Fusarium head blight at low levels in the northern grains region in 2016 – cause and implications

Steven Simpfendorfer[1], Daniele Giblot-Ducray[2], Diana Hartley[3] and Alan McKay[2]
[1]NSW DPI, Tamworth
[2]SARDI, Adelaide
[3]CSIRO National Collections and Marine Infrastructure, Canberra

Key findings

- A survey of 80 winter cereal crops across central and northern NSW in 2016 established that low levels (generally <1%) of Fusarium head blight (FHB) were evident with the causal pathogen predominantly being *Fusarium pseudograminearum* (*Fp*).
- This was the crown rot fungus (*Fp*), reminding growers that it does not disappear in a wet season.
- FHB infection caused by *Fp* has a reduced risk for mycotoxin accumulation in infected grain, but could have detrimental impacts on crop establishment if seed is retained for planting.
- Planting *Fusarium*-infected grain can also introduce seed-borne crown rot infection into clean paddocks, negating the rotational benefits associated with growing non-host crops.

Introduction

Above-average rainfall was experienced in many parts of northern NSW in the 2016 winter cropping season. While this was a major driver for increasing crop yield, unfortunately these conditions also favoured the development of a range of diseases. Of particular concern were low levels of head infections in durum and bread wheat crops.

A survey of symptomatic heads and grain samples was conducted in 2016 to determine the various causes and to address concerns around fusarium head blight (FHB) infections. FHB relates to the symptoms of head infection resulting in premature ripening of infected spikelets, generally caused by two fungi *F. graminearum* or *F. pseudograminearum*, following wet weather during flowering and/or grain-fill. White grain disorder, caused by *Eutiarosporella* spp. (formerly Botryosphaeria), produces similar visual symptoms that are not easily distinguished from FHB. These diseases are not uncommon in the northern grains region, with the last widespread occurrence in northern NSW and southern Qld in 2010.

NSW DPI conducted a similar study in 2010 with implications for mycotoxin production based on identification of causal species, issues with sowing infected grain and the potential role of seed treatments investigated. Some of this information will be presented here, as it is still relevant to the situation that occurred in 2016.

Survey details

NSW DPI, with assistance from agronomists and growers, conducted a survey of wheat crops with visible head infections during grain filling to determine the causal fungi. Head and grain symptoms were consistent with either Fusarium head blight or white grain disorder, so laboratory techniques concentrated on recovering these causal pathogens. Representative isolates collected from symptomatic heads or grain were identified to the species level using molecular techniques. Determining the exact causal pathogen has potential consequences for the risk of mycotoxin contamination and end use of affected grain.

Results

Head or grain samples were collected from a total of 80 paddocks from central and northern NSW in 2016 and causal pathogens identified to species. In 66% of cases, FHB was caused by *F. pseudograminearum* (*Fp*) only, 4% by *Fusarium graminearum* (*Fg*) only, 19% were a mixed infection of *Fp* + *Fg*, and 1% (one paddock) had a mixed infection from *Fp* and *F. cerealis* (*Fcer*)(Figure 1).

A total of 4% paddocks had white grain disorder with recovery of Eutiarosporella (*Eut*) only, with a further 4% having a mixed infection of Fp + Eut and 2% (two paddocks) having mixed infection by *Fp* + *Fg* + *Eut* (Figure 1).

Given the increased susceptibility of durum wheat to Fusarium infection, both FHB and crown rot, there was a slight dominance of samples coming from durum crops (54% of paddocks), but symptoms were also evident in many bread-wheat paddocks.

Fusarium pseudograminearum (*Fp*) is the main species usually causing crown rot. Hence, it appears that the low levels of FHB in 66% of paddocks surveyed in 2016 have come from *Fp*-producing spore masses (macroconidia) on the lowest nodes of tillers infected with crown rot. Rain-splash then disperses these spores up the canopy to infect heads at flowering and causes low levels of FHB symptoms in a wet year.

There are two other main species of Fusarium that can cause FHB: *F. graminearum* (*Fg*) and *F. culmorum* (*Fc*). *F. graminearum* has more commonly been associated with FHB in the northern region and has a life stage (perithecia) that is produced on maize, sorghum, grass weeds and winter cereals. The perithecia are full of smaller spores called ascospores, which are air-borne and hence more easily dispersed into wheat heads during flowering than macroconidia.

Fortunately, *Fp* does not readily produce perithecia in the paddock and is not hosted on maize or sorghum. Hence, it lacks an air-borne ascospore stage, which are easily dispersed into heads. A total of 23% of paddocks had FHB infection associated with *Fg*, which was most commonly in a mixed infection with *Fp*. Although *Fc* was not identified in any of the collected samples, another species, *F. cerealis* was identified in a durum sample from Terry Hie Hie in a mixed infection with *Fp* (Figure 1).

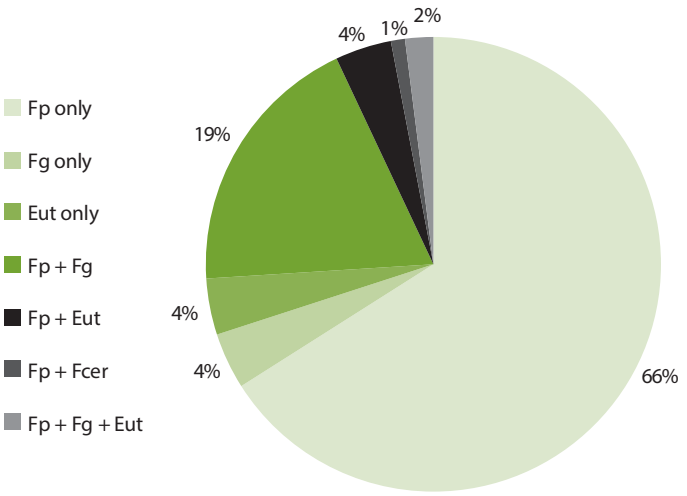

Figure 1. Fungal species associated with head infections in northern NSW in 2016. *Fp = Fusarium pseudograminearum, Fg = Fusarium graminearum, Eut = Eutiarosporella, Fcer = Fusarium cerealis*. Values represent the percentage of the 80 paddocks surveyed from which each pathogen was isolated.

Why is identifying the exact causal pathogen important?

Frequent rainfall in winter while cereals are flowering favours FHB infection. On these occasions, if it is *Fp* causing the infection, then the incidence is usually considerably lower than if *Fg* is the causal pathogen. This is due to that lack of an air-borne spore stage (ascospore) with *Fp*, with prolonged wet weather required for *Fp* to first produce spore masses (macroconidia) around lower nodes on infected stems. The macroconidia, although still microscopic, are considerably heavier than ascospores so they require splash dispersal to infect heads during flowering thus limiting their dispersal. In contrast, the ascospore stage in the *Fg* life cycle is not as reliant on moisture for initial maturation of perithecia, which contain the air-borne ascopsores. Rainfall during flowering is simply then required for the ascospores to be released, which wind then more readily disperses into heads during flowering. This was consistent with the very low incidence of infected heads in paddocks in 2016 with most having well below 1% of heads visually infected.

Identification to species level also has implications for potential mycotoxin issues within infected grain. The main mycotoxins produced by Fusarium are deoxynivalenol (DON) and nivalenol (NIV), with NIV being around 10 times more toxic than DON. DON is commonly called vomitoxin in the USA, with regulated limits of 1 ppm (1 mg/kg) in grain for human consumption, 5 ppm for pig feed and 10 ppm for beef/sheep/poultry feed. With *Fg*, DON

levels are closely linked to the incidence of visually infected white and pink grains at harvest as mycotoxins are concentrated in these damaged seeds (Sinha & Savard, 1997).

However, grain infected with *Fp* has been shown to accumulate much lower mycotoxin levels than that infected with *Fg* under laboratory conditions (Blaney & Dodman, 2002). This is supported by field sample analysis from a previous occurrence of FHB in Australia in 1984 with Burgess et al. (1987) finding that grain with 38% *Fp* infection only accumulated 0.6 ppm of DON. There are also two different forms (chemotypes) of DON, with 3ADON being half as toxic as the 15ADON form.

Similar research conducted by NSW DPI following an outbreak of FHB in northern NSW and southern Qld in 2010 determined that 92% of 137 *Fp* isolates examined were the 3ADON chemotype, 1.5% were 15ADON, 6.5% were a combination of 15 + 3ADON and none were NIV producers. In contrast, 93% of the 88 *Fg* isolates examined were 15ADON, 3.5% were 3ADON and 3.5% were the NIV chemotype.

Hence, determining which species of Fusarium is causing FHB is important as *Fg* generally produces larger quantities of more toxic forms of mycotoxins (NIV and 15ADON). Conversely, *Fp*, the main cause of FHB in 2010 and again in 2016 in this region, produces considerably lower quantities of a less toxic form of DON (3ADON) only. This has serious implications for sale and end use of the grain produced in these paddocks.

Eutiarosporella spp. (white grain disorder), also causes a head infection with symptoms appearing as premature bleaching of spikelets and production of white grains. These symptoms are hard to distinguish from FHB. However, it has been shown that there are no mycotoxins associated with this pathogen and that grain infected with Eutiarosporella caused no issues when fed to weaner pigs for four weeks (Kopinski & Blaney, 2010). Hence, distinguishing Eutiarosporella infection from FHB has important consequences for the potential end use of affected grain.

Are there issues of retaining Fusarium-infected seed for sowing in 2017?

Grain infected with Fusarium when sown in the following year can cause seedling death, which reduces emergence. Crown rot infection can also be introduced to the base of surviving healthy plants as infected grain is also an inoculum source. Grain infected with Fusarium only occurs as a result of FHB, which is favoured by wet conditions during flowering. Basal crown rot infection alone cannot directly result in grain infection, as the fungus does not grow up the entire stem and into heads within a season.

Additional experimental work at Tamworth in 2011 investigated the effect of grain infection with Fusarium on emergence, and subsequent crown rot infection in surviving plants (seed-borne crown rot infection). Four seed lots naturally infected with varying levels of Fusarium (19–73%) during an outbreak of FHB in 2010 were used in the study.

Grain infected with Fusarium had lower emergence (15–55%) as it caused severe infection in the seedlings and many died, which is commonly called seedling blight. However, the experiment also showed that plants which survived past the seedling-blight stage had also been infected with high levels of crown rot (average 35%). Seed-borne crown rot affects yield in the current crop and introduces infected stubble back into the paddock. Sowing Fusarium-infected seed, therefore, undoes any break-crop benefits that might have been obtained from growing non-host crops (such as chickpea, canola, faba bean, sorghum) in the previous season.

Some seed treatments were shown to improve emergence of Fusarium-infected grain by 10–30%, but had a limited effect on reducing levels of seed-borne crown rot in surviving plants. Ideally, growers should plant wheat seed that is free of Fusarium infection by targeting crops that were not infected with FHB. Grain infected with FHB is usually white and, if prolonged wet conditions occurred during grain-fill, infected grains will take on a pink appearance. However, it should be noted that if any white or pink grains are evident, then the levels of Fusarium infection can be significantly higher than that indicated by visual inspection – laboratory testing is recommended. This is because FHB infections that occur later during grain-fill might not cause any visual seed discolouration.

Conclusions

The low levels of FHB that occurred in bread wheat and durum crops across central and northern NSW in 2016 were predominantly related to *Fp* infection. These infections arose from spore masses produced around lower nodes of crown rot-infected tillers, which were then rain-splashed into heads during flowering.

Mild conditions during spring prevent the expression of crown rot as whiteheads as water supply to the developing head is not limited. Consequently, crown rot infections often go unnoticed in wetter years. The low levels of FHB evident in 2016 could be viewed as the crown rot fungus (*Fp*) reminding growers that it does not go away in a wet season.

Fortunately in 2016, the generally low incidence of FHB infection only resulted in a few instances of issues with harvested grain quality. Hence, the overall economic impact of FHB was relatively minor in 2016. However, if spring conditions in 2016 had been more stressed with limited rainfall and warmer temperatures during grain filling, then significant and widespread losses to crown rot are likely to have occurred. Growers should not be complacent about potential crown rot inoculum levels in 2017.

Avoid sowing winter cereals into paddocks that had FHB in the previous season as they are likely to represent a high risk for crown rot infection for the following year. All durum wheat varieties have increased susceptibility to Fusarium infection, both FHB and crown rot, hence durum production should be targeted to low-risk paddocks, preferably based on stubble or PreDicta B testing.

Growers who noticed or suspect that they had FHB or white grain disorder should get their planting seed tested to determine infection levels before sowing that seed in the next season.

This information can be used to guide appropriate seed treatment options or to source alternative cleaner seed with lower infection levels if required. This should be the preferred option compared with sowing seed with unknown Fusarium levels which, if moderately infected, will result in poor establishment and introduce significant crown rot levels into paddocks. This will compromise rotational benefits that might have been achieved by previously growing non-host crops.

References

Blaney BJ & Dodman RL (2002). *Australian Journal of Agricultural Research* 53: 1317–1326.

Burgess LW, Klein TA, Bryden WL & Tobin NF (1987). *Australasian Plant Pathology* 16: 72–78.

Kopinski & Blaney BJ (2010). *Journal of Animal Physiology & Animal Nutrition* 94: 44–54

Sinha RC & Savard ME (1997). Canadian Journal of Plant Pathology 19: 8–12

DON (vomitoxin) in wheat: Basic questions and answers (2005). PP-1302. North Dakota State University, Fargo, North Dakota 58105, USA. https://www.ag.ndsu.edu/pubs/plantsci/pests/pp1302.pdf

Acknowledgements

This experiment was part of the project *Northern NSW integrated disease management* (DAN00176; 2013–18), with joint investment by NSW DPI and GRDC. Assistance from growers and agronomists in collecting samples is greatly appreciated. Technical assistance provided by Karen Cassin and Rachel Bannister is gratefully acknowledged.