Quantitative Genetics of Disease Resistance in Wheat

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Abstract

Wheat (\textit{Triticum aestivum} L.) is one of the top three global food security crops. Fusarium head blight is one of the major constraints in sustainable wheat production and resistance to the disease is polygenic. This review provides an overview of recent efforts in mapping these genes/loci with the objective to aid marker-assisted selection breeding.

Bread wheat (\textit{Triticum aestivum} L.) is one of the top three stable food crops and accounts for 20\% of calories consumed by people (Brenchley et al., 2012). In terms of production, wheat ranked third with a global production of 729 million tonnes (Fig. 6.1A). Yield, maturity, grain quality and stress (biotic and abiotic) tolerance are the top four agronomic traits. The majority of these are polygenic and controlled by quantitative trait loci (QTLs). Wheat is an allohexaploid (2n = 6x = 42, AABBDD) containing three subgenomes: A-genome from \textit{T. urartu}, B-genome from \textit{Aegilops speltoides} and D-genome from \textit{A. tauschii}.

Fusarium head blight (Fhb) is one of the most devastating diseases of wheat. It is caused by \textit{Fusarium} species complex. The predominant species in North America is \textit{Fusarium graminearum} Shwabe. The pathogen secretes the secondary metabolites deoxynivalenol and nivalenol, and the estrogenic metabolite zearalenone, which accumulate in seeds during infection. These mycotoxins constitute a major concern for human and animal health (Champeil et al., 2004).

Recent developments in next-generation sequencing have accelerated the \textit{de novo} genome sequencing, re-sequencing and discovery of high-density molecular markers, especially single nucleotide polymorphism (SNP) markers. Despite this, the resolution of QTLs remains challenging for crops with complex genomes, such as bread wheat (~17 GB). Several genome complexity reduction-based genotyping approaches have been developed to overcome this bottleneck and used in

Inducible disease resistance in plants against pathogens is bilayered (Dangl and Jones, 2001). The first layer recognizes conserved microbial signatures, such as fungal chitin, using cell surface receptors and is known as microbe-associated molecular pattern-triggered immunity (MTI). MTI, also known as basal resistance, is incredibly effective against non-adapted pathogens, and therefore introgression of resistance from non-host of \textit{F. graminearum} through bridge crossing and embryo rescue could provide durable resistance against this pathogen (lineage-exclusion breeding) (Bhadauria and Banniza, 2014). The second layer detects race-specific pathogen effector molecules using intracellular receptors. A majority of these receptors possess nucleotide binding and leucine rich repeat (LRR) domains. The LRR domain forms a horseshoe shaped structure, which recognizes isolate-specific effectors (avirulence proteins). The concave surface of the structure contains β strands arranged in a crescent shape and surrounded by α-helices (Fig. 6.1B).
the discovery of high-density SNP markers in maize, wheat and barley. These require restriction enzymes to reduce genome representation and multiplexing prior to massively parallel sequencing. This was first demonstrated in stickleback fish and Neurospora crassa through restriction enzyme-associated genomic DNA (RAD) sequencing (Baird et al., 2008). More simplified and efficient versions of RAD, such as one and two restriction enzyme-based genotyping-by-sequencing (GBS) were developed. ApeKI-based GBS identified 25,185 and 24,186 biallelic SNPs in maize and barley recombinant inbred line (RIL) populations, respectively (Elshire et al., 2011). Poland et al. (2012) used PstI-MspI-based GBS to map 34,000 and 20,000 SNPs onto the barley and wheat reference maps, respectively. The GBS approach has the advantage over other approaches, such as microarrays in which cross-hybridization between homologous genes causes false positive signals, which leads to false QTL discovery. Arruda et al. (2016) genotyped a panel of 273 elite winter wheat lines using three sets of restriction enzymes PstI-MspI, PstI-HinP1I and PstI-BfAI for reducing genome complexity and mapped 19,992 high-quality SNP across 21 chromosomes. Using a compressed mixed linear model, the authors identified two SNP markers IWGSC_CSS_3B_scaff_10352272_5482 and IWGSC_CSS_3B_scaff_10699215_3620, which were in the same LD block as Fhb1, a major effect QTL controlling Fusarium head blight located on chromosome 3B. Landraces are an excellent source of genetic diversity for agronomic traits. Cai et al. (2016) phenotyped a recombinant inbred population originated from a cross between Fhb-resistant T. aestivum landrace Haiyanzhong and susceptible cultivar Wheaton for F. graminearum-infected spikelets within spikes. The authors genotyped the population using PstI-MspI-based genotyping approach and identified 6,232 SNPs mapped across 21 chromosomes. Composite interval mapping revealed six QTLs in the landrace linked to Fhb resistance. The SNPs including the one linked to the QTL on chromosome 5A explaining ~16% of the variance in Fhb resistance response across the population was converted to KASP (Competitive allele-specific PCR) marker to pyramid Fhb-resistant QTLs in wheat through marker-assisted selection breeding. So far, over 100 QTLs controlling resistance to F. graminearum have been identified (Buerstmayr et al., 2009) though candidate genes underlying these remain unidentified. Recently, Dhokane et al. (2016) identified six candidate genes underlying the QTL qFhb6B-2 located on the short arm of chromosome 6B through comparative metabolomics and transcriptomics. These include 4-coumarate:CoA ligase (4CL), callose synthase (CS), basic helix–loop–helix (bHLH) transcription factor, glutathione S-transferase (GST), ABC transporter-4 (ABC4) and cinnamyl alcohol dehydrogenase (CAD). 4CL, CS and CAD confer resistance by reinforcing the cell wall (cell wall apposition). ABC4 and GST may have a role in reducing DON accumulation. Functional
characterization of candidate genes through over-expression, silencing or knock-out is required to confirm their role in triggering Fhb-resistance.

References