Mungbean and Sorghum Disease Update

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Take home message
• Avoid paddocks with a history of Fusarium wilt in mungbean. Plant seed into well-drained soils and minimise plant stress
• To minimise the risk of halo blight and tan spot: use low risk planting seed, plant varieties with higher levels of resistance, clean harvesting equipment, control weeds and volunteers, and use suitable crop rotations
• Report phytoplasma outbreaks to Qld DAF Entomologists and Plant Pathologists
• Timely fungicide applications of Folicur® or Custodia® are effective at managing powdery mildew in mungbean. Crops should be sprayed at the first sign of disease and then again 14 days later, or sprayed when the disease is at 1/3 way up the plant and again 14 days later
• Sorghum stubble is a good reservoir for Fusarium thapsinum to survive between sorghum crops
• Level of F. thapsinum in sorghum stubble starts to decline after two months with standing stubble treatments appearing to decline at a slower rate than surface and buried treatments. By 18 months, F. thapsinum levels appear to be similar across all stubble treatments.
• PREDICTA B test for Macrophomina phaseolina is under evaluation.

Mungbean Background
Annual disease surveys, field observations, and diagnostic samples indicate that the dominant mungbean diseases continue to cause significant constraints to the mungbean industry. A
combination of poor host resistance, virulent pathogens, and ideal weather conditions will allow pathogens to infect crops and cause significant losses. Halo blight and tan spot are a common occurrence every season, and continue to be a major threat to the ‘clean’ seed scheme. Powdery mildew is typical in later planted summer crops, causing significant losses when left untreated. Stubble and soil-borne diseases, such as Fusarium wilt, appear to be increasing in incidence and severity, causing significant losses in individual paddocks. Recent cropping seasons have also seen major outbreaks in phytoplasma, which is usually considered a minor threat to growers.

**Phytoplasma**

In recent years there have been outbreaks of phytoplasma in a number of crops throughout the northern region. Phytoplasma are specialised bacteria that infect plants, and are spread from plant to plant by insect vectors, such as leaf hoppers. The phytoplasma responsible for these recent outbreaks is thought to be closely related to Pigeon Little Leaf Phytoplasma. Symptoms in severely affected plants infected prior to flowering are stunted with masses of small cupped leaves that don’t flower or produce pods. In plants infected after flowering, there are often masses of deformed flowers and small pods that remain green and fail to produce harvestable seed.

Over the last 3-4 years there have been localised outbreaks of phytoplasma in mungbean across central Queensland. On the central Downs during the 2015-16 autumn over 200ha of soybean were lost due to phytoplasma infection. Phytoplasma was widely reported from central Queensland to the Burnett to the Darling Downs to north-western NSW (Moree and Narrabri) in mungbean during the 2016 spring planting. During the 2016-17 summer, phytoplasma was widely reported in mungbean, soybeans, peanuts and pigeon pea from north Queensland to central NSW. In some of the worst crops 60-90% of plants were infected, whilst others had a lower infection of 10-15%. Prior to the succession of outbreaks in recent years, phytoplasma infection is estimated to have only occurred at a very low incidence (usually much less than 1%).

Phytoplasma is likely vectored by the common brown leafhopper, *Orosius orientalis*, although further research is needed to confirm this. The small (3mm) brown common leafhopper has been sighted in phytoplasma-affected crops in central Qld, the Darling Downs and the South Burnett.

It is unclear what the underlying reasons are for this sudden increase in phytoplasma disease incidence in recent years. One reason could be that spring rains have favoured weeds (e.g. *Datura*) that host the leafhopper. It is also possible that the phytoplasma may be vectored by another insect (most likely within the Cicadellidae family). Further research and paying close attention to any new infestations is vital to gain a better understanding of the disease and its insect vector and develop strategies to minimise damage.

Puffy pod symptoms have been observed in many mungbean crops infected with phytoplasma in recent outbreaks. Phytoplasma has also been detected in an archived sample of puffy pod from central Queensland collected in 2012. Symptoms of puffy pod include swollen, soft pods with a mottled appearance that contain brown seeds. It has previously been thought that the cause of puffy pod was abiotic, although there is a possibility that puffy pod may be linked with phytoplasma. Further research is needed to confirm this potential link.

It is currently unclear if controlling leafhoppers with an insecticide would prevent the transmission of phytoplasma. Previous experience from other insect-vectored diseases would indicate that spraying was an ineffective management technique because transmission of the disease usually occurs early in the crop growth, and at very low vector densities – often below the level of easy detection with traditional sampling methods. Attempting to control the vectors through the application of multiple prophylactic sprays of a non-selective insecticide (e.g. synthetic pyrethroids, organophosphates) will run the risk of flaring other pests like thrips, helicoverpa, and mirids. In addition, every application of SPs and OPs selects for resistance in helicoverpa.
Growers should monitor crops for leafhopper activity and phytoplasma infection, and report any outbreaks to Qld DAF Entomologists and Pathologists.

**Mungbean Powdery Mildew**

Powdery mildew in mungbean is caused by the fungus *Podosphaera fusca* (also known as *P. xanthii*). Yield losses of up to 40% have been demonstrated on susceptible varieties, such as Berken\(^1\) when the disease becomes established prior to flowering. Symptoms are first evident on the lower leaves as small, circular, white powdery colonies that can quickly move up plants and cover the surface of leaves under ideal conditions. If severe enough, powdery mildew will also cover the stems and pods of plants. Some varieties, e.g. Green Diamond\(^1\), develop a red-brown discoloration on the leaf surface at the site of infection. Infection may occur at any stage of plant growth when air-borne spores land on the leaf surface. The fungus then sends feeding structures (haustoria) into the cells of the leaf (epidermis), and then chains of spores develop from fungal strands, resulting in the white, powdery growth on infected tissues. These spores become air-borne, spreading long distances in the wind. Disease development is favoured by cool (22-26ºC), dry weather, and under ideal conditions the cycle of infection may be as short as five days.

*Podosphaera fusca* can only survive between seasons on a living host, and will not survive in seed, in soil, or in crop residues. Currently the only confirmed alternative hosts of *P. fusca* include phasey bean (*Macroptilium lathyroides*) and several members of the Asteraceae family.

Powdery mildew is best managed by planting varieties with higher levels of resistance and through the strategic use of fungicides. The variety Jade-AU\(^1\) currently has the highest levels of resistance to the powdery mildew pathogen. Tebuconazole, sulphur, and Custodia* are currently under permit by the APVMA for the control of powdery mildew in mungbean.

**Fungicide row spacing trials**

Previous research has indicated that the most effective time to spray Tebuconazole was at the first sign of disease and then 14 days later if needed. It has been unclear whether this fungicide strategy would apply to other fungicides, and on different row spacings. Two GRDC-funded trials were undertaken during the 2016-17 season to determine the best fungicide management strategy for the control of powdery mildew using different fungicides on the variety Jade-AU\(^1\) when grown on three different row spacings (25cm, 50cm, and 100cm). Both trials were located in southern Queensland, one at the DAF Hermitage Research Facility (HRF) at Warwick, and the second trial at Missen Flat, approximately 40km south of Toowoomba. The fungicides Folicur\(^*\) 430 EC (active ingredient tebuconazole 430g ai/L) and Custodia\(^*\) 320 SC (tebuconazole 200g ai/L plus azoxystrobin 120g ai/L) were applied at 145mL and 300mL product/ha, respectively, in >100L of water using a hand-held boom spray. Each trial consisted of six fungicide treatments (refer to Table 1 and Figures 1 and 2) for each of the three row spacings. Spreader rows of the variety Berken\(^1\) were used in both trials to encourage natural disease infection.

Trials were rated on six dates using a 1-9 scale of disease incidence, where 1 = no disease and 9 = colonies of powdery mildew on the top leaves of every plant with leaf defoliation. In addition, on two dates the trial was rated with an additional 1-5 scale to measure the severity of the disease across the whole plot, where 1 = the plot was all green with only small colonies of powdery mildew visible, 5 = the plot was severely bronzed with leaf defoliation and colonies of powdery mildew covering entire leaves. The trials were harvested at crop maturity, and the final yields were measured. Please note that only the results for one trial (Missen Flat) are presented in this paper as the data from the second trial is still being collected.
The development of powdery mildew over time averaged over the three row spacings at the Missen Flat trial is presented in Figure 1. Powdery mildew progressed rapidly in the untreated (no fungicide application) plots, reaching an incidence rating of 9 prior to crop maturity. All other fungicide treatments reached an incidence rating between 8 and 9 on their final rating (76 days after emergence [DAE]), although all disease in the other treatments developed at a slower rate compared to the untreated across the three row spacings. Disease development progressed more slowly in the treatments when the fungicides were applied only at the first sign of disease, or at the first sign of disease and then again 14 days later.

![Figure 1. Development of powdery mildew on mungbean variety Jade-AU in the 2017 Missen Flat trial](image)

The final severity rating (1-5) for the different treatments across the three row spacings is displayed in Figure 2. The incidence rating captured how the disease developed and spread over time, whereas the severity rating gave an indication of how much disease was present in individual plots. The severity of powdery mildew in plots sprayed prior to disease establishment (4 weeks post emergence treatment), or sprayed at the first sign of disease only, was no different to those in the untreated plots. Fungicide applications in the remaining treatments resulted in lower disease severity ratings. There was some indication that disease developed more quickly in the wider row spacings, however further research is needed to confirm this trend.
The percentage yield increase in each treatment compared to the untreated plots is shown in Table 1. Yields were highest in the treatments sprayed at the first sign of disease and then 14 days later, those sprayed once the disease reached 1/3 way up the plant, and in those sprayed twice after the fungus had reached 1/3 way up the plant, regardless of row spacing. These same treatments also had the lowest severity ratings at 76 days after emergence (Figure 2). Both fungicides resulted in large increases in yields compared to the untreated plots, particularly in the treatments sprayed more than once.

Table 1. Increases in yield in mungbean variety Jade-AU† under different fungicide applications during the 2017 Missen Flat trial across three row spacings

<table>
<thead>
<tr>
<th>Treatment (number of sprays)</th>
<th>25cm</th>
<th>Yield increase (%)</th>
<th>50cm</th>
<th>1m</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Custodia®</td>
<td>Folicur®</td>
<td>Custodia®</td>
<td>Folicur®</td>
</tr>
<tr>
<td>1/3 up plant (1)</td>
<td>42.8</td>
<td>1.5</td>
<td>39.5</td>
<td>16.2</td>
</tr>
<tr>
<td>1/3 up plant + 14 days later (2)</td>
<td>22.8</td>
<td>23.1</td>
<td>28.9</td>
<td>-11.4</td>
</tr>
<tr>
<td>First sign + 14 days later (2)</td>
<td>22.5</td>
<td>23.1</td>
<td>36.6</td>
<td>30.6</td>
</tr>
<tr>
<td>4 weeks post emergence (1)</td>
<td>10.9</td>
<td>-6.8</td>
<td>15.1</td>
<td>12.1</td>
</tr>
<tr>
<td>First sign (1)</td>
<td>10.7</td>
<td>3.9</td>
<td>28.8</td>
<td>-16.7</td>
</tr>
</tbody>
</table>

*Yield increase (%) = (mean yield of treatment – mean yield of untreated)*100/mean yield of untreated

The effectiveness of these treatments will differ between seasons based on weather conditions, and the timing of when powdery mildew first appears in a crop. The results of this trial suggest that both tebuconazole and Custodia® are effective at managing powdery mildew in mungbean. The effectiveness of specific spray regimes will differ from year to year, however the results from this trial support previous findings that found that spraying at the first sign of disease and again 14 days later was an effective management strategy. The results presented also indicate that there is an
option to spray once the disease is 1/3 way up the plant and then 14 days later if that initial sighting of disease is missed.

**Fusarium wilt in mungbean**

Fusarium wilt and root rot, caused by *Fusarium solani* and *F. oxysporum* is becoming an increasing threat to mungbean producers across the northern region. It is estimated that current infestations are costing the mungbean industry in the vicinity of at least $4.3 million per annum through lost production. Yield losses attributable to Fusarium wilt have been estimated to be as high as 80% in affected paddocks, causing huge losses to individual growers and the mungbean industry.

Affected plants often wilt and die. Surviving plants often remain stunted, develop a basal rot, experience leaf defoliation, and develop a brown discoulouration of the internal vascular tissues. The disease is often found at a low incidence (1-10%) in most crops, although individual paddocks have been reported to have incidence as high as 80% in affected areas. Fusarium wilt is more prevalent in heavy clay soils, particularly on the edge of crops and low-lying areas. Symptoms are more prevalent in crops experiencing stress, such as excess water.

The biology of the pathogens causing Fusarium wilt in Australia is poorly understood. Recent research suggests that the species, *F. solani* and *F. oxysporum*, are responsible for the disease in Australia, however further research is needed to gain a better understanding of the diversity of species involved. Current research, as part of the National Mungbean Improvement Program (NMIP), is underway to develop a reliable screening technique to assess the levels of resistance in the current commercial cultivars and advanced breeding lines. Preliminary trials have indicated that the black gram variety Regur may have higher levels of resistance to the pathogens, however further research is underway to confirm these findings. Future research is vital to investigate alternative hosts and other integrated disease management strategies.

Future disease management will be heavily reliant on crop rotations, careful paddock selection, and the use of more resistant cultivars. Both of these species will survive in the soil of affected paddocks for a number of years. It is recommended that growers avoid paddocks with a history of disease, sow seed into well-drained soils, and avoid plant stress where possible.

**Halo blight and Tan spot in mungbean**

Halo blight, caused by *Pseudomonas savastanoi pv. phaseolicola*, and tan spot, caused by *Curtobacterium flaccumfaciens pv. flaccumfaciens*, are two important bacterial diseases of mungbean. Both diseases are found in mungbean crops from all regions in every season. Tan spot is typically more prevalent in the hotter (>30ºC), drier seasons when plants are more stressed, whilst halo blight is more common during the cooler (18-23ºC), wetter seasons. The risk of an epidemic occurring in a crop can be minimised by;

- **Planting varieties with higher levels of resistance.** Celera II-AU has the best levels of resistance to the halo blight pathogen, whilst Jade-AU and Crystal offer the best resistance to the tan spot bacterium.

- **Planting low risk seed.** Both pathogens are highly seed-borne. Seed from an infected crop should be avoided. Australian Mungbean Association approved seed is sourced from crops that were inspected for symptoms of halo blight and tan spot during the growing season.

- **Crop rotation.** A number of hosts have been reported as potential hosts for one or both of the bacterial diseases, including cowpea and *Phaseolus* species. Mungbean should be rotated with a non-host crop for at least two years.
Control volunteers and weed hosts. The bacterium will survive between seasons on volunteer mungbean plants and weed hosts, such as cowvine, bellvine, morning glory, *Desmodium* and *Centrosema*.

Avoid unnecessary movement through the crop. Movement through the crop should be avoided to minimise wounding the foliage and spreading the bacteria further. Harvesting equipment should be thoroughly cleaned prior to entering a crop.

For further information on the biology, symptoms and management of these two bacterial diseases please refer to the paper from the 2016 June/July GRDC updates in the link below;


In recent years multiple pathotypes of the halo blight pathogen have been identified in NMIP trials. To assess the diversity of putative pathotypes present, over 250 isolates have been collected since July 2013 from grower’s paddocks across the northern region. A differential set, consisting of two commercial cultivars and four advanced breeding lines which display varying levels of resistance to the pathogen in the field, was developed to screen isolates. Currently over 200 isolates have been screened, and a total of twelve putative pathotypes have been identified. The results indicate that one particular putative pathotype has been the predominant strain found in each year and in each region. At least six of the putative pathotypes identified are virulent on lines which were previously resistant or immune to halo blight isolates prior to 2012. These differences in pathotypes could have huge impacts on the reliability of future host resistance. Further screening of halo blight isolates will be ongoing to confirm this diversity of pathotypes.

**Stalk Rot in Sorghum**

Sorghum stalk rots can be caused by either bacterial or fungal pathogens. Bacterial stalk rots (*Erwinia chrysanthemi*) are generally not common, cause a soft black fragrant rot inside the pith and are usually associated with insect damage and sustained hot wet conditions.

Fungal stalk rots, commonly found in sorghum, are considered to be a late-season disease caused by a suite of fungal organisms which often occur together. The two most important pathogens are *Macrophomina phaseolina*, causing charcoal rot and *Fusarium species*, causing fusarium stalk rot. Of the *Fusarium* species which have been associated with stalk rot, *Fusarium thapsinum* and *F. andiyazi* are the two most frequently isolated ones.

Stalk rot (fungal) causes yield loss through poor grain fill, but more commonly through plant lodging which impedes harvest and reduces grain quality. Although evidence indicates that infection occurs at any stage of plant development, the disease progresses rapidly after post flowering stress. Unfavourable environmental conditions are one of the key stressors known to exacerbate disease development. Charcoal rot commonly develops after a prolonged period of drought stress caused by hot dry conditions during grain development. Similarly, fusarium stalk rot also develops under these conditions, as evident in the 2016/17 summer season, but is often more severe when cool wet conditions occur in late maturity after this period of hot, dry weather. Agronomic factors that place the plant under any form of stress in the later stages of grain development, such as high plant populations and suboptimal nutrition, can also promote disease development. Further detail on current management strategies for charcoal rot and fusarium stalk rot can be found in the following GRDC link:
The ongoing research presented in this section of the paper aims to further our understanding of *M. phaseolina* and the *Fusarium* species involved in causing stalk rot, enabling us to develop the best management strategies for growers.

**Charcoal rot**

Lodging is the first obvious sign of charcoal rot as plants approach maturity. When stalks are split length-wise, characteristic symptoms include the internal shredding of the vascular tissue, which is ash-grey in colour and flecked with small “pepper-like” black microclerotia, the resting bodies of the pathogen (*M. phaseolina*). The micro sclerot es survive in the soil and on the stubble of over 400 crop and weed hosts for a significant length of time (4+ years). Due to its endemic nature and wide host range, management options are limited.

A collaborative GRDC research project between the South Australian Research Institute (SARDI) and USQ aimed to develop a molecular PREDICTA B test for the identification of *Macrophomina* from soil and stubble samples to determine the incidence and severity of the pathogen across the regions. The test, which has already been developed by SARDI, is now under field evaluation to develop appropriate sampling strategies for the test and to determine if inoculum loadings found in soil and stubble can be correlated with end of season disease levels found in a mature crop. If so, this will provide growers with a risk assessment for potential disease likelihood prior to planting.

This season (2016/17 summer season), thirty-two sorghum fields were sampled from across the south-east and central Queensland regions. At each field, three to five sampling strategies were evaluated, depending on the stubble management system employed by the grower:

The sampling strategies include:

1. Soil collected from previous crop row with no (0) stubble pieces
2. Soil collected from previous crop row with 15 stubble pieces
3. Soil collected from previous crop row with 30 stubble pieces
4. Soil collected from off the plant row
5. Soil collected from off the plant row with 30 pieces of stubble from weeds or previous crops at the location.

Data pertaining to location, crop history, stubble management, previous disease occurrences were also recorded. Samples were analysed by SARDI, and growers who participated were provided PREDICTA B reports which included results for a number of pathogens and diseases from commercially available tests including; (*Pratylenchus neglectus* and *Pratylenchus thornei* -root lesion nematodes, crown rot, take-all) and tests under evaluation (*Macrophomina*, common root rot, yellow spot, *Pythium* clade F and *Fusarium graminearum / culmorum*).

At the end of the growing season, all sites were re-visited and on-ground disease assessments of sorghum crops were conducted at each sample location. These data were combined with PREDICTA B results for further analysis. In central QLD, in particular, very few sorghum crops were planted this summer (2016/17) due to a large 2016 chickpea planting, low sorghum prices and little or no planting rain in January 2017. Despite the hot and dry conditions experienced, very little charcoal rot developed at the sampling sites. In central Queensland, the low level of charcoal rot was probably due to in-crop rainfall from ex-cyclone Debbie, which provided adequate follow up rain. In south east Queensland, particularly across the Darling Downs, crops experienced hot drought-like conditions and very little follow up rain after planting. The dry conditions limited crop yield and it is
likely that the low yielding crops were less impacted by charcoal rot at maturity than if yields had been higher.

Early results from soil and stubble samples analysed by SARDI confirmed the endemic nature of the pathogen, with *Macrophomina* being detected at every site sampled. Only 7% of samples had low population densities, while 66% of samples had high *Macrophomina* population densities (Figure 3). Correlations between these population densities and risk of disease could not be completed this season due to lack of disease pressure, however this work will be continued next summer (2017/18).

Differences in the *Macrophomina* population densities were also observed between sampling strategies, with higher levels of *Macrophomina* found in soil+stubble treatments (sampling strategies 2, 3 and 5), compared with soil only treatments (sampling strategies 1 and 4) (Figure 4). Samples containing stubble, taken off the plant row (sampling strategy 5), also had similar high levels of *Macrophomina* DNA compared with sampling on previous crop row with added stubble (sampling strategies 2 and 3). These preliminary results demonstrate that despite the soil borne nature of *Macrophomina*, stubble contributes substantially to the pathogen population.

![Figure 3. Percentage of field samples where *Macrophomina* could not be detected or that have *Macrophomina* population densities which are classified as “Low”, “Medium” or “High” (categories are devised by SARDI).](image_url)
**Fusarium stalk rot**

As with charcoal rot, diagnostic symptoms of fusarium stalk rot are seen by splitting sorghum stalks length wise. Infected tissue is red-brown in colour and the internal pith is often shredded in appearance. Symptoms are usually first observed at the crown of the plant spreading up the stalk as the disease progresses. Although the most obvious symptoms are expressed within the stalk, *F. andiyazi* and *F. thapsinum* can colonise all above ground plant parts (seed, stalk and leaf) and are commonly found in the roots. Once the crop is harvested, the pathogens survive on colonised sorghum residue, which act as an inoculum reservoir for future sorghum crops. *Fusarium* species are well known for their ability to colonise and survive saprophytically on dead stubble residue. Studies investigating the length of survival of *F. thapsinum* on sorghum stubble in Queensland are ongoing, with preliminary results being reported in this paper.

A field trial was established at Hermitage Research Station, Warwick, Queensland in 2014. The aim of this trial was to determine the length of survival of *F. thapsinum* on colonised sorghum residue and identify the implications of different stubble management practices (incorporated, surface, standing) on the survival of this pathogen. A second trial with identical treatments and aims was established at Kingaroy Research Station in 2015. Results presented in this paper are based on the trial at Hermitage Research Station only.

Treatments included: (i) standing un-inoculated stubble, (ii) standing inoculated stubble, (iii) surface un-inoculated stubble, (iv) surface inoculated stubble, (v) buried un-inoculated stubble, and (vi) buried inoculated stubble. The sorghum genotype IS8525 (known to be susceptible to *Fusarium* infection) was established at the Hermitage site and plants in the inoculated treatments were stalk-injected with a *F. thapsinum* spore suspension two weeks after flowering. At plant maturity, stalks from inoculated and un-inoculated plants were harvested, cut into 15 cm-long sections and the stubble treatments set up accordingly. Plants in standing stubble treatments were cut to a height of 60 cm, surface stubble was spread evenly across the soil surface of each plot and covered with netting to avoid loss of stubble, and buried stubble pieces were placed into mesh bags and buried between 5 and 10 cm deep.
Sorghum stubble was collected at 0 months (04/2015), 2 months (30/06/2015), 6 months (02/11/2015), 12 months (15/04/2016), 18 months (05/09/2016) and 24 months (06/04/2016). Stubble dry weights were recorded at each sampling date to assess the level of residue decomposition over time. Isolations were conducted on each removed stubble piece and *Fusarium* species isolated from the stubble pieces were identified using morphological and molecular techniques. Soil moisture and soil temperature data were collected during the trial period using dataloggers.

Stubble weight data showed that the stubble deteriorated rapidly within the first two months after harvest (36-70% weight loss). Surface stubble treatments deteriorated the most (62-70% reduction), while in the buried treatments the reduction in weight was much lower (36-40%) after two months. It is possible stubble from the surface treatments declined faster due to drying out quicker than the buried stalks. After six months the weight reduction in stubble was similar (71-80%) irrespective of the treatment. Twelve (12) months after the start of the trial the buried stubble showed the greatest deterioration (87.5%), followed by surface stubble (80.2%) with standing stubble having the lowest (74.7%) (Figure 5).

![Stubble weight loss over time at six sampling times](image)

**Figure 5.** Stubble weight loss over time at six sampling times; 0 months, 2 months, 6 months, 12 months, 18 months and 24 months after harvest

Preliminary results of isolations of *F. thapsinum* from inoculated stubble treatments indicate that after 2 months *F. thapsinum* levels had declined across all treatments (buried, standing and surface stubble) (Figure 6). At 12 months, the rate of recovery of *F. thapsinum* was lower in buried (33.8%) and surface treatments (42.5%), compared with standing stubble (74.5%). However, by 18 months, differences between treatments appear to have reduced, with *F. thapsinum* recovered the least from buried stubble (31.5%), followed by surface stubble (35.5%) and then standing stubble (40%).

These preliminary results suggest that under the weather conditions which occurred during the trial approximately one third of the initial *F. thapsinum* inoculum remained in the stubble after two years irrespective of the management of the stubble. The interrelationships between inoculum levels, fusarium stalk rot severity and weather is still unclear and needs to be investigated. The development of a molecular PREDICTA B test for *F. thapsinum* and *F. andiyazi* is underway, which
may provide an indication of future disease risk, although the role the weather plays in these outbreaks is significant and cannot be ignored or understated.

**Figure 6.** Recovery of *F. thapsinum* from inoculated stubble treatments over time. Note: 24 month data not presented.

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