

Serdc[™] GROWNOTES[™]



CHICKPEA SECTION 9

DISEASES

THE DISEASES | FUNGAL DISEASE MANAGEMENT STRATEGIES | INTEGRATED DISEASE MANAGEMENT | RISK ASSESSMENT | ASCOCHYTA BLIGHT | BOTRYTIS GREY MOULD | PHYTOPHTHORA ROOT ROT | SCLEROTINIA STEM AND CROWN ROT | ROOT ROTS INCLUDING DAMPING OFF (FUSARIUM, RHIZOCTONIA AND PYTHIUM SPP.) | COLLAR ROT (SCLEROTIUM ROLFSII) | VIRUS MANAGEMENT | FUNGAL DISEASE CONTROL | REGISTERED FUNGICIDES



October 2016

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SECTION 9 Diseases

A single disease management strategy rarely provides complete disease control. Using a number of integrated disease management techniques (IDM) is more likely to control diseases.

Controlling the major fungal diseases of chickpeas in the northern region requires an integrated approach to disease management and prevention.¹

9.1 The diseases

Ascochyta blight. The pathogen survives and spreads in infected seed, stubble and on volunteers; chickpeas are the only known host in Australia. Fruiting bodies (pycnidia) of *Phoma rabiei* (also known as *Ascochyta rabiei*) develop on infected plant tissue, and spores, which ooze from wet pycnidia, are spread short distances and cause new infections. Under ideal conditions, Ascochyta blight can reproduce as fast as 5–7 days.

Botrytis grey mould (BGM). Similar to the Ascochyta blight pathogen, the BGM pathogen (*Botrytis cinerea*) can survive and spread in infected seed and stubble, and some strains produce dark, hard sclerotia, which also aid survival and spread. However, the BGM pathogen has a very wide host range, and is able to colonise dead and dying tissue of virtually any plant. Huge numbers of spores are produced on BGM lesions and are spread on air currents. BGM can also cycle in 5–7 days.

Phytophthora root rot (PRR). *Phytophthora medicaginis* survives as thick-walled oospores, which develop in infected roots of chickpea and other plants including lucerne and annual medics. When the soil is saturated with moisture, the oospores germinate to produce zoospores, which swim to and infect chickpea roots. The pathogen is spread by movement of infected soil and water.

Sclerotinia rot. Both *Sclerotinia* species (*S. sclerotiorum* and *S. minor*) survive as hard black sclerotia, in soil or mixed with seed. Both species have a very wide host range, including many weeds and most broadleaf crops. Infection of chickpea plants occurs directly at the crowns (both species) or from airborne spores produced in fruiting bodies on germinated sclerotia (*S. sclerotiorum*) (Table 1).²

GRDC (2013) Chickpea disease management. GRDC Resources Fact Sheet. May 2013, <u>http://www.grdc.</u> com.au/Resources/Factsheets/2013/05/Chickpea-disease-management

K Moore, M Ryley, T Knights, P Nash, G. Chiplin, G Cumming (2011) Chickpeas—varietal selection, paddock planning and disease management in 2011. Northern region (Goondiwindi). GRDC Update Papers April 2011



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Table 1: Key facts about the biology of major chickpea diseases

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

Some of the disease terms that are useful to know when diagnosing chickpea diseases are pictured in Figure 1.

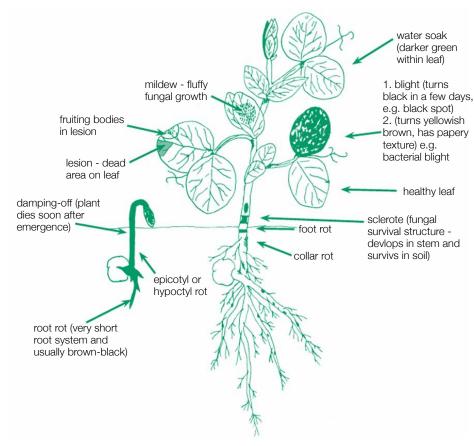


Figure 1: Chickpea disease diagnosis terms. (Source: Grain Legume Handbook).

Fungal disease management strategies 9.2

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 selfsown 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 Ascochyta 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

http://www.grdc. com.au/Resources/ Factsheets/2013/05/ Chickpea-diseasemanagement

M Fuhlbohm, M English, M Ryley, N Gannon, M Cahill (2004) Chickpea disorders: The Ute Guide. Department of Primary Industries and Fisheries, Queensland, and Grains Research and Development Corporation. http://www.grdc.com. au/GRDC-UteGuide-**ChickpeaDisorders**

http://grdc.com. au/Research-and-**Development**/ DAN00143

K Moore, L Jenkins, P Nash, G Chiplin, <u>S Bithell (2016),</u> Chickpeas: What we learnt in 2015 and recommendations for <u>2016</u>

P Matthews, D McCaffery, L Jenkins variety sowing guide 2016 p85

K Moore, K Hobson, A Rehman (2014), Chickpea varietal purity and implications for disease management p138

K Moore, K Hobson, S Bithell (2016), Is chickpea on chickpea worth it?



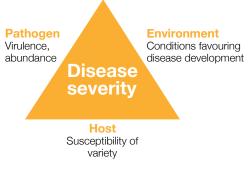
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critical points in disease development, and all can be represented by the disease triangle (Figure 2). Diseases such as Ascochyta blight and PRR rot can cause total crop failures very quickly, whereas the effects of BGM and root-lesion nematodes on crop performance and yield may unfold more slowly.





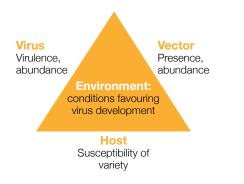


Figure 3: The disease triangle (Agrios 1988).

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 4 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.



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More information

http://www.nga. org.au/resultsand-publications/ download/137/ grdc-updatepapers-diseases/ botrytis-seedlingblight-in-chickpeas/ grdc-adviser-updatepaper-goondiwindi -march-2012.pdf

APVMA (2015), Permit to allow minor use of an agvet chemical product for the control of various fungal diseases in chickpeas and lentils

1 More information

http://www.grdc. com.au/Researchand-Development/ GRDC-Update-Papers/2013/02/ Developing-a-plan-forchickpeas-2013

http://www.grdc.com. au/Media-Centre/Media-News/North/2013/06/ Top-five-chickpeachallenges

Pulse Australia (2015), Chickpea: integrated disease management

Clean seed and care the recipe for chickpea success (2013)



- **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.
 - **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. Usually the virus spread has occurred by the time 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.³

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.

Paddock selection based on PRR is the first priority, followed by cropping history.

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.⁴

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

Integrated disease management 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:

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³ Pulse Australia (2013) Northern chickpea best management practices training course manual-2013. Pulse Australia Limited.

⁴ Pulse Australia (2011) Chickpea Integrated Disease Managment. <u>http://www.pulseaus.com.au/growing-pulses/bmp/chickpea/idm-strategies</u>



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- 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 (e.g. removal of diseased plants to prevent spread of infection). 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, for example, 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 life cycle.
- 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.

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.



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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.
- 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* and *Phytophthora medicaginis*.

- 1. Rotation
- Develop a rotation of no more than 1 year of chickpea in 4 years.
- Plant chickpea into standing stubble of previous cereal or sorghum stubble 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 and Phytophthora medicaginis.
- Ascochyta rabiei is chickpea-specific, whereas Botrytis cinerea has a wide host range including sunflower, bean, pea, and weeds (e.g. Euphorbia spp., groundsel and emu-foot).
- Lucerne, medics and chickpea are hosts for *Phytophthora medicaginis*, and *Phoma medicaginis* var. *pinodella* can be hosted by lucerne, clover, field pea, lupin and chickpea as well as *Phaseolus* spp.
- 2. History of chickpea diseases
- Previous occurrence of soil-borne diseases (PRR, Sclerotinia stem rot or *Pratylenchus* nematodes) constitutes a risk for subsequent chickpea crops for up to 10 years.



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http://www.dpi.nsw. gov.au/ data/assets/ pdf_file/0003/431274/ Moore-Chickpea-2011-Crops-and-Recommendations.pdf • At least 500 m from the previous year's chickpea crop.

3. Weeds

- Realise that 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 or sulfonylurea 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 2 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 about 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

9.4.4 Foliar fungicides

Foliar fungicides are essential for the management of Ascochyta blight in all varieties, and are an important tool for the management of BGM. Varieties with higher levels of



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Ascochyta blight resistance do not require as many sprays as susceptible varieties. The success of foliar fungicides depends on timeliness of spraying (hence the importance of regular crop monitoring), appropriate fungicide selection, and correct application (Table 2). Early detection and fungicide application is vital.

Table 2: Foliar fungicides for the control of Ascochyta blight and Botrytis grey mould in chickpea V Registered product label claim

Active ingredient:	Carbendazim	Chlorothalonil	Mancozeb
Example trade name:	Spin Flo®	Barrack [®] /Unite ^{® A}	Dithane [®] Rainshield
Ascochyta blight		\checkmark	\checkmark
Botrytis grey mould	\checkmark		\checkmark
Damping-off (Kabulis)			
Phoma root rot			
Phytophthora root rot			
Jurisdiction	All states	All states	All states

^AThese are the only registered chlorothalonil products. It is an offence to use any other product.

Refer to the current product label for complete 'Directions For Use' prior to application.

Prior to the use of any crop protection product, ensure that it is currently registered or that a current permit exists for its use in chickpeas.

9.5 Ascochyta blight

9.5.1 Background

Ascochyta blight, caused by the fungus *Ascochyta rabiei* (also known as *Phoma rabiei*), is a serious disease of chickpeas in Australia. The fungus can infect all aboveground parts of the plant and is most prevalent in areas where cool, cloudy and humid weather occurs during the crop season.

Ascochyta blight first caused widespread damage to chickpeas in Australia in 1998 when extremely wet conditions favoured disease development and spread. Ascochyta blight is now considered endemic in all growing regions of Australia. Unlike some insect-control strategies, there is no economic threshold for Ascochyta blight. Management strategies are aimed at preventing the occurrence of disease and limiting its spread.

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.

9.5.2 Economic importance

This disease is very serious, as it has caused severe damage and losses in chickpeas. In the very wet winter of 2010, many crops in north-west New South Wales (NSW) were wiped out completely by Ascochyta blight.

Biology and epidemiology

Ascochyta blight causes economic losses only on chickpea. There are no other known hosts of the pathogen in Australia, but different *Ascochyta* species infect faba beans, lentils and field peas. The pathogen survives between seasons on infected plant residues, on infected or contaminated seed and on infected volunteer chickpea plants (Figure 4).



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1 More information

Registered labels and current permits can be found on the APVMA website: <u>www.apvma.</u> gov.au.

http://www.grdc. com.au/Researchand-Development/ GRDC-Update-Papers/2014/03/ Chickpea-varietiesselecting-horses-forcourses



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K Moore, M Ryley, G Cumming, L Jenkins (2015), Chickpea: Ascochyta blight management

<u>K Moore, K Hobson,</u> <u>S Harden, P Nash, G</u> Chiplin, S Bithell (2015), Effect of chickpea Ascochyta on yield of current varieties and advanced breeding lines <u>– Tamworth 2014 p89</u>

<u>K Moore, K Hobson,</u> <u>S Harden, P Nash, G</u> <u>Chiplin, S Bithell (2015),</u> <u>Chickpea ascochyta -</u> <u>evidence that varieties</u> <u>do differ in susceptibility</u> <u>of pods</u>

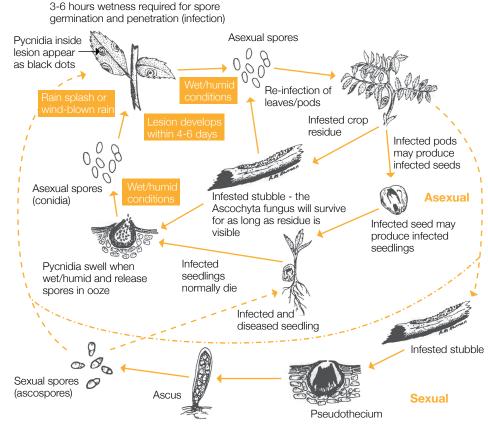


Ascochyta blight-infected stubble blown about during and after harvest is a major cause of short-medium-distance dispersal (metres to kilometres) along with movement of infected trash by water, machinery or animals. Spores of the fungus can survive a short time on skin, clothing and machinery.

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 3 years in the paddock.

Ascochyta blight can develop over a wide range of temperatures (5–30°C) and needs only 3 h of leaf wetness to infect. 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.



Drawings by R.M. Hannan (Can. J. Pl. Path. 19:215-224, 1997)

Figure 4: Life cycle of Ascochyta blight pathogen. Note: Only the asexual phase is known to occur in Australia at this time.

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9.5.3 Symptoms

Ascochyta blight infects the leaves, stems and pods of chickpea plants, causing tan/ brown, rounded lesions on affected plant parts.

Symptoms become visible in 7–10 days as a pale green–yellow discoloration on leaves, often referred to as 'ghosting' (Figure 5).

Toward the centre of the lesion, small, black fruiting bodies called pycnidia develop in 10–14 days, often in concentric rings (Figure 6). Spores ooze out of pycnidia and are spread by rain-splash upwards within the plant and sideways to nearby healthy plants.

Lesions often girdle the stems of the plant, causing them to weaken and subsequently break off, making later detection difficult (Figure 7). Circular 'hot spots' or 'foci' consisting of plants with severe infection can appear in crops, but by this stage considerable damage has occurred. Seeds can become infected after lesions develop on pods (Figure 8).



Figure 5: Ascochyta blight: leaf ghosting may appear 7–10 days after infection following rainfall or heavy dew.



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Figure 6: Note the concentric circles of brown–black dots in the centre of the lesions. These are the pycnidia or fruiting bodies, which are unique to Ascochyta blight.



Figure 7: 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.



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Figure 8: Pod lesions are similar in appearance to leaf lesion. They lead to infection of the seed. Do not keep planting seed from any crop that has been identified as having Ascochyta blight. (Photos: G. Cumming, Pulse Australia)

9.5.4 Management options

Follow the principles of IDM, which include:

- crop rotation and paddock selection
- reducing proximity to previous season chickpea stubble
- growing resistant varieties
- using clean seed and fungicide seed dressings
- regular crop monitoring
- strict hygiene on and off farm
- strategic use of foliar fungicides

Note: Chickpea seed dressings protect only 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*.

Differing spray programs have been developed based on each variety's Ascochyta blight rating.

Chickpea Ascochyta blight 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 blight spray program is regular monitoring combined with timely application of registered fungicides.

Resistant (R): Genesis[™] 090, Genesis[™] 425

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.

Monitor the crop 10–14 days after each rain event.

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.



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Moderately resistant (MR): PBA HatTrick⁽, PBA Boundary⁽), Genesis[™] 114

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.

Monitor the crop 10–14 days after each rain event.

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 (S): Jimbour^{(b}, Kyabra^{(b}, PBA Pistol^{(b})

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 postemergent 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.

Mancozeb is often the preferred fungicide for these first two applications because it can be applied with a Group A grass herbicide.

Continue monitoring 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 or longer since the last application, spray again just before the next rain event.

A fungicide program

A fungicide program needs to account for several factors.

Disease risk categories

Based on:

- varietal susceptibility or resistance
- source of seed and treatment of seed
- planting proximity to chickpea crops of previous seasons
- level of Ascochyta inoculum present from crop residue or volunteer plants
- climatic conditions in relation to disease infection

Registration status

The product must be registered or have a permit for the disease and use.

Withholding period

All products and timings used in the fungicide program must meet Australian withholding periods and export slaughter intervals to satisfy overseas markets.

Fungicide resistance management

The maximum number of sprays of a product must be adhered to, in order to minimise the risk of fungicide resistance developing.

Mode of action

Using products with a range of mode of actions for control of diseases further reduces the chance of fungicide resistance development and improves efficacy. Fungicides are



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M Ryley, K Moore, G

Cumming, L Jenkins

Managing Botrytis grey

(2015), Chickpea:

mould

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also recommended at times of the disease life cycle where they will be most effective according to their mode of action.

Early harvest

Harvest at maturity to minimise *Ascochyta* seed infection and potential down grading. Seed damage from Ascochyta blight is usually more severe when crops are harvested late. Moisture content allowable on delivery is 14%. Harvest losses, seed splitting and downgrading in quality can be substantial if chickpea is harvested at below 12% moisture.

9.6 Botrytis grey mould

9.6.1 Background

Botrytis grey mould in chickpea is caused by the fungus *Botrytis cinerea*, 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; however, *B. cinerea* does not infect cereals or grasses.

Botrytis cinerea has been recorded on over 138 genera of plants in 70 families. Legumes and asteraceous plants comprise about 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 blight, which is more dependent on inoculum, at least in the early phases of an epidemic.

Botrytis cinerea also causes pre- and post-emergent seedling death. This happens when chickpea seed, infected during a BGM outbreak, is used for sowing. This seedling disease does not need the wet conditions that favour BGM.

9.6.2 Economic importance

Botrytis grey mould 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.

9.6.3 Biology and epidemiology

Botrytis cinerea produces diffuse, white fungal growth, which later turns grey due to the production of huge numbers of spores borne in clusters at the ends of dark stalks.

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.

Botrytis grey mould is favoured by moderate temperatures (20–25°C) and frequent rainfall events. It does not become a risk until the average daily temperature is \geq 15°C. 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. ciner*ea.

The pathogen can survive on and in infected seeds, in infected stubble, on alternative hosts, in dead plant tissue and as sclerotia. The relative importance of these in Australia is unknown, but recent research in Victoria demonstrated that *B. cinerea* can survive



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for up to 18 months on infected stubble under field conditions. Other research from Western Australia suggests that sclerotia of *B. cinerea* may not be able to survive over summer because they lose their viability during hot weather.

Irrespective of its mode(s) of survival, the experience of the 2010 chickpea crop indicates that under conducive conditions, BGM can develop rapidly not only within crops, but also across districts and regions.

9.6.4 Symptoms

Often, the first symptom of BGM infection in a crop is drooping of the terminal branches. If groups of plants are infected, these may appear as yellow patches in the crop (Figure 9).

The diagnostic feature is a grey 'fuzz' (Figure 10), which under high humidity, develops on flowers (Figure 11), pods (Figure 12), stems and on dead leaves and petioles. Lesions can develop anywhere along the stem, but are usually first found on the lower part of the stems often starting in leaf axils.

Infected seeds are usually smaller than normal and are often covered with white to grey fungal growth (Figure 13).



Figure 9: If groups of chickpea plants are infected these may appear as yellow patches in a crop.



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Figure 10: The diagnostic feature for Botrytis grey mould is a grey 'fuzz', which develops under high humidity.



Figure 11: Botrytis grey mould on chickpea flowers.



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Figure 12: Botrytis grey mould on pods.



Figure 13: Botrytis grey mould on seed. (Photos: G. Cumming, 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. Small, dark brown–black resting bodies (sclerotia) of *B. cinerea* may develop on infected dead tissue, and are capable of producing spores on their surface.

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 sclerotia develop.

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



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9.6.5 Management options

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 in the air 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.

Volunteer control: the 'green bridge'

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

This will also reduce carryover of the Ascochyta blight pathogen.

Seed source

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.

Seed fungicides (dressings)

Thiram-based fungicide seed dressings are effective in significantly reducing, but not eliminating, BGM from infected seed.

Paddock selection

Paddocks in which chickpeas were affected by BGM should not be re-sown to chickpea, faba bean or lentil the following season. Irrespective of disease, the paddock should not be re-sown to chickpeas for at least 3 years. Nor should chickpea be sown beside paddocks where BGM was an issue the previous season.

As is the case 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.

Sowing time and row spacing

If long-term weather forecasts suggest a wetter than normal year (La Niña), consider sowing in the later part of the suggested sowing window for your district and on wider rows (e.g. 100 cm); the latter results in increased air movement through the crop and reduced humidity within the canopy.

Varietal resistance

All current commercial varieties suitable for the southern and western regions are susceptible to BGM.

Foliar fungicides

In seasons and situations favourable to the disease, a preventative spray of a registered fungicide immediately prior to canopy closure, followed by another application 2 weeks later, will assist in minimising BGM development in most years. This is particularly important, as often the seasons favourable to BGM will result in large crop canopies. This makes penetration of foliar fungicides very poor once the canopy has closed over.



http://www.publish. csiro.au/paper/ AR06120.htm

http://www.dpi.nsw. gov.au/ data/assets/ pdf file/0004/431266/ Chickpea-seed-tests.



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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.

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, initial timely and thorough application is critical.

9.7 Phytophthora root rot

Phytophthora root rot is not a major chickpea disease in southern and Western Australia, but it is a major disease north of central NSW and Queensland.

9.7.1 Background

Phytophthora root rot is a disease of chickpea caused by the fungus-like oomycete *Phytophthora medicaginis*, which is widespread in the cracking clay soils of northern NSW and southern Queensland. It can cause significant yield losses (Figure 14) in wetter than normal seasons or following periods of soil saturation in normal seasons. Lucerne, perennial and annual medics (*Medicago* spp.), and other leguminous plants including sulla (*Hedysarum* spp.) and sesbania (*Sesbania* spp.) can also host *P. medicaginis*.



Figure 14: Cultivated areas were killed by Phytophthora. Only plants on top of contours survived. (Photo: M. Schwinghamer, NSW DPI)



K Moore, K Hobson, S Harden, G Chiplin, S Bithell, L Kelly, W Martin, K King (2016), Phytophthora in chickpea varieties HER15 trial –resistance and yield loss

<u>S Bithell, K Moore,</u> <u>K Hobson, S Haden,</u> <u>W Martin, A McKay</u> (2016), A new DNA tool to determine risk of chickpea Phytophthora root rot

<u>K Moore, M Ryley,</u> <u>M Schwinghamer, G</u> <u>Cumming, L Jenkins</u> (2015), Chickpea: <u>Managing Phytophthora</u> root rot

<u>K Moore, T Knights,</u> <u>S Harden, P Nash, G</u> <u>Chiplin, K Hobson,</u> <u>M Ryley, W Martin, K</u> <u>King (2014), Response</u> <u>of chickpea genotype</u> <u>to Phytophthora root</u> <u>rot (Phytophthora</u> <u>medicaginis) – Warwick</u> <u>Qld 2013 p136</u>

K Moore, L Kelly, M Ryley, K Hobson, T Knights, S Harden, W Martin, K King, P Nash, G Chiplin, S Bithell (2015) Phytophthora in chickpea varieties resistance rankings and yield loss.

K Moore, M Ryley, K Hobson, T Knights, S Harden, W Martin, K King, P Nash, G Chiplin (2014), Phytophthora tolerance in chickpea varieties.



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Figure 15: Phytophthora in a water-course. (Photo: M. Schwinghamer, NSW DPI) Phytophthora and waterlogging (where roots die from low oxygen levels) are 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 (Figure 15), or where the soil has been compacted or has hard pans.

However, under very wet conditions, entire paddocks can be affected.

9.7.2 Economic importance

Phytophthora root rot is a serious disease of chickpeas in southern Queensland and northern NSW.

Although no economic losses have been reported in the southern regions, it remains a potential threat in areas with lucerne, medics or heavy textured soils.

9.7.3 Biology and epidemiology

Phytophthora medicaginis survives in soil mainly as thick-walled oospores (Figure 16), 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.



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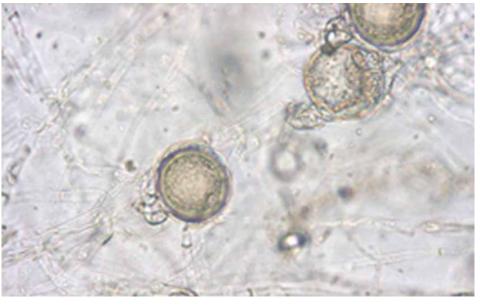


Figure 16: Phytophthora medicaginis oospores can survive in soil for up to 10 years. (Photo: G. Chiplin, NSW DPI)

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.

9.7.4 Symptoms

Infection by *P. medicaginis* can occur at any growth stage, causing seed decay, pre- and post-emergence damping off, loss of lower leaves (Figure 17), and yellowing, wilting and death of older plants.

Symptoms are sometimes delayed if temperatures are cool and the soil is moist. On young plants the lesions may extend up the stem for 10 mm or more above ground level (Figure 18). Lateral roots and tap root die (Figure 19) or dark brown/black lesions often girdle the taproots.



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Figure 17: Severely affected plants (left) have no lateral roots and defoliation below tips of stems. (Photo: J. Wessels, Qld Gov.)



Figure 18: Basal lesions extending up the plant stem. (Photo: M. Ryley, Qld Gov.)



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Figure 19: Phytophthora root rot-affected plant (right) with lateral and tap root death. (Photo: M. Ryley, Qld Gov.)



Figure 20: New roots forming from the top of the taproot (Phytophthora root rot). (Photo: M. Fuhlbohm, Qld Gov.)



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Plants with PRR 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 (Figure 20).

Symptoms of waterlogging can be confused with those of PRR (Table 3), but differ in the following ways:

- Plants are most susceptible to waterlogging at flowering and early pod-fill.
- Symptoms develop within 2 days of flooding, compared to at least 7 days for PRR.
- 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.

Table 3: Differences between Phytophthora root rot and waterlogging

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

9.7.5 Management options for PRR

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

There are no effective chemical sprays as there are for Ascochyta blight and BGM. Thus, PRR can only be managed by pre-sowing decisions and assessing risks for individual paddocks.

Development of the disease requires 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 not to sow chickpeas in high-risk paddocks, which are those with a history of:

- PRR noted in previous chickpea or lucerne crops
- lucerne or annual or perennial medics
- waterlogging or being flood-prone

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

Grow a chickpea variety with the highest level of resistance, particularly in mediumrisk situations where medic, chickpea or lucerne crops have been grown in the past 5–6 years. Current commercial varieties differ in their resistance to P. medicaginis, with Yorker and PBA HatTrick having the best resistance and are rated MR (historically Yorker has been slightly better than PBA HatTrick), while Jimbour is MS-MR, Flipper and Kyabra are MS and PBA Boundary has the lowest resistance (S). PBA Boundary should not be grown in paddocks with a history of PRR, lucerne, medics or other known hosts such as sulla.



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http://www.grdc.com. au/Media-Centre/ Ground-Cover/Ground-Cover-Issue-104-May-June-2013/Overcoming-Phytophthora-root-rotin-chickpeas

http://www.grdc. com.au/Researchand-Development/ <u>GRDC-Update-</u> Papers/2013/02/ Developing-a-plan-forchickpeas-2013 Although registered for use on chickpeas, metalaxyl seed treatment is expensive, does not provide season-long protection and is not recommended. ⁵

9.7.6 Management options for waterlogging

- Avoid poorly drained paddocks and those prone to waterlogging (Figure 21).
- Do not flood irrigate after podding has commenced especially if the crop has been stressed.
- A rule of thumb is that if the crop has started podding and the soil has cracked, do not irrigate.
- Overhead irrigation is less likely to result in waterlogging but consult your agronomist.



Figure 21: Waterlogged crop areas seen from the air.

9.8 Sclerotinia stem and crown rot

9.8.1 Background

Sclerotinia stem and crown rot of chickpea are caused by *Sclerotinia* spp. Three species of *Sclerotinia* are reported to cause the disease *S. sclerotiorum*, *S. minor* and *S. trifoliorum*. Of these, the most common is *S. Sclerotiorum*.

In 2010, Sclerotinia was more common than in previous years and in some paddocks caused serious damage, including 100% loss in one Kabuli crop near Dubbo (Figure 22).

⁵ K Moore, K Hobson, S Harden, G Chiplin, S Bithell, L Kelly, W Martin, K King (2016), Phytophthora in chickpea varieties HER15 trial –resistance and yield loss. <u>https://grdc.com.au/Research-and-Developme</u> GBDC-Lodate-Papers/2016/03/Phytophthora-in-chickpea-varieties-HER15-trial-Besistance-and-vield-Ir



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http://www.grdc. com.au/Researchand-Development/ GRDC-Update-Papers/2013/02/ Sclerotinia-stem-rot-Managing-the-diseasein-2013 Figure 22: Sclerotinia sclerotiorum killed these plants; eventually the whole crop was lost. (Photo: K. Moore, NSW DPI)

9.8.2 Economic importance

Sclerotinia can cause severe damage in chickpeas. This has occurred in Kabuli chickpeas in Victoria.

9.8.3 Biology and epidemiology

Among the three *Sclerotinia* spp., *S. sclerotiorum* has the widest reported host range. It infects >400 plant species, whereas *S. minor* causes diseases on crops in at least 53 plant genera and significant economic losses in peanut, sunflower and lettuce. *Sclerotinia trifoliorum* causes diseases on plants belonging to 21 plant genera and major economic losses in legumes, particularly forage legumes such as *Medicago* spp. and *Trifolium* spp.

All three species can survive in soil as sclerotia for 10–12 years without susceptible host plants. The crown can be infected by any of the three species, although usually one species dominates in a particular field.

The disease is favoured by cool, moist weather. Once established, the fungus can move rapidly to neighbouring healthy, tissue. A few days after infection, plants start to wither and die. The fungus is carried over to the next year in the infected plants. It is suspected, on the basis of optimal growth temperatures, that *S. trifoliorum* prefers cooler conditions than *S. sclerotiorum*.

9.8.4 Symptoms

Sclerotinia appears mainly on older plants. At first water-soaked patches (lesions) appear on the stems and leaves, and later affected areas develop a soft, slimy rot which exude droplets of brown liquid. The infected tissues dry out and they become covered with a fine white web of fungus growth. Small black spots, irregular in size and shape may sometimes be seen just below the surface, mingled with the white fungus growth.

Later on, stem lesions turn grey, the white growth disappears and the branch above the lesion dies. Affected plants wilt and die rapidly, without losing their leaves.



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A late infection can affect the pod and seeds. Infected seeds are smaller than normal and discoloured.

Sclerotinia sclerotiorum is the prevalent species in cooler, wetter regions, whereas *S. minor* is more common in warmer drier environments. Both species cause a basal stem rot when their sclerotia germinate in soil and infect the base of the plant.

Sclerotinia sclerotiorum (Figure 23) and S. trifoliorum produce large, irregular-shaped sclerotia 5–10 mm in diameter, as high up as 20–30 cm on the stem.



Figure 23: Sclerotia of S. sclerotiorum. (Photo: M. Ryley, Qld Gov.)



Figure 24: Sclerotia of S. minor. (Photo: K. Moore, NSW DPI) Sclerotinia minor (Figure 24) produces sclerotia that are angular and much smaller, rarely larger than 2–3 mm in diameter.



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In dense crops, during moist conditions, a white cottony fungal weft develops around the base of plants (Figure 25).



Figure 25: Fungal weft of Sclerotinia in the lower canopy. (Photo: K. Moore, NSW DPI) Under cool wet conditions, *S. sclerotiorum* sclerotia can germinate to produce small cup like structures (apothecia) at ground level (Figure 26).

These release air-borne ascospores that infect aboveground parts of the chickpea plant, often starting in leaf axils (Figure 27).

Stem tissues above and below the infection point initially remain green.



Figure 26: Apothecia are produced at ground level.



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Figure 27: Ascospore infection of chickpea stem by S. sclerotiorum. (Photo: G. Cumming, Pulse Australia)

9.8.5 Management options

Both *S. sclerotiorum* and *S. minor* have wide host ranges including many broadleaf weeds and crops such as canola, faba bean and sunflower.

Cotton and cereals are not hosts to either species.

Reduce the risk of losses from Sclerotinia rot by sowing seed free of sclerotia and by not sowing chickpea in paddocks that have had alternative host crops in the past 10 years, because the resting structures (sclerotia) can survive for that long.

It is acknowledged that 10 years, in most situations, is impractical, but do not sow chickpea in paddocks that had a broadleaf crop (other than cotton) last season.

The disease risk can be reduced by using disease-free seed. It is also important to avoid sowing chickpeas on areas where the disease is known to be present. If severe infection occurs, the area should be burnt and ploughed deeply to kill the fungus in the soil.

Crop rotation will reduce the risk of infection. Cereals, which are not a host, should be grown for several seasons before returning to chickpeas or other pulses or canola. Other hosts to *Sclerotinia* are the oilseed crops (e.g. canola, pulses and broadleaf weeds such as capeweed).

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

9.9.1 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 (Figure 28).



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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.

9.9.2 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.

9.9.3 Disease cycle

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.

9.9.4 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.



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Figure 28: Rhizoctonia root rot. Optimum soil temperature is 24–26°C; disease is worse on light sandy soils.



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9.10 Collar rot (Sclerotium rolfsii)

9.10.1 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, particularly in central Queensland, 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 (Figure 29); 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 (about 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.

9.10.2 Economic importance

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

9.10.3 Disease cycle

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 interrow 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.



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Figure 29: Webs of Sclerotium rolfsii mycelium at the base of an infected chickpea plant.

9.11 Virus management

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 aphids that fly in to crops do not stay long and do not normally colonise plants. Typical virus symptoms are bunching, reddening, yellowing, death of shoot tips and early death of whole plants. However, it should be remembered that none of these are diagnostic for viruses.

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 (Schwinghamer *et al.* 2009). Seed and foliar insecticides are not recommended for chickpea viruses. ⁶

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

Rotate pulse 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 that are a green bridge host for viruses and a refuge for aphids and their multiplication.

Aphids are the major means by which viruses enter chickpea crops. Winged aphids acquire viruses by feeding on alternative hosts (particularly lucerne) before landing on chickpeas. They feed briefly, thus transmitting viruses, and then fly on. Cucumber mosaic virus (CMV) and Alfalfa mosaic virus (AMV) are non-persistently transmitted by a range of aphid species. *Aphis gossypii* is one of many possible vectors of both. The luteoviruses are persistently aphid-transmitted, but are more vector-specific.

A Verrell (2013) Virus in chickpea in northern NSW 2012. GRDC Update Papers. 26 Feb 2013, <u>http://www.grdc.com.au/Research-and-Development/GRDC-Update-Papers/2013/02/Virus-in-chickpea-in-northern-NSW-2012</u>



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Aphids seldom colonise chickpeas or move between adjacent plants to feed. The result is that chickpea crops show a characteristic scattered distribution of individual virusinfected plant (Figure 30). This contrasts with crops such as peas and faba beans, which aphids do colonise, and patches of infected plants are common in these crops. The pea aphid (*Acyrthosiphon pisum*) appears to colonise chickpeas more readily than other aphid species and is likely to be an important vector.



Figure 30: Scattered appearance of virus-infected plants. (Photo: M. Schwinghamer, NSW DPI) Aphid activity is influenced by seasonal conditions and will require early monitoring in nearby crops and pastures.

Control measures for viruses in chickpea are not adequate at present. Application of seed and foliar insecticides, aimed at preventing feeding by aphids, has failed to prevent infection by viruses in field experiments.⁷

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

- Retain 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.
- Plant on time and at the optimal seeding rate. These practices result in early canopy closure, which reduces aphid attraction (see Figure 31).
- Ensure adequate plant nutrition.
- Control in-crop, fence line and fallow weeds. This removes in-crop and nearby sources of vectors and virus.
- Avoid planting adjacent to lucerne stands. Lucerne is a perennial host on which legume aphids and viruses, especially AMV and Bean leaf roll virus (BLRV), survive and increase.
- Seed treatment with insecticides (e.g. imidacloprid) are not effective for nonpersistently transmitted viruses but may be effective for luteoviruses. Unfortunately, local data supporting seed treatment are lacking.
- Given the high incidence of Beet western yellows virus (BWYV) sometimes found in canola, consider growing chickpeas (and other pulse crops) away from canola.⁸



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Pulse Australia (2013) Northern chickpea best management practices training course manual—2013. Pulse Australia Limited.

⁸ K Moore, M Ryley, M Sharman, J van Leur, L Jenkins, R Brill (2013) Developing a plan for chickpeas 2013. GRDC Update Papers 26 Feb 2013, <u>http://www.grdc.com.au/Research-and-Development/GRDC-Update-Papers/2013/02/Developing-a-plan-for-chickpeas-2013</u>





http://www.grdc. com.au/Researchand-Development/ **GRDC-Update-**Developing-a-plan-forchickpeas-2013

http://www.grdc. com.au/Researchand-Development/ **GRDC-Update**in-chickpea-in-northern-NSW-2012

http://grdc.com. au/Research-and-Development/ GRDC-Update-Papers/2010/05/ Chickpeas-in-2010-Recommendationsfor-2010

M Sharman, K Moore, J van Leur, M Aftab, A Verrell (2014), Impact and management of viral diseases in chickpeas

K Moore, A Verrell, M Aftab (2014), Reducing risk of virus disease in chickpeas through management of plant density, row spacing and stubble

Pulse Australia (2015), Managing viruses in pulses

A Verrell, K Moore (2015), Managing viral diseases in chickpeas through agronomic practices

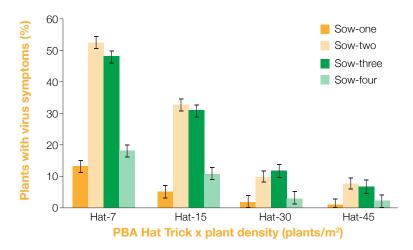


Figure 31: Proportion of plants with virus symptoms for sowing date by plant density for PBA HatTrick(D. 9

The northern experience in 2013 9.11.1

In 2013, virus infection was found in almost all chickpea crops inspected from southern Queensland to Wellington, NSW. The incidence of virus infection was generally lower than observed in 2012, with most crops inspected having <5% plants with symptoms, but it was as high as 30–50% in several crops from the Breeza–Werris Creek area and Edgeroi, NSW. Overall, the most prevalent virus was BWYV, and in some locations, >90% of symptomatic plants were infected with BWYV (Table 4).

Related virus species also react with the BWYV assay, so it is likely there was a mix of BWYV-like viruses present at many locations. Some of the main outcomes from the chickpea surveys in northern NSW were:

- A higher proportion of BWYV infections was found at, and north of, the Liverpool Plains. Higher proportion of AMV infections in the south (Table 4). Very low levels of BLRV and CMV.
- Up to 15% of non-symptomatic plants from the Liverpool plains still had BWYV infection.
- Accurate identification by polymerase chain reaction (PCR) has shown the aphidtransmitted Luteovirus species to have a wide geographical range in a number of alternative weed hosts (Table 5).
- Soybean dwarf virus (SbDV) was the major virus affecting several crops in the Edgeroi region in October 2013 and was confused with BWYV in the antibody test (Table 4).



A Verrell (2013) Virus in chickpea in northern NSW 2012. GRDC Update Papers. 26 Feb 2013. http://www.



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Table 4: Percentage infection of Beet western yellows virus (BWYV), Alfalfa mosaic virus (AMV),Bean leaf roll virus (BLRV) and Cucumber mosaic virus (CMV) from chickpeas displaying virussymptoms in northern NSW as determined by tissue blot immunoassay (TBIA) diagnostic

Virus identification was based on antibody reaction. Sample locations shown roughly from north to south. Note that the BWYV infections may be a complex of related viruses. Samples from most locations were also tested for Turnip mosaic virus (TuMV), but no positives were detected. n.t., Not tested

Location	No .of plants tested	% BWYV	% AMV	% BLRV	% CMV
Boomi	6	100	0	n.t.	n.t.
North Star	12	67	17	n.t.	n.t.
Moree	19	79	0	0	0
Edgeroi	32	62	0	n.t.	n.t.
Edgeroi	17	47	0	n.t.	n.t.
Tamworth	15	60	20	n.t.	0
Tamworth	30	87	10	n.t.	0
Breeza	18	89	0	5	6
Breeza	25	88	8	4	8
Breeza	26	77	0	0	0
Breeza	19	53	5	0	5
Liverpool Plains	20	90	10	5	0
Liverpool Plains	21	90	10	5	0
Werris Creek	15	73	13	n.t.	0
Pine Ridge	15	93	7	n.t.	0
Pine Ridge	15	80	13	n.t.	0
Blackville	15	13	67	n.t.	0
Gilgandra	14	7	78	n.t.	n/t
Gilgandra	38	21	71	0	3
Gilgandra	49	12	88	0	2
Wellington	30	10	73	n/t	n.t.
Wellington	16	19	63	0	0
Wellington	15	7	60	0	0
Wellington	20	5	55	0	0

Biology of significant viruses of pulses, particularly chickpeas

Accurate identification of viruses is critical for the long-term success of resistance breeding and for meaningful studies of how viruses survive in weed hosts and move into crops. To this end, improved accurate diagnostics are being developed for the luteoviruses, to help overcome uncertainty of virus identifications that can result from cross-reactions of viruses to some antibodies. PCR has been used for BWYV, BLRV, Phasey bean virus (PhBV) and SbDV to investigate the host range of the virus species from various locations (Table 5). Although testing continues, marshmallow weed is commonly found infected with BWYV from many locations and burr medic is a host for BLRV, PhBV and SbDV.



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Table 5:Identification of virus species in different plant hosts from different locations in the
northern region confirmed by species-specific polymerase chain reaction (PCR)Testing of selected samples from 2012 and 2013 surveys

Virus (by PCR or sequencing)	Plant host	Locations
Beet western yellows virus	Chickpea	Wellington, Breeza, North Star, Boomi
	Canola	Ardlethan, Burren Junction, Bellata
	Marshmallow	Wagga Wagga, Coolamon, Griffith, Hillston, Leeton, Narrandera, Wellington, Tamworth, Narrabri, Wee Waa, North Star, Goondoowindi, Grantham
	Turnip weed	Gravesend, Wee Waa, Burren Junction
	<i>Sonchu</i> s sp. Coolamon Shepherds purse	Kingsthorpe, Boomi
Bean leaf roll virus	Chickpea	Wellington, Edgeroi
	Burr medic	Wellington
Phasey bean virus	Chickpea	Kingsthorpe, Boomi, North Star, Edgeroi, Burren Junction, Breeza, Horsham
	Faba bean	Edgeroi
	Burr medic	Boomi, Burren Junction, Wee Waa
	Lentil	Breeza
	Vetch	Kingsthorpe
Soybean dwarf virus	Chickpea	Wellington, Gilgandra, Breeza, Edgeroi, Bellata, North Star, Boomi, Clifton

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 (Verrell 2013, Moore *et al.* 2014). 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 (Verrell 2013).

Inter-row planting into standing wheat stubble significantly reduced virus incidence in small trial plots of PBA HatTrick(^b compared with the same amount of stubble slashed low to the ground (Moore *et al.* 2014). 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 (Hawthorne 2008; Verrell 2013, 2014), 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^(b) and PBA HatTrick^(b), although both these varieties have been observed with high rates of infection under high disease pressure. 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 (van Leur *et al.* 2014), the



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1 More information

M Sharman, K Moore, J van Leur, M Aftab, A Verrell (2014) Viral diseases in chickpeas—impact and management. GRDC Update Papers 4 March 2014, <u>http://www.grdc.</u> com.au/Researchand-Development/ GRDC-Update-Papers/2014/03/ Viral-diseases-inchickpeas-impact-andmanagement

K Moore, A Verrell, M Aftab (2014) Reducing risk of virus disease in chickpeas through management of plant density, row spacing and stubble. GRDC Update Papers 4 March 2014, https://www. grdc.com.au/Researchand-Development/ <u>GRDC-Update-</u> Papers/2014/03/ Reducing-risk-of-virusdisease-in-chickpeas

Watch for chickpea viruses near canola (2013)



http://www.grdc. com.au/Researchand-Development/ GRDC-Update-Papers/2013/02/ Developing-a-plan-forchickpeas-2013 sometimes high incidence of BWYV in canola indicates it may be prudent to avoid planting chickpea and other pulse crops next to canola. ¹⁰

Proximity to canola plantings

Larger canola plantings may have been responsible for unusually severe outbreaks of viruses in the northern region's chickpea crops in 2012.

Plant pathologists say the link is unproven, but canola crops host BWYV, which could spread to chickpeas.

Canola and turnip weed close to surveyed chickpea paddocks showed high infections of BWYV and Turnip mosaic virus (TuMV) and could have played a role in the virus epidemic in chickpeas. The outbreak of BWYV was especially severe on the Liverpool Plains, NSW, and was costly as it wiped out several chickpea crops.

In 2012, the area sown to canola was about five times the long-term average because of better prices for canola and poor prices for other crops. Canola is sown earlier in the season than chickpeas. BWYV inoculum could build up in canola over winter then spread to chickpeas in spring and cause severe yield losses.

Growers need to pay attention to the whole farming system and growing environment of their crops to ensure that plants are healthy enough to fight incursions. Viruses are more severe in poor growing paddocks.

A healthy plant seems to have the ability to withstand the virus, so it is worth following recommended agronomic practices to reduce the chance of virus infection and increase the ability of plants to resist the virus. This includes sowing in standing stubble because virus-spreading aphids tend to be more attracted to plants that are in poor growing paddocks or growing in bare ground.

Researchers try to identify resistance as part of the breeding program in order to deliver varieties with improved resistance. ¹¹

9.11.2 Economic importance

Plant viruses occur in all states; however, they are a significant problem in chickpeas in northern NSW and southern Queensland, where total crop failures have occasionally occurred.

The damage caused by the viruses varies greatly from season to season and depends on the prevalence of aphids.

9.11.3 Virus types

Viruses that cause significant losses in chickpea include the following.

Luteoviruses:

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

Shoot tip virus complex:

- Alfalfa mosaic virus (AMV)
- Cucumber mosaic virus (CMV) (Figures 31 and 32)
- ¹⁰ M Sharman, K Moore, J van Leur, M Aftab, A Verrell (2014) Viral diseases in chickpeas—impact and management. GRDC Update Papers 4 March 2014, <u>http://www.grdc.com.au/Research-and-Development/</u> <u>GRDC-Update-Papers/2014/03/Viral-diseases-in-chickpeas-impact-and-management</u>.
- ¹¹ R Bowman (2013) Watch for chickpea viruses near canola. GRDC Media Centre May 2013, <u>https://grdc.</u> <u>com.au/Media-Centre/Media-News/North/2013/05/Watch-for-chickpea-viruses-near-canola</u>



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Other less common viruses:

- Lettuce necrotic yellows virus (LNYV)
- Clover yellow vein virus (CIYVV)

9.11.4 Symptoms

Luteoviruses will kill plants within 3-4 weeks of symptoms showing.

The diseased plants have a scattered distribution, usually occurring around the edges of a crop or in areas where plant numbers are low.

In Desi varieties of chickpeas, the leaves and stems become red or brown, whereas Kabuli varieties turn yellow. In older plants that are podding, premature death may be the only obvious symptom.

A shallow cut with a knife at the base of the main stem often reveals that the stem has turned brown, compared with a white or green colour in healthy plants.



Figure 32: Symptoms of Cucumber mosaic virus include reddening of the leaves and stunted growth. Symptoms are often confused with nutritional deficiency or herbicide damage. (Photo: G. Cumming, Pulse Australia)



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Feedback



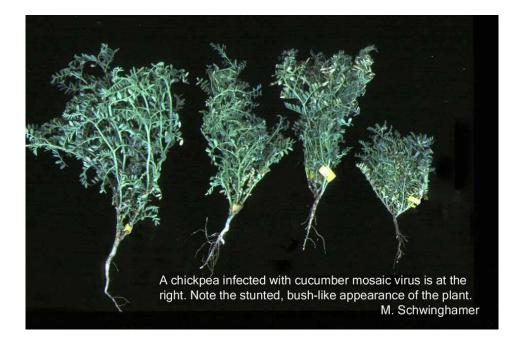


Figure 33: Shoot-tip virus complex. (Photo: M. Schwinghamer, NSW DPI)

The symptoms of AMV and some other viruses are similar to Luteovirus but more pronounced on the shoot tops. The symptoms include a pale colouration, bunching with small leaves, tip death, and the shoots are horizontal or even pointing downwards.

9.11.5 Chickpea virus-testing resource

As part of a new GRDC project, NSW DPI researcher Dr Jenny Wood is working on eliminating grain defects in Desi and Kabuli chickpeas (Figure 34). The information will assist chickpea breeders to breed for tolerance to seed markings in future varieties.

The two defects being examined are:

- seed markings (particularly seeds with tiger stripe or blotch markings); and
- weather-damaged seed (symptoms include light weight, brittle seeds or sprouting).

Samples are requested, whether they look clean or contain visibly diseased, marked or weathered seeds.

The grain will also be tested for germination, emergence, seed-borne diseases and moulds, by Dr Kevin Moore, Northern NSW Integrated Disease Management, NSW DPI, and results returned.

Please send a sample of harvested grain, ideally 1 kg, secured in two plastic bags (double-bagged). Do not hand pick the sample, as it must be representative of your entire harvested crop.

Send with the sample testing form, which includes:

- variety
- address of the crop paddock (GPS coordinates if possible)
- dates the crops was sown and harvested (plus flowering and maturity dates if you have them)
- information on any stressors the crops suffered in the field, including moisture or heat stress
- · observations of any reddening of the foliage
- details of other varieties nearby that were affected



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<u>M Schwinghamer, T</u> <u>Knights, K Moore (2009),</u> <u>Virus control in chickpea</u> <u>- special considerations</u>



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Samples should be addressed to: Dr Jenny Wood C/- Kate Keir Tamworth Agricultural Institute 4 Marsden Park Rd Calala NSW 2340

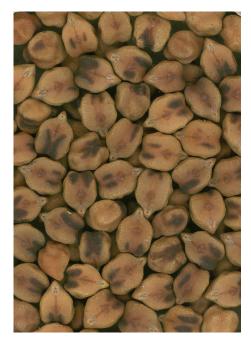


Figure 34: NSW DPI is now testing seed for germination, emergence, seed-borne diseases and moulds, as well as examining seed defects as part of a GRDC-funded project.

9.12 Fungal disease control

9.12.1 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.

9.12.2 Principles of spraying

A fungicide spray at the commencement of flowering protects early podset. 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 about 10 days.

Timing of fungicide sprays is critical (Table 6). As Ascochyta blight and BGM can spread rapidly, DO NOT DELAY spraying. A spray in advance of a rainy period is most desirable.



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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.

Table 6:	Principles of when to s	prav for fungal dis	ease control in chickpea

Occurrence	When to spray
First appears	Ascochyta blight is spread by rainfall.
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 \geq 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
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)
	First appears under wet conditions



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Table 7 illustrates the relative importance of various forms of carryover of fungal disease infection for chickpeas and other pulses.

Table 7: Carryover of major pulse diseases, showing their relative importance as sources of infection

Disease	Stubble	Seed	Soil
Ascochyta blight	***	**	*
Botrytis grey mould	***	***	*
Phytopthora root rot			***
Sclerotinia rot	*	*	***

9.13 Registered fungicides

Table 8 provides a list of registered seed dressings for chickpeas.

Refer to the current product label for complete 'Directions for use' prior to application.

Prior to the use of any crop protection product, ensure that is currently registered or that a current permit exists for its use in chickpeas.

Registered labels and current permits can be found on the APVMA website (<u>www.apvma.gov.au</u>).

Table 8: Seed dressings registered for use with chickpea ✓, Registered product label claim

Active ingredient	Thiram	Thiram + thiabendazole	Metalaxyl-M
Example trade name	Thiraflo®	P-Pickel [®] T	Apron [®] XL
Ascochyta blight	\checkmark	\checkmark	
Botrytis grey mould	\checkmark	\checkmark	
Damping-off (Kabuli)	\checkmark	\checkmark	\checkmark
Phytopthora root rot			\checkmark
Jurisdiction	All States	All States	Qld, NSW, Vic, SA, WA

Table 9 provides a summary of the main disorders affecting chickpea, their causes, transmission, symptoms and management.

Table 9: Key features of the main chickpea disorders

Disorder and cause	Seed- borne?	Symptoms	Distribution and occurrence	Survival and spread	Management
Seed-borne root rot: Botrytis cinerea Ascochyta rabiei (very rare)	Yes	Seedlings wilt and die, epicotyl rots	Random individual plants (not patches)	Seed	Quality seed; seed treatment
Phytophthora root rot (PRR): Phytophthora medicaginis	No	Rapid wilting and yellowing; defoliation from lower leaves; rotted roots; plants easy to pull up	Patches; poorly drained areas; heavy rainfall; can occur at any time; history of medics, lucerne or PRR	Oospores in soil and residue persist for many years; survives saprophytically; spread by water and soil	Varietal selection; avoid paddocks with history of PRR; rotation; seed treatment
Waterlogging: root anoxia	No	Very rapid death; little defoliation; roots not rotted but may be dark; plants hard to pull up	Patches; poorly drained areas; heavy rainfall; higher temperatures, i.e. later in season	Caused by insufficient supply of oxygen to roots	Avoid low-lying or poorly drained paddocks or areas within paddocks



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Sclerotinia root and stem rot: Yes (ad- mixed) Wilting and death; bleached root, colar and stem tissue; white cottony mould at site of lesion; sclerotia at lesions or inside stems Root and collar lesions result from direct infection from sclerotia; stem lesions result from patches; favoured by denser cancels; swett verents Sclerotia persist in soli for mary years; wide host ranges and broadbes, cancels, sufflowers patches; favoured by throadby present in most solis Avoid paddocks w history of Sclerotiu with elsions cancels with recreats; some succeptible Rhizoctonia rot: ? Death of seedlings, stumting of survivors damping-off of epicotyl Can be a problem in trigated crops grown imediately after contor. Often accurs in 1–5m stretches of row stumps; plant death Survives as sclerotia and on decomposition of trash. Allow time for most solis Accochyta blight: Accochyta grey mould (BGM): Botrytis crinerea Yes vers Ghosting of tissues; lesions with concertin rings of proridia; stem stumps; plant death Small patches enlarge rapidly in wet weather conp between the selesion; or patches Chickpea residue very important in solia Foldow chickpea management package publisher can blow arout, solia shr/vide host range; solia shr/vide host range; solia shr/vide host range; solia soli and/vide host range; solia soli and/vide host range; solia soli and/vide host range; solia soli and/vide host range; solia soli and/videhost range; solia soli and/vide host range; solia soli an	Disorder and cause	Seed- borne?	Symptoms	Distribution and occurrence	Survival and spread	Management
Rhizoctonia solanistutning of survivors due to root damage, re-shooting after damping-off of epicotylirrigated crops grown immediately after cotton. Often occurs in 1–5m stretches of rowand on decomposing trash. Probably present in most solisdecomposition of (preceding) crop debris. Tillage sho helpAscochyta blight: rabieiYesGhosting of tissues; lesions with concentric rings of pycnidia; stem stumps; plant deathSmall patches enlarge rapidly in wet weather torpChickpea residue wery important in spread especially surface weater flow; infected seed; wolunteersFollow chickpea Ascochyta blight magement package publishe and warm humid conditions persist; and warm humid conditions persist; in soilChickpea residue wery important in spread edue despecially volunteersFollow chickpea Ascochyta blight magement package publishe and warm humid conditions persist; individual plants or patchesChickpea residue very important in sclerotia can survive in soilFollow chickpea Ascochyta blight magement packageRoot-lesion nematodes: Pratylenchus spp.NoGeneral poor growth; small black lesions on lateral roots sometimes visibleOften affects large parts of crop; <i>P thornei</i> more prevalent on high clay solisWide host range sale alvorte solidViruses prevalent on high clay solisViruses verse (uke host range, survives and spreads allows nematodes to previdesFarn hygiene; state with resista spreadsRoot-lesion Pratylenchus spp.NoGeneral poor growth; shoot tips; later discoloration.Initially bunching,	Sclerotinia root and stem rot:	Yes (ad-	bleached root, collar and stem tissue; white cottony mould at site of lesion; sclerotia at lesions or	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	Sclerotia persist in soil for many years; wide host range including pulses, canola, sunflowers and broadleaf weeds but not cereals or	
Ascochyta (Phoma) rablellesions with concentric rings of pycnidia; stem stumps; plant deathrapidly in wet weather to kill large areas of cropvery important in spread especially and surface water flow; infected seed; volunteersAscochyta blight management anzually; includes foliar fungicidesBotrytis grey mould (BGM): Botrytis cinereaYes/ noStem, flower pod and leaf lesions covered in grey mouldOccurs later in season when canopy closes and warm humid conditions persist; individual plants or patchesCan flow-on from seed-borne root rot wide host range; and airborne spores can blow around; sclerotia can survive in soilAvoid highly susceptible varieti management packageRoot-lesion nematodes: Pratylenchus spp.NoGeneral poor growth; small black lesions on lateral roots sometimes visibleOften affects large parts of crop; P thornei more prevalent on high clay soilsWide host range; survives and spreads in soil, anhydrobiosis and multiply in weeds and pasture sperist for prolonged dry periodsFarm hygiene; rotate with resista species; grow tolerant varietiesAMV (Alfalfa mosaic virus), CMV (Cucumber mosaic virus)YesInitially bunching, reddening, yellowing, wilting or death of uiscolaration.Initially scattered plants often at edges of crop; more common in thin 		?	stunting of survivors due to root damage, re-shooting after damping-off of	irrigated crops grown immediately after cotton. Often occurs in	and on decomposing trash. Probably present in	decomposition of (preceding) crop debris. Tillage should
(BGM): Botrytis cinereanoleaf lesions covered in grey mouldwhen canopy closes and warm humid conditions persist; individual plants or patchesseed-borne root rot but pathogen has wide host range; and airborne spores can blow around; sclerotia can survive in soilsucceptible varieti plant on wider row follow chickpea Ascochyta blight management packageRoot-lesion nematodes: Pratylenchus spp.NoGeneral poor growth; small black lesions on lateral roots sometimes visibleOften affects large parts of crop; <i>P. thornei</i> more prevalent on high clay soilsWide host range; survives and spreads in soil; anhydrobiosis 	Ascochyta (Phoma)	Yes	lesions with concentric rings of pycnidia; stem	rapidly in wet weather to kill large areas of	very important in spread especially header dust and surface water flow; infected seed;	Ascochyta blight management package published annually; includes
nematodes: Pratylenchus spp.small black lesions on lateral roots sometimes visibleof crop; P. thornei more prevalent on high clay soilssurvives and spreads in soil; anhydrobiosis allows nematodes to persist for prolonged dry periodsrotate with resista species; grow tolerant varietiesAMV (Alfalfa mosaic virus), CMV (Cucumber mosaic virus)YesInitially bunching, reddening, yellowing, wilting or death of shoot tips; later discoloration.Initially scattered plants often at edges of crop; more common in thin stands.Viruses persist and multiply in weeds and pasture legumes; aphid- borne except for CpCDV (leafhopper).Establish uniform stand by using 	(BGM):		leaf lesions covered in	when canopy closes and warm humid conditions persist; individual plants or	seed-borne root rot but pathogen has wide host range and airborne spores can blow around; sclerotia can survive	susceptible varieties; plant on wider rows; follow chickpea Ascochyta blight management
virus), CMV (Cucumber mosaic virus)reddening, yellowing, wilting or death of shoot tips; later discoloration.often at edges of crop; more common in thin stands.and multiply in weeds and pasture legumes; aphid- borne except for CpCDV (leafhopper).stand by using recommended sowing rates and times; sowing into standing stubble.Phloem-limited viruses (luteoviruses): BLRV (Bean leaf roll SCRLV (SubterraneanDeath of entire plant; Luteovirus infected plants often have discoloured phloemClose to lucerne; seasons or districts with major aphid flightsCereal stubble deters aphid; gro resistant varieties	nematodes:	No	small black lesions on lateral roots	of crop; <i>P. thornei</i> more prevalent on high clay	survives and spreads in soil; anhydrobiosis allows nematodes to persist for prolonged	rotate with resistant species; grow
viruses (luteoviruses): BLRV (Bean leaf roll virus), SCRLV (SubterraneanLuteovirus infected plants often have discoloured phloemseasons or districts with major aphid flightsdeters aphids; gro resistant varieties	virus), CMV (Cucumber	Yes	reddening, yellowing, wilting or death of shoot tips; later	often at edges of crop; more common in thin	and multiply in weeds and pasture legumes; aphid- borne except for	stand by using recommended sowing rates and times; sowing into
clover red leaf virus), BWYV Beet western yellows virus, SCSV (Subterranean clover stunt virus)	viruses (luteoviruses): BLRV (<i>Bean leaf roll</i> <i>virus</i>), SCRLV (<i>Subterranean</i> <i>clover red leaf virus</i>), BWYV <i>Beet western</i> <i>yellows virus</i> , SCSV (<i>Subterranean</i>	No	Luteovirus infected plants often have	seasons or districts		deters aphids; grow
CpCDV (Chickpea ? Reddening, proliferation of axillary branching Individual or small ? (Leafhopper ? ? Maybe more at edges of crop for comp ? ?		?	proliferation of axillary	clusters of plants. Maybe more at edges		?



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