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REVIEW ARTICLE

Fusarium head blight in wheat: contemporary status and molecular approaches

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Abstract
Fusarium head blight (FHB) disease that occurs in wheat is caused by *Fusarium graminearum* and is a major risk to wheat yield. Although several research efforts focusing on FHB have been conducted in the past several decades, conditions have become more critical due to the increase in its virulent forms. In such a scenario, conferring complete resistance in plants seems to be difficult for handling this issue. The phenotyping for FHB and finding a solution for it at the genetic level comprises a long-term process as FHB infection is largely affected by environmental conditions. Modern molecular strategies have played a crucial role in revealing the host–pathogen interaction in FHB. The integration of molecular biology-based methods such as genome-wide association studies and marker-based genomic selection has provided potential cultivars for breeding programs. In this review, we aim at outlining the contemporary status of the studies conducted on FHB in wheat. The influence of FHB in wheat on animals and human health is also discussed. In addition, a summary of the advancement in the molecular technologies for identifying and developing the FHB-resistant wheat genetic resources is provided. It also suggests the future measures that are required to reduce the world's vulnerability to FHB which was one of the main goals of the US Wheat and Barley Scab Initiative.

Keywords Association mapping · Disease resistance · *Fusarium graminearum* · Fusarium head blight · Molecular breeding · Proteomics · QTL · Wheat

Introduction

Wheat with more than 700 million tons of annual production in year 2014/2015 is being used as a staple food crop by 35% of the world's population (FAOSTAT). With an expected population of 9 billion by 2050, wheat production is envisaged to increase while simultaneously meeting the projected food demand on a global basis. There are various threats to wheat production and among these, severe plant disease epidemics and climate change are considered

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as one of the most dangerous threats for wheat production (Friesen et al. 2008; Gurung et al. 2012). Plant diseases such as Fusarium head blight (FHB) serve as an obstacle in the production and value of significant food stuffs. Under the favorable conditions for disease development, a significant reduction in crop yield has been observed in different parts of the world. FHB, which is also known as scab, head scab, and ear blight, caused by FHB species complex dominantly *Fusarium graminearum* is a fungal disease affecting grain crops such as wheat, maize, and barley; and causes severe reduction in the quality and quantity of grain yield (Lilleboe and Roth 2011; Salgado et al. 2015; McMullen et al. 2012; Dweba et al. 2017). The FHB species complex includes more than 16 species including *F. chlamydosporum, F. boothii, F. scirpi, F. arthrosporioides, F. poae, F. avenaceum, F. culmorum, F. graminearum, F. verticillioides, F. asiaticum,* and *F. cortaderiae* (O'Donnell et al. 2004; van der Lee et al. 2015; Dweba et al. 2017).

According to the International Maize and Wheat Improvement Center (CIMMYT), FHB has been considered as one of the most destructive diseases impacting the production of wheat globally (Bottalico and Perrone 2002; McMullen et al. 1997; Yi et al. 2018; Dubin et al. 1997). Apart from decreasing the yield, it also affects the animal and human health. Thus, this review highlights the spread of FHB around the world and its effect on wheat production and human health. It also summarizes the molecular advancements that aid in developing FHB resistance in wheat genotypes. An outline of the existing research documentation on FHB is useful as it may facilitate future FHB control programs and also support the identification of novel FHB-resistant sources.

Prevalence of FHB

Considering several economic and scientific aspects, currently, ascomycetes *F. graminearum,* which grows in temperate climate conditions, is graded among the four crucial plant fungal pathogens (Dean et al. 2012) and it causes serious damage to wheat (Parry et al. 1995; Xu and Nicholson 2009). In 1884, FHB caused by *F. graminearum* was first identified in England (Goswami and Kistler 2004); and in subsequent years, it developed into a major risk factor for barley and wheat production (Del Ponte et al. 2017). Though there have been so many reasons behind severe FHB epidemics in Canada and the United States since 1993, the decreased level of resistance in cultivars, changes in weather conditions and modifications in crop management strategies are basically responsible for the issue (Dill-Macky and Jones 1997; McMullen et al. 1997). Since its evolution, different kinds of FHB have been destroying several wheat-growing regions of North America (Gilbert and Tekauz 2000; Ward et al. 2008). However, in recent years, FHB has become more prevalent in Asia, Europe, and South America, thereby resulting in increased economic loss (Parry et al. 1995; Bai and Shaner 2004; Zhu et al. 2019; O'Donnell et al. 2004; van der Lee et al. 2015).

The FHB epidemic has been reported to lead to a 10–70% of production loss during the epidemic years (Zhang et al. 2011). In China, though a 5–10% of yield loss is common due to FHB, it may reach up to 100% in epidemic years and around 7 million hectares of wheat fields would be affected (Cheng et al. 2012). From 1993 to 2001, a loss of 7.6 billion US dollars has been reported due to the FHB epidemic in the United States (Windels 2000; McMullen et al. 2012). After the FHB outbreak in United States and Canada, which happened from 1991 to 1996, a number of publications reported the spreading of the disease in other regions of the world, including the United States, Europe, and China (Elias et al. 2005; Oliver et al. 2007; McMullen et al. 2012; Giroux et al. 2016). The continuously changing environment and the increasing threat of global warming have led to an increase in the FHB epidemic (Shah et al. 2014). The variation in the environmental temperature and humidity in the atmosphere are the major factors affecting the spread of FHB infection (Rossi et al. 2001; De Wolf et al. 2003; Xu et al. 2007). However, this effect may vary according to the pathogen causing FHB. Different isolates can behave differently with regard to their aggressiveness in lower or higher temperatures. Several studies have reported an increase in mycotoxin production at higher temperatures at the moment of initial infection (Xu et al. 2007). Moreover, not only a separate occurrence of temperature and humidity stress enhances the FHB infection but also a combined occurrence of both the forms of stress increases its occurrence (Martínez et al. 2012). In addition, as FHB can be caused by numerous pathogens, competitive interaction between these pathogens may also affect their response. Thus, it is required to comprehend the influence of environmental factors on individual pathogens. Effective models should be developed and employed to estimate the actual extent of the FHB infection under individual and combined environmental stress conditions. This will direct us toward the dissemination of several pathogens related to the FHB species complex in different climatic conditions (Del Ponte et al. 2005; Martínez et al. 2012; Scala et al. 2016; Dweba et al. 2017).

Infection related to FHB and life cycle of *F. graminearum* in wheat

The FHB infection prevails when humid and warm conditions persist for a long duration. Plants are more sensitive to FHB at the flowering stage (Walter et al. 2010). The infection cycle of *F. graminearum* in wheat starts with the settling of airborne spores on wheat spikelets, which, after

germination, enter the plants via degenerated anther tissues or minute natural openings under the lemma. Further, the growth of fungus occurs between the cells and it passes from the xylem and the pith and colonizes with the tissue followed by necrosis (Trail 2009). At the cellular level, the cell wall, mitochondria, chloroplasts, and membranes are also damaged (Miller and Ewen 1997). This leads to the water soaking in chlorenchyma tissues leading to the production of shriveled kernels and premature bleaching that affects photosynthesis (Bai and Shaner 1994). After the infection, genes for deoxynivalenol (DON) biosynthesis are expressed by the fungus and this facilitates the spreading of the fungus from spikelet to rachis (Jansen et al. 2005). There is an association between the DON biosynthesis and the colonization of developing tissues leading to shriveled grains (Jansen et al. 2005).

The life cycle of *F. graminearum* consists of both sexual and asexual stages and haploid mycelial structures are formed in both stages (Ma et al. 2013; Goswami and Kistler 2004) (Fig. 1). Fusarium species possess three forms of mitotic (asexual) spores, chlamydospores from hyphae and macroconidia, macroconidia from sporodochium, and microconidia from conidiophores. The anamorph (asexual stage) and teleomorph (sexual stage) of this pathogen are *F. graminearum* and *Gibberella zeae, respectively*. In *F. graminearum,* asexual spores are called macroconidia, whereas sexual spores are called ascospores. Generally, *F. graminearum* is haploid during its life cycle. It is a hemibiotroph which spends its asexual cycle on infested crop debris and its sexual cycle on living wheat tissues (Gunupuru et al. 2017). Macroconidia formed on hyphae called sporodochia develop on infected crop residues under humid conditions and are largely responsible for short-distance dispersion (Deacon 2005). However, their sexual lifecycle is triggered by warm, humid, and wet conditions. As an ascomycota, its sexual life cycle consists of a prolonged dikaryotic phase that is homothallic and the two nuclei are genetically similar. These dikaryotic cells produce coiled cells, leading to the formation of ascus-filled perithecia. These asci consist of ascospores that are released outside via the mouth of the perithecium (Trail et al. 2002; Hallen and Trail 2008); these ascospores are the main inoculum of the infection (Trail 2009; Dweba et al. 2017).

Genetic diversity of the pathogen and its adaptive response to its surroundings is usually enhanced due to sexual reproduction as it allows genetic exchange in *F.*

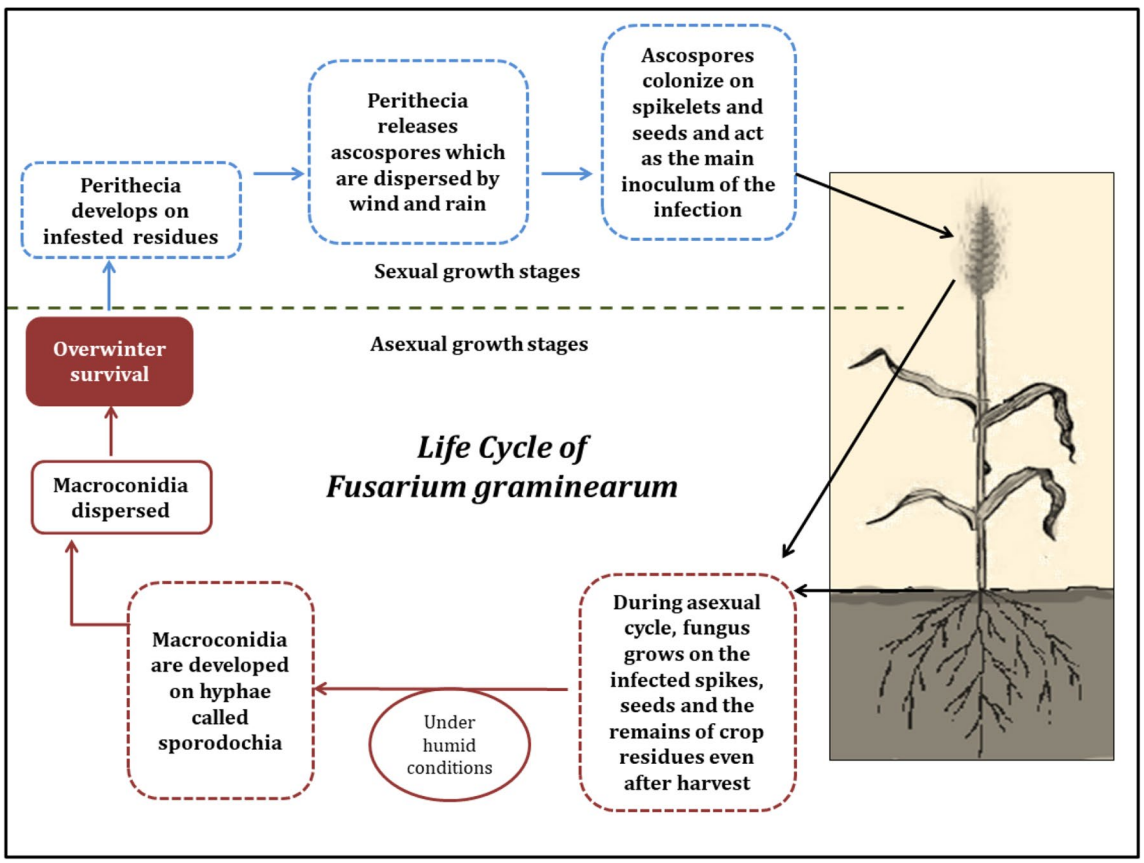

Fig. 1 Life cycle of *Fusarium graminearum* (red and blue outlined stages represent asexual and sexual growth stages, respectively)

graminearum populations via recombination (Lee et al. 2009; Cuomo et al. 2007). The distinct regions in the *F. graminearum* genome accompanied with a high genetic diversity of infection-related genes may increase the adaptability of the fungus toward diverse environmental conditions via genetic exchange during sexual reproduction (Carter et al. 2002; Cuomo et al. 2007). Such facts will aid researchers in implementing to arrange the managing strategies and at minimizing the overwintering of this notorious pathogen.

Mycotoxins-dangerous products of Fusarium

One of the major concerns associated with FHB is the release of the mycotoxins by the pathogens and consequently, their effect on wheat grains (da Rocha et al. 2014; Ponts 2015). Mycotoxins affect humans by raising teratogenic and immunological complications, and they inhibit weight gain in animals (Venkataramana et al. 2018). The consumption of FHB-infected food causes several health issues such as headache, food poisoning, abdominal pain, and diarrhea in humans as well as emaciation in animals (Wegulo 2012). The negative impacts of FHB-infected food on animals and human beings have been reported quite often (Darwish et al. 2014). A global assessment revealed 36%, 54% and 55% mycotoxins of Fusarium ZEN, fumonisins, and DON, respectively, infected food products, during 2004–2011, though most of the samples were according to the European Commission Regulation and Recommendations [(Perincherry et al. 2019); please have a look at Table A3 of Antonissen et al. (2014) for maximum tolerable concentrations of mycotoxins]. Zearalenone and trichothecenes exert economically negative impacts, resulting in agro-ecological zones in the world (Zain et al. 2012). The effect of FHB mycotoxins on livestock and human health can be monitored using chemotyping. Among the fungal toxins, DON, 4-acetyl nivalenol (4ANIV), 3-acetyl and 15-acetyl DON (3ADON and 15ADON), nivalenol (NIV), and Type B trichothecenes pose a significant source of damage to cereals. Among all, 15-acetyl DON is one of the most widespread and dominant FHB chemotypes (Boutigny et al. 2012). The intake of ZEN may cause health issues in animals, such as hindered conception and abortion as well as and hyper-estrogenic syndrome in pigs (Reddy et al. 2010). Trichothecenes can be assimilated into the body via the skin and they inhibit the protein synthesis (Zinedine and Mañes 2009). DON is immunosuppressive and may cause gastrointestinal stress, kidney issues, blood in stools, and throat and facial irritation (Reddy et al. 2010).

There are some groups that have cataloged FHB species complex distribution and the composition of their chemotypes from several regions of the world (Przemieniecki et al. 2014). The combined study of phylogenetic and chemophytic properties for distribution trials has provided an improved apprehension of the epidemiology of FHB. This would probably serve as a useful guide for formulating the disease management strategies. As a single strategy may not be sufficient to manage the FHB, combined implications of different control strategies such as chemical, cultural, and biological strategies, as well as the development of resistant host plant species, may support in coping with the issue.

The estimation of the environmental factors upholding the FHB pathotypes is important for their control, especially in developing and under-developed countries (Xu et al. 2008; Shin et al. 2014). FHB risk is increased when the relative humidity level in the atmosphere is about 70% or higher. The weather prediction and its precise updates should be provided to farmers to minimize the risk of disease management. This can greatly help to reduce the level of mycotoxins in food materials and a significant loss of yield and quality due to FHB can be controlled. Several prediction models employing stepwise logistic regression analysis, boosted regression trees (BRTs), and non-parametric correlation analysis have been established for the prediction of FHB (De Wolf et al. 2003; Shah et al. 2014). In these models, several factors including rainfall, relative humidity, and temperature combinations estimated at different periods were considered as promising predictor variables. These models had variable sensitivity, specificity, and prediction accuracy toward disease warning. However, some of these models were accurate up to 84% for 50 location-years (Shah et al. 2014; De Wolf et al. 2003). An FHB center has been developed in the University of Delaware: this center allows access to the FHB prediction tools (https://www.wheatscab.psu.edu/) after signing up on their website. The extent of Fusarium mycotoxins can also be reduced by the utilization of proper infrastructure such as processing, handling the technical issues in transportation and safe storage conditions, and, most importantly, skilled human resources.

In addition, as a defense strategy, plants are able to convert the chemical arrangement of mycotoxins for the inhibition of their xenobiotic consequences (Galaverna et al. 2009). For example, the mycotoxins produced by Fusarium, ZEA, and DON can be changed into non-virulent compounds, zearalenone-14-glucoside, and DON-3-glucoside, respectively, with the help of the enzyme glucosyltransferase (Berthiller et al. 2017).

Virulence in *F. graminearum*

Virulence is the capacity of microorganisms to cause disease. The identification of virulence factors of *F. graminearum* is necessary to regulate their biosynthesis and for developing the host's resistance towards this pathogen. It has been determined that there are regulatory signals in

infested plants that have an important role in triggering the biosynthesis of mycotoxins (Merhej et al. 2011; Mudge et al. 2006; Voigt et al. 2005; Kazan et al. 2012). The FHB has a close association with DON, which is the key factor for virulence, and it endows the pathogen with a stealthy ability of virulence. If the biosynthesis of DON is suppressed, then the virulence of infection can be reduced (Maier et al. 2006). There are several inducing or repressing agents such as polyamines, sugars, pH, and cobalt chloride that affect the virulence level of *F. graminearum* in culture by controlling the DON biosynthesis (Kazan et al. 2012). Across the fungoid domains, the secreted proteins also play a significant role in virulence. Because of the redundancy, several of these secreted proteins are accurately associated with the virulence of *F. graminearum* [(Yang et al. 2013), please read this review article for details]. The *F. graminearum*-secreted proteins play an important role in the degradation of the cell wall, starch, and proteins (Phalip et al. 2005; Paper et al. 2007; Yang et al. 2013; Gunnaiah et al. 2012). Various virulence factors have been determined by the in silico analysis of *F. graminearum* secretome (Brown et al. 2012). FgGPMK1, mitogen-activated protein (MAP) kinase, is an important factor for the virulence of *F. graminearum,* especially during the early infection stage as it controls the stimulation of secreted lipolytic, proteolytic, xylanolytic, and endoglucanase activities (Jenczmionka and Schäfer 2005; Salomon et al. 2012; Dilks et al. 2019). Filamentous growth, infection, stress, and sexual reproduction are the imperative factors that determine a wide range of virulence of *F. graminearum*. Nitrogen availability is an important factor affecting the virulence of pathogenic fungi (Walkowiak and Subramaniam 2014). Other virulence factors such as hydrolytic enzyme secretions are greatly responsible for the initial stage of infection. At the early and later infection stages, the biotrophic and necro-trophic life of *F. graminearum* is controlled by trichothecene synthesis; the pulling out of nutrients by fungus leads to cell death (Walter et al. 2015; Dweba et al. 2017). The efforts to find the pathways for manipulating the Fusarium genome with the aim of interrupting the trichothecene pathways may greatly diminish the virulence. Further, the manipulation of the host genome using techniques such as InFusion HD cloning, insertional mutagenesis, and fungal transformations can be used to develop tolerance towards necrosis that occurs along with the overexpression of transport proteins (Walter et al. 2010, 2015; Dweba et al. 2017).

Types of host resistance to FHB

There are five types of resistance to FHB: Type I and Type II are the primary and stable ones for the selection of FHB resistance in wheat breeding programs. Type I resistance, known as primary infection, is mostly estimated by scoring the number of infected spikelets after 7 to 21 days of spray inoculation. Type II resistance starts at the time of the spreading of the disease, and it is identified from the infected spikes after the point inoculation when the host plant prevents infection from spreading (Schroeder and Christensen 1963).

The mechanisms of Type IV and Type V resistance are not well understood; hence, they are not widely utilized for identifying FHB resistance in wheat (Zhang et al. 2011; Eldakak et al. 2018). Type III resistance is toward the infection of the kernel, Type IV resistance is toward FHB and DON, and Type V resistance is toward the DON accumulation when the host plant is capable of demeaning the involved mycotoxins (Gilbert and Tekauz 2000; Mesterházy et al. 1999; Mesterhazy et al. 2003; Gunupuru et al. 2017).

Type III resistance is a quantitative method that is based on the measurement of DON concentration. It is not based on the observation of symptoms. However, the DON concentration can be the result of pathogen invasion as well (Eudes et al. 2001). Association between the DON, disease development, and pathogen invasion is highly variable and complex (Jansen et al. 2005). With the aim of screening for the resistant germplasm, the formation of a quantitative strategy that is dependent on the establishment of a marker at the host–pathogen interface stage would largely facilitate the process.

Molecular strategies in developing the FHB resistance in wheat

Molecular marker-based studies on FHB

Marker-assisted selection is an advantageous strategy due to its quantitative nature and complex breeding for FHB resistance. After the discovery of FHB in England in 1884, several efforts have been made for the establishment of molecular markers for the disease. After the first mapping of QTL in Chinese spring wheat accession (Anderson et al. 2001; Buerstmayr et al. 2003), FHB-resistant genes were introgressed into adapted germplasm (Miedaner et al. 2006; Anderson et al. 2007; Salameh et al. 2011). Bai et al. (1999) determined a significant linkage between AFLP markers and scab resistance using recombinant inbred wheat lines (RILs) grown in greenhouse conditions. They found a 60% association between scab resistance and a major quantitative trait locus and emphasized the utility of AFLP markers in marker-assisted breeding to ameliorate wheat resistance to scab. Several chromosomal regions other than 3BS, such as 2AS and 2BL that are associated with scab resistance, were detected using microsatellite and AFLP markers (Zhou et al. 2002). In 2003, Liu and Anderson identified two Sequence

Tagged Sites (STS) markers, two novel Simple Sequence Repeats (SSR), and one Restriction Fragment Length Polymorphism (RFLP) marker mapped on *Fhb1*, a major QTL for FHB resistance. However, their study was directed toward the presence of some other novel FHB resistance genes in the tested genotypes.

A large number of studies have been focused on validating *Fhb1* gene on chromosome 3BS for FHB resistance. Throughout the world, SSR markers have been used to launch *Fhb1* in wheat breeding cultivars (Del Blanco et al. 2003; Miedaner et al. 2006; Pumphrey et al. 2007). Although the detection of the major quantitative trait locus (QTL) related to FHB resistance on chromosome 3BS facilitated the research in this direction, the marker density of SSRs (Xgwm493 and Xgwm533) in the QTL region is comparatively lesser than that required for the marker-assisted selection (MAS) and map-based cloning. Focusing on this, Yu et al. (2008a) identified single-strand conformational polymorphism (SSCP) markers derived from wheat-expressed sequence tags (ESTs) on 3BS to increase this marker density. They have suggested three potential SSCP markers, Xsscp6, Xsscp20, and Xsscp21, that showed a higher coefficient of determination than the used SSR markers and that can be used further for map-based cloning and marker-assisted assortment in breeding for FHB resistance.

The combined usage of marker-based selection and phenotypic selection for the incorporation of positive alleles for FHB resistance has been stressed by a number of researchers (Buerstmayr et al. 2009; Wilde et al. 2007). Yang et al. (2003) employed microsatellite markers from 3BS and 6B chromosome arms and determined up to 36% and 21% of phenotypic variation, respectively, in two double-haploid populations. Liu et al. (2019b) utilized SNP markers and determined that QTL *Fhb1* was associated with a 3.1% phenotypic variation. Haberle et al. (2007) determined 27% individual influence and 36% combined influence of two QTL on 6AL and 7BS on FHB resistance in European winter wheat cultivars.

Bernardo et al. (2009) identified a novel microarray-based type of marker, single feature polymorphism (SFP) associated with the *FHB1* region in 3BS. Though these EST-based markers efficiently identify the DNA sequence variation, these are not frequently used in MAS due to the difficulty in discovering these markers (Bernardo et al. 2012). However, SNP markers linked to these SFP markers can be developed to simplify the association mapping and MAS approach (Bernardo et al. 2012). The association genetics approach can also be beneficial for the detection of FHB resistance in wheat (Miedaner et al. 2011). In 2012, Bernardo et al. mapped seven Wheat EST-derived SNPs markers near *Fhb1*. Most of them accounted for about 50% of phenotypic variation for FHB resistance. An association between the 90 K SNP markers and phenotypic data for FHB resistance in Norwegian spring and winter wheat lines has also been determined (Jansen 2015). More than 100,000 SNP markers were determined by genotyping-by-sequencing (GBS) of more than 400 spring wheat breeding lines and the marker linked to QTL *Fhb1* described only 3.1% of the total phenotypic variation (Liu et al. 2019b). The association of *Fhb1* markers with Fusarium head blight resistance in wheat varies according to the type of inoculation, experimental environments, genetic context, and resistance level of the assessed genotypes (Bokore et al. 2017; Zhao et al. 2018; Liu et al. 2019b; Herter et al. 2019b; Miedaner et al. 2019). An analysis based on the number of SNP markers has been conducted to identify the QTLs and novel locus associated with FHB resistance (Petersen et al. 2017; Zhao et al. 2018; Yi et al. 2018; Liu et al. 2019b; Hu et al. 2020). These marker-based studies led to the identification of several wheat sources with *Fhb1* and provided high-resistance toward FHB. *Fhb1* has been introduced into several commercial cultivars especially in China, the United States, and Canada. Modern cultivars such as AAC Brandon, Prosper, and Alsen are obtained from the implication of the gene-pyramiding technique combining various resources of FHB resistance and high yield (Zhu et al. 2019).

QTLs associated with FHB resistance

The development of resistance against FHB in wheat genotypes can be largely performed by the association of molecular techniques with classical breeding methods. A number of QTLs have also been determined to be involved in providing resistance to FHB. *Fhb1* located on chromosome 3BS is a well-recognized QTL identified in different wheat cultivars, including the Chinese wheat cultivar and the line 'Sumai 3' and W14, respectively (Cuthbert et al. 2006; Chen et al. 2007; Zhao et al. 2018; Waldron et al. 1999).

For FHB resistance, another QTL on chromosome 3AS has been considered crucial with regard to wheat (Otto et al. 2002). *Fhb2* and *Fhb4* located on chromosome 6B and chromosome 4B, respectively, also regulate FHB resistance (Yang et al. 2003; Cuthbert et al. 2007; Xue et al. 2010). Another QTL, *Fhb5* located on chromosome 5A directs Type I resistance to FHB (Xue et al. 2011). In different alien species, *Leymus racemosus* and *Elymus tsukushiensis*, FHB resistance gene, *Fhb3,* and *Fhb6* were discovered on the short chromosome 7Lr#1 and 1EIS#1S, respectively (Qi et al. 2008; Cainong et al. 2015). In addition, a phenotypic variation of 22% and 24% for Type II and Type III resistance was determined in *Fhb7AC* QTL located on chromosome 7A (Jayatilake et al. 2011). A major QTL for FHB resistance was determined on 2DLc that overlapped with other QTLs for plant height and days to heading in synthetic hexaploid wheat Soru#1 (He et al. 2016).

Identifying major QTLs for FHB resistance in resistant cultivars and transferring them into susceptible cultivars comprise a major strategy for developing resistance toward FHB in wheat genotypes (Suzuki et al. 2012) (Table 1). A number of studies have reported the importance of 3B chromosome in providing Type II and DON resistance toward FHB in both durum and hexaploid wheat (Anderson et al. 2001; Buerstmayr et al. 2002; Somers et al. 2003; Cuthbert et al. 2006). Though hexaploid wheat has many better examples for FHB resistance (Bai and Shaner 2004; Mesterházy 1997; Ban and Suenaga 2000; Rudd et al. 2001; Singh et al. 1995; Mentewab et al. 2000), now durum wheat with FHB resistance has also been developed (Giancaspro et al. 2016). Recently, a major QTL *Qfhs.ifa-5A* associated with FHB resistance has been found to be linked with anther extrusion (Steiner et al. 2019). The studies reporting the overlapping

Table 1 Sources of FHB resistance and the location of the involved QTLs have been included in the article

Source	Chromosome	References
Sumai 3	7A, 3BS, 6BS	(Anderson et al. 2001; Liu and Anderson 2003; Liu et al. 2006; Cuthbert et al. 2006, 2007; Jayatilake et al. 2011; Waldron et al. 1999)
Stoa	2AL, 4BS	(Anderson et al. 2001; Waldron et al. 1999)
ND2603	3AL, 6AS, 3BS	(Anderson et al. 2001)
Ning 7840	2AS, 2BL, 3BS	(Bai et al. 1999; Zhou et al. 2002; Guo et al. 2003)
CM-82036	5A, 1B, 3BS	(Buerstmayr et al. 2002, 2003; Lemmens et al. 2005)
Ning 894037	3BS, 6BS	(Shen et al. 2003b)
Alondra	2DS	(Shen et al. 2003b)
Huapei 57-2	3AS, 3BS, 3BL	(Bourdoncle and Ohm 2003)
Patterson	5BL, 3D	(Bourdoncle and Ohm 2003; Shen et al. 2003a)
Wuhan 1	4BS, 2DL	(Somers et al. 2003)
Nyu Bai	5AS, 3BS, 2D	(Somers et al. 2003)
DH181	5AS, 3BS, 6BS, 7BL, 1DL, 2DS, 4DL	(Yang et al. 2005b)
W14	5A, 3BS	(Chen et al. 2006)
CS-SM3-7ADS	3BS, 2D, 4D	(Ma et al. 2006a)
CJ 9306	1AS, 5AS, 3BS, 7BS, 2DL	(Jiang et al. 2007b, a)
Gamenya	2DS	(Handa et al. 2008)
Wangshuibai	2A, 3AS, 5A, 7A, 1B, 2D, 3BS, 4B, 5B, 6B, 2DL, 3DL, 5DL	(Lin et al. 2004, 2006; Zhang et al. 2004; Zhou et al. 2004; Jia et al. 2005; Mardi et al. 2005; Ma et al. 2006b; Yu et al. 2008b)
Frontana	3A, 5A, 7AS 2B, 6B	(Steiner et al. 2004)
Remus	2A, 1B	(Steiner et al. 2004)
Seri82	1BL	(Mardi et al. 2006)
Chokwang	3BS, 4BL, 5DL	(Yang et al. 2005a)
Sincron	1DS	(Ittu et al. 2000)
Renan	2A, 5AL, 2BS	(Gervais et al. 2003)
Goldfield	2B, 7B	(Gilsinger et al. 2005)
Arina	4AL, 5AL, 1BL, 3BL, 6BL, 6BS, 4DS, 6DL	(Paillard et al. 2004; Semagn et al. 2007)
Forno	3AL, 5BL, 3DS	(Paillard et al. 2004)
NK93604	1AL, 2AS, 7AL	(Semagn et al. 2007)
Dream	6AL, 2BL, 7BS	(Schmolke et al. 2005)
Ernie	2B, 3B, 4BL, 5A	(Liu et al. 2007)
Triticum macha	4AS	(Steed et al. 2005)
Thinopyrum ponticum 7el2	7el	(Shen and Ohm 2007)
Triticum dicoccoides FA-15-3	3AS	(Chen et al. 2007)
Triticum durum cv. Strongfield	2BS	(Somers et al. 2006)
Triticum carthlicum cv. Blackbird	6BS	(Somers et al. 2006)
Triticum dicoccoides: PI478742	7AL	(Kumar et al. 2007)
Leymus racemosus	7Lr#1	(Qi et al. 2008)
Elymus tsukushiensis	1EIS#1S	(Cainong et al. 2015)

of QTLs for FHB resistance with the QTLs of other traits revealed that FHB resistance is regulated by many underlying genetic factors and involves pleiotropy. The identification of other components may facilitate the understanding of resistance of FHB and support the gene cloning and actual breeding programs. Additionally, the association of modern genetic tools such as gene editing and genomic selection with the available high-quality reference wheat genome may open new avenues for the development of FHB-resistant cultivars.

Proteomics-based studies on FHB resistance

Due to the devastating effects of Fusarium throughout the world, scientists are trying to understand its evolution, pathogenicity, population biology, and genetic basis of its life cycle. Several "omic" techniques are used to study the effects of Fusarium and their interactions with the host plants. Proteomics is the core technology that allows the interpretation of the function of genes, locations, interactions, modifications, determination of the abundance of proteins, and implications. Over the past few years, the analysis of the proteome of phytopathogenic fungi and their interactions with host species has increased (Perlikowski et al. 2016; Eldakak et al. 2018; Gunnaiah et al. 2012; Liu et al. 2019a). This interest in proteome analysis is due to an increase in the number of sequenced fungal genomes with the advancement in bioinformatics tools.

The main inquisitions in this field are the estimation of conidial, mycelial secreted proteins in the wide array of fungal species by the establishment of fungal structures from reference proteome maps. Proteome profiles of different races, mutants, species, developmental stages, growth stages, and different growth conditions are compared (Yang et al. 2013). These proteome profiles are mostly studied during the hyphal penetration, spore germination, toxin production, appressorium formation, and secretion (van Kan 2006). These are used to understand plant–fungal interactions of major crops such as maize, rice, wheat, and barley as well as to study the infection cycles and for the identification of pathogenicity factors that are responsible for the defense responses of plants (González-Fernández et al. 2010). The post-translational modifications (PTMs) can be investigated by employing proteomics. Some post-translational modifications such as glycosylation, phenylation, phosphorylation, acetylation, ubiquitylation, and S-nitrosylation are involved in transducing the signals during the interface of plants and microbes that have been examined by the proteomics (Jayaraman et al. 2012). Gunnaiah et al. (2012) implemented the combined metabolomics and proteomics techniques and revealed that the FHB resistance mechanism in the Nyubai wheat genotype can be due to the accumulation of phenolic glucosides, flavonoids, and hydroxycinnamic acid amides that lead to the thickening of the cell wall. As proteomics open new avenues for identifying differentially accumulated proteins (DAP) during host–pathogen interactions, several researchers conducted the proteome profiling to determine the FHB resistance mechanism in wheat (Wang et al. 2005; Zhou et al. 2005, 2006; Eggert et al. 2011; Zhang et al. 2013; Eldakak et al. 2018). Eldakak et al. (2018) determined the proteomic changes in spikelets of two contrasting wheat lines (with and without *Qfhb1*) during early infection of FHB. Employing 2D-DIGE and MALDI-MS/MS techniques, they identified 80 DAP that was involved in several mechanisms such as sucrose metabolism, photosynthesis, translation, and repairing of signaling molecules. In a similar study, Liu et al. (2019a) confirmed the presence of purple acid phosphatase and late embryogenesis abundant proteins in inoculated wheat accessions in response to *F. graminearum*. Proteomics requires the consequent functional investigation of the corresponding genes for the identification of fungal effectors of FHB that possibly either facilitate the infection or trigger the plant defense. Kazan et al. (2012) summarized about the *F. graminearum* genes which are related to the production of mycotoxins, metabolism, growth, and signal transduction. These genes have been studied in detail to understand their contribution toward pathogenicity and virulence using the proteomics. By following the proteomics-based techniques, the resistance to the FHB can be greatly improved. However, due to the dynamics and complexity of the proteome and the expensive nature of the technique, conducting an entire characterization is still challenging.

Gene silencing and transgenic studies on FHB resistance

Though various techniques have been developed to control the FHB, still its control is partially effective. The expression of harmful FHB genes can be regulated using the RNA interference method (Hu et al. 2015; Song et al. 2018; Werner et al. 2019; Koch et al. 2016; Majumdar et al. 2017; Machado et al. 2018). RNA silencing or RNA interference is a gene silencing mechanism that is post-transcriptional in nature. It involves the degradation of mRNA, which is sequence specific using small molecules of RNA (Fire et al. 1998). Host-induced gene silencing (HIGS) is employed in plants to silence the fungal genes so that both the chances and the level of the disease are reduced. The capacity of the host plant to produce small interfering RNA molecules that are complementary to the fungal genes and that are developed from the long double-stranded RNA may vary (Lin et al. 2010). In *F. graminearum*, Dicer protein (FgDicer2) and Argonaute protein (FgAgo1) are involved in (hairpin) hpRNA-induced gene silencing (Chen et al. 2015). HIGS can develop resistance in wheat to both FHB and Fusarium seedling blight (FSB) with regard to natural field infections

and also under controlled environmental conditions (Cheng et al. 2015). It directly affects the biosynthesis of chitin which is synthesized by chitin synthase enzymes and is an indispensable constituent of the fungal cell wall. Thus, employing the gene silencing of chitin synthase genes, chitin synthesis can be reduced and fungal growth can be controlled. It is predicted that *F. graminearum* genome contains eight genes for chitin synthesis Chs1, Chs2, Chs3a, Chs3b, Chs4, Chs5, Chs6 and Chs7. Among these genes, Chs3b had shown the highest expression level during the infection to the heads of wheat and the deletion of this gene was toxic *to F. graminearum* (Cheng et al. 2015). Fan et al. (2019) employed virus-induced gene silencing (VIGS) to determine that three genes engaged in the jasmonic acid (JA) signaling pathway, *TaAOC*, *TaAOS*, and *TaOPR3* positively control FHB resistance. Kage et al. (2017) confirmed the role of gene-encoding agmatine coumaroyl transferase, *TaACT* located on wheat-FHB QTL-2DL in the fortification of the cell wall using VIGS methods.

Though RNAi is a favorable substitute to the fungicides via the development of FHB-resistant wheat cultivars, it will not help in controlling the disease after the post-harvest stages, such as in dried seeds, fruits, roots, and leaves. It is due to the lesser metabolic and physiological activities in the desiccated parts of the plants. There is a public debate for the probability that dsRNA or siRNA could enter the bodies of mammals through the food chain and may affect the gene expression in animals and human beings. Specific studies have reported that siRNA could be delivered to the internal system of mammals via the digestive tract (Zhang et al. 2012). There is a need for a series of studies to sort out this issue.

It is likely to attain significant levels of resistance to FHB by the introduction of extraneous genes with remarkable effects that are transformed into elite genotypes (Makandar et al. 2006). Owing to the unlimited capacity for encoding the proteins, several genes have been suggested for their contribution to FHB resistance (Xue et al. 2011). The expression of the AtNPR1 gene of *Arabidopsis thaliana* developed heritable, type II FHB resistance in susceptible wheat cultivar, Bobwhite and was found to activate the systemic acquired resistance (Makandar et al. 2006). A few genes that have been inserted from the non-*Triticum* genomes have exerted negative influences on wheat physiology when expressed in the genome of *Triticum sp.* (Han et al. 2012). Even the incorporation of the NPR1 gene of Arabidopsis in the Yangmai 11 cultivar leads to enhanced susceptibility toward FHB (Gao et al. 2013). It has been suggested that the pathogen attack at different wheat development stages affects the functioning of the NPR1 gene (Gao et al. 2013). Thus, before recommending alien genes, specific differential effects depending on growth stages should be first confirmed. Mackintosh et al. (2007) revealed that overexpression of β-1, 3-glucanase gene increased the resistance of wheat cultivars towards FHB with a lesser DON concentration. For genetic engineering, some targeted genes that encode the enzymes for DON detoxification and responsible genes for the biosynthesis of antifungal proteins or possessing inhibitory actions for FHB are under special consideration (Ferrari et al. 2012; Hou et al. 2015; Mandalà et al. 2019). The barley HvUGT13248 gene involved in the glycosylation when expressed in durum and bread wheat plants led to enhanced DON-detoxification (Mandalà et al. 2019). Under the pathogen attack, some resistant genes may be overexpressed in the wheat genome, including the ones that encode the stress-responding hormones such as salicylic acid, ethylene, and methyl jasmonate (Makandar et al. 2012). This overexpression of genes possesses great potential to increase FHB resistance by affecting the signaling molecules and important transcription factors in plants (Bahrini et al. 2011). Numerous transgenic undertakings designate that foreign genes can possibly enhance the options to tackle the disease and genomic diversity. In addition, there are available tools for tracking the accounting genes for differential responses in cereals to the Fusarium attack. Using these tools, the influence and compatibility of such genes in wheat can be tested.

Genome-wide association studies (GWAS)

Genome-wide association studies (GWAS) facilitate the mapping of the potential genes so that the efficient markers tightly associated with a specific trait can be developed (Wang et al. 2014; Liu et al. 2019b). The development in high-throughput genotyping and genome sequencing methods has enabled the GWAS in large genome-size species such as wheat [International Wheat Genome Sequencing Consortium (IWGSC), 2018]. Several researchers have performed GWAS for FHB resistance in wheat and revealed important findings regarding the complex genetic mechanism (Mirdita et al. 2015b; Arruda et al. 2016b; Wang et al. 2017). It has been determined that a number of genes with variable effects add to FHB resistance in wheat besides well-defined QTLs. A number of studies emphasized the efficiency of genome-wide markers over the statistical markers for genomic selection (Rutkoski et al. 2012; Jiang et al. 2015, 2017; Mirdita et al. 2015a; Mamo and Steffenson 2015; Arruda et al. 2016a; Herter et al. 2019a). Arruda et al. (2016b) performed genotyping-by-sequencing (GBS) of 273 winter wheat breeding lines and identified that more than 19,000 SNPs lying on all 21 wheat chromosomes elucidated 8% of phenotypic variation. Tessmann and Van Sanford (2018) estimated the phenotypic response of 238 soft winter wheat lines grown during two different years in control and warmed conditions and determined 19 and 10 significant SNPs by employing the GWAS method. Wu et al. (2019) identified three and six loci linked with DON accumulation

and FHB resistance by GWAS performed on 213 Chinese accessions grown in four different environments.

FHB resistance associated with alien species

The usage of resistant cultivars is one of the effectual, economic, and environmental friendly strategies that can be employed to control FHB. Resistance to FHB is a quantitative trait that is regulated by multiple genes. As a result, breeding for resistant cultivars is not an easy task. In wheat, the QTLs for FHB were found to be present on all the chromosomes except the 7D (Buerstmayr et al. 2009). It has been determined that various wheat relatives are resistant to FHB. A high FHB resistance is found in *Aegilops*, *Agropyron*, *Elymus, Hystrix*, *Kengyilia, Thinopyrum ponticum, Th. elongatum, Th. intermedium, Dasypyrum, Leymus,* and *Roegneria* (Mujeeb-Kazi 1983; Cai et al. 2005; Wan et al. 1997; Yong-Fang et al. 1997; Oliver et al. 2005; Cai et al. 2008; Qi et al. 2008; Cainong et al. 2015).

These resistant wheat relatives having different ploidy levels extending from 2 to 10 × could be used as an important source to obtain FHB-resistant genes. As wheat is allopolyploid, the alien chromatin genes could be incorporated into the cultivated wheat by employing cytogenetic strategies and substitution, addition, translocation, and recombinant lines; these can also be developed via backcrossing with wheat cultivars (Oliver 2005; Bai et al. 2018). A high level of resistance has been developed in wheat cultivars by transferring FHB-resistant regions, *Fhb3*, *Fhb6,* and *Fhb7* from *Leymus racemosus*, 1Ets#1S of *Elymus tsukushiensis,* and *Thinopyrum ponticum* (Qi et al. 2008; Cainong et al. 2015; Guo et al. 2015). However, a pyramiding of *Fhb3* with *Fhb1* employing marker-assisted selection did not show FHB resistance in developed cultivars, thus, emphasizing that the efficiency of transferred alien genes in providing FHB resistance should be tested in different environments (Bai et al. 2018).

This transfer of FHB-resistant alien genes can significantly increase the genetic diversity of wheat genotypes toward FHB resistance (Han et al. 2012). In wheat, more than 100 fragments of alien chromosomes from *Roegneria kamoji, Triticum macha, T. ponticum, Elytrigia intermedia, Elymus racemifar,* and *Leymus racemosus* related to FHB resistance have been efficaciously integrated (Oliver et al. 2005). In a detailed screening of 293 lines obtained from the crosses of wheat and its relatives, 74 lines showed potential significance toward FHB (Oliver et al. 2005). However, meiotic lines, linkage drag, and chromosome instability on the discrete alien chromosomes limit the utilization of substitution and addition lines (Cai et al. 2005; Bai et al. 2018). Thus, it is problematic for breeders to rightly utilize the substitutional, addition or amphiploid lines in breeding programs.

To reduce the chances of linkage drag, wheat alien translocation can be used as it only conveys the alien chromosomal segment associated with the wheat chromosome. Thus, alien translocation in wheat can be considered an effective approach toward introducing the FHB-resistant genes that have been extracted from the alien species (Cai et al. 2005). More alien genes that are resistant to FHB should be identified by the breeders and cryptic translocation techniques should be utilized for the integration of these genes into wheat genomes for higher FHB resistance. Moreover, genomic and phenotypic selection and gene editing techniques can facilitate the combining of several QTLs to develop FHB-resistant cultivars.

Status of FHB-resistant genetic resources and developed cultivars

To date, a number of differentially FHB-resistant wheat landraces around the world have been selected by local farmers (Talas et al. 2011; Zhu et al. 2019; Li et al. 2011, 2016; Jia et al. 2018). FHB resistance in several cultivars such as 'Wangshuibai', 'Sumai 3', 'Gamenya', 'Alondra', 'Nyubai', 'Romanus', 'Frontana', 'Spark', 'Wangshuibai', 'Arina' and 'F201R' has been determined using specific locus markers such as Xgdm35-2DS and Xbarc19-3AS (Buerstmayr et al. 2009) (Table 1). Mapping has played a crucial role in identifying the QTL Fhb1, which is one of the most important loci known for FHB resistance and is found to be located on the 3BS chromosome of the Chinese cultivar Sumai 3 for the first time (Bernardo et al. 2012; Gunnaiah et al. 2012; Rawat et al. 2016). Many cultivars such as Glenn, Wanshuibai, Frontana, and L699 have been identified to be FHB resistant due to the presence of loci other than Fhb1, such as *Fhb2, Fhb4,* and *Fhb5* (Zhang et al. 2011; Yang et al. 2016; Liu et al. 2015; Lin et al. 2006; Mergoum et al. 2006; Bokore et al. 2017; Cai et al. 2016; Steiner et al. 2004). ND2710 is one of the initially developed FHB-resistant lines that evolved from Sumai 3 via the NDSU breeding program (Frohberg et al. 2004).

Future perspectives for FHB

By controlling FHB, major destructions in terms of both quality and quantity of wheat can be controlled to a very great extent. The utilization of gene-derived markers and diagnostic features can be effective for developing superior cultivars that can tolerate FHB. Management practices for host plant resistance can be promising to control the FHB pathogen. A few strategies will not help to completely cover up the losses arising due to the virulence shifts of *F. graminearum*, so there is a need for international collaboration to control the existing species and to tap the genetic and

genomic resources to manage the FHB. The need for this collaboration was recognized by the US Wheat and Barley Scab Initiative (USWBSI) where a prediction center for FHB was developed so that growers may understand the risk of FHB outbreak in their region (https://www.wheatscab.psu.edu/riskTool.html). In 2005, the International Maize and Wheat Improvement Centre (CIMMYT), started research on FHB that significantly assisted international communication via the Japan-CIMMYT FHB project. By taking such initiatives, the exchange of synthetic derivatives of wheat for genetic characterization and identification of FHB-resistant sources from the accessions available in gene banks will be easier. It may also facilitate the evaluation of the incidence and the distribution of notorious pathogens so that farmers may stay alert for the upcoming FHB outbreak. Several efforts have been made to understand the evolution of virulence, the effect of the changing environment, and toxin biosynthesis related to FHB to develop some strategies for disease control. If all the available information can be integrated with genetic engineering and plant pathology, there can be significant chances to develop much more reliable strategies for disease prevention and, consequently, food production and quality can be enhanced. In addition, the identification of novel loci that are responsible for FHB resistance and the development of SNP-based diagnostic markers for these regions to be used in marker-assisted breeding can prove to be potential techniques. The integration of speed breeding and molecular markers into conventional breeding programs may open new avenues in this direction.

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Compliance with ethical standards

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

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