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Review

A review of wheat diseases—a field perspective

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SUMMARY

Wheat is one of the primary staple foods throughout the planet. Significant yield gains in wheat production over the past 40 years have resulted in a steady balance of supply versus demand. However, predicted global population growth rates and dietary changes mean that substantial yield gains over the next several decades will be needed to meet this escalating demand. A key component to meeting this challenge is better management of fungal incited diseases, which can be responsible for 15%–20% yield losses per annum. Prominent diseases of wheat that currently contribute to these losses include the rusts, blotches and head blight/scab. Other recently emerged or relatively unnoticed diseases, such as wheat blast and spot blotch, respectively, also threaten grain production. This review seeks to provide an overview of the impact, distribution and management strategies of these diseases. In addition, the biology of the pathogens and the molecular basis of their interaction with wheat are discussed.

Keywords: blotch, fungal pathogens, Fusarium head blight/scab, Helminthosporium, *Magnaporthe*, rusts, wheat diseases.

INTRODUCTION

Wheat is one of the world's most important staple grains and is the leading source of calories and plant-derived protein in human food (Curtis *et al.*, 2002). In 2015/2016, 735 million tonnes of wheat were produced globally, worth approximately US$ 145 billion. A recent assessment of wheat production by the Food and Agricultural Organization of the United Nations indicates that current wheat supply is ample for global demand (http://www.fao.org/worldfoodsituation/csdb/en/). Nevertheless, future production must increase as the global population is estimated to exceed nine billion people by 2050. As such, it is predicted that annual cereal production must grow by almost one billion tonnes. Furthermore, increased consumption of wheat products throughout many countries in Asia and changes to the grain quality requirements to meet the 'hidden hunger' objectives demand additional crop production (Anonymous, 2017; Shewry *et al.*, 2016).

The continual drive to match yield and quality increases is not without its challenges. Decreasing availability of suitable farm land, climate change and a variety of unpredictable abiotic and biotic stresses continually pose threats to wheat production locally and globally. The decline in the genetic diversity of wheat, in the pursuit of elite high-performing cultivars, has contributed to a perfect storm in pathogen emergence to the point at which diseases threaten global wheat supplies. Pathogenic fungi represent a significant constraint to wheat production. This review considers the key diseases and causal pathogenic fungi affecting crop production, as well as those emerging as threats. In each case, we consider the geographical distribution, impact (if information available), disease management strategies and briefly address the current status of the molecular understanding of each interaction.

THE WHEAT RUSTS

Rust pathogens have hindered global wheat production since the domestication of the crop and continue to threaten the world's wheat supply (Roelfs *et al.*, 1992). It is estimated that global annual losses to wheat rust pathogens range between US$ 4.3 to 5.0 billion (P. Pardey, University of Minnesota, unpublished).

Rust fungi are obligate biotrophic organisms that are completely dependent on nutritional resources obtained from living host cells for growth and reproduction (Cummins & Hiratsuka, 2004; Duplessis *et al.*, 2012). Rust species vary in their ability to infect certain hosts and this differential biology is reflected in the classification of formae speciales (ff. spp.) (Eriksson, 1894). There are three wheat rust diseases, namely stem, stripe and leaf rust, all caused by members of the Basidiomycete family, genus *Puccinia*, named *P. graminis* f. sp. *tritici* (*Pgt*), *P. striiformis* f. sp. *tritici* (*Pst*) and *P. triticina* (*Pt*), respectively (Fig. 1) (McIntosh *et al.*, 1995) (Fig. 1).

Wheat stem rust

Puccinia graminis f. sp. *tritici* Ericks and Henn. (*Pgt*), the causal agent of wheat stem (black) rust, is widely distributed around the world, although less common than the other two wheat rusts

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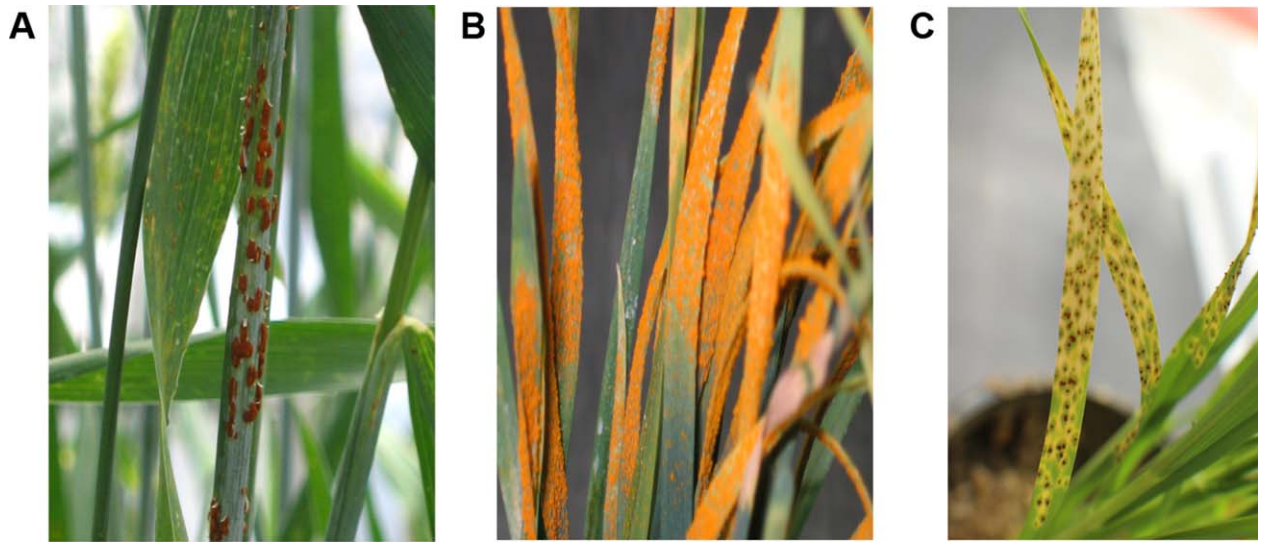

Fig. 1 Symptoms of wheat rust diseases caused by *Puccinia graminis* f. sp. *tritici* (A), *Puccinia striiformis* f. sp. *tritici* (B) and *Puccinia triticina* (C). Photographs courtesy of Rohit Mago.

(Leonard & Szabo, 2005; Singh *et al.*, 2015). *Pgt* is usually found in regions in which warm and moist conditions prevail, and symptoms of infection are typically manifested as masses of red-brick urediniospores on leaf sheaths, stems, glumes and awns of susceptible plants (Kolmer, 2005). Yield losses caused by stem rust are associated with a reduction in grain size and lodging of the plant (Leonard & Szabo, 2005).

Historically, stem rust epidemics have occurred throughout major wheat-producing areas, and the need to control this disease served as a cornerstone to the Green Revolution which led to the introduction of semi-dwarf, stem rust-resistant wheat varieties (Figueroa *et al.*, 2016). Although stem rust has been well controlled in many parts of the world, forecasting models assuming the absence of durable resistance estimate that global losses would average 6.2 million metric tons per year or higher under severe epidemics (Pardey *et al.*, 2013).

In recent years, stem rust has gained significance as new virulence traits have evolved in *Pgt* populations, demonstrating the vulnerability of broadly used wheat cultivars across the globe (Pretorius *et al.*, 2000; Singh *et al.*, 2015). The emergence of the Ug99 race in Uganda in 1998, its subsequent geographical expansion within Africa, to the Middle East, and the appearance of Ug99 variants illustrate the imminent threat to wheat production (Singh *et al.*, 2015). Estimates suggest that 90% of wheat varieties in the world are susceptible to Ug99, justifying elevated concerns about food security (Singh *et al.*, 2011). Likewise, other Ug99 unrelated races have appeared in various parts of the world, reducing the efficacy of the newly identified and deployed sources of resistance. The 'Digalu' race caused a devastating epidemic in Ethiopia in 2014 and a similar race has been reported in Germany (Olivera Firpo *et al.*, 2015, 2017). In 2016, another 'broadly' virulent race was detected in an outbreak in Sicily (Bhattacharya, 2017).

Wheat stripe rust

The disease wheat stripe (yellow) rust is caused by *P. striiformis* Westend. f. sp. *tritici* (*Pst*), a pathogen highly prevalent in temperate regions with cool and wet weather conditions (Chen *et al.*, 2014). Stripe rust is currently the most economically important wheat rust disease with yield losses reaching 100% in susceptible cultivars (Chen, 2005). Approximately 88% of the world's wheat varieties are susceptible to *Pst* and global losses inflicted by the disease are nearly US$ 1 billion annually (Beddow *et al.*, 2015; Wellings, 2011). In Australia, losses caused by stripe rust are estimated at AU$ 127 million (Murray & Brennan, 2009).

Wheat stripe rust has been reported in more than 60 countries and evidence suggests a significant global geographical expansion of *Pst* in the last 50 years (Beddow *et al.*, 2015; Chen, 2005). Since 2000, aggressive races of *Pst* adapted to higher temperature climates have spread to parts of the world that were previously less affected by this disease (Ali *et al.*, 2014). Although populations of *Pst* appear to be clonal in Europe, Australia and North America, there are significant levels of genotypic diversity within some pathogen populations (Chen *et al.*, 2014). Such polymorphic populations are evident in western China and Central Asia, consistent with the Himalayan and nearby regions as the centre of pathogen diversity where sexual recombination appears to be common (Ali *et al.*, 2014; Hovmøller *et al.*, 2011). More recently, new race groups have emerged and swept through Europe in 2011, 2012/13 and 2015, and genetic analysis places their origin in Himalayan regions, indicating the role of incursions in altering

the population structure of the pathogen (Hovmøller *et al.*, 2015; Hubbard *et al.*, 2015).

Wheat leaf rust

Puccinia triticina Eriks. (*Pt*) is the causal agent of leaf rust (Anikster *et al.*, 1997; Bolton *et al.*, 2008), the most common and widely distributed of the three wheat rust diseases (Bolton *et al.*, 2008; Huerta-Espino *et al.*, 2011). The pathogen is prevalent in areas with mild temperatures and moist conditions. Yield losses associated with infection are attributed to a reduction in kernel weight and numbers of grain per head. Although grain losses caused by leaf rust display temporal and geographical variation, the economic significance of the disease is substantial (Huerta-Espino *et al.*, 2011; Kolmer, 2005). From 2000 to 2004 in the USA, estimated losses caused by *Pt* reached over US$ 350 million (Huerta-Espino *et al.*, 2011). In Australia, losses ascribed to this disease are calculated at AU$ 12 million (Murray & Brennan, 2009).

Leaf rust is a problematic disease because the pathogen displays high diversity, there is a constant emergence of new virulence profiles and the pathogen exhibits high adaptability to a wide range of climates (Huerta-Espino *et al.*, 2011; Kolmer, 2005; McCallum *et al.*, 2016). The centre of origin of *Pt* is in the Fertile Crescent region of the Middle East, where both primary and alternative hosts exist; however, in most parts of the world, the population of *Pt* is clonal (Bolton *et al.*, 2008; Kolmer, 2005).

General overview of the life cycle of wheat rust fungi

The full life cycle of wheat rust fungi involves the production of five types of spores associated with either asexual or sexual reproduction, which occurs in wheat or another unrelated non-cereal host, respectively (Jin *et al.*, 2010; McIntosh, 2009). The devastating asexual reproductive phase is driven by urediniospores, which mediate infection through multiple developmental stages, such as haustoria formation (Harder & Chong, 1984; Staples & Macko, 2004). Haustoria are structures essential not only to acquire nutrients, but to deliver effectors into the plant cell, allowing the suppression of plant defences and cell reprogramming to accommodate fungal growth (Garnica *et al.*, 2014; Panstruga & Dodds, 2009; Ramachandran *et al.*, 2016). Several *Mahonia* and *Berberis* spp. (barberry) serve as alternative hosts for *Pgt* (Leonard & Szabo, 2005; Roelfs, 1985) and *Pst* (Jin *et al.*, 2010; Wang & Chen, 2017). The alternative hosts for *Pt* include species of *Thalictrum*, *Anchusa*, *Clematis* and *Isopyrum* (Bolton *et al.*, 2008; Huerta-Espino *et al.*, 2011).

Molecular understanding of the rust–wheat interactions

The molecular and genetic basis underpinning wheat rust pathogenicity is not well characterized. The lack of efficient genetic transformation methods and the inability to generate *in vitro* fungal cultures have limited progress to define the underlying molecular factors governing rust resistance or susceptibility. Nevertheless, genetic resistance studies have provided a strong foundation to understand the basic components of these interactions.

Genetic resistance to rust infection has been identified as either race-specific (also known as seedling or qualitative resistance) or non-race-specific resistance (Periyannan *et al.*, 2017). More than 150 wheat rust resistance genes have been genetically defined in wheat or wild relatives, most conferring race-specific resistance (McIntosh *et al.*, 1995, 2013). At least 50 of these genes are designated *S*tem *r*ust (*Sr*) resistance genes that are responsible for reactions to *Pgt* (McIntosh *et al.*, 1995, 2013). *Sr31* is one of the most widely utilized race-specific genes against *Pgt* (Singh *et al.*, 2006). Unfortunately, the evolution of virulence to *Sr31* led to the emergence and spread of Ug99. Other important genes, such as *Sr21*, *Sr24*, *Sr36*, *Sr38* and *SrTmp*, have also been overcome by the Ug99 lineage or Digalu races (Jin *et al.*, 2008, 2009; Olivera Firpo *et al.*, 2015; Pretorius *et al.*, 2010). To date, the genes *Sr2*, *Sr25*, *Sr23*, *Sr33*, *Sr35*, *Sr45* and *Sr50* are considered to be the most valuable to protect against the newly evolved races (Singh *et al.*, 2015). More than 70 genes are designated *Y*ellow *r*ust (*Yr*) resistance genes and these have been shown to condition reactions to *Pst* (Chen, 2005; McIntosh *et al.*, 2013). However, in many parts of the world, *Pst* virulence has been reported for the majority of these genes. Resistance to *Pt* is conditioned by 68 *L*eaf *r*ust (*Lr*) genes, with *Lr1*, *Lr3*, *Lr10* and *Lr20* being commonly used in global wheat cultivars (Dakouri *et al.*, 2013; McIntosh *et al.*, 1995). In general, the constant emergence of new rust races thwarts the maintenance of effective sources of genetic resistance in the field and highlights the challenges to control these diseases solely with genetic resistance (Ellis *et al.*, 2014).

The cloning of 10 race-specific genes in wheat (*Sr22*, *Sr33*, *Sr35*, *Sr45*, *Sr50*, *Yr10*, *Lr1*, *Lr21*, *Lr10*, *Lr22*) has demonstrated that, as in other plants, these genes encode nucleotide-binding site leucine-rich repeat (NBS-LRR) proteins (Ellis *et al.*, 2014; Mago *et al.*, 2015; Steuernagel *et al.*, 2016; Thind *et al.*, 2017), and hence resistance responses must be governed by the direct or indirect recognition of cognate Avr factors. So far, no Avr factor has been characterized in the wheat rust fungi; however, genome sequencing and transcript predictions indicate the presence of rich effector repertoires (Bruce *et al.*, 2013; Cantu *et al.*, 2013; Duplessis *et al.*, 2011; Garnica *et al.*, 2013; Upadhyaya *et al.*, 2015). In addition, limited numbers of sexual crosses showing genetic segregation for virulence and avirulence phenotypes support the hypothesis that rust–wheat interactions conform to the gene-for-gene model (Samborski & Dyck, 1968; Zambino *et al.*, 2000). The evolution of physiological races is also consistent with this model. Physiological races in wheat rust fungi are defined by standard

classification systems which establish relationships between disease phenotypes and specific race-specific genes present in fixed sets of differential wheat cultivars (Chen *et al.*, 2002; Jin *et al.*, 2008; Long & Kolmer, 1989; McIntosh *et al.*, 1995). A general feature in populations of wheat rust fungi is the appearance of virulence traits which overcome deployed race-specific resistance in the field, a phenomenon known as 'boom-and-bust cycles' (Hulbert & Pumphrey, 2014). These pathogenicity changes can be the result of genetic recombination by sexual reproduction or somatic hybridization and serial mutations in the absence of alternative hosts (Lei *et al.*, 2016; Park & Wellings, 2012; Wang & McCallum, 2009).

Non-race-specific resistance, often referred to as adult plant resistance (APR), confers quantitative resistance against wheat rusts (Periyannan *et al.*, 2017). In these cases, the characteristic partial resistance phenotypes limit inoculum build-up and the likelihood of the occurrence of epidemics. Examples of this type of resistance include *Sr2*, *Lr34*, *Lr46*, *Lr67*, *Lr68* and *Yr36* (Ellis *et al.*, 2014). The mechanism by which these genes exert their function is not well understood. Cloning of *Yr36* (Fu *et al.*, 2009) revealed the role of a cytoplasmic protein kinase in mediating resistance. In contrast, *Lr34* and *Lr67* encode an ATP-binding cassette transporter and hexose transporter, respectively (Dodds & Lagudah, 2016; Krattinger *et al.*, 2009; Moore *et al.*, 2015).

Management strategies for wheat rust diseases

Management strategies to mitigate the effect of wheat rust diseases include cultural control practices, in addition to chemical and genetic control (Ellis *et al.*, 2014). The removal of inter-crop 'green bridges' with tillage and the eradication of alternative hosts are some of the cultural practices to help manage wheat rust diseases (Kolmer *et al.*, 2007; Zadoks & Bouwman, 1985). The use of genetic resistance has traditionally been the method of choice because fungicide treatments can be costly, weather dependent and raise environmental and health concerns. Nevertheless, the recent emergence of new rust races for which genetic resistance is unavailable has led to a more widespread use of fungicides. There are several chemical formulations approved to control wheat rusts. In general, quinone outside inhibitors (QoIs), 14α-demethylation inhibitors (DMIs) and the recent use of succinate dehydrogenase inhibitors (SDHI) are effective (Oliver, 2014). In Australia, during the period 2003–2005, approximately AU$ 40–90 million per year was spent on chemical control to prevent stripe rust epidemics (Wellings, 2007). These regular chemical applications pose a potential risk of the reduction or loss of fungicide sensitivity (Arduim *et al.*, 2012; Oliver, 2014).

Both race-specific and non-race-specific resistances are utilized to manage all three wheat rust diseases (Ellis *et al.*, 2014). The value of non-race-specific resistance genes is highly recognized because of their durability and protection against multiple pathogens or races. To date, breeding programmes have favoured the use of either gene stacking or pyramiding in order to achieve resistance durability, and often generate combinations of race-specific and non-race-specific resistance genes with additive effects to optimize protection.

The airborne nature of rust pathogens, in combination with the local evolution of new races and the documented consequences of exotic rust incursions, means that coordinated international surveillance programmes are crucial to guide management strategies (Park *et al.*, 2011). The information obtained via pathogen surveys informs policies, research and development investments, as well as crop protection and breeding approaches. In response to the emergence of Ug99, a global cereal rust monitoring system was created to collect geospatial and time-sensitive data on rust prevalence and race structure (Hodson *et al.*, 2009; Park *et al.*, 2011). Such a resource has proven to be extremely valuable and will serve as a model to monitor other important pathogens.

THE BLOTCH DISEASES

The Ascomycete fungi *Zymoseptoria tritici*, *Parastagonospora nodorum* and *Pyrenophora tritici-repentis* are the causal agents of Septoria tritici blotch (STB), Septoria nodorum blotch (SNB) and tan spot (TS), respectively (Fig. 2). Collectively, these diseases are described as the blotch diseases. Although these diseases are known to form complexes, each will be summarized independently.

Septoria tritici blotch

Zymoseptoria tritici (formerly known as *Mycosphaerella graminicola* or *Septoria tritici*) (*Zt*) is the causal agent of STB, the primary leaf disease of wheat in temperate growing regions. In Europe, STB is currently regarded as the primary threat to wheat production, and is estimated to cost growers throughout the EU €280–1200 million per annum, including direct losses and control costs (Fones & Gurr, 2015). Actual losses associated with STB are less clear in other growing regions. In Australia, STB is reported to be responsible for only AU$ 20 million in losses per year (Murray & Brennan, 2009). However, the prevalence of this disease has increased in recent years (Milgate *et al.*, 2014).

Molecular understanding of the Zt–wheat interaction

The colonization pattern of *Zt* is unique amongst the blotch pathogens because the sparse apoplastically dwelling hyphae undergo a protracted asymptomatic growth phase (7–11 days post-infection). During this latent phase, the pathogen grows slowly and does not appear to actively acquire nutrients (Keon *et al.*, 2005; Sánchez-Vallet *et al.*, 2015). This phase is followed by the onset of necrotic symptoms as the host cells lyse, which increases nutrient availability for the pathogen to sporulate and complete its infection cycle.

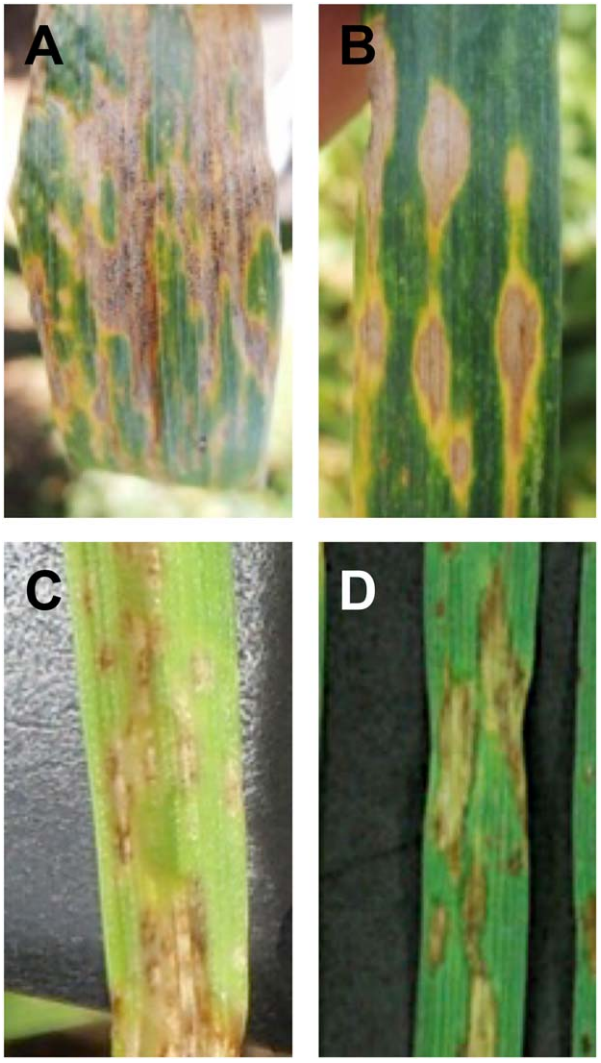

Fig. 2 Symptoms of foliar blotch diseases. (A) Septoria tritici blotch. (B) Tan spot. (C) Septoria nodorum blotch. (D) Spot blotch. Photographs courtesy of Dr Megan McDonald (A, B) and Erin Hill (C, D).

The molecular basis of the infection cycle described above is poorly understood. A myriad of approaches, including forward genetics, reverse genetics, functional genomics and next-generation sequencing, have attempted to identify the key molecules facilitating pathogen establishment and host specificity. The study by Marshall *et al.* (2011) has demonstrated that *Zt* actively subverts host defence mechanisms during the latent phase of the infection through the secretion of the chitin-binding effector protein Mg3LysM. The inactivation of *Mg3LysM* resulted in the abolishment of apoplast colonization of susceptible wheat leaves.

A seminal advance in our understanding of this interaction has been reported recently through the cloning of the first avirulence gene from *Zt*. Zhong *et al.* (2017) used a combination of genome-wide association studies and traditional map-based cloning approaches to identify *AvrStb6*. AvrStb6 is a small secreted protein which elicits a resistance phenotype in *Stb6* wheat lines. AvrStb6 has no similarity to other known proteins and displays evidence of undergoing strong diversifying selection, typical of genes involved in gene-for-gene interaction. The contribution though of *AvrStb6* to virulence remains elusive.

Management strategies for STB

STB is primarily managed through two classes of fungicide: SDHIs and DMIs. Multi-site inhibitor fungicides (e.g. chlorothalonil) are also used, although these lack the efficacy of the SDHIs and DMIs and are only useful as protectants. Other fungicide chemistries, previously efficacious against STB (e.g. QoIs), are no longer used because of the evolution of resistance (Torriani *et al.*, 2015). The efficacy of existing chemistries is also now being re-considered because of the performance reduction of DMIs in the field (Dooley *et al.*, 2016; Heick *et al.*, 2017). Furthermore, the future availability of some DMIs and SHDIs remains in doubt because of the impending EU Regulations and the emergence of new SHDI semi-resistant strains (Jess *et al.*, 2014). Despite this, chemical control, often involving two or three sprays per season, remains the primary mechanism for STB control in Europe. Renewed emphasis has now been placed on the use of alternative management strategies (Arraiano & Brown, 2017; Torriani *et al.*, 2015).

Another major component of STB management is through genetic resistance. Twenty major genes have been mapped that contribute to qualitative resistance to STB (Brown *et al.*, 2015). Sources of quantitative resistance have also been identified. Although only accounting for a moderate percentage of disease resistance, quantitatively inherited resistance is more durable under field conditions and often confers a broad-spectrum resistance that is effective against multiple *Zt* genotypes. Brown *et al.* (2015), who provide a comprehensive background to the genetics of STB resistance, reported that 167 genomic regions harbouring quantitative trait loci (QTLs) have been identified. Phenotyping of these QTLs has demonstrated their involvement in different stages of disease progression, including sporulation, necrosis and latency. Unlike many other pathogens, numerous genetically different *Zt* genotypes can be routinely found infecting a single wheat leaf or a single field. In a comprehensive comparative study involving individual lesions, fields and regions, *Zt* genotype diversity in Switzerland, Texas and Israel was found to be high; for example, variation within a single field ranged from 79% to 100% of maximum possible values (Linde *et al.*, 2002). These findings indicate a significant potential risk for the spread of *Zt* mutant alleles that would enable the breakdown of single major resistance genes.

Septoria nodorum blotch

Parastagonospora nodorum (*Pn*) is the causal agent of SNB. The disease is prominent throughout Australia, costing the industry approximately AU$ 100 million per annum (Murray & Brennan, 2009). Outside of Australia, the significance of SNB is less conclusive with documented evidence of direct losses difficult to source. Anecdotally, reports have emerged that the disease is prevalent throughout parts of France and the Scandinavian countries. In the UK, SNB was fully replaced by STB in the 1980s (Bearchell *et al.*, 2005).

Molecular understanding of the Pn–wheat interaction

In contrast with *Zt*, symptom development as a result of *Pn* infection on susceptible cultivars develops rapidly, and the pathogen can complete its infection cycle in a week if conditions are suitable (Solomon *et al.*, 2006). Recent studies have demonstrated that *Pn* secretes host-specific effector proteins that facilitate *in planta* growth (Oliver *et al.*, 2012). Although it is well documented that each of these effectors induces host cell death in a susceptible genetic background, evidence also suggests that each protein has a role in the suppression of host defence responses. For example, the Tox1 protein, which displays homology to chitin-binding proteins, protects *Pn* from wheat chitinases induced as part of the host defence response (Liu *et al.*, 2016). Similarly, Tox3 and ToxA interact with wheat PR-1 proteins (Breen *et al.*, 2016; Lu *et al.*, 2014). Although evidence suggests that these interactions may serve independent functions, there is support for the hypothesis that the Tox3–PR-1 interaction mediates host defence and facilitates disease (Breen *et al.*, 2016, 2017).

The *Pn* effectors induce cell death and necrosis as an outcome of their interaction with a cognate dominant susceptibility gene (*ToxA–Tsn1*, *Tox1–Snn1* and *Tox3–Snn3*). *Tsn1* encodes a protein of the typical NBS-LRR resistance gene structure that does not directly interact with ToxA, but is required for ToxA-induced necrosis (Faris *et al.*, 2010). *Snn1* encodes a membrane-bound wall-associated kinase which interacts directly with Tox1 to induce cell death (Shi *et al.*, 2016). Collectively, the cloning and characterization of these effectors and corresponding host-interacting proteins have fundamentally advanced our understanding of host-specific necrotrophic diseases.

Management strategies for SNB

SNB is effectively controlled by a combination of host genetics, chemical control and cultural practices, such as crop rotations (Francki, 2013). Most commercially available fungicides successfully manage SNB and, despite intensive use, fungicide resistance has been detected rarely. However, like many other foliar pathogens, resistance to the QoI fungicide azoxystrobin has been reported in Scandinavia (Blixt *et al.*, 2009).

Host genetics play a significant role in controlling SNB, and multiple QTLs have been reported to confer both quantitative and qualitative resistance (Francki, 2013). However, the discovery of the genotype-specific effector proteins ToxA, Tox1 and Tox3 has impacted significantly on cultivar development and deployment (Friesen *et al.*, 2006; Liu *et al.*, 2009, 2012). The purified effector proteins have been exploited commercially (through leaf infiltrations) to rapidly identify and disregard lines sensitive to specific effectors, and therefore susceptible to the pathogen (Vleeshouwers & Oliver, 2014). This application illustrates how breakthroughs in understanding molecular plant–pathogen interactions can expedite disease management practices.

Tan spot

Tan spot (TS) is caused by the ascomycete fungal pathogen *Pyrenophora tritici-repentis* (*Ptr*) and results in decreased kernel weight and numbers of grains per head (Shabeer & Bockus, 1988). TS is found in most parts of the wheat-growing world, including Europe, North America and Australia. In Asia, this pathogen is a component of the Helminthosporium leaf blight complex (Duveiller *et al.*, 2007). The rise of TS as a significant disease in affected areas has been attributed to the use of minimum or zero tillage practices (Bockus & Claasen, 1992; Rees & Platz, 1979). However, the worldwide impact of the disease is difficult to assess because of a lack of available data. For example, there is anecdotal evidence that TS is prevalent within the UK, but there are no data publically available to support this. Disease surveys conducted in Australia in 2009 concluded that TS was the primary cause of yield loss, costing the local wheat industry in excess of AU$ 200 million in losses per annum (Murray & Brennan, 2009)

Molecular understanding of the Ptr–wheat interaction

The pathogenicity of *Ptr* is largely attributed to three necrotrophic effectors: ToxA, ToxB and ToxC. The products of each of these genes interact specifically in an inverse gene-for-gene manner with the host susceptibility genes *Tsn1*, *Tsc2* and *Tsc1*, respectively, to facilitate disease (Ciuffetti *et al.*, 2010). It has been hypothesized that *ToxA* was acquired by *Ptr* through a horizontal gene transfer event from *Pn*, and that this acquisition provided a significant fitness advantage to *Ptr* (Friesen *et al.*, 2006). *ToxB* also encodes a small secreted protein that induces a chlorotic response in the presence of the host susceptibility locus *Tsc2* (Ciuffetti *et al.*, 2010). Unlike *ToxA*, *ToxB* is a multi-copy gene within the *Ptr* genome and virulence has been reported to correlate with copy number. Potential *ToxB* homologues have been detected in a variety of other pathogens, including within *Bipolaris*, *Alternaria* and species in the *Pyrenophora* (Ciuffetti *et al.*, 2010). The product of *ToxC* has not yet been identified, but it is thought to interact with *Tsc1* in wheat resulting in a chlorotic phenotype (Effertz *et al.*, 2002).

Fig. 3 Symptoms of Fusarium head blight/scab. (A) Early infection signs manifested as a partially bleached wheat head. (B) Advanced infection of *Fusarium graminearum* manifested as an almost completely bleached wheat head.

Management strategies for TS

Given the relatively recent emergence of the disease (or, at least, its recognition), the attention to TS management appears to be minimal compared with the diseases described above. Host genetics and the understanding of the *Ptr* race structure are important management tools. There are currently eight races of pathogen that have been identified, which are defined by the presence/absence of *ToxA*, *ToxB* and *ToxC*. *ToxA* is harboured in races 1, 2, 7 and 8, whereas the *ToxB* gene is present in races 5, 6, 7 and 8 (Strelkov & Lamari, 2003). Infiltration studies using culture filtrates have shown that ToxC is produced in races 1, 3, 6 and 8. The recognition of this race structure has implications for resistance breeding. For example, *ToxB* is lacking from all races of the pathogen in Australia. Consequently, breeders can focus on counter-selection for *Tsn1* (i.e. removal of ToxA sensitivity) to improve TS resistance, rather than divert resources towards *Tsc2* (Antoni *et al.*, 2010; Liu *et al.*, 2017).

Chemical control also has a significant role in TS management. Fungicide applications result in yield increases ranging from 0.8 to 4.4 tonnes/ha depending on the level of tillage (Jørgensen & Olsen, 2007). Efficacy tests on different fungicides have demonstrated that the QoI chemicals (e.g. pyraclostrobin) are more effective than the DMIs (e.g. propiconazole).

FUSARIUM HEAD BLIGHT/SCAB DISEASE

Fusarium head blight (FHB) disease (also known as wheat scab or ear blight) leads to premature senescence of the wheat head and is caused primarily by the Ascomycete fungus *Fusarium graminearum* (*Fg*) (Fig. 3). In combination with other cereal-infecting *Fusarium* species, many regionally unique species complexes exist to cause severe FHB epidemics [reviewed in Brown & Proctor (2013); http://scabusa.org/]. Globally, FHB is the most serious and hazardous floral disease of wheat. In the USA, China, the EU, UK, Africa, Brazil and elsewhere, severe FHB epidemics occur at a minimum of every fourth or fifth year. In the USA, yield losses as a result of FHB were estimated to be US$ 3 billion between the early 1990s and 2008 (Schumann & D'Arcy, 2009). Wheat crops are particularly prone to FHB if rain prevails just prior to and during crop anthesis. The main consequences of FHB disease are three-fold: grain yield and quality are reduced, which compromises the overall harvest and subsequent marketability; moreover, the accumulation of various sesquiterpenoid trichothecene mycotoxins [such as the type B toxin deoxynivalenol (DON)] in the grain presents a major food safety risk and health hazard to humans, animals and natural ecosystems. In many countries, legal limits are in place on the permitted mycotoxin levels for the various end uses. For example, in the EU and USA, for human consumption, the respective permitted levels are 1250 and 2000 ppb for unprocessed products, and between 200–750 and 1000 ppb for finished products (http://scabusa.org). In North America, new highly virulent *Fg* strains have recently emerged that produce two novel type A trichothecene mycotoxins, namely NX-2 and NX-3, which present additional health risks (Varga *et al.*, 2015).

Molecular understanding of the Fg–wheat interaction

The process by which Fg infects wheat heads is unique when compared with other fungal–plant interactions. Infections start with

direct entry into open florets, followed by penetration of the various floral tissues with or without the formation of infection cushions (Boenisch & Schäfer, 2011). *Fg* infections continue with extracellular hyphae advancing between live host cells without causing visible disease symptoms, reminiscent of an apoplastic biotroph (Brown *et al.*, 2010). This symptomless infection phase continues and extends for over a centimetre beyond any visibly bleached wheat tissues. The symptomatic phase occurs when surrounding fungal hyphae enter the wheat cells and is accompanied by host death. This intracellular *Fg* colonization of dead wheat cells has all the hallmarks of a necrotrophic pathogen (Brown *et al.*, 2010). Complete wheat head colonization and bleaching take between 10 and 14 days.

During the symptomless phase, extensive *Fg* transcriptional changes occur, which are distinct from those observed during the symptomatic phase (Brown *et al.*, 2017; Lysøe *et al.*, 2011). These include the rapid activation of various *Tri* genes, responsible for trichothecene and DON production, other co-regulated gene clusters and non-clustered genes of known and unknown functions. DON production by *Fg* is known to be essential for disease formation in wheat spikes (Cuzick *et al.*, 2008). DON inhibits protein translation in eukaryotes (reviewed in Varga *et al.*, 2015). *In planta*, the presence of DON may reduce early induced plant defence responses. At higher concentrations, DON induces plant programmed cell death (PCD) and activates specific plant defences (Desmond *et al.*, 2008). Plant PCD could assist nutrient acquisition by *Fg*.

Several other small secreted enzymes and secondary metabolites are required for disease formation. These are the iron-scavenging secreted siderophore triacetyl fusarinine C (TAFC) (Oide *et al.*, 2006), the secreted lipase Fgl1 (Blümke *et al.*, 2014) and several carbohydrate-active enzymes (CAZymes) that can modify and deconstruct plant cell walls and induce host cell death (Brown *et al.*, 2012; Sperschneider *et al.*, 2016). At least 390 *Fg* genes with a wide range of evolutionarily conserved functions in intracellular signalling, enzyme reactions and transcription are known to be required for pathogenicity or to contribute to virulence. Full details of each of these genes are available in the Pathogen–Host Interactions database (PHI-base) (Urban *et al.*, 2017).

So far, only a small region on wheat chromosome 3BS, where a major QTL conferring FHB resistance, known as *Fhb1*, is located, has been molecularly and functionally characterized in detail. Early studies revealed that the *Fhb1* locus potentially either encodes or regulates the expression of a DON-glucosyltransferase involved in DON detoxification (Lemmens *et al.*, 2005). Recent findings have shown that a single gene in the *Fhb1* locus, encoding a pore-forming toxin-like protein (*PFT*), plays a major role in FHB resistance that is unrelated to DON detoxification (Rawat *et al.*, 2016). However, at least two reported, but as yet unpublished, studies have indicated that other gene types residing within the QTL containing *Fhb1* may also contribute to host resistance.

Management strategies for FHB

The use of genetic-based resistance to FHB represents the most cost-effective control strategy (Wegulo *et al.*, 2015), but is proving to be slow and complex to achieve in elite commercial wheats. To date, only a few unrelated moderately resistant sources have been identified: for example, the cultivars Sumai-3 from China and Frontana from Brazil. FHB resistance is controlled by multiple major and minor QTLs that are often associated with a fitness cost or yield penalty (Gilbert & Haber, 2013). The two commercially important types of FHB resistance are classified as type I (resistance to initial infection) and type II (resistance to the spread of FHB in the host) (Cuthbert *et al.*, 2006; Kubo *et al.*, 2010; Niwa *et al.*, 2014; Schroeder & Christensen, 1963). An alternative breeding approach is to select against known susceptibility targets in wheat, for example, a locus residing near one of the 'Green revolution' semi-dwarfing *Rht* genes (Srinivasachary *et al.*, 2009).

Fungicides are an integral part of the FHB disease management strategy in Europe. However, triazole (DMIs) mixtures applied during the optimal crop flowering period can only provide approximately 30%–60% FHB control (McMullen *et al.*, 2012) as a result of *Fg*'s intrinsic resistance to triazoles (Fan *et al.*, 2013). Fungicide efficacy is reduced further when adverse weather conditions delay applications. In addition, technical challenges exist in the effective treatment of both the early- and late-flowering tillers with a single application.

Cultural practices include tillage to bury infected crop residues, thereby preventing the development of the next generation of ascospores. Crop rotation with non-host species is used to reduce FHB intensity and DON accumulation (Dill-Macky & Jones, 2000). For example, the DON content of harvest grain in soybean–wheat rotations was 25% lower than in wheat–wheat rotations and 49% lower than in maize–wheat rotations. The ripe crop can also be left in the field to stand to wash out the mycotoxins prior to harvest (R. Dill-Macky, unpublished). Post-harvest operations to reduce mycotoxin contamination in feed wheats include grain cleaning to remove damaged, pink-coloured or light grains which harbour the highest mycotoxin levels.

Host-induced gene silencing (HIGS) has emerged recently as a novel transgenic approach to control FHB (Koch & Kogel, 2014). In this approach, transgenic plants express long double-stranded or hairpin RNAs, with sequences identical to parts of the coding regions of *Fg* essential genes, which induce the plant's RNA silencing machinery to generate small interfering RNAs (siRNAs). The latter are mobile and can enter fungal cells [using unknown mechanism(s)] where they trigger the sequence-specific degradation of fungal mRNA targets. Selected *Fg* HIGS targets include the *Tri5* gene and specific members of the chitin synthetase gene

family. In particular, the study by Cheng *et al.* (2015) indicated the high potential of HIGS under field conditions to reduce FHB disease and mycotoxin contamination to minimal levels.

EMERGING AND 'UNDER THE RADAR' DISEASES

Spot blotch and Helminthosporium leaf blight

Bipolaris sorokiniana (syn. *Helminthosporium sativum*, teleomorph *Cochliobolus sativus*) (*Bs*) is a devastating pathogen causing both foliar and root diseases (Fig. 2). The foliar diseases have the most impact on wheat production and these can be delineated into spot blotch (SB) and also Helminthosporium leaf blight (HLB), a disease complex comprising both SB and TS (Duveiller *et al.*, 2005). These diseases are the major biotic constraint to wheat production in the Eastern Gangetic Plains encompassing the main growing regions in India, Bangladesh and Nepal (Duveiller & Sharma, 2009; Saari, 1985; Singh *et al.*, 2004). Losses to these diseases can reach up to 50% under conditions conducive to infection (Sharma & Duveiller, 2004; Singh *et al.*, 2004). Significant losses have also been recorded in the warmer humid growing climates in South America (Duveiller & Sharma, 2012). Notably, the distribution of the disease appears to be comparable to wheat blast through South America and Asia (described below).

Molecular understanding of the Bs–wheat interaction

The molecular basis of the *Bs*–wheat interaction is poorly understood. Currently, only a handful of genes have been characterized from the pathogen and no Avr proteins or small molecules have yet been identified.

Nevertheless, recent genome sequencing efforts have made progress towards an understanding of the basis of the disease. McDonald *et al.* (2017) sequenced three *Bs* isolates from eastern Australia and identified that one of these isolates contained a gene nearly identical to *ToxA* as described in *Pn* and *Ptr*. Further analysis revealed that *ToxA* was present in approximately 30% of Australian *Bs* isolates and the existence of two haplotypes differing by only 1 bp. The gene was harboured within a larger 12-kb region that is also shared between these blotch pathogens, suggesting that this mobile genetic element plays a key role in facilitating pathogenicity in these related fungi. *ToxA* has also been reported in North American isolates of the pathogen (T. Friesen, personal communication); however, it is unknown whether the gene is present in isolates from wheat-growing areas affected by the disease. If *ToxA* is dominant within pathogen populations in affected areas, concerted efforts could be made to eliminate the *Tsn1* susceptibility gene from local wheat varieties.

Management strategies for SB

The control of these diseases is problematic and the integration of complementary management approaches is often applied. Resistance breeding, chemical management and agronomic practices play key roles in the management of SB and HLB. As with most diseases, breeding for resistance is preferred. Given that most wheat growers in the affected areas are often small holders, fungicides are not always available, leaving growers reliant on resistance breeding (Duveiller, 2004). The genetics of SB and HLB resistance in wheat is quantitative (Joshi *et al.*, 2004; Singh *et al.*, 2016). Sources of resistance to both HLB and SB have included wheat germplasm from Brazil, Zambia and China, together with progenies from wide cross derivatives, including the use of synthetic hexaploids and classical hexaploid wheat and Chinese materials. The exploitation of these lines and others via QTL and association mapping has confirmed that many genes play a role in the resistance to these diseases (Singh *et al.*, 2016). A detailed list of SB-resistant genotypes is described by Duveiller & Sharma (2012).

Fungicides contribute to the control of SB and HLB, with the treatment of seeds and foliar symptoms shown to be effective in the management of disease. In a recent study by Sharma-Poudyal *et al.* (2016), a combination of seed treatment, together with a single foliar spray, was found to be particularly effective. Initial treatment of wheat seed with carboxin + thiram resulted in 9% and 8% grain yield increases in successive years compared with control treatment. A subsequent single foliar post-flowering spray further increased grain yields by up to 15%, demonstrating the potential of this complementary fungicide approach (Sharma-Poudyal *et al.*, 2016).

Planting time is also crucial in minimizing the losses caused by SB and HLB, presumably because of the increased growth rate of *Bs* at higher temperatures and also the increased likelihood of temperatures exceeding the threshold level of 28 °C when host resistance is known to break down (Duveiller *et al.*, 2005; Nema & Joshi, 1973). Nutrient and water stresses also contribute to disease severity, with yield losses almost doubling when wheat is grown under suboptimal conditions (Sharma & Duveiller, 2006).

Wheat blast

Wheat blast (WB) is a devastating disease caused by the pathogenic fungus *Magnaporthe oryzae Triticum* pathotype (*MoT*) (synonym *Pyricularia oryzae Triticum* pathotype and *Pyricularia graminis-tritici*) (Castroagudín *et al.*, 2016; Cruz & Valent, 2017). WB was first identified in Paraná State in Brazil in 1985, and was subsequently disseminated to Bolivia, Argentina and Paraguay (Igarashi *et al.*, 1986). The disease was confined to South America until its discovery in Bangladesh in 2016 (Islam *et al.*, 2016), and more recently in India (Bhattacharya & Pal, 2017). A phylogenomics and populations genetics study indicated that the disease did not evolve independently in South Asia, but was probably caused by an incursion of a South American lineage of *MoT* (Islam *et al.*, 2016).

WB is primarily a head disease. The typical symptoms range from small elliptical lesions to complete bleaching and empty spikes (Igarashi *et al.*, 1986, 1990). Yield losses of 40%–100% have been reported (Igarashi, 1990). Foliar lesions caused by *MoT* have also been described; however, their role and significance in grain yield losses remain unknown (Cruz *et al.*, 2016a; Igarashi, 1990). Warm and humid conditions are required for WB development (i.e. temperatures at 25 °C with at least a 10-h wetting period) (Cardoso *et al.*, 2008). Accordingly, a recent study in the USA listed Louisiana, Mississippi and Florida as the most high-risk states to a WB outbreak (Cruz *et al.*, 2016a).

Management strategies for WB

Given the recent emergence of WB in developing countries, disease control has been a priority. The efficacy of chemicals to control WB remains questionable. Reported studies have indicated that the performance of foliar fungicides only reduces disease incidence by 50% (compared with non-fungicide treatments), and that this efficiency is typically far lower in the weather conditions, described above, that favour disease development (Maciel, 2011). Little information is available on the chemistries used in the field, although QoI fungicides, either as the sole active ingredient or in mixtures, have been documented for WB control in Brazil (Castroagudín *et al.*, 2015). However, resistance to QoIs in South America has increased dramatically over the last 10 years to the point at which 90% of tested isolates carry a resistant allele. As such, there are no currently documented effective fungicide strategies in Brazil (Castroagudín *et al.*, 2015)

Seed treatment with fungicides has also been tested to control the initial establishment of the disease. However, the length of time from seed germination to heading has led to questions with regard to the efficacy of seed treatments. Nonetheless, seed treatment trials in Brazil and also the USA have proven to be successful, at least in the management of seed-borne infections (Bockus *et al.*, 2015; Goulart & Paiva 1991; Igarashi, 1990).

A more desirable approach to control WB is through the use of host genetics. Unfortunately, these approaches have been only partially effective because of the lack of identifiable genetic resistance. Trials to screen for genetic resistance have also been hampered by the need to undertake field testing (i.e. no correlation between seedling resistance and yield losses) and the localized nature of the disease.

Recently, Cruz *et al.* (2016b) have assessed the effect of the 2NS translocation from *Aegilops ventricosa* (Zhuk.) Chennav on WB (head and leaf) resistance. This translocation carries a 25–38-cM distal segment of chromosome arm 2NS from *A. ventricosa* to the distal region of chromosome arm 2AS in wheat (Helguera *et al.*, 2003). Using near-isogenic lines with and without 2NS, phenotyping in the field revealed that 2NS conferred a significant decrease in WB symptoms on heads, but, interestingly, not foliar symptoms. However, a small percentage of MoT isolates tested appeared to overcome the 2NS-conferred resistance. However, these data are promising and provide the only robust evidence to date of a natural source of resistance to WB.

Several other wheat genes have been identified to play a role in resistance to *MoT*, including *Rmg2*, *Rmg3*, *Rmg7* and *Rmg8* (Anh *et al.*, 2015; Tagle *et al.*, 2015; Zhan *et al.*, 2008). Although these may be promising candidates for resistance breeding, any potential impact is difficult to determine as disease screening was undertaken on either seedlings or detached heads, and field confirmation would be needed prior to any conclusion that these genes may have an effective role in WB management.

CONCLUDING REMARKS

It is, without question, the case that fungal diseases provide a significant challenge to the maximization of wheat yields, now and into the future. In this review, we have attempted to briefly summarize the key aspects of some of the most significant foliar and floral wheat diseases currently threatening production. We acknowledge that there are many other diseases that also threaten production (e.g. powdery mildew, take-all, eyespot, bare patch, crown rot); however, space limitations restricted our selection of diseases to those currently considered to have the greatest impact on yield. Although clearly not exhaustive, this review provides a reference point for colleagues in molecular plant pathology to appreciate the complexity of these diseases and to consider them in a more holistic manner.

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