The flecks on leaves enlarge to lesions and the surrounding tissue yellows. Within the lesions numerous pinhead-sized black fruiting bodies of the fungus develop. On the stem, similar but more elongated lesions form.

Black lesions may completely girdle the base of the stem and root where infection is severe (Photo 19). Pod lesions are sunken, with pale centres and dark margins, and may be covered by small black spots. The fungus may penetrate the pod and infect developing seeds. Badly affected plants may be totally defoliated when infected leaflets senesce and fall.

**Photo 19:** Stem lesions caused by Phoma infection.

Source: CropPro

9.8.2 Conditions favouring development

Phoma can survive on infected seed, in soil and on crop residue from one season to the next. Infection can occur at any stage of plant growth provided conditions are favourable. Moisture is essential for infection to occur. During wet weather, the disease may spread further when spores of the fungus are carried by wind and rain splash onto neighbouring plants (Figure 9). Pod infection can occur when the fungus penetrates the pod wall and infects developing seeds late in the season.

The only serious outbreaks of this disease on chickpea in Australia have occurred in very wet years. However, it is usually a more damaging disease on field pea than chickpea.
Figure 9: Disease cycle of Phoma stem rot of chickpeas.
Illustration by Kylie Fowler, CropPro

9.8.3 Managing Phoma

Before sowing

Use clean seed

The use of disease-free seed and crop rotation will help prevent the establishment and build-up of this disease.

Crop rotation

Where chickpeas have been badly infected, a two-year break from host crops will minimise the disease risk. Crops which host Phoma include field pea, chickpea, faba bean, lupin, lentil, vetch and legume pasture species. Cereal and oilseed crops will provide a good disease break.

Chemical control

Seed-borne disease infection can sometimes be controlled with fungicide seed dressings. No fungicides are known to manage this disease in crop.  

9.9 Root rots including damping-off (*Fusarium*, *Rhizoctonia* and *Pythium* spp.)

All fungi responsible for root rot are soil dwellers. They can survive from crop to crop in the soil, either on infected plant debris or as resting spores. In wet soils, these fungi can invade plant roots and cause root rot. Wet conditions also encourage the spread of disease within a field.

9.9.1 Economic importance

Root rot diseases can occasionally be serious especially when soils are wet for prolonged periods. The reduced root development causes the plants to die when they are stressed.

9.9.2 Symptoms

Affected seedlings gradually turn yellow and leaves droop. The plants usually do not collapse. The taproot may become quite brittle, except in *Pythium* root rot when they become soft. When plants are pulled from the ground the portion of the root snaps off and remains in the soil. The upper portion of the taproot is dark, shows signs of rotting and may lack lateral roots. Distinct dark-brown to black lesions may be visible on the taproot (Photo 20). The leaves and stems of affected plants are usually straw-coloured, but in some cases may turn brown. Older plants dry-off prematurely and are often seen scattered across a field. In some cases, especially with kabuli, seeds may rot before they emerge.

Photo 20: *Rhizoctonia* root rot. Optimum soil temperature is 24–26°C; disease is worse on light sandy soils.
9.9.3 Management options

Root rot disease can be reduced by crop rotation. As this disease may also affect other pulses, chickpeas should be sown in rotation with another non-legume crop. Chickpeas should not be grown in areas subject to waterlogging. Damping-off in kabuli chickpeas can be controlled using fungicide seed treatment.

9.10 Collar rot (*Sclerotium rolfsii*)

9.10.1 Economic importance

Collar rot is generally a minor disease in chickpea. However, the disease has been particularly severe in irrigated Macarena (kabuli).

9.10.2 Symptoms

This disease is commonly observed at very low levels in chickpea crops (up to 6 weeks after sowing) sown during warmer conditions, as isolated dead seedlings with a coarse web of white fungal threads encasing the tap root. However, in irrigated systems, the fungus can kill significant numbers of plants. The coarse threads of the fungus can be seen on or just under the soil surface, colonising decomposing trash or on the plant itself (Photo 21); these webs of mycelium can cover quite a substantial area around plants. On chickpea, plants will be killed outright and quite rapidly as the fungus invades around the soil level and girdles the vascular tissue. Plants will wilt and become bleached (a result of a toxin produced by the fungus), younger seedlings may collapse but older plants may simply dry (without collapse). The characteristic signs of the pathogen will be the webs of coarse mycelium and the small (~1–2 mm) spherical brown sclerotia (survival and resting structures) of the fungus that attach to the fungal threads. The sclerotia look like canola seeds.

![Photo 21: Webs of Sclerotium rolfsii mycelium at the base of an infected chickpea plant.](Photo: K McCosker)

9.10.3 Conditions favouring development

The fungus has a very wide host range including monocots (such as millet and barley) and dicots (such as cotton). The pathogen is also the causal agent of white mould in peanuts.

The pathogen rarely occurs where average winter temperatures fall below 0°C. The fungus survives in the soil mainly as sclerotia that remain viable for 2–3 years, but occasionally it persists as mycelium in infected tissues or plant residues. Sclerotia germinate by hyphal or eruptive germination. Hyphal germination is characterised
by the growth of individual hyphae from the sclerotial surface, while eruptive germination is characterised by plugs or aggregates of mycelium bursting through the sclerotial surface.  

9.10.4 Management options

The disease is favoured by the presence of undecomposed organic matter on the soil surface and excessive moisture. If possible, avoid wetting and drying cycles during warmer periods, as this promotes germination of the sclerotia, and try to minimise inter-row cultivation, which pushes soil up around the base of plants. The fungus is a very effective saprophyte of cotton trash, so allowing time for cotton trash to break down prior to planting will reduce the activity of the fungus. Similarly, trash from other crops such as barley and millet are attractive substrates for the fungus.

9.11 Fungal disease control

9.11.1 When to spray

Sprays will control fungal disease, but when and how often to spray will depend on the varietal resistance, amount of infection, the impending weather conditions and the potential yield of the pulse crop.

Fungal disease control is geared around protection rather than cure. The first fungicide spray must be applied as early as necessary to minimise the spread of the disease. Additional sprays are required if the weather conditions favour the disease.

9.11.2 Principles of spraying

A fungicide spray at the commencement of flowering protects early pod-set. Additional protection may be needed in longer growing seasons until the end of flowering. Fungicides last around 2–3 weeks.

Remember all new growth after spraying is unprotected. Coverage and canopy penetration is critical, as only treated foliage will be protected. Translocation is very low in most products.

In periods of rapid growth and intense rain (50 mm over several days), the protection period will reduce to ~10 days.

Timing of fungicide sprays is critical (Table 12 and 13). As Ascochyta blight and BGM can spread rapidly, DO NOT DELAY spraying. A spray in advance of a rainy period is most desirable.

Despite some fungicide washing off, the disease will be controlled. Delaying until after a rainy period will decrease the effectiveness of the fungicide as the disease has started to spread.

Repeat fungicide sprays depend on:

- amount of unprotected growth
- rainfall since spraying
- likelihood of a further extended rainy period

Unprotected crops can lose >50% in yield. In severe cases, the crop may drop all of its leaves.  

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Table 12: Principles of when to spray for fungal disease control in chickpea.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Occurrence</th>
<th>When to spray</th>
</tr>
</thead>
</table>
| Ascochyta blight       | First appears under wet conditions              | Resistant variety. Fungicide sprays are unlikely to be required before podding. Despite good foliar resistance to Ascochyta blight, the flowers and pods of resistant varieties can be infected which can result in poor quality, discoloured seed or seed abortion and, in extreme situations, yield loss.
|                        |                                                 | Moderately resistant variety. In most seasons, disease development will be slow and there will be no or minimal yield loss. In such seasons there is no cost benefit in applying a fungicide during the vegetative stage. Despite good foliar resistance to Ascochyta blight, the flowers and pods of MR/R rated varieties can be infected, which can result in poor quality, discoloured seed or seed abortion and yield loss in severe situations. However, under high disease pressure, a reactive foliar fungicide strategy may be warranted during the vegetative period of the crop. If Ascochyta blight is present in the crop, apply a registered fungicide at early podding prior to rain to ensure pods are protected, and high quality, disease-free seed is produced.
|                        |                                                 | Susceptible variety. If the season favours Ascochyta blight, regular fungicide sprays will be needed from emergence until 4 weeks before maturity. Do not wait until you find the disease. Timing of the first two sprays is critical, because control is difficult or impossible after the disease has taken hold. The first spray must be applied before the first post-emergent rain event, or 3 weeks after emergence or at the 3-leaf stage, whichever occurs first. The second spray should be applied 3 weeks after the first spray. However, apply the second spray if 2 weeks have elapsed since the first spray and rain is forecast.
|                        |                                                 | Continue to monitor the crop 10–14 days after each rain event. If Ascochyta blight is found, additional sprays will be required. If it has been ≥2 weeks since the last application, spray again just before the next rain event.
|                        |                                                 | For all varieties regardless of resistance. If Ascochyta blight is detected, apply a registered fungicide at early podding prior to rain. In high-rainfall or high-risk situations and where there is an extended pod-filling period, further applications may be required.
| Botrytis grey mould    | Develops during warm (15–20°C), humid (>70%) conditions, usually at flowering | During early to mid-flowering as a protective spray. Additional sprays may be necessary through flowering and pod-filling if disease progresses. Disease is favoured by warm weather (15–20°C) and high humidity (>70% RH).

Table 13: Carryover of major pulse diseases, showing relative importance as sources of infection. 64

<table>
<thead>
<tr>
<th>Disease</th>
<th>Stubble</th>
<th>Seed</th>
<th>Soil</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ascochyta blight</td>
<td>***</td>
<td>**</td>
<td>*</td>
</tr>
<tr>
<td>Botrytis grey mould</td>
<td>***</td>
<td>***</td>
<td>*</td>
</tr>
<tr>
<td>Phytophthora root rot</td>
<td></td>
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<td>***</td>
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<tr>
<td>Sclerotinia</td>
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</tbody>
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9.12 Viruses

Key points:
- Chickpea is distinct from other pulses in respect to virus diseases and how viruses spread in crops.
- Aphicide sprays and some other control strategies that are effective in other pulses are less effective.
- At present, the best control options for chickpea are the current best agronomic practices: retaining standing stubble, using optimal sowing rates and times, and controlling in-crop and fallow weeds. 65
- Virus management aims at prevention through integrated management practice that involves controlling the virus source, aphid populations and virus transmission into pulse crops.
- Rotate legume crops with cereals to reduce virus and vector sources and where possible avoid close proximity to perennial pastures (e.g. lucerne) or other crops that host viruses and aphid vectors.
- Eliminate summer weeds and self-sown pulses ‘green bridge’ that are a host for viruses and a refuge for aphids.
- Aphid activity is influenced by seasonal conditions and will require early monitoring in nearby crops and pastures and possible use of an aphicide or cultural controls to reduce numbers.
- Sow directly into cereal stubbles (preferably standing), and encourage rapid canopy cover through early planting, high planting density as bare soil is more attractive to some aphid species.
- Purchase virus-tested seed or have farmer seed virus tested as PSbMV, CMV, BYMV and AMV depend largely on seed transmissions for survival.
- Gaucho® 350SD is now registered and when applied as seed treatment will help protect faba bean, field pea and lentil seedlings from early season aphid attack and reduce virus spread.

Viruses differ from most fungal diseases in that they infect plants systematically and no curative treatment is available. Virus infections are spasmodic and levels depend heavily on seasonal conditions and differ greatly between years and locations. Early infection can lead to stunting, reduced tillering and plant death and losses can be high. Late infections have less impact, but can still affect seed quality. 66

There are more than 14 species of virus that naturally infect chickpeas. These viruses are spread by airborne insects, with aphids being the predominant vector. The occurrence of virus in chickpeas is episodic and changes dramatically from season to season and location. Clovers, medics, canola/mustard, weeds and other pulses can host viruses that infect chickpea. The best control strategies to reduce risk of viruses are agronomic. These include retaining cereal stubble, sowing on time, establishing a

uniform closed canopy and controlling weeds. Seed and foliar insecticides are not recommended for chickpea viruses.

9.12.1 Symptoms

Viruses exhibit a varied range of symptoms and severity from relatively unapparent to plant death. The intensity and symptoms depend on virus and pulse species and to a lesser extent on virus strain, pulse variety, climatic conditions and plant stage at infection. Plants infected at an early stage or through seed will usually show more uniform discolouration and stunting, but when infected at the later stage will usually occur at the leaf tip before the whole plant starts to deteriorate (Photo 22).

Photo 22: Kabuli chickpea (centre) with low plant stand and high virus infection compared to kabuli (right) and desi (left) with good canopy.

Foliage symptoms are often more visible on young leaves and can include yellowing (sometimes reddening), vein clearing, leaf mottle, leaf distortion, curling of leaves, reduced size, chlorotic or necrotic spotting, or more widespread necrosis (Photo 23). Shoot symptoms may be seen as bunching of young leaves, growth of auxiliary shoots, bending over of the growing point, tip or apical necrosis, streaking of stems, stunting and wilting or plant death.
Symptoms such as leaf yellowing, veining, mottling, and wilting can often be confused with nutrient deficiencies, herbicide damage or water stress unless sufficiently distinct. It is also difficult to tell which virus is present without resorting to laboratory tests on plant samples.

It is best to collect living tissue samples and collection and packaging of fresh samples is simple. Instructions from local agronomists or Pulse Australia need to be heeded. Immediately place the sample with paper towelling into a plastic bag, seal it and refrigerate it until dispatched. Send the sample by priority post and do not leave it sitting around.

9.12.2 Conditions favouring development

High levels of virus infections have occurred in recent years resulting from infected plants in the previous spring as a virus source and a ‘green bridge’ of summer plant material to carry over these viruses and as a refuge for aphids. Warm dry conditions during autumn have favoured increased aphid activity and virus transmission.

Some aphid species prefer to land on plants surrounded by bare ground and favour thin crop stands or areas within the crop which have low plant densities.

Stressed plants are also more attractive to aphids, possibly due to a higher level of plant sugars, and are vulnerable to colonisation and can become a source of virus spread. Environmental factors that impacted on chickpeas in 2009 were extremely dry conditions early in the season that favoured aphid build and this was particularly evident in vetch crops. Then followed cold and wet conditions that included some transient waterlogging that stressed plants making them more venerable to root diseases and aphid attack.

Chickpea that border lentil, canola or lucerne crops can be subjected to larger numbers of aphids, as they can readily colonise these crops and multiply quickly.


Photo 23: Wilting and necrosis of shoot tip in desi chickpea cv. Amethyst infected naturally with Tomato spotted wilt virus (TSWV).
Controlling aphids in these nearby host crops can potentially decrease aphid numbers moving through chickpea crops.

**Types of transmission**

Pulse viruses are transmitted either in a persistent or non-persistent manner by insects (mostly aphids). The mode of transmission has implications for the way a virus develops in the field and its management.

**Persistently transmitted viruses:**

- Bean leafroll virus (BLRV)
- Beet western yellows virus (BWYV) (Photo 24)
- Subterranean clover red leaf virus (SCRLV)
- Subterranean clover stunt virus (SCSV)  

![Photo 24: Beet Western Yellows Virus in kabuli (top) and in desi (bottom).](image)

Source: CropPro.

The general symptoms of BLRV on pulses are interveinal chlorosis, yellowing, stunting and leaf rolling (Photo 25). These symptoms could easily be confused with subterranean clover stunt virus (SCSV) or other luteoviruses such as beet western yellows virus (BWYV) and subterranean clover red leaf virus (SCRLV) or nutrient stress symptoms.  


Persistent transmission means that once the insect becomes infectious, it remains so for the rest of its life. After an insect vector feeds on an infected plant, the virus has to pass through its body and lodge in the salivary glands before it can be transmitted to healthy plants. Not all aphid species are vectors of this kind of virus in pulses so the identification of aphid species is very important.

BWYV is the main virus and most common occurring in chickpea and lentil crops. It has a diverse natural host range including canola, pasture plants, lucerne and many weeds such as paddy melons, wild radish and some native legumes. BLRV is another but is limited to fabaceae (faba bean, field pea, chickpea, and lentil), lucerne, clovers and summer legumes.

Persistently transmitted viruses typically start with a random distribution of infected plants in autumn and increase during the season as vectors colonise the crop. Transmission rates can dramatically increase with large aphid flights that will often coincide with aphid activity and build-up prior to sowing.

Non-persistently transmitted viruses:
- Alfalfa mosaic virus (AMV)
- Bean yellow mosaic virus (BYMV)
- Cucumber mosaic virus (CMV)
- Pea seed-borne mosaic virus (PSbMV)

Non-persistently transmitted viruses can be seed-borne (depending on the virus/crop combination), but require aphid vectors to spread during the season. 73

9.12.3 Reducing risk of viral diseases

Controlling virus disease in chickpeas is difficult. Chickpea plants that become infected with a virus invariably die. GRDC-funded field trials have shown no benefit of seed-applied insecticides or regular foliar-applied insecticides or a combination of both against chickpea viruses. The best and at this stage only, control strategies to reduce risk of viruses in chickpeas are agronomic. These include; retaining cereal stubble, sowing on time, establishing a uniform closed canopy, providing adequate nutrition and controlling weeds. Reduce risk of viruses in chickpea crops by planting between rows of standing cereal stubble, sowing on time and targeting at least 25 plants/m². 74

9.12.4 Management of viruses

A virus management strategy to reduce the risk of infection may require a number of control measures relevant to the various virus and pulse types.

Better agronomy – better chickpeas

Field trials from 2012 and 2013 have shown that chickpea crops are at risk of increased damage from viruses when plant density is <20 plants/m². Significantly fewer plants are infected when plant densities are higher, and it is recommended to aim for >25 plants/m².

Trial crops deficient in nitrogen, potassium, phosphorus or all three have been shown to have significantly more virus-affected plants than a crop with adequate nutrition.

Inter-row planting into standing wheat stubble significantly reduced virus incidence in small trial plots of PBA HatTrick compared with the same amount of stubble slashed low to the ground. The mechanism for this difference is unclear, but these results are in agreement with many field observations in large crops during virus outbreaks.

Although differences in virus resistance have been observed for different varieties, further screening is needed to strengthen confidence in these results under high disease pressure in different growing regions, and to identify for which virus species resistance is effective. Under low virus pressure in field trials, some of the better performing varieties included FLIPPER and PBA HatTrick, although both these varieties have been observed with high rates of infection under high disease pressure. The variety Gully is very susceptible to Ascochyta blight, but has moderate virus resistance so may be useful for breeding resistance into future varieties.

While a link could not be confirmed in the 2013 season between BWYV infections in canola and subsequent spread into nearby chickpea crops, the sometimes high incidence of BWYV in canola indicates it may be prudent to avoid planting chickpea and other pulse crops next to canola.

Best agronomic management can help to reduce damage by viruses and includes:

- Retaining standing stubble, which can deter migrant aphids from landing. Where possible, use precision agriculture to plant between stubble rows. This favours a uniform canopy, which makes the crop less attractive to aphids.
- Planting on time and at the optimal seeding rate. These practices result in early canopy closure, which reduces aphid attraction (Figure 10).
- Ensuring adequate plant nutrition.
- Controlling in-crop, fence line and fallow weeds. This removes in-crop and nearby sources of vectors and virus.
- Avoiding planting adjacent to lucerne stands. Lucerne is a perennial host on which legume aphids and viruses, especially AMV and BLRV, survive and increase.
- Seed treatment with insecticides, e.g. imidacloprid, is not effective for non-persistently transmitted viruses but may be effective for luteoviruses. Unfortunately, local data supporting seed treatment are lacking.
- Given the high incidence of BWYV sometimes found in canola, consider growing chickpeas (and other pulse crops) away from canola.

References:

**Row spacing and incidence of plants with virus symptoms**

Row spacing had a significant effect on incidence of plants with virus symptoms in a 2013 trial. On 11 October 2013, there were more than twice as many symptomatic plants/m² in plots with 40 cm rows compared to those with 80 cm rows (Figure 11). Both row configurations were sown at 30 plants/m² so plant density per unit area cannot account for the difference. Rather, plant density within each row appears to be responsible (12 plants/m row @ 40 cm and 24 pl/m row @ 80 cm).

**Stubble management and incidence of plants with virus symptoms**

Planting into standing cereal stubble is known to help reduce risk of virus in lupin crops. Retaining standing winter or summer cereal is believed to be useful in reducing risk of virus in chickpea crops, though research providing such evidence is limited.

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Two trials were conducted in 2013 to compare standing versus flat (slashed) wheat stubble on incidence of plants with virus symptoms. One trial was sown at 80 cm row spacing; the other at 40 cm spacing; both were sown with PBA HatTrick chickpea at 30 plants/m². The 80 cm trial was assessed on 11 October and the 40 cm trial was assessed on 9 October and again on 16 October. In both trials, incidence of plants with virus symptoms was lower where the chickpeas had been sown into standing stubble (Figure 12). Individual plots in these trials were small, 2 m × 10 m for the 80 cm trial and 4 m × 10 m in the 40 cm trial. 79

![Figure 12: Effect of stubble management (flat v. standing) on incidence of chickpea plants with virus symptoms. Source: GRDC](https://grdc.com.au/)

**Non-persistently transmitted viruses (e.g. CMV, BYMV, AMV, and PSbMV)**

The initial and main source of infection is contaminated seed, with further transmission in-crop by aphids.

Management steps include:

- Sourcing healthy seed that has been tested free of CMV, BYMV, AMV and PSbMV virus. Tested seed should have less than 0.1% virus infection and field peas should have less than 0.5% for PSbMV.
- Farmer-retained seed should only come from crops with no visible virus symptoms and seed testing should be a priority.
- Some cultivars have virus resistance such as CMV in many new lupin varieties and in JENABILLUP (available in 2011). Yarrum field pea has resistance to BLRV and PSbMV. Increased emphasis on virus resistance is a priority of Pulse Breeding Australia.
- Controlling aphids in-crop is not an effective means of controlling non-persistently transmitted types of viruses.
- Sowing direct into retained cereal stubble and preferably standing as some aphid species are attracted to bare earth. This has been effective in minimising CMV spread in lupins.

**Persistently transmitted viruses (e.g. BLRV, BWYV, SCSV)**

These viruses are not seed-borne, and the virus is transmitted from live infected plants to healthy plants primarily by aphids or other insect vectors.

An integrated management strategy involves the use of both cultural and chemical measures that aim to eliminate any virus sources, minimise aphids and deter aphids from entering the crop. Often by the time aphids are detected, the virus spread has already occurred.

Management steps include:

- Minimising the ‘green bridge’ for virus and aphid survival over summer. Control volunteer pulses, legumes and weeds well before sowing and early crop weeds that may carry viruses and aphids.
- Minimising bare earth through sowing into previous cereal stubbles and early sowing with adequate plant population (germ and vigour test seed). Use narrow rows in the absence of stubble to minimise exposed bare soil to deter aphids entering the crop.
- Avoiding crop stress through good paddock selection (soil type, no hard pan, low weed burden) adequate nutrition, no herbicide stresses and good inoculation.
- Avoiding sowing pulses close to each other and broadleaf crops such as canola, and being aware of proximity to perennials (e.g. lucerne).
- Monitoring crops and neighbouring areas using a sweep net or beat sheet. Yellow sticky traps on crop perimeters can also be a handy check for aphid presence. Identify the species present and be prepared to use a ‘soft’ insecticide such as pirimicarb if there is a chance of localised flights.
- Use of ‘soft’ insecticides soon after emergence has been shown to help control persistently transmitted viruses only. Use of an SP is controversial as while it prevents early colonisation due to ‘anti feed’ properties, it can also agitate aphids not controlled and increase virus spread. It should not be used when GPA is present as this major vector for BWYV has resistant populations. Impact on natural beneficials could also lead to higher aphid build-up.  

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