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Chapter

Breeding Wheat for Biotic Stress Resistance: Achievements, Challenges and Prospects

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Abstract

Wheat (*T. aestivum*) is one of the key food grain crops and is a prominent source of calories and proteins globally. In addition to mushrooming population and rising abiotic stresses in this ongoing climate change era, biotic stresses pose a great threat to wheat production over the globe. Fungal diseases such as rusts, mildew, along with pests like aphid, hinder the potential yield performance of the elite wheat cultivars to a huge extent. The complex nature of plant-parasite interactions is shown to be the decisive factor for the ultimate resistance expression in wheat. However, the advancement of molecular genetics and biotechnology enabled the replacement of the tedious, time and resource consuming cytogenetic analyses of locating APR and ASR genes using molecular mapping techniques. Continuous efforts have been made to mine resistance genes from diverse genetic resources such as wild relatives for combating these diseases and pests, which are repositories of R genes. Additionally, they offer a promising source of genetic variation to be introgressed and exploited for imparting biotic stress tolerance in cultivated wheat. Though just a handful of R-genes are cloned and molecularly characterized in wheat so far, more than 350 resistance genes for various diseases have been identified and successfully introgressed into elite varieties around the globe. Modern genomics and phenomic approaches coupled with next-generation sequencing techniques have facilitated the fine-mapping as well as marker aided selection of resistance genes for biotic stress resistance wheat breeding.

Keywords: Biotic stress, Durable resistance, Genomics, R-genes, Wheat rusts, Wild relatives

1. Introduction

Wheat is one of the most important cereal staple food crops in the world, both in terms of food production and for providing the total amount of food calories and protein in the human diet [1]. It is believed that bread wheat originated in south western Asia from where it spread to other regions of Asia, Europe, Africa and America [2]. Wheat has adapted itself to diverse climatic conditions and, as such, is grown over a range of altitudes and latitudes under irrigated, severe drought and wet conditions. The global demand for wheat is projected to rise by 60% by 2050 because of the increase in the world's human population and changing livelihoods.

Wheat production has been threatened by unexpected abiotic and biotic stresses due to abrupt environmental changes or movement of pathogens. The monoculture of modern wheat cultivars with low genetic diversity has resulted in pathogen resurgences, which threaten wheat supplies [3].

Biotic stress in plants is caused by several living organisms namely fungi, virus, insects, nematodes, arachnids and weeds. Unlike the stresses caused by environmental factors i.e. abiotic stresses (heat and drought), the biotic stress agents directly affect the host growth and development by depriving them of nutrition resulting into reduced plant vigor and in extreme cases, even death of the host. From the agricultural context, biotic stress has major contribution in pre as well postharvest losses. Of the nearly 200 diseases and pests that have been documented, 50 are considered economically important because of their potential to damage crops and affect farmers' incomes [4]. Among biotic stresses, pathogenic fungi represent a significant challenge to wheat production globally. The major diseases in wheat involves stripe rust, stem rust, leaf rust, powdery mildew, head blight etc. Historically, yellow rust has caused and is presently causing significant and severe losses in susceptible wheat cultivars worldwide [5]. The major insect-pests attacking wheat are aphid, hessian fly, green bug and borers etc.

In this chapter, the major diseases and pests detrimental to wheat crop along with the molecular basis of stress resistance will be discussed. Moreover, the remarkable global milestones being achieved along with some important tools and prospects for mitigating with these economically important diseases and pests will be focused.

2. Biotic stress resistance in wheat

2.1 Types of disease resistance

There are basically two types of genetic resistances as described by Vander Plank [6] for the different diseases in wheat i.e. Qualitative/Vertical resistance and Quantitative/Horizontal resistance.

2.1.1 Qualitative (vertical) resistance

It is specified to pathogen races controlled by a single or few genes i.e. monogenic or oligogenic. Race-specific is used to describe resistance that interacts differentially with different pathogenic races i.e. it is applied both to complete resistance and components of incomplete resistance that so interact [7]. This kind of resistance is easily detectable with specific pathogenic races or pathotypes which are controlled by genes with major effects. In wheat rust pathosystems, these resistances are recognized by characteristic low infection types. Most of these genes can be detected in seedling evaluations using specific pathotypes. For every resistance gene in the host plant, there is a corresponding virulent gene in the pathogen as stated by gene for gene hypothesis. However the ability of a virulent gene to mutate to avirulent gene, no longer recognizable by the corresponding resistance gene, implies a type of resistance termed race-specific resistance.

2.1.2 Quantitative (horizontal) resistance

This kind of resistance varies in continuous way among the different phenotypes of the host population, ranging from almost imperceptible to quite strong resistance response. The resistance expression depends upon the genotype and environment, where pathogen is the part of that environment. The environment can considerably

affect its durability also [7]. Partial resistance is supposed to be under polygenic control and such resistance will be race-nonspecific. Being controlled by minor genes, the quantitative resistance has complex genetic basis which operates against all the pathotypes/races of that specific pathogen. Race-nonspecific resistance is mainly effective at the post-seedling and adult plant stages and adult plant resistance (APR) is often detected as field resistance [8]. The best known APR genes in wheat are *Sr2* (stem rust resistance gene) and *Lr34*, a gene that provides resistance to leaf and stripe rust and powdery mildew. These genes have been used in commercial wheat varieties for almost 100 years. *Sr2* and *Lr34* have provided partial resistance for decades over large areas and under prolonged disease pressure in the field, proving their durability. Adding to complexity, Ug99 had a very wide spectrum of virulence towards most of the commonly used R genes and rapidly evolved virulence to the important R genes (*Sr24* and *Sr36*) which has impeded the initial emergency breeding response to incorporate resistance to this strain [9].

2.2 Types of insect resistance

Insect resistance on the other hand is typically governed by three main mechanisms.

2.2.1 Single or oligogenic resistance

Single or oligogenic resistance has been observed against some insects such as Hessian fly in wheat. Such resistance is governed by a single or few major genes. This type of resistance has also been reported against Russian wheat aphid and green bug.

2.2.2 Polygenic resistance

Several genes with small additive effects govern the resistant response against some insects. The resistance observed against cereal leaf beetle in wheat is of this type.

2.2.3 Cytoplasmic resistance

Cytoplasmic resistance against insects has not been reported in case of wheat. However, in maize resistance against European corn borer is governed by cytoplasmic genes. Another case of cytoplasmic resistance is observed in lettuce against root aphid.

3. Major diseases of wheat

There are many diseases found in wheat caused by different microorganisms from fungi to bacteria and viruses. But only a few of them caused by pathogenic fungi are economically important with global implications. The major diseases in wheat (**Table 1**) are stripe rust, leaf rust, stem rust, powdery mildew, loose smut, Fusarium head blight (FHB) and more recently wheat blast (WB) also. Besides Stem rust, which is under control to some extent, Leaf rust and yellow rust have the potential to affect production levels up to 60 and 43 million hectares respectively in Asia if susceptible cultivars were grown [10]. Though fungicidal applications offer control, their use is an added cost to farmers besides being unsafe environmentally. Hence growing resistant cultivars is the most effective and efficient control strategy

S no	Disease	Causal Pathogen	Behavior	No. of R-genes identified
1.	Stripe rust (yellow)	*Puccinia striiformis* f. sp. *tritici*	Biotrophic	95
2.	Leaf rust (brown)	*Puccinia triticina*	Biotrophic	80
3.	Stem rust (black)	*Puccinia graminis* f. sp. *tritici*	Biotrophic	67
4.	Powdery mildew (PM)	*Blumeria graminis* f. sp. *tritici*	Biotrophic	70
5.	Karnal Bunt (KB)	*Tilletia indica*	Biotrophic	6
6.	Fusarium head blight (FHB)	*Fusarium graminearum*	Necrotrophic	7
7.	Wheat blast (WB)	*Magnaporthe oryzae* pathotype *triticum*	Necrotrophic	5
8.	Loose Smut (LS)	*Ustilago tritici*	Biotrophic	10

Table 1.
Major diseases of wheat with their respective behavior and number of resistance genes identified for each disease (up to 2020).

[11]. The rusts and mildew diseases are caused by biotrophic fungi (survive by obtaining nutrients from living plant tissues). Among these, *Puccinia* rusts continue to affect and threaten the world's wheat production [12], although powdery mildew has also emerged as an economically important disease. In case of stem rust, the emergence of *Ug99* group of stem rust races placed it among one of the most significant threats to global wheat production [13].

The other diseases like FHB and WB are caused by necrotrophic fungi (facultative parasites feeding on dead tissue during unavailability of living plants). Wheat blast was first identified in Parana, Brazil in 1985 [14]. It is also of utmost significance as WB outbreaks in Bangladesh [15] and more recently in Africa [16] have attracted immediate global attention from the wheat scientists.

Another economically significant disease, Karnal Bunt (KB) of wheat was first reported in Karnal, India [17], soon extended to Northern and Central India. Later, KB was found to occur in Nepal, Pakistan, Iraq, Afghanistan, South Africa, Mexico and USA [18]. The pathogen is seed, soil and airborne in nature, therefore difficult to control after it is introduced and then established over a region. Although host plant resistance is the most effective and economic method of its management but development of KB resistance varieties is difficult task owing to limited genetic variability in hexaploid wheat [19], quantitative inheritance and considerable impact of environment on KB resistance screening [20].

4. Major insect-pests of wheat

Various insect pests delimit the yields of wheat crop in different agro-climatic zones. Some of these insect pests are foliar aphid complex in irrigated wheat, root aphids in loose soils, pink stem borers in fields having rice stubbles, cut worms in residues, termites in raised beds and brown mites in rainfed conditions [21]. Six different species of aphids are reported to attack cereals. Out of these, Russian wheat aphid and bird cherry-oat aphid are important pests of wheat. The Russian wheat aphid (*Diuraphic noxia*) is a sucking pest of wheat. Aphid attack is characterized

by leaf rolling which is the result of toxic injection by the aphid. The rolled leaves serve as a protection site for the insects. Yield losses up to 40% have been reported in case of aphid infection [22]. The bird cherry oat aphid (*Rhopalosiphum padi*) has been reported to affect wheats all over the world. Feeding symptoms are almost absent. Yield losses due to *R. padi* dependent upon the crop stage at which insect attacks. High yield losses upto 24–65% have been reported in case the attack occurs at seedling stage. Losses decrease if attack occurs at later stages [23]. The aphid is also reported to cause significant indirect losses as it is a vector of Barley Yellow Dwarf Virus (BYDV), which is the most important viral disease in cereals. Greenbug (*Schizaphis graminum*) is another sucking pest of the wheat aphid complex. The green bug feeds on wheat leaves and stems, extracting sap from the phloem. Injection of toxins concomitant with feeding further reduces the chlorophyll content thereby inversely affecting the carbon assimilation and overall plant development [23–25].

Cephus spp., the wheat stem sawfly has also been reported to cause major losses in wheat. The adult females oviposit into the young stems of wheat. Upon hatching within the stem, the larvae feed voraciously moving up and down in the stem. When the plant attains maturity, larvae migrate to the basal portion of the stem and build a hibernaculum. The stem above the hibernaculum weakens and breaks [26]. The Hessian fly (*Mayetiola destructor*) is another major pest of wheat crop. Larvae damage stems of plants, thereby preventing internode elongation and disrupting nutrient transport. Significant losses (upto 40%) have been reported upon sawfly attack [27].

5. Molecular basis of disease resistance in wheat

Wheat is an allopolyploid, means a polyploid species that resulted from interspecific or intergeneric hybridization of two or more genomes from different species. Polyploidy, a common form of plant evolution, is associated with promoting the genetic diversity that facilitates adaptation to a range of environments. Because wheat is a global crop, it is under continuous exposure to a large variety of parasite species and strains, many of which have the ability to move around the globe. Long-term co-evolution between plants and their pathogens has equipped plants with a sophisticated multi-layered immune system to guard themselves against pest and pathogens [28]. Specificity between pathogenic variants (races) and plant genotypes (cultivars) follows gene for gene-for-gene interactions, whose outcome is conditioned by alleles of a gene regulating resistance (*R* gene) in plant and alleles of its corresponding gene regulating avirulence (*Avr* gene) in pathogen [29]. The plant immune system is typically described in terms of two components: pattern triggered immunity (PTI) which is activated by recognition of microbial or pathogen-associated molecular patterns (MAMPs or PAMPs) and effector-triggered immunity (ETI) involving gene for gene kind of resistance [30, 31]. ETI is often based on the recognition of cytosolic effectors by immune receptors with a conserved nucleotide-binding domain (NBARC) and a leucine-rich repeat domain (LRR) also called NLRs. This type of resistance is usually associated with a hypersensitive response (HR) localized to infection sites. To date, only a handful of these biotic stress resistance genes have been isolated and cloned in wheat (*T. aestivum*). Donors of the *R* genes are genetically diverse, including species in the primary gene pool (*Triticum* spp.), secondary gene pool (e.g. *T. timopheevii*), and tertiary gene pool (e.g. *Aegilops*, *Secale*, and *Thinopyrum*).

5.1 NBS: LRR proteins - basis of race-specific/seedling/all stage resistance (ASR)

A few resistance genes have been cloned for race-specific resistance in wheat so far, which belong to a conserved gene family encoding NBS-LRR (Nucleotide binding site-leucine-rich repeat) proteins, also known as R-proteins (NLR) [30]. For example, powdery mildew genes, *Pm3* and *Pm8* and leaf rust resistance genes *Lr10* and *Lr21*. These R-proteins impart complete but race specific resistance. NBS-LRR proteins are a conserved class of immune receptors that directly or indirectly recognize pathogen-specific effector proteins. These proteins are secreted by pathogens into the host cell to suppress defense response and to establish infection. Recognition of effectors by NBS–LRR proteins triggers a signaling cascade resulting in a strong resistance response called hypersensitive reaction (HR) [31]. HR eventually leads to death of the infected host cell by this means preventing further spread of the pathogen [32]. Since this type of disease resistance depends on the recognition of specific pathogen effectors, even point mutations within effector genes or their loss can disrupt recognition by the corresponding NBS–LRR protein. Such mutations in pathogen effectors result in the emergence of new virulent pathogen races and breakdown of disease resistance. Mutated pathogen spores that avoid recognition by the corresponding R gene will have a huge selective advantage facilitating their rapid multiplication. Dispersal of fungal pathogens by wind over long distances adds to the quick spread of newly evolved virulent pathogen strains. Ug99, for instance, spread out from Kenya to South Africa and the Near East in less than a decade.

So far, only 31 genes have been cloned (**Table 2**) for biotic stress resistance (30 for disease resistance) from bread wheat and its wild relatives. Among these, most of the genes impart race specific resistance to the plant. These R-genes encode proteins with an NBS-LRR domain with a coiled-coil (CC) domain. This type of gene typically shows a greater degree of variation in LRR-encoding sequences [60, 61]. This is consistent with the idea that the LRR-encoding sequence is important for target specificity [61, 62]. The sequence variation in NBS-encoding region can also play significant role in specificity. For powdery mildew resistance, *Pm3* locus encodes seven alleles (*Pm3a–Pm3g*) providing resistance to different races of *Blumeria graminis* f. sp. *tritici* [63]. Sequence analysis indicated that the *Pm3* alleles evolved either by gene conversion/recombination or by single point mutations within the NBS and LRR regions [61].

5.2 Transporter proteins: basis of durable/adult plant resistance (APR)

Due to rapid pathogen evolution, R gene resistance is often not durable. One strategy to increase the longevity of disease resistance in wheat cultivars is to pyramid several R genes in one cultivar. To overcome such resistance gene stacks, simultaneous mutations in several effector genes would be required in one single pathogen spore. Race-non-specific resistance is supposed to be more durable when deployed in agriculture. Such kind of resistance mechanism sometimes may also be effective against multiple pathogens. These are normally quantitative traits conferring partial resistance that is able to slow down disease development. For example *Lr34*, *Yr36*, and *Pm21*. *Lr34* confers non-specific, partial, and slow rusting resistance, and has been deployed worldwide, maintaining its effectiveness in agriculture for decades. Due to its role in conferring resistance to pathogens other than leaf rust, it is also known as *Yr18, Pm38, Sr57* and *Bdv1* for resistance to stripe rust, powdery mildew, stem rust, and barley yellow dwarf virus, respectively [64]. The successful cloning of *Lr34, Yr36, and Lr67* revealed these APRs encode an ABC transporter, a kinase-START protein, and a hexose transporter, respectively (**Table 2**). They appear to each have their own resistance mechanism, function

S.no	Gene	Biotic stress	Protein type	Reference
1.	*Lr21*	Leaf rust	NLR	[33]
2.	*Lr10*		NLR	[34]
3.	*Lr1*		NLR	[35]
4.	*Lr34/Yr18/Sr57/Pm38*		ABC[1] transporter	[36]
5	*Lr67/Yr46/Sr55/Pm46*		Hexose transporter	[37]
6	*Lr22a*		NLR	[38]
7	*Yr36/WKS1*	Stripe/yellow rust	Kinase-START[2]	[39]
8	*Yr7*		NLR	[40]
9	*Yr5a*		NLR	[40]
10	*Yr5b*		NLR	[40]
11	*Yr15*		Tendem kinase-pseudokinase	[41]
12	*YrAS2388*		NLR	[42]
13	*Sr33*	Stem rust	NLR	[43]
14	*Sr35*		NLR	[44]
15	*Sr50*		NLR	[45]
16	*Sr22*		NLR	[46]
17	*Sr45*		NLR	[46]
18	*Sr13*		NLR	[47]
19	*Sr21*		NLR	[48]
20	*Sr46*		NLR	[49]
21	*SrTA1662*		NLR	[49]
22	*Sr60/WTK2*		Tendem kinase	[50]
23	*Sr26*		NLR	Zhang et al. (under review)
24	*Sr61*		NLR	Zhang et al. (under review)
25	*Pm3*	Powdery Mildew	NLR	[51]
26	*Pm8*		NLR	[52]
27	*Pm2*		NLR	[53]
28	*Pm21*		serine/threonine protein kinase	[54, 55]
29	*Pm60*		NLR	[56]
30	*WFhb1–1 (Qfhb1)*	Fusarium head blight (FHB)	PFT[3]- chimeric lectin	[57, 58]
31	*H13*	Hessian fly	CC-NB-ARC-LRR	[59]

[1]*ABC- ATP binding cassette.*
[2]*START- Steroidogenic acute regulatory protein-related lipid transfer domain.*
[3]*PFT- Pore-forming toxin.*

Table 2.
List of major cloned resistance genes in wheat for different biotic stresses.

constitutively and often increase the basal level of resistance of the host, which is different from the recognition based NLRs.

6. Insect resistance in wheat

6.1 Resistance categories

Responses which govern insect resistance in plants can be classified into three categories. Tolerance can be defined as the response of plant which allows the plant to survive insect damage with low or no damage to the yield. Tolerance is generally governed by a complex set of genetic traits. Tolerance does not affect the overall survival of insects thereby poses no selection pressure. Tolerance has been reported in a number of crops [65, 66]. The non-preference of a plant by insect pest or antixenosis is another mechanism used by plants against insects. Generally, antixenosis is manifested by some morphological or chemical factors which hinder feeding of the pest and sometimes rejection as host. Antibiosis, the third category, can be defined as the condition when pest health and reproduction are negatively affected by the resistant plant. Most of the resistance observed in field (up to 90%) is due to antibiosis.

6.2 Resistance mechanisms

Over the due course of evolution traits for direct and indirect defense mechanisms against insect attacks have developed in plants. The classification of these mechanisms has been further done as direct mechanisms and indirect mechanisms. Structural barriers constitute the direct defenses. Tissue toughness, glandular and non-glandular trichomes and plant pubescence are included in these types of defenses. Allelochemicals in plant tissues are also included in direct defenses. These exhibit toxic, anti-feedant, and repellent effects on the attacking arthropods. The digestive enzyme inhibitors, cyanogenic glycosides, glucosinolates, lectins, glucosinolates, terpenoids and alkaloids are involved in this [67, 68]. An extensive review of constitutive & induced morphological & chemical plant defenses has been done [65, 66, 69, 70]. These defenses mediate antixenosis & antibiosis. Volatile organic compounds constitute the indirect defenses. The plants which are damaged by pest arthropod release these compounds. These compounds lead to attraction of arthropod predators & parasitoids or the ones that cause repelling of oviposition of pest arthropods [71]. The specific plant indirect defense responses are represented by herbivore associated molecular patterns (HAMPs). These are the responses to the specific herbivore derived elicitors. This occurs in the in oral or ovipositor secretions. These facilitate indirect defenses against herbivores [72]. The widely researched HAMPs are the insect fatty acid plant amino acid conjugates. These are obtained from the lepidopterous larvae [71, 73].

6.3 Constitutive and induced resistance genes

The arthropod selects host plant tissue substrate based on well-coordinated interactions occurring within evolutionarily conserved protein(s) which are encoded by attacking arthropod & responding host plant. The arthropod successfully manipulates the host plant as a result of suitable arthropod-plant interactions. When there is incompatibility in the arthropod-plant interaction, the arthropod does not succeed resulting in the survival of the plants attacked [74]. The plant and

fungal endophytic genes are expressed in both the interactions. These are expressed constitutively or via induced defense responses. These occur following herbivory and find involvement in arthropod resistance [75, 76].

Under field conditions, resistance has been explained more clearly by the effects which are controlled by the constitutive genes. This is concluded based on the limited research done till date. The effects owing to the induced gene expression do not contribute much in this [77, 78]. The generation of reactive oxygen species and the signal cascades which involve salicylic acid (SA), jasmonic acid (JA), abscisic acid, ethylene and gibberellic acid occurs in plants as a response to arthropod herbivory. Direct and indirect defense proteins are resulted by the downstream production [79–82]. The aphid bacterial endosymbionts could also lead to defense signals [83]. Jasmonic acid based transcriptomes are elicited by the plant tissue damage caused by arthropods with chewing mouthparts. On the contrary, arthropods with piercing-sucking mouthparts induce the jasmonic acid- salicylic acid-based transcriptomes [71]. Recent documentation has been done of the jasmonic acid- salicylic acid signaling induced by both types of herbivory and jasmonic acid- salicylic acid cross talk [68, 74, 84, 85]. The expression of several plant genes which are produced in the initial responses to arthropod herbivory are controlled by the JA, 12- oxo-phytodienoic acid, and jasmonoyl-amino acid conjugates (which are governed by zinc finger protein expressed in inflorescence meristem) repressor proteins [86]. Several defense allelochemicals are produced by the defense response gene upregulation. This occurs via JA and some other pathways [69]. Scanty information is available regarding the arthropod induced expression of the plant metabolism genes. There are very few evidences indicating the down regulation of few of these genes. This is reported to occur in the beginning just after the arthropod herbivory sets in and later on upregulated in the ensuing days [84, 87].

The identification of arthropod pest elicitors of resistance genes is yet to done. An undefined elicitor protein of *Diuraphis noxia* is recognized by the wheat plant receptors. *D. noxia* is recognized by plant-signaling gene products feeding in incompatible interactions [88]. Secondary metabolites possessing the Hydroxamic acids (Hx) (1,4-benzoxazin-3-ones) group, find involvement in the resistance of certain cereals against bacteria, fungi and several insects including aphids [89]. In the seed, Hydroxamic acids (Hx) are absent. This increases after germination. The young seedlings exhibit the concentration peak [90]. This is basically located in the mesophyll protoplasts, the vascular bundles [91] and in the sieve elements [92]. In the mature plants, the Hx levels decline after the seedling stage. Even then, the young tissue still exhibits a high concentration of Hx [90]. In the plants, the Hx compounds occur as 2-β-O-D-glucopyranosides [90]. When the tissue is injured, these are enzymatically hydrolyzed by endo-β-glucosides to DIMBOA (2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3- one) [92]. DIMBOA is the main Hx aglucone in the wheat extracts. It leads to antibiosis, decreased performance, feeding deterrence and reduced reproduction in aphids [93].

An enhancement in the overall activity of several enzymes was observed. All the enzymes such as superoxide dismutase, phenylalanine ammonia lyase glutathione reductase, and polyphenol oxidase have a major role in the defense of plants towards the feeding of aphid [94]. An early defense strategy is mounted by the Hessian fly-resistant *Ae. tauschii*. The production of anti-feedant proteins (lectins), secondary metabolites and ROS radicals is involved in this strategy. These successfully counter the larval extra oral salivary plant cell degrading proteases, lead to fortification of the cell wall and prevention of the Hessian fly larvae from establishing permanent feeding sites [95].

There are different types of carbohydrate binding proteins known as lectins which are present in tissues of plants. Resistance building potential is possessed by these lectins for wheat against insects. To tackle HF, the identification of

genes leading to production of this type of lectins seems a potential method. The genes include Hfr-2 called as HF destructor. This is expressed in the leaf sheaths of the resistance genotypes [96] On similar lines, the mannose binding lectins serve as storage protein and accumulate in the phloem sap. This might act against HF. Anti-insect properties are possessed by these lectins. This is attributed to the accumulation of lectin in the midgut of insects, killing them instantly. Another defensive mechanism present in resistant varieties of wheat is the production of Wci-1 mRNAs and Hfr-1. This occurs in response to the attack of HF larvae. The Hfr-1 gene is known as the defender gene against HF. It has the ability to control crop from severe attack [97]. The identification of arthropod pest elicitors of resistance genes is yet to done. An undefined elicitor protein of *Diuraphis noxia* is recognized by the wheat plant receptors. *D. noxia* is recognized by plant-signaling gene products feeding in incompatible interactions [88]. An Avirulence (*Avr*) gene is there on the parasites side. This encodes one of the several effector proteins that the parasite applies to the plant to help in colonization. A Resistance (R) gene is there on the plant's side. It mediates a surveillance system which detects the Avr protein. The detection is done either directly or indirectly. It triggers effector-triggered plant immunity. The arthropods are responsible for a significant proportion of plant biotic stress but even then they have not been integrated into important models of plant immunity that arise from plant pathology. The absence of molecular evidence for arthropod Avr effectors has been a limiting factor. This evidence was discovered in a plant pathogen around thirty years back. Now, there is evidence for arthropods with the cloning of the Hessian fly's vH13 *Avr* gene. Resistance against RWA is supposed to be induced by gene-for-gene model. The resistant gene produces a protein in this mechanism. This protein contains nucleotide binding site-leucine rich repeat (NBSLRR) domain [98, 99]. Firstly, this NBSLRR domain recognizes and then interacts with cognate Avr protein which is produced by the respective insect [100]. It has been reported that another domain (serine/threonine-protein kinases: STKs) is produced by *Dn* genes. This confers resistance against the RWA [101].

7. Sources of biotic stress resistance in wheat

Wheat belongs to the kingdom Plantae and family Poaceae. It is a long day and a self-pollinated crop. The bread wheat (*Triticum aestivum*) genome is one of the most challenging plant genomes to study. It is highly repetitive (~85%) and approximately 15.4–15.8 Gbp in size, which is five times larger than the human genome [102]. The genus Triticum contains 10 species, out of which six are cultivated and four are wild. Hexaploid wheat (*T. aestivum*) genome (2n = 6x = 42) encompasses A, B and D sub-genomes which is advantageous for providing useful genetic diversity for crop improvement. There are three ploidy levels in *Triticum* and *Aegilops* (encompassing cultivated wheats and their progenitors) genera with 2n chromosomes 14, 28, 42 and the basic chromosome x = 7 in all the species. Other genera of Poaceae such as Secale, Hordeum, Dasopyrum, Agropyron, Elymus, Leymus, Elytrigia, and Thinopyrum are also important for introgression of useful variability into cultivated wheats. On the basis of their genomic constitution, the wild relatives of wheat can be classified into primary, secondary, and tertiary gene pools [103, 104]. These gene pools are affluent source of genes for disease and pest resistance, mitigating abiotic stresses and micronutrient enrichment in wheat. These three gene pools of wheat as sources of resistance can be described as follows:

1. The primary gene pool consists of species sharing homologous genomes with cultivated wheat. This group includes land races of *T. aestivum*, *T. turgidum* and donor species of the A and D genomes of bread wheat-*T. monococcum, T. urartu, T. boeoticum and Ae. tauschii*. Gene transfer from these species can be achieved by direct hybridization, backcrossing, and selection [104]. Just embryo rescue in certain cases is necessary to produce F_1 hybrid. Many genes conferring resistance to diseases and insect pests have been transferred using this method and several of them are still being exploited in cultivar improvement [105, 106]. Among genetic resources, landraces has been reported a crucial germplasm pool contributing to the genes for grain yield [107, 108] high protein content and tolerance to biotic/abiotic stresses [109]. The green revolution semi-dwarfing genes (*Rht- B1b* and *Rht-D1d*) [110] and other semi-dwarfing gene, *Rht8c*, has been a significant contribution of the landraces. The *Rht* dwarfing gene that was available through the Japanese variety 'Norin10' originating from a Japanese landrace Shiro Daruma [111]. Later, these dwarfing genes were utilized by Dr. Norman E. Borlaug to develop the high-yielding semi-dwarf wheat varieties triggering the Green Revolution in late 1960s. At Punjab Agricultural University (PAU), Ludhiana, India, an active collection of 280 *Ae. tauschii* accessions is being maintained. These accessions have been found to carry resistance genes for various biotic stresses including leaf rust, stripe rust, powdery mildew, and Karnal bunt. Ae. tauschii has a very high level of KB resistance.

2. The secondary gene pool of bread wheat includes the polyploid *Triticum* and *Aegilops* species that have at least one genome in common with wheat. Gene transfer from these species by homologous recombination is possible, if the target gene is located on a homologous chromosome. However, if the genes are present in a non-homologous genome, special cytogenetic manipulations are required. These species have contributed many resistance genes that are being used in cultivar development [103]. At PAU, the genes for disease resistance and HMW glutenin subunits have been successfully transferred from several *Triticum* and *Aegilops* species into wheat and durum cultivars with direct hybridization and backcrossing [112, 113].

3. Species belonging to the tertiary gene pool are more distantly related. Their chromosomes are not homologous to those of wheat. Gene transfer from these species cannot be achieved by homologous recombination, chromosome pairing, and recombination between wheat chromosome and alien chromosomes [103, 104]. Special cytogenetic techniques (in-situ hybridization) are required to ensure compensating transfers with least linkage drag for commercial exploitation of introgressed derivatives. Even though such transfers may include an entire chromosome arm or part of an arm, these have been successfully bred into commercial wheat cultivars because the alien chromosome segment genetically compensates for the missing wheat segment.

8. Major techniques for inducing biotic stress resistance

The route maps followed for a trait improvement particularly stress resistance, both biotic and abiotic remain the same. The **Figure 1** graphically depicts various tools and techniques that can be utilized with efficient and effective manner for tackling different biotic stresses in wheat.

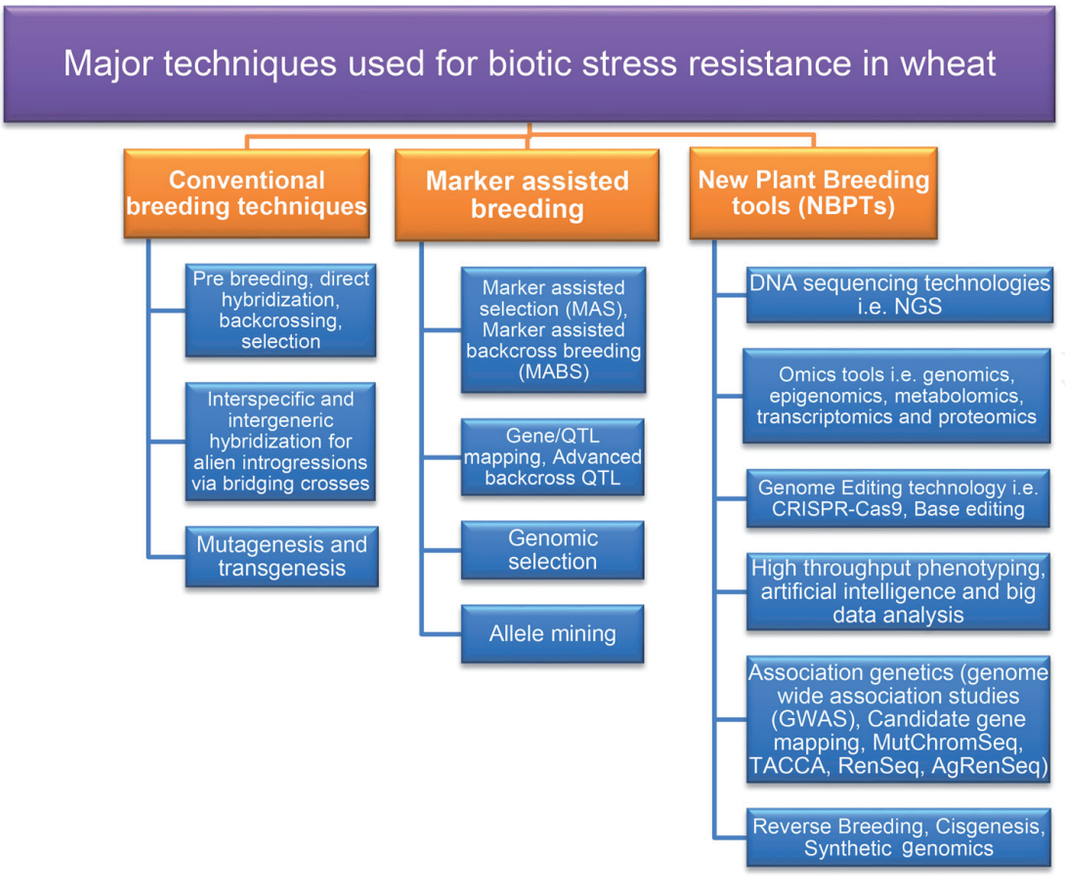

Figure 1.
Some major tools and techniques (both in use and under exploration) in wheat breeding for biotic stress resistance.

9. Present scenario

9.1 Fungal diseases

So far, more than 240 rust resistance genes have been characterized and formally designated in wheat or its relatives; most being race-specific resistance genes. At least 67 of these genes are designated as *Sr* resistance genes [105, 114, 115]. *Sr31* was one of the most widely utilized race-specific *Sr* resistance genes [116]; however, its presence at the International Maize and Wheat Improvement Center (CIMMYT) has been drastically reduced following testing against Ug99 races in Kenya. Evolution of virulence against *Sr31* with the emergence of Ug99 led to stem rust susceptibility in most of the wheats grown around the globe. After its new races overcame a number of resistance genes, the genes *Sr2*, *Sr23*, *Sr25*, *Sr33*, *Sr35*, *Sr45*, *Sr47*, and *Sr50* are presently the most efficient for protection against newly evolved races [117]. The QTL-controlling stripe rust resistance in *T. monococcum* was mapped on chromosome 2A (*QYrtm.pau-2A*), whereas the QTL from *T. boeoticum* was mapped on 5A (*QYrtm.pau-5A*). One stripe rust-resistant gene from *T. boeoticum* acc. pau5088 was confirmed to be introgressed in cultivated wheat which was indicated by co-introgression of *T. boeoticum* sequences linked to stripe rust-resistant QTL, *QYrtb.pau-5A* [118].

For stripe (yellow) rust resistance, 95 genes have been characterized and formally named [105, 114, 115]. However, most of these genes have been rendered ineffective with emergence of virulent races around the globe with exception of a few combinations, such as the combination of *Yr5* and *Yr15* that remain effective worldwide. At Punjab Agricultural University, Ludhiana, India, about 200 accessions of *T. monococcum* and *T. boeoticum* were screened for leaf rust and stripe rust

resistance for several years and we found that all the *T. monococcum* accessions, most of the *T. boeoticum* and a few *T. urartu* accessions, were completely resistant to leaf rust. However, a lot of variation was observed for stripe rust resistance. Leaf and stripe rust resistance genes have also been introgressed from diploid species *Ae. umbellulata* and *Ae. caudata* using *T. durum* as bridging species [118, 119].

Similarly, 80 *Lr* resistance genes have been genetically characterized and documented [115]. Out of these, *Lr1*, *Lr3*, *Lr10*, and *Lr20* have been commonly deployed in wheat cultivars [120]. Generally, ASR genes are rendered ineffective with continual emergence of new virulent races of rust pathogens through mutation and recombination [121]. It has been well documented through cloning of 11 race-specific genes in wheat (*Sr22*, *Sr33*, *Sr35*, *Sr45*, *Sr50*, *Yr5*, *Yr10*, *Lr1*, *Lr10*, *Lr21*, and *Lr22*) that these genes encode NLR proteins [122–126].

Till date, only seven race non-specific APR genes have been genetically characterized and formally designated in wheat namely *Sr2*/*Yr30*, *Lr34*/*Yr18*/*Sr57*/*Pm38*, *Lr46*/*Yr29*/*Sr58*/*Pm39*, *Lr67*/*Yr46*/*Sr55*/*Pm46*, *Lr68*, *Sr56*, and *Yr36* [127–133]. Cloning of the APR genes *Yr36*, *Lr34*/*Yr18*/*Sr57*/*Pm38* and *Lr67*/*Yr46*/*Sr55*/*Pm46* has revealed the roles of cytoplasmic protein kinase, adenosine triphosphate (ATP)-binding cassette transporter, and hexose transporter, respectively in mediating resistance [134–136].

Growing resistant cultivars is the most cost-effective strategy for tackling PM. To date, 70 PM resistance genes have been formally cataloged; most of these provide race-specific resistance in wheat [114, 115]. It is desirable to know the virulence pattern of isolates to generate effective combinations of race-specific resistance genes [137]. More effective method would be deployment of combinations of race non-specific resistance genes is a promising method. As discussed above in the section for rust resistance, only three race non-specific resistance genes have been identified, out of which two pleiotropic genes (*Lr34*/*Yr18*/*Sr57*/*Pm38* and *Lr67*/*Yr46*/*Sr55*/*Pm46*) have been cloned [135, 136].

Genetic resistance to FHB is mainly quantitative and is controlled by multiple moderate to minor genes [138]. Although genetic resistance is the most cost-effective method, it is hard to accomplish in commercial cultivars due to its complex behavior. This complexity is further enhanced by various resistance mechanisms, e.g., invasion (type I), fungal spread (type II), toxin accumulation (type III), kernel infection (type IV) and yield reduction (type V) [139]. FHB resistance also displays significant correlations with heading, plant height, and anther extrusion of the wheat plant [140]. To date, seven genetic loci designated as *Fhb1*, *Fhb2*, *Fhb4* and *Fhb5* from wheat, and *Fhb3*, *Fhb6* and *Fhb7* from wild relatives, have been formally named as FHB resistance genes [141]. The cultivars Sumai 3 from China and Frontana from Brazil have been identified as sources of moderate resistance to FHB.

Karnal bunt is among the few quarantine diseases that restrict free trade among countries due to quarantine regulations [142]). Resistance to Karnal bunt has been reported in durum wheat (*Triticum turgidum*), common wheat, *Aegilops*, rye and barley under artificial conditions [143, 144]. Susceptibility of *T. aestivum* to Karnal bunt might be due to presence of an additional D genome [145, 146]. Sharma et al. [147] at PAU developed high yielding Karnal bunt resistant wheat lines by introgression of Karnal bunt resistance from KBRL 22 into the background of high yielding PBW343. Studies on deciphering genetics of resistance have indicated the presence of quantitative rather than qualitative resistance [145, 146, 148]. Fuentes-Davila et al. [145] suggested six genes, designated Kb1, Kb2, Kb3, Kb4, Kb5, and Kb6, while Villareal et al. [149] postulated a minimum of three genes for resistance. Studies on deciphering genetics of resistance have indicated the presence of quantitative rather than qualitative resistance [145, 148].

For loose smut, the majority of genetic studies carried out thus far have demonstrated simple inheritance with one, two or three major genes in hexaploid wheat

controlling resistance to several races of *U. tritici*. The first four loose smut resistance genes *Ut1* to *Ut4* were named based on segregation of avirulence in *U. tritici* [150, 151]. Genes *Ut1* and *Ut3* have no chromosome assignment. Based on pedigree, the gene symbol *Ut2* was assigned to the resistance gene on chromosome 6A to race T19 [152]. *Ut4* associated with the Thatcher derived differential line TD12A, was located on chromosome 7B [153, 154]. *Ut5* was located on chromosome 2BL [155], *Ut6* was initially reported on chromosome 5B by Kassa et al. [156] which was later validated by Knox et al. [153]. A gene located to chromosome 7A by Dhitaphichit et al. [157] was subsequently named *Ut7* [153]. Knox et al. further identified genes *Ut8* on chromosome 3A, *Ut9* on chromosome 6B and *Ut10* on chromosome 6D. Several studies revealed the additive nature of resistance genes, while in some cases, duplicate complementary action of multiple genes was also implicated [158].

Finally, the genetic resistance to wheat blast at the seedling stage follows a gene-for-gene interaction model [159] and five resistance genes namely *Rmg2, Rmg3, Rmg7, Rmg8*, and *RmgGR119* have been identified in wheat against the *Magnaporthe oryzae* pathotype *triticum* [160–164].

Various molecular markers have been widely used to tag and map resistance genes in wheat; however, SSRs have emerged as the choice of marker in gene-mapping studies. These markers can be strategically used for selection of desirable gene combinations along with phenotypic assays. Wheat has more than 3000 SSR markers mapped so far [165]. Molecular markers can be used for alien gene transfers and understanding the mechanism of gene transfer. Such markers ensure selection of a target gene based on the presence of the linked genotype. The success of selection depends on the close genetic association and robustness of a given marker across different genetic backgrounds. At PAU, a number of genes/QTLs have been mapped for different wheat diseases including stripe rust, cereal cyst nematode, and Karnal bunt. Two QTLs, one each in *T. monococcum* acc. pau14087, and *T. boeoticum* acc. pau5088, were detected for resistance in the RIL population. The QTL in *T. monococcum* mapped on 2A in a 3.6 cM interval between *Xwmc407* and *Xwmc170*, whereas the QTL from *T. boeoticum* mapped on 5A in 8.3 cM interval between *Xbarc151* and *Xcfd12* [166–168].

9.2 Insect-pests

In the last 50 years or so, the HPR concept has been extended to insect-host interactions. As a result, insect resistant cultivars are now in the picture. The variables, both biotic and abiotic which play a major role in deciding the plant reaction to pest, along

Insect-pest	Order	Gene(s)	Category	References
Aceriatosichella	Acari	*Cmc*(4)	Ab	[169]
Cephuscinctus	Hymenoptera	*Qssmsub* (2); QTL	Ab, Ax, Tol	[170, 171]
Diuraphisnoxia	Hemiptera	*Dn* (10); QTL	Ab, Ax, Tol	[172, 173]
Mayetiola destructor	Diptera	*H* (>33)	Ab	[174]
Schizaphis graminum	Hemiptera	*Gb* (>10); QTL	Ab, Ax, Tol	[175]
Sitodiplosis mosellana	Diptera	*Sm* (1); QTL	Ab	[176, 177]

Ab: antibiosis; Ax: antixenosis; QTL: quantitative trait loci; Tol: tolerance.

Table 3.
Genes identified for insect resistance in wheat and their respective categories.

with mechanisms and categories of resistance are now better understood. Drawing analogy from plant-pathogen interactions, pest-host relationships are now being viewed as (susceptible plant) and incompatible (resistant plant) interactions [74].

Deployment of insect resistance genes in wheat along with other field crops has increased steadily over the years from mid 60s. Marker assisted selection (MAS) and breeding has sped up the process of identification of resistance loci and QTLs and understanding of the mechanisms governing the resistance. **Table 3**. depicts the genes identified for insect resistance in wheat and their respective categories.

10. Key challenges

Wheat is an allopolyploid resulted from interspecific or intergeneric hybridization of two or more genomes from different species. Being one of the most consumed and cultivated crop globally, it is under continuous exposure to a large variety of parasite species and strains, many of which have the ability to move around the globe. Long-term co-evolution between plants and their pathogens has equipped plants with a sophisticated multi-layered immune system to guard themselves against pest and pathogens [178]. Despite this, there are a few important challenges which are required to be addressed for effectively mitigating with different biotic stresses in wheat:

1. New strains of pathogens like the rusts continue to evolve rapidly. It is well documented that the rust pathogens have great pathogenic variability and the frequent emergence of new virulent strains that overcome resistance genes present in cultivated wheat varieties has hindered efforts to achieve durable resistance to these pathogens.

2. The complex nature of plant–parasite interactions can be overwhelming while breeding for disease resistance in wheat. The standard models of plant pathology i.e. gene for gene model and the expanded model of plant immunity do not elucidate plant immunity and parasite adaptation explicitly in such natural interactions.

3. The bread wheat (*Triticum aestivum*) genome is one of the most challenging plant genomes to study. It is highly repetitive (~85%) and approximately 15.4–15.8 Gbp in size [179]. Much of the desirable genetic diversity is present in the wild relatives of wheat, both in progenitors and non-progenitor species. The genomic complexity of bread wheat and various hybridization barriers hinder the potential use of resistance alleles present in that germplasm.

4. Despite the versatility of transgenic technology with unlimited scope for application in wheat resistance breeding, it has faced increasing public dissent especially against its use in food crops. Other issues include rigorous risk assessments of crop, which are time-consuming and cost-intensive. Such modifications lead to integration of transgenes randomly into plant genomes along with their selection marker genes. Due to which, there is a possibility of pleiotropic effects, potential silencing and varied gene expression in modified plants

5. Traditional map based/positional cloning is not viable for target genes derived from wild relatives of wheat and which are located in introgressed genome segments that do not recombine with wheat chromatin. Applying this strategy on genes that are located in centromeric regions is also extremely challenging (low recombination rates there).

6. The foremost challenge in breeding against insect pests is finding sources with reasonable levels of resistance against the pest. Secondly another major hurdle is the difference between resistance at field and protected conditions, since evaluation is carried out in protected conditions, results vary when evaluation is carried out *in vivo.* Lack of efficient evaluation and selection tools against insects also hinders the insect resistance breeding. Finally, transfer of resistance is often accompanied by linkage drag which sometimes becomes cumbersome to break.

11. Conclusion and future prospects

Genetic control is considered as the most effective and environmentally friendly strategy to control rust disease and involves breeding effective disease resistance genes into wheat cultivars. Many rust resistance genes have been identified genetically, and introgression into wheat lines is increasingly being facilitated by the development of robust molecular markers. However, the massive and complex genome of wheat poses major challenges for the isolation of individual genes. As revealed by the increasing number of newly available whole genome sequences and the more precise bioinformatic pipelines developed for identifying NLR genes, the number of NLR genes varies greatly between species. Based on an analysis of the IWGSC RefSeq v1.0 assembly, a total of 3,400 full-length NLR loci have been documented [180]. The approaches for identifying effective resistance genes therefore, must consider both classical R-genes (immune receptor class genes) as well as other novel classes that may operate via different mechanisms.

Cloning of the genes that controlling resistance to rust pathogens will significantly advance our understanding of the molecular basis underlying expression of disease resistance in wheat. Only a small number of rust resistance genes have been cloned and had their molecular functions studied (**Table 2**). To overcome the limitations of the map-based cloning strategy in the large genome of wheat, alternative approaches were developed and validated by the rapid cloning of several genes using Target-sequence Enrichment and Sequencing (TEnSeq) pipelines. These include MutRenSeq (Mutagenesis and the Resistance gene Enrichment and Sequencing), AgRenSeq (Association genetics with R gene enrichment Sequencing), MutChromSeq (Mutgenesis Chromosome flow sorting and short-read Sequencing), and TACCA (Targeted Chromosome based Cloning via long-range Assembly). The common component among all these approaches is the intent to reduce the genome complexity prior to the use of next generation sequencing (NGS). Such insight into the molecular mechanisms will be the foremost step towards the functional characterization of the wheat-rust interaction and allow engineering of new resistance by exploiting novel techniques like allele mining and genome editing. Also, approaches like TILLING (Targeting Induced Local Lesions IN Genomes) can be adopted for more precise and efficient characterization of the function of targeted wheat genes for different fungal and bacterial diseases.

The rich genetic diversity available in wheat is a source of numerous novel alleles for both disease resistance and tolerance to abiotic stress. However, there is still a huge gap in characterization of the available genetic resources and their utilization in breeding programs. Over the years, traditional breeding strategies have successfully incorporated novel alleles into elite germplasm, which has significant impacts on production globally. Use of advanced technologies, marker-assisted selection (MAS), genomic selection, transgenics and genome editing will help to increase the efficiency of wheat breeding for biotic stress resilience around the world.

To escape the boom and bust cycle, resistance gene stewardship and deployment strategies such as gene pyramiding, gene stacking (transfer of gene cassettes) could

prove to be effective against deadly diseases of wheat (rusts, blight). It is widely reported and agreed upon fact that the most effective and durable means for genetic control of wheat rusts is the use of combinations of multiple broadly effective ASR and APR genes. Using this, the desirable combinations of effective resistance genes can be combined and transformed into wheat as gene cassettes or stacks. This can result in faster improvements in disease resistance of current high-yielding varieties. Also, the advancements in R-gene cloning pipeline like TEnSeq will provide many more tools for MAS in wheat breeding as well as the raw gene sequences to pursue gene stacking (via transgenic gene cassettes). Combining with advances in identifying genetic variation in rust *Avr* genes, these new tools will lead to more effective deployment strategies to maximize resistance durability.

Genomic selection (GS) is considered one of the best strategies for selection of multiple minor-effect loci in comparison with MAS. Using GS, a training population (after phenotyping and genotyping) is used to standardize a prediction model, which is further used to predict breeding values, thus enabling selection of candidates prior to phenotyping [181]. Recent studies have reported that greater genetic gains can be obtained by using genomic selection than by using MAS [182] and phenotypic selection [183].

More recently, genome editing has emerged as a prominent new plant breeding technique, which involves targeted modification of a native DNA sequence. For instance, it has been observed that a single amino acid substitution (Arg144Gly) in a hexose transporter in wheat results in the gene *Lr67* conferring resistance. This substitution evolved recently after common wheat polyploidization. Introduction of the *Lr67* transgene into barley conferred seedling and adult plant resistance to the barley leaf rust pathogen [184, 185]. The orthologue sequence of *Lr67* exists in the barley genome; hence altering the Arg144Gly by genome editing would be expected to produce resistance to rust in barley. Similarly, a number of homologs/orthologues of the isolated genes exist in related species. Isolating a rust resistance gene from other related species thus can provide deeper insight into rust resistance in the wheat.

Therefore, under a changing global climate, it is of paramount importance to breed for durable and broad-spectrum disease resistance in wheat at a faster pace to reduce losses from attack by rapidly evolving new virulent pathogenic races. Moreover, this would lead to reduction of the use of agrochemicals (fungicides), escaping environmental and human health hazards, an essential component of modern sustainable crop production systems.

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References

[1] Gupta P K, Mir R R, Mohan A and Kumar J. Wheat Genomics: Present Status and Future Prospects. International Journal of Plant Genomics Volume 2008. p. 1-36

[2] Bertholdsson NO. Early vigor and Allelopathy- Two useful traits for enhancing barley and wheat competitiveness against weeds. Weed Research. 2005;**45**:94-102

[3] Figueroa M, Hammond-Kosack KE, Solomon PS. A review of plant diseases—a field perspective. Molecular Plant Pathology. 2017;**19**:1523-1536

[4] Weise MV, editor. Compendium of wheat diseases. 2nd ed. American Phytopathology Society,St. Paul 1987

[5] Wellings CR. Global status of stripe rust: a review of historical and current threats. Euphytica 2011;**179**:129-141

[6] Plank J. E. van der. Plant diseases: epidemics and control. 1963. New York: Academic. 349 pp

[7] Priyamvada, Saharan M S, Tiwari R. Durable resistance in wheat. International Journal of Genetics and Molecular Biology. 2011; **3(8)**:108-114

[8] Hovmøller SM, Sørensen CK, Walter S, Justesen A F. Diversity of Puccinia striiformis on Cereals and Grasses. Annual Review Phytopathology.2011; **49**:197-217

[9] Singh R P, Hodson DP, Huerta-Espino J et al. The 61 Emergence of Ug99 Races of the Stem Rust Fungus is a Threat to World Wheat Production. Annual Review Phytopathology. 2011; **49**:465-482

[10] Singh RP, William HM, Huerta-Espino J, Rosewarne G. Wheat rust in Asia: meeting the challenges with old and new technologies. New dimensions for a diverse planet. In: Proceedings of the 4th International Crop Science Congress, 26 Sep–1 Oct 2004, Brisbane

[11] Rizwan S, Ahmad I, Ashraf M, Mirza JI, Sahi GM, Rattu AR, Mujeeb-Kazi A. Evaluationof synthetic hexaploid wheats and their durum parents for stripe rust resistance. Rev MexFitopatol 25:152-160

[12] Roelfs AP, Singh RP, Saari EE. Rust diseases of wheat: concepts and methods of diseasemanagement. CIMMYT, Mexico DF. http://hdl.handle.net/10883/1153

[13] Singh RP, Hodson DP, Huerta-Espino J, Jin Y, Njau P. Will stem rust destroy theworld's wheat crop? Advances in Agronomy. 2008;**98**:271-309

[14] Cruz CD, Valent B. Wheat blast disease: danger on the move. Tropical Plant Pathology. 2017;**42(3)**:210-222

[15] Islam MT, Croll D, Gladieux P. Emergence of wheat blast in Bangladesh was caused by a South American lineage of *Magnaporthe oryzae*. BMC Biology. 2016; **14:**84

[16] Tembo B, Mulenga RM, Sichilima S, Msiska KK, Mwale M, Chikoti PC. Detection and characterization of fungus (*Magnaportheoryzae pathotype Triticum*) causing wheat blast disease on rain-fed grown wheat (*Triticum aestivum* L.) in Zambia. Plosone. 2020;**15**(9): e0238724

[17] Mitra M. A new bunt on wheat in India. Annals of Applied Biology. 1931; **18**:178-179

[18] Rush CM, Stein JM, Bowden RL, Riemenschneider R, Boratynski T, Royer MH. Status of Karnal bunt of wheat in United States 1996-2004. Plant Disease. 2005;**89**:212-222

[19] Dhaliwal HS, Singh H, Singh KS, Randhawa HS. Evaluation and cataloguing of wheat germplasm for disease resistance and quality. In: Damania AB ed. Biodiversity and wheat improvement. Wiley, London,1993. pp 123-140

[20] Dhaliwal HS, Singh H. Breeding for resistance to bunts and smuts: Indian scenario. In:Proceedings bunts and smuts of wheat: an international symposium. North Carolina, North American Plant Protection Organization, Ottawa 1997, pp 327-347

[21] Katare S, Singh B,Patil SD, Tiwari R, Jasrotia P, Saharan MS, Sharma I. Evaluation of new insecticides for management of foliar aphid complex in wheat. Indian Journal of Entomology 2015;79(2):185-190.

[22] Kieckhefer RW, Gellner JL. Yield losses in winter wheat caused by low-density cereal aphid populations. Agron J 1992; 84:180-183.

[23] Kieckhefer R W, Gellner, JL, Riedell WE. Evaluation of the aphid-day standard as a predictor of yield loss caused by cereal aphids. Agronomy J 1995;87(5):785-788.

[24] RyanJD, Dorschner KW, Eikenbary RD, Johnson RC. Drought/ greenbug interactions: photosynthesis of greenbug resistant and susceptible wheat. Crop Sci.1987;27:283-288.

[25] BurtonRL. Effect of greenbug (Homoptera: Aphididae) damage on root and shoot biomass of wheat seedlings. J. Econ. Entomol.1986; 79633636

[26] Capinera JL, editor. Encyclopedia of entomology, 2nd ed. Springer, Netherlands; 2008. 4242 p.

[27] Beres BL, Dosdall LM, Weaver DK. Biology and integrated management of wheat stem sawfly and the need for continuing research. Can Entomol. 2011;143:105-125. DOI: 10.4039/n10-056

[28] Andersen E J, Ali S, Byamukama E, Yen Y, Nepal P. Disease resistance mechanisms in plants. Genes. 2018;**9**:339.

[29] Flor H H. Current status of the gene-for-gene concept. Annual Review of Phytopathology. 1971; **9**:275-296

[30] Jones JDG, Vance RE, Dangl JL. 2016. Intracellular innate immune surveillance devices in plants and animals. Science. 2016;**354:**aaf6395-1-8

[31] Dodds PN, Rathjen JP. Plant immunity: towards an integrated view ofplant–pathogen interactions. Nature Reviews Genetics. 2010;**11**:539-548

[32] Singla J,Krattinger S G.Biotic Stress Resistance Genes in Wheat. Encyclopedia of food grains. 2016; 388-392

[33] Huang L, Brooks S A, Li W, Fellers J P, Trick H N, Gill B S. Map-based cloning of leaf rust resistance gene Lr21 from the large and polyploid genome of bread wheat. Genetics. 2003;**164**:655-664.

[34] FeuilletC, Travella S, Stein N, Albar L, Nublat A, Keller B. Map-based isolation of the leaf rust disease resistance gene Lr10 from thehexaploid wheat (*Triticum aestivum* L.) genome. Proc. Natl. Acad. Sci. U.S.A. 2003;**100**:15253-15258.

[35] Cloutier S, McCallum B D, Loutre C, Banks T W, Wicker T, Feuillet C (2007). Leaf rust resistance gene Lr1, isolated from bread wheat (Triticumaestivum L.) is a member of the large psr567 gene family. Plant Molecular Biology. 2007;**65**:93-106.

[36] Krattinger S G, Lagudah E S, Spielmeyer W, Singh R P,

Huerta-EspinoJ, McFadden H. A putative ABC transporter confers durableresistance to multiple fungal pathogens in wheat. Science. 2009;**323**:1360-1363.

[37] Moore J W, Herrera-Foessel S, Lan C, Schnippenkoetter W, AyliffeM,Huerta-Espino J. A recently evolved hexose transporter variantconfers resistance to multiple pathogens in wheat. Nature Genetics. 2015;**47**:1494-1498.

[38] Thind A K, Wicker T, Simkova H, Fossati D, Moullet O, Brabant C. Rapid cloning of genes in hexaploid wheat using cultivar-specific longrangechromosome assembly. Nature Biotechnology. 2017;**35**: 793-796.

[39] Fu D, Uauy C, Distelfeld A, Blechl A, EpsteinL, Chen X. Akinase-START gene confers temperature-dependent resistance to wheat striperust. Science. 2009;**323**:1357-1360.

[40] Marchal C, Zhang J, Zhang P, Fenwick P, Steuernagel B, Adamski N M. BED-domain-containing immune receptors confer diverse resistancespectra to yellow rust. Nature Plants. 2018;**4**:662-668.

[41] Klymiuk V, Yaniv E, Huang L, Raats D, Fatiukha A, Chen S S.Cloning of the wheat Yr15 resistance gene sheds light on the plant tandemkinase-pseudokinase family. Nature Communication. 2018;**9**:3735

[42] Zhang C, Huang L, Zhang H, Hao Q, Lyu B, Wang M. Anancestral NB-LRR with duplicated 3'UTRs confers stripe rust resistance inwheat and barley. Nature Communication. 2019;**10**:4023

[43] Periyannan S, Moore J, Ayliffe M, Bansal U, Wang X, Huang L.The gene Sr33, an ortholog of barley Mla genes, encodes resistance to wheatstem rust race Ug99. Science. 2013;**341**:786-788

[44] Saintenac C, Zhang W, Salcedo A, Rouse M N, Trick H N, Akhunov E. Identification of wheat gene Sr35 that confers resistance to Ug99 stemrust race group. Science. 2013;**341**:783-786.

[45] MagoR, Zhang P, Vautrin S, Simkova H, Bansal U, Luo M C. The wheat Sr50 gene reveals rich diversity at a cereal disease resistance locus.Nature Plants. 2015;**1**:15186.

[46] Steuernagel B, Periyannan SK, Hernandez-Pinzon I, Witek K, Rouse M N,Yu G. Rapid cloning of disease-resistance genes in plants usingmutagenesis and sequence capture. Nature Biotechnology. 2016;**34**:652-655.

[47] Zhang W, Chen S, Abate Z, Nirmala J, Rouse M N,DubcovskyJ. Identification and characterization of Sr13, a tetraploid wheat gene that confersresistance to the Ug99 stem rust race group. Proc. Natl. Acad. Sci. U.S.A. 2017;**114**:E9483–E9492.

[48] Chen S, Zhang W, Bolus S, Rouse M N, DubcovskyJ.Identification and characterization of wheat stem rust resistance gene Sr21effective against the Ug99 race group at high temperature. PlosoneGenetics. 2018;**14**:e1007287.

[49] Arora S, Steuernagel B, Gaurav K, Chandramohan S, Long Y M, Matny O. Resistance gene cloning from a wild crop relative by sequencecapture and association genetics. Nature Biotechnology. 2019;**37**:139-143

[50] Chen S, Rouse M N, Zhang W, Zhang X, Guo Y, Briggs J.Wheat gene Sr60 encodes a protein with two putative kinase domains thatconfers resistance to stem rust. New Phytology. 2019;**225**:948-959.

[51] Yahiaoui N, Srichumpa P, Dudler R, Keller B. Genome analysis atdifferent

ploidy levels allows cloning of the powdery mildew resistance genePm3b from hexaploid wheat. Plant Journal. 2004;**37**:528-538

[52] Hurni S. Rye Pm8 and wheat Pm3 are orthologous genes and showevolutionary conservation of resistance function against powdery mildew.Plant Journal. 2013;**76**:957-969

[53] Sanchez-Martin J. Rapid gene isolation in barley and wheat by mutantchromosome sequencing. Genome Biology. 2016;**17**: 221

[54] Xing L P. Pm21 from Haynaldiavillosa encodes a CC-NBS-LRR proteinconferring powdery mildew resistance in wheat. Molecular Plant.2018; **11**:874-878

[55] He H G. Pm21, encoding a typical CC-NBS-LRR protein, confersbroad-spectrum resistance to wheat powdery mildew disease. Molecular Plant. 2018;11,:879-882

[56] Zou S H, Wang H, Li Y W, Kong Z S, Tang D Z. The NB-LRR genePm60 confers powdery mildew resistance in wheat. New Phytology. 2018;**218**:298-309

[57] Rawat N, Pumphrey M, Liu S.(2016) Wheat *Fhb1* encodes a chimeric lectin with agglutinin domains and a pore-forming toxin-like domain conferring resistance to Fusarium head blight. Nature Genetics. 2016;**48:**1576-1580

[58] Paudel B, Zhuang Y, Galla A, Dahal S, Qiu Y, Ma A, Raihan TYenY.*WFhb1-1* plays an important role in resistance against Fusarium head blight in wheat. Scientific Reports. 2020; 10:7794

[59] Joshi A. Map-based cloning of the Hessian fly resistance gene H13 in Wheat [thesis]. Kansas State University; 2018.

[60] Dodds PN, Lawrence GJ, Ellis JG. Six amino acid changes confined to the leucine-rich repeat beta-strand/beta-turn motif determine the difference between the P and P2 rust resistance specificities in flax.The Plant Cell. 2001;**13**:163-178.

[61] Yahiaoui N, Brunner S, Keller B. 2006. Rapid generation of newpowdery mildew resistance genes after wheat domestication. The Plant Journal. 2006;**47**:85-98.

[62] Ashfield T, Redditt T, Russell A, Kessens R, Rodibaugh N, GallowayL, Kang Q, Podecheti R, Innes RW. Evolutionary relationshipof disease resistance genes in soybean and *Arabidopsis* specific for the *Pseudomonas syringae* effectors *Avr B*and *Avr Rpm1*. Plant Physiology. 2014;**166**:235-251

[63] Tommasini L, Yahiaoui N, Srichumpa P, Keller B. 2006. Development of functional markers specific for seven *Pm3* resistance alleles and their validation in the bread wheat gene pool. Theoretical and Applied Genetics. 2006; **114**:165-175

[64] Lagudah ES, Krattinger SG, Herrera-Foessel S, Singh RP, Huerta-Espino J, Spielmeyer W, Brown-Guedira G, Selter LL, Keller B. Gene-specific markers for the wheat gene *Lr34/Yr18*/Pm38 which confers resistance to multiple fungal pathogens. Theoretical Applied Genetics. 2009;**119**:889-898

[65] Panda N, Khush GS. Host Plant Resistance to Insects.Wallingford, UK: CABI/IRRI. 1995. 431 p.

[66] Smith CM. Plant Resistance to Arthropods: Molecular and Conventional Approaches. Dordrecht, The Netherlands: Springer. 2005. 423 p.

[67] Sadasivam S, Thayumanavan B. Molecular Host Plant Resistance to Pests. New York:Marcel Dekker. 2003. 479 p.

[68] Smith CM, Liu XM, Wang LJ, Liu X, Chen MS. Aphid feeding activates expression of a transcriptome of oxylipin-based defense signals in wheat involved in resistance to herbivory. J. Chem. Ecol. 2010;36:260-276.

[69] Chen MS. Inducible direct plant defense against insect herbivores: a review. Insect Sci.2008;15:101-114.

[70] Peshin R, Dhawan AK, editors. Integrated Pest Management: Innovation-Development Process New York/Heidelberg: Springer Science + Business Media; 2009; 690 p.

[71] Kessler A, Baldwin IT. Plant responses to insect herbivory: the emerging molecular analysis. Annu. Rev. Plant Biol.2002;53:299-328.

[72] Mithofer A, Boland W. Recognition of herbivory-associated molecular patterns. Plant Physiol.2008;146:825-831.

[73] Schmelz EA, Engelberth J, Alborn HT, Tumlinson JH, Teal PEA. Phytohormone-based activity mapping of insect herbivore-produced elicitors. Proc. Natl. Acad. Sci. USA2009;106:653-57.

[74] Kaloshian I. Gene-for-gene disease resistance. bridging insect pest and pathogen defense. J. Chem. Ecol.2004;30:2419-2438.

[75] Sullivan TJ, Rodstrom J, Vandop J, Librizzi J, Graham C. Symbiont-mediated changes in *Lolium arundinaceum* inducible defenses: evidence from changes in gene expression and leaf composition. New Phytol. 2007;176:673-679.

[76] Underwood N, Rausher M. Comparing the consequences of induced and constitutive plant resistance for herbivore population dynamics. Am. Nat. 2002;160:20-30.

[77] Huang J, McAuslane HJ, Nuessly GS. Resistance in lettuce to *Diabroticabalteat a* (Coleoptera: Chrysomelidae): the roles of latex and inducible defense. Environ. Entomol.2003;32:9-16.

[78] Underwood NC, Rausher M, Cook W. Bioassay versus chemical assay: measuring the impact of induced and constitutive resistance on herbivores in the field. Oecologia2002;131:211-219.

[79] Couldridge C, NewburyHJ, Ford-Lloyd B, Bale J, Pritchard J. Exploring plant responses to aphid feeding using a full *Arabidopsis* microarray reveals a small number of genes with significantly altered expression. Bull. Entomol. Res.2007;97:523-532.

[80] Kielkiewicz M. Influence of carmine spider mite TetranychuscinnabarinusBoisd. (Acarida: Tetranychidae) feeding on ethylene production and the activity of oxidative enzymes in damaged tomato plants. In Acarid Phylogeny and Evolution: Adaptation in Mites and Ticks—Proc. IV Symp. Eur. Assoc. Acarol., ed. F Bernini, R Nannelli, G Nuzzaci, E de Lillo;2002; Dordrecht, The Netherlands: Kluwer;2002. p. 389-92

[81] Li Y, Zou J, Li M, Bilgin DD, Vodkin LO. Soybean defense responses to the soybean aphid. New Phytol. 2008; 179:185-195.

[82] Liu X, Bai J, Huang L,Zhu L, Liu X. Gene expression of different wheat genotypes during attack by virulent and avirulent Hessian fly (*Mayetiola destructor*) larvae. J. Chem. Ecol. 2007;33:2171-2194.

[83] Kaloshian I, Walling L. Hemipterans as pathogens. Annu. Rev. Phytopathol.2005;43:491-521.

[84] Smith CM, Boyko EV. The molecular bases of plant resistance and defense responses to aphid feeding: current status. Entomol. Exp. Appl. 2007;122:1-16.

[85] van Eck L, Schultz T, Leach JE, Scofield SR, Peairs FB. Virus-induced gene silencing of *WRKY53* and an inducible *phenylalanine ammonia-lyase* in wheat reduces aphid resistance. Plant Biotechnol. J.2010;8:1023-1032.

[86] Howe GA, Jander G. Plant immunity to insect herbivores. Annu. Rev. PlantBiol.2008;59:41-66.

[87] Zhu L, Liu X, Liu XM, Jeannotte R, Reese J. Hessian fly (*Mayetiola destructor*) attack causes a dramatic shift in carbon and nitrogen metabolism in wheat. Mol. Plant-Microbe Interact.2008;21:70-78.

[88] Lapitan NLV, Li YC, Peng JH, Botha AM. Fractionated extracts of Russian wheat aphid eliciting defense responses in wheat. J. Econ. Entomol. 2007;100:990-999.

[89] Thackray DJ, Wratten SD, Edwards PJ, Niemeyer HM. Hydroxamic acids - potential resistance factors in wheat against the cereal aphids *Sitobionavenae* and *Rhopalosiphumpadi*. Proceedings of 1990 Brighton Pest Control Conference-Pests and Diseases-1990.1991;p215-220.

[90] Gianoli E, Ríos JM, Niemeyer HM. Allocation of a hydroxamic acid and biomass during vegetative development in rye. Acta Agriculture Scandinavica, Section B. Soil and Plant Science. 2000;50:35-39.

[91] Givovich A, Niemeyer HM. Comparison of the effect of hydroxamic acids from wheat on five species of cereal aphids. EntomologiaExperimentalis et Applicata. 1995;74:115-119.

[92] Givovich A, Niemeyer HM. Effect of hydroxamic acids on feeding behavior and performance of cereal aphids on wheat. European Journal of Entomology 1994;91:371-374.

[93] Figueroa C, Simon J, Gallic J, Prunier–letermeN, Briones lM, DedryverC,Niemeyer hM.Effect of host defense chemicals on clonal distribution and performance of different genotypes of the cereal aphid *Sitobionavenae.* Journal of Chemical Ecology. 2004;30(12):2515-2525.

[94] Kaur H, Salh P.SinghB. Role of defense enzymes and phenolics in resistance of wheat crop (Triticum aestivum L.) towards aphid complex. J.PlantInteractions. 2017;12(1):304-311.

[95] Nemacheck JA, Schemerhorn BJ, Scofield SR, Subramanyam S. Phenotypic and molecular characterization of Hessian fly resistance in diploid wheat, *Aegilops tauschii*. BMC Plant Biol. 2019;19(1):439. DOI:10.1186/s12870-019-2058-6

[96] Puthoff DP, Sardesai N, Subramanyam S, Nemacheck JA, Williams CE. Hfr-2, a wheat cytolytic toxin-like gene, is up-regulated by virulent hessian fly larval feeding. Mol Plant Pathol. 2005;6:411-423.

[97] Subramanyam S, Sardesai N, Puthoff DP, Meyer JM, Nemacheck JA, Gonzalo M, Williams CE. Expression of two wheat defense-response genes, Hfr-1 and Wci-1, under biotic and abiotic stresses. Plant Sci. 2006;170:90-103.

[98] FeuilletCS, TravellaNS, teinAlbarL,Nublatand A, Keller B. Map-based isolation of the leaf rust disease resistance gene Lr10 from the hexaploid wheat (*Triticum aestivum* L.) genome. Proc. Natl. Acad. Sci. U.S.A. 2003;100(25):15253-15258.

[99] Botha AM, Lacock L, van Niekerk C, Matsioloko MT, du Preez FB, Loots S, Venter E, Kunert KJ, Cullis CA. Is photosynthetic transcriptional regulation in Triticum aestivum L. cv. 'TugelaDN' a contributing factor for tolerance to Diuraphisnoxia (Homoptera: Aphididae)? Plant Cell

Rep. 2006;25(1):41-54. DOI: 10.1007/s00299-005-0001-9.

[100] Keenn T. Gene-for-gene complementarity in plantpathogen interactions. Annu. Rev. Genet. 1900;24:425-429.

[101] Boyko EV, Smith CM, Thara VK, Bruno JM, Deng Y, Starkey SR, Klaahsen DL. Molecular basis of plant gene expression during aphid invasion: wheat Pto- and Pti-like sequences are involved in interactions between wheat and Russian wheat aphid (Homoptera: Aphididae). J Econ Entomol. 2006;99(4):1430-1445. DOI: 10.1603/0022-0493-99.4.1430.

[102] Appels R, Eversole K, Feuillet C, Keller B, Rogers J, Stein N.Shifting the limits in wheat research and breeding using a fully annotatedreference genome. Science. 2018;**361**:eaar7191.

[103] Jiang J, Friebe B, Gill BS (1994) Recent advances in alien gene transfer in wheat. Euphytica 73:199-212

[104] Friebe B, Jiang J, Raupp WJ, McIntosh RA, Gill BS (1996) Characterization of wheat-alien translocations conferring resistance to diseases and pests: current status. Euphytica 91:59-87

[105] McIntosh RA, Wellings CR, Park RF (1995) Wheat rusts. An atlas of resistance genes. CSIRO Publishing, Melbourne

[106] McIntosh RA, Yamazaki Y, Dubcovsky J, Rogers J, Morris C, Appels R, Xia XC (2013) Catalogue of gene symbols for wheat. In: 12th international wheat genetics symposium, Yokohama, Japan

[107] Lopes, M.S., El-Basyoni, I., Baenziger, P.S., Singh, S., Royo, C., Ozbek, K., et al., 2015b. Exploiting genetic diversity from landraces in wheatbreeding for adaptation to climate change. Journal of Experimental Botany. 66, pp: 3477-3486.

[108] Yang, J. and Liang, Q., 1995. Yinchun 3 wheat germplasm with high protein content and resistance to drought. Crop Genetic Resources, 1, pp: 44.

[109] Li, X., Sun, F., Guo, B., Liu, L. and Pang, C., 1997. Evaluation of abiotic stress resistance in hebei winter wheat (*Triticum aestivum*) genetic resources.*Wheat Information Service*, 85, pp: 1-6.

[110] Reynolds, M.P. and Borlaug, N.E., 2006. Impacts of breeding on international collaborative wheat improvement. *J Agric Sci*., 144, pp: 3

[111] Reitz, L. P.and Salmon, S. C. 1968. Origin, history, and use of Norin 10 Wheat1. Crop Sci., 8, pp: 686-689.

[112] Dhaliwal HS, Singh Harjit, William M (2002) Transfer of rust resistance from Aegilops ovata into bread wheat (Triticum aestivum L.) and molecular characterization of resistant derivatives. Euphytica 126:153-159

[113] Dhaliwal HS, Chhuneja P, Gill RK, Goel RK, Singh H (2003) Introgression of disease resistance genes from related species into cultivated wheats through interspecific hybridization. Crop Improv 29:1-18

[114] McIntosh RA, Dubcovsky J, Rogers J, Morris C, Appels R, Xia XC (2016) Catalogue of gene symbols for wheat: 2015-16 supplement.

[115] McIntosh RA, Dubcovsky J, Rogers JW, Morris C, Xia CX (2017) Catalogue of gene symbols for wheat: 2017 supplement.

[116] Singh RP, Hodson DP, Jin Y, Huerta EJ, Kinyua M, Wanyera R, Njau P, Ward RW (2006) Currentstatus, likely migration and strategies to

mitigate the threat to wheat production from race Ug99(TTKS) of stem rust pathogen. CAB Rev 1:54

[117] Singh RP, Hodson DP, Jin Y, Lagudah ES, Ayliffe MA et al (2015) Emergence and spread of newraces of wheat stem rust fungus: continued threat to food security and prospects of geneticcontrol. Phytopathology 105:872-884

[118] Chhuneja P, Kaur S, Garg T, Ghai M, Kaur S, Prashar M, Bains NS, Goel RK, Keller B, Dhaliwal HS, Singh K (2008a) Mapping of adult plant stripe rust resistance genes in diploid a genome wheat species and their transfer to bread wheat. Theor Appl Genet 116:313-324

[119] Riar AK, Kaur S, Dhaliwal HS, Singh K, Chhuneja P (2012) Introgression of a leaf rust resistance gene from Aegilops caudata to bread wheat. J Genet 91:155-161

[120] Dakouri A, McCallum BD, Radovanovic N, Cloutier S. Molecular and phenotypic characterization of seedling and adult plant leaf rust resistance in a world wheat collection. Molecular Breeding. 2013;**32**:663-677

[121] Randhawa MS, Singh RP, Lan C. Interactions among genes *Sr2*/*Yr30*, *Lr34*/*Yr18*/*Sr57* and *Lr68* confer enhanced adult plant resistance to rust diseases in common wheat (*Triticum aestivum*L.) line 'Arula. Australian Journal of Crop Science. 2018;**12**:1023-1033

[122] Ellis JG, Lagudah ES, Spielmeyer W, Dodds PN. The past, present and future of breeding rust resistant wheat. Frontier Plant Science. 2014;**5**:641.

[123] Mago R, Zhang P, Vautrin S. The wheat *Sr50* reveals a rich diversity at a cereal disease resistance locus. Nature Plants. 2015;**1**:15186.

[124] Steuernagel B, Periyannan SK, Hernandez-Pinzon I. Rapid cloning of disease-resistance genes in plants using mutagenesis and sequence capture. Nature Biotechnology. 2016;**34**:652-655

[125] Thind AK, Wicker T, Simkova H, Fossati D. Rapid cloning of genes in hexaploidy wheat using cultivar-specific long-range chromosome assembly. Nature Biotechnology. 2017;**35**:793-796.

[126] Marchal C, Zhang J, Zhang P, Fenwick P. BED-domain containing immune receptors confer 2 diverse resistance spectra to yellow rust. Nature Plants. 2018;**4**:662.

[127] Bansal U, Bariana H, Wong D, Randhawa M, Wicker T, Hayden M, Keller B. Molecular mapping of an adult plant stem rust resistance gene *Sr56* in winter wheat cultivar Arina. Theoretical Applied Genetics. 2014;**127**:1441-1448.

[128] Dyck PL. The association of a gene for leaf rust resistance with the chromosome 7D suppressor of stem rust resistance in common wheat. Genome. 1987;**29**:467-469

[129] Hare RA, McIntosh RA. Genetic and cytogenetic studies of durable adult-plant resistances in 'Hope' and related cultivars to wheat rusts. Z Planzenzuchtung. 1979;**83**:350-367

[130] Herrera-Foessel SA, Lagudah ES, Huerta-Espino J. New slow-rusting leaf rust and stripe rust resistance genes *Lr67* and *Yr46* in wheat are pleiotropic or closely linked. Theoretical Applied Genetics. 2011;**122**:239-249

[131] Herrera-Foessel SA, Singh RP, Huerta-Espino J.*Lr68*: a new gene conferring slow rusting resistance to leaf rust in wheat. Theoretical Applied Genetics. 2012; **124**:1475-1486

[132] Singh RP, Mujeeb-Kazi A, Huerta-Espino J.*Lr46*: a gene conferring slow rusting resistance to leaf rust in

wheat. Phytopathology. 1998;**88**:890-894

[133] Uauy C, Brevis JC, Chen X, Khan I, Jackson L, Chicaiza O, Distelfeld A, Fahima T, Dubcovsky J. High-temperature adult-plant (HTAP) stripe rust resistance gene *Yr36* from *Triticum turgidum* ssp. *Dicoccoides* is closely linked to the grain protein content locus *Gpc-B1*. Theoretical Applied Genetics. 2005;**112**:97-105

[134] Fu D, Uauy C, Distelfeld A, Blechl A, Epstein L, Chen X, Sela H, Fahima T, Dubcovsky J (2009) A kinase-START gene confers temperature dependent resistance to wheat stripe rust. Science. 2009; **323**:1357-1360

[135] Krattinger SG, Lagudah ES, Spielmeyer W, Singh RP, Huerta-Espino J, McFadden H, Bossolini E, Selter LL, Keller B. A putative ABC transporter confers durable resistance to multiple fungal pathogens in wheat. Science. 2009;**323**:1360-1363.

[136] Moore JW, Herrera-Foessel S, Lan C. A recently evolved hexose transporter variant confers resistance to multiple pathogens in wheat. Nature Genetics. 2015;**47**:1494-1498.

[137] Wang ZL, Li LH, He ZH, Duan XY, Zhou YL, Chen XM, Lillemo M, Singh RP, Wang H, Xia XC(2005) Seedling and adult plant resistance to powdery mildew in Chinese bread wheat cultivarsand lines. Plant Dis 89:457-463

[138] Singh RP, Singh PK, Rutkoski J. Disease impact on wheat yield potential and prospects of genetic control. Annual Review Phytopathology. 2016;**54**:303-322

[139] Mesterhazy A, Bartok T, Kaszonyi G, Varga M, Toth B, Varga J. Common resistance to different *Fusarium* spp. causing *Fusarium* head blight in wheat. European Journal of Plant Pathology. 2005;**112**:267-281

[140] Buerstmayr H, Adam G, Lemmens M. Resistance to head blight caused by *Fusarium* spp. Inwheat. In: Sharma I (ed) Disease resistance in wheat. CABI, Wallingford. 2012; pp 236-276

[141] Guo J, Zhang X, Hou Y, Cai J, Shen X, Zhou T, Xu H, Ohm HW, Wang H, Li A, Han F, Wang H, Kong L. High-density mapping of the major FHB resistance gene *Fhb7* derived from *Thinopyrum ponticum* and its pyramiding with *Fhb1* by marker-assisted selection. Theoretical Applied Genetics. 2015; **128**:2301-2316

[142] Rush CM, Stein JM, Bowden RL, Riemenschneider R, Boratynski T, Royer MH. Status of Karnal bunt of wheat in the United States 1996 to 2004. Plant Disease. 2005;**89**:212-223.

[143] Dhaliwal HS,Navarete MR, Valdez JC. Scanning electron microscope studies of penetration mechanism of *Tilletia indica* in wheat spikes. Review Mexican Fitopathology. 1988;**7:**150-155.

[144] Gill KS, Aujla SS, Sharma I. Karnal Bunt and Wheat Production. Punjab Agricultural University Ludhiana, India. 1993; pp. 1-153.

[145] Fuentes-Davila G, Rajaram S, Singh G. Inheritance of resistance to Karnal bunt (*Tilletiaindica*Mitra) in bread wheat (*Triticum aestivum* L). Plant Breeding. 1995; **114**:252-254

[146] Sharma I, Bains NS, Singh K, Nanda GS. Additive genes at nine loci govern Karnal bunt resistance in a set of common wheat cultivars. Euphytica. 2005;**142**:301-307.

[147] Sharma I, Bains NS, Nanda GS.Inheritance of Karnal bunt free trait in bread wheat. Plant Breeding. 2004; **123**:96-97.

[148] Sharma I, Bala R, Kumar S, Bains NS. Development of near isogenic lines (NILS) using backcross method of breeding and simultaneous screening against Karnal bunt disease of wheat. Journal Applied Natural Science. 2016;**8**(3)1138-1145.

[149] Villareal RL, Fuentes- Davila G, Mujeeb-Kazi A, Rajaram S. Inheritance of resistance to *Tilletia indica* (Mitra) in synthetic hexaploids × *Triticum aestivum* crosses. Plant Breeding. 1995;**114:**547-548.

[150] Nielsen J. Inheritance of virulence of loose smut of wheat, *Ustilago tritici*, onthe differential cultivars Renfrew, Florence x Aurore, Kota, and little Club. Canadian Journal of Botany. 1977;**55**:260-263.

[151] Nielsen J. Inheritance of virulence of *Ustilagotritici* on the differential cultivars Carma, red bobs, and a derivative of the cross Thatcher x regent spring wheat. Canadian Journal of Botany. 1982;**60**:1191-1193.

[152] Knox RE, Howes NK. A monoclonal antibody chromosome marker analysisused to locate a loose smut resistance gene in wheat chromosome 6A.Theoretical Applied Genetics. 1994;**89:**787-93.

[153] Knox RE, Campbell HL, Clarke FR, Menzies JG, Popovic Z, ProcunierJD,Clarke JM, DePauw RM, Cuthbert RD, Somers DJ. Quantitative trait loci forresistance in wheat (*Triticum aestivum*) to *Ustilago tritici*. Canadian Journal of Plant Pathology.2014;**36**:187-201

[154] Knox RE, Campbell H, Menzies JG, Popovic Z, Procunier JD, Clarke JM,DePauw RM, Singh AK. Quantitative trait locus for loose smut resistance (*Ustilago tritici*) in wheat (*Triticum aestivum*). Lethbridge: XVI Biennial Workshop on the Smuts and Bunts; 2010

[155] Procunier JD, Knox RE, Bernier AM, Gray MA, Howes NK. DNA markers linkedto a T10 loose smut resistance gene in wheat (*Triticum aestivum* L.).Genome. 1997;**40**:176-179

[156] Kassa MT, Menzies JG, McCartney CA. Mapping of the loose smut resistancegene Ut6 in wheat (*Triticum aestivum* L.). Molecular Breeding. 2014;**33**:569-576

[157] Dhitaphichit P, Jones P, Keane EM. Nuclear and cytoplasmic gene control ofresistance to loose smut (*Ustilagotritici* (Pers.) Rostr.) in wheat (*Triticum aestivum* L.). Theoretical Applied Genetics. 1989;**78**:897-903.

[158] Syukov VV, Porotkin SE. Genetics of common wheat's (*Triticum aestivum* L.) resistance to loose smut (*Ustilagotritici* (Pers.) Jens.) review. Russian JournalGeneticsApplied Research. 2015;**5**:55-9.

[159] Takabayashi N, Tosa Y, Oh H S,Mayama S. A gene-for-gene relationship underlying the species-specific parasitism of *Avena/Triticum* isolates of *Magnaporthe grisea* on wheat cultivars. Phytopathology.2002;**92**:1182-1188

[160] Zhan S W, Mayama S, Tosa Y. Identification of two genes for resistance to *Triticum* isolates of *Magnaporthe oryzae* in wheat. Genome. 2008;**51**:216-221

[161] Tagle A G, ChumaI, Tosa Y. Rmg7, a new gene for resistance to Triticum isolates of *Pyriculariaoryzae* identified in tetraploid wheat. Phytopathology. 2015 https://doi.org/10.1094/PHYTO-06-14-0182-R

[162] Anh V L. Rmg8, a new gene for resistance to Triticum isolates of *Pyricularia oryzae* in hexaploid wheat. Phytopathology. 2015;**105**:1568-1572

[163] Anh V L.Rmg8 and Rmg7, wheat genes for resistance to the wheat blast

fungus, recognize the same avirulence gene AVRRmg8. Molecular Plant Pathology.2018;**19**:1252-1256

[164] Wang S.A new resistance gene in combination with Rmg8 confers strong resistance against triticum isolates of *Pyricularia oryzae* in a common wheat landrace. Phytopathology. 2018;**108**: 1299-1306

[165] Song QJ, Shi JR, Singh S, Fickus EW, Costa JM, Lewis J, Gill BS, Ward R, Cregan PB (2005) Development and mapping of microsatellite (SSR) markers in wheat. Theor Appl Genet 110:550-560

[166] Chhuneja P, Kaur S, Garg T, Ghai M, Kaur S, Prashar M, Bains NS, Goel RK, Keller B, Dhaliwal HS, Singh K (2008a) Mapping of adult plant stripe rust resistance genes in diploid a genome wheat species and their transfer to bread wheat. Theor Appl Genet 116:313-324

[167] Chhuneja P, Kaur S, Goel RK, Aghaee-Sarbarzeh M, Prashar M, Dhaliwal HS (2008b) Transfer of leaf rust and stripe rust resistance from Aegilops umbellulata Zhuk. to bread wheat (*Triticum aestivum* L.). Genet Resour Crop Evol 55:849-859

[168] Chhuneja P, Kaur S, Singh K, Dhaliwal HS (2008c) Evaluation of *Aegilops tauschii* (L.) germplasm for Karnal bunt resistance in a screen house with simulated environmental conditions. Plant Genet Resour Charact Util 6:79-84

[169] Malik R, Brown-Guedira GL, Smith CM, Harvey TL, Gill BS. Genetic mapping of wheat curl mite resistance genes Cmc3 and Cmc4 in common wheat. Crop Sci. 2003;43:644-650.

[170] Lanning SP, Fox P, Elser J, Martin JM, Blake NK, Talbert LE. Microsatellite markers associated with a secondary stem solidness locus in wheat. Crop Sci. 2006;46:1701-1793.

[171] Sherman JD, Weaver DK, Hofland ML, Sing SE, Buteler M. Identification of novel QTL for sawfly resistance in wheat. Crop Sci. 2010 50:73-86.

[172] Lapitan NLV, Peng J, Sharma V. A high-density map and PCR markers for Russian wheat aphid resistance gene Dn7 on chromosome 1RS/1BL. Crop Sci. 2007;47:811-820.

[173] Liu XM, Smith CM, Gill BS, Tolmay V. Microsatellite markers linked to six Russian wheat aphid resistance genes in wheat. Theor. Appl. Genet. 2001;102:504-510.

[174] BerzonskyWA, Ding H, Haley SD, Harris MO, Lamb RJ. Breeding wheat for resistance to insects.Plant Breed. Rev. Vol. 22. 2010. DOI: 10.1002/9780470650202.ch5

[175] Zhu LC, Smith CM, Fritz A, Boyko EV, Voothuluru P, Gill BS. Inheritance and molecular mapping of new greenbug resistance genes in wheat germplasms derived from *Aegilops tauschii*. Theor. Appl. Genet.2005;111:831-837.

[176] Gharalari AH, Fox SL, Smith MAH, Lamb RJ. Oviposition deterrence in spring wheat, Triticum aestivum, against orange wheat blossom midge, *Sitodiplosismosellana*: implications for inheritance of deterrence. Entomol. Exp. Appl.2009;133:74-83.

[177] Thomas MB. Ecological approaches and the development of "truly integrated" pest management. Proc. Natl. Acad. Sci. USA. 1999; 96:5944-5951.

[178] Andersen E J, Ali S, Byamukama E, Yen Y, Nepal M P. Disease resistance mechanisms in plants. Genes. 2018; **9**:339.

[179] Appels R, Eversole K, Feuillet C, Keller B, Rogers J, Stein N.Shifting the

limits in wheat research and breeding using a fully annotatedreference genome. Science. 2018;**361**:eaar7191.

[180] Steuernagel, B.,Witek, K., Krattinger, S. G., Ramirez-Gonzalez, R. H., Schoonbeek, H.-J., Yu, G., et al. (2018). Physical and transcriptional organisation of the bread wheat intracellular immune receptor repertoire. bioRxiv [Preprint].

[181] Lorenz AJ, Chao S, Asoro FG, Heffner EL, Hayashi T, Iwata H, Smith KP, Sorrells ME, Jannink JC. Genomic selection in plant breeding: knowledge and prospects. Advance Agronomy. 2011;**110**:77-123

[182] Rutkoski J, Poland JA, Singh RP, Huerta-Espino J, Bhavani S, Barbier H, Rouse MN, Jannik J-L,Sorrells M. Genomic selection for quantitative adult plant stem rust resistance in wheat.Plant Genome. 2014;**7**(3):1-10

[183] Mirdita V, He S, Zhao Y, Korzun V, Bothe R, Ebmeyer E, Reif JC, Jiang Y. Potential and limitsof whole genome prediction of resistance to *Fusarium* head blight and *Septoria tritici*blotchin a vast central European elite winter wheat population. Theoretical Applied Genetics. 2015;**128**:2471 2481

[184] Milne RJ, Dibley KE, Schnippenkoetter W, Mascher M, Lui ACW, Wang L, Lo C, Ashton AR, Ryan PR, Lagudah ES (2019) The wheat gene from the sugar transport protein 13 family confers multipathogen resistance in barley. Plant Physiol 179:1285

[185] Moore JW, Herrera-Foessel S, Lan C, Schnippenkoetter W, Ayliffe M, Huerta-Espino J, Lillemo M, Viccars L, Milne R, Periyannan S (2015) A recently evolved hexose transporter variant confers resistance to multiple pathogens in wheat. Nat Genet 47:1494-1498