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**Abstract:** Fusarium head blight (FHB), caused primarily by *Fusarium graminearum*, has rapidly become the most devastating wheat disease worldwide. Host resistance is the most effective and sustainable strategy to combat the disease. To date, more than 50 unique quantitative trait loci (QTLs) have been identified in diverse wheat populations, and *Fhb1* consistently shows the highest level of resistance in different genetic backgrounds. In spite of worldwide efforts to deploy *Fhb1* through wheat breeding, only a few cultivars currently used in commercial production carry *Fhb1*. Recently, *Fhb1* has been cloned and diagnostic markers have been developed, which will facilitate successful deployment of *Fhb1* in wheat breeding. Using marker-assisted backcross, *Fhb1* has been successfully transferred into locally adapted cultivars that carry minor QTLs, and selected *Fhb1* lines in different backgrounds showed high levels of FHB resistance. This approach may provide a quick solution to improvement of FHB resistance in commercial cultivars. In addition, gene editing that knocks down the susceptible allele of *Fhb1* in commercial cultivars may also improve wheat FHB resistance. Most other QTLs identified to date show much smaller effects than *Fhb1*. These QTLs are distributed in many locally adapted cultivars, and thus are important QTLs for FHB resistance improvement. Currently, diagnostic markers are still not available for most of these minor QTLs; therefore, genomic selection may improve selection accuracy for these QTLs to create highly FHB-resistant cultivars.

**Keywords:** *Fhb1*, *Fusarium graminearum*, marker-assisted backcross, QTLs for FHB resistance

**Résumé**: La brûlure de l'épi causée par le fusarium (BEF), engendrée principalement par *Fusarium graminearum*, est rapidement devenue la maladie du blé la plus dévastatrice au monde. La résistance de l'hôte est la stratégie la plus efficace et la plus durable pour la combattre. À ce jour, plus de 50 locus à caractère quantitatif (QTL) ont été répertoriés dans différentes populations de blé, et le gène *Fhb1* affiche invariablement le plus haut taux de résistance des différents fonds génétiques. En dépit des efforts déployés partout dans le monde pour utiliser le gène *Fhb1* en vue d'améliorer le blé, seuls quelques cultivars commerciaux utilisés couramment le portent. Récemment, le gène *Fhb1* a été cloné et des marqueurs de diagnostic ont été développés pour en faciliter l'utilisation efficace afin d'améliorer le blé. En recourant au rétrocroisement assisté par marqueurs, le gène *Fhb1* a été transféré avec succès dans des cultivars locaux adaptés, porteurs de QTL de moindre importance, et des lignées *Fbh1* choisies dans différents fonds ont affiché un taux élevé de résistance à la BEF. Cette approche peut se révéler une solution rapide pour ce qui est d'améliorer la résistance à la BEF des cultivars commerciaux. De plus, la manipulation génétique qui invalide l'allèle réceptif du gène *Fbh1* chez les cultivars commerciaux peut aussi contribuer à améliorer la résistance du blé à la BEF. La majorité des autres QTL identifiés à ce jour affiche des effets beaucoup plus modestes que le gène *Fbh1*. Ces QTL sont répartis dans plusieurs cultivars locaux adaptés et, par conséquent, sont essentiels à l'amélioration de la résistance à la BEF. Actuellement, les marqueurs de diagnostics ne sont pas encore disponibles pour la majorité de ces QTL, par conséquent, la sélection génomique peut contribuer à améliorer la précision du choix à l'égard de ces QTL afin de créer des cultivars hautement résistants à la BEF.

**Mots clés:** *Fusarium graminearum*, *Fhb1*, QTL pour la résistance à la BEF, rétrocroisement assisté par marqueurs

## Introduction

Fusarium head blight (FHB), also called scab, is a damaging disease of cereal crops worldwide (Bai & Shaner, 2004). It is primarily incited by *Fusarium graminearum* Schwabe but other *Fusarium* species have also been reported as pathogens (Buerstmayr et al., 2009). FHB can cause significant reductions not only in grain yield but also in grain quality (Bai & Shaner, 2004). The pathogen produces mycotoxins during infection, in particular deoxynivalenol (DON). DON-contaminated grain is harmful to the health of human beings and livestock after consumption (Jiang et al., 2007a; Ferrigo et al., 2016). In the USA, Canada, Europe and several other countries, governments have imposed strict upper limits on the level of DON allowed in wheat grain and grain products used for food and feed (Buerstmayr et al., 2009; Ferrigo et al., 2016).

Epidemics of FHB were first reported in 1884 in England where it was called 'wheat scab' (Dubin et al., 1997). The disease has spread to other major wheat growing areas worldwide and has been considered a major threat to wheat production in many countries since the early 20th century (Bai & Shaner, 1994; Goswami & Kistler, 2004). In the USA, Arthur (1891) first reported a FHB epidemic in Indiana. FHB epidemics have spread to all major wheat production states in the last several decades (De Wolf et al., 2003; McMullen et al., 2012). The economic losses in wheat and barley caused by FHB were estimated at about $7.67 billion from 1993 to 2001 for nine major FHB epidemic states in the northern Great Plains and the central USA (Illinois, Indiana, Kentucky, Michigan, Missouri, Minnesota, Ohio, South Dakota and North Dakota) (Nganje et al., 2004). In Kansas, FHB losses were valued at $57 million in 2008 alone (McMullen et al., 2012).

FHB epidemics have been reported in most of the wheat production areas worldwide (Dubin et al., 1997; Bai & Shaner, 2004; Buerstmayr et al., 2009; McMullen et al., 2012). Recent changes in climate and cropping systems have made FHB epidemics more frequent and severe, even in the regions where FHB has not been previously reported, such as in Oklahoma, Montana and Idaho in the USA and the Yellow and Huai River Valleys in China. Increased acreages of maize in these regions might contribute to expanded epidemics by providing rich sources of inoculum for initial infection (Gilbert & Haber, 2013). Applying fungicides to control FHB remains challenging because of the short time window for application, potential environmental contamination and increased cost of wheat production (McMullen et al., 2012). Growing resistant cultivars is the most effective and environment-friendly approach to minimizing the damage caused by the disease (Bai & Shaner, 2004; McMullen et al., 2012).

FHB resistance is a quantitative trait that is controlled by multiple quantitative trait loci (QTLs). Among more than 50 unique QTLs reported to date (Liu et al., 2009), *Fhb1* was the first QTL reported from Chinese cultivars 'Sumai3' and 'Ning7840' (Bai et al., 1999; Waldron et al., 1999; Zhou et al., 2003; Cuthbert et al., 2006). Since then, several reviews have been published to summarize these QTLs from different sources (Buerstmayr et al., 2009; Liu et al., 2009; Loffler et al., 2009). The aim of this review is to provide a recent update on wheat FHB resistance with emphasis on several major FHB resistance QTLs and their potential application in FHB control in wheat.

## Wheat responses to FHB infection

FHB infection in wheat spikes is a complicated process. Plant morphological and developmental factors and their growing environments all affect FHB infection. Plant height, spikelet density, awn character, flowering date, degree of floret opening during flowering, and grain filling rate have all been related to FHB resistance (Rudd et al., 2001; Ban, 2003). For example, wheat susceptibility decreases from early flowering to later grain filling, and thus genotypes with quick grain filling may have better resistance than these of slow grain filling genotypes. The *Rht-D1b* semi-dwarf allele in European wheat cultivars may have pleiotropic effects on FHB susceptibility; thus, tall plants may show better resistance than short ones (Holzapfel et al., 2008). Those morphological and developmental traits most likely help plants escape initial infection to provide passive resistance, whereas physiological resistance that produces chemical barriers through biochemical pathways to prohibit pathogen penetration to other tissues and growth in plants after initial infection might play a more important role in wheat defence against FHB (Pritsch et al., 2000; Browne & Brindle, 2007; Li & Yen, 2008; Ding et al., 2011; Zhang et al., 2013).

Based on host response to pathogen infection, Schroeder & Christensen (1963) first observed two types of resistance: resistance to initial pathogen penetration (Type I) and resistance to spread of FHB symptoms within a spike (Type II). Later, Miller et al. (1985) added low mycotoxin accumulation as Type III resistance. These three types of resistance are widely accepted (Bai & Shaner, 1994); however, only type II resistance has been extensively characterized and used in breeding programmes because it is the most stable type of resistance and easiest to be assessed in wheat compared with other types (Bai & Shaner, 2004). Type II resistance is usually evaluated by single floret (spikelet) inoculation that injects inoculum into a central spikelet of a spike in

greenhouse experiments or grain-spawn inoculation in the field (Bai & Shaner, 1994). Percentage of symptomatic spikelets in a spike (PSS) is used to measure the disease severity (Bai & Shaner, 1994). However, accurate assessment of type I resistance is more difficult than type II resistance. Usually plants are inoculated by spraying conidiospores over spikes or grain-spawn inoculation to provide natural infection. Infection rate is measured by percentage of initially infected spikelets in a spike or FHB incidence (percentage of initially infected spike in a plot) for type I resistance (Bai & Shaner, 1994). Many factors may affect type I resistance, including inoculation methods (spraying vs. grain-spawn inoculation) and time and inoculum concentration if spraying.

Degrees of FHB spread within an infected spike vary with levels of host resistance. The pathogen can successfully establish initial infection in any wheat genotype when it is point-inoculated by placing spores into one floret of a spike (Bai & Shaner, 1994). In a highly resistant genotype, FHB infection is usually confined in the inoculated spikelet or floret and does not spread to neighbouring spikelets, whereas in a highly susceptible genotype, FHB symptoms spread to uninoculated neighbouring spikelets until the entire spike is bleached (*Fig. 1a*), designated type II resistance (Schroeder & Christensen, 1963). However, in moderately resistant to susceptible genotypes, FHB symptoms start spreading upward and downward to neighbouring spikelets about 4–5 days after needle inoculation. The number of spikelets to which FHB can spread varies with host genotypes and environmental conditions where plants are grown. In moderately resistant genotypes, many spikelets in an inoculated spike remain uninfected, whereas in highly susceptible genotypes, the entire inoculated spike is bleached within 7–10 days after inoculation (Ribichich et al., 2000). Therefore, FHB severity score (PSS) evaluates type II resistance under greenhouse conditions. In the field, initial infection may occur in multiple spikelets of a single spike, and the genotypes with fewer initially infected spikelets in a spike have higher type I resistance (*Fig. 1b*). Under field conditions, final PSS ratings usually reflect both type I and type II resistance of a genotype (*Fig. 1c*) (Burt et al., 2015).

Type III resistance is important to grain end-use quality (Rudd et al., 2001), but it is highly correlated with type I and type II resistance (Paul et al., 2006); therefore, PSS can be used to predict DON contents in harvested wheat grains (Bai et al., 2001). Besides, accuracy of type III resistance assessment is significantly affected by threshing method used. In most cases, *Fusarium* damaged kernels (FDK) that contain a high level of DON can be blown out during threshing (Mesterhazy et al., 1999; Bai & Shaner, 2004); thus the

DON measurement may not reflect actual content of DON produced in the lines tested. In addition, low FDK and FHB tolerance were described as two additional types of resistance (Mesterhazy, 1995). However, FDK is highly correlated with FHB severity, and may not be an independent type of resistance (Bai et al., 2001). For FHB tolerance, an evaluation method is not available; therefore, it has not been pursued further.

Biochemical mechanisms for FHB resistance have been frequently reported, but the conclusions remain equivocal. Resistant wheat plants may either produce physical barriers (such as a thickened cell wall) or accumulate toxic phenolic compounds and triticens to prevent or delay the mycelial growth after initial infection (Ribichich et al., 2000; Zhang et al., 2013). *Fusarium graminearum* may induce defence responsive genes that encode defence-related proteins, including PR-1, PR-2 (β-1,3-glucanase), PR-3 (chitinase), PR-4 and PR-5 (thaumatin-like protein) during early infection in wheat spikes (Pritsch et al., 2000). Some of the proteins are differentially expressed between resistant and susceptible genotypes (Zhang et al., 2013), but other studies observed that *Fusarium* inoculation induced biosynthesis of more jasmonic acid (JA) and higher expression of lipoxygenase (LOX2) and chalcone synthase in resistant wheat plants than in susceptible plants (Li & Yen, 2008; Ding et al., 2011). Ethylene (ET) production was also higher in resistant plants than in susceptible plants, which can cause plant organ senescence, cell wall degradation and finally cell death (Li & Yen, 2008). In addition, many other biochemical compounds, including choline, betaine, and amino acids glutamine, glutamate alanine, trans-aconitate and sucrose have also been associated with fungal hyphal growth (Browne & Brindle, 2007).

## FHB resistance sources in wheat

Germplasm sources with various levels of resistance have been reported worldwide, although those with immunity to FHB have not been found (He et al., 2013). Among these FHB resistant germplasm lines, only a few showed a high level of FHB resistance, which are mostly found in China and Japan (Bai & Shaner, 2004; Yu et al., 2008a). In the 1980s, the Chinese Wheat Scab Cooperative Project identified 1765 accessions with various levels of resistance after screening 34 571 wheat landraces and other germplasm materials (Bai & Shaner, 1994; He et al., 2013). Among them, 'Sumai3' and its derivatives are improved cultivars with high FHB resistance and have received extensive attention by breeders and geneticists worldwide (Bai & Shaner, 1994; Rudd et al., 2001; Liu

**Fig. 1** (Colour online) **a**, Wheat type II resistance. Wheat plant with a high level of type II resistance (right head) usually confines FHB infection to the inoculated spikelet or floret and does not spread to neighbouring spikelets; whereas in a highly susceptible genotype (left head), FHB symptoms spread from the inoculated spikelet to uninoculated neighbouring spikelets until the entire spike is bleached. Both genotypes were inoculated by injecting fusarium spores into a central spikelet of a spike under greenhouse conditions. **b**, Wheat type I resistance. Under field conditions, the FHB nursery was inoculated by scattering Fusarium infected corn kernels to induce natural infection. The plants with type I resistance show only one or a few infected spikelets (right head), whereas the type I susceptible plant shows multiple independently infected spikelets in a spike (left head). **c**, Comparison between type I and type II resistance under field conditions. Plant with good type II resistance has lower final disease severity even when multiple florets are infected under heavy disease pressure (left head); whereas plant without type II resistance has entire spike bleached even if only a single spikelet was initially infected (right head).

et al., 2009). A major QTL for FHB resistance on chromosome arm 3BS was first reported from 'Sumai3' and 'Ning7840' (Bai et al., 1999; Waldron et al., 1999). Later, the QTL, designated as *Fhb1* (Cuthbert et al., 2006), was reported in landraces 'Wangshuibai', 'Baishanyuehuang', 'Huangcandou', 'Huangfangzhu' from China and 'Nyu Bai' from Japan (Ban, 2001; Bai & Shaner, 2004; Jia et al., 2006; Lin et al., 2006; Yu et al., 2008b). However, direct use of these landraces or cultivars as resistant parents in conventional breeding has not been successful due to their poor agronomic traits.

Many locally adapted cultivars may also carry some minor QTLs for FHB resistance (Zhang et al., 2012a, 2012b). These minor QTLs individually show much smaller effects than *Fhb1* from 'Sumai3', but they may show additive effects when combined (Cai, 2016; Li et al., 2016). These cultivars have good adaptation and desirable agronomic traits, and thus can be easily included in elite breeding lines without penalty of yield and other agronomic traits. The examples of these cultivars include 'Chokwang' from Korea (Yang et al., 2005a), 'Frontana' (Mardi et al., 2006) and 'Encruzilhada' from Brazil (Mesterhazy, 1995; Ban, 2001), 'Ernie', 'Freedom' and 'Roane' from the USA

(Rudd et al., 2001; Liu et al., 2005, 2007; Jin et al., 2013), and 'Arina', 'Renan' and 'Fundulea 201R' from Europe (Gervais et al., 2003; Paillard et al., 2004; Somers et al., 2004; Steiner et al., 2004). These sources may carry some different QTLs from those present in Asian sources (Jin et al., 2013) and can be used to pyramid FHB resistance QTLs from Asian sources.

FHB resistance was also reported in alien species, including tetraploid wheat species, such as *Ae. ventricosa, Ae. speltoides, Thinopyrum ponticum, Th. elongatum, Th. intermedium, Dasypyrum villosum, Leymous racemosus* and *Elymus tsukushiensis* (Oliver et al., 2005; Cai et al., 2008; Qi et al., 2008; Cainong et al., 2015). Various cytogenetic approaches have been used to transfer resistance genes from these alien sources to generate addition, substitution, translocation or recombinant lines by backcrossing them with adapted common wheat. Three alien FHB resistant fragments, designated as *Fhb3, Fhb6* and *Fhb7*, have been successfully transferred into wheat from alien species *Leymus racemosus* (Qi et al., 2008), 1E$^{ts}$#1S of *Elymus tsukushiensis* (Cainong et al., 2015), and *Thinopyrum ponticum* (Guo et al., 2015), respectively, and selected lines were reported to show a high level of resistance in wheat backgrounds. Among

them, *Fhb3* has been used to pyramid with *Fhb1* by marker-assisted backcross. Evaluation of FHB resistance of BC₂F₄ progenies indicated that *Fhb3* did not reduce FHB severity in both greenhouse and field experiments, either alone or together with *Fhb1* (Fatima, 2016). Therefore, the effects of these alien genes on FHB resistance need to be evaluated in different backgrounds before they can be deployed in breeding.

## QTLs for FHB resistance

FHB resistance is a quantitative trait and controlled by several QTLs (Bai & Shaner, 2004). To date, more than 100 QTLs for different types of resistance have been reported from different studies (Buerstmayr et al., 2009). Further comparisons of the chromosome locations of these QTLs from different studies narrowed the list down to about 50 QTLs with unique chromosome locations (Liu et al., 2009). These unique QTLs have been reported in at least two populations, in particular those on chromosomes 3A, 3B, 4B, 5A and 6B that were reported in several populations of different geographic origins (Buerstmayr et al., 2009; Liu et al., 2009). Seven QTLs have been formally assigned with a gene name. *Fhb1* on chromosome arm 3BS from 'Sumai3' was named first (Cuthbert et al., 2006). *Fhb2* on chromosome 6B from 'Sumai3' (Cuthbert et al., 2007), *Fhb4* (Xue et al., 2010) and *Fhb5* (Xue et al., 2011) on chromosomes 4B and 5A, respectively, from 'Wangshuibai'. As mentioned above, *Fhb3* on chromosome 7AS, *Fhb6* on chromosome 1A and *Fhb7* on chromsome 7D were all from alien species.

Among the seven named QTLs, *Fhb1* shows the largest effect on FHB type II resistance among all QTLs identified to date and is the only QTL that shows stable type II resistance across multiple backgrounds (Bai et al., 1999; Waldron et al., 1999; Zhou et al., 2002; Buerstmayr et al., 2003; Bai & Shaner, 2004; Cuthbert et al., 2006; Yu et al., 2008b; Schweiger et al., 2016). This QTL has also been reported in many wheat landraces that are not related to 'Sumai3' (Somers et al., 2003; Cuthbert et al., 2006; Lin et al., 2006; Yu et al., 2008b; Li et al., 2012; Zhang et al., 2012b; Cai & Bai, 2014). We surveyed a worldwide collection of wheat landraces and cultivars and only found *Fhb1* resistance alelle in landraces from east Asia (southern China and Jappan), not from other parts of the world; therefore, *Fhb1* most likely originated from southern China.

Due to its major and stable effect on FHB type II resistance, *Fhb1* has been extensively studied and utilized in wheat breeding programmes. It was first prioritized for map-based cloning (Rawat et al., 2016; Schweiger et al., 2016). Previous fine mapping identified Xumn10 as a

tightly linked marker for marker-assisted selection (MAS) of *Fhb1* (Liu et al., 2008). This marker has been sucessfully used as the near diagnostic marker of *Fhb1* in US breeding programmes for about a decade. However, Xumn10 is not diagnostic in Chinese germplasm because many susceptible Chinese landraces and cultivars carry the 'Sumai3' allele at the marker locus. As more Chinese germplasm sources are used in US wheat breeding programmes, Xumn10 becomes less diagnostic for *Fhb1* in these programmes. SNP markers such as SNP319 and SNP8 (Bernardo et al., 2011) have been developed as alternative markers for *Fhb1*, but false positive alleles were also reported for these markers. More recently, significant progress has been made in *Fhb1* cloning. Schweiger et al. (2016) sequenced a 1 Mb contig harbouring *Fhb1* in 'CM-8203', a 'Sumai3' derivative, and found that *Fhb1* is in a suppressed recombination region of 703 kb. Rawat et al. (2016) further characterized one of the candidates in the region, a chimeric lectin with agglutinin domains and a pore-forming toxin-like domain (*PFT*), for *Fhb1* and reported that the genotype with the resistance allele of *Fhb1* carried the functional *PFT*, whereas the genotypes with susceptible allele had no *PFT* or a malfunctioned *PFT* in a set of wheat materials they used. However, when a large collection of wheat samples, especially Chinese landraces, was analysed for *PFT*, many susceptible genotypes were found to carry functional *PFT*, suggesting *PFT* may not be the determinant of *Fhb1*. We conducted map-based cloning and EcoTILLING of candidate genes in the *Fhb1* region and found a hypothetical protein, designated *TaHRC*, that is responsible for *Fhb1* resistance (Su et al., 2017). We found that the wild type allele of the gene confers FHB susceptibility, and a large deletion mutation in the open reading frame of the gene caused a loss-of-function of the gene and thus results in FHB resistance. We developed diagnostic markers to capture the deletion mutation for genotyping using either gel-based or Kompetitive allele specific PCR (KASP) protocols (Su et al., 2016). These markers are highly diagnostic for *Fhb1* when they were analysed in a large worldwide wheat collection; therefore, they are ideal markers for MAS of *Fhb1* in breeding.

For the other three named genes from wheat, *Fhb2* on chromosome 6B explained a wide range of the phenotypic variation (4.4–23%) for mainly type II FHB resistance (Waldron et al., 1999; Anderson et al., 2001; Yang et al., 2005b; Semagn et al., 2007; Bonin & Kolb, 2009; Li et al., 2011, 2012), but type I and type III resistance was also reported (Szabó-Hevér et al., 2012; Ágnes et al., 2014). *Fhb2* is located in the interval between Xgwm508 and Xbarc79 with Xgwm644 as a common marker for most populations (Liu et al., 2009). It is

present not only in Chinese germplasm, but also in germplasm sources from different continents, including 'Arina' and 'Apache' from Europe, 'Patton' from the USA, and 'DH181' from Canada.

*Fhb4* on chromosome 4B showed type I resistance in 'Wangshuibai' (Jia et al., 2006; Lin et al., 2006; Xue et al., 2010), and type II resistance in 'Ernie' (Liu et al., 2005, 2007), 'Chokwang' (Yang et al., 2005a), and 'Wuhan1' (Somers et al., 2003) etc., and explained 4.7% (Yang et al., 2005a) to 17.5% (Lin et al., 2006) of the phenotypic variation. It was located between Xbarc20 and Xwmc349 (Liu et al., 2009). *Fhb4* is not only present in Chinese germplasm, but also in several US germplasm sources, including 'Becker' and 'IL-95-1653′' (Liu et al., 2009).

*Fhb5* has been associated with type I resistance (Lin et al., 2006), but it was also associated with type II resistance in several populations (Liu et al., 2009). *Fhb5* is present in more than 10 populations including the resistant landraces/cultivars from China ('Wangshuibai'), Japan ('Nyu Bai'), Europe ('F201R') and America ('Frontana' and 'Ernie'). It shows much smaller effects than *Fhb1* on average but can explain up to 30% of the phenotypic variation in some studies (Buerstmayr et al., 2011, 2012). Based on the consensus maps, this QTL is located in a ~ 10 cM interval between Xgwm293 and Xbarc180 on chromosome 5A (Buerstmayr et al., 2009; Liu et al., 2009). Chu et al. (2011) mapped *Qfhb.rwg-5A.1* in PI277012 at the same chromosome position as *Fhb5*, showing a major effect on type II resistance. They also found another QTL, designated *Qfhb.rwg-5A.2*, on chromosome arm 5AL that showed an even larger effect on type II resistance than *Qfhb.rwg-5A.1*. The QTL peak for *Qfhb.rwg-5A.2* is at Xcfd39, flanked by Xwmc470 and Xbarc48, and explained up to 32% of phenotypic variation for type II resistance. This QTL is in the same interval as the Q gene, but recombination was identified between Q and *Qfhb.rwg-5A.2*. In previous studies, QTLs in the vicinity of *Qfhb.rwg-5A.2* have also been reported from several European cultivars, including 'Renan', 'Apache', 'Pirat' and 'Arina', but with a much smaller effect than in *Qfhb.rwg-5A.2* (Gervais et al., 2003; Holzapfel et al., 2008; Buerstmayr et al., 2009). Currently, we are pyramiding *Qfhb.rwg-5A.2* with *Fhb1* to achieve highly FHB resistant germplasm lines for breeding or cultivars for production.

Besides seven named QTLs, QTL on 2DL also shows a major effect on type II resistance and was reported in several cultivars, including 'Wuhan1' (Somers et al., 2003), 'Soru#1' (He et al., 2016), 'Kenyon' (McCartney et al., 2016), 'Sumai3' (Suzuki et al., 2012; Yang et al., 2005b), and 'CJ9306' (Jiang et al., 2007a, 2007b). This QTL is located between Xgwm539 and Xwmc245 with Xgwm539 as a common marker among these mapping studies (Buerstmayr et al., 2009; Liu et al., 2009). It confers both type II and type III resistance and showed an additive effect with *Fhb1* in different backgrounds (Clark et al., 2016; Jiang et al., 2007a; 2007b).

QTLs on 3AS have been detected in germplasm from several countries including 'F201R' from Europe (Shen et al., 2003), 'Huapei57-2' (Bourdoncle & Ohm, 2003), 'Wangshuibai' (Yu et al., 2008b) and several other landraces (Zhang et al., 2012b; Cai & Bai, 2014; Cai, 2016) from China, 'Heyne' from the USA (Zhang et al., 2012a), 'DH181R' (Yang et al., 2005b) and 'Frontana' from Brazil (Steiner et al., 2004). A QTL near centromere region of 3BS (3BSc) has been reported in 'Baishanyuehuang' (Zhang et al., 2012b), 'Wangshuibai' (Zhou et al., 2004) from China, 'Nyu Bai' from Japan (Somers et al., 2003), 'DH181R' (Yang et al., 2005b), and 'Ernie' from the USA (Liu et al., 2007). These QTLs with either a major effect or a minor effect detected in multiple genetic backgrounds can be good sources of resistance for gene pyramiding.

## Breeding for FHB resistance

Many QTLs for FHB resistance show additive effects (Bai et al., 2001; Jiang et al., 2007a, 2007b), and pyramiding major QTLs from different sources to locally adapted cultivars may achieve a high level of resistance (Rudd et al., 2001). *Fhb1* is the QTL showing the largest effect identified to date and usually shows additive effects when it is combined with other QTLs. Therefore, transferring *Fhb1* into locally adapted moderately susceptible or moderately resistant cultivars may significantly improve FHB resistance in commercial wheat cultivars (Kolb et al., 2001). Direct use of *Fhb1* from Chinese sources in US hard winter wheat breeding programmes has not been successful because they usually have many undesired agronomic traits. To remove these poor agronomic traits and reduce the linkage drag in *Fhb1* carrying Chinese landraces, marker-assisted backcross was used to transfer *Fhb1* into locally adapted backgrounds. Recently, the US Department of Agriculture Central Small Grain Genotyping Laboratory has transferred *Fhb1* to 16 locally adapted hard winter wheat cultivars or breeding lines from five hard winter wheat states using marker-assisted backcross. *Fhb1* usually reduces FHB severity by 20–50% in different genetic backgrounds compared with their recurrent parents. Significant variations in FHB severity were observed between *Fhb1* lines within a population and across populations. The differences among *Fhb1* lines might be due to segregation of other

minor QTLs in *Fhb1* lines. However, the minor QTLs in the backgrounds usually are unknown and markers for these QTLs are not available; therefore, phenotypic selection of *Fhb1* lines is necessary for selecting highly resistant lines. Most of the selected *Fhb1* lines showed moderate to high FHB resistance. These lines have been used in the hard winter wheat breeding programmes as *Fhb1* donor parents (Jin et al., 2013, 2014). One of the selected lines, 'OverlandFHB-10', has been promoted to the 2017 Northern Regional Performance Nursery for yield trails. Using marker-assisted backcrossing, *Fhb1* has also been transferred into a durum wheat and shows a large major effect on type II resistance (Prat et al., 2017), which is an important achievement for durum breeding because QTL with high FHB resistance has not been found in durum to date. These selected *Fhb1* lines with different genetic backgrounds are useful resources for breeders to deploy *Fhb1* in wheat and durum.

Significant progress in using *Fhb1* resistance has been made in US hard spring wheat (HSW) and soft winter wheat breeding programmes. Several hard spring wheat cultivars with *Fhb1*, including 'Bacup' and 'Sabin' from Minnesota, and 'Alsen', 'ND2710' and 'Glenn' from North Dakota, have been released for production (Mergoum et al., 2007; Anderson, 2012a; Anderson et al., 2012b). In this region, moderate FHB-resistant cultivars occupied about 50% of the total spring wheat acreage (Anderson, 2012a) and these *Fhb1*-derived cultivars have played a significant role in reducing FHB damage in US hard spring wheat. For soft winter wheat in the USA, some cultivars harbouring *Fhb1* such as Pioneer Brands '25R18', '25R42' and '25R51' have been released for commercial production (Brown-Guedira et al., 2008).

Besides *Fhb1* from China, many cultivars from different countries may contain native resistance QTLs. Several moderately FHB resistant cultivars without *Fhb1* have been released in US soft winter wheat (SWW) regions, including 'Truman', 'Massy', 'Ernie', 'Roane' and 'Freedom' (Rudd et al., 2001; Sneller et al., 2012; Jin et al., 2013; Liu et al., 2013). 'Everest', 'Overland', 'Lyman', 'Heyne' and 'Hondo' are hard winter wheat (HWW) that carry native resistance QTLs (Bockus et al., 2009; Zhang et al., 2012a; Jin et al., 2013). To improve FHB resistance in these wheat cultivars, they can serve as recurrent parents for pyramiding major QTLs from Asian sources. Also, since these cultivars may carry different QTLs, combining these QTLs together may provide highly resistant cultivars by selecting transgressive segregants. One example is a HWW cultivar 'Everest' (HBK1064-3/ Betty 'S'//VBF0589-1/IL89-6483) from Kansas. It has moderate FHB resistance under field conditions, especially

type I resistance, but none of its parents (HBK1064-3/ Betty 'S'//VBF0589-1/IL89-6483) have any noticeable resistance. Maintaining FHB screening nursery with proper, but consistent, disease pressure across years may be critical for successful selection of these types of resistance. To improve selection efficiency, genomic selection may be a promising strategy for combining these minor QTLs from adapted cultivars in breeding programmes and provide moderate to high prediction accuracies for FHB resistance (Arruda et al., 2015).

Creating transgenic wheat expressing resistance genes provides another approach to improve FHB resistance. For example, Li et al. (2015, 2017) transferred a barley UDP-glucosyltransferase into wheat. The transgenic plants showed significantly higher type II resistance and lower toxin content than non-transformed controls. In another study, transgenic plants overexpressing a β-1,3-glucanase showed enhanced type II, type III and type IV resistance (Mackintosh et al. 2007). Although the transgenic approach has been used to enhance wheat resistance for decades, transgenes showing consistent resistance against FHB are still not available for breeding. Recently, identification of *TaHRC* (Su et al., 2017) as *Fhb1* opens another avenue for using gene editing to improve FHB resistance. *TaHRC* is a susceptibility gene for FHB and is a conserved gene present not only in wheat, but also in durum, barley and other cereal crops. Using gene editing, we can modify the gene sequence of *TaHRC* in susceptible wheat to improve FHB resistance (Su et al., 2017). Therefore, gene editing shows promise to improve wheat FHB resistance by knockdown of susceptible genes in commercial cultivars.

## Prospectives for future FHB research

Use of wheat resistance to FHB is the most effective and sustainable strategy to manage FHB. To date, many QTLs for different types of resistance have been identified in wheat, but only *Fhb1* shows high and stable FHB resistance in different genetic backgrounds. Recently, *Fhb1* has been cloned and diagnostic markers have been developed for marker-assisted breeding. Direct transfer of *Fhb1* to adapted wheat backgrounds using marker-assisted backcrossing can provide a quick solution to improve FHB resistance of commercial wheat cultivars. Resistance genes from alien species may be useful, but experience suggests it may be ineffective in some wheat backgrounds. For the minor QTLs in adapted cultivars, diagnostic markers are not available for marker-assisted breeding. Phenotypic selection and genomic selection may be able to combine several QTLs from different sources to create highly resistant cultivars. In addition,

gene editing may provide another alternative to improve wheat resistance.

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