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Integrated disease management of wheat and barley

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Diseases affecting wheat: tan spot

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1 Introduction

Tan spot is an economically important disease of wheat worldwide and is found throughout the major wheat-growing areas of the world. This foliar fungal disease is a challenge to wheat production as it reduces green leaf area, restricting photosynthesis and subsequently the ability to fill grain and set yield. Initially, leaf lesions appear as small tan-coloured brown flecks, which expand into oval or diamond-shaped spots, often with a yellow chlorotic margin (Fig. 1a). As the disease progresses, these spots increase in size, forming large blotches and irregular areas of dead tissue. A small dark brown eyespot near the centre of the lesion is common (Fig. 1b). The disease is particularly severe on older leaves, and entire leaves, spikes and whole plants may also be affected.

The causal agent is the necrotrophic fungal pathogen, *Pyrenophora tritici-repentis* (Died.) Drechs. [anamorph: *Drechslera tritici-repentis* (Died.) Shoem. (sym. *Helminthosporium tritici-repentis* (Died)]. *P. tritici-repentis* is commonly abbreviated to Ptr (or Dtr in some areas) and is a filamentous ascomycete fungus belonging to the class Dothideomycetes and order Pleosporales. Though initially described as an occasional wheat pathogen, the incidence of tan disease has increased markedly in recent times, and the pathogen exhibits a wide geographic range. Countries affected include those in North America (Canada, the USA and Mexico) (Ali and Francl, 2003; Singh and Hughes, 2006; Lamari et al., 1998; Friesen et al., 2005); South America (Colombia, Ecuador, Peru, Brazil, Uruguay and Argentina) (Gurung et al., 2013; Dubin, 1983; Gamba et al., 2012; dos Santos et al., 2002; Moreno et al., 2008); North Africa (Morocco, Algeria and Tunisia) (Lepoint et al., 2010; Cherif et al., 1994; Lamari et al., 1995); Europe (UK, Belgium, Luxembourg, Germany, Denmark, Czechia, Slovakia, Hungary, Poland, Romania, Bulgaria Lithuania, Latvia, Sweden and Finland) (Todorova, 2006; Sidrat et al., 2016; Abdullah et al., 2017; Gurung et al., 2013; Hudcovicova et al., 2015;

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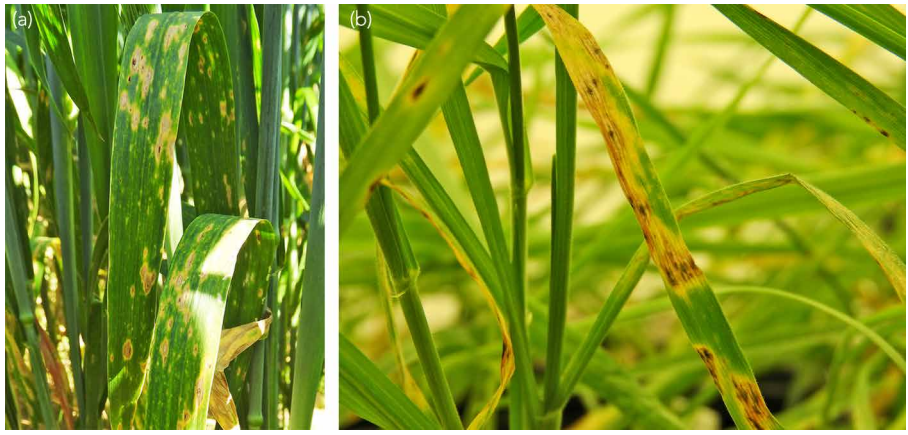

Figure 1 (a) Typical early tan spot symptoms showing necrotic leaf lesions surrounded by chlorosis. (b) Dark eyespots visible within lesions.

Sierotzki et al., 2007; Cook and Yarham, 1989; Jalli et al., 2011; Leisova et al., 2008; Lepoint et al., 2010); Asia (Turkey, Syria, Georgia, Armenia, Azerbaijan, Russia, Iran, Turkmenistan, Uzbekistan, Tajikistan, Kyrgyzstan, Kazakhstan, Pakistan, India, Nepal, Bangladesh and Japan) (Singh, 2007; Ali and Francl, 2001; Gurung et al., 2013; Aboukhaddour et al., 2011; Momeni et al., 2014; Lamari et al., 2003, 2005; Mironenko et al., 2016; Lepoint et al., 2010) and Australasia (Australia and New Zealand) (Murray and Brennan, 2009; Hampton and Matthews, 1978). Both bread wheat (*Triticum aestivum*) and durum wheat (*T. turgidum*) are susceptible to tan spot. Furthermore, Ptr has been isolated from a number of other species including barley (*Hordeum vulgare*), oat (*Avena sativa*) and rye (*Secale cereale*), as well as other grasses which may serve as sources of inoculum. Initial infections early in the season originate from affected grass hosts or diseased crop residue in the soil, with symptoms usually visible five days after infection.

The tan spot disease cycle consists of both sexual and asexual stages. Ptr is able to survive saprophytically on wheat stubble and straw, upon which small, black, raised fruiting bodies (pseudothecia) containing sexual ascospores develop and mature (Fig. 2). These are commonly observed around the stem nodes. Ascospores are present throughout the growing season and serve as the primary inoculum infecting the lower leaves, particularly during tillering and early jointing. The lower, more mature leaves are usually infected first (Fig. 3), with disease radiating to the upper leaves and leaf sheaths when conditions are favourable. Infection occurs over a wide temperature range, with a longer wet period required during cold conditions. Free moisture on the leaf surface is crucial for infection. Under these damp conditions, asexual spores (conidia), with their distinct snakehead-like appearance, form within mature leaf lesions and act as a secondary inoculum, sustaining disease throughout the growing season. Rain and wind disperse the spores and spread the disease within fields and beyond. Although leaf infections are most common, seed infections can also occur usually following prolonged wet periods that promote disease during grain development. Infected seeds exhibit a red discoloured seed coat (red smudge) that will usually downgrade market grain.

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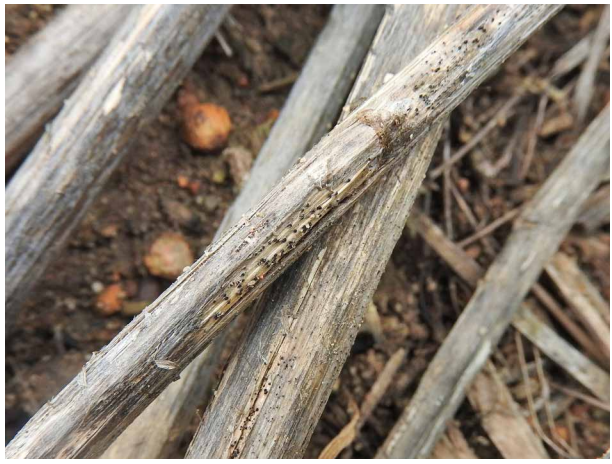

Figure 2 Pseudothecia on wheat stubble.

Tan spot is a major constraint to maintaining wheat yield and quality, resulting in shrivelled grain and lower test weights. Yield losses of up to 50% can occur under exceptionally wet seasons, although losses of around 15% are more common, with the effect on yield greatest during grain fill, particularly if the upper leaves are affected.

2 Disease management considerations

A number of strategies are available for the management of tan spot, consisting of cultural modifications, chemical control and wheat variety genetics. However, a single strategy rarely provides total protection, and growers need to consider a combination of strategies to provide effective and consistent long-term management.

2.1 Cultural practices

Prior to seeding, disease information from previous seasons should be assessed and consideration be given to growing an alternative crop or variety, or using a different field with a lower disease risk. Field location, topography and prevailing wind direction should also be taken into consideration, as sowing downwind from a field with a history of tan spot may present a high disease risk since condia can travel up to 200 km (De Wolf et al., 1998). Maintaining quality pathogen-free seed with good germination and vigour is also an important part of a holistic disease management programme.

Tan spot has gained more importance with the increased adoption of minimum and no till agronomic practices which has coincided with disease outbreaks (Ciuffetti et al., 2014). Therefore, the management of crop debris is an effective means to limit tan spot by eliminating potential sources of primary inoculum. Practices that reduce stubble density, such as tillage, burning or grazing, will reduce the level of inoculum, but must be balanced against the increased risk of soil erosion and reduction in soil moisture.

Figure 3 Characteristic tan spot infection, with the lower leaves most severely affected.

Stubble management will not, however, reduce disease caused by spores blown in from surrounding fields.

Another effective strategy is to diversify crops through rotation, which helps to break disease cycles and enable an overall rise in crop productivity. Monoculture of continuous wheat or wheat–fallow–wheat put the crop at an increased risk of infection, yet economic considerations tempt growers to restrict rotations and perpetuate the disease.

2.2 Chemical control

Fungicide application can be used to manage tan spot when environmental conditions favour disease development, particularly on susceptible varieties. However, prior to any fungicide use, it is essential to first ensure that observed symptoms are indeed due to tan spot and not caused by other factors such as nutritional deficiencies, environmental stress or physiological yellowing (Moffat et al., 2015). Seed treatment with a registered fungicide can address potential early season disease problems, particularly under continuous wheat

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production or if a susceptible variety is grown. Early crop scouting combined with readily available weather information can be indicative of whether an in-crop application of fungicide is warranted. The overall incidence of disease is strongly influenced by rainfall. If forecasts suggest that prolonged wet conditions will occur, a spray may provide up to three weeks of protection. However, little benefit will be gained from spraying when conditions are warm and dry.

Proper timing of fungicide application is essential to maximise yield and grain weight. A high disease pressure early in the season may require spraying between early tillering and stem elongation. This may be followed up by a second spray after flag leaf emergence, particularly when conditions are wet. For mid- to late-season disease, a single spray between flag leaf emergence and head emergence is generally the most profitable. Where there is minimal rainfall, fungicide applied after flag-leaf emergence is unlikely to provide economic returns.

2.3 Wheat variety choice

Another strategy for managing tan spot is to utilise variety resistance. Variety choice has a major influence on disease development with tan spot resistance ratings varying widely. Wheat breeding entities continue to release improved varieties that combine enhanced agronomic, quality and disease traits. However, highly resistant commercial varieties are not readily available and continue to be a breeding priority. Thus, varietal improvement offers the best approach for a long-term solution to yield stability. Increased germplasm resistance also reduces the use of fungicides which are costly, time-consuming and potentially hazardous. When choosing a variety, growers should aim to sow those with the best available disease resistance package, while considering adaptation to local growing conditions as well as agronomic and marketing requirements.

2.4 Case study: management of tan spot in Brazil

In Brazil approximately 6 million tonnes of wheat is grown over 2 million hectares, with around 90% of the production from the southern region (the states of Paraná, Santa Catarina and Rio Grande do Sul) (CONAB, 2017). Yields typically are between 2500 kg/ha and 3000 kg/ha. Losses due to tan spot have been reported as high as 48% (Reis and Casa, 2007), and it is estimated that for every 1% increase in tan spot incidence, yield can be reduced by 17 kg/ha (Reis et al., 2016).

For over 20 years, a no-till management system has been in place in Brazil to maximise soil conservation. Stubble is left in place and allowed to decompose, thus promoting the supply of organic material to the soil. Growers are urged to apply crop rotation, mainly soybean and maize during the summer, and wheat, oat, barley, triticale and canola during the winter. However, not all growers follow this, and consequently severe outbreaks of tan spot do occur, more frequently where wheat is grown in monoculture.

The application of chemicals is widely practised for tan spot control in Brazil, with growers applying fungicides, most typically triazoles and strobilurins, at least once or twice a season from tillering onwards. In Paraná, local practice is to spray fungicide up to five times a season when considering all possible diseases (Santana, unpublished). The threshold for application at the elongation stage is around 6%, meaning that fungicide should be applied before a 6% disease incidence in order to be most effective. Despite this, growers have started to report lower fungicide efficacy and tan spot appears to be becoming more problematic.

In terms of germplasm, currently there are over 100 commercially available wheat varieties in Brazil, which exhibit resistance to a wide range of diseases. Breeders based at Embrapa Trigo have developed and released over 30 wheat varieties, of which 15 are moderately resistant to tan spot. However, no molecular markers for tan spot resistance have yet been incorporated, and Brazilian variety selection follows a more traditional method via natural infection through field trials.

3 The host–pathogen interaction

Necrosis and chlorosis are two distinct symptoms of tan spot and result from highly specialised interactions between Ptr and the wheat host. Ptr is able to secrete necrotrophic effectors (also called host-selective toxins) that play a primary role in the development of disease, by inducing plant cell death and enabling fungal colonisation (Tan et al., 2010). So far, three Ptr effectors have been described: ToxA, ToxB and ToxC. ToxA has been particularly well studied and is responsible for the tan-coloured necrosis on sensitive wheat varieties, while ToxB and ToxC cause the yellowing chlorosis.

Just as wheat varieties differ in their susceptibility to tan spot, Ptr isolates (or strains) differ in their pathogenicity. Individual isolates of Ptr are currently classified into one of eight races based on their ability to produce the three effectors, either alone or in combination (Table 1). Races 1 and 2 appear to be the most predominant races, while races 3 and 4 are found at lower frequency. Race 5 has a relatively wide distribution, while races 6, 7 and 8 are comparatively rare.

ToxA appears to be the most prevalent Ptr effector worldwide and is rapidly internalised into mesophyll cells of sensitive wheat genotypes, where it localises to the chloroplast. Reduced photosynthesis follows, via the light-dependent accumulation of reactive oxygen species (ROS) and photosystem disruption (Pandelova et al., 2012). Cell death is rapid and has been detected as early as 9 h following ToxA treatment. Like ToxA, the chlorosis induced by ToxB is also light-dependent and involves chloroplast damage, ROS accumulation and inhibition of photosynthesis. However, ToxB-induced cell death is slower than that of ToxA, occurring after 24 h and does not eventuate in tissue collapse (Pandelova et al., 2012).

Table 1 The current race classification system of Ptr isolates is based on presence or absence of ToxA, ToxB or ToxC

Race	Effectors
1	AC
2	A
3	C
4	–
5	B
6	BC
7	AB
8	ABC

ToxC remains to be characterised with little information currently known and is an obvious target for researchers. It is thought to be a metabolite and has been described as a low-molecular weight, polar and non-ionic molecule (Effertz et al., 2002). Since ToxA and ToxB are both proteins and products of identified genes, Ptr isolates can be tested for their presence/absence through simple molecular detection (PCR). However, currently the lack of an identified biosynthetic pathway for ToxC excludes PCR-based screening. Instead, the presence of ToxC in Ptr isolates of interest relies on spore inoculation of a particular differential wheat line (6B365). The elucidation of ToxC through metabolome-based studies will undoubtedly facilitate significant advances in tan spot research.

Currently, Ptr isolate races are determined by infection of a set of differential wheat lines that are sensitive to specific effectors. These are Glenlea (ToxA sensitive), 6B662 (ToxB sensitive), Katepwa (ToxA and ToxB sensitive), 6B365 (ToxC sensitive) and Auburn or Salamouni (insensitive to all three effectors). However, the ability to detect new races is limited by the effectiveness of the differential lines, and expansion of the differential varieties will facilitate the identification of new races. Indeed, there have been a number of reports of additional unidentified effectors that extend beyond the eight-race model and are the focus of ongoing research efforts (Moffat et al., 2014; Manning and Ciuffetti, 2015).

A number of studies have also examined the genetic diversity in Ptr populations. These have employed various molecular markers to assess regional or global collections of isolates and have revealed high population differentiation and genetic variability. This is further supported by comparative genomics which is suggestive of high levels of genome plasticity among isolates (Moolhuijzen et al., In Press).

The Ptr genome of a North American race 1 isolate was sequenced several years ago, and its release marked a significant evolution in tan spot research (Manning et al., 2013). However, extraordinary progress has been made in genome sequencing technologies in the years since, and the focus of current research will be to release new high-quality reference genomes that can resolve whole chromosomes. Capturing genomes of different races from diverse geographic regions will be important in the identification and isolation of novel effectors.

4 The genetics of host resistance

Dominant host sensitivity genes confer sensitivity to Ptr effectors and are considered as susceptibility loci as they are highly correlated with disease. The single dominant gene *Tsn1* (*Tan spot necrosis 1*) located on the long arm of chromosome 5B confers sensitivity to ToxA (Faris et al., 2010). Similarly, a single dominant gene *Tsc2* (*Tan spot chlorosis 2*) confers sensitivity to ToxB, on the short arm of chromosome 2B but is yet to be cloned. *Tsc1* confers sensitivity to ToxC; however, the *Tsc1*–ToxC interaction appears to be more complex. A quantitative trait locus (QTL) on the short arm of chromosome 1A has been associated with race 3-induced chlorosis (ToxC) and partially purified ToxC (Liu et al., 2017; Effertz et al., 2002); yet the mode of inheritance of sensitivity/insensitivity to ToxC remains to be determined. Identification of the sensitivity genes for both ToxB and ToxC with the release of perfect markers remains a high priority for researchers.

Numerous reports of additional QTLs with effects ranging from minor to significant, as well as QTLs for race-nonspecific resistance/susceptibility (Faris et al., 2013) exist. Many of these have been detected via plant infection assays and are not yet associated with

reaction to specific effectors. The recent development of genetic manipulation tools in Ptr is a game changer, allowing researchers to manipulate and delete genes in Ptr in the hunt for novel effectors. For example, removal of the ToxA gene means researchers can tap into ToxA-sensitive wheat mapping populations to uncover QTLs, where previously ToxA-induced necrosis masked any additional symptoms (Moffat et al., 2014; Manning and Ciuffetti, 2015). As more evidence emerges it is becoming apparent that the genetics of tan spot resistance/susceptibility is more complicated than first thought, with both major and minor genes likely to be involved.

5 Case study: ToxA-assisted breeding for tan spot resistance in Australia

In Australia, annual wheat production is around 24 million tonnes, with approximately 12 million hectares under cultivation for wheat. Tan spot (also called yellow spot in Australia) is arguably the most costly wheat disease to growers, with yield losses estimated at $212 million (AUD) per annum, and annual control costs of $463 million (AUD) (Murray and Brennan, 2009). Wheat breeding programmes here have enthusiastically adopted an effector-based germplasm screening approach, in addition to the conventional use of molecular markers through marker-assisted selection. This exploitation of fungal effectors as functional markers has proven to be beneficial in selecting for disease resistance, particularly in the case of ToxA.

Insensitivity to ToxA generally correlates with higher levels of tan spot resistance in Australian wheat varieties (See et al., 2018). ToxA-sensitive varieties are consistently rated as very susceptible (VS) to moderately susceptible/susceptible (MSS), while insensitive varieties are found in the moderately susceptible (MS) to moderately resistant (MR) range, with only a handful of exceptions. Thus, a priority for breeders has been the elimination of the *Tsn1* sensitivity gene, since sensitivity to ToxA is sufficient to determine significant disease susceptibility.

ToxA has been synthetically produced via a microbial expression system and delivered to Australian wheat breeders since 2009, with many thousands of doses dispatched annually. By simply infiltrating wheat leaves with ToxA, breeders can quickly determine which lines are sensitive (i.e. necrotic) (Fig. 4). These leaf infiltration effector assays are flexible and can be carried out in glasshouses and field plots, on seedlings and adult plants. One breeder is able to test several thousand plants in a single day and obtain clear results within a few days (Oliver et al., 2016). Those lines identified as sensitive are likely to be susceptible to tan spot (and septoria nodorum blotch which also produces ToxA) and can be removed early to streamline the variety selection process. Effector-assisted screening can be undertaken at any stage in the breeding process, enabling screening for disease susceptibility in varieties almost ready for release, as well as in new crosses still early in the development pipeline. It offers substantial advantages over field testing approaches, saving time, space and resources. Furthermore, a wheat variety trial over two years and four locations in the Western Australian wheat belt found that no yield penalties were observed in ToxA-insensitive varieties, suggesting that the absence of the *Tsn1* gene has no obvious downside in Australian conditions (Oliver et al., 2014).

The uptake of effector-assisted selection by breeders in Australia has been rapid and the impact can already be seen. Between the 2009/10 and 2015/16 seasons, the area in Western Australia sown with ToxA-sensitive varieties was reduced by approximately

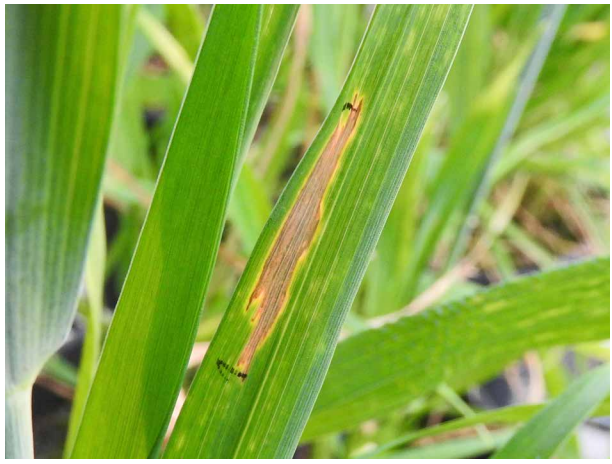

Figure 4 ToxA leaf infiltration of a sensitive wheat variety showing necrosis one week after infiltration.

1.4 million hectares from 37.5% to 8.3%. Based on a typical yield reduction of 0.3 t/ha and average wheat prices of $250 (AUD)/t, this equates to 438 000 t, translating to saving of approximately $109 million (AUD). Further savings are expected as effector assays become more widespread, and new effectors are discovered and made available.

The use of effectors to screen seedlings early in cereal breeding programmes provides a direct and optimised approach to identify insensitive varieties for advancement, while molecular marker assays require DNA to be prepared and costly assays performed. Markers for tan spot are often derived from weak disease QTLs and require substantial validation and optimisation prior to adoption by the breeders. In contrast, all of the major Australian wheat breeders now include ToxA screening in their selection strategies (Vleeshouwers and Oliver, 2014). This enthusiastic adoption has substantially reduced the cost of screening for disease resistance, allowing breeders to concentrate their efforts and resources on other traits, such as drought or frost resistance, or improved yield. Australia has welcomed a new era of effector-assisted selective breeding for improving wheat resistance to necrotrophic pathogens, whilst maintaining or improving other desirable traits with no observable disadvantages. The challenge now lies in extending this approach to other major wheat-growing countries.

6 Conclusion and future trends

Implementing an effective, economic and lasting tan spot management strategy is not a simple task, with many factors and risks to consider. Growers must make informed decisions based on a combination of management practices encompassing cultural practices, fungicides and varietal resistance. While chemicals may prove effective in the short term, a viable and enduring approach to tan spot control lies in the improvement of varietal disease resistance. Research is continually advancing our understanding of the mechanisms of pathogenicity, with a particular emphasis on effectors. As the success of ToxA-assisted

breeding in Australia has proven, this knowledge can truly be translated out of the laboratory and into the field to provide quantifiable and significant progress. Genomics will no doubt aid in the search for novel effectors, and Ptr genetic manipulation will enable functional testing of these in the laboratory. The validation of QTLs and the incorporation of diagnostic molecular markers into breeding programmes, combined with effector-assisted selection, are likely to prove very effective towards long-term control of tan spot.

7 Where to look for further information

An abundance of disease fact sheets and management information is available from local agricultural offices, state departments and industry professionals. Proprietary decision guides are usually available from crop protection companies.

The UK-based Agriculture and Horticulture Development Board website (AHDB) is well worth exploring and publishes a wheat disease management guide which has useful information for control of a comprehensive number of wheat diseases (https://cereals.ahdb.org.uk/media/176167/g63-wheat-disease-management-guide-february-2016.pdf).

The CABI (Centre for Agriculture and Biosciences International) Plantwise Knowledge Bank (http://www.plantwise.org/KnowledgeBank/home.aspx) is also worth a visit as a source of tan spot factsheets, including diagnostic resources and best-practice control advice.

The Grains and Research Development Corporation (GRDC) is a great resource for tan (yellow) spot research in Australia and includes variety trials and disease research reports (https://grdc.com.au/).

For a comprehensive and thorough review of the genetics underlying tan spot resistance, see Faris et al. (2013).

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