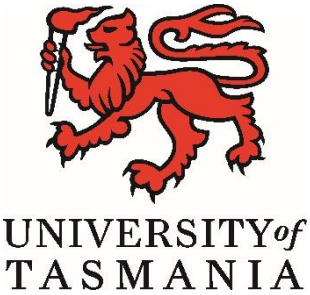

Potential strategies for control and treatment against powdery mildew of wheat

By

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Declaration of Originality

The thesis contains no material which has been accepted for a degree or diploma by the University of Tasmania or any other institution, and to the best of my knowledge and belief, no material previously published or written by any other person except where due acknowledgement is made in the text of the thesis.

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Conference

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Statement of Co-authorship

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Author contributions:

Wrote the manuscript: Candidate

Contributed to the manuscript writing: Author 1, Author 2, Author 3

Paper 1: Located in Chapter 3

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Author contributions:

Conceived and designed experiment: Candidate, Author 1

Performed the experiments: Candidate, Author 4

Analysed the data: Candidate

Wrote the manuscript: Candidate

Contributed to the manuscript writing: Author 1, Author 2, Author 3

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Table of Contents

Abstract

Improvement of both quantity and quality of wheat production is of great importance for food security as wheat is the most traded cereal crop and third most produced globally. Wheat diseases represent a constant threat affecting grain production, therefore cost-effective, sustainable control strategies are required. Airborne powdery mildew (*Blumeria graminis* f. sp. *tritici*) is the sixth most damaging pathogen of wheat and can reduce yield from 13-30%. Characteristics such as short propagation period, easy dispersal and rapid establishment on the host makes management of this pathogen difficult. A thorough understanding of wheat powdery mildew control with a focus on sustainable, non-chemical approaches is a research high priority. This thesis presents studies which explore two such sustainable means of reducing the disease – genetic resistance and use of symbiotic fungi which alter plant response to disease.

To facilitate marker assisted selection for breeding resistant wheat varieties, a review of current knowledge was undertaken to produce a genetic map-based reference which integrates records of all known powdery mildew resistance genes and quantitative trait loci (QTL). Over 200 powdery mildew genes (permanently and temporally designated genes) and QTL were mapped to 21 chromosomes of common bread wheat, which is expected to benefit future molecular resistance breeding.

A genome-wide association study (GWAS) was conducted on a total set of 329 wheat varieties with diverse genetic backgrounds. Based on disease incidence data from three Tasmanian trials from 2016 to 2018, and variety genotypes obtained by single-nucleotide polymorphism (SNP) array, 14 QTL associated with wheat powdery mildew resistance were identified on 11 different chromosomes. Out of these, four QTL on chromosome 3A, 3B, 6D and 7D were believed to be novel. High confidence gene candidates underlying new QTL involved in wheat powdery mildew resistance include a member of major facilitator superfamily (MFS) which is a new class of plant-defence related proteins, genes encoding disease resistance protein and regulating early response to fungal infection.

In addition to exploring genetic resistance, the influence of beneficial fungi on the expression of resistance was also investigated. Arbuscular mycorrhizal fungi (AMF) are widely known for their benefits in plant growth, as well as reported evidence of disease mitigation. This thesis presents a study conducted in three wheat cultivars that were inoculated with the AMF species

Rhizophagus irregularis. Results showed that AMF inoculation had no significant effects on wheat powdery mildew incidence or plant growth. These results contrasted from previous studies which found that this AMF species was associated with a 34% decrease in powdery mildew incidence or severity in wheat. This difference may be explained by the different wheat genotypes used in this study, which highlights the specific of plant-pathogen-AMF interactions.

The findings provide insight into wheat powdery mildew control. Greater understanding in genetic resistance, in particularly durable resistance, may enable more utilization of identified QTL. The current findings also suggest the impact of an AMF species on disease cannot be generalised to all host genotypes, but rather AMF effects should be evaluated with a case by case basis.

Chapter 1: General introduction

Wheat is cultivated on over 220 million hectares and approximately 750 metric megatons is produced annually (Balfourier et al., 2019), and is essential for global food supply. Powdery mildew can lead to wheat yield loss of 13% to 30% on average, and frequently occurs in wheat growing regions worldwide (Yao et al., 2007, Savary et al., 2019). Effective control of powdery mildew is required for securing wheat production. Like most pathogenic fungi, wheat powdery mildew can be reduced through a range of strategies, the most effective being using resistant cultivars and fungicide application. Due to its airborne distribution and short life cycle, wheat powdery mildew is easily dispersed across hosts and can become established rapidly. It is therefore important to control wheat powdery mildew with preventative measures to reduce disease impact and yield penalty.

Currently, deployment of a wide range of powdery mildew resistant wheat varieties is central to integrated disease control programs. The resistance obtained in most of these varieties is driven by a single or a few wheat powdery mildew race-specific resistance genes (Pm genes). Although there is high risk that the pathogen will overcome this resistance and effectiveness will erode, resistant varieties are still highly valued control option for farmers and breeders. In contrast to resistance conferred by such race-specific genes, quantitative resistance is also valuable for its broad-spectrum effectiveness. Quantitative resistance has no specificity for pathotype or even pathogen species, as exemplified by the Lr34/Yr18/Pm38 and Lr46/Yr29/Pm39 genes, which display resistance to wheat leaf rust and yellow rust, in addition to powdery mildew (Lillemo et al., 2008). In this context, selection pressure imposed on pathogen populations is not as strong as that by race-specific resistance and it is therefore more durable. Identification of quantitative resistance has been conducted extensively through association mapping and linkage mapping at the population level (McIntosh et al., 2014, McIntosh et al., 2016, McIntosh et al., 2017, Liu et al., 2017b). Knowledge of wheat powdery mildew resistance loci is increasing greatly due to connection between molecular markers and disease incidence data.

The growing number of identified wheat genome-wide powdery mildew resistance loci reflects rich resistance sources and genetic diversity. However, breeding cultivars with quantitative resistance is still slow and challenging, due to control by several minor-effect resistance genes (Pilet-Nayel et al., 2017). In addition, the connections between identified QTL and gene

candidates remain poorly established, therefore how QTL are responsible for the trait of interest is still not well understood. Identification of resistance sources and determining the underlying resistance mechanisms to harness quantitative resistance for breeding objectives is a must for a better manipulation of wheat resistance to powdery mildew.

A complete strategy for wheat disease control should never rely on plant resistance alone. Cultural and chemical controls are also required to prevent crop disease incidence and epidemics. The benefits of some microorganisms, particularly arbuscular mycorrhizal fungi (AMF), are increasingly evident in crop production system, addressed in a wide range of areas such as promoting crop growth (Tran et al., 2019), boosting yield (Zhang et al., 2019) and mitigating against diseases (Xavier, 2004). Interactions between AMF and host plants rely on symbiosis and mutual effects can enhance nutrient and water uptake for the host in exchange for photosynthetic carbon and lipids (Klironomos, 2003, Luginbuehl et al., 2017, Jiang et al., 2017).

Root disease reduction has been often reported in arbuscular mycorrhizal plants (Whipps, 2004). Although AMF colonize plant root parts, they have been reported to display inhibitory effects against some aboveground plant pathogen diseases, including for tomato *Alternaria solani* and rice blast fungus *Magnaporthe oryzae* diseases (Fritz et al., 2006, Campos-Soriano et al., 2012). The ability of AMF to reduce powdery mildew has also been investigated, but responses of mycorrhizal plants have not led to consistent results. An earlier study reported a 78% reduction of powdery mildew in wheat, of which mycorrhiza-induced resistance (MIR) was proposed as the mechanism to trigger rapid plant defence (Mustafa et al., 2017). In the case of plant foliar diseases, the biocontrol effects of arbuscular mycorrhizal fungi are suggested to involve improvement of plant nutrition and productions of phytoalexins and antimicrobial substances. The commercial utility of AMF as crop inoculants in field crops is still under evaluation. The efficacy generated from single studies cannot be generalised in all cases; factors such as host species and cultivars, life cycle of host, environment conditions like phosphorus levels, species and isolates of AMF, and the ratio of individual species/isolates (if using a mixture of mycorrhizae), can all account for various performances of mycorrhizae in crop disease control (Eke et al., 2016, Mustafa et al., 2016).

This thesis focuses on the understanding of wheat genetic resistance against powdery mildew, as well as control strategies based on the identification of quantitative resistance and application of beneficial AMF. Broadly, the objectives of the research were to:

- Thoroughly review previously reported powdery mildew resistance genes and quantitative resistance loci in wheat, with the aim of integration and visualisation in a genetic map to facilitate marker assisted selection for resistance breeding.
- Identify potential quantitative resistance for wheat powdery mildew resistance via association mapping using more than 300 wheat cultivars, mainly core collections from China.
- Explore the effects of arbuscular mycorrhizal fungi on powdery mildew disease in wheat cultivars varying in resistance level.

Chapter 2: Literature review

2.1. Background of wheat powdery mildew disease

Security of wheat production is of great economic importance globally as it is a key staple food crop. Wheat cultivation can be found in many areas of the world, particularly in regions with maritime and semi-continental climates, and this diversity presents multiple constraints on productivity (Alam et al., 2014). Despite the growing scarcity of arable land and shifting of agriculture to more marginal lands, the production volume of wheat is projected to increase (Placido et al., 2013, Peng et al., 2011). Many factors can reduce the fitness and productivity of wheat including drought (Mwadzingeni et al., 2016), osmotic stress from salinity (Oyiga et al., 2016), and diseases which represent a potential production loss of 15% in wheat globally (Oerke, 2006). Powdery mildew (caused by *Blumeria graminis* f. sp. *tritici*) ranks the sixth out of ten fungal pathogen priorities in wheat (Dean et al., 2012), and results in eighth most yield loss by pests and pathogens worldwide (Savary et al., 2019). Powdery mildew can occur year-round in many wheat growing regions, with crop production losses of up to 35%, 62% and 40% recorded in Russia, Brazil and China respectively (Mehta, 2014).

Characteristics of the pathogen which promote rapid spread and adaptation include the short life cycle, air-borne spores easily transported long distances, and potential sexual recombination for generating new virulent races. *Bgt* provides two types of pathogenic inoculum for infection, asexual conidia and sexual ascospores. The ascospores are released from chasmothecia which enable *Bgt* survival in the absence of a living crop, the ascosporic infection process of *Bgt* has recently been characterised by Jankovics et al. (2015). Both conidia and ascospores are infectious and ready to germinate when surface moisture is available or environment is humid. Mild temperatures (10-22°C) further enhance the infection process (Beest et al., 2008). Upon adhesion of conidia or ascospores to a photosynthetically-active wheat leaf surface, a specialized germ tube is produced and elongates to form thread-like hypha with appressoria and this process can complete within two hours (Acevedo‐Garcia et al., 2017). Shortly afterwards, digitate hypha gives rise to a penetration peg and ramifies a haustorium to allow the breach of host epidermal cell, via turgor pressure and dynamic enzymes (Glawe, 2008).

2.2. Wheat cultivars with resistance to powdery mildew

Germplasm resistance is among the most useful means to control powdery mildew and is considered the most economically efficient for growers (Summers & Brown, 2013, Xin et al., 2012). Crops are diverse in defence capacity against pathogen, and the genetic status of both host and pathogen determines the outcome of the interaction. The plant defence system can be considered as a two-layered network, in which "nonhost" pathogen-associated molecular pattern (PAMP)-triggered immunity and "gene for gene" based effector-triggered immunity are fundamental (Jones & Dangl, 2006, Gozzo, 2003). Different plant resistance phenotypes depend on how the host interacts with the pathogen, generally either broad-spectrum or race-specific resistance.

2.2.1. Race-specific resistance

The mechanism of race-specific resistance is well-accepted to due to presence of a major resistance gene (R gene) and cognate pathogen avirulence gene (avr gene) (Flor, 1971) and has proved an integral part of crop breeding for resistance in wheat for many decades (Shamanin et al., 2019, Wang et al., 2005, Lillemo et al., 2010). The plant R gene codes for a receptor, which is activated by a pathogen effector, irrespective of plant stages. The outcome of a recessive allele in one or both the host and pathogen is disease susceptibility, with a resistant outcome only resulting from a dominant R gene and dominant Avr gene.

In wheat, arrays of powdery mildew R genes (Pm genes and temporally designated genes) and alleles have been documented over 30 years, and in this review we have provided a definitive list based on current knowledge (Table S2.1). Of the 144 resistance genes documented, only Pm2, Pm3, Pm8 and Pm21 have been cloned to date (Yahiaoui et al., 2004, Cao et al., 2011, Hurni et al., 2013, McIntosh et al., 2014, McIntosh et al., 2016, McIntosh et al., 2017, Xing et al., 2018). Most powdery mildew resistance genes in Table S2.1 are those of the nucleotide-binding site-leucine-rich repeat (NBS-LRR) genes and are race-specific as they only recognize complementary avr genes. The gene for gene interaction always results in a hosts' full resistance rating to pathogen. However, R gene resistance is no longer effective when the predominant pathogen genotype is an evolved isolate or pathotype mutations. Due to this, race-specific genes typically lead to "boom-bust" disease dynamics over time, as the disease is controlled by a new gene for some time, then is eventually overcome by changes in the pathogen population. Defeated Pm genes have been seen in major wheat-producing areas like

Australia (Golzar et al., 2016), China (Ma et al., 2015, Wang et al., 2005) and USA (Cowger et al., 2018). An earlier field test in Western Australia detected a breakdown in powdery mildew resistance present in fewer than four years (Cowger et al., 2018, Golzar et al., 2016, Ma et al., 2015). To extend the lifespan of race-specific resistance, strategies based on gene stacking with a range of Pm genes together, regional allocation or temporal deployment of R gene are recommended for durable breeding (Li et al., 2014b, Burdon et al., 2014).

In natural environments, different pathogen isolates tend to exist simultaneously. This might increase the disease epidemic risk as a result from genetic diversity in the pathogen population. Therefore, gene stacking may be insufficient to prevent pathogenic ingress for long periods and the cultivars are eventually no longer effective once these genes have been overcome. To add value to gene stacking, allelic mining is put forward for crop improvement (Bhullar et al., 2010). Allelic diversity at a resistance locus results in genetic variation in a corresponding phenotype or trait. The mixture of lines with different alleles of a specific resistance gene (Pm3) has shown to be a promising strategy for an effective and sustainable use of race specific genes (Ma et al., 2016b, Brunner et al., 2012) which might be an evolutionary response to selection by a specific pathotype (Yahiaoui et al., 2006). In wheat, studies on the allele series at the Pm3 locus, which prescribes isolate-specific resistance to each functional allele, provides an exceptional mode. Wheat Pm3 resides on the short arm of chromosome 1A and 20 functional alleles of Pm3 (Pm3a to Pm3t) have been reported so far. As Pm3h, Pm3i and Pm3j alleles are identical to Pm3d, Pm3c and Pm3b respectively, it is thus actually 17 different alleles (Pm3a to Pm3g, Pm3k to Pm3t) (Bhullar et al., 2009, Yahiaoui et al., 2006, Bhullar et al., 2010, Huang et al., 2004, Bougot et al., 2002).

Evolution analysis suggested that Pm3 alleles originate from Pm3CS, a susceptible allele in domesticated tetraploid wheat and broadly spread in bread wheat cultivars (Yahiaoui et al., 2006). Transgenic lines with Pm3 allelic series gave more superior PM resistance in field tests compared to the parental lines with only a single Pm3 allele (Koller et al., 2018). This enhanced resistance came from allelic combination and additive expression levels of alleles. However, combining different Pm genes or alleles does not always enhance resistance. The presence of Pm3, a rye (*Secale Cereale*) Pm8 orthologue in the same cultivar will subvert Pm8 resistance (Hurni et al., 2014). To explain, Pm8 is from 1RS rye chromosome, resistant to Pm8 avirulent *Bgt* isolates (Zeller, 1973). Pm8 gene in 1BL.1RS chromosome wheat translocation showed no resistance to Pm8 avirulent *Bgt* isolates when Pm3 exists. Further studies showed that Pm3

interferes with Pm8 induced signal transmission, where non-function heterometric protein complex forms at the post-translational stage (Hurni et al., 2014). Suppression also exists in combination between two resistance alleles; an allele pyramid of Pm3b and Pm3f interfered with Pm3 based resistance in wheat F_1 hybrid (Stirnweis et al., 2014).

To better distil Pm3-*Bgt* interaction and multi-allelic phenomenon, corresponding Avr loci of Pm3 alleles have been characterized recently (Bourras et al., 2015). In this system, three major Avr loci in *Bgt* were found to associate with avirulence, where locus_2 accounted for specificity and locus_1 generally involved in all AvrPm3-Pm3 interactions. It is quite interesting that *Bgt* avirulence was not simply determined by interactions between R locus and Avr loci, but also found to involve a third component – a suppressor of avirulence (Svr) (Bourras et al., 2016). The presence of Svr in mildew will, together with avirulence allele and R allele, regulate plant resistance in cereal-powdery mildew pathosystem. An active suppressor allele blocks out the function of a recognized avirulence allele, in which case disease occurs. The Avr-R-Svr interactions have led to an extension of the classical gene-for-gene model, based on the fact that genetic background underpinning resistance to cereal powdery mildew is much more dimensional.

2.2.2. Broad-spectrum resistance - Quantitative resistance

Different from race-specific resistance, quantitative resistance has very contrasting characteristics. The arbitrary terminology for *Bgt* quantitative resistance is as rich as the interactions it encompasses, including the terms partial resistance, horizontal resistance, background resistance, slow-mildewing, or adult plant resistance (APR) for resistance present in stages after the seedling stage (Bennett, 1984, Tucker et al., 2007). Quantitative resistance operates horizontally on an action of multigenic, simultaneously controlled by a few to several genes (Miedaner & Flath, 2007). This type of resistance does not lead to complete absence of infection, instead it minimizes fungal sporulation and duration (Li et al., 2014b, Burdon et al., 2014).

Based on the number and effects of genes controlling resistance, one may expect to distinguish quantitative resistance from race-specific resistance. However, some APR cultivars such as Genesee and Knox which are widely cultivated in USA show qualitative patterns (Bennett, 1984). APR in Genesee was reported owing to a single dominant gene in controlled conditions (Ellingboe, 1976), from which case is reminiscent of qualitative resistance with pathotype

specificity involved. Host × *Bgt* isolate differential interactions were observed in Knox (Rouse et al., 1980), actually isolate-specificity for quantitative resistance was also noticed in wheat against leaf rust (Azzimonti et al., 2013). According to Niks et al. (2015) there are four categories of resistance based on quantitative/qualitive performance in phenotype/genetic inheritance. Questions arise such as: why quantitative resistance has race-specific pattern? what mechanisms lead to different performance of resistance since both qualitative and quantitative resistances could be expressed by a single gene?

The answer to these questions lies in consideration that reactions of host plants to pathogens are basically either sensitive, partially resistant or completely resistant. A recent report proposed the term partial resistance to refine the concept of quantitative resistance, proposing that it can be a consequence from any of the following genetic actions: (1) incomplete expression of major R genes, (2) APR genes and (3) resistance genes with minor effects (Burdon et al., 2014). It is not surprising that in the case (1) what appears to be quantitative resistance phenotypically is based on race-specific resistance, whereas (2) and (3) act in a manner of non-race specificity. APR is single gene controlled, but effective towards all or most *Bgt* subsets so very different to race-specific R genes (Burdon et al., 2014). Some attributes of APR overlap with those of types (1) and (3), but are not exactly the same. APR Pm38 (Lr34/Yr18/Pm38) shows broad-spectrum resistance to different *Bgt* pathotypes, also a gene for leaf rust and yellow rust (Lillemo et al., 2008). But it follows Mendelian inheritance, though large effects enough to treat Pm38 as a major gene having its own gene symbol.

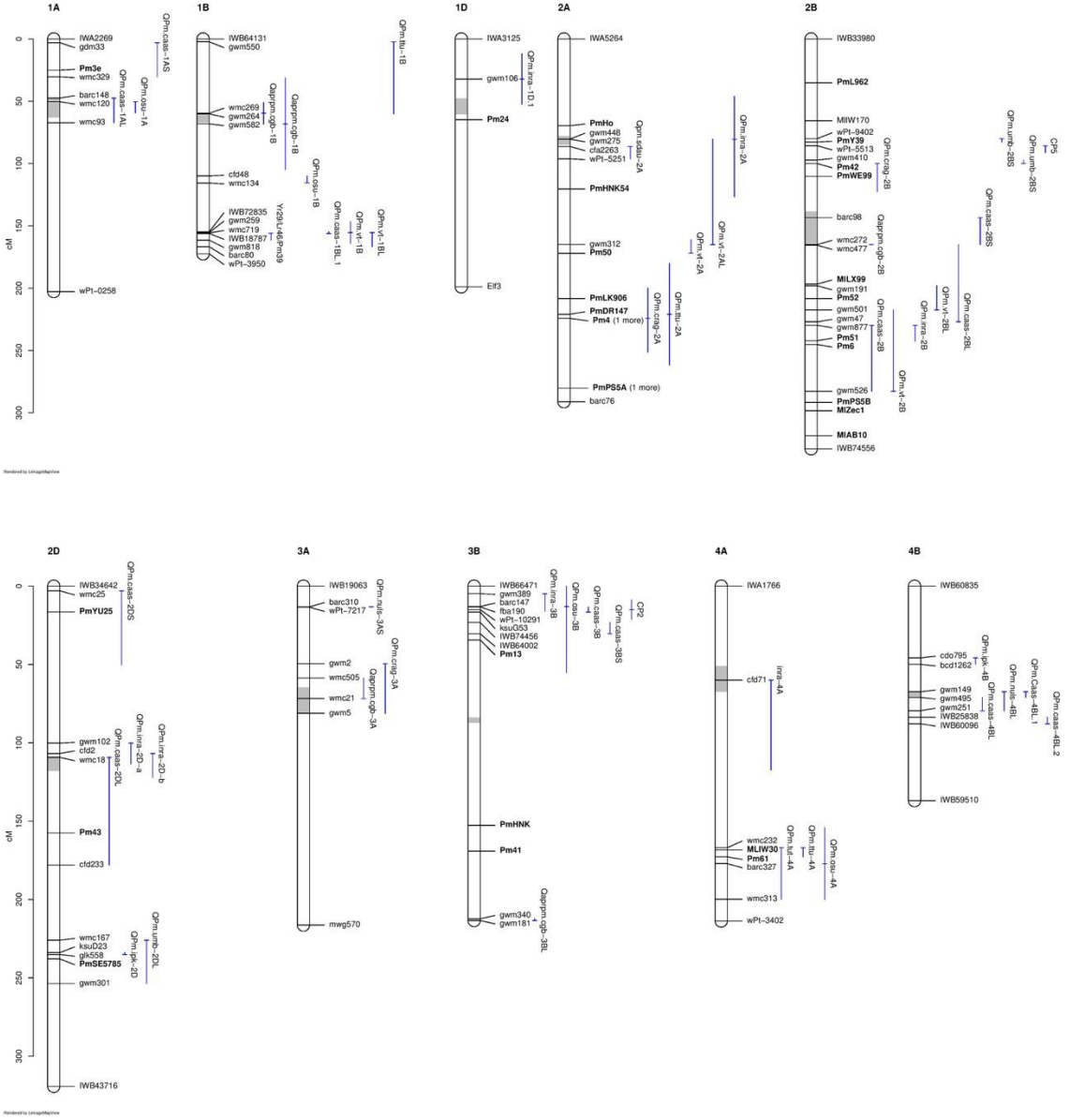
Quantitative Trait Locus (QTL) mapping is an effective approach to detect quantitative resistance to powdery mildew. Hitherto, more than one hundred *Bgt* QTL (Table S2.1) have been mapped to all homoeologous chromosome groups from different mapping studies, with some of them being positioned in the same marker intervals (Figure 2.1), which is more than that found in any other species by far. Even so, new QTL detection is still believed to increase with construction of high-resolution genetic map facilitated by genome-wide genotyping markers. Assisted by single-nucleotide polymorphisms (SNP) array, four powdery mildew resistance QTL have been identified in elite wheat line Zhou8425B, together explaining 27% of the total resistance variance (Jia et al., 2018). Amongst, *QPm.caas-3BS* resided in the same position as *QLr.hebau-3BS* (Zhang et al., 2017a), assumed to be a new APR QTL that confers resistance to both wheat powdery mildew and leaf rust. Normally, a suite of QTL could be discovered through genome-wide association mapping or linkage mapping using biparental

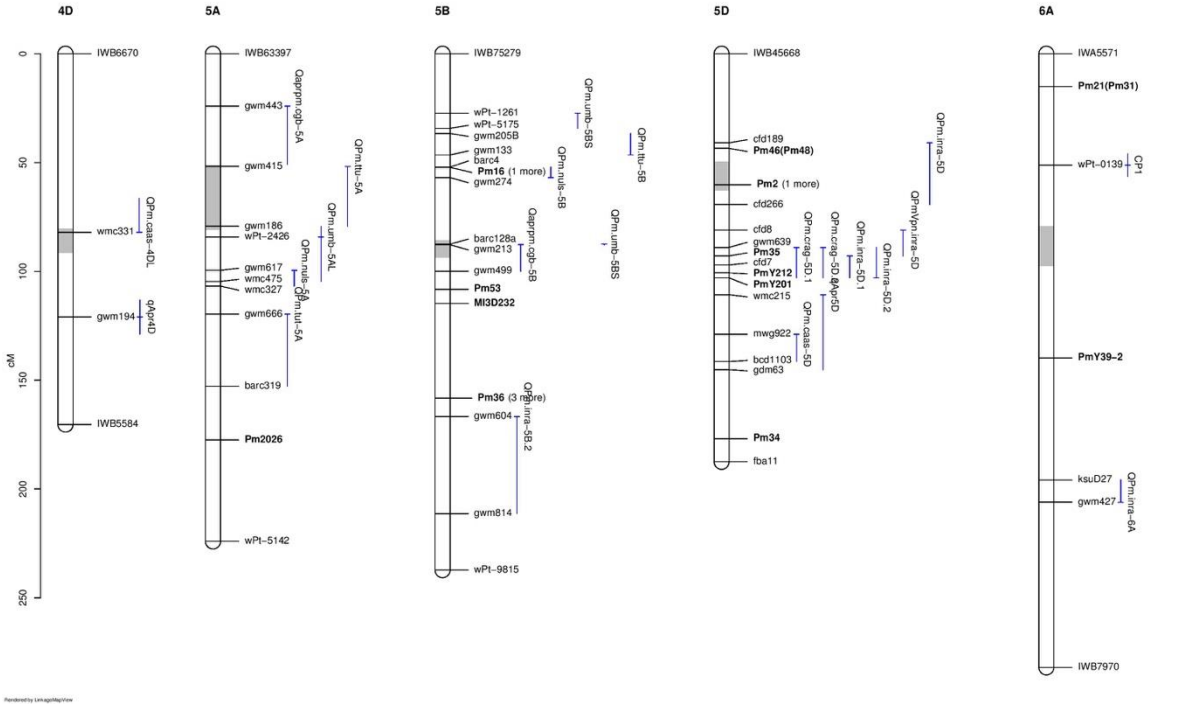
population, but 4-5 QTL can be adequate for powdery mildew resistance in wheat under slight to moderate pathogen pressure (Asad et al., 2012, Jia et al., 2018). Co-localisation of QTL has been suggested to imply a peroxidase gene family in barley resistance to powdery mildew and rusts (Gonzalez et al., 2010), fully sequenced and annotated wheat genome (Appels et al., 2018) will benefit exploration of certain gene functional groups underlying powdery mildew QTL that mapped to similar region.

Most studies have found that quantitative resistance is more durable than qualitative resistance, given there is less selection pressure on the pathogen (Li et al., 2014b, Liu et al., 2001). For instance, cultivar Knox has persistently displayed APR efficacy over 20 years of cultivation (Liu et al., 2001). Complex genetic basis makes it hard for breeders to manipulate quantitative resistance. If quantitative resistance impacts basal non-host defence, then it is reasonable to expect it being more durable (Mundt, 2014). Increasing pathogen aggressiveness has been found to erode quantitative resistance (McDonald, 2010, Mundt, 2014). This has been seen in wheat rust disease, for which some Australian cultivars known harbouring APR had greater infections to isolates from Western Australia (Bariana et al., 2007). Increased virulence from ongoing pathotypic evolution implies potential deterioration of race non-specific resistance. Then again, studies suggest possible residual effects on quantitative resistance from defeated major R genes, as described for *Qpm.crag*-2A as a residual effect of the defeated Pm4b (Mingeot et al., 2002). Perhaps tight links between R genes and QTL forms a cluster as a resistance unit (Kou & Wang, 2010), such that it is almost unable to distinguish the resistance contribution from R gene and the QTL nearby. Prediction of durability is difficult in practice because assessment is made after distribution of a cultivar in a conducive environment (Johnson, 1981). Therefore, a plant breeder does not know how a cultivar will perform over the long-term until multi-year field trials have been done.

Normally, race-specific resistance and quantitative resistance to powdery mildew co-present in a given cereal cultivar (Miedaner & Flath, 2007). In this case, genetic interactions are multi-dimensional, with possible interaction of quantitative resistance and qualitative resistance. Some research already discovered the potential that in combination with quantitative resistance, durability of qualitative R gene can be improved (Brun et al., 2010). This gives promise for breeding durable resistance cultivars by taking advantage of both types of resistance but yielding more than additive benefits. However, this co-existing genetic profile makes it challenging to analyse genetic effects, because quantitative resistance can only be

screened when the host lacks race-specific genes (Miedaner & Flath, 2007, Burdon et al., 2014).

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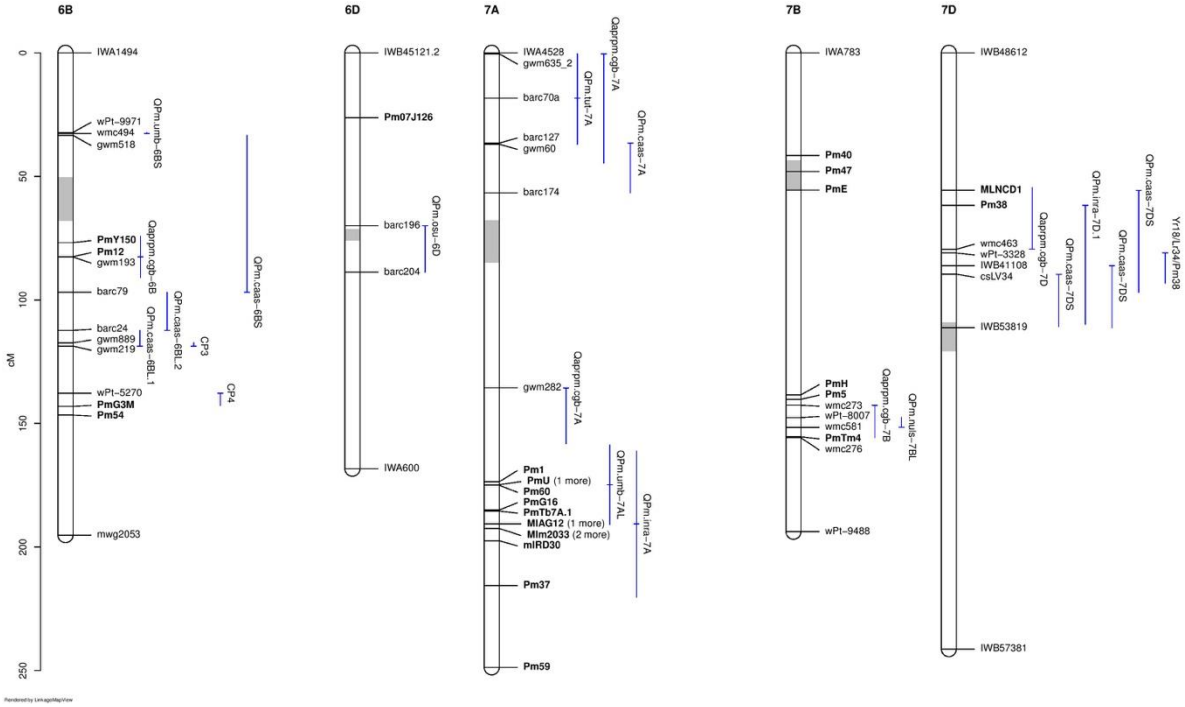
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Qaprpm.cgb-6B
QPm.caas-6BL.2
QPm.caas-6BL.1
CP3
CP4
QPm.caas-6BS
IWB45121.2
Pm07J126
barc196
barc204
IWA600
QPm.osu-6D
IWA4528
gwm635_2
barc70a
barc127
gwm60
barc174
gwm282
Pm1
PmU (1 more)
Pm60
PmG16
PmTb7A.1
MIAG12 (1 more)
MIm2033 (2 more)
mIRD30
Pm37
Pm59
Qaprpm.cgb-7A
QPm.tut-7A
QPm.caas-7A
Qaprpm.cgb-7A
QPm.umb-7AL
QPm.inra-7A
IWA783
Pm40
Pm47
PmE
PmH
Pm5
wmc273
wPt-8007
wmc581
PmTm4
wmc276
wPt-9488
Qaprpm.cgb-7B
QPm.nuis-7BL
IWB48612
MLNCD1
Pm38
wmc463
wPt-3328
IWB41108
csLV34
IWB53819
IWB57381
Qaprpm.cgb-7D
QPm.caas-7DS
QPm.inra-7D.1
QPm.caas-7DS
QPm.caas-7DS
Yr18/Lr34/Pm38

Figure 2.1: Projection of wheat Pm genes (in bold) and QTL (blue bars) onto wheat chromosomes, grey section on each chromosome represents centromere. The positions of these genes and QTL are estimated from their flanking molecular markers from previous publications in integrated map (Maccaferri et al., 2015). For those with either flanking marker or close marker not available, are excluded from map. Some Pm genes which are placed at the same position, only one gene randomly chosen to be present, the number of other genes in this case is given in bracket after.

2.2.3. Broad-spectrum resistance - Recessive mlo resistance

While resistance genes or QTL convey disease resistance to plants, susceptibility genes (S-genes) also determine disease outcomes. Inactivation or knock-down of these susceptible factors can lead to reliable resistance to powdery mildew in both monocot and dicot plants (Appiano et al., 2015b, Pessina et al., 2016, Consonni et al., 2006, Wang et al., 2014b). A well-studied type of PM susceptibility gene is MLO (Mildew-Locus-O), which is conserved across great numbers of species but was first demonstrated in barley towards *Blumeria graminis* f.sp. *hordei* (*Bgh*) in 1942 (Jorgensen, 1992). Since then, MLO genes have been reported in other monocots, namely *OsMLO3* in rice, *TaMLO_A1* and *TaMLO_B1* in wheat (Devoto et al., 2003, Varallyay et al., 2012). Contrary to prototypical R gene-mediated qualitative resistance, impaired MLO (the recessive *mlo* form) confers resistance, characterized by broad-spectrum and long-standing efficacy (Jorgensen, 1992). PM-specific resistance separates MLO from another type of negative regulator, which is the enhanced disease resistance 1 (EDR1) (Zhang et al., 2017b), of which mutation also causes PM resistance but exhibits a more general resistance (Huckelhoven, 2005). Zhang et al. (2017b) created *Taedr1* mutants by editing wheat EDR1 with clustered regularly interspaced short palindromic repeats/CRISPR-associated 9 (CRISPR/Cas9) technology, these mutant plants displayed *Bgt* resistance which was independent of mildew-induced cell death. *Bgh* virulence towards *mlo* was postulated to be under the control of at least three genetic loci - one major virulence gene and two minor genes with additive effects (Lyngkjaer et al., 2000). For barley itself, full resistance of the mutant allele *mlo* requires *Ror1* and *Ror2* genes (Freialdenhoven et al., 1996). This sort of protection reinforces spontaneous callose deposition and results in cell death following infection (Wolter et al., 1993). Despite large-scale cultivation of commercial barley varieties, *mlo* mutation has granted resistance towards most *Bgh* isolates for more than 30 years (Jorgensen, 1992,

Lyngkjaer et al., 2000). In wheat, no natural occurrence of an *mlo* gene has been discovered (Acevedo‐Garcia et al., 2017).

MLO proteins are plasma membrane localised plant specific protein possessing seven transmembrane domains (Qiu et al., 2015). Phylogenetic relationships group these MLO proteins into seven clades (I to VII), where clade IV harbors all characterized susceptibility proteins from monocots such as wheat (Pessina et al., 2016). Even with different conservation patterns, orthologue MLO genes of monocots and dicots are functionally conserved in respect to interaction with powdery mildew (Appiano et al., 2015a). MLO proteins appear to negatively modulate vesicle-associated and SNARE (soluble N-ethylmaleimide-sensitive factor attachment protein receptor) protein-dependent defence pathways at penetration site attempts (Bhat et al., 2005). Based on histological analysis, *mlo*-regulated resistance in barley requires actin cytoskeleton to function at the cell periphery (Miklis et al., 2007). The role of actin cytoskeleton is to mediate secretory vesicle trafficking, participating in basal resistance against powdery mildew in Arabidopsis (Kwon et al., 2008). Contents inside these vesicles are presumed to work on papilla formation, so delivery of secretory vesicles will facilitate focal accumulation of cell-wall appositions (CWA) referred to as papillae beneath the encounter site (Pessina et al., 2016, Underwood & Somerville, 2008). In line with these results, a fast and early fortification of papillae was observed in *mlo* resistant barley, indicated as one of the mechanisms involved in *mlo* resistance (Gold et al., 1986, Skou et al., 1984). Since papillae formation is a feature of basal resistance towards *Bgh*, *mlo* resistance expected to share the same aspect of defence with basal resistance (Aghnoum et al., 2010). Both barley *Mlo* and *mlo* genotypes constructed papillae, but papillae were in general bigger and had more failures of fungal penetration in *mlo* plants (Stolzenburg et al., 1984). The role of MLO protein is not confined to the interaction with powdery mildew. Recent systematic dissection of the MLO gene family in rice and wheat suggests tissue expression preference of MLO genes and diversity in stress, heat/cold and osmotic stresses (Konishi et al., 2010, Nguyen et al., 2016, Acevedo-Garcia et al., 2014).

Three candidate homologs of barley Mlo in wheat were initially reported on chromosomes 5AL, 4BL and 4DL at syntenic positions of barley, all sharing high sequence similarity (Elliott et al., 2002). Overexpression of *TaMlo*-B1 (*Triticum aestivum* Mlo in B genome) not only enhanced wheat susceptibility to appropriate powdery mildew *formae speciales* but increased haustorium development of inappropriate powdery mildew form as well (Elliott et al., 2002).

The association between *TaMlo* and the resistance or susceptibility was further confirmed through functional validation of *TaMlo* by VIGS (virus-induced gene silencing) to disable *TaMlo* (Varallyay et al., 2012). With arrested *Bgt* development, subsequent proof for the role of *TaMlo* associated resistance was gained. Recently, *TaMlo* mutants have been generated based on different technologies, all showing good *Bgt* resistance (Wang et al., 2014b, Acevedo-Garcia et al., 2017). *Mlo* genes are largely conserved among the plant kingdom with comparative studies showing that wheat and barley reflect conserved similarity in genome structure. Likewise, the host-specific pathogens *Bgt* and *Bgh also* co-evolved with each host and display gene collinearity (Mayer et al., 2011, Oberhaensli et al., 2011). The functional characterization of barley *Mlo* genes should be able to assist exploration of wheat *mlo*-based resistance, since *TaMlo* shows about 88% similarity to that of barley (Elliott et al., 2002).

2.3. Prospects and problems in current resistance breeding

2.3.1. Utility of exotic resistance sources

Bread wheat and wheat relatives possess untapped diversity for powdery mildew resistance. To increase the potential of disease resistance for wheat breeding, cross-species breeding is valuable. Rye and *Dasypyrum villosum*, close and wild relatives of common wheat respectively have been used for transmitting a variety of resistance genes, especially against powdery mildew and rust fungi (Graybosch, 2001, Li et al., 2018, Chen et al., 2013). Rye-wheat 1RS.1BL translocation, for example, successfully introduced resistance to powdery mildew and stripe rust from rye into modern wheat germplasm (Ren et al., 2017a). For common wheat, its progenitor wild emmer is also a rich donor of resistance alleles and can be exploited for trait improvement. Many confirmed Pm genes originated from wild species and primitive forms including wild emmer, incorporating Pm genes into commercial cultivars is made possible as wild emmer is crossable with both hexaploid common wheat and tetraploid durum wheat (Rong et al., 2000, Elkot et al., 2015). On the other hand, landraces of bread wheat have been cultivated for thousands of years under extreme environments, more genetically polymorphic in disease resistance (Li et al., 2016, Talas et al., 2011) and adaptive traits to abiotic stressors (Lopes et al., 2015b, Reynolds et al., 2007). Compared to distant relatives, landraces are ready for direct crossing of interested traits into new cultivars. A set of wheat landraces have exhibited highly significant resistance to powdery mildew, formally designated as Pm2c (Xu et al., 2015), Pm3b (Yahiaoui et al., 2004), Pm5d (Hsam et al., 2001), Pm5e (Huang et al.,

2003b), Pm24a (Huang et al., 2000b), Pm24b (Xue et al., 2012b), Pm47 (Xiao et al., 2013), Pm59 (Tan et al., 2018) and Pm63 (Tan et al., 2019).

2.3.2. Marker assisted selection (MAS)

Conventional breeding is still the cornerstone in the crop breeding pipeline. It is conducted by crossing plants with interested characteristics and selecting the offspring with the optimal combination. Although resistance is an objective in resistance breeding, crossing includes risk of pleiotropic effects since introduction of alien chromosomal fragment harbouring resistance might also carry undesirable genes. Yield penalties of disease resistance in wheat have been reported for *Pch1* (Johnson, 1992, Groos et al., 2003) and Pm16 (Chen et al., 2005). A segment introgressed from *Aegilops ventricose* carries both eyespot resistance gene *Pch1* and genes reducing yield in wheat (Groos et al., 2003, Doussinault et al., 1983). This potential "linkage drag" greatly limits the direct use of Pm genes in breeding program. In this case, conventional method could take multiple generations to evaluate phenotypes and obtain the target recombination of genes, typically six years for traditional breeding of self-pollinating crop is obviously time consuming (Cowling, 2013). Even so, considerable donor DNA materials can still be found along interested gene in generations after multiple backcrosses (Young & Tanksley, 1989). These attributes reduce the utility and efficiency of conventional breeding.

Alternatively, marker assisted selection (MAS) uses molecular markers to assist trait selection. Since many agronomically important traits are quantitative traits, like yield and disease resistance, QTL mapping is applied to discover marker-trait association (Collard & Mackill, 2008). Valuable markers are then used in downstream marker assisted breeding to screen individuals. MAS shortens the breeding cycle and is more reliable in selecting disease resistant plants compared to simply relying on phenotyping (Tanweer et al., 2015). In this context, many resistance-related QTL have been found in wheat, including for resistance to powdery mildew (Liang et al., 2006), *Fusarium* head blight (Giancaspro et al., 2016), sharp eyespot (Wu et al., 2017) and spot blotch (Kumar et al., 2009). With QTL-MAS, interested genes and alleles can be introduced to commercially favoured cultivars. MAS can also be combined with genomic selection (Nakaya & Isobe, 2012) to make the breeding cycle more efficient. Unlike MAS which mainly selects for QTL with modest to large effects, genomic selection captures all minor-effect QTL as well identifying individuals with high estimated genetic value for the selected traits (Desta & Ortiz, 2014). It relies on genomic prediction, so theoretically the breeding pace is faster since no phenotyping is required. Inspired by optimum contribution

selection model for animal breeding, a plant model for breeding selfing crop was proposed by Cowling et al. (Cowling et al., 2015). This model not only reduces breeding cycle time but also increases genetic response to selection, and able to be combined with genomic selection.

2.3.3. Genetic tools other than MAS

Genetic modification (GM) also delivers genetic improvement for wheat breeding, as it enables the transfer of resistance genes from another species. The effectiveness of GM has been exemplified in transgenic wheat lines, which express antifungal barley-seed class Π chitinase and have enhanced resistance against powdery mildew (Bliffeld et al., 1999). Due most likely to the polyploid nature of common wheat, often it is challenging to obtain stable inheritance of traits developed by DNA-editing tools to induce mutations. The advent of forward genetic screening makes it feasible. The use of SSN (sequence-specific nucleases) with introduction of TALEN (transcription activator-like effector nuclease), together simultaneously edited three MLO homoeoalleles in the same wheat individual; resistance in this triple mutant is complete and heritable (Wang et al., 2014b). However, *TaMlo* modifications caused leaf chlorosis in plants. Limitations of the *mlo* mutant include the common observation of coupling to undesirable traits, for example spontaneous leaf decay which has been a sign of yield penalty, potential of enhancing sensitivity to some other pathogens, as well as reduced plant size (Acevedo‐Garcia et al., 2017, McGrann et al., 2014, Zheng et al., 2013, Jarosch et al., 1999).

CRISPR/Cas9 has been around for years since it is first exploitation for fungal resistance in plant, wheat powdery mildew resistance (Wang et al., 2014b), afterwards this system was also successfully applied to other fungal resistance such as powdery mildew resistance in tomato (Nekrasov et al., 2017) and rice blast disease (Wang et al., 2016a). It was adapted from bacteria, based on simpler RNA-guided DNA recognition to encode the new trait in plants (Knott & Doudna, 2018). Evidence of CRISPR gene free in edited plant progeny indicated a potential strategy for producing non-transgenic crop (Char et al., 2017, Chen et al., 2018). More recently, progress in Targeting Induced Local Lesions IN Genomes (TILLING) technology has been applied to production of commercial PM resistant wheat varieties. Using TILLING, partially resistant bread wheat lines carrying several combinations of mutant alleles of *TaMlo*, the orthologue of barley *Mlo,* have been created (Acevedo‐Garcia et al., 2017). These loss-of-function *TaMlo* homoeologues overcame the disturbance of pleiotropic phenotypes, with no evident abnormality in plant growth. TILLING combines high-throughput genotyping for

mutations with traditional chemical mutagenesis, which is more efficient to unravel single nucleotide mutations in regions of interest (McCallum et al., 2000). It is considered non-transgenic, which is an advantage for addressing consumer concerns about transgenic food.

2.3.4. Considerations for genetic tools in breeding practice

MAS is largely conducted along with linkage mapping and genome-wide association mapping, both mapping methods require genotype and phenotype data to process. Obtaining reliable phenotype data is pivotal for finding out true trait-associated marker. In the case of powdery mildew APR, the phenotype is commonly disease severity, measured either as disease index (i.e. 0-9 scale) or percentage at a specific adult stage. However, resistance response might change as plants age, which is seen in some powdery mildew-infected cereals including wheat (Carver & Adaigbe, 1990, Duggal et al., 2000). This was also observed in QTL mapping of powdery mildew resistance in mungbean, in which a QTL was found to be effective 85 days after sowing while no resistance was expressed 20 days earlier (Young et al., 1993). Multi-year and environment field trials are necessary for QTL detection as it is common for a QTL identified in a one year- environment scenario not to appear in another year-environment combination. For durable resistance breeding, resistance QTL with consistent performance over several years, environments and plant growth would be more valuable. Considering phenotyping, disease severity at a single time point is not the only component relating to resistance, the length of the latent period, percent survival, and area under the disease progress curve (AUDPC) also have potential to discover resistance locus though these traits, though very likely controlled by overlapping QTL (Wang et al., 1994, Chung et al., 2010, Muranty et al., 2009). Inspired by this, future QTL mapping could address more of these resistance relevant components.

QTL mapping clarifies significant markers that are beyond an assigned threshold, referred to as log-likelihood (LOD) in linkage mapping and P-value in genome-wide association study. However, not every QTL exceeding these criteria is a true candidate region, it might be a false positive. In linkage mapping, QTL of powdery mildew resistance in wheat always function with additive effects, but in some mapping studies epistatic interactions between these QTL also appear (Goldringer et al., 1997). This should be carefully considered when estimating QTL effects, to control potential bias of QTL findings.

Failure in transgenic plants has often been reported, such as poor or even no expression and inheritance of transgene (Rajeevkumar et al., 2015). Also, transgene expression could be affected by environment. For example, field condition generally is more complicated than a controlled environment, in this sense more genetic factors could be involved in biological and physiological activities and interacts with transgene expression (Ueda et al., 2006). Genetic modification is always associated with concerns about potential hazards from transgenic plants; many countries, especially in Europe, have announced a ban on planting transgenic seeds. Release of cisgenic approach is considered similar in risk to conventional breeding, because cisgenic plants only have genes from the same species or from a crossable relative (Schouten et al., 2006). It takes a step forward from introgression breeding, as it directly transfers functional genes without multiple transfer steps which involve linkage of other genes (Jacobsen & Schouten, 2007). Further wheat breeding projects can utilise cisgenic methods, as wheat close relatives and progenitors are a great resource of resistant genes.

Resistance alone is not the full answer to control disease. Concerning protecting cultivars at different susceptible levels and short-lived Pm genes, there are some other things farmers can do in field. Cultural controls such as fine-tuning of use of nitrogen fertiliser saves budget but also reduces disease occurrence. Well nitrogen fertilized plants can have more prolific and soft foliar growth (Fois et al., 2009, Ahern et al., 2007), which in turn may affect disease susceptibility as reported decreased phenolic compounds in leaves responding to excess of nitrogen (Larbat et al., 2012, Leser & Treutter, 2005, Li et al., 2008). Tillage can also eliminate inoculum carried on crop stubbles or volunteers. To help avoid strong selection pressure for a certain fungal strain, planting multi-lines or cultivar mixtures is recommended for controlling airborne fungus, especially of small grain cereals (Mundt, 2002, Wolfe, 1985, Villareal & Lannou, 2000). Chemical control should still be a part of wheat management, but used judiciously by plant development stage rather than according to a calendar-based schedule. It is also valuable to monitor fungicide resistance in field, with high-throughput screening method by digital PCR as described in Zulak et al. (2018).

2.4. Other control methods of powdery mildew disease

2.4.1. Chemical control

The ideal management strategy for powdery mildew in cereals is utilisation of resistant cultivars, but efficient disease control is also in need of other complementary approaches (Hilje

et al., 2001, Fry, 2012). Fungicides have been a primary disease control to routinely target powdery mildew, adopted extensively in intensive planting systems (McGrath, 2001, Ishii, 2006). Although perceived environmental contamination and human health problems justify considered use of fungicides, overall, the benefits of fungicides outweigh the risks. With no fungicide control, powdery mildew occasionally reduces wheat yield by nearly 40% (Jørgensen, 1988).

In Australian cereals, FRAC (Fungicide Resistance Action Committee) code 3 (Demethylation inhibitors, DMIs) and code 11 (Strobilurins) fungicides are used for major foliar disease control including powdery mildew. Azoles from DMIs dominate global agrochemical market for the control of plant fungal pathogens (Morton & Staub, 2008), first released in 1970s (Russell, 2005). The mode of action disrupts the biosynthesis of ergosterol which is required for fungal cell membrane integrity (Wong & Midland, 2007). Strobilurins for the control of cereal *Erysiphe graminis* were introduced 20 years later than triazole (Russell, 2005), now the second largest group of fungicides on cereal (Morton & Staub, 2008). They inhibit fungal respiration chain (Leroux et al., 2010), affecting spore germination and penetration (Balba, 2007). So strobilurins are mainly effective at early stage before fungus rooted inside the leaf, providing protectant activity (Balba, 2007). While triazole is more active to target mycelia than spores, functioning as curative fungicides (Balba, 2007).

In wheat, fungicides are applied as seed dressings, on fertiliser in-furrow or foliar spray for powdery mildew control. Chemical foliar spray is normally the best for disease control and economic return. But with increased incidence of powdery mildew, early protection makes sense. Seed (Leath & Bowen, 1989) and in-furrow treatments (Khan & Young, 1989) were found to reduce crop powdery mildew severity. However, when disease pressure is high or crop is susceptible, seed dressing or in-furrow fungicides sometimes cannot even delay onset of disease. To enhance control effectiveness, crops need spray adjuvant. Since fungicides are expensive and can have negative impacts on environmental and human health, therefore they should be used judiciously by plant development stage rather than according to a calendar-based schedule. To optimise crop yield, timing of chemical spray is key for protecting plant parts that affect yield potential or directly contribute to yield. Flag leaf contributes 30-50% of photosynthates to wheat grain milking (Sylvester-Bradley et al., 1990). Best powdery mildew control and yield response were achieved by sprays at emergence of flag leaf before boots visible or ear emerged (Hardwick et al., 1994). Yield loss of 50% can be met at heading and

milking stages if flag leaf is heavily diseased (Griffey et al., 1993). Because well-controlled infection in flag leaf can help minimise disease on head which is almost impossible to be managed. Proper fungicide schedule should be put in place to keep disease on flag leaf to minimum, hence delay leaf aging and prolong grain filling. Particularly in disease prone area and high yield scenario, multiple spray is necessary to protect flag leaf and head.

Many crop pathogens are likely to develop fungicide resistance after long term exposure and this is rather common in powdery mildew. For example, fungicide resistance to group 11, appeared rapidly in *Bgt* in Europe (Chin et al., 2001). Reports of *Bgh* resistance to DMIs also continue to mount as resistant isolates grow fast across West Australia (Tucker et al., 2015). Alteration of the target site is a common mechanism of fungicide resistance, mutations affect the binding of fungicides to the enzyme. A single nucleotide mutation in the *Bgh* Cyp51 (sterol 14α-demethylase) gene was found to give rise to survive DMIs fungicides (Zulak et al., 2018). As for wheat powdery mildew, amino acid substitution of a glycine for an alanine at point 143 (G143A) compromised resistance to strobilurins (Fraaije et al., 2000). Above mutation-based resistance is qualitative, where higher fungicide rate no longer helps control disease as resistant individuals will not be affected (Deising et al., 2008). Frequent application of same fungicide will constantly select for fungal mutants, in doing so, selected strains are insensitive to this fungicide (McGrath, 2001). Quantitative resistance in most cases, is pronounced to be restorable by increasing fungicide concentration and frequency (McGrath, 2001). Currently high reliance on DMIs and strobilurins for disease control will add more pressure on fungicides within group 3 and 11. Powdery mildew evolved resistance after 2 and 4 years of the introduction of strobilurins and DMIs respectively (Deising et al., 2008), as both of them have only one target site which makes fungus easier to overcome. For all fungicides in the same group, they target at the same site which may result in another problem so called cross-resistance. Occurrence of this in one fungicide will result in resistance to another fungicide within the group. For these reasons, DMIs and strobilurins are considered high-risk fungicides. As a matter of fact, cross resistance has already shown in FRAC group 11 (Wyenandt & Maxwell, 2010). To slow down the isolate selection by fungicides, crop producers are advised to limit the spray from the same fungicide group, implement mixture of fungicides from different groups and rotation of chemical classes.

2.4.2. Cultivation practices

In spite of the introducing resistant varieties, yield loss caused by pathogens worldwide is still considerable in major food crops (Oerke & Dehne, 2004). This is mainly due to changes in pathogen populations which evolve to be resistant to existing fungicide and resistant genes. For this reason, other practices which reduce disease incidence are also important. Worldwide, trials pertaining to plant density, irrigation schedules, cropping systems, and tillage management have been conducted for decades to see how they affect powdery mildew incidence, as expected, some of their results were not statistically consistent (Knops et al., 1999, Burdon & Chilvers, 1982). In a field experiment, more severe infection by powdery mildew was observed with high seed rate, because of more dense wheat canopy (Tompkins et al., 1992). This positive relationship is also seen in grapevine, more vigorous development of powdery mildew was found in a higher leaf population (Valdes-Gomez et al., 2011).

Wheat powdery mildew prefers mild and humid circumstances to favour germination, as outlined in section 2.1. Higher frequency of irrigation provides a longer time of humid environment, this favoured powdery mildew infection (Sharma et al., 2004). When relative moisture was over 85%, however conidia germination began to reduce, so did disease severity (Carroll & Wilcox, 2003). On the other hand, water drops may remove conidia from leaf surfaces, diminishes spore numbers (Merchan & Kranz, 1986). Deployment of multi-lines or cultivar mixtures is recommended for control of airborne fungal pathogens, especially of small grain cereals (Mundt, 2002, Wolfe, 1985, Villareal & Lannou, 2000). Cropping cultivar mixtures can provide more interactions between different host genotypes and pathogen population in both genetic and ecological respects, avoid strong selection pressure for a certain fungal strain as well (Finckh et al., 2000). However, in a long-term scenario, cultivar mixtures might also give selective force to pathogen adaption which was independent of the cognate virulence gene in isolates.

Tillage practice can change soil circumstance, for soil-borne fungus, sequential alterations in soil texture, temperature, moisture and aeration can significantly influence their activities and epidemics as well (Janvier et al., 2007). A large body of work has emphasized that reduced tillage activities were more effective against soil-borne diseases, while with a neutral or even negative impact on foliar disease control (Bailey et al., 1992, Janvier et al., 2007). In some cases, despite less wheat powdery mildew ratings were observed under a no-tillage system compared with conventional tillage or ploughed application, but results were not consistent

over a long-term field study (Bailey et al., 1992, Elen, 2002, Ditsch & Grove, 1991). Even so, the potential of tillage in foliar disease control is still valuable. As powdery mildew can persist on stubbles, so stubbles or green volunteer carry-over are disease potential for next season. Tillage that reduces stubble is supposed to reduce the presence of inoculum. In addition, tillage affects content of soil organic reserves and below-ground root development, these changes directly relate to crop growth which may modify tolerance towards foliar pathogen (Munkholm et al., 2008, Havlin et al., 1990).

Balanced nutrition is crucial for plant resistance to biotic stresses, plants suffering nutrient limits tend to be more disease susceptible than those away from malnutrition (Amtmann et al., 2008, Walters & Bingham, 2007). Farmers can resort to fertiliser use to enhance plant immunity, enabling disease damage below a designated economic threshold (Dordas, 2008). But not all nutrients are expected to be effective. For example, nitrogen in plant growth is profound and becomes a constraint in crop production in many agricultural regions (Eickhout et al., 2006, Spiertz, 2010), but excess nitrogen can increase plant susceptibility. This is especially well-accepted in powdery mildew case, where wheat varieties received nitrogen rate above recommendation were more vulnerable than that planted at low nitrogen levels (Sander & Heitefuss, 1998, Chen et al., 2007, Olesen et al., 2000, Olesen et al., 2003a, Oerke & Schönbeck, 1990). This is because more nitrogenous matter will be accumulated in leaves, which could be taken in favour of multiplication and development of causative powdery mildew (Olesen et al., 2003b, Hoffland et al., 2000). Moreover, extra nitrogen can interfere the production of phytoalexin like phenolics, and weaken the physical defence of host cell walls (Dordas, 2008).

Interestingly, use of some nutrients alone plays a control role, but when supplied with other nutrients the nutrient interactions may increase disease incidence. A number of studies has seen effectiveness of phosphorus for powdery mildew control in many horticultural and grain crop species (Mitchell & Walters, 2004, Reuveni et al., 1996, Reuveni et al., 1998, Mostafa & El Sharkawy, 2017). This is claimed to associate with induced systemic resistance, where phosphate could boost activities of endogenous enzymes such as peroxidase and lipoxygenase (Walters & Bingham, 2007). However, combination of phosphorus and nitrogen fertilisers made wheat even more susceptible to powdery mildew (Rowaished, 1980).

Other macronutrients such as potassium (Perrenoud, 1977) and sulphur (Hussain & Leitch, 2005), and micronutrient silicon were also reported to reduce wheat powdery mildew. Silicon

is not proposed as a generally essential element, but its prophylactic role in enhancing plant resistance to fungal diseases has drawn great attention (Ma, 2004, Debona et al., 2017). Ample literatures have suggested that silicon induced resistance closely connects to one such localized deposition of silicon-containing substance in epidermal cells of leaves, this reinforces physical strength against foreign objects (Guevel et al., 2007, Belanger et al., 2003). Apart from that, osmotic effects of silicate solution and increased level of fungitoxic materials, e.g., phenolics, phytoalexins and momilactones on pathogen activities are also a part of defence (Luyckx et al., 2016, Cote-Beaulieu et al., 2009). In mildewed wheat, phenolic-like (PhL) deposits in epidermis of wheat after silicon treatment have been identified to restrict fungal further invasion (Remus-Borel et al., 2005).

2.4.3. Arbuscular mycorrhiza, an eminent source for PM control

2.4.3.1 Background context of arbuscular mycorrhiza

Arbuscular mycorrhizal fungi (AMF) are ubiquitous soil microorganism existing in the majority of habitats. Being important members of soil micro biota, they are present in roots of over four-fifths of terrestrial plants with high species richness (Hamel, 1996). AMF are characterised endomycorrhiza by way of interaction with root cells of hosts and morphology, because their hyphae are inside cells in the roots and form specialised intracellular structures vesicles and arbuscles (Bonfante & Genre, 2010). It used to employ term vesicular arbuscular mycorrhizae (VAM), now AMF instead since not all endomycorrhiza form vesicles but all produce arbuscules (Strack et al., 2003). These attributes distinguish it from ectomycorrhiza, another broad type of mycorrhizae, of which fungal hyphae do not penetrate root cells but occupy intercellular space. AMF are group of beneficial fungi, form widespread intimate symbiosis with host plants, of which history is revealed by fossil evidence and dated to more than 400 million years ago (Remy et al., 1994). This reciprocal association has prompted application of AMF in plant improvement for agricultural production. Advantages taken from AMF symbionts are multifaceted, mostly known for enhancement of nutrient and water access, and better performance towards many external stresses (Marschner & Dell, 1994, Birhane et al., 2012, Sheng et al., 2008, Xavier, 2004). Whereas in exchange, AMF obtain photosynthetically carbon, predominantly glucose and lipids from hosts to complete their growing and multiplication (Shacharhill et al., 1995, Luginbuehl et al., 2017, Helber et al., 2011). This is attributable to their obligate biotroph scenario, strictly dependent on feed from their green hosts.

Entry to root epidermis starts from attachment of elongated germ tubes of fungal spores and extended mycelium on root surface (Peterson & Bonfante, 1994), results in formation of hyphopodia (Bonfante & Genre, 2010). Structure so called prepenetration apparatus (Bonfante & Genre, 2010) is then produced beneath hyphopodia to guide fungal hypha into cortical cells, this eventually forms highly branched arbuscule which is the key site for nutrient exchange between two symbiotic partners (Harrison, 2005). In the bulk soil, extra-radical mycelium (ERM) develops and establishes giant underground mycelial network many times (up to 40 times) the size of plant root system (Pepe et al., 2018). Fine ERM network explores soil to increase host water and nutrient uptake in particular P away from P-depletion zone.

2.4.3.2. Response of mycorrhizal plants to fungal disease

Bio-protection role of AMF against deleterious pathogens is a matter of considerable interest. Crop biotic stress tolerance can be evoked by the exploitation of AMF, especially against soil-borne pathogens. Reduction of root fungal disease by AMF has been reported in an array of plant species, to name a few, *Fusarium* in tomato (Akkopru & Demir, 2005) and common bean (Eke et al., 2016), *Phytophthora nicotianae* var. *parasitica* in tomato (Trotta et al., 1996), *Rhizoctonia solani* in oxeye daisy (Lewandowski et al., 2013). Singh et al. (2000) reviewed previous study in root pathogenic fungus-AMF interactions; AMF were found to effectively alleviate infection in most cases, however, no effect and occasional increase in severity were also present. Comparatively, studies of plant response to aboveground fungal disease to AMF is less addressed, and AMF is more likely to be protective in disease caused by necrotrophic fungi than biotrophic ones (Jung et al., 2012).

Conversely, enhanced disease symptom is often found in mycorrhizal plants attacked by foliar biotrophic fungus (Pozo & Azcon-Aguilar, 2007). With respect to powdery mildew, effects of AMF on disease from bibliographic data are really inconsistent. Disease reduction of powdery mildew has been recorded in mycorrhizal pea (Singh et al., 2004), barley (Molitor et al., 2011), wheat (Mustafa et al., 2017) and Begonia hiemalis (Feldmann & Boyle, 1998). Mustafa et al. (2016) experimentally examined control of wheat powdery mildew by AMF, protection comprised of increased peroxidase activity in both roots and shoots, papillae formation and reduced pathogen haustorium. This is confirmed by their follow-up work, a strong accumulation of polyphenolic compounds and H_2O_2 was manifested at penetration site in AMF wheat, as well as increase in expression of defence marker genes such as phenylalanine ammonia lyase (PAL) and OsNPR1(Mustafa et al., 2017). However, even with higher activity

of peroxidase, mycorrhizal standing milkvetch still suffered more infection than the control (Liu et al., 2018). AMF colonization underground may not be the determinant in disease control, better AMF colonization but came with less prevention from powdery mildew has been noticed in wheat (Mustafa et al., 2016). Mycorrhiza colonization though is required for MIR, however might not be sufficient to activate mycorrhiza-induced resistance (MIR). This is supported by Mora-Romero et al. (2015), he found that normal AMF establishment did not confer leaf MIR when PvLOX2, an oxylipin biosynthetic gene involved in JA biosynthesis was silenced in roots of common bean. Since PvLOX2 is involved in AMF-induced signal translocation from roots to shoots, disruption of signalling process will terminate activation of leaf MIR. These together suggest that AMF colonization is essential for MIR but not a guarantee, because MIR requires systemic signalling to be activated and spread in parts distal from roots. On the other hand, Mustafa et al. (2016) found that AMF species was more significant than its colonization level in determining wheat response to powdery mildew. Despite proof of impact of AMF species on disease control, no AMF specificity for that was also noted. This conflict has been found in, for example Fusarium oxysporum f. sp. cubense in banana, two AMF species had no difference in lessening damage (Jaizme-Vega et al., 1998). Although much has been done in investigating AMF for plant disease control, those variable evidences imply a need to evaluate a specific AMF species in a specific case.

Chapter 3: Genome-wide association mapping for adult resistance to powdery mildew in common wheat

3.1. Introduction

Biotic factors, especially weeds and diseases, are major growth constraints for crop production. Powdery mildew (caused by *Blumeria graminis* f. sp. *tritici*) is an important foliar wheat disease occurring in most places where wheat is grown, from tropical areas near the equator to temperate latitudes as high as 60°N and 44°S (Singh et al., 2008). Infection with powdery mildew can cause yield reductions of up to 40%, particularly under humid rainfed and irrigated high input conditions (Bennett, 1984). To meet future food demands, it has been estimated that wheat yield increases of 2.4% per year are required by 2050, however only approx. 0.9% yield increases are being achieved (Ray et al., 2013). Therefore reduction in wheat production due to powdery mildew greatly challenges food security.

To reduce powdery mildew disease, the use of resistant varieties is most effective within a comprehensive disease management program and can minimize both financial and labour inputs for growers. Plant resistance to diseases is typically classified as "qualitative", being dominated by a major resistant gene (R gene), or "quantitative", in which it is manifest by resistance-related QTL (quantitative trait loci) (Pilet-Nayel et al., 2017). Wheat powdery mildew R genes can be highly effective and are the foundation in resistance breeding programs that have led to many commercial wheat varieties. However, the functional mode of R genes in a gene for gene system (Flor, 1971), in which specificity of R gene to avirulence genes from a complementary pathotype, imposes strong selection pressure on the pathogen population. Evolving pathogens will eventually adapt to varieties carrying a particular R gene, leading to loss of resistance function (Golzar et al., 2016, Cowger et al., 2018). QR (quantitative resistance) to powdery mildew is frequently observed with several QTL contributing to the resistance. Lower selection pressure of QR on pathogens results in more durable, but partial resistance. In some cases, QTL with large effects can confer very strong resistance, for example the Lr34/Yr18/Pm38 locus on the short arm of wheat chromosome 7D (Lillemo et al., 2008). This locus explains up to 72% of the phenotypic variation for powdery mildew resistance and is also a resistance source for leaf rust and yellow rust. QTL with minor effects also contribute to resistance when combined additively with other QTL/genes. In fact, there is a great

abundance in minor-effect QTL detected in mapping studies (Niks et al., 2015, Pilet-Nayel et al., 2017), functioning collectively with major QTL.

Both linkage mapping (Asad et al., 2012, Mingeot et al., 2002) and association mapping (Liu et al., 2017b) have been used to identify QTL for wheat powdery mildew resistance. Conventional linkage mapping uses a segregating population derived from parents differing in performance for a trait of interest (Huang & Han, 2014). However, recombination events and genetic variation are limited in linkage mapping, only a few QTL could be detected. Advanced wheat populations such as MAGIC (Multiparent Advanced Generation Inter-Cross) can improve the resolution of linkage mapping (Huang et al., 2012a), but they also come with challenges in validation of map order and recombination analysis due to use of bi-allelic markers (Huang et al., 2015). Association mapping, known as GWAS (Genome-wide association studies), is conducted on diverse varieties instead and does not need the development of populations (Buckler & Thornsberry, 2002). It uses a larger sample size, which facilitates the exploration of more genetic variations, including those with small effect and allelic diversity (Juliana et al., 2018, Zhu et al., 2008). GWAS can provide higher resolution mapping for identification of candidate genes (Nelson et al., 2018).

By taking advantages of SNP (single-nucleotide polymorphism) for high-throughput genotyping, agronomic traits and disease resistance in wheat have been extensively studied. For example, QTL have been reported for yield-related traits (Wang et al., 2017), leaf rust/stripe rust/tan spot (Juliana et al., 2018), and eyespot diseases (Lewien et al., 2018). So far in wheat, a wealth of QTL have been discovered for powdery mildew resistance, distributed on all chromosomes (McIntosh et al., 2014, McIntosh et al., 2016, McIntosh et al., 2017, Liu et al., 2017b). However, the majority of these QTL result from bi-parental mapping with only limited attempts with GWAS (Li et al., 2019, Liu et al., 2017b). The association analysis conducted by Li et al. (2019) targeted disease reactions to certain *Bgt* isolates. Liu et al. (2017b) revealed twelve QTL at adult-plant stage with three of them being suggested as novel QTL, located on chromosomes 1A, 1B and 6A.

Detection of QTL, especially with minor-effects, varies between the environments and populations used, which offers opportunity to find new QTL under different experimental conditions. The current study aims to identify potentially novel QTL that are effective against powdery mildew at the adult plant stage, using 335 wheat accessions collected worldwide. With landmark projects such as high-density 90K SNP array (Wang et al., 2014a) and

Ensemble plants database (Bolser et al., 2016), potential candidate genes associated with markers identified from GWAS were investigated to address this aim.

3.2. Materials and methods

3.2.1. Plant materials and marker genotyping

A set of 335 wheat genotypes from different countries of origin were used to evaluate their resistance to powdery mildew. DNA was extracted from leaf tissue collected at the 2-leaf seedling stage from a single plant per accession and genotyped using the Illumina iSelect 90,000 SNP bead chip assay described in (Wang et al., 2014a). Genome Studio polyploid clustering V1.0 software (Illumina Ltd.) was used to export normalized NormR and Theta values for each accession for SNPs that produced well-separated clusters for unambiguous scoring and had been previously genetically mapped (Wang et al., 2014a). SNP genotype calling was performed using a custom Perl script that assigned a genotype to each accession based on the Euclidian distance of the sample data point to the centre of pre-defined clusters having known allelic relationships, considering the standard deviations of the defined clusters. A total of 38,379 SNPs was identified to be polymorphic in the population. For quality control, we first used the R package snpReady to prune markers with a call rate no greater than 5% and a maximum of 0.1 of MAF (minor allele frequency) and individuals with missing data in genotyping more than 50% were also excluded from the present study (Granato et al., 2018). Out of these, markers were manually removed either due to 1) no position information on a 90K wheat consensus map was available, 2) only having phenotype data for a single year, which resulted in 329 varieties for further association mapping. Missing value of markers were imputed by the synbreed package (Wimmer et al., 2012). The PIC (polymorphism information content) of each SNP marker assessing their genetic diversity was extracted from population genetic summary calculated in snpReady.

3.2.2. Resistance assessment for powdery mildew

Trials under field and glasshouse conditions were carried out at Mt. Pleasant Laboratory in Launceston, Tasmania, Australia over three years. In glasshouse experiments in 2016 and 2018, two plants of each of the 329 varieties were raised in small pots filled with pine bark/loam-based potting mix. Each experiment had two replications. Average temperatures for daytime and night in glasshouse were 25±2° and 16±2° respectively, no supplemental light throughout

experiment. A field trial comprising of three replicates was set up in the 2017 growing season. Each genotype was sown in a 120 cm row with row spacing of 35 cm. Crop management followed local practice. All the trials were infected by naturally spread powdery mildew which is a mixture of S509 wild type and T509 mutant (tested in Curtin University, Australia).

Phenotype data of powdery mildew disease was recorded at the anthesis stage. A modified disease rating scale of 0-5 as described in Sijaona et al. (2001), was used for disease evaluation on the whole plant as follows; 0 = indicated no visible symptoms, 1 = <1% leaf area colonized, 2 = 1-10% leaf area colonized, 3 = 11-25% leaf area colonized, 4 = 26-50% leaf area colonized and 5 = >50% leaf area colonized (Sijaona et al., 2001). Phenotype data that was subjected to GWAS consisted of mean response across replications of each wheat variety for each environment (year-location combination, three environments in total). Analysis of variance components for disease response was calculated using R software (https://www.r-project.org/) (R Core Team, 2014). To estimate broad-sense heritability (H^2) of each trait, an equation to calculate the ratio of genotypic variance to the total phenotypic variance was used as below:

$$H^2 = \sigma_g^2/(\sigma_g^2 + \frac{\sigma_{gl}^2}{nl} + \frac{\sigma_{gy}^2}{ny} + \sigma^2/(nl * ny))$$

where nl is the number of locations, ny represents number of years. Genotypic variance (σ_g^2), genotype × location (σ_{gl}^2), genotype × year (σ_{gy}^2) and residual (σ^2) variances were obtained from linear mixed model fitting to ascribe random effects to genotype, location, year, the interaction of these factors and replication nested in "year-location" using lme4 package (Bates et al., 2015). Additionally, BLUE (best linear unbiased estimator) across three environments for each variety was also computed for GWAS, in which genotype accounted for fixed effects in the linear mixed model.

3.2.3. Linkage disequilibrium and population structure

Squared correlation coefficient (r^2) was used to indicate LD (linkage disequilibrium), ranging from 0 to 1; the higher r^2 indicates stronger LD between tested markers. The r^2 for pairs of loci along the same chromosome was calculated with the LDcorSV package (Mangin et al., 2012). The ggplot2 package was used to visualise the local LD patterns (Ginestet, 2011), plotting r^2 against corresponding genetic distance among these intra-chromosome SNPs. Curve of LD decay over distance was fitted by nonlinear regression per chromosome in R, following

adjustments based on the Hill and Weir the expected value of r^2 was calculated as below (Remington et al., 2001, Hill & Weir, 1988):

$$E(r^2) = \left[\frac{10 + C}{(2 + C)(11 + C)}\right]\left[1 + \frac{(3 + C)(12 + 12C + C^2)}{n(2 + C)(11 + C)}\right]$$

where n is the sample size, and C is a coefficient for the genetic distance to be estimated. A cut-off value of $r^2 = 0.2$ was used to determine LD decay, adjacent SNPs with $r^2 > 0.2$ were assumed to be in LD and likely co-segregate. Wheat collections comprising diverse genotypes have complex genetic backgrounds and the presence of population stratification and cryptic relatedness among individuals (i.e., kinship, the "K matrix") could be problematic to detect true MTA (marker-trait association) (Hoffman, 2013). To address this, different methods were used to determine structural effects (e.g., subpopulation Q matrix, Principal component of marker genotypes) and K matrix in association models. The GAPIT (Genome Association and Prediction Integrated Tool) package implemented in R was chosen for computing both population and family structures. Its ability to exploit EMMA (efficient mixed model association) and P3D (population parameters previously determined) algorithms to improve statistical power and computational efficiency was also a bonus (Hoffman, 2013, Kang et al., 2010, Kang et al., 2008). For population correction, PCA (principal component analysis) was calculated using SNPRelate package (Zheng et al., 2012), the optimum number of PCs (Principal components) was fixed in association models.

3.2.4. Genome-wide association analysis

For GWAS, an ECMLM (enriched compressed mixed linear model) approach has been made in GAPIT to optimize traditional MLM (mixed linear model) (Tang et al., 2016). ECMLM association model was carried out for three environments (year-location combination) as well as BLUEs, with or without the optimum number of PCs included. A chi-squared test based naïve model was also investigated as a baseline model to assess the fitness of above association model for each trial and BLUEs. Q-Q (quantile-quantile) plots (expected $-\log_{10}(P)$ values to observed $-\log_{10}(P)$ values) were created to check the distributions of $-\log_{10}(P)$ values generated from each test, together with genomic inflation factor, denoted by λ_{GC} using package GenABEL for model evaluation (Aulchenko et al., 2007). Genomic control factor λ_{GC} was used to reflect inflation of the test statistic (Tsepilov et al., 2013), in which keeping its value close to 1 is desired (Sharma et al., 2018). A threshold of *P* value at 0.001 ($-\log_{10}(P) \geq 3$) was set up

to indicate significant MTA. *P* value and likelihood ratio based R^2 of model with and without marker were retrieved from GWAS output from GAPIT for each significant SNP, the difference in R^2 was approximate estimation of phenotypic variation explained by that SNP.

3.2.5. Pyramiding effects of positive/negative tag-SNP alleles on disease response

The allele at the locus of the detected SNP associated with increasing disease response is referred to as "negative allele", while an allele associated with reduced infection is the "positive allele". Based on genotype data for all individuals by a SNP and crude means of disease response of each variety across three environments, the effects of SNP bi-alleles were determined. A single putative QTL was assigned when detected intra-chromosomal SNPs were in LD ($r^2 > 0.2$) and with consistent direction of the effects. Best QTL-tag SNP was selected on the basis of *P*-values, allelic effects and explained variance (Maccaferri et al., 2015). Regarding all QTL-tag SNPs in our study, the number of positive alleles and negative alleles for each variety was summarized and connected to disease response by linear regression and Pearson's correlation analysis.

3.2.6. Search for putative candidate genes

The flanking sequence of detected SNP marker was used for a query against wheat reference genome assembly IWGSC (International Wheat Genome Sequencing Consortium) RefSeq v1.0 to obtain its position on the reference physical map (Appels et al., 2018). The interval was then subject to JBrowse (Skinner et al., 2009) from URGI (Unité de Recherche Génomique Info/research unit in genomics and bioinformatics) to explore candidate gene overlapping with this genomic region. For significant SNP which had no gene found in the same interval, 4000 base pairs were added to either side of the SNP to search for candidate gene. Potential candidate gene and its functional annotation were obtained from IWGSC Annotation v1.1. As annotation was not available for some wheat genes, gene sequence was then retrieved for a more reliable collinearity analysis and BLASTn implemented Ensembl search (http://plants.ensembl.org/index.html) (Kinsella et al., 2011) against *Triticum Urartu* (A-genome donor), *Aegilops tauschii* (D-genome donor), *Brachypodium distachyon* and *O. sativa Japonica.* Based on the query coverage, E-val and identity percentage (% ID), orthologous genes with highly similar sequences were considered for prediction of gene function in wheat.

3.2.7. Comparisons of QTLs with previous mapping results

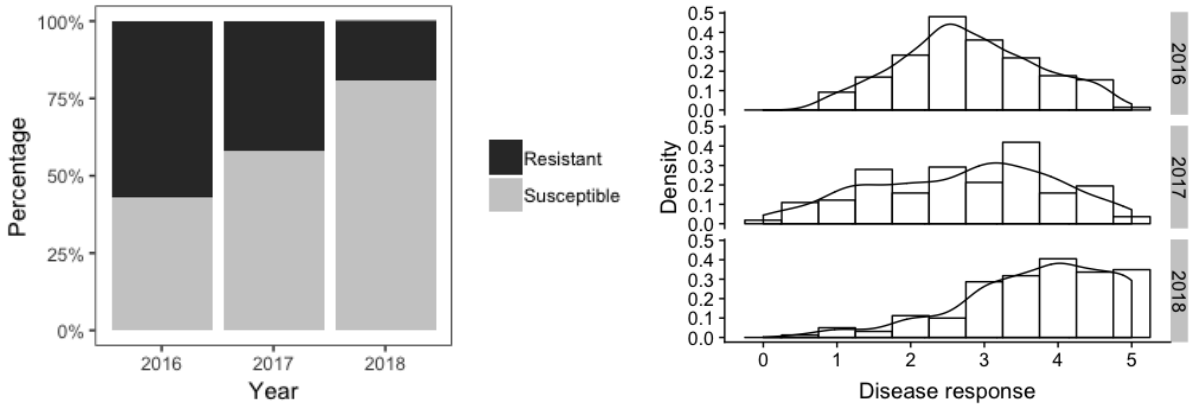
IWGSC RefSeq v1.0 is the first and also the latest repository of nearly complete wheat genome sequence at chromosome scale (Appels et al., 2018). Considering that most mapping studies were genetic map based and misalignment of some genetic markers to a sequence-based physical map, an integrated map by Maccaferri et al. (2015) was used for a rough mapping comparison. The integrated map was based on common markers across different genetic maps and incorporated multiple marker types including SNPs, GBS and SSR markers (Maccaferri et al., 2015), which enabled QTL comparisons across publications using different genotyping methods. To visualise mapping results, previously identified wheat Pm genes and QTL, together with QTL from our study, were projected onto an integrated map. The confidence interval of published QTL was determined by its flanking markers. If these were not given in the original reference, the peak was characterised by the closest flanking marker. For all QTL identified in our study, their peaks were represented by corresponding tag SNPs. The confidence intervals were obtained according to local chromosome LD decay, as ± D cM (D was genetic distance where chromosomal r^2 dropped to threshold 0.2) for QTL detected by a single SNP, with respect to QTL harbouring more than one SNP, while positions of these SNPs on integrated map were ordered to determine its confidence interval. The locations of wheat powdery mildew resistance genes (Pm genes and temporarily designated genes) were based on near flanking markers available in the integrated map.

3.3. Results

3.3.1. Wheat resistance to powdery mildew

Evaluation of the disease response of wheat in the three environments showed a wide variation in resistance between the 329 varieties (Figure 3.1). The average disease response of all wheat varieties for 2016 trial was 2.8 but was skewed towards a higher percentage of susceptibility, given there was an increased disease rating (3.7) during the 2018 trial (Table S3.1). The analysis of variance suggested that estimated variance components for genotype and the genotype x environment interaction were both highly significant ($P \leq 0.001$) for disease response to powdery mildew (Table 3.1). This was also reflected by correlations of disease response among three environments, where positive but moderate correlation coefficients (0.4-0.5) implied interaction between genotype and environment (Figure 3.2). Estimation of broad-

sense heritability for wheat resistance to powdery mildew in our study was 0.67 which suggested a moderate heritability.



(A) **(B)**

Figure 3.1: Phenotypic analysis across three environments in association panel. **(A)** Percentage of resistant and susceptible wheat varieties for the years 2016, 2017 and 2018. **(B)** Distribution of genotype means for disease response evaluated in three environments.

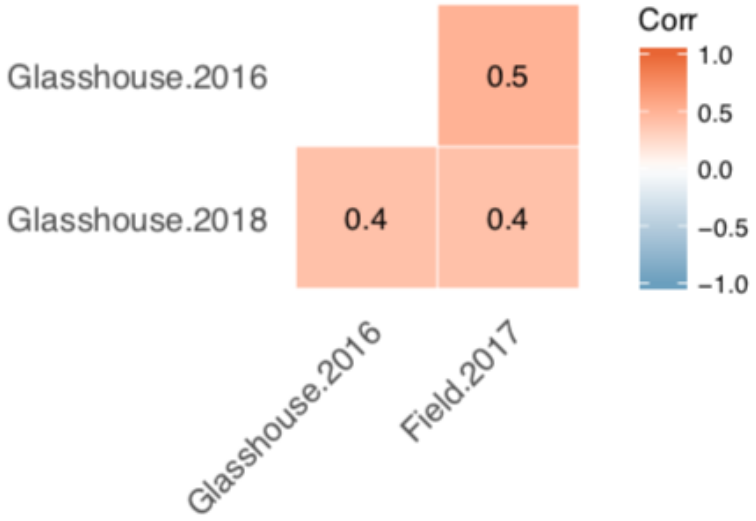


Figure 3.2: Pearson correlation coefficient of disease response among three environments.

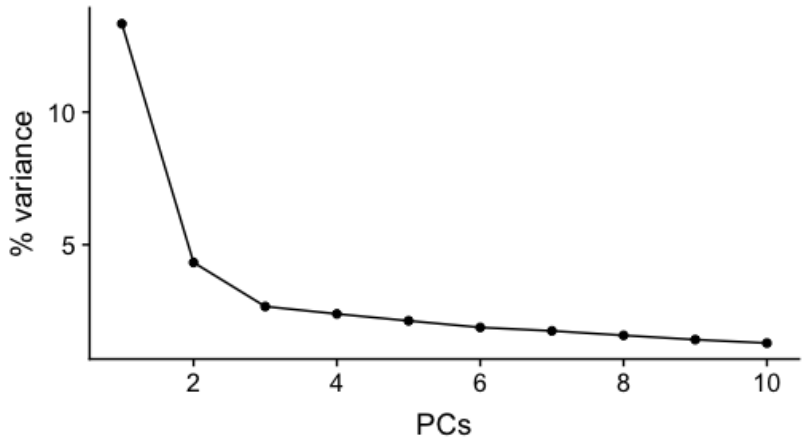
Table 3.1: Analysis of variance for disease response in three environments.

Effect	Df	F-value	P-value
Genotype	328	7.993	<0.001
Environment	2	316.904	<0.001
Genotype × environment	611	2.41	<0.001

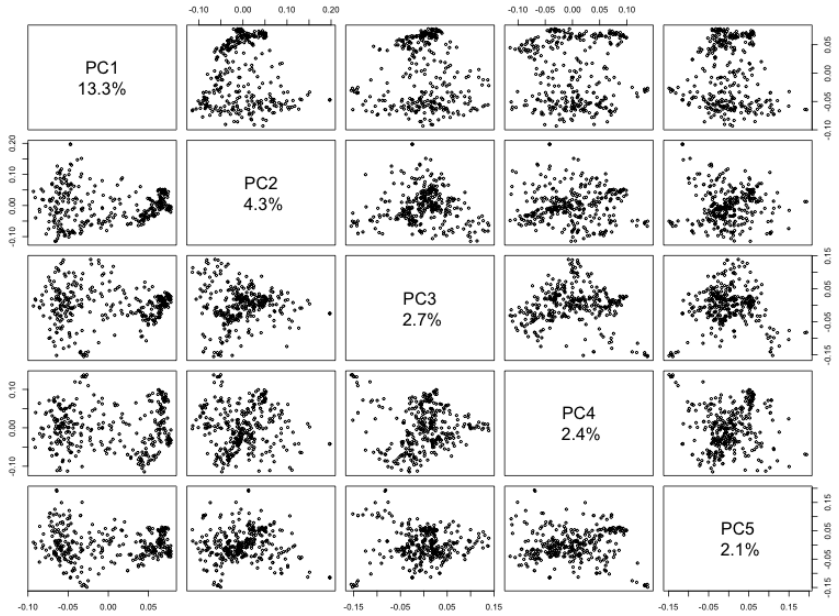
3.3.2. Genotyping data, linkage disequilibrium and PCA

All wheat varieties were genotyped with a dataset of 33381 SNPs using 90K Infimum SNP chip. After removing markers with MAF<0.1 (2406) and call rate < 95% (19441), 12718 SNPs remained. As 2161 SNPs out of these were unmapped to 90K consensus map, a total of 10557 SNPs covering all chromosomes with position information were used for the association study. More than half of SNPs were mapped on the B genome (5331) and fewest SNPs were found on the D genome (917) (Table S3.2). The average PIC value for A, B and D genomes was 0.31, 0.3 and 0.27 respectively (Table S3.2). The PIC value across all chromosomes varied from 0.25 to 0.38 with an average of 0.29 (Table S3.2). Most SNPs (~77%) were considered moderately informative with PIC value between 0.25 to 0.5 (Table S3.3) (Botstein et al., 1980). SNP showed an imbalance distribution on each chromosome with the number of markers ranging from 20 for chromosome 4D to 1051 for chromosome 5B (Table S3.2). All the markers spanned a genetic distance of 10684 cM in the wheat genome, with an average density of 1 marker/cM (Table S3.2).

The r^2 between adjacent intra-chromosomal SNP pairs was estimated and displayed in scatter plots against genetic distance (Figure S3.1) and the average rate of LD decay was also quantified for each chromosome. Considering a critical r^2 value of 0.2, the extent of LD varied from 1 cM to 28 cM (Table S3.2). The D sub-genome had the highest LD decay about 11.2 cM, triple the A (3.7 cM) and B (3.3 cM) sub-genomes. PCA was implemented to infer population structure for the total of 329 accessions. Both scree and pairwise plots implied that most variance was accounted for in the top two PCs, which explained 13.3% and 4.3% of the variance respectively (Figure 3.3). Since the first two PCs contributed to less than 20% of total variance, the effect of population structure in our study was not strong and the first two PCs only were retained in the association model.



(A)



(B)

Figure 3.3: Population structure analysis. **(A)** Scree plot for the first ten PCs and the amount of variance explained by each PC. **(B)** Pairwise scatter plot of the first five PCs.

3.3.3. Analysis of association scan

An ECMLM with adjustment for population structure and relatedness was selected to detect MTA; its fitness for wheat powdery mildew resistance was present by Q-Q plots for all environments (Figure 3.4). In general, no inflation of GWAS results by population structure or relatedness was evident, despite a slight overcompensation (black line below null-hypothesis

red line), which again supported that population stratification in our study was not significant. Since deviation from straight line was very small in Q-Q plot with only a drift at the end, and ECMLM correcting with PCA and kinship yielded inflation factor λ_{GC} across three environments between 0.95 to 1.0, model performance was fairly appropriate (Figure 3.4).

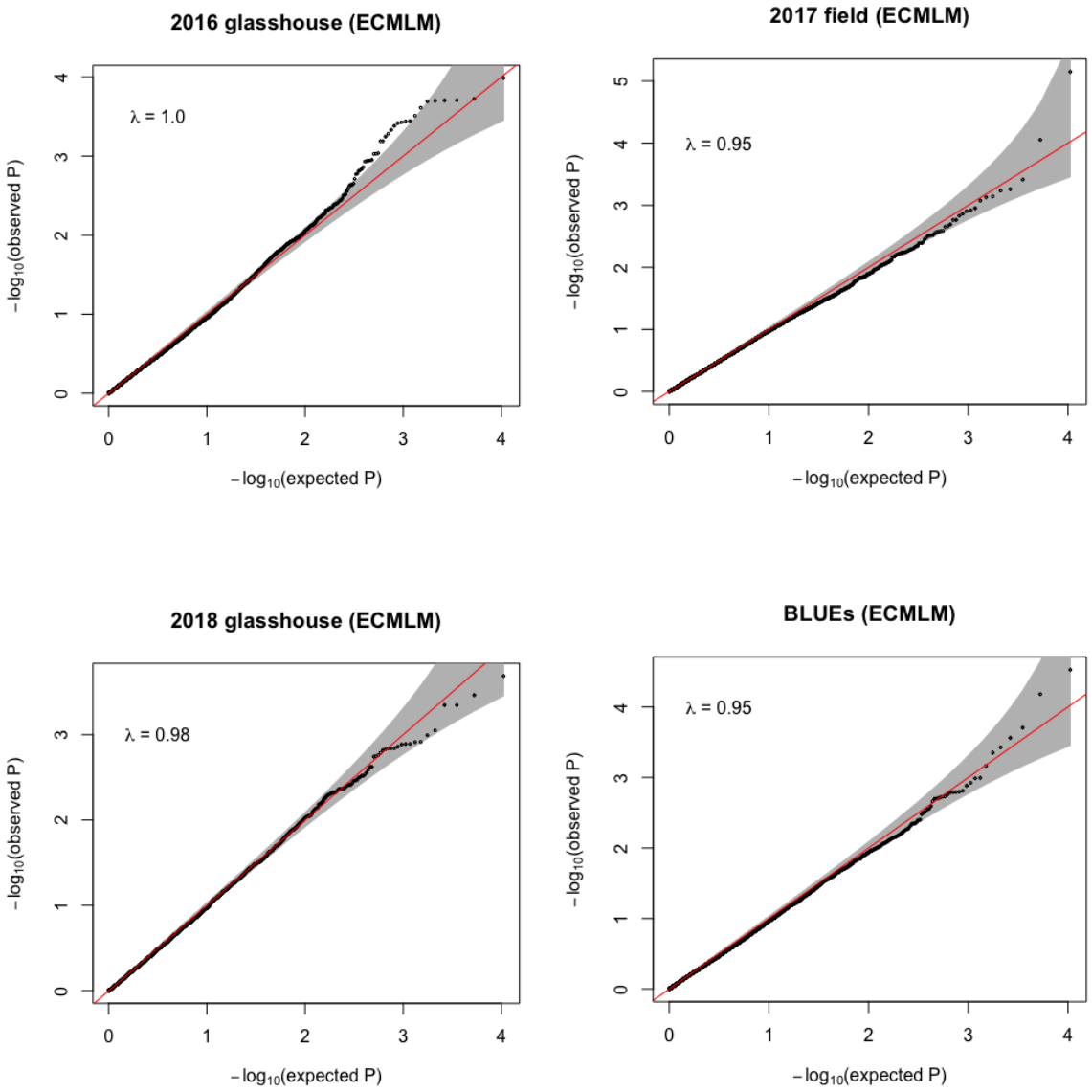
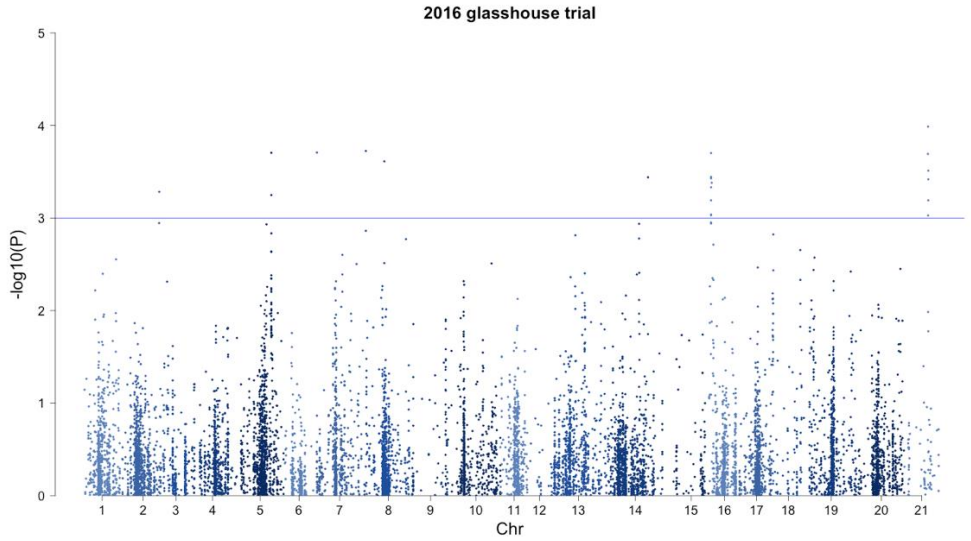
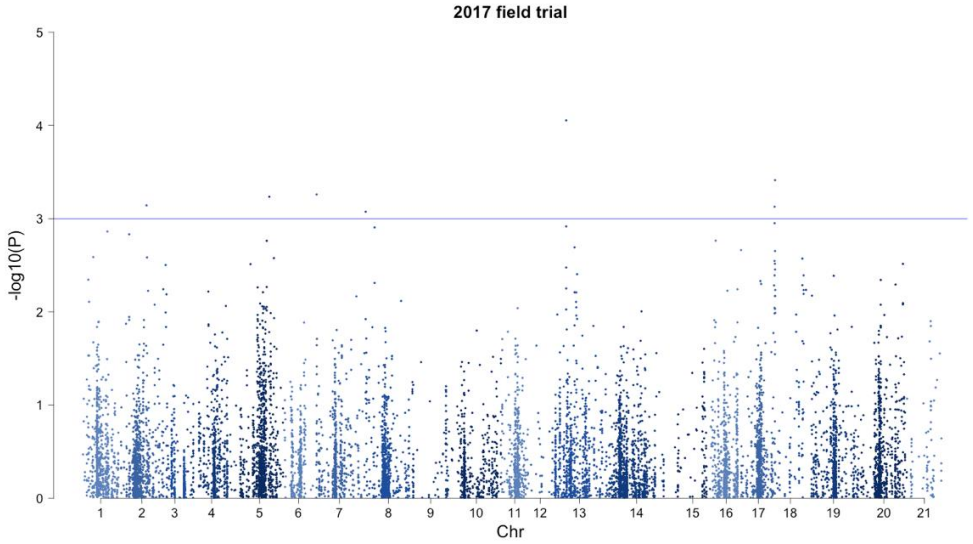


Figure 3.4: Q-Q plots of *p*-values generated from ECMLM model correcting for PCs and kinship for all test datasets. Red line indicates the expected normal distribution of *p*-values.

A total of 33 SNP hits on chromosomes 1B, 2B, 2D, 3A, 3B, 5A, 5B, 6A, 6B, 7D associated with powdery mildew resistance were identified from the three individual environments while considering population structure and relative kinship (Table S3.4), as shown in Manhattan plots (Figure 3.5). At a minimum, six associations each on chromosome 6A and 7D were detected

from a single trial. From the range of likelihood ratio based R^2, the SNP loci explained a small percentage of phenotypic variance. Of these, most SNPs were only present in one trial, with the exception of IWB67770 at position of 47.94 cM on chromosome 3A which was shared between 2016 glasshouse and 2017 field conditions. Inconsistent detection of MTAs across environments revealed QTL × environment interaction. Since there was poor repeatability of QTL among environments, association mapping based on BLUEs was also analysed. MTA tagged by IWB67770 was again detected by BLUEs, meanwhile two new associations to SNP IWB48794 on chromosome 3A and IWB59264 on chromosome 6D were uncovered, which made a total of 35 MTAs (Table S3.4). The most significant MTA was identified in 2017 field and coordinated to IWB74 (-log10(P) = 5.15) on chromosome 2D, described the largest variation among all detected SNPs. Based on position, LD, R^2 and effect direction of these significant SNPs, 20 QTLs and their representative SNPs (Tag-SNP) within QTL region were defined, details were shown in Table S3.4.


2016 glasshouse trial
-log10(P)
0
1
2
3
4
5
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
Chr


2017 field trial
-log10(P)
0
1
2
3
4
5
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
Chr

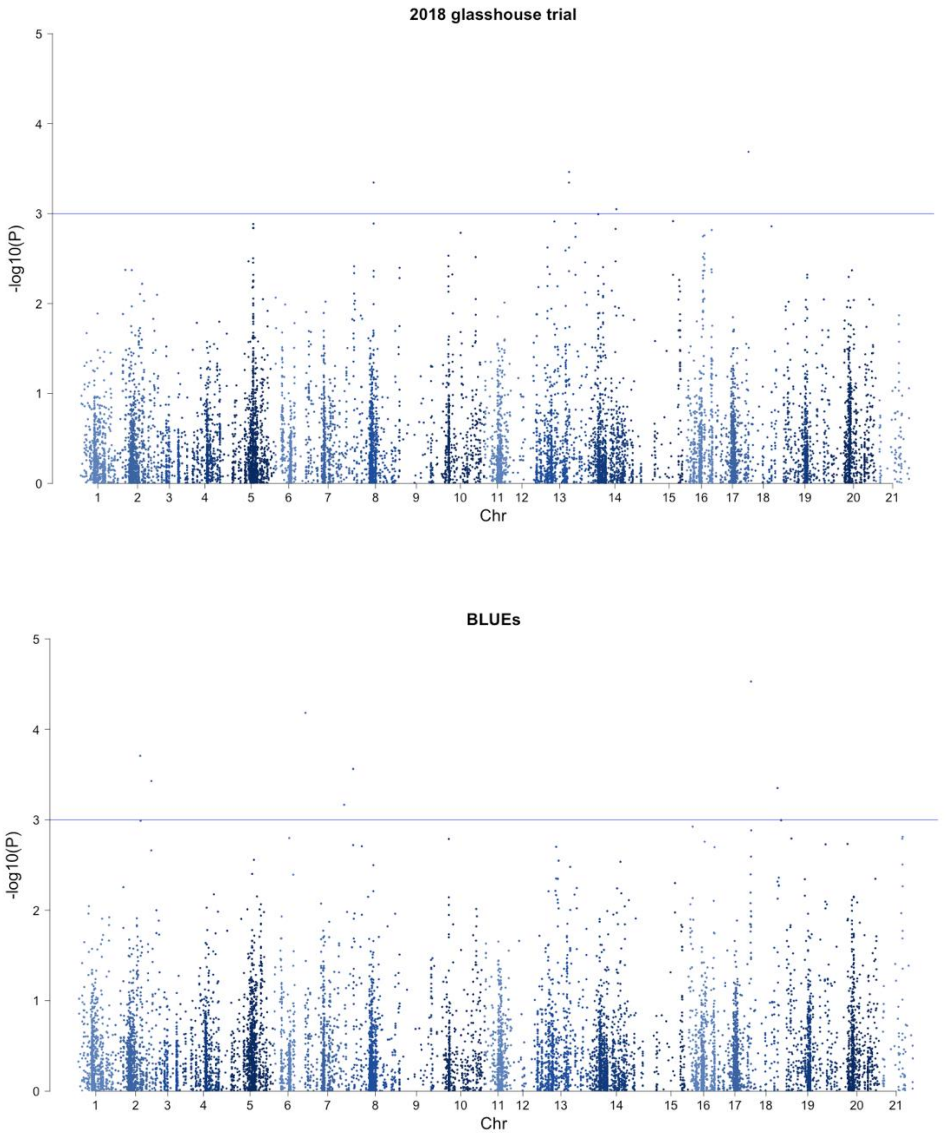
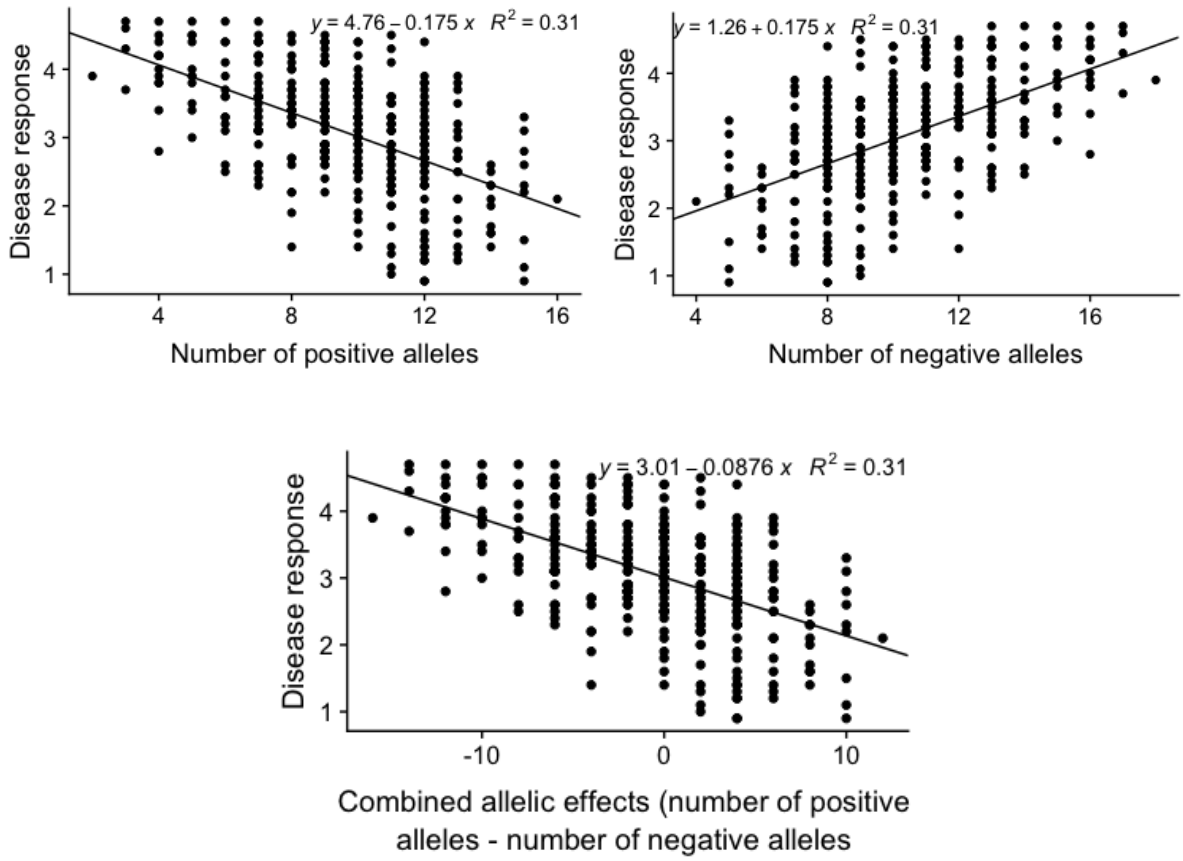


Figure 3.5: Manhattan plots of genome association scan for different environments as well as BLUEs. The horizontal blue line suggests the cut-off of $-\log_{10}(P)$ at 3.

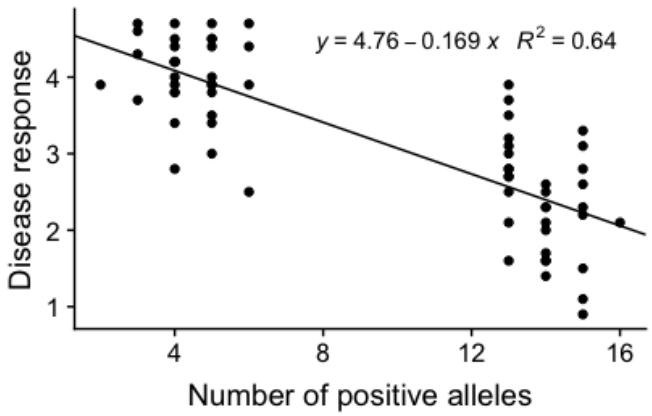
3.3.4. Cumulative effects of alleles on disease response

The number of positive and negative alleles of tag-SNPs was given for each individual. Most varieties had more negative alleles than positive alleles, this was supported by the ratio of resistant and susceptible varieties based on their average scores across three environments where less than half of them were classified as resistant type (Table S3.5). Up to 16 positive and 18 negative alleles were pyramided in KOPARA73 and X-258 respectively (Table S3.5). The difference in average disease response at a scale of 0-5 was 1.8; KOPARA73 showed superior resistance compared to X-258 with disease rating at 3.9. However, the number of

positive and negative alleles did not relate to increased resistance or susceptibility at least in some cases. As seen in H-137, for example, it had 12 positive alleles and 8 negative alleles but was much more susceptible (disease rating 4.4) than Hartog carrying 4 positive alleles, of which disease response rated 2.8 (Table S3.5). Even with these exceptions, varieties with more positive alleles in general have stronger disease resistance. Exploration of the relationship between the number of positive (negative) alleles and disease resistance was conducted by correlation analysis. Results suggested that allele number of both positive (correlation coefficient = -0.56) and negative (correlation coefficient = 0.56) ones associated with disease response. The scatter plot also indicated somewhat of a downhill (uphill) linear relationship between the number of positive (negative) alleles and disease response (Figure 3.6). Further relationships were estimated by linear regression models and in all cases better disease resistance was likely achieved by increasing positive alleles and decreasing negative alleles. This was best demonstrated by comparing the 10% genotypes with great or low number of positive alleles, strong correlation (correlation coefficient = -0.8) and way better fitness of linear model (R^2 = 0.64) proposed a significant dependency of disease response on number of positive alleles and additive effects of these alleles (Figure 3.6).



(A)



(B)

Figure 3.6: (A) Linear regression of disease response to the number of positive and negative alleles of tag-SNPs, as well as combined allelic effect. **(B)** Linear regression of disease response of 10% wheat varieties carrying high or low number of positive alleles of tag-SNPs.

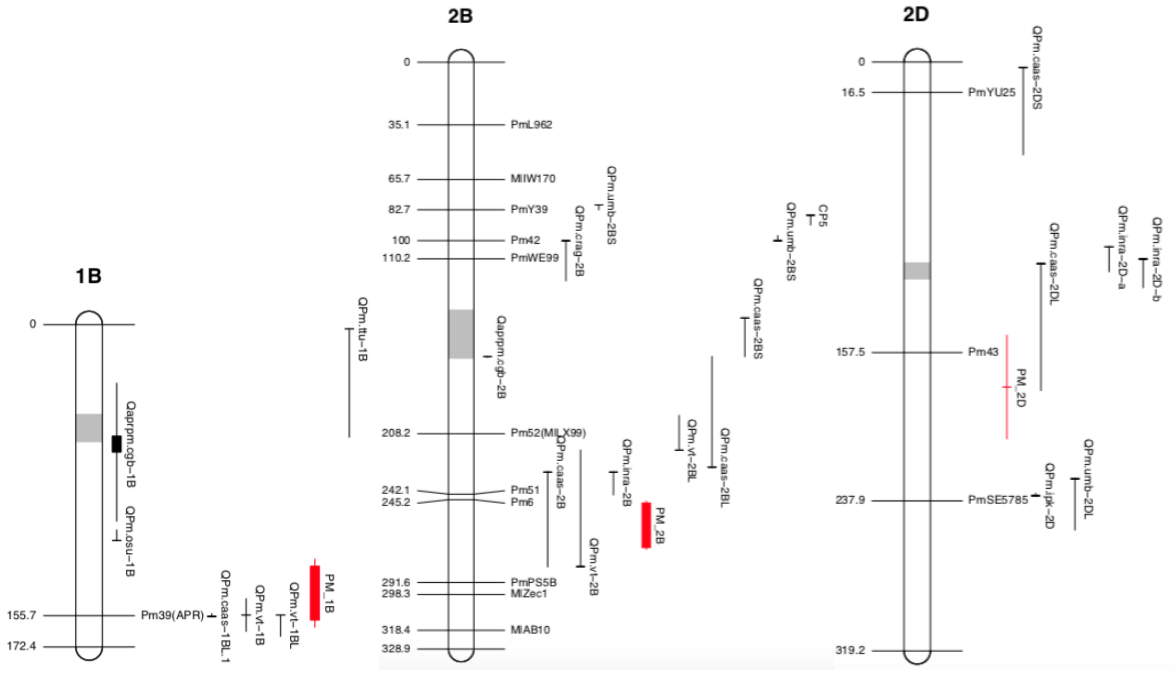
3.4. Discussion

Reliable genotype and phenotype data are key for accurate GWAS analysis. The SNPs in our association mapping were saturated among most of the chromosomes with a mean PIC value of 0.29 and large proportion of moderately informative markers indicated our association population had good genetic diversity for GWAS (Botstein et al. 1980). Analysis in degree of genetic variation and PIC for three sub-genomes was in parallel with previous findings (Liu et al. 2017a; Lopes et al. 2015), in which the D genome was reported to have the fewest polymorphic markers mapped to due to low frequency of recombination event (Chao et al. 2009). Resistance scores showed a high level of variation among trials and replicates, especially among the susceptible accessions, with 2018 trial showing generally severer symptoms than the other two trials. This led to relatively low correlations (r ranged from 0.37 to 0.49), which was mainly due the variation of some sensitive genotypes. For example, the resistance scores of B-T-18 ranged from 2 in 2016 to 5 in 2018. However, some resistant lines showed resistance in all the trials/replicates, with no symptom scores being over 2 (Supplementary Table 1). Thus, it is crucial to obtain phenotypic data from multiple trials.

PCA was used to infer population stratification where top PCs reflect genetic variation due to population structure. Small fraction of total phenotype variation explained by PC1 and PC2 suggested an insignificant effect of population stratification on genetic variation. This was also supported by slightly deflated Q-Q plots for ECMLM without correction for population structure. Q-Q plot offers a simple explanation for the distribution of P values; deflation of Q-Q plot is often justified as a result of overpowered association model, mostly attributable to the control of population structure that some other association mappings also encountered (Alomari et al. 2019; Cao et al. 2016). However, the pattern of Q-Q plot and GWAS result was little changed after excluding ancestry representative PCs (Supplementary Table 6), as seen in ECMLM without PC correction (Supplementary Figure 2), therefore the population structure might not be the reason for overfitting. For this reason, PCs were retained in the ECMLM. Although the kinship was reported to be sufficient and effective to control false positives (Pasam et al. 2012; Sharma et al. 2018), it was also unlikely the case in our study as per deflations from naïve models which assumed no any covariate (Supplementary Figure 3). To confirm MTAs from three individual environments, we further applied association study on BLUEs and some consistent results supported model reliability.

3.4.1 Comparison of identified QTL to previous publications

Copious powdery mildew resistance related QTL have been documented from bi-parental linkage mapping, spread-out over all chromosomes. Based on locations of 20 QTL, 14 putative QTL were further defined and discussed (Table 3.2, Figure 3.7). Figure 3.7 shows the position of all these QTL in comparison with reported genes/QTL. Most of the QTL identified from this study were located at similar positions to those previous reported genes/QTL. The QTL on 1B (PM_1B) was located at similar positions to QPm.vt-1B (Liu et al., 2001), QPm.vt-1BL (Tucker et al., 2007), QPm.caas-1BL.1 (Jia et al., 2018) and an adult-plant resistance (APR) gene Yr29/Lr46/Pm39 which is a pleiotropic gene responsible for the resistance to not only powdery mildew but also stem rust and stripe rust (Lillemo et al., 2008). Another QTL (QPm.osu-1B) has also been reported close to this region (Chen et al., 2009). The relationship between PM_1B and Pm8 (Mohler et al., 2001) and Pm32 (Hsam et al., 2003) were not clear as the exact positions of Pm8 and Pm32 on 1BL are not available. Resistance genes and adjacent QTL have been suggested to function as a resistance cluster (Kou & Wang, 2010), so there is likely a segment rich in powdery mildew resistance sources in the distal end of chromosome 1B.



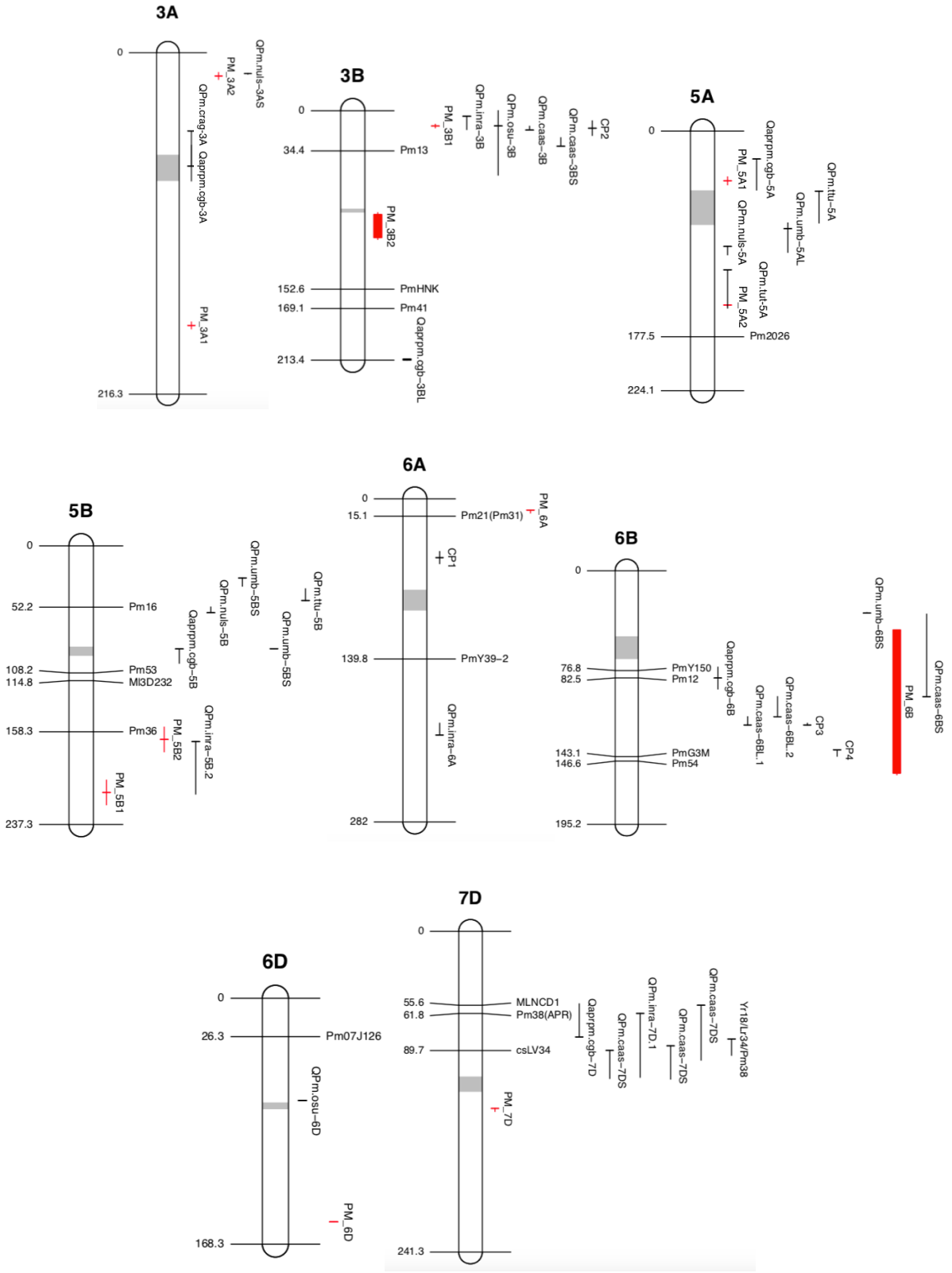


Figure 3.7: Chromosome locations of wheat powdery mildew resistance associated QTL (red bars) identified in this study. The positions of previously reported powdery mildew resistance genes and QTLs are estimated from their flanking molecular markers from published integrated map (Maccaferri et al., 2015).

Chromosome 2B is known for comprising the most powdery mildew resistance genes and QTL so far; the chromosome region where PM_2B mapped to has also localised several other QTL and catalogued Pm genes (Figure 3.7). PM_2B is in the QTL interval of QPm.vt−2B (Liu et al., 2001) and QPm.caas−2B (Liang et al., 2006), close to QPm.inra-2B (Bougot et al., 2006), Pm 6 (Qin et al., 2011) and Pm 51 (Zhan et al., 2014). A similar case was also seen in PM_2D on chromosome 2D as it partially overlapped with several reported QTL (Lan et al., 2010) and Pm 43 (He et al., 2009). PM-3A2 is located at the same position to the previously reported QPm.nuls-3AS (Lillemo et al., 2008). Positions of QPm.sfr-3A (Keller et al., 1999) and Pm 44 (Chen et al., 2011) (unpublished work) on the short arm of 3A are not available due to the lack of markers which can be located to integrated map. PM_3B1 is expected to be similar to QPm.osu-3B (Chen et al., 2009), CP2 (Marone et al., 2013), QPm.caas-3BS (Jia et al., 2018) and QPm.inra-3B (Bougot et al., 2006). The resistance loci on chromosome 5A and 5B identified in this study were all found in regions of QTL from previous linkage mapping studies. Pm21, introgressed from an alien source *Haynaldia villosa,* physically resides in chromosome bin 6AS 0.67-1.00 (Xie et al., 2012a) which is very close to PM_6A. PM_6B shared a similar position with QPm.caas-6BS, Pm54 (Hao et al., 2015) and a few other reported QTL.

Several new QTL were identified in this study. PM_3A1 is located on the long arm of 3A. However, the chromosomal-assignment of PM_3A1 on physical assembly was 7D, which was inconsistent with the genetic map. The inconsistencies of genetic markers in position and chromosome information between genetic map and sequence-based physical map have been reported, possibly due to genotyping error in genetic map or issues in physical assembly (DeWan et al., 2002). Further studies are needed to confirm the position of PM_3A1. PM_3B2 was mapped to centromere region, unlikely to be linked to any Pm gene or QTL reported. PM_6D was mapped to the end of long arm, possibly a novel QTL. As a few previously reported genes/QTL on chromosome 7D lack marker information and/or position on the chromosome, these were not able to be projected onto the integrated map. Thus it is unclear if PM_7D detected in this study coincides with any of those early findings.

3.4.2. Mining gene candidates underlying potential new QTL

PM_3B2 was in the coding region of a high confidence gene TraesCS3B02G398200, a member of major facilitator superfamily (MFS). MFS is known to play a pivotal role in transmembrane transport of a wide spectrum of ions and solutes and their homeostasis (Haydon & Cobbett,

2007, Li et al., 2014a), found to enhance plant salinity and drought tolerance in *Arabidopsis* (Remy et al., 2013, Cubero et al., 2009). It has also been implicated in plant-pathogen interactions and now accepted as a new class of plant-defence related proteins (Simmons et al., 2003). The functions of MFS in plant were first characterised in maize, exemplified by the corn leaf blight pathogen induced MFS *Zm-mfs1* (Simmons et al., 2003). Its defence strategy involved mutual export of antimicrobial toxins in pathosystem, as well as plant potassium pumps which helped plant re-uptake potassium in response to pathogen attack. On the other hand, in causative agents, MFS transporter appeared to be a determinant for pathogenesis and virulence (Lin et al., 2018, Chen et al., 2017). Pathogens are able to regulate oxidative stress and fungal multidrug resistance through MFS (Chen et al., 2017). One of the mechanisms for improved resistance to azoles is the control of fungicide efflux by fungal MFS and ATP-binding cassette transporters (Frenkel et al., 2015, Kretschmer et al., 2009). Azoles and strobilurin represent the predominant fungicide classes used for major foliar disease control including wheat powdery mildew. Disrupted MFS in wheat *Mycosphaerelle graminicola* evidently increased sensitivity to strobilurin (Roohparvar et al., 2007). Intriguingly, an earlier transcriptomic analysis in wheat reported a gene of MFS giving distinct expression trends in powdery mildew resistant and susceptible genotypes before inoculation (Xin et al., 2012). The dual roles of MFS in both counterparts of plant-pathogen system highlight the disease control strategy based on harnessing plant MFS transporters to enhance innate resistance, and inhibition of fungicide efflux MFS in pathogen.

GWAS scan of BLUE values identified a QTL *PM_3A1* on the long arm of chromosome 3A, however there was no available annotation of gene TraesCS7D02G190300 in this region. According to its orthologous gene in *Aegilops tauschii*, TraesCS7D02G190300 is predicted to be myosin-J heavy chain-like protein. TraesCS6D02G401500 harboured by QTL *PM_6D* is from peptidase family M3. Function of this protein family in plant was poorly understood. As proteases from M3 family were witnessed by their presences in an early immune response to fungal infection by biotrophic *Puccinia recondite* and necrotrophic *Stagonospora nodorum* in wheat (Balakireva et al., 2018), peptidase family M3 could be a potential candidate involved in wheat defence against powdery mildew. As for PM_7D detected in 2016 glasshouse trial, four wheat genes were found in the region with two of them being annotated. TraesCS7D02G310200 is a B56 subunit of protein phosphatase 2A (PP2A). Trimeric protein complex PP2A belonging to the family of serine/threonine phosphatase comprises of a scaffolding subunit A (PP2Aa), a variable regulatory subunit B (PP2Ab) and a catalytic subunit

C (PP2Ac) (Janssens & Goris, 2001). Significantly, there are multiple isoforms of B and C subunits in plant and a diverse combination of PP2A complex has shown regulation in plant development or response to multiple abiotic and biotic cues (Yu et al., 2003, Xu et al., 2007, Ahn et al., 2011, Durian et al., 2016). PP2Abs play a key role in directing the phosphatase to specific substrates and subcellular locations (Ballesteros et al., 2013), studies in PP2Abs were mostly performed on mammals while very little is known about their functions in plant. In *Arabidopsis*, PP2Ab has three subunit families based on the functional domains including WD-40 repeat (B), B56 domain (B') and EF-hand domain (B'') (Farkas et al., 2007). B56-regulated PP2A in *Drosophila melanogaster* was required for preventing apoptosis (Li et al., 2002). However, B'-type regulatory subunit in wheat is currently not well characterised. Identification of *Aegilops tauschii* resistance genes has been suggested to help create durable resistance genes in wheat (Andersen et al., 2015). As TraesCS7D02G291700 is orthologous to F775_19584 encodes disease resistance protein, we hypothesize that TraesCS7D02G291700 participated in wheat response associated with resistance to powdery mildew.

3.4.3. Application of detected QTL to breeding

Like most other quantitative traits, wheat powdery mildew resistance is also sensitive to environments, as indicated by only one QTL (5%) which is PM_3A2 that appeared in both 2016 glasshouse and 2017 field environments (Table S3.4) among the 20 QTL (undefined) detected in current study. QTL × environment interaction was found in many previous studies, where QTL effect magnitudes and detections were inconsistent across different environments. For example, environment-specific QTL have been identified for both rice plant height and heading date (Li et al., 2003). A yield QTL on wheat chromosome 3B was also reported to be highly dependent on environmental conditions (Parent et al., 2017). Such inconsistent detection of QTL due to genotype by environment interaction has important implications on manipulating QTL for resistance breeding, because it relates to the efficiency and stability of resistance under different conditions.

Therefore, the use of phenotypic data from multiple years and environments are necessary for detecting QTL which can be effectively used in breeding programs for improving wheat powdery mildew by MAS.

3.5. Conclusions

This study has demonstrated that GWAS is a useful tool for studying quantitative traits to discover responsive genes in a genetically diverse background, as a complement to linkage mapping at the population level. We have identified 14 putative QTL associated with wheat powdery mildew resistance. Most of the QTL were located on similar positions to previously reported genes/QTL for powdery mildew resistance. PM_3A1, PM_3B2, PM_6D and PM_7D were most likely novel QTL. These QTL need to be further confirmed through segregating populations.

Chapter 4: The potential of arbuscular mycorrhizal fungi in wheat PM control

4.1. Introduction

Arbuscular mycorrhizal fungi (AMF), as root symbionts of many terrestrial plant species, provide several important roles in agricultural systems (Thirkell et al., 2017, van de Staaij et al., 2001), including for cereal crops such as wheat. These roles include improved nutrient acquisition and water uptake, better soil structure and mycorrhizal-induced resistance (MIR) to plant pathogens. While the host-fungal interaction is typically beneficial for the plant, it is a delicate balance given the AMF seek carbohydrates and lipids in return. Different plant species have variable dependencies on AMF symbiosis, and interactions also differ with AMF species (Sawers et al., 2008). In some cases, such as under conditions of nitrogen deficiency, the presence of AMF may decrease plant growth due to competition for nitrogen (Puschel et al., 2016).

AMF are broadly suggested to improve plant growth and productivity due to increased host nutrient uptake (Berruti et al., 2016). Dispersed hyphae of AMF allow more acquisition of mineral nutrition from bulk soil to feed resident plants (Hodge & Storer, 2015). For example, extended mycorrhizal hyphae have been shown to transfer phosphorus and zinc almost 15 centimetres away from the root area (Jansa et al., 2003), whereas root hairs are small and have limited ability to seek resources away from the root. Absorption of phosphorus by AMF hyphae is also much quicker than diffusion across soil (Smith & Read, 2008). For example, the mycorrhizal phosphorus uptake pathway can account for more than 80% of the total phosphorus foraging in wheat (Li et al., 2006). Abundant phosphorus has been found to accumulate in AMF mycelium, that indicated AMF ability to uptake phosphorus (Hijikata et al., 2010). These features of increased nutrient uptake area and uptake efficiency reflect the importance of AMF in complementing root function.

Presence of AMF can alter plant disease outcomes (Veresoglou & Rillig, 2012, Jacott et al., 2017) and have been reported for a range of different plant diseases. For example, mycorrhizal symbiosis significantly reduced disease in tomato caused by *Alternaria solani* (Fritz et al., 2006), in maize caused by *Fusarium verticillioides* (Olowe et al., 2018) and in apple caused by

the nematode *Pratylenchus penetrans* (Ceustermans et al., 2018). A range of underlying mechanisms for this AMF-mediated disease reduction are proposed, including improved plant nutrition (notably phosphorus), enhanced tolerance to pathogens, altering rhizosphere conditions to benefit antagonists, direct competition with pathogens, initiation of systemic resistance, and facilitating the recruitment of defence substances such as phytoalexins and hydrolases (Xavier, 2004).

High phosphorus availability in soil has been found to reduce the abundance and development of AMF (Olsson et al., 1997, Breuillin et al., 2010) and in this context AMF benefits might be reduced in terms of disease control and plant growth. Reduced wheat yield due to leaf rust tightly relates to decreased plant photosynthetic capacity (Bolton et al., 2008). Enhanced plant tolerance to pathogens by AMF does not reduce disease incidence, rather it compensates for yield loss by improving plant vigour and growth despite pathogen damage (Xavier, 2004). By the same token, AMF influence on plant disease reduction might not relate to improved host nutrition. AMF were associated with improved fungal disease resistance in cucumber (Hu et al., 2010) and common bean (Eke et al., 2016) caused by *Fusarium*, and apple by *Neonectria ditissima* (Berdeni et al., 2018), irrespective of AMF impact on plant growth. Competition with plant pathogens and alteration of the rhizosphere microbial community are particularly evident in arbuscular mycorrhizal plants diseased by soil-borne pathogens (Vierheilig et al., 2008). However, competition and rhizosphere community changes are unlikely to be the mechanisms for foliar pathogens reduction, as there is no direct interaction between AMF and pathogen.

Some studies claimed that AMF were actually able to trigger systemic resistance to pathogens at both the root system level (Zhu & Yao, 2004) and distal parts of host plant (Campos-Soriano et al., 2012). This induced systemic resistance is known as mycorrhiza-induced resistance (MIR). AMF *Rhizophagus irregularis* induced MIR in grapevine (Cameron et al., 2013) and *Medicago truncatula* (Liu et al., 2007) was associated with priming of jasmonic acid and salicylic acid inducible defence genes and enhanced expression of stress-related genes respectively. Moreover, MIR also coincides with cell wall defences as reported in tomato (Pozo et al., 2002) and common bean (Abdel-Fattah et al., 2011).

MIR has also been reported for powdery mildew control in wheat, where the disease was reduced by 78% and priming effects on papillae formation and hypersensitive reaction-like responses were detected in AMF plants (Mustafa et al., 2017). Another study also reported that mycorrhizal pea showed reduction of powdery mildew severity by up to 55% (Singh et al.,

2004). However, the positive role of AMF in plant disease control, particularly for powdery mildew, cannot be generalised as the magnitude of AMF-induced response is conflicting and inconsistent in some cases. For example, when standing milkvetch was inoculated with AMF, increased susceptibility to powdery mildew was recorded (Liu et al., 2018). Factors including host species and cultivars might account for such contrasting results as they have been suggested to result in varied arbuscular mycorrhizal response (Tawaraya, 2003). Additional phosphorus status and identity of arbuscular mycorrhizal fungi were also found to affect pathogen protection (Mustafa et al., 2016, Maherali, 2007).

Powdery mildew is a major disease problem in wheat production which is hard to control once established. Management strategies such as use of resistant varieties and foliar fungicides also face challenges due to breakdown of host resistance and fungicides. Based on Mustafa's findings (Mustafa et al., 2016, Mustafa et al., 2017), AMF appears promising as a sustainable tool of disease control in a general wheat-powdery mildew pathosystem. We need to learn more about any specificity of AMF species in control effectiveness and to what degree the outcome is affected by wheat genotype. Therefore, the aims of the current study were to investigate: (1) the impacts of phosphorus supply on AMF colonisation; (2) AMF impacts on wheat-powdery mildew disease severity and incidence; (3) the growth responses of different wheat cultivars to AMF.

4.2. Materials and methods

4.2.1. Plant material and soil

Grains of wheat were surface sterilised with 10% commercial bleach, then rinsed with tap water thoroughly. A pine bark/load-based potting mix was screened through 2mm sieves then autoclaved twice for one hour each time under high pressure at 120℃ before sowing to eliminate indigenous AMF propagules. Grains were germinated and grown in clean 2L pots filled with potting mix, later thinned to 5 seedlings per pot. Low phosphorus soluble fertiliser (total of 5% phosphorus, 25% nitrogen, 8.8% potassium, w/w) was supplied fortnightly with 1% concentration (1g of fertiliser into 1L of tap water) to all pots, at amount of 100ml solution per pot.

4.2.2. Preculture and application of arbuscular mycorrhizal fungi

A commercial inoculum of the AMF species *Rhizophagus irregularis* was extracted according to the protocol provided by the manufacturer (MycAgro Lab, France), as it was embedded in gel for long-distance transport. AMF spores were placed in the vicinity of roots of 3-week old spring onion seedlings planted in fumigated potting mix with low phosphorus fertiliser as described above. Two months after AMF inoculation, spring onion roots were harvested and checked for AMF colonisation under the microscope. Onion roots with 50% of the root length AMF colonized were used as inoculum for the wheat plants. Around 5g inoculum was mixed with autoclaved potting mix for each pot which received AMF treatment before sowing. Equivalent amounts of non-mycorrhizal spring onion roots were introduced to pots with no AMF treatment.

4.2.3. Treatment scenario and powdery mildew inoculation

The trial was carried out at Mt. Pleasant Laboratories, Launceston (41° S, 147° E), in a greenhouse conducive to powdery mildew, from November 2018 to January 2019. The experimental design was a three (wheat cultivars) by four (AMF and phosphorus treatments) factorial using a randomised block design with three replicates. Three pots were used per 'plot' giving a total of 108 pots. Wheat cultivars were chosen for contrasting powdery mildew resistance; SFR036 (resistant), Revenue (intermediate resistant) and Wedgetail (susceptible). AMF and phosphorus treatments were: (1) Control where seedlings did not receive AMF inoculation or extra phosphorus (A-P-), (2) Seedlings inoculated with AMF but grown with no extra phosphorus (A+P-), (3) Seedlings received both AMF and extra phosphorus (A+P+), (4) Seedlings received extra phosphorus but no AMF inoculation (A-P+). Extra phosphorus was given at a rate of 50µg phosphorus/g potting mix (dry weight) in the form of KH_2PO_4 solution. For the control, the same volume of tap water was added instead of the phosphorus solution. To ensure strong infection pressure inside the glasshouse, heavily powdery mildew infected wheat plants were manually shaken over 2-week old experimental seedlings.

4.2.4. Data collection

4.2.4.1. Colonisation rate of AMF

At harvest (10 weeks after sowing), roots of wheat seedlings were collected and washed thoroughly with tap water to remove potting mix, then stored in 50% ethanol at -20°C until

assessment. For staining, stored roots were drained, placed in Schott bottles covered with 5% KOH and heated until boiling for 2-3 minutes in a fume hood. After heating, roots were rinsed with tap water and then 3.5% HCI. Root samples were then placed back to clean Schott bottles and stained with solution made from 5% black ink (Schaeffer) in HCI, then boiled for another 3 minutes. Stained roots were rinsed with running water and de-stained with a few drops of 5% HCI in tap water.

To better handle samples under the microscope, processed wheat roots were cut into 1.5cm fine segments with a sterilised blade. Estimation of AMF colonisation on wheat roots followed the method as described by Abobaker et al. (2018), which was modified from McGonigle et al. (1990). Instead of following intersections between microscope eyepiece crosshair and roots, a gridded slide (two grids 20mm × 20mm each with 1 mm line spacing) was used in this study. AMF colonisation rate on each root segment was estimated by inspecting 10 intersections between root and grid lines. For each 'plot', 100 intersections were assessed to calculate percent colonisation as below:

$$\text{Colonisation rate} = \frac{X}{100} \times 100\%$$

Where X is the presence of AMF structure hypha (H), vesicle (V) or arbuscular (A).

4.2.4.2. Assessment of disease severity

Powdery mildew disease severity was evaluated on whole plants according to a modified rating scale of 0-5 (Sijaona et al., 2001): where 0 = no visible symptoms, 1 = <1% leaf area colonized, 2 = 1-10% leaf area colonized, 3 = 11-25% leaf area colonized, 4 = 26-50% leaf area colonized and 5 = >50% leaf area colonized. Disease was scored twice, at 7 days post pathogen inoculation (7 dpi) and harvest respectively (56 dpi).

4.2.4.3. Plant growth measurements

All plant growth parameters were measured at harvest, including number of dead and living leaves, tiller numbers, plant height (cm), total biomass (g) which were determined by weight after oven drying at 60℃ for 72 hours. Death rate was calculated by the ratio of number of dead leaves to total leaves.

4.2.4.4. Leaf chlorophyll content

Leaf chlorophyll content was quantified at harvest, as a SPAD index with a portable device SPAD-502 chlorophyll meter (Konica Minolta, Osaka, Japan), from the middle of the lamina of second uppermost wheat leaves.

4.2.4.5. Statistical analysis

For each parameter, measurements were conducted on three biological replicates, and averaged for statistical analysis. All data analysis and visualisation were performed with the R software (R Core Team, 2014). The significance of main effects (cultivar and AMF/phosphorus treatments) and interactions were tested by analysis of variance, and a post-hoc analysis by Tukey's test was also conducted to investigate differences between treatment means. A p-value below 5% level was used to indicate differences in the means.

4.3. Results

4.3.1. AMF colonisation of wheat roots

AMF colonisation of sample roots recorded the presence of colonisation structures which could be vesicles, arbuscules or hyphae. In non-AMF treatments, plants stayed mycorrhizal fungi free. We found AMF inoculation in all wheat cultivars with AMF application, with no significant difference in colonisation rate across cultivars (Table 4.1), which suggested successful establishment of *Rhizophagus irregularis* on wheat roots independent cultivar. Despite no significant difference, SFR036 tended to have greater AMF colonisation. As expected, AMF colonisation in wheat cultivars without additional phosphorus tended to be higher than plants with additional phosphorus supplied, though the difference between A+P- and A+P+ was not statistically significant (Table 4.1).

Table 4.1. Main effects of cultivar and treatment on arbuscular mycorrhizal fungi (AMF) colonisation rate, powdery mildew disease severity at 7 and 56 days post inoculation (dpi), dead leaf and green leaf number, death rate of leaves (as a proportion of dead leaves to total leaf number), tiller number, plant height, total dry matter per plant and leaf chlorophyll content (SPAD). Significance code *, ** and *** suggested significance at 0.05, 0.01 and 0.001 level respectively. A different letter next to the mean indicates the parameter differed significantly (P = 0.05) using Tukey's post hoc test.

Main effect	AMF colonisation rate	PM severity (7 dpi)	PM severity (56 dpi)	Dead leaves (number/plant)	Green leaves (number/plant)	Death rate (dead leaf number:total leaf number)	Tillers (number/plant)	Plant height (cm)	Total dry matter (g/plant)	Leaf chlorophyll content (SPAD)
Cultivar										
Revenue	22.3	1.8	3.4ab	17	25.2	40.4	7.6a	45.1b	9.9	32.9
SFR036	29.3	2.0	3.9a	16.2	22.9	40.7	5.2b	48.4a	9.4	32.6
Wedgetail	20.7	2.0	2.9b	14.3	21.8	39.7	6.8ab	48.0ab	8.5	32.6
p-value	0.244	0.839	0.007**	0.338	0.385	0.797	0.011*	0.041*	0.403	0.944
Treatment										
A-P-		1.8	3.4	15.3	23.8	39.6 b	7	46.6	9.4	33.1
A-P+		1.5	3.7	15.5	24.6	38.2 b	6.5	48.5	9.4	32.8
A+P-	27.1	2.0	3.2	14.3	24.1	37.3 b	6.6	46.3	8.8	33.0
A+P+	21.1	2.2	3.3	17.9	21.0	45.5 a	6.3	47.1	9.5	32.1
p-value	0.18	0.382	0.478	0.401	0.59	<0.001***	0.92	0.498	0.909	0.86

4.3.2. Powdery mildew infection

All wheat cultivars presented symptoms of powdery mildew infection one week after pathogen inoculation (Table 4.1), with average disease score between 1-2. Disease severity was not affected by wheat cultivar, treatment or interaction. Disease became more severe over the seven-week period between the first and harvest assessment, with average disease scores increasing to between 2-4 (Table 4.1). An increase in disease was detected regardless of AMF and phosphorus application. For each cultivar, there was no treatment effects. Wheat genotype affected disease severity (Table 4.1). The Wedgetail cultivar, typically classed as susceptible, exhibited moderate powdery mildew resistance having a mean score of 2 and 2.9 at 7dpi and 56dpi respectively (Table 4.1). In contrast, SFR036 which is rated as most resistant, was the most susceptible to powdery mildew at harvest.

4.3.3. Plant growth responses

Overall there were no significant interactions between cultivar and treatment, nor were there main effects of treatment or cultivar for most plant growth parameters tested (Table 4.1). Plant height and tiller number differed according to wheat cultivar (Table 4.1). Although no treatment effect was evident for both number of dead leaves and green leaves, there was a significant difference in death rate across four treatments (Table 4.1). Plants that received additional phosphorus and AMF had the highest death rate than other treatments (Table 4.1).

4.4. Discussion

4.4.1. AMF colonisation and wheat growth

Elevated phosphorus supply has previously shown adverse effects on AMF colonisation and AM benefits (Tavarini et al., 2018, Balzergue et al., 2013, Liu et al., 2016, Urcoviche et al., 2015). Our study presented a different outcome, in which phosphorus level did not influence either the extent of AMF colonisation or the impact of AMF on plant growth in glasshouse. Generally, AMF are still able to colonize plant roots in the presence of phosphorus application but might provide no additional benefit to host nutrition and growth. AMF are obligate biotrophs and utilise 4-20% of the host photosynthate, so their colonisation comes at a cost (Lerat et al., 2003), this results in a functional diversity of AMF effects on host plants from parasitic to mutualistic ranges. Reduced growth of AM wheat has been evident in several studies (Graham & Abbott, 2000, Li et al., 2006, Hetrick et al., 1993), thus it is plausible that

there could be no growth gain or even decreased host growth when host carbon allocation to AMF outweigh other benefits of AMF.

On the other hand, a number of studies have found that host genotypes and species showed different ability of colonisation by AMF (Berdeni et al., 2018, Parke & Kaeppler, 2000, Azcon, 1981). A foundational experiment investigating response of different wheat cultivars to inoculation with *Glomus mosseae* found different degrees of AMF colonisation and mycorrhizal dependency across wheat genotypes (Azcon, 1981). This sort of host genotype-dependency has also been observed in AMF-mediated growth including wheat (Hetrick et al., 1992, Hetrick et al., 1993), which suggests that AMF impacts on crop growth could be dependent on the plant genotype. Wheat cultivars used in our study did not affect colonisation by *Rhizophagus irregularis* as all cultivars showed similar root colonisation; low mycorrhizal dependency of these cultivars is presumably the primary reason that AMF presence did not improve growth. However, growing environment factor should not be ignored in affecting outcome of such host-pathogen-AMF interaction. A meta-analysis uncovered that total plant biomass from crop families Cucurbitaceae and Poaceae gave the strongest positive response to AMF inoculation (Van Geel et al., 2016), but successful AMF colonisation cannot guarantee beneficial effects on plant growth. In fact, AM wheat has been previously reported for growth depression in both field (Ryan et al., 2005) and glasshouse experiments (Graham & Abbott, 2000), it did not occur in our study though but does suggest that AMF presence is not always beneficial for plant growth.

Moreover, host root morphology and the AMF genera could modify AMF function. Newsham et al. (1995) proposed that plants with simple rooting systems could get more nutrient benefits from AM fungus than those with complex root architecture. Root architecture variation was identified among different wheat cultivars, cultivars with longer and finer roots, larger surface area, more tips and greater branching angle were deemed to improve efficiency of nutrient uptake (Chapagain et al., 2014). In this context, the AMF pathway of nutrient uptake may be less important in wheat cultivars with complex root system, although our current study did not look into root architecture components. In addition to host species, meta-analysis also implied effects of AMF genera on host growth response; AMF *Glomus* and *Funneliformis* species typically provide their host with more biomass returns than other AMF genus including *Rhizophagus* (Van Geel et al., 2016). The growth response of Poaceae to AMF from *Rhizophagus* was zero which indicated that *Rhizophagus* does not always benefit crops from

the family Poaceae. The optimal growth enhancement in a given crop species is suggested to be obtained from a specific arbuscular mycorrhizal taxa (Van Geel et al., 2016), and it follows that wheat growth may be more responsive to other AMF species than *Rhizophagus irregularis.* Impact of mycorrhizal inoculum type on wheat growth and architecture has been demonstrated before - results showed that *Rhizophagus* led to larger decrease in wheat plant height and less increase in root dry weight than *Funneliformis* inoculation, but *Rhizophagus* increased the most in shoot dry weight (Mustafa et al., 2016).

Taken together, potentials such as imbalance between photosynthate cost for AMF colonisation and nutrient gain, low mycorrhizal dependency of wheat cultivars, and AMF genera could affect AMF effects and account for non-response of wheat growth to AMF and AMF coupled with phosphorus supply in our glasshouse study.

4.4.2. AMF colonisation and powdery mildew disease

It has been proposed that the trend of AMF presence leading to reduced incidence and severity of disease caused by aerial pathogens depend upon the pathogen lifestyle type and pathosystem (Pozo & Azcon-Aguilar, 2007). Abundant evidence of enhanced host resistance towards necrotrophs and hemibiotrophs has been shown associated with *Rhizophagus irregularis* colonisation, for example tomato early blight caused by *Alternaria solani* (Fritz et al., 2006) and rice blast by *Magnaporthe oryzae* (Campos-Soriano et al., 2012). However, there is less evidence that AMF colonisation enhances resistance to biotrophic pathogens. It is not surprising that co-culturing with AMF did not give positive disease control in our case as responses of AM plants to foliar biotrophic diseases are really varied and contrasting, with both positive and negative feedbacks regarding powdery mildew disease (Liu et al., 2018, Singh et al., 2004, Chandanie et al., 2006, Mustafa et al., 2017, Lowe et al., 2012). Recently Comby et al. (2017) reviewed literature related to AMF effects on airborne phytopathogens and pests. Concerning powdery mildew control, application of *Rhizophagus irregularis* was found in part with contrasting outcomes from negative effect in flax (Dugassa et al., 1996), zero effect in cucumber (Larsen & Yohalem, 2004) to positive control in wheat, though effectiveness was less pronounced than other AMF identities (Mustafa et al., 2016). Amount of protection to wheat powdery mildew disease varied among tested AMF species, of which *Funneliformis mosseae* provided the best control (74%), twice that (34%) by *Rhizophagus irregularis* (Mustafa et al., 2016). This suggests the ability to enhance disease resistance differs among AMF species. In fact, AMF function does vary with identity, for example, species from the

family Glomeraceae are claimed to be good protectants against root pathogens while Gigasporaceae species are more efficient in phosphorus foraging (Maherali, 2007).

Effective AMF symbioses requires establishment on the host root, though high AMF colonisation may not result in increased resistance to pathogens. For example, Toth et al. (1990) found maize inbred lines that had greater susceptibility to a number of fungal pathogens also had higher AMF root colonisation. Also, an aboveground fungal pathogen infection has been demonstrated to induce resistance and hence inhibit root colonisation with AMF in common bean (Ballhorn et al., 2014). In contrast to both results, we neither observed impact of wheat genotype nor powdery mildew disease severity on AMF colonisation, though there was a significant impact of wheat genotype on disease severity. AMF colonisation does not necessarily result in less disease incidence, as more mildew fungus noticed in AMF standing milkvetch (Liu et al., 2018) and barley (Gernns et al., 2001) than control plants. Moreover, there is also no certainty that AMF colonisation rate associates with powdery mildew severity. At least, good AMF colonisation did not promise good disease protection in some reports. For example, higher AMF colonisation was found in AMF plants with more powdery mildew (Liu et al., 2018, Mustafa et al., 2016), though it could be owing to AMF coloniser identity rather than colonisation rate that predominately accounted for disease protection.

Improved plant tolerance to pathogens as a potential mechanism of AMF-mediated disease resistance has been suggested to relate to compensation for growth inhibition. AMF induced tolerance has not been found in this study to be the basis for reduced powdery mildew disease incidence, though AM wheat showed both biomass increase and powdery mildew decrease in work done by Mustafa et al. (Mustafa et al., 2016, Mustafa et al., 2017). More severe powdery mildew infection in AMF flax (Dugassa et al., 1996) and barley (Gernns et al., 2001) was associated with lower reduction of shoot fresh weight, assimilation of carbon dioxide, grain number, ear yield and thousand-grain weight than control plants. AMF induced tolerance in these experiments was attributed to maintained photosynthetic capacity and physiological activities. This compensation was also present in AMF standing milkvetch diseased by powdery mildew (Liu et al., 2018), where AMF mitigated yield loss by enhancing both foliage and root growth though disease susceptibility increased. Unlike these findings, no AMF effect on total biomass was found in the current study.

4.5. Conclusion

Our results suggested that the presence of AMF did not affect wheat powdery mildew disease, independent of phosphorus addition. Also, plant growth was unaffected among treatments that indicated the potential absence of an AMF induced powdery mildew tolerance, however more evidence is required to support the hypothesis that there was no AM compensation. It is possible that the outcome from interaction of powdery mildew-wheat-AMF is affected by wheat genotype and symbiont species which led to ineffectiveness of AMF in this case, contrasting with previous studies. These results suggest that for the AMF species *Rhizophagus irregularis*, wheat cultivars and environment used in this current experiment, there was no benefit to powdery mildew control.

Chapter 5: General conclusions and discussion

The purpose of current study was to explore two methods of wheat powdery mildew control, by detecting genetic resistance for breeding objectives and examining the application of AMF species *Rhizophagus irregularis* in disease control. This chapter includes a discussion of challenges of deploying QTL and APR as related to the publications in breeding program and what implications may be valuable for manipulating AMF for disease control.

5.1. Application of association mapping in breeding durable resistance

In this thesis, both R genes and APR were identified for resistance to powdery mildew in wheat. The relative priority of both types needs to be considered to inform how this information can be utilised in practical breeding efforts. Breeders tend to focus on a combination of APR genes to prolong resistance durability, rather than prioritising specific individual APR genes. However, the selection of APR can be influenced by the presence of resistance from R gene, so breeding for APR is more difficult than breeding exclusively for R genes (Ellis et al., 2014). The main way of identifying APR is by population-based QTL mapping, either linkage analysis using a population with known pedigree mostly biparental crosses, or association mapping based on linkage disequilibrium in a population derived from an unknown pedigree (Bartholome et al., 2016).

Through linkage mapping, more than 100 QTL for wheat powdery mildew resistance have been reported over recent decades (McIntosh et al., 2014, McIntosh et al., 2016, McIntosh et al., 2017). The fact is that locating APR on chromosome underlying these QTL is still challenging, as resolution of linkage mapping was quite low in most previous studies due to limited recombinants. Association mapping, in contrast, generally has higher resolution mapping with dense markers spanning the whole genome, so this approach is more likely to detect causal genetic variants instead of estimating a large confidence interval for QTL which could harbour abundant genes. Such an improvement of narrowing down candidate region was also implicated in the findings in this thesis, where some powdery mildew resistance associations detected by SNP markers were found to reside in previously identified QTL. However, most QTL defined in current association mapping overlapped with previous ones, four QTL on respective chromosome 3A, 3B, 6D and 7D were believed to be new QTL responsible for powdery mildew resistance in common bread wheat. This suggested the

potential of association mapping as a complementary tool to linkage mapping in detecting QTL.

Resistance mechanisms behind these QTL are not well elucidated as gene complexes, as candidate genes have neither been proposed nor validated in most previous linkage mappings. Although a couple of potential genes involved in powdery mildew defence were suggested in the studies in this thesis, it needs further experimental evidences to confirm their functions and effects. Unlike R genes which could be isolated from a resistant line and deployed in breeding schemes, identified QTL are difficult for breeding resistance. An important feature of QTL is that resistance effect is of a wide range and only APR genes that underly large-effect QTL are more valuable for sufficient resistance and currently possible for map-based cloning. In terms of "weak" QTL which are often seen in mapping studies, their application in breeding projects has revealed more technical and practical limitations due to polygenic control by multiple minor-effect genes (Ellis et al., 2014). Pyramiding has been an interesting strategy of enhancing resistance of QTL, and in theory combining QTL with minor to moderate phenotypic effect could increase resistance sufficiently. Even so, concerns like unfavourable linkages which could result in physiological costs for the plant slow down breeding progress (Brown & Rant, 2013). For these reasons, successful deployment of powdery mildew resistance QTL in wheat is reported except for a few APR genes (Zhang et al., 2018, Lillemo et al., 2008, Jia et al., 2018).

To target QTL for durable resistance breeding, additional factors need to be considered. A challenge of QTL deployment is environment-sensitive expression, even when using the same populations. This has also been demonstrated with the data presented in this thesis from the consecutive three-year experiment, where consistency of identified QTL across environments was really poor. Such a variability of resistance performance reduces the value of QTL in breeding programs and highlights a need for evaluation over time, particularly under field conditions. Moreover, identification of QTL by either linkage mapping or association mapping is assisted by genetic markers, so finding out reliable markers linked to resistance loci is important for breeding with assistance of MAS.

5.2. Arbuscular mycorrhizal fungi for management of wheat powdery mildew

The role of arbuscular mycorrhizal fungi (AMF) in reducing air-borne plant pathogens has been shown to vary with pathogen lifestyle and the pathogen-host combination (Pozo & Azcon-Aguilar, 2007). Specifically, more positive effects have typically been found against necrotrophs and hemibiotrophs, with more variable outcomes for biotrophic pathogens. Investigations of AMF effects on plant powdery mildew have been done in pea (Singh et al., 2004), standing milkvetch (Liu et al., 2018), cucumber (Larsen & Yohalem, 2004), flax (Dugassa et al., 1996), barley (Gernns et al., 2001) and wheat (Mustafa et al., 2016, Mustafa et al., 2017) and results varied from disease enhancement through to reducing disease or no effect. These contrasting results suggested that the host-powdery mildew-AMF interaction is complex and may be influenced by other factors.

AMF colonization is already known to vary widely between host species and among genotypes (Tawaraya, 2003). Although no direct correlation between AMF colonization rate and powdery mildew incidence was previously indicated, establishment of AMF is a prerequisite for host to benefit from symbiosis with AMF. AMF colonization comes with costs as biotrophic AMF compete for carbon compounds with their host which could take up to 20% of a host's total carbon budget (Lerat et al., 2003). It is more likely the balance of resource exchange between two partners rather than net AMF colonization rate that determines host performance. A curvilinear relationship between AMF colonization and host plant benefits was predicted by a previous model (Gange & Ayres, 1999) based on empirical data which was later also found in the annual plant *Datura stramonium* (Garrido et al., 2010). In both cases, plant fitness was maximized at a certain level of AMF colonization, where colonization densities lower and higher than optimum level were parasitic to the host plant. Such curvilinear relationship might also be implied in AMF colonization and disease incidence, to help explain why more powdery mildew infections occur in plants with higher AMF colonization in some occasions. It will be interesting to explore relationship between AMF colonization rate and wheat powdery mildew severity before conclude the value of AMF in wheat powdery mildew management. Moreover, genotype-dependent plant defences have been shown in mycorrhizal responsiveness in strawberry (Mark & Cassells, 1996) and tomato (Steinkellner et al., 2012). The genetic basis in wheat AM colonization rate has been studied earlier (Lehnert et al., 2017), but little is known about wheat genotypic variation in AM-mediated disease response. However, the different

wheat cultivars used, compared to those by Mustafa et al. (2017), could be a potential reason for insignificant AMF effects on powdery mildew disease obtained from current study.

In addition to host species and cultivar, AMF identity has also been found to affect mycorrhizal roles thus should be factored into variable AMF effects on powdery mildew control (Maherali, 2007). Supported by Mustafa et al. (2016), control effectiveness of wheat powdery mildew was different among three applied AMF species, where *Funneliformis mosseae* outperformed *Rhizophagus irregularis*. As a result, the role of AMF in plant disease response is unclear as different host, AMF species and pathogens have been tested in individual studies. So even for a general wheat-powdery mildew pathosystem, evaluations of different AMF species in a cultivar of interest will be required for optimizing management under certain condition.

Regardless of the direct impact of AMF on disease incidence, their role in promoting host growth may compensate for fungal damage and therefore enhance plant disease tolerance. Tolerance is an important plant defence strategies against pathogens, in addition to resistance (Pagan & Garcia-Arenal, 2018). On the contrary, vigorous plant growth might also favour fungal development as new leaves can be more susceptible to infection. In this sense, AMF-induced disease tolerance seems to be absent in our study as mycorrhizal wheat showed no growth increase compared with that of no AMF inoculation under powdery mildew infection. However, we did not test how much powdery mildew infection supressed growth of AM wheat in our experiment as all experimental plants suffered pathogen pressure. It is possible that AMF did enhance disease tolerance, but such compensation was overruled by the impact of a large pathogen load on plant growth.

5.3. Effects of genotype by AMF interaction on disease response

The ‘disease triangle’ concept in plant pathology demonstrates that a specific pathogen-host plant interaction is affected by given environment. Resistance variations due to environmental differences were detected in our association study, where not only the resistance distribution of wheat population has a clear shift towards a more susceptible population but also resistance performance of individual varieties varied across years and growing environments. Such evidence, taken together, implies that while resistant crop genotype can be effective in a certain environment, resistance expression can be quite different in another environment. A conducive environment for disease potentially could lead to a less effective defensive response or even disease susceptibility in resistant host genotype. Velasquez et al. (2018) critically reviewed the

influence of a few important environmental factors relating to climate change in plant-pathogen interactions and interpreted insightful molecular mechanisms underlying these environmental impacts on plant immunity, which highlight the challenge of disease prediction.

Regarding plant growing environment, artificial systems such as an enclosed glasshouse does not mirror field conditions. Most of the research on AMF application (as for that in this thesis) has been carried out inside a glasshouse where overall environment was not as dynamic as that actually in nature, so conclusions from glasshouse experiments are not directly applicable to field outcomes. For interaction between pathogen and environment, powdery mildew development in open field is not only affected by climate and soil conditions, but also other diseases present that cause multi-diseased wheat plants. Competition with other pathogenic organisms for host availability may reduce damage caused solely by powdery mildew. Evidently, control of multiple crop diseases instead of exclusively on powdery mildew through AMF would be a more practical concern. However, this requires thorough understanding of the interplay between AMF as an environmental component and aboveground pathogens before understanding AMF for disease control in cropping system. In addition, soil rhizosphere in the field is more complex than a glasshouse experiment, such as used in this thesis, in which wheat was grown in sterilized potting mix with one species of AMF inoculant. In a field soil, communications between artificially supplied AMF with existing indigenous AMF species, other soil microbial flora as well as foliar pathogens add to difficulty of demonstrating artificial AMF influence on crop disease control in field.

On the other hand, the ‘disease triangle’ does include the concept that the plant also interacts with environment. To some extent plant genotype is determinant for resistance, however, environment also influences the expression of plant resistance. For example, wheat APR gene Yr36 confers non-specific resistance to stripe rust at temperatures up to 35°C but not below 20°C (Fu et al., 2009). Studies in such plant genotype by environment interaction in disease resistance expression have been conducted in different crop-pathogen systems. Greater impact of environment than plant genotype on resistance variation was found in rice (Zeng et al., 2017) and pea (Das et al., 2019) responses to sheath blight and rust respectively under field condition. This increasing evidence suggested that plant resistance expression could be very sensitive to environmental fluctuations, therefore genotype is less reliable for resistance selection. In this context, effects of wheat genotype, AMF and their interactions on powdery mildew control

identified in an individual study are dependent on experimental conditions which cannot be fully translated to different agricultural conditions.

5.4. Future research directions

The focus of an association study is molecular marker-trait association based on linkage disequilibrium between a marker and a candidate gene, where the most associated marker is likely to be in candidate gene region. However, in association study, strictly speaking there is not a confidence interval of QTL, rather, basically a statistically significant marker with known sequence. Moreover, potential candidate genes in association mapping in this thesis were given by their coding regions overlapping with associated markers, which have not been confirmed yet and could be nothing to do with true candidate genes.

A quantitative polymerase chain reaction-based protocol to quantify expression of these potential candidate gene would help to work out if they contribute to resistance. Since association mapping cannot determine the distance between candidate gene and associated marker, a follow-up linkage mapping study in segregation population could be an option to locate candidate gene region. If there is more than one candidate gene, a quantitative cell-based RNA interference-based functional characterisation on systematic scale might be worth trying to reveal functions of interested genes within QTL as well as the key biological pathways underlying disease resistance (Blattmann et al., 2013).

AMF *Rhizophagus irregularis* did not demonstrate any benefit in either growth or powdery mildew control in wheat cultivars used in current experiment. As suggested, mycorrhizal effect is not dependent on a single factor rather is affected by a complex of components involved in interaction of powdery mildew-wheat-environment. In this sense, the role of AMF in wheat powdery mildew control could be inconsistent across individual studies, and successful application might not be generalised in all cases. Evaluation of AMF in terms of disease control effectiveness, control stability over years and environments will be required to find out the best combination of wheat cultivar and AMF species for local practice.

Last but also importantly, the application of AMF in cropping systems is not an easy task, as positive results obtained from a controlled environment such as lab-bench and glasshouse experiments might not be copied under natural environments. Wheat fields are much more complicated agroecosystem where multiple natural and artificial factors can influence the

abundance and activities of AMF inoculation, which should be taken into consideration to evaluate the potential of AMF field application.

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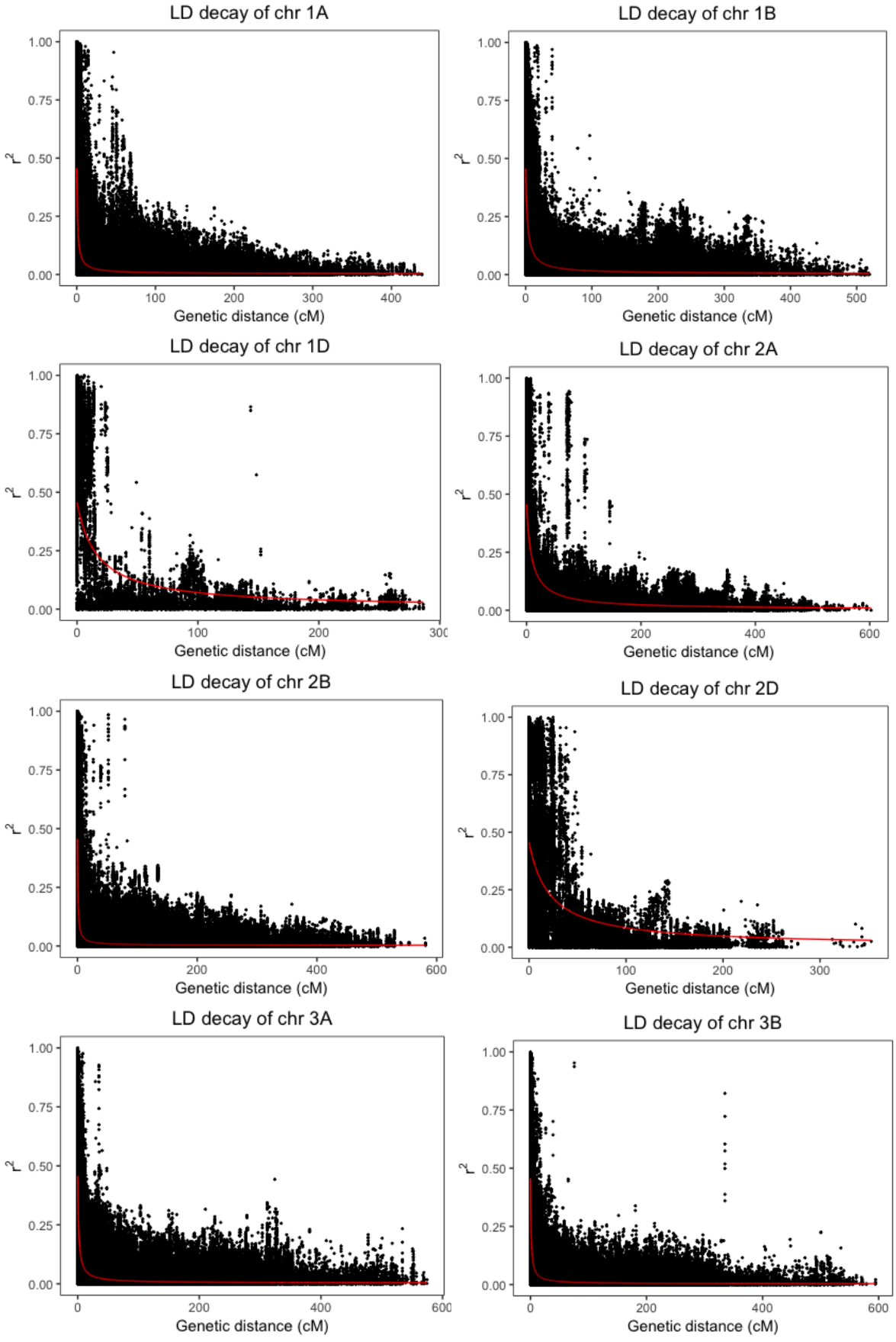
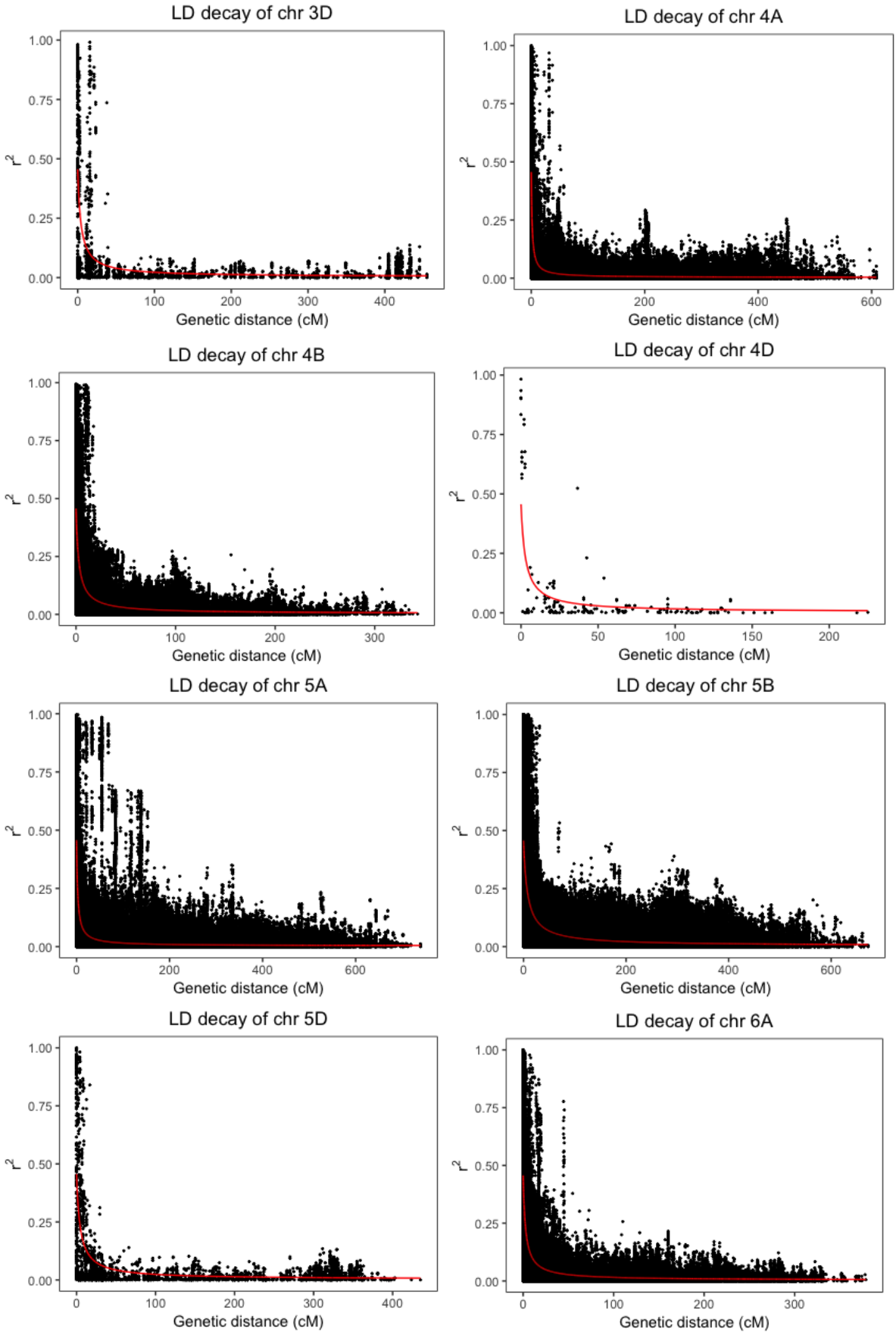

LD decay of chr 1A
LD decay of chr 1B
LD decay of chr 1D
LD decay of chr 2A
LD decay of chr 2B
LD decay of chr 2D
LD decay of chr 3A
LD decay of chr 3B
r2
Genetic distance (cM)


LD decay of chr 3D
LD decay of chr 4A
LD decay of chr 4B
LD decay of chr 4D
LD decay of chr 5A
LD decay of chr 5B
LD decay of chr 5D
LD decay of chr 6A
r^2
Genetic distance (cM)

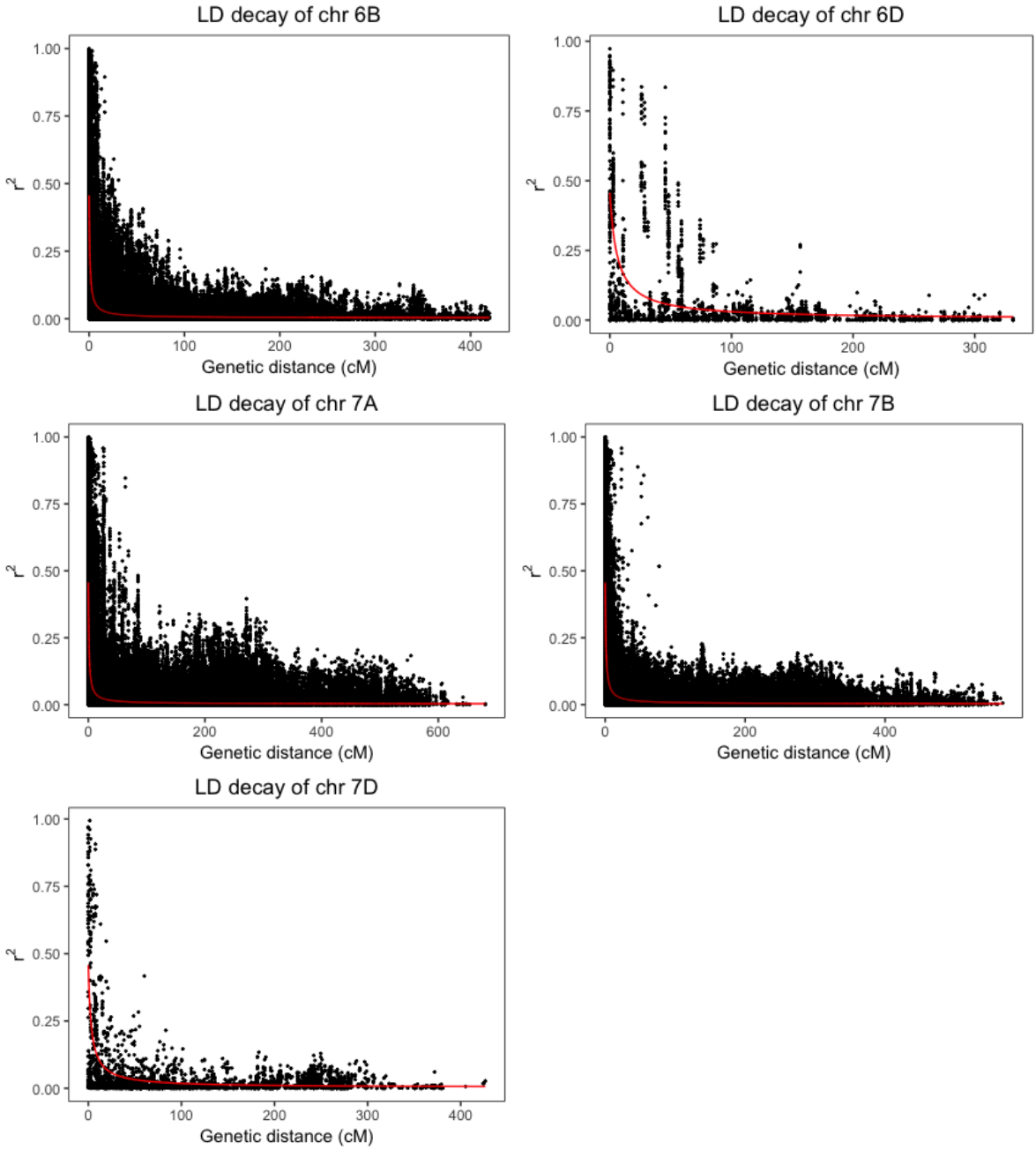


Figure S3.1: Scatter plots of pair-wise SNP r^2 of the linkage disequilibrium (LD) against genetic distance (cM) for all chromosomes in wheat. The trend line (in red) illustrates the LD decay threshold ($r^2 = 0.2$).

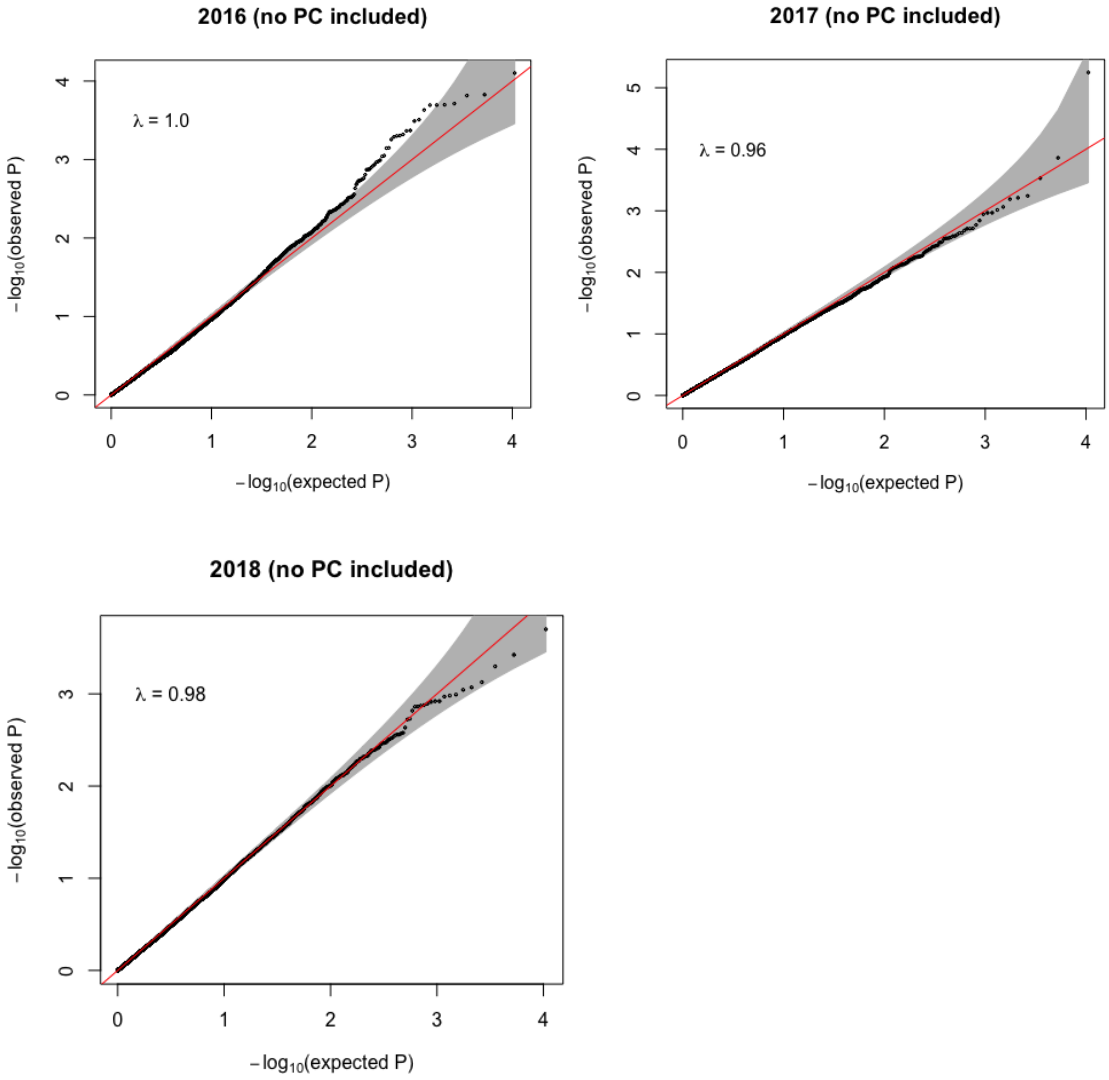


Figure S3.2: Q-Q plots of enriched compressed mixed linear model (ECMLM) without covariate of principal component (Fraaije et al.) for all test datasets. Red line indicates the expected normal distribution of *p*-values. Grey band suggests a 95% confidence interval. λ indicates genomic control factor.

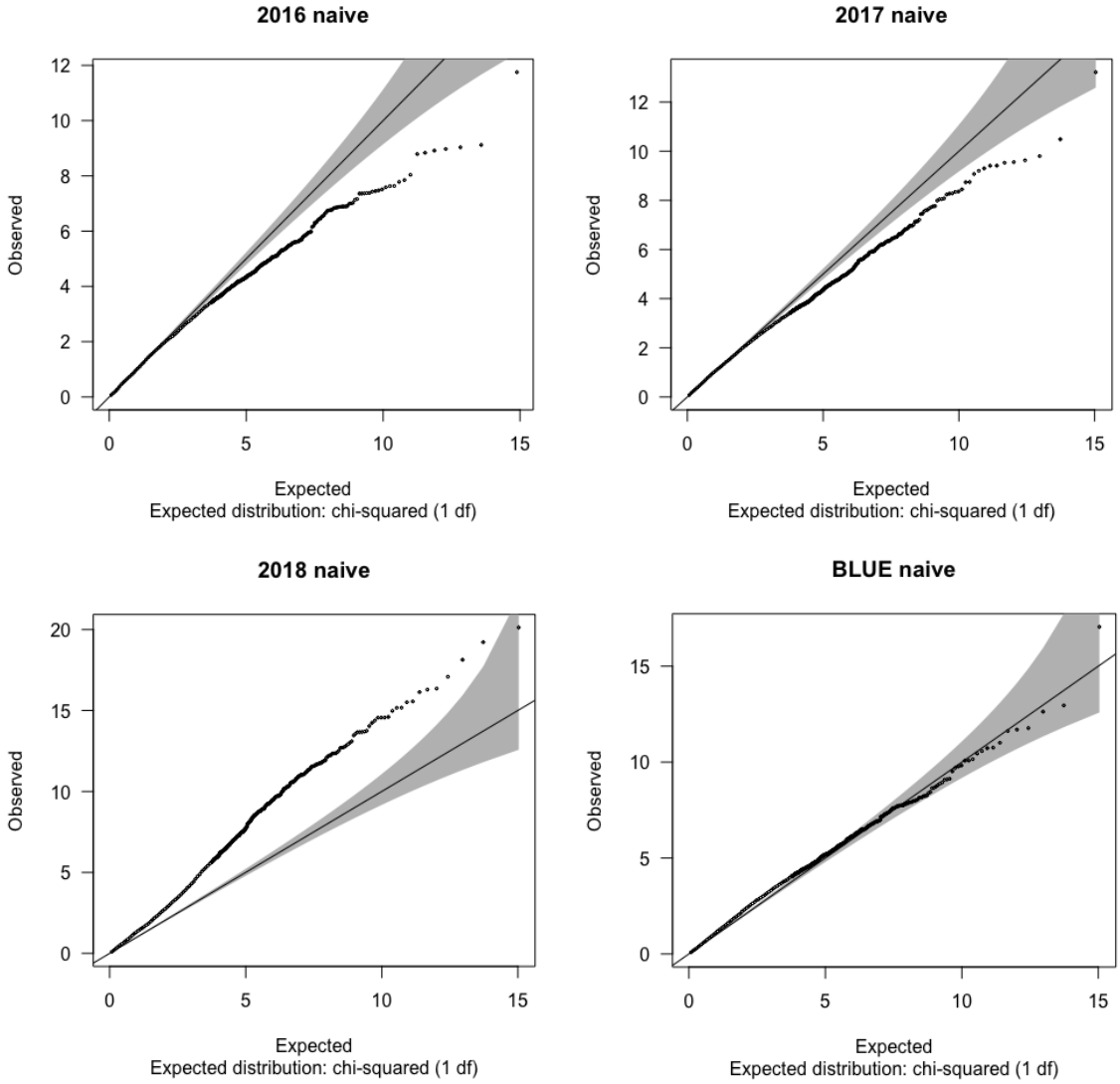


Figure S3.3: Q-Q plots of a chi-squared test based naïve model for all test datasets. Black line indicates the *p*-values under null-hypothesis. Grey band indicates a 95% confidence interval.

Table S2.1: Summary of reported wheat powdery mildew gene and QTL

Chromosome	Gene/QTL	Donor	Flanking markers or marker interval	References for flanking markers	R^2 *
1A	Pm25	NC96BGTA5	OPA04950	(Shi et al., 1998)	
1RS.1AL	Pm17	Amigo (Heun et al., 1990)	Xmwg68-1R, Sec-1 in 1RS, Lr26	(Mater et al., 2004, Hsam et al., 2000)	
1AL	QPm.sfr-1A	Oberkulmer	Xpsr1201b-Xpsr941	(Keller et al., 1999)	7.7%
1AL	QPm.crag-1A	RE714	cdo572b-bcd442	(Mingeot et al., 2002)	39.3–43.0%
1AL	QPm.caas-1AL	Bainong 64	Xbarc148-Xwmc550	(Lan et al., 2009)	7.4–9.9%
1AS	Pm3a	Asosan (Briggle & Sears, 1966)	Xwhs179	(Hartl et al., 1993)	
	Pm3b(Pm3j)	Chul (Briggle, 1966)	BCD1434	(Ma et al., 1994)	
	Pm3d	Kolibri		(Zeller et al., 1993)	
	Pm3e	W150	Xwmc818	(Zeller et al., 1993)	
	Pm3f	Michigan Amber		(Zeller et al., 1993)	
	Pm3g	Aristide (Zeller, 1998)	Gli-A5	(Sourdille et al., 1999)	
	Pm3h(Pm3)	Abessi (Zeller, 1998)	Xgwm905	(Huang et al., 2004)	
	Pm3i(Pm3c)	N324 (Zeller, 1998)	Xgwm905	(Huang et al., 2004)	
	Pm3j	GUS 122 (Zeller, 1998)	Xgwm905	(Huang et al., 2004)	
1AS	QPm.osu-1A	2174	Pm3a	(Chen et al., 2009)	61%
1AS	QPm.caas-1AS	Fukuho-komugi	Xgdm33-Xpsp2999	(Liang et al., 2006)	19.9–26.6%
1B	Pm28	Meri		(Peusha et al., 2000)	
1B	Qaprpm.cgb-1B	Hanxuan 10	WMC269.2-CWM90	(Huang et al., 2008)	20.3%
1B	Qaprpm.cgb-1B	Hanxuan 10	P4133-170-Xgwm582	(Huang et al., 2008)	4.8%
1BL	Pm39(APR)	Saar	Xwmc719-Xhbe248	(Lillemo et al., 2008)	
1RS·1BL	Pm8	Disponent (Hsam & Zeller, 1997)	IAG95	(Wricke et al., 1996)	
			OPJ07-1200, OPR19-1350	(Iqbal & Rayburn, 1995)	
			SEC-1b-412bp	(de Froidmont, 1998)	
			STSiag95-1050	(Mohler et al., 2001)	
1BL.1SS	Pm32	L501		(Hsam et al., 2003)	
1BL	QPm.osu-1B	2174	WMC134	(Chen et al., 2009)	5–14%
1BL	QPm.caas-1BL.1	Zhou8425B	IWB72835–IWB18787	(Jia et al., 2018)	7.2%

1BL	QPm.vt-1B	USG3209	WG241	(Liu et al., 2001)	17%
1BL	QPm.vt-1BL	Massey	Xgwm259-Xbarc80	(Tucker et al., 2007)	15%–17%
1BL	Yr29/Lr46 /Pm39	Saar	Xwmc719-Xhbe248	(Lillemo et al., 2008)	7.3–35.9%
1BS	QPm.sfr-1B	Forno	CD9b-Xpsr593a	(Keller et al., 1999)	11.6%
1BS	QPm.ttu-1B	*Triticum militinae*	Xgwm3000	(Jakobson et al., 2006)	4.0–5.0%
1D	Pm10	Norin 26		(Tosa et al., 1987)	
1DL	QPm.sfr-1D	Forno	Xpsr168-Xglk558b	(Keller et al., 1999)	9.5%
1DL	QPm.heau-1DL	Francolin	wPt-5721-wPt-1865	(Ren et al., 2017b)	6.1–8.5%
1DS	Pm24a	Chiyacao (Huang et al., 2000b)	Xgwm789,Xgwm603 Xgwm1291, Xbarc229	(Huang & Roder, 2011)	
	Pm24b	Baihulu	Xgwm789, Xgwm603, Xbarc229	(Xue et al., 2012b)	
1DS	QPm.inra-1D.1	RE9001	Xgwm106	(Bougot et al., 2006)	12.6%
2A	Mltd1055	TA1055 (wild emmer *T. dicoccoides* accessions)		(Ahmadi & Moore, 2007)	
2A	Qpm.sdau-2A	LM21	cfa2263—D-1395795	(Qu et al., 2018)	8.6–9%
2AL	Pm4a	Khapli (McIntosh & Bennett, 1979)	Xgwm356	(Ma et al., 2004)	
			BCD1231, CDO678	(Ma et al., 1994)	
			4aM1	(Hartl et al., 1999)	
			STSbcd1231-1.7kb	(Liu et al., 1998)	
	Pm4b	Armada (McIntosh & Bennett, 1979)	STS_241, Me8/Em7_220, Xgwm382	(Yi et al., 2008)	
	Pm4d	Tm27d2	Xgwm526-Xbarc122	(Schmolke et al., 2012)	
2AL	Pm23(Pm4c)	82-7241 (McIntosh et al., 1998)	Xbarc122- Xgwm356	(Hao et al., 2008)	
2AL	Pm50	K2	Xgwm294	(Mohler et al., 2013)	
2AL	PmDR147	DR147	Xgwm311, Xgwm382	(Zhu et al., 2004)	
2AL	PmPS5A	Am4	Xgwm356	(Zhou et al., 2005)	
2AL	PmE	Xiaohan	Xgwm265, Xgwm311, Xgwm382	(Zhou et al., 2005)	
2AL	PmLK906	Lankao 90 (6)	Xgwm265, Xgdm93	(Niu et al., 2008)	
2AL	PmYm66	Yumai 66	XKsum193	(Tiezhu et al., 2008)	
2AL	TaAetPR5	EU082094	p9-7p1, p9-7p2	(Niu et al., 2010)	
2AL	PmZB90	ZB90		(Yi et al., 2013)	
2AL	PmHo	Mv Hombár	XwPt-3114 –	(Komaromi et al.,	

			XwPt-665330	2016)	
2AL	Ml92145E8-9	92145E8-9	Xsdauk13 - Xsdauk682	(Yu et al., 2018)	
2AL	PmHNK54	Zheng 9754	Xbarc5-Xgwm312	(Xu et al., 2011)	
2AL	PmX	Xiaohongpi	Xhbg327-Xsts-bcd1231 – XresPm4/Xgpw4456	(Fu et al., 2013)	
2AL	QPm.crag-2A	RE714	Pm4b-gbxG303	(Mingeot et al., 2002)	22.7–39.2%
2AL	QPm.ttu-2A	*Triticum militinae*	Xgwm311-Xgwm382	(Jakobson et al., 2006)	5%
2AL	QPm.vt-2A	USG3209	Xgwm304-Xgwm294	(Tucker et al., 2007)	26–29%
2AL	QPm.vt-2AL	Massey	Xgwm304a-Xgwm312	(Liu et al., 2001)	29%
2AS	QPm.sfr-2A	Oberkulmer	Xpsr380-Xglk293b	(Keller et al., 1999)	7.7%
2AS	QPm.inra-2A	Courtot	Xgwm275	(Bougot et al., 2006)	7.4%
2B	QPm.sdau-2B	SN0431	D-1054006—D-1114401	(Qu et al., 2018)	12.6–13.4%
2B	Qaprpm.cgb-2B	Hanxuan 10	Xwmc477-Xwmc272	(Huang et al., 2008)	5.4%
2BL	Pm6	TP 114 (Jorgense.Jh & Jensen, 1973)	BCD135-9 kb-EcoRV	(Tao et al., 2000)	
			CINAU123-CINAU127	(Qin et al., 2011)	
			NAU/$STS_{BCD135-1}$, NAU/ $STS_{BCD135-2}$	(Ji et al., 2008a)	
2BL	Pm33	PS5		(Zhu et al., 2005)	
2BL	Pm51	CH7086	Xwmc332 – Xwmc317	(Zhan et al., 2014)	
2BL	Pm52 (MlLX99)	Liangxing 99	Xcfd73, Xwmc441, XBE604758, Xgwm120	(Zhao et al., 2013)	
2BL	Pm57	Line 89-346(TA5108)/ Line 89(5)69 (TA5109)	X2L4g9p4/HaeIII	(Liu et al., 2017c)	
2BL	MlAB10	NC97BGT AB10	Xwmc445, Xwmc317, Xwmc361, Xwmc149	(Maxwell et al., 2010)	
2BL	MlLX99	Liangxing 99	Xgwm120, BE604758	(Zhao et al., 2013)	
2BL	MlZec1	Zecoi 1	Xwmc356	(Mohler et al., 2005)	
2BL	PmPS5B	Am9	Xgwm111, Xgwm382, Xwmc317	(Zhou et al., 2005)	
2BL	QPm.caas-2B	Fukuho-komugi	Xgwm877.1-Xgwm435.1	(Liang et al., 2006)	5.7–8%
2BL	QPm.vt-2B	Massey	WG338-Xgwm526a	(Liu et al., 2001)	11%
2BL	QPm.inra-2B	RE9001	Xrtp114R-Xcdf267b	(Bougot et al., 2006)	10.3–36.3%
2BL	QPm.vt-2BL	USG3209	Xgwm501-Xgwm191	(Tucker et al., 2007)	11–15%
2BL	QPm.caas-2BL	Lumai 21	Xbarc1139-Xgwm47	(Lan et al., 2010)	5.2–10.1%
2BS	Pm26	TTD140	Xwg516	(Rong et al.,	

				2000)	
2BS	Pm42	G-303-1M	BF146221 – Xgwm148	(Hua et al., 2009)	
2BS	Pm49 (Ml5323)	MG5323	Xcau516-XCA695634	(Piarulli et al., 2012)	
2BS	MlIW170 (located in the same region as Pm26)	IW170	Xcfd238, Xwmc243	(Liu et al., 2012)	
2BS	PmWE99	WE99	Xgwm148 – Xbarc55	(Ma et al., 2016a)	
2BS	PmL962 (transferred from *Thinopyrum Intermedium*)	Line L962	Xwmc314 - BE443737	(Shen et al., 2015)	
T2BS.2VL#5	Pm62(APR)	NAU1823		(Zhang et al., 2018)	
2U(2B)	PmY39	Am9	Xgwm257, Xgwm296, Xgwm319	(Zhou et al., 2005)	
2BS	QPm.crag-2B	Festin	Xgwm148-gbxG553	(Mingeot et al., 2002)	23.6–71.5%
2BS	QPm.caas-2BS	Lumai 21	Xbarc98-Xbarc1147	(Lan et al., 2010)	10.6–20.6%
2BS	QPm.umb-2BS	Folke	wPt-9402	(Lillemo et al., 2012)	3.9–13%
2BS	QPm.umb-2BS	Folke	Xgwm410b–Xgwm148	(Lillemo et al., 2012)	8–10.2%
2BS	CP5	Pedroso	wPt-5513	(Marone et al., 2013)	12.3%
2D	Pm43	CH5025	Xwmc41-Xbarc11	(He et al., 2009)	
2DL	PmYU25	TAI7047	Xgwm210 (Ma et al., 2007)	(Ma et al., 2007)	
2DL	PmSE5785	SE5785	Xbarc59 – Xwmc817	(Wang et al., 2016b)	
2DL	QPm.sfr-2D	Oberkulmer	Xpsr932-Xpsr331a	(Keller et al., 1999)	10%
2DL	QPm.ipk-2D	W7984	Xglk558-XksuD23	(Borner et al., 2002)	
2DL	QPm.caas-2DL	Lumai 21	Xwmc18-Xcfd233	(Lan et al., 2010)	5.7–11.6%
2DL	QPm.umb-2DL	Folke	Xwmc167–Xgwm301	(Lillemo et al., 2012)	4.3–9.5%
2DS	Pm58	*Ae. tauschii* TA1662	K-TP338253-K-TP159900	(Wiersma et al., 2017)	
2DS	QPm.caas-2DS	Libellula	Xcfd51-Xcfd56	(Asad et al., 2012)	2.3–3.4%
2DS	QPm.inra-2D-a	RE9001	Xgwm102	(Bougot et al., 2006)	19%
2DS	QPm.inra-2D-b	RE9001	Xcfd2e	(Bougot et al., 2006)	16.5%
3A	Qaprpm.cgb-3A	Hanxuan 10	Xwmc21-Xwmc505.2	(Huang et al., 2008)	9.8%
3AS	Pm44	Hombar		(Chen et al., 2011)	
3AS	QPm.sfr-3A	Forno	Xpsr598-Xpsr570	(Keller et al., 1999)	10.4%

3AS	QPm.crag-3A	Festin	Xpsr598-Xgwm5	(Mingeot et al., 2002)	21.4–25.9%
3AS	QPm.nuls-3AS	Saar	Xstm844tcac-Xbarc310	(Lillemo et al., 2008)	8.1–20.7%
3BL·3SS-3S 3DL·3SS-3S	Pm13	C strans.Line (Ceoloni et al., 1992)	Xpsr305, Xpsr1196	(Donini et al., 1995)	
			cdo460, utv135, OPV13800, UTV13, OPX12570, UTV14	(Cenci et al., 1999)	
3B	Qaprpm.cgb-3BL	Hanxuan 10	Xgwm181-Xgwm340	(Huang et al., 2008)	13.3%
3BL	Pm41	IW2	BE489472 – Xwmc687	(Li et al., 2009)	
3BL	PmHNK	Zhoumai 22	Xgwm108-Xwmc291	(Xu et al., 2010)	
3BS	QPm.inra-3B	Courtot	Xgwm389	(Bougot et al., 2006)	22.7%
3BS	QPm.osu-3B	2174	WMS553	(Chen et al., 2009)	10%
3BS	QPm.caas-3B	Opata 85	XksuG53-Xfba190	(Huo et al., 2005)	7.3%
3BS	QPm.caas-3BS	Zhou8425B	IWB21064–IWB64002	(Jia et al., 2018)	7.1%
3BS	CP2	Creso	F103	(Marone et al., 2013)	10.6%
3DS	QPm.inra-3D	RE9001	Xcfd152, Xgwm707	(Bougot et al., 2006)	9.3–15.2%
3DS	QPm.sfr-3D	Oberkulmer	Xpsr1196a-Lrk10_6	(Keller et al., 1999)	15.7%
4A	QPm.tut-4A	8.1	Xwmc232–Xrga3.1	(Jakobson et al., 2012)	24–46%
4AL	MLIW30	2L6 (derived from wild emmer wheat accession IW30)	XB1g2000.2, XB1g2020.2	(Geng et al., 2016)	
4AL	Pm61	Xuxusanyuehuang	Xgwm160-Xicsx79	(Sun et al., 2018)	
4AL	QPm.sfr-4A.1	Forno	Xgwm111c-Xpsr934a	(Keller et al., 1999)	14.7%
4AL	QPm.sfr-4A.2	Forno	Xmwg710b-Xglk128	(Keller et al., 1999)	14.3%
4AL	QPm.ttu-4A	*Triticum militinae*	Xwmc232–Xgwm160	(Jakobson et al., 2006)	35–54%
4AL	QPm.inra-4A	RE714	XgbxG036	(Chantret et al., 2001)	4.9–6.9%
4AL	QPm.crag-4A QPm.	RE714	XgbxG036-XgbxG542	(Mingeot et al., 2002)	22.3%
4AL	inra-4A	Courtot	Xcfd71b	(Bougot et al., 2006)	8.9%
4AL	QPm.osu-4A	2174	WMS160	(Chen et al., 2009)	12%
4B	Mld	Maris Dove		(Bennett, 1984)	
4BS.4BL-2RL	Pm7	Transec		(Friebe et al., 1994)	
4BL	QPm.sfr-4B	Forno	Xpsr593b-Xpsr1112	(Keller et al., 1999)	7.5%
4BL	QPm.ipk-4B	W7984	Xcdo795-Xbcd1262	(Borner et al., 2002)	
4BL	QPm.caas-4BL	Oligoculm	Xgwm375-Xgwm251	(Liang et al., 2006)	5.9%

4BL	QPm.nuls-4BL	Avocet	XwPt1505-Xgwm149	(Lillemo et al., 2008)	21–40.2%
4BL	QPm.Caas-4BL.1	Libellula	Xgwm149-Xgwm495	(Asad et al., 2012)	9.1–14.7%
4BL	QPm.caas-4BL.2	Zhou8425B	IWB35851–IWB60096	(Jia et al., 2018)	8.77%
4D	qApr4D	Yumai 57	Xgwm194-Xcfa2173	(Zhang et al., 2008)	20%
4DL	QPm.sfr-4D	Forno	Xglk302b-Xpsr1101a	(Keller et al., 1999)	14.4%
4DL	QPm.caas-4DL	Bainong 64	Xbarc200-Xwmc331	(Lan et al., 2009)	15.2–22.7%
5A	QPm.tut-5A	8.1	Xgwm666–Xcfd30-Xbarc319	(Jakobson et al., 2012)	14–22%
5A	Qaprpm.cgb-5A	Hanxuan 10	P3616-185-P3616-195	(Huang et al., 2008)	13.2%
5AL/5DL	Pm55	NAU421	5EST-237	(Zhang et al., 2016)	
5AL	Pm2026	TA2026	Xcfd39, Xcfd1493, Xmg2170, Xgwm126	(Xu et al., 2008)	
5AL	QPm.sfr-5A.2	Oberkulmer	Xpsr1194-Xpsr918b	(Keller et al., 1999)	16.6%
5AL	QPm.sfr-5A.3	Oberkulmer	Xpsr911-Xpsr120a	(Keller et al., 1999)	10.5%
5AL	QPm.nuls-5A	Saar	Xgwm617b-Xwmc327	(Lillemo et al., 2008)	4.2–15.2%
5AL	QPm.nau-5AL	TA2027	Xcfd39/Xmag1491-Xmag1493	(Jia et al., 2009)	59%
5AL	QPm.umb-5AL	Folke	wPt-2426	(Lillemo et al., 2012)	4–9.7%
5AS	QPm.ttu-5A	*Triticum militinae*	Xgwm186–Xgwm415	(Jakobson et al., 2006)	4–6%
5AS	QPm.sfr-5A.1	Oberkulmer	Xpsr644a-Xpsr945a	(Keller et al., 1999)	22.9%
5B	Pm16 (Pm30 may be the same)	Norman rec. line (Reader & Miller, 1991)	Xgwm159	(Chen et al., 2005)	
5B	Qaprpm.cgb-5B	Lumai 14	Xgwm213-Xgwm499	(Huang et al., 2008)	19.8%
5BL	Pm36	MG29896	Xcfd7, EST BJ261636, Xwmc75	(Blanco et al., 2008)	
5BL	Pm53	NC09BGTS16	Xwmc759, Xgwm499, IWA6024, IWA2454, Xgwm408	(Petersen et al., 2015)	
5BL	PmAs846	N9134 (Wang et al., 2007), N9738 (Xue et al., 2012a)	XMAG2498–Pm36/XBJ261635 – XFCP1	(Xue et al., 2012a)	
5BL	MlWE29	WE29	Xgwm415, Xwmc75 Xwmc525, Xcfa2040, Xwmc273, XE13-2, Xmag1759, MlWE18, Xcfa2240	(Zhang et al., 2009)	
5BL	Ml3D232	3D232	Xwmc415- CJ832481	(Zhang et al., 2010)	
5BL	PmG25	N0308		(Alam et al.,	

				2013)	
5BL	QPm.inra-5B.2	Courtot	Xgwm790b	(Bougot et al., 2006)	11.1%
5BL	QPm.sfr-5B	Oberkulmer	Xpsr580b-Xpsr143	(Keller et al., 1999)	12.6%
5BS	Pm30	C20	gwm159-460, gwm159-500	(Liu et al., 2002)	
5BS	QPm.nuls-5B	Saar	Xbarc4-Xgwm274b	(Lillemo et al., 2008)	9.7%
5BS	QPm.umb-5BS	T2038	wPt-1261	(Lillemo et al., 2012)	3.1%
5BS	QPm.umb-5BS	Folke	Xbarc128a–Xgwm213	(Lillemo et al., 2012)	8.1–12.9%
5BS	QPm.ttu-5B	Tahti	Xgwm133.mi6-Xgwm205.mi1	(Jakobson et al., 2006)	4–6%
5D	QPm.caas-5D	W7984	Xmwg922-Xbcd1103	(Huo et al., 2005)	5.9%
5D	qApr5D	Yumai 57	Xwmc215-Xgdm63	(Zhang et al., 2008)	1.3%
5DL	Pm34	NC97BGTD7	Xbarc177-Xbarc144	(Miranda et al., 2006)	
5DL	Pm35	NC96BGTD3	Xcfd26	(Miranda et al., 2007)	
5DL	PmM53	M53		(Li et al., 2005)	
5DL	PmY201	Y201	Xgwm174	(Sun et al., 2006)	
5DL	PmY212	Y212	Xcfd57	(Sun et al., 2006)	
5DL	PmAeY2	Y189		(Zhang & Lang, 2007)	
5DL	QPm.crag-5D.1	RE714	Xgwm639a-Xgwm174	(Mingeot et al., 2002)	22.2–54.3%
5DL	QPm.crag-5D.2	RE714	Xcfd8B9-Xcfd4A6	(Mingeot et al., 2002)	37.8%
5DL	QPm.inra-5D.1	RE714	Xcfd26	(Chantret et al., 2001)	28.1–37.7%
5DL	QPm.inra-5D.2	RE714	XgbxG083c	(Chantret et al., 2001)	37.7%
5DL	QPmVpn.inra-5D	Courtot	Xcfd8	(Bougot et al., 2006)	11%
5DS	Pm2a	Ulka/XX 194 (Lutz et al., 1995a)	Xcfd81	(Qiu et al., 2006)	
	Pm2b	KM2939 (Ma et al., 2015)	Xcfd81, Xbwm25, Xbwm21, Xbwm20	(Lu et al., 2015)	
	Pm2c	Niaomai	Xcfd81 – Xcfd78	(Xu et al., 2015)	
5DS	Pm46 (Pm48)(APR)	Tabasco	Xgwm205, Xmp510(BE498794), Xcfd81	(Gao et al., 2012)	
5DS	PmLX66	Liangxing 66	SCAR203, Xcfd81	(Huang et al., 2012b)	
5DS	PmW14 (allelic with Pm2)	Wennong 14		(Sun et al., 2015)	
5DS	QPm.inra-5D	RE9001	cfd189	(Bougot et al., 2006)	9%
6VS.6AL	Pm21(Pm31)(Chen et al., 1995)	Yangmai 5 line	Xgwm459, Xgwm334, Xgwm1009,	(Xie et al., 2012a)	

			Xgwm1040, Xbarc171, Xgwm1296		
6AL	QPm.inra-6A	RE714	MIRE(Xgwm427)	(Chantret et al., 2001)	8.8–13.4%
6AL	QPm.crag-6A	RE714	MlRE	(Mingeot et al., 2002)	19.8–53.9%
6AS	Pm56	LM47-6		(Hao et al., 2018)	
6AS	PmY39-2	N9628-2	Xwmc553, Xwmc684 Xcfd39, Xgwm126, MAG1491, MAG1493, MAG1494, MAG2170	(Liu et al., 2008)	
6AS	CP1	Pedroso	MAG1200b	(Marone et al., 2013)	12.6%
6B-6G	Pm27	146-155-T	Xpsp3131	(Jarve et al., 2000)	
6B/6S	PmY150	*Ae. longissima*	Xgwm325, Xwmc382, Xwmc397	(Zhou et al., 2005)	
6B	Qaprpm.cgb-6B	Hanxuan 10	Xgwm193-P3470-210	(Huang et al., 2008)	21%
6BL	Pm54	AGS 2000	Xgpw2344, wPt-9256, Xbarc134	(Hao et al., 2015)	
6BL	PmG3M	G-305-3M (wild emmer accession)	Xuhw213	(Xie et al., 2012b)	
6BL	QPm.caas-6BL.1	Huixianhong	Xgwm219-Xbarc24	(Asad et al., 2012)	2.5–5.2%
6BL	QPm.caas-6BL.2	Huixianhong	Xbarc24-Xbarc345	(Asad et al., 2012)	0.5–1.9%
6BL	CP3	Pedroso	Xgwm219-Xgwm889	(Marone et al., 2013)	14.8–18.5%
6BL	CP4	Pedroso	wPt-5270	(Marone et al., 2013)	13.4%
6BS	Pm11	Chinese Spring		(Tosa et al., 1988)	
6BS-6SS·6SL	Pm12	Trans.line 31 (Jia et al., 1996)	Xwmc105 - Xcau127	(Song et al., 2007)	
6BS	Pm14	Norin 10		(Tosa & Sakai, 1990)	
6BS.6RL	Pm20	KS93WGRC28		(Friebe et al., 1994)	
6BS	QPm.sfr-6B	Forno	Xpsr167b-Xpsr964	(Keller et al., 1999)	8.7%
6BS	QPm.umb-6BS	Folke	wPt-6437-Xwmc494	(Lillemo et al., 2012)	6.5–10.3%
6BS	QPm.caas-6BS	Bainong 64	Xbarc79-Xgwm518	(Lan et al., 2009)	10.3–16.0%
6DS	Pm45	D57	Xmag6176	(Ma et al., 2011)	
6DS	Pm07J126	07jian126	Xbarc183, Xgpw7425, Xwmc75, Xgwm408, Xwmc810, Xbarc232, Xbarc142	(Yu et al., 2012)	
6DS	QPm.osu-6D	2174	BARC196	(Chen et al., 2009)	5%
7A	QPm.tut-7A	8.1	Xgwm635–Xbarc70-Waxy	(Jakobson et al., 2012)	5–28%
7A	Qaprpm.cgb	Hanxuan 10	CWM462.2-	(Huang et al.,	8%

	-7A		Xgwm635.2	2008)	
7A	Qaprpm.cgb -7A	Hanxuan 10	Xgwm282-P1111-202	(Huang et al., 2008)	15.7%
7AL	Pm1a	Axminister (Hsam et al., 1998)	UBC320420, UBC638550	(Hu et al., 1997)	
			WHS178-9.4 kb-EcoRI	(Liu et al., 2001)	
			CDO347	(Ma et al., 1994)	
			Xmwg2062, Xcdo347, Xpsr121, Xpsr148, Xpsr680, Xpsr687, wir148, C607, STS638542, Xksuh9	(Neu et al., 2002)	
7AL	Pm1b	MocZlatka		(Hsam et al., 1998)	
7AL	Pm1c(Pm18)	Weihestephan M1N (Hsam et al., 1998)	Xwhs178-15 kb-EcoRI, OPH-111900	(Liu et al., 2001)	
			S19M22-325/200	(Hartl et al., 1999)	
			S14M20-137/138		
7AL	Pm1d	*T. spelta* var. *duhamelianum*		(Hsam et al., 1998)	
7AL	Pm1e(Pm22)	Virest	GWM344-null–S13M26-372	(Singrun et al., 2003)	
7AL	Pm9	Normandie		(Bennett, 1984)	
7AL	Pm37	NC99BGTAG11	Xgwm332, Xwmc790	(Perugini et al., 2008)	
7AL	Pm59	PI 181356	Xmag1759 – Xmag1714	(Tan et al., 2018)	
7AL	Pm60	PI428309	Xwmc273.3	(Zou et al., 2018)	
7AL	mlRD30(seedling stage, distal to Pm1)	RD30	Xgwm344-Xksuh9	(Singrun et al., 2004)	
7AL	PmU	UR206	Xgwm273, Xpsp3003	(Qiu et al., 2005)	
7AL	Mlm2033 (likely allelic to Mlm80)	TA2033	Xgwm344, Xmag2185	(Yao et al., 2007)	
	Mlm80	M80		(Yao et al., 2007)	
7AL	mllW72	IW72	Xmag1759, Xmag2185, Xgwm344	(Ji et al., 2008b)	
7AL	MlWE18	3D249		(Han et al., 2009)	
7AL	MlAG12	NC06BGTAG12	Xwmc273, Xwmc346	(Maxwell et al., 2009)	
7AL	PmG16	G18-16 (wild emmer accession)	Xgwm1061, Xgwm344, wPt-1424, wPt6019, wPt-0494, wPt9217, Xwmc809	(Ben-David et al., 2010)	
7AL	PmTb7A.1	pau5088	wPt4553, Xcfa2019(Ta7AL-4556232)	(Chhuneja et al., 2012)	
7AL	PmTb7A.2	pau5088 (Chhuneja et al., 2012)	7AL-4426363	(Elkot et al., 2015)	
7AL	QPm.umb-	T2038	Xgwm428–Xcfa2040	(Lillemo et al.,	6.4–13%

	7AL			2012)	
7AS	QPm.inra-7A	RE714	Xfba069-Xgwm344	(Chantret et al., 2001)	2.9–6.4%
7AS	QPm.caas-7A	Bainong 64	Xbarc127-Xbarc174	(Lan et al., 2009)	6.3–7.1%
7B	Mljy	Jieyan 94-1-1		(Huang et al., 2002)	
7B	Mlsy	Siyan 94-2-1		(Huang et al., 2002)	
7B	Qaprpm.cgb-7B	Lumai 14	Xwmc273-Xwmc276	(Huang et al., 2008)	12.6%
7BL	Pm5a	Hope		(Law & Wolfe, 1966)	
7BL	Pm5b	Ibis		(Hsam et al., 2001)	
7BL	Pm5c	Kolandi		(Hsam et al., 2001)	
7BL	Pm5d	IGV 1-455 (Hsam et al., 2001)	Xgwm611, Xgwm577, Xwmc581	(Nematollahi et al., 2008)	
7BL	Pm5e	Fuzhuang 30 (Huang et al., 2003a)	GWM1267-136	(Huang et al., 2003b)	
7BL	mlxbd(Pm5)	Xiaobaidong		(Huang et al., 2000a)	
7BL	PmTm4	Tangmai 4	Xgwm61, Xbarc1073, Xbarc82	(Hu et al., 2008)	
7BL	PmH	Hongquanmang	Xgwm611, Xpsp3033	(Zhou et al., 2005)	
7BL	QPm.sfr-7B.1	Forno	Xpsr593c-Xpsr129c	(Keller et al., 1999)	11.3%
7BL	QPm.sfr-7B.2	Forno	Xglk750-Xmwg710a	(Keller et al., 1999)	31.8%
7BL	QPm.inra-7B	RE714	Xgwm577	(Chantret et al., 2001)	1.7%
7BL	QPm.crag-7B	RE714	XpdaC01-XgbxR035b	(Mingeot et al., 2002)	22.8–33.5%
7BL	QPm.nuls-7BL	Saar	Xwmc581-XwPt8007	(Lillemo et al., 2008)	4.9%
7BS	Pm40	GRY19	Xwmc426, Xwmc334, Xgwm297, Xwmc364	(Luo et al., 2009)	
7BS	Pm47	Hongyanglazi	Xgpw2097-Xgwm46	(Xiao et al., 2013)	
7BS	PmE	TAI7047	Xgwm297	(Ma et al., 2007)	
7D	Pm19	XX 186		(Lutz et al., 1995a)	
7D	Qaprpm.cgb-7D	Hanxuan 10	Xwmc436-Xgwm44	(Huang et al., 2008)	3.8–4.6%
7D	Qaprpm.cgb-7D	Hanxuan 10	Xgdm88-WMC463	(Huang et al., 2008)	14.2%
7D	QPm.caas-7D	Opata 85	Xwg834-Xbcd1438	(Huo et al., 2005)	29.6%
7DL	Pm29	Pova	S24M13-233, S19M23-240, S22M26-192, S25M15-145, S13M23-442,	(Zeller et al., 2002)	

			S22M21-217, S17M25-226		
7DS	Pm15	Norin 26		(Tosa & Sakai, 1990)	
7DS	Pm38(APR)	RL6058	Xgwm1220, Xgwm295	(Spielmeyer et al., 2005)	
7DS	MLNCD1	NC96BGTD1	Xgwm635-Xgpw328	(Maxwell et al., 2012)	
7DS	QPm.caas-7DS	Libellula	XcsLV34-Xgwm295	(Asad et al., 2012)	7.6–13.8%
7DS	QPm.ipk-7D	Optata	Xwg834-Xbcd1872	(Borner et al., 2002)	
7DS	QPm.inra-7D.1	Courtot	Xgpw1106	(Bougot et al., 2006)	10.6%
7DS	QPm.caas-7DS	Chinese Spring	IWB41108–IWB53819	(Jia et al., 2018)	4.21%
7DS	QPm.caas-7DS	Fukuho-komugi	Ltn-Xgwm295.1	(Liang et al., 2006)	12%
7DS	Yr18/Lr34/Pm38	Saar	Xgwm1220-Xswm10	(Lillemo et al., 2008)	19–56.5%
	Ml-Ad	Adlungs Alemannen		(Lutz et al., 1995b)	
	Ml-Br	Bretonischer		(Lutz et al., 1995b)	
	Ml-Ga	Garnet		(Lutz et al., 1995b)	
	PmP	Xiaobing	XM55P66, XM55P37	(Zhou et al., 2005)	

*Phenotypic variance explained by QTL given in corresponding reference.

Table S3.1: Disease response on a scale of 0-5 evaluated in 329 wheat cultivars across three environments.

Cultivar	2016	2017	2018
073-44	1.5	2.3	0.5
19401	3	2	NA
19720	2	2	NA
92FS-16	3	0.7	1
AEGYLOPS14257	4	4.3	4
AFGHANISTAN-7	3	3	2.5
AHGAF	3	3	3.5
ALBIDUM-24	1.5	1	1
Al-wheat	4	4.7	4.5
ARTEMOVKA	2.5	3	5
AUS19392	2.5	1.3	4.5
AUS19393	2.5	3	4
AUS19394	3	1	4
AUS19395	4.5	3	4.5
AUS19396	2	0	2
AUS19397	1	0.3	1.5
AUS19398	1.5	0.3	1
AUS19399	2	0.3	0.5
AUS19400	2	1	2.5
AUS19402	2	3	4
AUS19403	4.5	3	4.5
AUS19719	3	1	3.5
B-T-17	2	2.7	5
B-T-35	1.5	0.3	4
B-T-38	1	1.3	5
B-T-51	1.5	1.3	4
BUKOVINKA	1.5	0	2
CAZ53	4.5	3.3	4
E-M-S-SUMMIT70-8	3.5	4.7	5
EXITOB	4	3.3	5
FERRUGINEUM	4	4	4.5
FRETES1	4	5	4.5
FRETES2	4	3.7	4.5
FRETES3	4	5	4.5
Glover	2.5	1.3	3.5
H-001	2.5	3.3	4
H-002	NA	3.3	3.5
H-003	4	4	5
H-004	5	4.7	4.5
H-005	3.5	4.3	4

H-006	4.5	4.7	5
H-007	NA	3	4.5
H-008	3	0.3	3
H-009	3	2.7	4.5
H-010	4	4.3	5
H-011	3.5	3.7	4
H-012	NA	3	5
H-013	1.5	1	4.5
H-014	2.5	2.7	3.5
H-015	NA	1.3	3
H-016	NA	1.7	4
H-017	NA	3	4
H-019	2.5	2.7	NA
H-020	1	1.3	4
H-021	3	1.3	3.5
H-022	2.5	3.3	2.5
H-023	1.5	4.7	3
H-024	2	3.7	5
H-025	NA	2.7	3.5
H-027	2	4.7	3
H-028	1.5	1.3	5
H-030	NA	2	1
H-031	NA	0.7	2
H-033	1.5	2.7	3.5
H-034	3	4	3.5
H-037	1	2.7	4.5
H-038	4.5	2.7	3
H-039	3.5	3.3	4.5
H-040	NA	1	4.5
H-041	2.5	4	4
H-042	3	1.3	4.5
H-043	1.5	4	3.5
H-044	2.5	4	5
H-045	4	4.7	4.5
H-046	4.5	5	4
H-047	3	3.7	4
H-048	4	4.3	4.5
H-049	2	3.7	4.5
H-051	2.5	3.3	2.5
H-052	3	3.3	4
H-053	3	2.7	4
H-054	2.5	3.7	3
H-055	NA	3	4.5

H-056	2.5	3.7	NA
H-057	2.5	1.7	4
H-058	2.5	3.7	4
H-059	2	3.3	3.5
H-060	3.5	3	3
H-061	2.5	3	3
H-062	3	4	3.5
H-063	2.5	3	4.5
H-064	3	3.3	3.5
H-065	3	4	4
H-066	2.5	2	5
H-067	NA	1.7	2.5
H-068	3	4	3.5
H-069	4	1.7	3
H-070	4.5	4.7	4
H-071	4.5	3.7	5
H-073	NA	4	5
H-074	2.5	4	NA
H-075	3.5	4	5
H-076	2.5	3.3	NA
H-077	3	4.7	3
H-078	2.5	1.7	3.5
H-079	1	1.3	1
H-080	4	2	1.5
H-081	3	1.7	3
H-082	2.5	2	3
H-083	2	2.7	3
H-084	3	3.3	4
H-085	2.5	4.3	5
H-086	3	3	4
H-087	2	2.7	4.5
H-088	2.5	3.3	4
H-089	2.5	2	4
H-090	3	4	5
H-091	3.5	3	4
H-092	1	1.7	4
H-093	3	3.7	4
H-094	2.5	4.7	3.5
H-095	2.5	2	4.5
H-096	2	3	3
H-097	NA	3.3	5
H-098	1.5	2.3	5
H-099	NA	2.3	5

H-100	2	3	5
H-101	NA	2.7	5
H-103	NA	3	2
H-104	NA	1.3	4.5
H-105	2	1.5	1.5
H-106	2	1	3.5
H-107	2.5	4	4
H-108	3	3.7	4
H-109	2.5	4	5
H-110	3.5	5	5
H-111	4	4.3	4
H-112	2.5	3	4.5
H-113	3	5	4.5
H-114	NA	1.7	3.5
H-115	3.5	1.3	4.5
H-116	1.5	0.3	2.5
H-117	1	1	2
H-118	2	1.3	4
H-119	2.5	3.3	5
H-120	4.5	3	4.5
H-121	3.5	3.7	4.5
H-122	3.5	3	4
H-125	NA	3.7	5
H-126	4	1.3	5
H-127	2.5	3	4
H-129	2	4.7	4.5
H-130	1.5	3	3
H-131	4.5	3.7	4.5
H-132	2.5	3.3	3.5
H-133	3	3.3	4.5
H-135	1.5	1.3	5
H-136	NA	3.3	5
H-137	4.5	3.7	5
H-138	3.5	1.3	5
H-141	1	1	4.5
H-142	1.5	1	3
H-143	3	2.7	3.5
H-144	2.5	3.3	4
H-145	2.5	2.3	3.5
H-146	1.5	2.3	3
H-147	2.5	3.3	4
H-148	2	2.3	4
H-149	4	3.3	5

H-150	1	1.3	3
H-151	1.5	1.3	1.5
H-152	2.5	3	2
H-153	3.5	1	3.5
H-154	3.5	1.7	3.5
H-157	NA	3.7	4.5
H-159	4.5	3.7	5
H-160	2.5	0.7	3.5
H-161	2	2	2
H-163	3	1.7	5
H-164	1	4.3	4
H-165	3.5	4.7	5
H-167	2.5	4	2
H-168	2.5	4	3.5
H-169	2.5	2	3.5
H-170	3	2	4.5
H-171	2.5	3.7	5
H-172	2	2.3	2.5
H-176	3	4	4
H-177	3.5	3.3	4.5
H-178	4.5	3.7	3.5
H-179	4	4.7	4
H-180	3	2	4.5
H-181	3.5	4.7	5
H-183	3.5	1.7	4.5
H-184	4	3.7	5
H-185	2.5	3.3	4
H-186	4.5	4.7	4
H-187	2	1.3	1
H-189	3.5	3.3	4
H-191	3	3.3	5
H-192	3	3	4
H-193	2	2	3
H-194	2.5	1.7	2.5
H-195	4.5	4	2
H-196	3	2.3	3
H-197	4.5	4.3	5
H-199	4.5	3.7	5
H-200	5	4	NA
H-201	2.5	2	3
H-202	3.5	3	4.5
H-203	1	1.3	2
H-204	3	4.7	4.5

H-205	3.5	4.7	5
H-207	2	1	4
H-208	1.5	1	3.5
H-209	2.5	3.3	5
H-210	2	2	3.5
H-211	2	0.3	1
H-212	4	2.3	5
H-213	3.5	1	3
H-214	2.5	3.7	4
H-215	3.5	3.3	3.5
H-216	2.5	2.3	4
H-217	4	1.7	3.5
H-218	2	1.3	3
H-220	2.5	1.3	2.5
H-221	2	3.3	3
H-222	3	2.7	5
H-223	2.5	2.7	3
H-224	1	3.3	3
H-225	3	2.7	2
H-227	3.5	4.3	3.5
H-228	3	1.3	3.5
H-229	3	3	3.5
H-230	1.5	1.3	2
H-231	4.5	4.7	5
H-232	2.5	3.7	2
H-233	3.5	2.3	NA
H-234	4	3.3	4
H-235	3.5	2.7	5
H-236	3.5	3.3	4.5
H-237	3	2	4.5
H-238	3.5	2.7	3
H-239	3.5	2.7	2.5
H-240	2.5	4	4
H-241	3.5	4	3
H-242	3.5	2.7	4.5
H-243	2.5	2	2
H-246	3.5	3.3	5
H-247	2	0	1
H-249	3.5	1.7	4
H-250	4.5	3.7	4.5
H-251	3	3.7	3
Hartog	3	2.3	3
HB88I-172	4	3.7	3.5

IG43428	2	1	2
KARAGAN	1.5	1.3	2
KAZAKHSTANSKAJA126	4	2.7	4
KHARCHIA65	2	0.5	1.5
KOPARA73	2	0.3	4
KORDCLPLUS	2.5	1	3
KZYL-SARK	NA	2.7	3.5
LU26S	3	1	4.5
Mac	2.5	1.7	5
MAHONDEMIAS	2	2.7	5
NAJAH	NA	2.7	3
NAPOSTA	NA	3.7	4
ONOHOISKAJA4	4	4	3
PALESTINE8	3	3.3	4
PI178012	3	3.3	3.5
PI178704	2.5	2.7	3
PI180988	2.5	4.3	2.5
PI264952	3	0.3	2.5
POBEDA	NA	0.3	2
PretoAmarelo	2.5	2.7	2.5
Revenue	2.5	1.3	3.5
SEAGULL	NA	2	3.5
SELOFHOURANI27-1	4	3	3
SKALA	NA	2	3
SOCIEDADNACIONALDEAGRICULTURA	2.5	2.3	3.5
SONNENWEIZEN	NA	3.3	3.5
SURHAK5688	4.5	3.7	4.5
SURHAKMESTNYJ	NA	3.7	3
SW9550101	2.5	2	3
SW9550192	3	3	3
SW9550213	3.5	3.7	3.5
SW9550292	2.5	4.3	3.5
TAJAZNAJA4	1.5	1.3	3
TURCICUM1	3	4.3	4
VYS	2	0.3	4.5
W5013EY-11-1	4.5	5	4.5
W5228HZ-1	2.5	3.3	3
W5924DT-34	4.5	4	4.5
wheat-2HBYDV	2.5	4	4.5
WL-wheat	3.5	2.3	5
X-165	NA	0.7	2.5
X-253	NA	3	4
X-254	NA	4	5

X-255	NA	3.7	3.5
X-256	NA	3	4.5
X-257	NA	4.3	5
X-258	NA	4.3	3.5
X-259	NA	3.3	4
X-26	NA	3	4
X-260	NA	1.3	3.5
X-261	NA	3	3.5
X-B	NA	3	3
X-C	NA	2.3	3
X-D	NA	1	2
X-E	NA	2.3	3
X-F	NA	2.7	3.5
Yang17	3.5	2	4
Yannong15	3	1	3.5
Yannong19	2.5	1.7	4
YRODY1006	2	2.3	3.5
Yu-01	3.5	2.3	4
Yu-02	1.5	0.3	3
Yu-03	1.5	2.3	4.5
Yu-04	2.5	2	3.5
Yu-05	2	2.3	5
Yu-06	3.5	1	2.5
Yu-07	2	2	3
Yu-08	3	2	4
Yu-09	2	2.3	4
Yu-10	2.5	2	4.5
Yu-11	2	2	4.5
Yu-12	2.5	1.3	5
Yu-13	1	0.7	2.5
ZHONG4	2	3.3	4
Min	1	0	0.5
Median	2.5	3	4
Max	5	5	5
Mean	2.8	2.7	3.7

Table S3.2: Basic analysis of SNP markers used for genome-wide association study (GWAS).

Chromosome	No. of SNP	Chromosome length (cM)	Marker density (Marker/cM)	Average PIC	Average LD decay (cM)
1A	688	481.08	1.4	0.29	1.42
1B	876	550.71	1.6	0.31	3.71
1D	249	291.84	0.9	0.26	22.21
2A	678	614.77	1.1	0.33	11.81
2B	952	583.38	1.6	0.30	1.01
2D	321	368.68	0.9	0.25	28.01
3A	521	617.24	0.8	0.29	2.17
3B	695	617.82	1.1	0.30	1.45
3D	83	454.76	0.2	0.31	5.53
4A	497	641.89	0.8	0.29	2.14
4B	448	343.89	1.3	0.29	3.77
4D	20	255.65	0.1	0.26	3.83
5A	736	742.53	1.0	0.31	3.41
5B	1051	676.14	1.6	0.30	10.48
5D	93	575.13	0.2	0.27	6.07
6A	601	387.9	1.5	0.32	3.56
6B	649	419.8	1.5	0.31	1.13
6D	74	352.15	0.2	0.31	7.93
7A	588	708.87	0.8	0.30	1.44
7B	660	570.06	1.2	0.30	1.45
7D	77	429.77	0.2	0.26	4.48
A genome	4309	4194.28	1.1	0.31	3.71
B genome	5331	3761.8	1.4	0.30	3.29
D genome	917	2727.98	0.4	0.27	11.15
Whole genome	10557	10684.06	1.0	0.29	6.05

SNP = Single-nucleotide polymorphism; PIC = Polymorphism information content; LD = Linkage disequilibrium.

Table S3.3: Summary of PIC values for 10557 genotyping markers.

Chromosome	No. of SNP with PIC < 0.25	No. of SNP with PIC 0.25-0.5
1A	225	463
1B	147	729
1D	146	103
2A	84	594
2B	177	775
2D	133	188
3A	158	363
3B	147	548
3D	16	67
4A	126	371
4B	134	314
4D	10	10
5A	115	621
5B	204	847
5D	36	57
6A	84	517
6B	111	538
6D	11	63
7A	128	460
7B	160	500
7D	29	48
Total	2381	8176

SNP = Single-nucleotide polymorphism; PIC = Polymorphism information content.

Table S3.4: SNPs significantly associated with wheat powdery mildew resistance.

QTL	Tag-SNP	Chromosome	Genetic position (cM)	$\log_{10}(P)$	R^2	Other detected SNPs	Environment
PM_1B1	IWB73714	1B	491.82	3.28	0.04		2016,BLUE
PM_1B2	IWB52607	1B	348.9	3.14	0.03		2017,BLUE
PM_2B1	IWB36753	2B	431.19	3.7	0.05	IWB11366	2016
PM_2B2	IWB6167	2B	413.97	3.23	0.03		2017
PM_2D	IWB74	2D	208.94	5.15	0.06		2017
PM_3A1	IWB48794	3A	541.15	3.17	0.03		BLUE
PM_3A2	IWB67770	3A	47.94	3.71	0.05		2016, 2017,BLUE
PM_3B1	IWB64989	3B	38.79	3.72	0.05		2016
PM_3B2	IWB67768	3B	38.79	3.07	0.03	IWB67768	2017,BLUE
PM_3B3	IWB42046	3B	269.78	3.61	0.04	IWB42046	2016
PM_3B4	IWA7225	3B	297.01	3.35	0.04	IWA7225	2018
PM_5A1	IWA6287	5A	210.44	4.05	0.04	IWA6287	2017
PM_5A2	IWB10765	5A	493.65	3.46	0.04	IWA674	2018
PM_5B1	IWA22	5B	498.31	3.44	0.04	IWA22	2016
PM_5B2	IWB7206	5B	359.4	3.05	0.03	IWB7206	2018
PM_6A	IWB23521	6A	32.37	3.7	0.05	IWB72957, IWB43805, IWB67415, IWB58271, IWB882, IWB67416, IWB35219	2016
PM_6B1	IWB60950	6B	417.51	3.41	0.03	IWB4385	2017
PM_6B2	IWB28017	6B	417.51	3.69	0.04	IWB28017	2018,BLUE
PM_6D	IWB59264	6D	335.87	3.35	0.03	IWB59264	BLUE
PM_7D	IWA5557	7D	297.62	3.99	0.05	IWB49398, IWB34836, IWB6064, IWB36226, IWB44273	2016

Tag-SNP = representative SNP marker of QTL; *P* = probability of type-I error measured by enriched compressed mixed linear model (ECMLM) correcting with principal components and kinship; R^2 = the difference in the likelihood ratio calculated by ECMLM which could be interpreted as variation explained by the SNP.

Table S3.5: Distribution of positive and negative alleles of tag-SNP in 329 wheat cultivars.

Cultivar	No. of positive allele	No. of negative allele	Mean disease response	Type
073-44	13	7	1.4	R
19401	13	7	2.5	R
19720	14	6	2.0	R
92FS-16	14	6	1.6	R
AEGYLOPS14257	11	9	4.1	S
AFGHANISTAN-7	9	11	2.8	R
AHGAF	8	12	3.2	S
ALBIDUM-24	12	8	1.2	R
Al-wheat	9	11	4.4	S
ARTEMOVKA	12	8	3.5	S
AUS19392	11	9	2.8	R
AUS19393	7	13	3.2	S
AUS19394	8	12	2.7	R
AUS19395	5	15	4.0	S
AUS19396	13	7	1.3	R
AUS19397	15	5	0.9	R
AUS19398	12	8	0.9	R
AUS19399	12	8	0.9	R
AUS19400	13	7	1.8	R
AUS19402	5	15	3.0	R
AUS19403	4	16	4.0	S
AUS19719	6	14	2.5	R
B-T-17	8	12	3.2	S
B-T-35	12	8	1.9	R
B-T-38	9	11	2.4	R
B-T-51	12	8	2.3	R
BUKOVINKA	13	7	1.2	R
CAZ53	6	14	3.9	S
E-M-S-SUMMIT70-8	5	15	4.4	S
EXITOB	8	12	4.1	S
FERRUGINEUM	4	16	4.2	S
FRETES1	5	15	4.5	S
FRETES2	9	11	4.1	S
FRETES3	9	11	4.5	S
Glover	7	13	2.4	R
H-001	10	10	3.3	S
H-002	7	13	3.4	S
H-003	3	17	4.3	S
H-004	4	16	4.7	S
H-005	5	15	3.9	S
H-006	6	14	4.7	S

H-007	5	15	3.8	S
H-008	13	7	2.1	R
H-009	5	15	3.4	S
H-010	6	14	4.4	S
H-011	8	12	3.7	S
H-012	10	10	4.0	S
H-013	12	8	2.3	R
H-014	9	11	2.9	R
H-015	11	9	2.2	R
H-016	11	9	2.9	R
H-017	8	12	3.5	S
H-019	10	10	2.6	R
H-020	10	10	2.1	R
H-021	10	10	2.6	R
H-022	11	9	2.8	R
H-023	10	10	3.1	S
H-024	12	8	3.6	S
H-025	11	9	3.1	S
H-027	11	9	3.2	S
H-028	8	12	2.6	R
H-030	12	8	1.5	R
H-031	8	12	1.4	R
H-033	7	13	2.6	R
H-034	11	9	3.5	S
H-037	9	11	2.7	R
H-038	9	11	3.4	S
H-039	13	7	3.8	S
H-040	13	7	2.8	R
H-041	13	7	3.5	S
H-042	9	11	2.9	R
H-043	10	10	3.0	R
H-044	12	8	3.8	S
H-045	8	12	4.4	S
H-046	8	12	4.5	S
H-047	8	12	3.6	S
H-048	9	11	4.3	S
H-049	9	11	3.4	S
H-051	9	11	2.8	R
H-052	8	12	3.4	S
H-053	9	11	3.2	S
H-054	10	10	3.1	S
H-055	12	8	3.8	S
H-056	9	11	3.1	S

H-057	8	12	2.7	R
H-058	9	11	3.4	S
H-059	9	11	2.9	R
H-060	10	10	3.2	S
H-061	9	11	2.8	R
H-062	8	12	3.5	S
H-063	9	11	3.3	S
H-064	8	12	3.3	S
H-065	13	7	3.7	S
H-066	9	11	3.2	S
H-067	14	6	2.1	R
H-068	11	9	3.5	S
H-069	9	11	2.9	R
H-070	6	14	4.4	S
H-071	7	13	4.4	S
H-073	11	9	4.5	S
H-074	10	10	3.3	S
H-075	8	12	4.2	S
H-076	9	11	2.9	R
H-077	6	14	3.6	S
H-078	9	11	2.6	R
H-079	11	9	1.1	R
H-080	7	13	2.5	R
H-081	9	11	2.6	R
H-082	14	6	2.5	R
H-083	12	8	2.6	R
H-084	8	12	3.4	S
H-085	12	8	3.9	S
H-086	8	12	3.3	S
H-087	9	11	3.1	S
H-088	6	14	3.3	S
H-089	10	10	2.8	R
H-090	8	12	4.0	S
H-091	12	8	3.5	S
H-092	11	9	2.2	R
H-093	9	11	3.6	S
H-094	11	9	3.6	S
H-095	12	8	3.0	R
H-096	11	9	2.7	R
H-097	9	11	4.2	S
H-098	12	8	2.9	R
H-099	12	8	3.7	S
H-100	10	10	3.3	S

H-101	13	7	3.9	S
H-103	13	7	2.5	R
H-104	10	10	2.9	R
H-105	11	9	1.7	R
H-106	8	12	2.2	R
H-107	9	11	3.5	S
H-108	10	10	3.6	S
H-109	10	10	3.8	S
H-110	7	13	4.5	S
H-111	9	11	4.1	S
H-112	11	9	3.3	S
H-113	7	13	4.2	S
H-114	14	6	2.6	R
H-115	11	9	3.1	S
H-116	11	9	1.4	R
H-117	12	8	1.3	R
H-118	11	9	2.4	R
H-119	10	10	3.6	S
H-120	8	12	4.0	S
H-121	10	10	3.9	S
H-122	10	10	3.5	S
H-125	10	10	4.4	S
H-126	8	12	3.4	S
H-127	8	12	3.2	S
H-129	6	14	3.7	S
H-130	12	8	2.5	R
H-131	4	16	4.2	S
H-132	11	9	3.1	S
H-133	11	9	3.6	S
H-135	15	5	2.6	R
H-136	10	10	4.2	S
H-137	12	8	4.4	S
H-138	7	13	3.3	S
H-141	11	9	2.2	R
H-142	10	10	1.8	R
H-143	7	13	3.1	S
H-144	10	10	3.3	S
H-145	10	10	2.8	R
H-146	11	9	2.3	R
H-147	12	8	3.3	S
H-148	10	10	2.8	R
H-149	7	13	4.1	S
H-150	12	8	1.8	R

H-151	12	8	1.4	R
H-152	12	8	2.5	R
H-153	11	9	2.7	R
H-154	11	9	2.9	R
H-157	9	11	4.1	S
H-159	9	11	4.4	S
H-160	9	11	2.2	R
H-161	11	9	2.0	R
H-163	11	9	3.2	S
H-164	13	7	3.1	S
H-165	10	10	4.4	S
H-167	11	9	2.8	R
H-168	10	10	3.3	S
H-169	12	8	2.7	R
H-170	13	7	3.2	S
H-171	9	11	3.7	S
H-172	14	6	2.3	R
H-176	10	10	3.7	S
H-177	4	16	3.8	S
H-178	5	15	3.9	S
H-179	7	13	4.2	S
H-180	10	10	3.2	S
H-181	7	13	4.4	S
H-183	12	8	3.2	S
H-184	7	13	4.2	S
H-185	9	11	3.3	S
H-186	9	11	4.4	S
H-187	10	10	1.4	R
H-189	7	13	3.6	S
H-191	9	11	3.8	S
H-192	11	9	3.3	S
H-193	7	13	2.3	R
H-194	10	10	2.2	R
H-195	7	13	3.5	S
H-196	9	11	2.8	R
H-197	3	17	4.6	S
H-199	4	16	4.4	S
H-200	5	15	4.5	S
H-201	10	10	2.5	R
H-202	8	12	3.7	S
H-203	14	6	1.4	R
H-204	6	14	4.1	S
H-205	7	13	4.4	S

H-207	12	8	2.3	R
H-208	11	9	2.0	R
H-209	10	10	3.6	S
H-210	11	9	2.5	R
H-211	15	5	1.1	R
H-212	7	13	3.8	S
H-213	10	10	2.5	R
H-214	10	10	3.4	S
H-215	12	8	3.4	S
H-216	12	8	2.9	R
H-217	12	8	3.1	S
H-218	12	8	2.1	R
H-220	13	7	2.1	R
H-221	9	11	2.8	R
H-222	11	9	3.6	S
H-223	13	7	2.7	R
H-224	12	8	2.4	R
H-225	12	8	2.6	R
H-227	8	12	3.8	S
H-228	11	9	2.6	R
H-229	12	8	3.2	S
H-230	14	6	1.6	R
H-231	3	17	4.7	S
H-232	12	8	2.7	R
H-233	9	11	2.9	R
H-234	9	11	3.8	S
H-235	10	10	3.7	S
H-236	9	11	3.8	S
H-237	11	9	3.2	S
H-238	6	14	3.1	S
H-239	7	13	2.9	R
H-240	9	11	3.5	S
H-241	8	12	3.5	S
H-242	10	10	3.6	S
H-243	8	12	2.2	R
H-246	7	13	3.9	S
H-247	11	9	1.0	R
H-249	7	13	3.1	S
H-250	4	16	4.2	S
H-251	6	14	3.2	S
Hartog	4	16	2.8	R
HB88I-172	7	13	3.7	S
IG43428	14	6	1.7	R

KARAGAN	10	10	1.6	R
KAZAKHSTANSKAJA126	6	14	3.6	S
KHARCHIA65	11	9	1.3	R
KOPARA73	16	4	2.1	R
KORDCLPLUS	15	5	2.2	R
KZYL-SARK	7	13	3.1	S
LU26S	13	7	2.8	R
Mac	15	5	3.1	S
MAHONDEMIAS	9	11	3.2	S
NAJAH	12	8	2.9	R
NAPOSTA	4	16	3.9	S
ONOHOISKAJA4	10	10	3.7	S
PALESTINE8	8	12	3.4	S
PI178012	11	9	3.3	S
PI178704	10	10	2.7	R
PI180988	7	13	3.1	S
PI264952	8	12	1.9	R
POBEDA	12	8	1.2	R
PretoAmarelo	6	14	2.6	R
Revenue	11	9	2.4	R
SEAGULL	12	8	2.8	R
SELOFHOURANI27-1	9	11	3.3	S
SKALA	10	10	2.5	R
SOCIEDADNACIONALDEAGRICULTURA	9	11	2.8	R
SONNENWEIZEN	8	12	3.4	S
SURHAK5688	4	16	4.2	S
SURHAKMESTNYJ	4	16	3.4	S
SW9550101	11	9	2.5	R
SW9550192	10	10	3.0	R
SW9550213	7	13	3.6	S
SW9550292	9	11	3.4	S
TAJAZNAJA4	10	10	1.9	R
TURCICUM1	4	16	3.8	S
VYS	14	6	2.3	R
W5013EY-11-1	5	15	4.7	S
W5228HZ-1	9	11	2.9	R
W5924DT-34	11	9	4.3	S
wheat-2HBYDV	10	10	3.7	S
WL-wheat	7	13	3.6	S
X-165	12	8	1.6	R
X-253	5	15	3.5	S
X-254	4	16	4.5	S
X-255	7	13	3.6	S

X-256	10	10	3.8	S
X-257	7	13	4.7	S
X-258	2	18	3.9	S
X-259	3	17	3.7	S
X-26	11	9	3.5	S
X-260	10	10	2.4	R
X-261	15	5	3.3	S
X-B	13	7	3.0	R
X-C	9	11	2.7	R
X-D	15	5	1.5	R
X-E	9	11	2.7	R
X-F	7	13	3.1	S
Yang17	12	8	3.2	S
Yannong15	10	10	2.5	R
Yannong19	13	7	2.7	R
YRODY1006	7	13	2.6	R
Yu-01	6	14	3.3	S
Yu-02	13	7	1.6	R
Yu-03	15	5	2.8	R
Yu-04	12	8	2.7	R
Yu-05	12	8	3.1	S
Yu-06	11	9	2.3	R
Yu-07	15	5	2.3	R
Yu-08	10	10	3.0	R
Yu-09	12	8	2.8	R
Yu-10	12	8	3.0	R
Yu-11	9	11	2.8	R
Yu-12	10	10	2.9	R
Yu-13	12	8	1.4	R
ZHONG4	7	13	3.1	S

Type indicates resistance type, either resistant (R) or susceptible (S).

Table S3.6: Comparison of GWAS results from ECMLM with and without correction for population structure.

	ECMLM (with PCs)		ECMLM (without PC)	
	Detected SNP	$-\log_{10}(P)$	Detected SNP	$-\log_{10}(P)$
2016	IWA5557	3.99	IWA5557	4.10
	IWB64989	3.72	IWB36753	3.83
	IWB67770	3.71	IWB49398	3.81
	IWB36753	3.70	IWB42046	3.71
	IWB23521	3.70	IWB67770	3.70
	IWB49398	3.69	IWB64989	3.69
	IWB42046	3.61	IWB23521	3.69
	IWB34836	3.51	IWB34836	3.63
	IWB67415	3.44	IWA22	3.51
	IWA22	3.44	IWB6064	3.49
	IWB58271	3.43	IWB67415	3.37
	IWB6064	3.42	IWB58271	3.37
	IWB882	3.38	IWB11366	3.32
	IWB67416	3.33	IWB882	3.31
	IWB73714	3.28	IWB73714	3.30
	IWB11366	3.25	IWB36226	3.29
	IWB36226	3.19	IWB67416	3.26
	IWB35219	3.19	IWB35219	3.15
	IWB72957	3.04	IWB44273	3.14
	IWB43805	3.03	IWB71391	3.05
	IWB44273	3.03	IWB41405	3.04
2017	IWB74	5.15	IWB74	5.25
	IWA6287	4.05	IWA6287	3.86
	IWB60950	3.41	IWB60950	3.53
	IWB67770	3.26	IWB4385	3.24
	IWB6167	3.23	IWB6167	3.21
	IWB52607	3.14	IWB67770	3.19
	IWB4385	3.13	IWB48842	3.06
	IWB67768	3.07	IWB67768	3.01
2018	IWB28017	3.69	IWB28017	3.70
	IWB10765	3.46	IWB10765	3.42
	IWA7225	3.35	IWA674	3.30
	IWA674	3.35	IWA7225	3.13
	IWB7206	3.05	IWB7206	3.07
			IWB66080	3.04

ECMLM = enriched compressed mixed linear model; PC = principal component.