Breeding Wheat for Powdery Mildew Resistance

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Abstract

Powdery mildew (*Blumeria graminis* f. sp. *tritici*) is one of the diseases of wheat and causes economic loss in wheat production. Powdery mildew can be managed through an array of methodologies; however genetic/host resistance is the most economical, reliable, efficient, sustainable, and environment-friendly approach. Genetic resistance is imparted either through race-specific/qualitative or non-race-specific/quantitative or combination of both in the host. Sources of powdery mildew resistance include cultivated and wild species comprising of primary, secondary, and tertiary gene pool. Identified resistance is transferred to elite genetic background with minimum linkage drag using breeding techniques involving from backcrossing, marker-assisted selection, gene pyramiding to the advanced CRISPER, gene cassettes, etc. This chapter discusses on the abovementioned subjects/topics along with breeding challenges and future prospects.

Keywords

Wheat powdery mildew · Resistance breeding · Marker-assisted selection · PM resistance gene

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12.1 Introduction

Wheat is one of the most important sources of food. At global level, approximately 95% of wheat cultivated is bread wheat (*T. aestivum*, hexaploid), while the remaining 5% being durum wheat (*T. durum*., tetraploid) and few other less important types (Shewry 2009). Wheat yield has been constantly under threat due to biotic and abiotic factors. In the event of abiotic stress, wheat cultivars are inclined by stress because of humidity, salt, temperature, and micronutrient. Continuous experience to high temperatures in rain-fed areas leads to drought stress and osmotic stress and higher salt concentrations in soil created by rapid evaporation of water, while the major biotic stress in wheat is due to diseases such as rusts, powdery mildew, karnal bunt, loose smut, blast, etc. Among the diseases, powdery mildew (PM), caused by the biotrophic fungus *Blumeria graminis* f. sp. *tritici* (Bgt), is one of the most serious threats limiting wheat production in several regions of the world including India. PM reduces the photosynthetic leaf area and the available nutrients, thereby reducing the yield significantly (by up to 25%). Breeding and developing PM-resistant cultivars is commonly viewed as the most efficient, powerful, and ecologically friendly technique to manage the disease. PM management using resistance genes enhances the stability and durability of the cultivars. Such durable resistance is exceedingly beneficial to farmers as it leads to increase in yield along with reduced cost of cultivation and is environmentally pleasant (Shah et al. 2017).

12.2 Disease

Powdery mildew is a wind-borne disease favored by the presence of disease in the preceding season. Disease infection can start during early crop growth when conditions are cool and wet. As the temperature rises and the humidity falls, the incidence and severity tend to diminish. The disease is preferred by mild temperatures (10–22 °C) (Beest et al. 2008), and 100% relative humidity (RH) favors the conidium germination. Prolonged cloudy weather fastens the disease development. During winter, spores survive in the host tissue after infection and may come from earlier infections within the field or from fields farther away. The disease is most common in dense early sown crops with high nitrogen fertility and rapid plant growth. Good soil moisture with potassium deficiency promotes canopy humidity which in turn favors the pathogen infection. Warm weather with alternate dry and wet conditions with wind may lead to epidemics during which even a resistant variety can become susceptible (Cunfer 2002).

12.2.1 Causal Organism

Powdery mildew is a fungal leaf disease caused by *Blumeria graminis* (DC.) E.O. Speer f. sp. *tritici* Em. Marchal (Bgt) = *Erysiphe graminis* DC. Ex Merat f. sp. *tritici* Em. Marchal. The pathogen is an obligate fungus which is host specific.

Blumeria has been previously treated as a species of *Erysiphe*. But it differs from all species of *Erysiphe* because its anamorph possesses unique features such as digitate haustoria, secondary mycelium with bristle-like hyphae, and bulbous swellings of the conidiophores and unique structure of the ascocarps (Braun 1987). As a biotrophic parasite, *B. graminis* has evolved to specialize on particular hosts of Poaceae family. The wind-borne polycyclic pathogen greatly reduces yield and grain quality in wheat varieties that are susceptible. *Blumeria* is a true ascomycete fungus, forming the order of Erysiphales with only one family, the Erysiphaceae.

12.2.2 Geographical Distribution

PM of cereals is globally distributed. The disease is more common in regions with frequent rain and relatively cool temperature (Kashyap et al. 2021; Bennett 1984). Powdery mildew has been reported in several countries like the United Kingdom, Russia, Germany, Japan, Africa, and all parts of West Asia (Bennett 1984). Powdery mildew is a rampant disease in the cooler regions of China, Japan, and Central Asia, in North and East Africa, in northern Europe, and in eastern North America (Roelfs 1977; Saari and Wilcoxson 1974). In warmer, humid regions with mild winters such as parts of South America and the southeastern United States, the disease tends to be severe. But in regions where rain is frequent and heavy, the occurrence of powdery mildew is usually less as the spores are washed away from the leaves (Merchan and Kranz 1986). In India, PM is increasingly becoming problematic particularly in the northern and southern hill zone and some parts of north western plain zone.

12.2.3 Symptoms

Powdery mildew, caused by *Blumeria graminis* f. sp. *tritici* (syn. *Erysiphe graminis*), attacks wheat exclusively. The disease is characterized by white cottony powdery patches of fungal mycelium and conidia on the surface of the leaf. Initially, the symptoms appear as yellow flecks on the leaves. As the growing season progresses, the fluffy white fungal colonies can also affect the stem and earheads. Young plants having shorter tillers can become easily susceptible as they remain lower in the canopy. In severe condition it also appears on the awns. At maturity, the fluffy white colonies turn tan or brown in color. Symptoms usually progress from the lower to the upper leaves. As the infection gets older, small, black sexual fruiting bodies called chasmothecia (previously named cleistothecia) appear as distinct black dots on the maturing plants. When severe, the individual colonies often merge together and eventually cover most part of the stem and leaf surface. Moderate to severe infections can result in the death of the leaf tissues. Crop infected with powdery mildew usually appears yellow when seen from far due to the early death of leaves (Cunfer 2002).

12.2.4 Epidemiology

Powdery mildew severity occurs during periods of rapid crop growth, i.e., when plants begin to joint. The disease is first observed during tillering but does not continue after ear emergence. The sexual fruiting bodies known as chasmothecia produced during the late spring are highly resistant to extreme temperature and are thus an important source of inoculum for the following season. The chasmothecia survives on the wheat straw or as mycelium on infected wheat. During humid weather conditions, chasmothecia release ascospores which can germinate and infect plants under cool, humid conditions. Conidia are easily discharged from the lesions and are disseminated over long distances by wind and rain. An optimal temperature of 20°C is conducive for the production of conidia, and it declines rapidly above and below that temperature (Ward and Manners 1974). Fresh pustules with conidia are produced every 7–10 days at 97–100% relative humidity (Esmail and Draz 2017; Piarulli et al. 2012). The cycle is repeated for the continuous production of spores. As per the reports of Friedrich and Boyle (1993), relative humidity below 92% reduces the germ tube growth and appressorium production. Wheat powdery mildew infection and development tend to diminish when the temperatures rise above 25 °C and as humidity declines. Frequent and heavy rains slow the development of established pustules as the conidia are washed away by the rain water (Merchan and Kranz 1986).

12.2.5 Disease Cycle

The mildew fungus has multiple, rapid life cycles in a growing season. The fungus survives on the stubble as chasmothecia. Windborne conidia cause initial infections on the leaf surface which leads to secondary infections. The life cycle of powdery mildew includes both sexual (between seasons) and asexual (within season) stages particularly adapted to specific host habitats (McDonald and Linde 2002). Also, powdery mildew propagates very efficiently on wild plants and forms a huge reservoir of deeply rooted parasites that could serve as a source of inoculums to initiate epidemics in the fields (Dinoor 1974).

12.2.6 Yield Losses due to Powdery Mildew

Heavy economic losses around the globe have been reported to be due to powdery mildew diseases of wheat (Alam et al. 2012; Chen 2005). Powdery mildew can cause serious crop damage under severe conditions. Generally, the yield losses range from 12 to 34% (Griffey et al. 1993; Conner et al. 2003). But greatest yield losses up to 50% may occur if the disease occurs on the flag leaf during the heading and grain filling stage (Griffey et al. 1993; Leath and Bowen 1989). Based on the time of disease, epidemic onset, and its severity, the yield losses can reach up to 60% (Oerke et al. 1994). The pathogen reduces photosynthesis, decreases leaf assimilation index,

and negatively affects grain yield components in wheat crop (Bowen et al. 1991; Henry and Kettlewell 1996; Samobor et al. 2005). At later growth stages, heavily colonized leaves can be killed prematurely which can significantly reduce yield up to 25% by reducing photosynthetic leaf area and crop available nutrients. In the colonized plants, the infection increases the metabolism of the attacked plants producing smaller shriveled grains. Also, during the attack of powdery mildew, the ability of the plant to resist other pathogens is decreased (Paulech 1995). Even low level of powdery mildew infection leads to the production of a greater number of nonproductive tillers which leads to reduced yield. Severe infection of powdery mildew can also cause delayed maturity, which increases the chances of reinfection and can also cause crop lodging through weakened stems. The earlier the infection, the larger is the potential yield loss.

In India, powdery mildew disease of wheat has caused serious consequences especially in parts of North Western Plain Zone, Northern Hill Zone, and Southern Hill Zone (Singh et al. 2009). However, sporadic incidence of powdery mildew has been reported from Rajasthan, Maharashtra, and Karnataka (Arya and Ghemawat 1953; Gadore and Patwardhan 1965; Patil et al. 1969). The disease was reported for the first time from Bombay (Maharashtra) (Gadore and Patwardhan 1965) and from Karnataka (Patil et al. 1969). From time to time, disease has been observed in severe form in U.P., Punjab, Haryana, Rajasthan, and Delhi (Swaminathan et al. 1971). In India, accurate data regarding the losses caused by mildew are not available since the disease mainly occurs in hills where wheat area is very limited.

12.2.7 Powdery Mildew Disease Assessment

Precise assessment of wheat powdery mildew is necessary for identifying the resistant and susceptible plants. Assessment of powdery mildew severity is done both in the seedling and adult plant stages using a visual scoring scale. Seedlings are generally assessed in the glasshouse under controlled conditions. The infection types (IT) of 10–15 days post-inoculation (dpi) of wheat leaves were scored using the 0–4 scale (Zhang et al. 2010). According to this scale, the scoring of "0," "0;", "1," "2," "3," and "4" indicate "no visible symptoms," "necrotic flecks without sporulation," "highly resistant," "resistant," "susceptible," and "highly susceptible," infection types, respectively. In field conditions, powdery mildew is generally assessed using a 0–9 scale (Saari and Prescott 1975), based on the progression of symptoms. This scale is divided into three classes of infection types (ITs). 0–3 is considered as resistant, ITs 4–6 is considered as intermediate, and ITs 7–9 is considered as susceptible. The adult plant scoring was done once per season when the powdery mildew symptoms fully developed around GS-75 (Zadoks et al. 1974) and the most susceptible cultivars reached maximum severity (Table 12.1).

Table 12.1 Adult plant scale for powdery mildew disease scoring in wheat

Host response (class)	Infection type	Disease symptoms
Immune	0	Free from infection
Very resistant	1	Few scattered colonies on the lowest most leaves only
Resistant	2	Few colonies on both second and first leaves which infected at light intensity
Moderately resistant	3	Light intensity of infection at lower third leaves of plant
Low intermediate	4	Moderate to severe infection of lower leaves with scattered to light infection extending to the leaf immediately below the mid-point of the plant
Intermediate	5	Moderate to light infection extending to the mid-point of the plant with severe infection of lower leaves and upper leaves free. Infections do not extend beyond mid-point of plant
High intermediate	6	Severe infection of lower third leaves of plant, moderate degree on middle leaves, and scattered colonies beyond the mid-point of the plant
Moderately susceptible	7	Severe infection on both lower and middle leaves with light infection extending to the leaf below the flag leaf with few colonies on the flag leaf
Susceptible	8	Severe infection on lower and middle leaves with moderate to severe infection of upper third of plant. Flag leaf infected in amounts more than a trace
Very susceptible	9	Severe infection on all leaves and the spike infected to some degree. Spike infections are scored as a modified scale (1–9) or as the percentage of the total area covered. The spike infection score is separated from the foliar score
–	N	Used to indicate no scoring possible due to necrosis as a result of other diseases or factors

12.3 Disease Management

Many strategies are involved to control PM in wheat. However, genetic/host resistance is the most economical, reliable, sustainable, and environmentally safest way to control the PM disease.

12.3.1 Genetic Resistance to Powdery Mildew

Genetic resistance is among the most useful means to control powdery mildew (Xin et al. 2012; Summers and Brown 2012). Crops are diverse in their defense capacity against pathogens, and the genetic status of both host and pathogen determines the outcome of the interaction. The resistance depends on the interaction between the host and the pathogen. In general, there are two types of resistance to powdery

mildew, i.e., quantitative resistance (horizontal or polygenic) and qualitative resistance (complete, vertical, and race-specific).

Each cell of the plants has innate immune system with systemic signaling capability from the site of infection (Jones and Dangl 2006). Upon infection, pathogens produce elicitors which are called pathogen-/microbe-associated molecular patterns (PAMP/MAMP), which includes peptides, metabolites, cell wall components, enzymes, and toxins (Dodds and Rathjen 2010; Giraldo and Valent 2012). These elicitors suppress the plant's defense mechanisms. Post-infection, the host produces certain signal molecules known as damage-associated molecular patterns (DAMP) (Boller and Felix 2009). These elicitors or PAMP/MAMP/DAMP are recognized by the specific receptors (PRRs) in the plasma membrane (Frescatada-Rosa et al. 2015). As a primary level of defense response, the PAMP/MAMP triggers downstream genes resulting in no symptoms or hypersensitive response, generally referred to as the PAMP/pattern-triggered immunity (PTI) or non-host resistance (Baxter et al. 2014; Dodds and Rathjen 2010).

Certain pathogens produce race-specific intracellular elicitors known as effectors which are produced by specific avirulence (AVR) genes (Boller and Felix 2009). These effectors are recognized by plant-produced specific receptors (R proteins), encoded by R genes (Du et al. 2015; Sarris et al. 2015; Jones and Dangl 2006). These effectors suppress other PAMPs and also the host resistance genes to become more virulent (Lo Presti et al. 2015). As a secondary level of defense response, the effectors trigger downstream genes resulting in race-specific hypersensitive response to contain the pathogen, generally referred to as the effector triggered immunity (ETI) or qualitative resistance or vertical resistance (Boller and Felix 2009; Giraldo and Valent 2012).

In contrast, a weaker immune response, PTI and ETI response along with lack of hypersensitive response due to reduced or non-functionality of genes producing effectors and PAMP/PRR proteins and production of enzymes and toxins by pathogens, facilitating the pathogen to advance further is considered as incomplete resistance or quantitative resistance (Kim and Hwang 2015; Waszczak et al. 2015).

12.3.1.1 Race-Specific Resistance

Race-specific resistance or qualitative resistance has proved to be an integral part of crop breeding for resistance in wheat for many decades (Lillemo et al. 2010; Shamanin et al. 2019). This type of resistance is usually linked to immunity and constitutes resistance at all stages. Both PAMP/pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) are to be considered as qualitative resistance, where the plant immune response is either a complete resistance with hypersensitive response or susceptibility. Race-specific resistance or vertical resistance to powdery mildew is controlled by major genes that is effective for only few Bgt isolates but is ineffective for others. Race-specific resistance is mainly via a hypersensitive foliar reaction directly involving single major R genes, which follows a gene-for-gene model (Bennett 1984; Hsam and Zeller 2002). More than 70 formally designated powdery mildew resistance genes (Pm) have been cataloged thus far (McIntosh et al. 2014). Major genes are expressed in seedlings and throughout the life stages of

wheat. The widely used powdery mildew resistance gene *Pm6*, derived from *T. timopheevi* (Kuckuck 1970), is best expressed from the three-leaf stage onward (Jorgensen and Jensen 1972), and it is moderately effective.

Cultivars with race-specific resistance genes generally provide immunity or near-immunity to disease. This exerts a selection pressure on the pathogen that often leads to the rapid build-up of isolates with matching virulence genes (McDonald and Linde 2002). The R genes encode a class of resistance proteins which are the nucleotide-binding site leucine-rich repeat (NLR)-type receptors (Jones and Dangl 2006). He et al. (2018) reported that the powdery mildew resistance gene *Pm21* encodes a typical NLR protein, yet it confers a broad-spectrum resistance at both seedling and adult plant stages to PM. To date, 11 mildew resistance genes, viz., *Pm3* (Srichumpa et al. 2004), *Pm38* (Krattinger et al. 2009), *Pm8* (Hurni et al. 2012), *Pm46* (Moore et al. 2015), *Pm2* (Sánchez-Martín et al. 2016), *Pm21* (Cao et al. 2011), *Pm17* (Singh et al. 2018), *Pm60* (Zou et al. 2018), *Pm5* (Xie et al. 2020), *Pm24* (Lu et al. 2020), and *Pm41* (Li et al. 2020a, b) all encoding the nucleotide binding sites and leucine-rich repeat (NBS-LRR) proteins have been cloned.

Change in the virulence frequency is mainly influenced by the resistance genes present in the cultivars grown in a particular area. This has led to the rapid increase in virulent strains within the pathogen population, and consequently many of the major genes were overcome by new virulent pathotypes in a short span of time. For instance, a widely used gene *Pm17* was overcome by new virulent pathotypes (Persaud et al. 1994). Recent ineffectiveness of some genes such as *Pm17*, *Pm3a*, and *Pm4a* in Eastern and mid-Atlantic regions of the USA (Cowger et al. 2009) and *Pm8* in China (Wang et al. 2005) has urged the breeding community to further enrich the resistance sources to PM by identification and mobilization of new genes (Wallwork 2009).

Though many resistance genes have been identified from wheat and wheat-related species, most of them are race-specific and can be easily be overcome by new Bgt isolates (Li et al. 2014). Development of powdery mildew ceases when the day time temperature exceeds 26 °C, and thus even a moderate level of host resistance is adequate. In areas prone to severe epidemics, a common approach to control powdery mildew is to adopt host resistance by deploying one or more *Pm* genes in a better genetic background. Another strategy is to deploy non-race-specific quantitative resistance conferring more durable broad-spectrum resistance.

12.3.1.2 Non-race-Specific Resistance

Another type of resistance to powdery mildew is called non-race-specific resistance or quantitative or adult plant resistance (APR) or horizontal resistance which is governed by genes which is expressed in adult plants but not in seedlings. APR is quantitative in nature, and Keller et al. (1999) reported nearly 18 QTLs to govern powdery mildew resistance. APR is non-race-specific and is also referred as "slow mildewing" (Shaner 1973) and "partial resistance" (Hautea et al. 1987). APR is more durable than race-specific resistance to powdery mildew. Non-race-specific or quantitative resistance is commonly effective at the post-seedling stage. The resistance conferred by these genes is the result of the small effects of many genes and does not

lead to complete absence of infection; instead, it reduces the fungal sporulation and duration (Burdon et al. 2014; Li et al. 2014).

Poland et al. (2009) reported that the molecular mechanism underlying quantitative resistance is that there is a multigenic basis, but the recent evidence suggests a likely diversity of mechanisms, some overlapping with race-specific resistance. Depending on the number and effects of genes controlling resistance, it is possible to distinguish quantitative resistance from race-specific resistance. According to Cowger et al. (2012), APR to powdery mildew has been identified in numerous widely cultivated cultivars which have been effective for more decades.

Hsam et al. (2003) reported that *Pm12*, *Pm16*, and *Pm20* genes confer most effective defense response against powdery mildew in the adult plant stage. Majority of the APR genes have been mapped in the winter wheat. However, some of the APR genes such as *Pm38* located at the *Lr34/Yr18* locus and *Pm39* located at the *Lr46/Yr29* locus have been identified in spring wheat sources. Majority of the QTLs governing APR to powdery mildew have been reported on the chromosomes 1B, 2A, 2B, 2D, 3A, 3B, 4A, 4D, 5A, 5D, 6A, 7B, and 7D.

It is difficult to recognize plants with both race-specific resistance and APR to powdery mildew based on phenotype variation. With the advent of the molecular markers closely associated with genes such as *Pm1* (Hu et al. 1997), *Pm2* and *Pm3* (Ma et al. 1994), *Pm4* (Hartl et al. 1999), *Pm12* (Jia et al. 1996), *Pm12* (Cenci et al. 1999), *Pm21* (Qi et al. 1996), and *Pm25* (Shi et al. 1998) revealed that these genes have been reported with both race-specific resistance and APR to powdery mildew.

Several quantitative trait loci (QTLs) governing APR to wheat powdery mildew have been mapped near loci where the defeated major genes such as *Pm4*, *Pm5*, and *Pm6* have been located (Keller et al. 1999; Liu et al. 2001). Keller et al. (1999) observed that the resistance conferred by such QTLs may be the result of the residual effects of defeated major genes and partial resistance may be the result of alternate alleles present at the R gene loci. Thind et al. (2017) reported that some genes involved in the quantitative resistance such as adult plant resistance (APR) gene also encode an NLR protein.

Humid and high rainfall conditions generally favor the rapid spread of powdery mildew throughout the plant canopy until senescence. Powdery mildew infection in the early plant growth stages reduces the tillering, whereas infection in the flowering stages reduces the losses in grain yield and quality (Everts et al. 2001). Thus control of powdery mildew in late plant growth stages is necessary for maximum protection of grain yields (Bowen et al. 1991; Griffey et al. 1993). Recently, additional sources of APR to powdery mildew have been identified, characterized, and validated. In order to develop durable and effective resistance to powdery mildew in wheat, it is necessary to combine QTLs having additive and complementary effects and that express during different growth stages.

However, the effective use of APR to powdery mildew resistance was largely limited due to lack of knowledge of effective sources of resistance, the quantitative nature, and precise tools for selection. This approach is cost-effective and time-consuming. Development of large mapping population segregating for the

quantitative trait is required. Also the linkage analysis is restricted to loci in genomic regions containing polymorphisms between the two parental lines (Tanksley 1993).

12.3.2 Sources of Resistance to Powdery Mildew

Transferring PM-resistant genes to hexaploid wheat cultivars has been considered as an effective way to contain the disease. Most of the powdery mildew resistance genes actually come from wild relatives of wheat such as *Triticum monococcum*, *T. urartu*, *T. turgidum* ssp. dicoccoides, *Aegilops speltoides*, *Ae. tauschii*, etc. (Hsam and Zeller 2002; Jiang et al. 1994). Within the so far identified *Pm* genes derived from alien species or sparsely cultivated subspecies, 22 are assigned on the B genome, while only 14 are assigned on the A genome and 8 on the D genome (Tang et al. 2018).

12.3.2.1 Diploid Sources

Diploid *Ae. tauschii* Coss (2n = 2x = 14, DD), a distant relative of wheat, has proved to be a valuable source of powdery mildew resistance (Gill et al. 1986; Cox et al. 1992). Two genes, namely, *Pm2* and *Pm19*, were transferred from *A. tauschii* into common wheat (Hsam and Zeller 2002). *Ae. speltoides* and *Ae. longissima* are both diploid species which were the donor of *Pm1d*, *Pm12*, *Pm32*, and *Pm12* (Hsam and Zeller 2002; Hsam et al. 2003; Cenci et al. 1999). *Pm29* gene was introduced from *Ae. ovate* to hexaploid wheat (Hsam et al. 2003). Similarly, *Pm57* was identified on chromosome 2BL in *Ae. searsii* (Liu et al. 2017). More distantly related species which includes *Ae. caudate*, *Ae. ovate*, *Ae umbellulate*, *Ae triuncialis*, and *Ae variabilis* are also reported as valuable sources for PM resistance (Chen et al. 1995; He et al. 2009).

12.3.2.2 Tetraploid Sources

Tetraploid *T. carthlicum* (2n = 4x = 28, AABB genomes) was the source of *Pm4b* and *Pm33* (Hsam and Zeller 2002; Zhu et al. 2005). Tetraploid species such as *T. timopheevii* and *T. araraticum* constitute the secondary gene pool. *T. timopheevii* and its wild form, *T. araraticum* (2n = 4x = 28, AAGG), contributed *Pm6*, *Pm27*, and *Pm37* (Mains 1934; Järve et al. 2000; Hsam and Zeller 2002; Murphy et al. 2002; Perugini et al. 2008). Though tetraploid *T. durum* (2n = 4x = 28, AABB) is a less valuable source of resistance to powdery mildew (Mains 1934; Hsam and Zeller 2002), it contributed *Pm3h* (Zeller and Hsam 1998).

For common wheat, its progenitor wild emmer is also a rich donor of adaptive diversity to various diseases and can be exploited for trait improvement (Huang et al. 2016). Many confirmed *Pm* genes originate from wild species and primitive forms including wild emmer. Incorporating *Pm* genes into commercial cultivars is made possible as wild emmer is easily crossable with both hexaploid common wheat and tetraploid durum wheat (Rong et al. 2000; Elkot et al. 2015). Tetraploid wild emmer wheat (*T. dicoccoides*) (2n = 4x = 28, AABB) is the progenitor of common tetraploid and hexaploid wheats (Liu et al. 2002). Some of the powdery mildew

resistance genes such as *Pm16*, *Pm30*, *Pm31*, *Pm36*, *Pm41*, *Pm42*, *Pm49*, and *Pm50* have been transferred from wild emmer into common hexaploid wheat (Piarulli et al. 2012; Mohler et al. 2012). Cultivated species, *T. dicoccum*, is the source of resistance gene, *Pm5*, which is a recessive gene for powdery mildew (Mclntosh 1973).

12.3.2.3 Hexaploid Sources

Hsam and Zeller (2002) reported that old wheat cultivars, landraces, and related species were screened for resistance to powdery mildew early in the 1930s. PM genes were identified in many different, widely distributed wheat cultivars and landraces cultivated for thousands of years under extreme environments which are more genetically polymorphic in disease resistance and widely adapted to abiotic stresses (Talas et al. 2011; Li et al. 2016). Landraces can be readily crossed for the desired traits into new cultivars in comparison to distant relatives. Globally, many spring and winter wheat genotypes having seedling and adult plant resistance to powdery mildew have been identified and utilized in breeding programs. A total of 22 resistance alleles at 10 loci including *Pm1*, *Pm2*, *Pm3* (*3a*, *3b*, *3c*, *3d*, *3e*, and *3f*), *Pm9*, *Pm18*, *Pm22*, and *Pm45* were identified in *T. aestivum* indicating the presence of more PM genes in cultivated wheat (Hsam and Zeller 2002). So far, nearly 33 designated genes have been identified from *T. aestivum*.

12.3.2.4 Other Sources

Li et al. (2018) and Chen et al. (2012) have reported that rye (*Secale cereale* L.) and *Haynaldia villosa* (*H. villosa*, syn. *Dasypyrum villosum*) had been used as a source of powdery mildew resistance genes. Genes for resistance to powdery mildew such as *Pm8* and *Pm17* have been successfully transferred into commercial wheat cultivars from rye (Jiang et al. 1994; Kim et al. 2004).

Pm8 is one of the most widely used genes in wheat breeding (Ren et al. 1997). This gene has played a major role in reducing the wheat yield loss due to powdery mildew infection. *Pm8* was originally transferred from the "Petkus" rye into hexaploid wheat. Lutz et al. (1992) reported the emergence of new Bgt isolates that overcame the resistance of *Pm8* during the 1990s. But the use of *Pm8* in wheat breeding programs continued, especially in the twenty-first century, because the wheat-rye 1BL/1RS translocation carried other agronomic traits such as wide adaptability and high yield potential together with multiple disease resistance (Luo et al. 2009; El-Shamy et al. 2016). Globally, *Pm8* has played an important role in wheat breeding and has been effective against the powdery mildew pathogen (Hurni et al. 2012). *Pm17* is another resistance gene located on the short arm of the 1R chromosome in rye. Another gene *Pm17* is also from rye identified in 1AL/1RS wheat-rye translocations (Friebe et al. 1994).

Chen and Liu (1982) reported another potential source of alien wheat powdery mildew resistance gene, *Pm21* derived from *H. villosa* during the early 1980s. *Pm21* showed a broad spectrum of resistance against most of the isolates of Bgt (Liu et al. 2015) and has remained effective for more than 40 years. Also, the resistance gene *Pm51* was identified on chromosome 2BL in *D. villosa* (Zhan et al. 2014).

Oettler et al. (2005) reported that the hexaploid triticale (× Triticosecale Wittmack, AABBRR, 2n = 6x = 42), synthesized artificially by combining the genomes of *Triticum turgidum* (AABB, 2n = 4x = 28) and *S. cereale* (RR, 2n = 2x = 14), is an excellent source of powdery mildew resistance. Easy transfer of rye chromosomes into common wheat is possible as the rye components in triticale have been adapted to the wheat nucleus and cytoplasm (Ma and Gustafson 2008). As direct cross between wheat and rye requires precise embryo rescue techniques (Oettler et al. 2005), triticale serves as an alternative source for transferring the resistance contained in the rye chromosome to the hexaploid wheat. Hybridization of several triticale lines resulted in the development of triticale cultivar that varied in the rye genomes. As it combines the broad stress tolerance of different triticale lines, it can be effectively used to improve the powdery mildew and rust resistance of wheat in a short time.

Other species with potentially useful powdery mildew resistance genes are *Ae. markgrafii*, *Ae. umbelluata*, *Ae. variabilis*, *Ae. triuncialis*, and *Ae. mutica*, as well as the perennial subspecies of Triticeae, such as *Elymus*, *Leymus*, *Elytrigia*, and *Thinopyrum* (Jiang et al. 1994; Eser 1998; Hsam and Zeller 2002; Luo et al. 2009). Therefore, the identification of new and effective alien genes from wild relatives of wheat and their further translocation into crops may be a significant contribution to develop durable resistance to a broad spectrum of pathogen (Tester and Langridge 2010). Developing more resilient cultivars thereby solves the problem of low resistance of crops to powdery mildew of cereals and grasses and other fungal diseases (Pietrusińska et al. 2019).

According to Ma et al. (2018), the significance of breeding for powdery mildew resistance depends not only on its effectiveness for disease control but also on the agronomic performance of its donor (Zhao et al. 2012). Thus the identification of novel genes from elite wheat germplasm is a smart outlook for the rapid genetic improvement of resistance.

12.3.3 Effectiveness of Powdery Mildew Genes in Resistance Breeding

Cultivation of disease-resistant cultivars/varieties is an efficient method for commercial breeding, and disease control by the introgression of resistance genes enhances the durability of the variety. Host resistance is more likely to be durable when two or more resistance genes are pyramided in a single wheat variety. Information about the genetic diversity and distribution of Pm genes in a set of wheat varieties is required for the pyramiding of resistance genes.

Until now, 68 Pm genes/alleles (*Pm1–Pm68*) (*Pm8* is allelic to *Pm17*, *Pm18* = *Pm1c*, *Pm22* = *Pm1e*, *Pm23* = *Pm4c*, *Pm31* = *Pm21*) have been identified in 60 loci from common wheat and its wild relatives (Li et al. 2019; McIntosh et al. 2019) (Table 12.2). Genes encoding resistance to powdery mildew have multi-allelic sites, where selected PM genes that respond differently to Bgt isolates are located at the same locus in different genotypes. Such genes include *Pm1* (*Pm1a-1e*), *Pm2* (*Pm2a-*

Table 12.2 Genes associated with powdery mildew resistance, source, and their chromosomal location

Gene/ allele	Location	Source	References
Pm1a	7AL	*T. aestivum*	Briggle and Sears (1966)
Pm1b	7AL	*T. monococcum*	Hsam et al. (1998)
Pm1c (*Pm18*)	7AL	*T. aestivum*	Hsam et al. (1998)
Pm1d	7AL	*T. spelta*	Hsam et al. (1998)
Pm1e (Pm22)	7AL	*T. aestivum*	Ch et al. (2003)
Pm2	5DS	*T. aestivum/Ae. tauschii*	McIntosh and Baker (1970) and Briggle and Sears (1966)
Pm3a	1AS	*T. aestivum*	Briggle and Sears (1966)
Pm3b	1AS	*T. aestivum*	Briggle and Sears (1966)
Pm3c	1AS	*T. aestivum*	Briggle and Sears (1966)
Pm3d	1AS	*T. aestivum*	Zeller et al. (1993)
Pm3e	1AS	*T. aestivum*	Zeller et al. (1993)
Pm3f	1AS	*T. aestivum*	Zeller et al. (1993)
Pm3g	1AS	*T. aestivum*	Zeller and Hsam (1998)
Pm3h	1AS	*T. durum*	Zeller and Hsam (1998)
Pm3i	1AS	*T. aestivum*	Zeller and Hsam (1998)
Pm3j	1AS	*T. aestivum*	Zeller and Hsam (1998)
Pm4a	2AL	*T. dicoccum*	The et al. (1979)
Pm4b	2AL	*T. carthlicum*	The et al. (1979)
Pm4c (*Pm23*)	2AL	*T. aestivum*	Hao et al. (2008) and Mcintosh (1998)
Pm4d	2AL	*T. monococcum*	Schmolke et al. (2011)
Pm5a	7BL	*T. dicoccum*	Law and Wolfe (1966)
Pm5b	7BL	*T. aestivum*	Hsam et al. (2001)
Pm5c	7BL	*T. aestivum* ssp. sphaerococcum	Hsam et al. (2001)
Pm5d	7BL	*T. aestivum*	Hsam et al. (2001)
Pm5e	7BL	*T. aestivum*	Huang et al. (2003)
Mlxbd (*Pm5* allele)	7BL	*T. aestivum*	Huang et al. (2000)
Pm6	2BL	*T. timopheevii*	Marone et al. (2013) and Jensen and Jensen (1973)
Pm7	4BS 4BL-2RL	*S. cereale*	Hsam et al. (2003) and Friebe et al. (1994)
Pm8	1RS 1BL	*S. cereale*	Hsam et al. (1998)
Pm9	7AL	*T. aestivum*	Hsam et al. (1998)
Pm10	1D	*T. aestivum*	Tosa et al. (1987)
Pm11	6BS	*T. aestivum*	Tosa et al. (1988)
Pm12	6BS-6SS,6SL	*Ae. speltoides*	Jia et al. (1996)

(continued)

Table 12.2 (continued)

Gene/ allele	Location	Source	References
Pm12	3BL 3SS-3S, 3DL 3SS-3S	*Ae. longissima*	Ceoloni et al. (1992)
Pm14	6BS	*T. aestivum*	Tosa and Sakai (1990)
Pm15	6BS	*T. aestivum*	Tosa and Sakai (1990)
Pm16	4A	*T. dicoccoides*	McIntosh et al. (2007) and Reader and Miller (1991)
Pm17	1RS1AL	*S. cereal*	Hao et al. (2015), Heun et al. (1990) and Hsam et al. (1998)
Pm19	7D	*Ae. tauschii*	Hsam et al. (2003) and Lutz et al. (1995)
Pm20	6BS6RL	*S. cereale*	Friebe et al. (1994)
Pm21	6VS6AL	*Haynaldia villosa*	Chen et al. (1995)
Pm23	5A	*T. aestivum*	Hao et al. (2008) and McIntosh (1998)
Pm24	1DS	*T. aestivum*	Huang et al. (2000)
Pm25	1A	*T. boeoticum*	Shi et al. (1998)
Pm26	2BS	*T. dicoccoides*	Rong et al. (2000)
Pm27	6B-6G	*T. timopheevii*	Järve et al. (2000)
Pm28	1B	*T. aestivum*	Peusha et al. (2000)
Pm29	7DL	*A. ovata*	Hsam et al. (2003)
Pm30	5BS	*T. dicoccoides*	Liu et al. (2002)
Pm31 (MIG)	6AL	*T. dicoccoides*	Xie et al. (2003)
Pm32	1BL,1SS	*Ae. speltoides*	Hsam et al. (2003)
MlTd1055		*T. dicoccoides*	Ahmadi Firouzabad and Moore (2003)
Pm33	2BL	*T. carthlicum*	Zhu et al. (2005)
Pm34	5DL	*Ae. tauschii*	Miranda et al. (2006)
Pm35	5DL	*Ae. tauschii*	Miranda et al. (2007)
Pm36	5BL	*T. dicoccoides*	Blanco et al. (2008)
Pm37	7AL	*T. timopheevii*	Perugini et al. (2008)
Pm38	7DS	*T. aestivum*	Lillemo et al. (2005)
Pm39	1BL	*T. aestivum*	Lillemo et al. (2008)
Pm40	7BS	*Elytrigia intermedium*	Marone et al. (2013) and Luo et al. (2009)
Pm41	3BL	*T. dicoccoides*	Li et al. (2009)
Pm42	2BS	*T. dicoccoides*	Hua et al. (2009)
Pm43	2DL	*T. intermedium*	Marone et al. (2013) and He et al. (2009)
Pm44	3AS	*T. aestivum*	Alam et al. (2012)
Pm45	6DS	*T. aestivum*	Ma et al. (2011)
Pm46	5DS	*T. aestivum*	Gao et al. (2012)
Pm47	7BS	*T. aestivum*	Xiao et al. (2013)

(continued)

Table 12.2 (continued)

Gene/ allele	Location	Source	References
Pm49	2BS	*T. dicoccum*	Piarulli et al. (2012)
Pm50	2AL	*T. dicoccum*	Mohler et al. (2012)
Pm51	2BL	*Thinopyrum ponticum*	Zhan et al. (2014)
Pm54	6BL	*T. aestivum*	Hao et al. (2015)
Pm55	5VS	*Dasypyrum villosum*	Zhang et al. (2016)
Pm57	T2BS.2BL-2S	*Ae. Searsii*	Liu et al. (2017)
Pm58	2DS	*Aegilops tauschii*	Wiersma et al. (2017)
Pm59	7A	Afghanistan landrace PI 181256	Tan et al. (2018)
Pm60	7AL	*Triticum urartu*	Zou et al. (2018)
Pm61	4AL	*Triticum aestivum*	Sun et al. (2018)
Pm62	2VL	*Dasypyrum villosum*	Zhang et al. (2018)
Pm63	2B	Iranian landrace PI 628024	Tan et al. (2019)
Pm64	2BL	*Triticum turgidum* var. dicoccoides	Zhang et al. (2019)
Pm65	2AL	Facultative wheat cultivar Xinmai 208	Li et al. (2019)
Pm66	T4Sl S·4BL	*Aegilops longissima*	Li et al. (2020)
Pm68	2BS	*Triticum turgidum* L. var. durum Desf.	He et al. (2020)

2c, *PmX3986-2*, *PmWFJ*, *PmD57-5D*, *PmLX66*, and others), *Pm3* (*Pm3a-3j*), *Pm4* (*Pm4a-4e*), *Pm5* (*Pm5a-Pm5e*), and *Pm24* (*Pm24a-Pm24b*) (Ma et al. 2016; McIntosh et al. 2017). However, only a few PM genes have been successfully utilized in developing powdery mildew-resistant wheat cultivars such as *Pm2*, *Pm4*, *Pm6*, *Pm21*, and *Pm30* which confer resistance against the pathogen isolates (Huang et al. 2012; Li et al. 2011; Liu et al. 2008).

Pm3, existing in seven functionally distinct alleles (*Pm3a* to *Pm3g*), is the first wheat powdery mildew resistance gene to be cloned (Srichumpa et al. 2005; Yahiaoui et al. 2006). *Pm3* alleles being the largest allelic series provide additional diversity for resistance toward different Bgt isolates while enriching the genetic basis for PM resistance breeding in wheat. The *Pm4* locus is one of the most widely recognized loci of genetic resistance to powdery mildew, and *Pm4a* resistance allele has been used in breeding for several decades (Li et al. 2017). Further studies on the function characterization of this gene might provide an insight into the effectiveness of this gene toward the newly emerging Bgt pathotypes.

Pm6 is another important gene transferred from *T. timopheevi*, and the resistance conferred by *Pm6* exhibits moderate resistance at the seedling stage and high resistance at the adult plant stage in a distinctly developmental stage-dependent manner (DDSDM) (Bennett 1984). Purnhauser et al. (2011) reported that *Pm6* resistance has been found to be enhanced especially when used in combination

with *Pm2*. The fine mapping and further cloning of *Pm6* will facilitate not only the better utilization of *Pm6* in wheat breeding but also a better understanding of the molecular mechanism of DDSDM resistance in plants.

Another popular gene *Pm8* derived from the 1RS chromosome of rye has made a significant contribution to powdery mildew resistance in wheat since the 1990s. But the linked secalin glycopeptide in 1RS resulted in a poor flour quality (Friebe et al. 1989; Lee et al. 1995) making it unsuitable for breeding program. Other effective PM resistance genes that continue to be resistant include *Pm1c*, *Pm12*, and *Mlxbd*. But they have not been exploited in the present breeding program due to the poor agronomic traits associated with either alien chromosome segments or un-adapted genetic backgrounds (Duan et al. 1998; Qiu and Zhang 2004). Also, the PM genes derived from landraces usually have been reported to be linked with poor agronomic performance and require several backcrosses to eliminate the associated linkage drag (Xu et al. 2015; Li et al. 2020a, b). Thus, breeding for PM resistance depends not only on the effectiveness of the gene but also on the agronomic performance of its donor source (Zhao et al. 2012; Ma et al. 2018).

Though most of the resistant genes continue to be resistant, it could not be exploited due to poor agronomic traits associated with the alien translocations. For instance, *Pm16* has been reported to give broad-spectrum resistance to wheat PM, but the linkage drag associated with this gene caused physiological deficiencies of yield leading to 15% yield loss (Summers and Brown 2012) which is the major constraint in the deployment of this gene.

Other factors such as the number of Bgt isolates virulent to particular gene should be considered while evaluating the putative resistance genes in bread wheat. *Pm2a*, which is believed not to be highly effective against Bgt isolates, remained effective in some parts of the world (Miedaner and Flath 2007); thus, this allele offers a novel source to be pyramided with other PM resistance genes in future breeding programs. Similarly, the recently identified *Pm41* gene was reported to be highly resistant to Bgt isolates, and it is as a valuable and never exploited powdery mildew resistance gene. Thus, *Pm41* could make an important contribution to wheat breeding through gene stacking.

An increased effort is required to explore new powdery mildew resistance genes and to improve the agronomic traits with currently identified genes. Though conventional wheat breeding has been remarkably successful, it is generally subjective, inefficient, and unable to achieve stable improvement (Gupta et al. 2010). Marker-assisted selection can successfully provide a valuable complement to conventional breeding (Gupta et al. 2010). With the advent of closely linked molecular markers, marker-assisted selection (MAS) can facilitate the elimination of adverse genes and accelerate breeding progress (Jiang 2015).

12.3.4 Molecular Markers Linked to Powdery Mildew Resistance Gene

Molecular markers that are tightly linked to the resistance genes can be used to identify the resistance of the wheat varieties in early generations (Gupta et al. 2010). Molecular markers tightly linked to the QTLs and R genes greatly facilitate in marker-assisted breeding and pyramiding of QTLs (Tucker et al. 2006). Molecular markers aid in the selection of resistant lines during the early growth stages and could be evaluated for high yield with high heritability, and they can easily be found by the genetic linkage of the desirable gene group on the chromosome associated with disease resistance (Hua et al. 2009). Molecular mapping allows for the accurate detection of molecular markers that are closely linked to the resistance to powdery mildew pathogen Bgt (Yao et al. 2007). Development and application of molecular markers in crop genetics are impactful in parental selection, genetic diversity estimation, reducing linkage drag, etc. and therefore of paramount importance in genetic mapping and gene discovery (Rasheed et al. 2017).

Different molecular techniques have been used to characterize and manipulate resistance genes and to dissect different types of resistance. RFLPs were the first molecular markers that developed and used in genetic analysis, initially in humans (Botstein et al. 1980), since the early 1980s, RFLPs have been used successfully for a wide range of plant species. The RFLP was also one of the first methods used for genetic typing and also known as genetic fingerprinting or profiling or testing. Despite that the RFLP have many benefits, it is still a slow and more tedious process to screen the mapping of genes compared to some of the newer DNA analysis techniques. In this manner, RFLPs have restricted application in wheat improvement programs. More number of powdery mildew resistance genes are identified by RFLP markers in wheat, such as *Pm1*, *Pm2*, *Pm3b*, *Pm4a* (Ma et al. 1994), *Pm2* (Mohler and Jahoor 1996), *Pm6* (Tao et al. 2000), *Pm12* (Jia et al. 1996), *Pm12* (Cenci et al. 1999), *Pm17* (Hsam et al. 2000), *Pm29* (Zeller et al. 2002), etc.

In the most recent decade, the RAPD procedure dependent on the polymerase chain reaction (PCR) has been one of the most commonly used procedures for detection of resistance genes. The RAPD analysis gives a speedy and proficient screen for DNA grouping-based polymorphism at uncountable loci. The significant favorable position of RAPD incorporates that it doesn't need pre-sequencing of DNA. It is inexpensive and informal to use. Numerous powdery mildew resistance genes marked with RAPD markers, such as *Pm1* (Hu et al. 1997), *Pm1*, *Pm2*, *Pm3*, *Pm3a*, *Pm3b*, *Pm3c*, *Pm4a*, *Pm12* (Shi 1997), *Pm25* (Shi et al. 1998), *Pm12* (Cenci et al. 1999), etc.

Microsatellites or simple sequence repeated (SSR) loci, and simple sequence length polymorphisms (SSLPs), are co-dominant marker which found mostly in eukaryotes and to a lesser extent in prokaryotes more suitable for screening large populations than RFLPs. They are tandemly repeated (normally 5–20 times) in the genome with a minimum repeat length of 12 base-pairs. In recent times, several powdery mildew resistance genes are identified and mapped using SSR markers

such as *Pm27* (Järve et al. 2000), *Pm30* (Liu et al. 2002), *Pm33* (Zhu et al. 2005), *Pm34* (Miranda et al. 2006), *Pm43* (He et al. 2009), and *Pm45* (Ma et al. 2011), etc.

12.3.5 Marker-Assisted Gene Pyramiding

Marker-assisted gene pyramiding entails introducing more than one resistance gene in order to increase durability and broad-spectrum resistance. Successful accumulation/stacking of multiple powdery mildew resistance genes is possible by rational application of marker-assisted gene pyramiding. Liu et al. (2008) successfully pyramided three powdery mildew resistance gene combinations, *Pm2* + *Pm4a*, *Pm2* + *Pm21*, and *Pm4a* + *Pm21*, into elite wheat cultivar 'Yang047'. Plants deployed with single *Pm2* gene had lower resistance, while those with *Pm4a* showed moderate resistance to powdery mildew. Notably, plants with *Pm2* + *Pm4a* showed enhanced resistance than those with single *Pm2* or *Pm4a* alone. Elkot et al. (2015) transferred two powdery mildew resistance genes *PmTb7A.1* and *PmTb7A.2* from *T. boeticum* into *T. aestivum* via *T. durum* (three-way cross) through marker-assisted backcross. Pietrusińska et al. (2011) pyramided *Pm21* gene along with two leaf rust resistance genes into *T. aestivum*. Zheng et al. (2020) stacked powdery mildew resistance gene, yellow rust resistance (*Yr26*), and high-quality glutenin subunits Dx5 + Dy10 into the dwarf mutant wheat cultivar. Using the gene-linked markers, among 60 Chinese wheat cultivars, 24 cultivars are detected to carry *Pm4b* gene in combination with other Pm resistance genes (*Pm2* + *Pm4b* + *Pm8*) in cultivars Xinxuan 2039, Lankao 008, and Zhengmai 366, while Yumai 368 was detected with (*Pm2* + *Pm4b* + *Pm6*) multiple genes (Mwale et al. 2017).

Zhang et al. (2002) identified 11 wheat lines stacked with multiple genes *Pm4b*, *Pm12*, and *Pm21* that showed high degree of resistance toward Bgt than single gene. Zhou et al. (2005) developed a resistant line (VPM1/7∗Bainong 3217 F4) against Bgt which had promising agronomic traits in addition to powdery mildew resistance. Later, Wu et al. (2018) characterized the stability of PM resistance gene, *Pm4b*, in wheat line VPM1/7∗Bainong 3217 F4 using 46 Bgt isolates and also developed 4 SNP and 3 SSR markers using BSR-Seq technique. Koller et al. (2018) pyramided transgenic lines with *Pm3* allelic series and identified enhance powdery mildew resistance in field conditions compared to the parental lines transformed with single *Pm3* alleles. Pyramiding of quantitative trait loci (QTLs) can be an alternate effective approach for developing durable resistance to powdery mildew in wheat. Simeone et al. (2020) mapped 18 QTL for APR powdery mildew resistance from 1AS, 2BS, 3BL, 4BL, and 7BS and 3 QTL for SR on 3BS chromosome region. Bai et al. (2012) identified QTLs responsible for adult plant resistance (APR) to powdery mildew in superior Chinese cultivars Bainong 64 and Lumai 21 and pyramided the same four QTLs and three QTLs, respectively, which showed high degree of resistance against Bgt. Strategy of pyramiding different PM resistance genes offers better protection for wheat against powdery mildew and provides a way of utilizing resistance gene resources for breeding new types of resistance lines and cultivars, which will have significance not only in breeding practice but also in theoretical research.

12.4 Advance in Powdery Mildew Resistance Breeding

Expanding the genetic tools by using genetic modification (GM) and genome editing is one of the foremost ways to enhance disease resistance. Genetic improvement for wheat breeding through GM enables easy transfer of resistance genes from one species to another compared to conventional crossing and also overcomes sexual incompatibilities. Obtaining stable inheritance of traits is often challenging due to the polyploid nature of common wheat. High-throughput genotyping platforms have established their potential role within the estimation of genetic diversity, construction of the high-density genetic maps, dissecting polygenic traits, and better understanding their interactions through GWAS (genome-wide association studies), QTL mapping, isolation of R genes, etc.

Wheat genome sequence (17 Gb) in a good quality is the hindrance of exploring the relationship between genotype and phenotype as it is 40 times bigger than rice (0.43 Gb) and 126 times bigger than *Arabidopsis thaliana* (0.125 Gb) (Brande and Moscou Matthew 2014). This poses a research barrier over the years. Until then, various technologies have been developed to reduce the complexity of genomes before sequencing. High-throughput genotyping platforms such as DArT-Seq, SNPs, GBS markers, and population-specific tGBS (targeted genotyping-by-sequencing) have accelerated the precise mapping of genomic regions sustaining rust and powdery mildew resistance (Qureshi et al. 2018; Nsabiyera et al. 2020).

12.4.1 Genome-Wide Association Studies (GWAS)

Genome-wide association studies (GWAS) is primarily focused on quickly screening germplasm collection for genes of interest and to capture historical recombination to obtain high-resolution mapping at a selected locus (Babu et al. 2020). As an advantage over mapping in bi-parental populations, this approach makes use of already existing natural populations, accounting for more genetic diversity at a given locus among the varied individuals. It relies on the recombination events that occur throughout the evolutionary process of germplasm (Yao et al. 2019). Four powdery mildew resistance QTLs have been identified using SNP genotyping-based genetic linkage analysis (Jia et al. 2018). Simeone et al. (2020) evaluated 221 accessions of wild and cultivated genotypes belonging to seven *T. turgidum* subspecies against Bgt; among them three QTL for SR (QPm.mgb-3BL.3, QPm.mgb-5AL.2, QPm.mgb-7BS.2) were mapped on chromosome assumed to be a new source conferring resistance to wheat powdery mildew.

Focused Identification of Germplasm Strategy (FIGS) is a rational method that provides information of specific plant traits along with the geographic and agro-climatic information. Using the FIGS strategy, Bhullar et al. (2009) identified 7 alleles for PM resistance from 1320 bread wheat landraces out of large collection of 16,089 accessions. Similarly, Vikas et al. (2020) exploited FIGS strategy to identify collection of accessions which had the maximum probability of gene of interest from a collection of 19,460 accessions of wheat.

12.4.2 Exome Capture

A high-throughput next-generation sequencing (NGS) technology can be used to assess genome-wide diversity in a single step. High-density SNP genotyping arrays and NGS have aided in the molecular detection of powdery mildew resistance genes/QTLs in wheat (Chao et al. 2019). Exome capture is an alternate genomic approach to SNP arrays also known as whole exome sequencing (WES) (King et al. 2015). The bread wheat exome constitutes only 1–2% of the total genome size of the targeted sequencing of the protein-coding portion of the genome. Such sequence can be specifically accessed by "exome capture." Similar to Southern blotting, exome capture tolerates a high mismatch, thereby allowing efficient capture of diverged homologous sequence space in tetraploid and hexaploid wheat sequences (Saintenac et al. 2011; Henry et al. 2014). Wendler et al. (2014) studied the targeted genome complexity reduction strategy focused on exome sequencing, resulted in the discovery of markers in the cultivated and wild relatives of barley and in wheat (Allen et al. 2012). Genetic diversity and variations in wheat and barley genomes have been extensively explored using exome capture assays (Mascher et al. 2012). Ingvardsen et al. (2019) used a TILLING population for which the captured exome sequence of >1500 lines is available to obtain *Mlo*-based powdery mildew mutations in tetraploid wheat "Kronos." The most common *R* gene encoded products such as nucleotide-binding leucine-rich repeat domain-containing (NB-LRR) proteins are confined to a smaller fraction of a plant exome. A typical plant genome is populated by several hundred *R* genes of the *NB-LRR* class (Meyers et al. 2003), and thus exome capture is an efficient method to identify and explore the plant exomes in mutant populations (Henry et al. 2014; King et al. 2015).

As the wheat genome (17 Gb in size) is too large to work, exome capture and sequencing is one of the approaches which greatly reduces sequencing volume and is highly cost efficient. It also covers the entire coding regions and reveals sufficient mapping information (Mo et al. 2018).

12.4.3 Genotype-by-Sequencing

Genotype-by-sequencing (GBS), an alternative genotypic approach, was presented by Elshire et al. (2011). In contrast to exome capture arrays, it does not rely on a fixed set of SNPs and its reference genome, whereas GBS involves genome complex reduction strategy followed by restriction enzyme-based sequencing allowing marker discovery focused on population-specific and genome wide studies. GBS-based labeling genomic regions conferring resistance genes in germplasm collections enables the discovery of resistance genes absent in reference genomes (Sanchez-Martin and Keller 2019). Exome capture or SNP-based arrays relying on reference genomes will miss out germplasm collection-specific resistance genes, while in this regard GBS acts an excellent genomics-assisted breeding approach for de novo platform. Cheng et al. (2020) studied the diversity of known powdery

mildew resistance gene loci among Chinese wheat germplasm against the whole genome using genotyping-by-sequencing (GBS).

12.4.4 TILLING

Polyploid wheat tolerates a high mutational load compared with diploid species (Uauy et al. 2017). TILLING (Targeting Induced local Lesions in Genomes) reverse genetics methodology that integrates chemical mutagenesis with a high-throughput detection of single nucleotide mutations of target of interest in mutagenized populations. McCallum and coworkers were the first to introduce Targeting Induced Local Lesions in Genomes (TILLING), 20 years ago (McCallum et al. 2000). Acevedo-Garcia et al. (2016) reported the advantage of the non-transgenic TILLING technology to select partial loss-of-function alleles of TaMlo, the orthologue of the barley Mlo (mildew resistance locus o) gene which is known to confer durable broad-spectrum powdery mildew resistance.

Eco-tilling (Ecotype-Targeting Induced Local Lesions IN Genome) is a modified procedure of TILLING, and it relies on the enzymatic cleavage of hetero-duplexed DNA with single-strand-specific nuclease followed by detection through Li-Cor genotypes (Gokidi et al. 2017). It can be used to identify polymorphisms from within a naturally occurring population of crop plants (Comai et al. 2004). It can be used to characterize phylogenetic diversity, and the technique can help to identify important alleles within cereal crops. Eco-tilling is a relatively underexplored method now possible for the characterization of wheat disease resistance (Bhullar et al. 2009).

12.4.5 Mlo Proteins

The mildew resistance locus o (MLO)-based resistance trait was first characterized in barley where a loss of function mutation in an MLO gene conferred broad resistance against Bgt pathotypes which was later reported in wheat (Acevedo-Garcia et al. 2016). In 1942, Jorgensen was first to demonstrate and exploit the MLO-susceptible gene in barley toward *Blumeria graminis f.* sp. hordei (Bgh) (Jorgensen 1992). On resistant mlo mutant plants, *Blumeria graminis* pathogenesis is terminated at penetration stage, and consequently, fungal sporelings do not form haustoria inside host cells (Aist et al. 1987).

Freialdenhoven et al. (1996) identified two genes responsible for MLO resistance in barley, *Ror1* and *Ror2*. Thenceforth, MLO genes have been reported in rice (OsMLO3) and TaMlo-A1, B1, and D1 (Konishi et al. 2010) in *Triticum aestivum*, located on chromosomes 5AL, 4BL, and 4DL (Elliott et al. 2002).

In wheat, in contrast to barley, no mlo mutants in natural condition were reported (Acevedo-Garcia et al. 2016). *Mlo* genes are largely conserved eukaryotic gene family among the plant kingdom, with comparative studies showing that wheat and barley reflect conserved similarity in genome structure (Kang et al. 2020).

Similarly, coevolution of host-specific pathogens Bgt and Bgh occurred displaying gene collinearity (Mayer et al. 2011; Oberhaensli et al. 2011). The functional characterization of barley *Mlo* genes should be able to assist exploration of wheat *mlo*-based resistance because *TaMlo* (MLO in Barley Orthologue of Wheat) shows about 88% similarity to that of barley (Elliott et al. 2002).

In hexaploid wheat, three (*TaMlo* homologs) orthologs of the barley *Mlo* gene were located on ABD genome (Elliott et al. 2002). As of now eight bread wheat MLO members were identified (Konishi et al. 2010). Rakszegi et al. (2010) identified *Tamlo* mutants in spring bread wheat cv. Cadenza by TILLING approach using ethyl methane sulfonate (EMS). In hexaploid wheat, TILLING-derived *Tamlo* (TaMlo-A1, TaMlo-B1, and TaMlo-D1) missense mutants provided partial protection against Bgt while enhanced the resistance toward parental type (Acevedo-Garcia et al. 2016). Recently, Ingvardsen et al. (2019) tested *mlo* mutants in tetraploid durum wheat (*Triticum turgidum* var. *durum*). TILLING partial loss-of-function mutants of "susceptibility genes" showed mild pleiotropy (Huckelhoven et al. 2012). Gruner et al. (2020) observed no undesired pleiotropic phenotypes such as early signs of leaf senescence or spontaneous callose deposits in leaf mesophyll cells in *Tamlo* triple mutants in contrary to the mlo mutants of barley.

12.4.6 TALENS-Derived Bgt Resistance

In hexaploid bread wheat, the transgenic approach mediated by TALEN (transcription activator like effector nucleases) leads to complete resistance against the pathotypes of *Bgt* (Wang et al. 2014). TALEN (transcription activator-like effector nuclease) genome editing technology was used to generate transgenic winter wheat plants containing simultaneous knockout lesions in the three TaMlo homologs (Acevedo-Garcia et al. 2016). Gruner et al. (2020) reported that TALEN-derived triple mutant was completely resistant toward Bgt (5% host cell entry) and was highly heritable, whereas the respective susceptible parental wild-type line, cv. KN199, had an entry rate (71%) and cv. Cadenza (78%). Study also showed that TALENS derived TaMlo mutants when subjected to study the infection phenotypes against pathogens *Zymoseptoria tritici*, and *Magnaporthe oryzae* pv. *Triticum* (*MoT*), respective mutants were highly susceptible to the pathogens and showed high degree of resistance towards Bgt.

12.4.7 CRISPER

CRISPR/Cas9 (clustered regularly interspaced short palindromic repeats)-mediated knockout facilitates enhanced powdery mildew resistance in wheat (Wang et al. 2014). In wheat, the first successful experiment using the CRISPR/Cas9 system was editing of *TaMLO*, a powdery mildew resistance locus (Wang et al. 2014). In the above study, all six copies of TaMlo were simultaneously mutated, and the edited plants exhibited resistance toward the powdery mildew fungus *Blumeria graminis f.*

sp. *tritici* (Bgt). The Cas9-mediated gene was used to generate and induce variation in hexaploid wheat. CRISPR/Cas9 technology is less expensive, more versatile, and easier to design and has thus largely overtaken the other genome editing technologies (Hilscher et al. 2017).

12.4.8 Taedr1-Basal Resistance for Bgt

EDR1 (enhanced disease resistance 1) is another type of negative regulator apart from MLO which plays a negative role in defense mechanism against powdery mildew. Dangl and Jones (2001) suggested basal resistance toward pathogen infection as quantitative disease resistance. It is highly conserved in many species of the plant kingdom (Frye et al. 2001). Mutation of EDR1 was found to confer resistance to PM but more of a basal resistance (Huckelhoven 2005). Zhang et al. (2017) found that EDR1 would be a source for enhancing powdery mildew resistance in wheat. Resistance in EDR1 is accompanied by the accumulation of callose marginal growth reduction and mildew-induced mesophyll cell death (Frye and Innes 1998).

Zhang et al. (2017) exploited CRISPR/Cas9 technology and generated *Taedr1* wheat plants by targeting all three homologs of wheat. The *Taedr1* mutants were resistant to *Bgt* but without mildew-induced cell death. Wang et al. (2014) speculated that *Taedr1* plants might confer resistance to other wheat pathogens also. Thus, the targeted knockout of negative regulators and/or susceptibility genes via genome editing represents a powerful approach for plant disease resistance breeding.

12.4.9 Candidate Gene Approach in Wheat

In plant kingdom, the major classes of disease resistance genes include NLR proteins and protein kinases (PKs). The candidate gene approach has emerged as a promising method of merging QTL analysis with the extensive data available on the cloning and characterization of genes involved in plant defense (Faris et al. 1998). Hitherto, more than 100 Bgt QTLs have been mapped in homologous chromosome groups from different mapping studies, with some of them being positioned in the same marker intervals (Kang et al. 2020). Infection of Bgt in wheat may be suppressed by host immune responses leading through the massive secretion of small virulence proteins called effectors (Bourras et al. 2019). Wheat plant genome encodes hundreds of cloned *R* genes that code for NB-LRR proteins which directly or indirectly recognize these effectors from pathogens to activate defense responses.

To date, seven R genes, namely, *Pm3* allelic series (Brunner et al. 2010), *Pm8* (Hurni et al. 2012), *Pm2* (Sánchez-Martín et al. 2016), *Pm17* (Singh et al. 2018), *Pm21* (Cao et al. 2011; He et al. 2018; Xing et al. 2018), *Pm60* (Zou et al. 2018), and *Pm1a* (Hewitt et al. 2021), for powdery mildew resistance have been cloned. These genes encode NB-LRR immune receptors that recognize pathogen effectors and activate effector-triggered immunity (ETI) with known resistance for powdery

mildew specificities. R-gene enrichment sequencing (AgRenSeq) approach is reference-free which was successfully demonstrated to clone R genes from alien sources of domesticated crops which expedite the discovery of new NLR genes and counteracts the pathogen profile when most pathogenic strains are used (Martin et al. 2019). These cloned genes confer high level of resistance against Bgt usually culminating in the induction of a type of programmed cell death known as the hypersensitive response (HR) (Collier and Moffett 2009). However, *Pm38/Yr18/Lr34/Sr57* and *Pm46/Yr46/Lr67/Sr55* also code for NLR proteins, but they provide partial resistance to powdery mildew and rust diseases in adult plants. These genes encode an ATP binding cassette (ABC transporter) and an altered hexose transporter, respectively.

12.4.10 R Gene Cassettes

Stacking of PM genes is an important strategy to extend the life span of race-specific resistance genes (Li et al. 2014; Burdon et al. 2014). Taking benefit from effector-assisted breeding, stacking of multiple R gene-based resistance provides robust and broad-spectrum disease resistance (Martin et al. 2019). Molecular stacking is an effective alternative to conventional gene stacking through marker-assisted selection. Through molecular stacking, multiple R gene cassettes can be assembled on to one plasmid and then introduced as a cluster at a single genetic locus through plant transformation (Que et al. 2010) in routine breeding (Ainley et al. 2012). But the length of the DNA insert delimits the number of genes to be inserted into the vector (Que et al. 2010).

12.4.11 Virus-Induced Gene Silencing

The resistance action of candidate genes can be studied by the complementary functional assay—virus-induced gene silencing (VIGS) (Lee et al. 2012). VIGS offers a fast and rapid transient assay for silencing of gene expression. The most widely used vectors for VIGS in wheat are those derived from barley stripe mosaic virus (BSMV), a plant virus with a tripartite RNA genome (RNAα, RNAβ, and RNAγ) that readily spreads throughout tissues following mechanical rub-inoculation onto the leaves.

Bhullar et al. (2009) used combined strategy of VIGS and transient transformation assay to assign the function of previously undescribed *Pm3* alleles. Moreover, through VIGS, silencing of the TaMlo homologs leads to powdery mildew resistance in wheat (Varallyay et al. 2012). Zhang et al. (2017) reported the knockdown of TaEDR1 (negative regulator of MLO protein) in mutant lines with VIGS and observed that these lines were showing enhanced resistance to powdery mildew. Xing et al. (2018) used barley stripe mosaic virus-induced gene silencing (BSMV-VIGS) for targeting NBS domain and the LRR domain to evaluate the function of NLR1-V gene in *Pm21*. Later, he and his coworkers identified and investigated two

candidate genes, viz., DvRGA1 and DvRGA2 (CC-NBC-LRR proteins), for *Pm21*-mediated resistance in wheat variety Yangmai 18 using BSMV-VIGS. Results suggested that silencing of DvRGA2 allowed abundant development of Bgt colonies with disease symptoms and fungal sporulation on the leaves, rather than DvRGA1 gene. Thus it was identified that DvRGA2 is associated with *Pm21* resistance (He et al. 2018). Zou et al. (2018) validated the function of *Pm60*-NB LRR gene through VIGS and transient expression assays. VIGS was also useful in wheat genotypes that were difficult to transform and in those for which mutant/TILLING populations were unavailable. It can be also be used for simultaneous silencing of all homologs without the need for further genetic crosses.

12.5 Challenges in Breeding for Powdery Mildew Resistance

For the past few decades, developing disease-resistant wheat varieties mainly relied on the conventional breeding. Increased yield with desirable agronomic characters is the main prioritization of the modern wheat breeding community. This has led to the loss of genetic diversity for disease resistance. Resistant genes from alien sources of wheat were transferred to commercial cultivars through conventional breeding approaches. However, introduction of alien segments always was associated with a linkage drag. The deleterious effect associated with the alien gene may indirectly affect the yielding components of the host plant. Thus the potential linkage drag limits the transfer of PM genes to wheat. Also it might take several years for fixing a resistant gene in a particular wheat background through conventional breeding methods (Cowling 2012). Thus isolation of resistance genes from alien sources and recombination between alien chromosomes and wheat chromosomes are largely limited (Lukaszewski 2000; Mago et al. 2002; Qi et al. 2007).

These limitations have been largely overcome by combining genetic, cytogenetic, and molecular methods together. Recently, more number of molecular markers linked to the resistance genes has been identified. Precise physical mapping of powdery mildew resistance genes is very crucial for the identification of candidate genes (Kang et al. 2020). Locating the inserted alien segments in the wheat genome is also important for the successful development of resistant cultivar (Dundas et al. 2007). Genetic and cytogenetic methods need to be put into practice to promote recombination and minimize the adverse effects brought by the alien chromatin.

Precise genotyping and phenotyping are necessary for the identification of novel PM-resistant genes. Changing climatic conditions often influence the phenotypic data. In case of quantitative resistance, response of the host is very difficult to be scored compared to qualitative resistance. Time of scoring and nature of phenotypic expression are very crucial in assessing accurate level of severity. Visual scoring for the estimation of disease severity is always subjective and error prone particularly when large populations are screened (Poland and Nelson 2011).

Transgenic-based technologies are also influenced by the environmental factors as there might be biological and physiological interaction of genetic factors with the transgene expression (Ueda et al. 2006). Also, there are limitations in

broad-spectrum application of molecular techniques in developing mildew-resistant lines as it is more expensive. Certain uncontrolled transgene insertion might also be associated with detrimental effects on plant growth and development (Kang et al. 2012). Yet, this approach involves the direct transfer of the functional genes eliminating the linkage drag (Jacobsen and Schouten 2007) and thus offers a long-term solution to global agricultural challenges. Identified trait-linked SNPs can be converted into allele-specific PCR assays which can be breeder friendly. Using R gene cassettes, despite the advantages of molecular stacking, the number of genes that can be introduced through molecular stacking is often restrained by the limit in the length of the DNA insert that can be put into a vector (Que et al. 2010). This limitation can be overcome if DNA fragments can be sequentially inserted at the same genomic target. Recent breakthroughs in genome editing technologies in plants enable such targeted insertion of DNA fragments in diverse crop species (Kumar et al. 2016; Voytas 2012).

12.6 Conclusions and Future Prospects

Significant breakthrough in the development of powdery mildew-resistant wheat varieties was achieved after the de novo sequencing of whole wheat genome. Marker-based approach is an effective method in the selection of target genes. Also, these markers might make it possible to identify novel genes for the development of powdery mildew-resistant wheat varieties. In future, strategies that combine conventional and molecular approaches for easy and rapid characterization of useful germplasm might be necessary for categorizing resistant genes.

Discovering and introducing novel sources of resistance to powdery mildew either from common wheat background or other wheat-related species should be the major objective of the wheat breeding. Chromosome micromanipulation and microinjection techniques are new and effective technology to be utilized in the future for the introduction of resistance to mildew into common wheat.

Generally, selection of lines with quantitative genes is difficult as extensive tests on selected mildew population are required and there are no proper methods to select the appropriate plant. The process is time-consuming and laborious. Thus a novel rapid selection technique for phenotyping seedling and adult plants is the need of the hour. Additionally, information regarding the genetic diversity and distribution of PM genes in hexaploid wheat is necessary for the enriching the resistance basis to PM in wheat. Interspecific and wide crosses will still continue as an effective strategy for developing resistant lines to mildew.

References

Acevedo-Garcia J, Spencer D, Thieron H, Reinstädler A, Hammond-Kosack K, Phillips AL (2016) mlo-based powdery mildew resistance in hexaploid bread wheat generated by a non-transgenic TILLING approach. Plant Biotechnol J 15:367–378. https://doi.org/10.1111/pbi.12631

Ahmadi Firouzabad A, Morre K (2003) Chromosomal location of powdery mildew resistance gene Td1055 in wild emmer wheat (T. dicoccoides) accessions TA1055 and TA1150. Proceedings 10th International Wheat Genetics Symposium Instiuto Sperimentale per la Cerealcoltura, Rome, Italy, (Pogna NE, Romano N, Pogna EA & Galterio G eds.) Vol 3: 1090-1092

Ainley WM, Sastry-Dent L, Welter ME, Murray MG, Zeitler B, Amora R, Corbin DR, Miles RR, Arnold NL, Strange TL et al (2012) Trait stacking via targeted genome editing. Plant Biotechnol J 11:1126–1124

Aist JR, Gold RE, Bayles CJ (1987) Evidence for the involvement of molecular components of papillae in mlo resistance to barley powdery mildew. Phytopathology 77:17–32. https://doi.org/10.1016/0885-5765(88)90040-9

Alam MA, Mandal MSN, Wang C, Wanquan J (2012) Chromosomal location and SSR markers of a powdery mildew resistance gene in common wheat line N0308. Afr J Microbiol Res 7(6): 477–482. https://doi.org/10.5897/ajmr12.1816

Allen AM, Barker GLA, Wilkinson P et al (2012) Discovery and development of exome-based, co-dominant single nucleotide polymorphism markers in hexaploid wheat (*Triticum aestivum* L.). Plant Biotechnol J 11:279–295. https://doi.org/10.1111/pbi.12009.

Arya HC, Ghemawat MS (1953) Occurrence of powdery mildew of wheat in neighbourhood of Jodhpur. Indian Phytopathol 15:127–122

Babu P, Baranwal DK, Harikrishna et al (2020) Application of genomics tools in wheat breeding to attain durable rust resistance. Front Plant Sci 11:567147. https://doi.org/10.3389/fpls.2020.567147

Bai B, He ZH, Asad MA, Lan CX, Zhang, Xia XC, Yan J, Chen XM, Wang CS (2012) Pyramiding adult-plant powdery mildew resistance QTLs in bread wheat. Crop Pasture Sci 63:606–611. https://doi.org/10.1071/CP12183

Baxter A, Mittler R, Suzuki N (2014) ROS as key players in plant stress signalling. J Exp Bot 65: 1229–1240. https://doi.org/10.1093/jxb/ert375

Beest D, Paveley N, Shaw M, Bosch F (2008) Disease–weather relationships for powdery mildew and yellow rust on winter wheat. Phytopathology 98:609–617. https://doi.org/10.1094/PHYTO-98-5-0609

Bennett FGA (1984) Resistance to powdery mildew in wheat: a review of its use in agriculture and breeding programmes. Plant Pathol 33:279–300. https://doi.org/10.1111/j.1265-3059.1984.tb01224.x

Bhullar NK, Street K, Mackay M, Yahiaoui N, Keller B (2009) Unlocking wheat genetic resources for the molecular identification of previously undescribed functional alleles at the Pm3 resistance locus. Proc Natl Acad Sci U S A 106:9519–9524. https://doi.org/10.1073/pnas.0904152106

Blanco A, Gadaleta A, Cenci A, Carluccio AV, Abdelbacki AMM, Simeone R (2008) Molecular mapping of the novel powdery mildew resistance gene Pm36 introgressed from T. turgidum var. dicoccoides in durum wheat. Theor Appl Genet 117:135–142

Boller T, Felix G (2009) A renaissance of elicitors: perception of microbe-associated molecular patterns and danger signals by pattern-recognition receptors. Annu Rev Plant Biol 60:379–406. https://doi.org/10.1146/annurev.arplant.57.032905.105346

Botstein D, White R, Skolnick M, Davis RW (1980) Construction of a genetic map in man using restriction fragment length polymorphisms. Am J Hum Genet 32:314–331

Bourras S, Kunz L, Xue M et al (2019) The *AvrPm3-Pm3* effector-NLR interactions control both race-specific resistance and host-specificity of cereal mildews on wheat. Nat Commun 10:2292. https://doi.org/10.1038/s41467-019-10274-1

Bowen KL, Everts KL, Leath S (1991) Reduction in yield on winter wheat in North Carolina due to powdery mildew and leaf rust. Phytopathology 81:503–511

Brande WBH, Moscou Matthew J (2014) Strategies for transferring resistance into wheat: from wide crosses to GM cassettes. Front Plant Sci 5:692. https://doi.org/10.3389/fpls.2014.00692

Braun U (1987) A monograph of the Erysiphales (powdery mildews). Beihefte zur Nova Hedwigia 89:700

Briggle LW, Sears ER (1966) Linkage of resistance to Erysiphe graminis f. sp. tritici (Pm3) and hairy glume (hg) on chromosome 1A of wheat. Crop Sci 6:559–561. https://doi.org/10.2135/cropsci1966.0011183X000600060017x

Brunner S, Hurni S, Streckeisen P, Mayr G, Albrecht M, Yahiaoui N, Keller B (2010) Intragenic allele pyramiding combines different specificities of wheat *Pm3* resistance alleles. Plant J 64(3): 433–445. https://doi.org/10.1111/j.1265-312X.2010.04342.x

Burdon JJ, Barrett LG, Rebetzke G, Thrall PH (2014) Guiding deployment of resistance in cereals using evolutionary principles. Evol Appl 7:609–624. https://doi.org/10.1111/eva.12175

Cao A, Xing L, Wang X, Yang X, Wang W, Sun Y et al (2011) Serine/threonine kinase gene *Stpk-V*, a key member of powdery mildew resistance gene *Pm21*, confers powdery mildew resistance in wheat. Proc Natl Acad Sci U S A 108:7727–7732. https://doi.org/10.1073/pnas.1016981108

Cenci A, D'Ovidio R, Tanzarella OA, Ceoloni C, Porceddu E (1999) Identification of molecular markers linked to *Pm12*, an *Aegilops longissima* gene conferring resistance to powdery mildew in wheat. Theor Appl Genet 98:448–454. https://doi.org/10.1007/s001220051090

Ceoloni C, Del Signore G, Ercoli L, Donini P (1992) Locating the alien chromatin segment in common wheat- Aegilops longissima mildew resistance transfers. Hereditas 116:239–245

Ch S, Hsam SL, Hartl L, Zeller FJ, Mohler V (2003) Powdery mildew resistance gene Pm22 in cultivar Virest is a member of the complex Pm1 locus in common wheat (Triticum aestivum L. em Thell.). Theor Appl Genet 106(8):1420–1424. https://doi.org/10.1007/s00122-002-1187-7

Chao KX, Su WW, Wu L, Su B, Li Q, Wang B et al (2019) Molecular mapping of a recessive powdery mildew resistance gene in wheat cultivar Tian Xuan 45 using bulked segregant analysis with polymorphic single nucleotide polymorphism relative ratio distribution. Phytopathology 109:828–838

Chen XM (2005) Epidemiology and control of stripe rust (*Puccinia striiformis f. sp. tritici*) on wheat. Can J Plant Pathol 27:314–337. https://doi.org/10.1080/07060660509507230

Chen P, Liu D (1982) Cytogenetic studies of hybrid progenies between *Triticum aestivum* and *Haynaldia villosa*. J Nanjing Agric Coll 5(4):1–16. https://doi.org/10.1007/s11427-008-0048-2

Chen PD, Qi LL, Zhou B, Zhang SZ, Liu DJ (1995) Development and molecular cytogenetic analysis of wheat-*Haynaldia villosa* 6VS/6AL translocation lines specifying resistance to powdery mildew. Theor Appl Genet 91:1125–1129. https://doi.org/10.1007/BF00223930

Chen SQ, Huang ZF, Dai Y, Qin YY, Zhang LL, Gao Y et al (2012) The development of 7E chromosome-specific molecular markers for *Thinopyrum elongatum* based on SLAF-seq technology. PLoS One 8:e65122. https://doi.org/10.1271/journal.pone.0065122

Cheng B, Ding YQ, Gao X et al (2020) The diversity of powdery mildew resistance gene loci among wheat germplasm in Southwest China. Cereal Res Commun 48:65–70. https://doi.org/10.1007/s42976-020-00015-2

Collier SM, Moffett P (2009) NB-LRRs work a "bait and switch" on pathogens. Trends Plant Sci 14:521–529

Comai L, Young K, Till BJ, Reynolds SH, Greene EA, Codomo CA, Enns LC, Johnson JE, Burtner C, Odden ARE (2004) Efficient discovery of DNA polymorphisms in natural populations by Eco TILLING. Plant J 37:778–786

Conner RL, Kuzyk AD, Su H (2003) Impact of powdery mildew on the yield of soft white spring wheat cultivars. Can J Plant Sci 83:725–728

Cowger C, Parka R, Marshall O (2009) Appearance of powdery mildew of wheat caused by *Blumeria graminis f. sp. tritici* on Pm17-bearing cultivars in North Carolina. Plant Dis 93: 1219. https://doi.org/10.1094/PDIS-93-11-1219B

Cowger C, Miranda L, Griffey C, Hall M, Murphy JP, Maxwell JJ (2012) Wheat powdery mildew. In: Sharma I (ed) Disease resistance in wheat. CAB International, Cambridge, pp 84–119

Cowling WA (2012) Sustainable plant breeding. Plant Breed 122:1–9. https://doi.org/10.1111/pbr.12026

Cox TS, Raupp WJ, Wilson DL, Gill BS, Leath S, Bockus WW, Browder LE (1992) Resistance to foliar diseases in collection of *Triticum tauschii* germplasm. Plant Dis 76(10):1061–1064. https://doi.org/10.1094/PD-76-1061

Cunfer BM (2002) Powdery mildew. In: Curtis B, Rajaram S, Macpherson H (eds) Bread wheat: improvement and production (FAO Plant Production and Protection Series No. 30). Food and Agriculture Organization of the United Nations, Rome

Dangl JL, Jones JDG (2001) Plant pathogens and integrated defence responses to infection. Nature 411:826–833. https://doi.org/10.1038/35081161

Dinoor A (1974) Role of wild and cultivated plants in the epidemiology of plant diseases in Israel. Annu Rev Phytopathol 12:412–436. https://doi.org/10.1146/annurev.py.12.090174.002212

Dodds PN, Rathjen JP (2010) Plant immunity: towards an integrated view of plant–pathogen interactions. Nat Rev Genet 11:539–548. https://doi.org/10.1038/nrg2812

Du J, Verzaux E, Chaparro-Garcia A, Bijsterbosch G, Keizer LP, Zhou J, Liebrand TW, Xie C, Govers F, Robatzek S (2015) Elicitin recognition confers enhanced resistance to Phytophthora infestans in potato. Nat Plants 1:15034. https://doi.org/10.1038/nplants.2015.34

Duan X, Sheng B, Zhou Y, Xiang Q (1998) Monitoring of the virulence population of *Erysiphe graminis* f. sp. tritici. Acta Phytophylac Sin 25:31–36

Dundas IS, Anugrahwati DR, Verlin DC, Park RF, Bariana HS, Mago R et al (2007) New sources of rust resistance from alien species: meliorating linked defects and discovery. Aust J Agric Res 58:545–549. https://doi.org/10.1071/AR07056

Elkot AF, Chhuneja P, Kaur S, Saluja M, Keller B, Singh K (2015) Marker assisted transfer of two powdery mildew resistance Genes PmTb7A.1 and PmTb7A.2 from *Triticum boeoticum* (Boiss.) to *Triticum aestivum* (L.). PLoS One 10:e0128297. https://doi.org/10.1271/journal.pone.0128297

Elliott C, Zhou F, Spielmeyer W, Panstruga R, Schulze-Lefert P (2002) Functional conservation of wheat and rice *Mlo* orthologs in defense modulation to the powdery mildew fungus. Mol Plant-Microbe Interact 15:1069–1077. https://doi.org/10.1094/MPMI.2002.15.10.1069

El-Shamy MM, Emara HM, Mohamed ME (2016) Virulence analysis of wheat powdery mildew (*Blumeria graminis f. sp. tritici*) and effective genes in Middle Delta, Egypt. Plant Dis 100:1927–1930. https://doi.org/10.1094/PDIS-01-16-0120-RE

Elshire RJ, Glaubitz JC, Sun Q et al (2011) A robust, simple genotyping-by-sequencing (GBS) approach for high diversity species. PLoS One 6:e19379. https://doi.org/10.1271/journal.pone.0019379

Eser V (1998) Characterization of powdery mildew resistant lines derived from crosses between *Triticum aestivum* and *Aegilops speltoides* and *Ae. mutica*. Euphytica 100:269–272. https://doi.org/10.1023/A:1018372726968

Esmail SM, Draz IS (2017) Fungal morphogenesis tracking of *Blumeria graminis* f. sp. *tritici* on leaf freed of epicuticular wax using scanning electron microscopy. Int J Microbiol Biotechnol 2:181–188. https://doi.org/10.11648/j.ijmb.20170204.16

Everts K, Leath S, Finney P (2001) Impact of powdery mildew and leaf rust on milling and baking quality of soft red winter wheat. Plant Dis 85:423–429. https://doi.org/10.1094/PDIS.2001.85.4.423

Faris JD, Li WL, Liu DJ, Chen PD, Gill BS (1998) Candidate gene analysis of quantitative disease resistance in wheat. Theor Appl Genet 98:219–225

Freialdenhoven A, Peterhänsel C, Kurth J, Kreuzaler F, SchulzeLefert P (1996) Identification of genes required for the function of non-race-specific mlo resistance to powdery mildew in barley. Plant Cell 8:5–14. https://doi.org/10.1105/tpc.8.1.5

Frescatada-Rosa M, Robatzek S, Kuhn H (2015) Should I stay or should I go? Traffic control for plant pattern recognition receptors. Curr Opin Plant Biol 28:23–29. https://doi.org/10.1016/j.pbi.2015.08.007

Friebe B, Heun M, Bushuk W (1989) Cytological characterization, powdery mildew resistance and storage protein composition of tetraploid and hexaploid 1BL/1RS wheat-rye translocation lines. Theor Appl Genet 78:425–432. https://doi.org/10.1007/BF00265307

Friebe B, Heun M, Tuleen N, Zeller FJ, Gill BS (1994) Cytogenetically monitored transfer of powdery mildew resistance from rye into wheat. Crop Sci 34:621–625. https://doi.org/10.2125/cropsci1994.0011183X003400030003x

Friedrich S, Boyle C (1993) Wirkung unterschiedlicher Luftfeuchten auf die Production und Keimung der Konidien von *Erysiphe graminis f. sp. tritici* in vitro. J Plant Dis Prot 100:180–188. http://www.jstor.org/stable/43386159

Frye CA, Innes RW (1998) An *Arabidopsis* mutant with enhanced resistance to powdery mildew. Plant Cell 10:947–956

Frye CA, Tang D, Innes RW (2001) Negative regulation of defense responses in plants by a conserved MAPKK kinase. Proc Natl Acad Sci U S A 98:373–378. https://doi.org/10.1105/tpc.10.6.947

Gadore WY, Patwardhan PG (1965) Occurence of *Erysiphe graminis var. Tritici* in Bombay (Maharashtra). Curr Sci 34:89

Gao H, Zhu F, Jiang Y, Wu J, Yan W, Zhang Q, Jacobi A, Cai S (2012) Genetic analysis and molecular mapping of a new powdery mildew resistant gene Pm46 in common wheat. Theor Appl Genet 125:967–973

Gill BS, Raupp WJ, Sharma HC et al (1986) Resistance in *Aegilops squarrosa* to wheat leaf rust, wheat powdery mildew, greenbug, and Hessian fly. Plant Dis 70:553–556. https://doi.org/10.1094/PD-70-553

Giraldo MC, Valent B (2012) Filamentous plant pathogen effectors in action. Nat Rev Microbiol 11:800–814. https://doi.org/10.1038/nrmicro3119

Gokidi Y, Nishant Bhanu A, Chandra K, Singh MN, Hemantaranjan A (2017) Allele mining—an approach to discover allelic variation in crops. J Plant Sci Res 33(2):167–180

Griffey CA, Das MK, Stromberg EL (1993) Effectiveness of adult-plant resistance in reducing grain-yield loss to powdery mildew in winter wheat. Plant Dis 77:618–622. https://doi.org/10.1094/PD-77-0618

Gruner K, Esser T, Acevedo-Garcia J, Freh M, Habig M, Strugala R, Stukenbrock E, Schaffrath U, Panstruga R (2020) Evidence for allele-specific levels of enhanced susceptibility of wheat *mlo* mutants to the hemibiotrophic fungal pathogen *Magnaporthe oryzae* pv. *triticum*. Genes 11:517. https://doi.org/10.3390/genes11050517

Gupta PK, Langridge P, Mir RR (2010) Marker-assisted wheat breeding: present status and future possibilities. Mol Breed 26:145–161. https://doi.org/10.1007/s11032-009-9359-7

Hao Y, Liu A, Wang Y, Feng D, Gao J, Li X et al (2008) Pm23: a new allele of Pm4 located on chromosome 2AL in wheat. Theor Appl Genet 117(8):1205–1212. https://doi.org/10.1007/s00122-008-0827-y

Hao Y, Parks R, Cowger C, Chen Z, Wang Y, Bland D, Murphy JP, Guedira M, Brownguedira G, Johnson J (2015) Molecular characterization of a new powdery mildew resistance gene Pm54 in soft red winter wheat. Theor Appl Genet 128:465–476

Hartl L, Mohler V, Zeller FJ, Hsam ALK, Schweizer G (1999) Identification of AFLP markers closely linked to the powdery mildew resistance genes *Pm1*c and *Pm4*a in common wheat (*Triticum aestivum* L.). Genome 42:322–329. https://doi.org/10.1129/g98-129

Hautea RA, Coffman WR, Sorrells ME, Bergstrom GC (1987) Inheritance of partial resistance to powdery mildew in spring wheat. Theor Appl Genet 73:609–615. https://doi.org/10.1007/BF00289202

He R, Chang Z, Yang Z, Yuan Z, Zhan H, Zhang X, Liu J (2009) Inheritance and mapping of powdery mildew resistance gene *Pm43* introgressed from *Thinopyrum* intermedium into wheat. Theor Appl Genet 118:1173–1180. https://doi.org/10.1007/s00122-009-0971-z

He H, Zhu S, Zhao R, Jiang Z, Ji Y, Ji J et al (2018) *Pm*21, encoding a typical CC-NBS-LRR protein, confers broad-spectrum resistance to wheat powdery mildew disease. Mol Plant 11:879–882. https://doi.org/10.1016/j.molp.2018.03.004

He H, Liu R, Ma P, Du H, Zhang H, Wu Q et al (2020) Characterization of Pm68, a new powdery mildew resistance gene on chromosome 2BS of Greek durum wheat TRI 1796. Theor Appl Genet. https://doi.org/10.1007/s00122-020-03681-2

Henry RJ, Kettlewell PS (1996) Cereal grain quality. Chapman & Hall, London, p 488
Henry IM, Nagalakshmi U, Lieberman MC, Ngo KJ, Krasileva KV, Vasquez-Gross H, Akhunova A, Akhunov E, Dubcovsky J, Tai TH, Comai L (2014) Efficient genome-wide detection and cataloging of EMS-induced mutations using exome capture and next-generation sequencing. Plant Cell 26:1282–1297. https://doi.org/10.1105/tpc.112.121590
Heun M, Friebe B, Bushuk W (1990) Chromosomal location of the powdery mildew resistance gene of Amigo wheat. Phytopathology 80:1129–1133
Hewitt T, Müller MC, Molnár I, Mascher M, Holušová K, Šimková H, Kunz L et al (2021) A highly differentiated region of wheat chromosome 7AL encodes a *Pm1a* immune receptor that recognizes its corresponding *AvrPm1a* effector from *Blumeria graminis*. New Phytol. https://doi.org/10.1111/nph.17075
Hilscher J, Burstmayr H, Stoger E (2017) Targeted modification of plant genomes for precision crop breeding. Biotechnol J 12:1600173. https://doi.org/10.1002/biot.201600173
Hsam SLK, Zeller FJ (2002) Breeding for powdery mildew resistance in common wheat *(T. aestivum L. em Thell.)*. In: Bélanger RR, Bushnell WR, Dik AJ, Carver TLW (eds) The powdery mildews: a comprehensive treatise. APS Press, St. Paul, pp 219–238
Hsam SLK, Huang XQ, Ernst F, Hartl L, Zeller FJ (1998) Chromosomal location of genes for resistance to powdery mildew in common wheat (Triticum aestivum L. em. Thell.). 5. Alleles at the Pm1 locus. Theor Appl Genet 96:1129–1134
Hsam SLK, Mohler V, Hartl L, Wenzel G, Zeller FJ (2000) Mapping of powdery mildew and leaf rust resistance genes on the wheat-rye translocated chromosome T1BL.1RS using molecular and biochemical markers. Plant Breed 119:87–89. https://doi.org/10.1046/j.1439-0523.2000.00444.x
Hsam SLK, Huang XQ, Zeller FJ (2001) Chromosomal location of genes for resistance to powdery mildew in common wheat (Triticum aestivum L. em. Thell.). 6. Alleles at the Pm5 locus. Theor Appl Genet 102:127–133
Hsam SLK, Lapochkina IF, Zeller FJ (2003) Chromosomal location of genes for resistance to powdery mildew in common wheat (*Triticum aestivum* L. em Thell.). 8. Gene *Pm32* in a wheat *Aegilops speltoides* translocation line. Euphytica 123:367–370. https://doi.org/10.1023/A:1025738512638
Hu XY, Ohm HW, Dweikat I (1997) Identification of RAPD markers linked to the gene *Pm1* for resistance to powdery mildew in wheat. Theor Appl Genet 94:832–840. https://doi.org/10.1007/s001220050484
Hua W, Liu Z, Zhu J, Xie C, Yang T, Zhou Y, Duan X, Sun Q, Liu Z (2009) Identification and genetic mapping of *Pm42*, a new recessive wheat powdery mildew resistance gene derived from wild emmer (*Triticum turgidum var. dicoccoides*). Theor Appl Genet 119:223–230. https://doi.org/10.1007/s00122-009-1031-4
Huang XQ, Hsam SLK, Zeller FJ (2000) Chromosomal location of two novel genes for resistance to powdery mildew in Chinese landraces (Triticum aestivum L. em. Thell.). J Genet Breed 54:311–317
Huang XQ, Wang LX, Xu MX, Roder MS (2003) Microsatellite mapping of the powdery mildew resistance gene Pm5e in common wheat (Triticum aestivum L.). Theor Appl Genet 106:858–865
Huang J, Zhao ZH, Song FJ, Wang XM, Xu HX, Huang Y, An DG, Li HJ (2012) Molecular detection of a gene effective against powdery mildew in the wheat cultivar Liangxing 66. Mol Breed 30:1737–1745. https://doi.org/10.1007/s11032-012-9757-0
Huang L, Sela H, Feng LH, Chen QJ, Krugman T et al (2016) Distribution and haplotype diversity of WKS resistance genes in wild emmer wheat natural populations. Theor Appl Genet 129:921–934. https://doi.org/10.1007/s00122-016-2672-8
Huckelhoven R (2005) Powdery mildew susceptibility and biotrophic infection strategies. FEMS Microbiol Lett 245(1):9–17. https://doi.org/10.1016/j.femsle.2005.03.001
Huckelhoven R, Eichmann R, Weis C, Hoefle C, Proels RK (2012) Genetic loss of susceptibility: a costly route to disease resistance? Plant Pathol 62:56–62. https://doi.org/10.1111/ppa.12103

Hurni S, Brunner S, Buchmann G, Herren G, Jordan T, Krukowski P, Wicker T, Yahiaoui N, Mago R, Keller B (2012) Rye *Pm8* and wheat *Pm3* are orthologous genes and show evolutionary conservation of resistance function against powdery mildew. Plant J 76(6):957–969. https://doi.org/10.1111/tpj.12345

Ingvardsen CR, Massange-Sánchez JA, Borum F, Uauy C, Gregersen PL (2019) Development of mlo-based resistance in tetraploid wheat against wheat powdery mildew. Theor Appl Genet 122: 3009–3022. https://doi.org/10.1007/s00122-019-03402-4

Jacobsen E, Schouten HJ (2007) Cisgenesis strongly improves introgression breeding and induced translocation breeding of plants. Trends Biotechnol 25:219–223

Järve K, Peusha HO, Tsymbalova J, Tamm S, Devos KM, Eno TM (2000) Chromosomal location of a Triticum timopheevii-derived powdery mildew resistance gene transferred to common wheat. Genome 43:377–381. https://doi.org/10.1129/g99-141

Jensen HJ, Jensen CJ (1973) Gene Pm6 for resistance to powdery mildew in wheat. Euphytica 22: 423. https://doi.org/10.1007/BF00022656

Jia JZ, Jia J, Miller TE, Reader SM, Devos KM, Gale MD (1996) Homoeologous group 6 chromosomes maps of wheat and their application in the tagging of a mildew resistance gene *Pm12*, transferred from *Aegilops speltoides*. Theor Appl Genet 92:559–565. https://doi.org/10.1007/BF00224558

Jia A, Ren Y, Gao F, Yin G, Liu J, Guo L et al (2018) Mapping and validation of a new QTL for adult-plant resistance to powdery mildew in Chinese elite bread wheat line Zhou8425B. Theor Appl Genet 121:1063–1071

Jiang L (2015) Molecular marker-assisted breeding: a plant breeder's review. In: Advances in plant breeding strategies: breeding, biotechnology and molecular tools. pp 431–472. https://doi.org/10.1007/978-3-319-22521-0_15

Jiang J, Friebe B, Gill B (1994) Recent advances in alien gene transfer in wheat. Euphytica 73:199–212. https://doi.org/10.1007/BF00036700

Jones JD, Dangl JL (2006) The plant immune system. Nature 444(7117):323–329. https://doi.org/10.1038/nature05286

Jorgensen JH (1992) Discovery, characterization and exploitation of Mlo powdery mildew resistance in barley. Euphytica 63:141–152. https://doi.org/10.1007/BF00023919

Jorgensen JH, Jensen CI (1972) Genes for resistance to wheat powdery mildew in derivatives of *Triticum timopheevii* and *Triticum carthlicum*. Euphytica 21:121–128. https://doi.org/10.1007/BF00040557

Kang M, Heuvelink E, Carvalho SMP, de Reffye P (2012) A virtual plant that responds to the environment like a real one: the case for chrysanthemum. New Phytol 195:384–395. https://doi.org/10.1111/j.1469-8127.2012.04177.x

Kang Y, Zhou M, Merry A, Barry K (2020) Mechanisms of powdery mildew resistance of wheat—a review of molecular breeding. Plant Pathol 69:601–617. https://doi.org/10.1111/ppa.12166

Kashyap PL, Kumar S, Sharma A, Kumar RS, Jasrotia P, Singh GP (2021) Diversity and genetic architecture of resistance to powdery mildew in wheat. In: Bishnoi SK, Khan H, Singh SK, Singh GP (eds) Breeding frontiers in wheat. Agrobios (INDIA), pp 207–239

Keller M, Keller B, Schachermayr G, Winzeler M, Schmid JE, Stamp P, Messmer MM (1999) Quantitative trait loci for resistance against powdery mildew in a segregating wheat × spelt population. Theor Appl Genet 98:903–912. https://doi.org/10.1007/s001220051149

Kim NH, Hwang BK (2015) Pepper pathogenesis related protein 4c is a plasma membrane localized cysteine protease inhibitor that is required for plant cell death and defense signaling. Plant J 81: 81–94. https://doi.org/10.1111/tpj.12709

Kim W, Johnson JW, Baenziger PS, Lukaszewski AJ, Gaines CS (2004) Agronomic effect of wheat–rye translocation carrying rye chromatin (1R) from different sources. Crop Sci 44:1254–1258. https://doi.org/10.2125/cropsci2004.1254

King R, Bird N, Ramirez-Gonzalez R, Coghill JA, Patil A, Hassani-Pak K, Uauy C, Phillips AL (2015) Mutation scanning in wheat by exon capture and next-generation sequencing. PLoS One 10:e0127549

Koller T, Brunner S, Herren G, Hurni S, Keller B (2018) Pyramiding of transgenic *Pm3* alleles in wheat results in improved powdery mildew resistance in the field. Theor Appl Genet 121(4): 861–871. https://doi.org/10.1007/s00122-017-3043-9

Konishi S, Sasakuma T, Sasanuma T (2010) Identification of novel Mlo family members in wheat and their genetic characterization. Genes Genet Syst 85:167–175. https://doi.org/10.1266/ggs.85.167

Krattinger SG, Lagudah ES, Spielmeyer W, Singh RP, Huerta-Espino J, McFadden H, Bossolini E, Selter LL, Keller B (2009) A putative ABC transporter confers durable resistance to multiple fungal pathogens in wheat. Science 323:1260–1263. https://doi.org/10.1126/science.1166453

Kuckuck H (1970) Primitive wheats. In: Frankel OH, Bennett E (eds) Genetic resources in plants—their exploration and conservation. Blackwell Scientific Publications, Oxford, pp 249–266

Kumar S, Stecher G, Tamura K (2016) MEGA7: molecular evolutionary genetics analysis version 7.0 for bigger datasets. Mol Biol Evol 33:1870–1874. https://doi.org/10.1093/molbev/msw054

Law CN, Wolfe MS (1966) Location of genetic factors for mildew resistance and ear emergence time on chromosome 7B of wheat. Can J Genet Cytol 8:462–479

Leath S, Bowen KL (1989) Effects of powdery mildew, triadimenol seed treatment, and triadimefon foliar sprays on yield of winter wheat in North Carolina. Phytopathology 79(2):152–155

Lee JH, Graybosch RA, Peterson CJ (1995) Quality and biochemical effects of a 1BL/1RS wheat-rye translocation in wheat. Theor Appl Genet 90:105–112. https://doi.org/10.1007/BF00221002

Lee WS, Hammond-Kosack KE, Kanyuka K (2012) *Barley stripe mosaic virus*-mediated tools for investigating gene function in cereal plants and their pathogens: virus-induced gene silencing, host-mediated gene silencing and virus-mediated over expression of heterologous proteins. Plant Physiol 160:582–590. https://doi.org/10.1104/pp.112.203489

Li G, Fang T, Zhang H, Xie C, Li H, Yang T, Nevo E, Fahima T, Sun Q, Liu Z (2009) Molecular identification of a new powdery mildew resistance gene Pm41 on chromosome 3BL derived from wild emmer (Triticum turgidum var. dicoccoides). Theor Appl Genet 119:531–539

Li T, Zhang ZY, Hu YK, Duan XY, Xin ZY (2011) Identification and molecular mapping of a resistance gene to powdery mildew from the synthetic wheat line M53. J Appl Genet 52:127–143. https://doi.org/10.1007/s12353-010-0006-0

Li ZF, Lan CX, He ZH, Singh RP, Rosewarne GM, Chen XM et al (2014) Overview and application of QTL for adult plant resistance to leaf rust and powdery mildew in wheat. Crop Sci 54:1907–1925. https://doi.org/10.2125/cropsci2014.02.0162

Li G, Xu X, Bai G, Carver BF, Hunger R, Bonman JM (2016) Identification of novel powdery mildew resistance sources in wheat. Crop Sci 56(4):1817. https://doi.org/10.2125/cropsci2015.09.0551

Li N, Jia HY, Kong ZX, Tang WB, Ding YX, Liang JC et al (2017) Identification and marker-assisted transfer of a new powdery mildew resistance gene at the *Pm4* locus in common wheat. Mol Breed 37:79. https://doi.org/10.1007/s11032-017-0670-4

Li F, Li Y, Cao L, Liu P, Geng M, Zhang Q et al (2018) Simultaneous transfer of leaf rust and powdery mildew resistance genes from hexaploid triticale cultivar Sorento into bread wheat. Front Plant Sci 9:85. https://doi.org/10.3389/fpls.2018.00085

Li G, Cowger C, Wang X, Carver BF, Xu X (2019) Characterization of Pm65, a new powdery mildew resistance gene on chromosome 2AL of a facultative wheat cultivar. Theor Appl Genet 132(9):2625–2632. https://doi.org/10.1007/s00122-019-03377-2

Li H, Dong Z, Ma C, Xia Q, Tian X, Sehgal S et al (2020) A spontaneous wheat- Aegilops longissima translocation carrying Pm66 confers resistance to powdery mildew. Theor Appl Genet. https://doi.org/10.1007/s00122-020-03538-8

Li M, Dong L, Li B, Wang Z, Xie J, Qiu D, Li Y, Shi W, Yang L et al (2020a) A CNL protein in wild emmer wheat confers powdery mildew resistance. New Phytol 228:1027–1037. https://doi.org/10.3389/fpls.2018.00085

Li YH, Shi XH, Hu JH, Wu PP, Qiu D, Qu YF et al (2020b) Identification of a recessive gene *PmQ* conferring resistance to powdery mildew in wheat landrace Qingxinmai using BSR-Seq analysis. Plant Dis 104:743–751. https://doi.org/10.1094/PDIS-08-19-1745-RE

Lillemo M, Skinnes H, Singh RP, van Ginkel M (2005) Genetic analysis of partial resistance to powdery mildew in bread wheat line Saar. Plant Dis 90:225–228

Lillemo M, Asalf B, Singh RP, Huerta-Espino J, Chen XM, He ZH, Bjørnstad A (2008) The adult plant rust resistance loci Lr34/Yr18 and Lr46/Yr29 are important determinants of partial resistance to powdery mildew in bread wheat line Saar. Theor Appl Genet 116:1155–1166

Lillemo M, Skinnes H, Brown JKM (2010) Race specific resistance to powdery mildew in Scandinavian wheat cultivars, breeding lines and introduced genotypes with partial resistance. Plant Breed 129:297–303. https://doi.org/10.1111/j.1439-0523.2009.01691.x

Liu S, Griffey CA, Maroof MA (2001) Identification of molecular markers associated with adult plant resistance to powdery mildew in common wheat cultivar Massey. Crop Sci 41:1268–1275. https://doi.org/10.2125/cropsci2001.4141268x

Liu ZY, Sun QX, Ni ZF, Nevo E, Yang TM (2002) Molecular characterization of a novel powdery mildew resistance gene *Pm30* in wheat originating from wild emmer. Euphytica 123:21–29. https://doi.org/10.1023/A:1014471112511

Liu J, Liu D, Wenjing T, Wei L, Wang S, Chen P, Cheng S, Gao D (2008) Molecular marker-facilitated pyramiding of different genes for powdery mildew resistance in wheat. Plant Breed 119:21–24. https://doi.org/10.1046/j.1439-0523.2000.00431.x

Liu NZ, Liu G, Gong M, Zhang X, Wang Y, Zhou et al (2015) Virulence structure of *Blumeria graminis f. sp. tritici* and its genetic diversity by ISSR and SRAP profiling analyses. PLoS One 10(6):e0120881. https://doi.org/10.1271/journal.pone.0120881

Liu W, Koo DH, Xia Q et al (2017) Homoeologous recombination-based transfer and molecular cytogenetic mapping of powdery mildew-resistant gene *Pm57* from *Aegilops searsii* into wheat. Theor Appl Genet 120:841–848. https://doi.org/10.1007/s00122-017-2855-y

Lo Presti L, Lanver D, Schweizer G, Tanaka S, Liang L, Tollot M et al (2015) Fungal effectors and plant susceptibility. Annu Rev Plant Biol 66:512–545. https://doi.org/10.1146/annurev-arplant-043014-114623

Lu P, Guo L, Wang Z, Li B, Li J, Li Y, Qiu D, Shi W, Yang L, Wang N et al (2020) A rare gain of function mutation in a wheat tandem kinase confers resistance to powdery mildew. Nat Commun 11(1):680. https://doi.org/10.1038/s41467-020-14294-0

Lukaszewski AJ (2000) Manipulation of the 1RS.1BL translocation in wheat by induced homoeologous recombination. Crop Sci 40:216–225. https://doi.org/10.2125/cropsci2000.401216x

Luo PG, Luo HY, Chang ZJ, Zhang HY, Zhang M, Ren ZL (2009) Characterization and chromosomal location of *Pm40* in common wheat: a new gene for resistance to powdery mildew derives from *Elytrigia intermedium*. Theor Appl Genet 18:1059–1064. https://doi.org/10.1007/s00122-009-0962-0

Lutz J, Limpert E, Bartoš P, Zeller FJ (1992) Identification of powdery mildew resistance genes in common wheat (*Triticum aestivum* L.). Plant Breed 108(1):33–39. https://doi.org/10.1111/j.1439-0523.1992.tb00097.x

Lutz J, Hsam S, Limperti E, Zeller F (1995) Chromosomal location of powdery mildew resistance genes in Triticum aestivum. Heredity 74:152–156

Ma XF, Gustafson JP (2008) Allopolyploidization-accommodated sequence changes in triticale. Ann Bot 101:825–832. https://doi.org/10.1093/aob/mcm331

Ma ZQ, Sorrels ME, Tanksley SD (1994) RFLP markers linked to powdery mildew resistance genes *Pm1, Pm2, Pm3* and *Pm4* in wheat. Genome 37:871–875. https://doi.org/10.1129/g94-123

Ma H, Kong Z, Fu B, Li N, Zhang L, Jia H, Ma Z (2011) Identification and mapping of a new powdery mildew resistance gene on chromosome 6D of common wheat. Theor Appl Genet 123:1099. https://doi.org/10.1007/s00122-011-1651-3

Ma P, Xu H, Li L, Zhang H, Han G, Xu Y, Fu X, Zhang X (2016) Characterization of a new *Pm2* allele conferring powdery mildew resistance in the wheat germplasm line FG-1. Front Plant Sci 7:546. https://doi.org/10.3389/fpls.2016.00546

Ma PT, Xu HX, Xu YF, Song LP, Liang SS, Sheng Y et al (2018) Characterization of a powdery mildew resistance gene in wheat breeding line 10V-2 and its application in marker-assisted selection. Plant Dis 102:925–931. https://doi.org/10.1094/PDIS-02-17-0199-RE

Mago R, Spielmeyer W, Lawrence J, Lagudah S, Ellis G, Pryor A (2002) Identification and mapping of molecular markers linked to rust resistance genes located on chromosome 1RS of rye using wheat-rye translocation lines. Theor Appl Genet 104:1217–1224. https://doi.org/10.1007/s00122-002-0879-3

Mains EB (1934) Inheritance of resistance to powdery mildew, *Erysiphe graminis tritici* in wheat. Phytopathology 24:1257–1261

Marone D, Russo MA, Laido G, Vita PD, Papa R, Blanco A, Gadaleta A, Rubiales D, Mastrangelo AM (2013) Genetic basis of qualitative and quantitative resistance to powdery mildew in wheat from consensus regions to candidate genes. BMC Genomics 14:562

Martin AR, Kanai M, Kamatani Y, Okada Y, Neale BM et al (2019) Clinical use of current polygenic risk score may exacerbate health disparities. Nat Genet 51:584–591. https://doi.org/10.1038/s41588-019-0379-x

Mascher M, Muehlbauer GJ, Rokhsar DS, Chapman J, Schmutz J, Barry K et al (2012) Anchoring and ordering NGS contig assemblies by population sequencing (POPSEQ). Plant J 76:718–727. https://doi.org/10.1111/tpj.12319

Mayer KFX, Martis M, Hedley PE, Šimková H, Liu H, Morris JA et al (2011) Unlocking the barley genome by chromosomal and comparative genomics. Plant Cell 23:1249–1263. https://doi.org/10.1105/tpc.110.082537

McCallum CM, Comai L, Greene EA, Henikoff S (2000) Targeted screening for induced mutations. Nat Biotechnol 18:455–457. https://doi.org/10.1038/74542

McDonald BA, Linde C (2002) The population genetics of plant pathogens and breeding strategies for durable resistance. Euphytica 124:163–180. https://doi.org/10.1023/A:1015678432355

McIntosh RA (1998) Breeding wheat for resistance to biotic stresses. Euphytica 100:19–34

McIntosh RA, Baker EP (1970) Cytogenetic studies in wheat IV chromosomal location and linkage studies involving the *Pm2* locus for powdery mildew resistance. Euphytica 19:71–77. https://doi.org/10.1007/BF01904668

McIntosh RA, Hart GE, Gale MD, Rogers WJ (1998) Catalogue of gene symbols for wheat. In: Slinkard AE (ed) Proceedings of the 9th international wheat genet symposium, vol 5. University Extension Press, University of Saskatchewan, Saskatoon, Canada, p 235

McIntosh RA, Devos KM, Dubcovsky J, Rogers WJ, Morris CF, Appels R, Somers DJ, Anderson OA (2007) Catalogue of gene symbols for wheat: 2007 supplement. http://wheat.pw.usda.gov/ggpages/awn/53/Textfile/WGC.html

McIntosh RA, Dubcovsky J, Rogers WJ, Morris C, Appels R, Xia XC (2014) Catalogue of gene symbols for wheat: 2012-2014 supplement. Online publication

McIntosh RA, Dubcovsky J, Rogers WJ, Morris CF, Xia XC (2017) Catalogue of gene symbols for wheat. Suppl Annu Wheat Newsl 53:1–20

McIntosh RA, Dubcovsky J, Rogers WJ, Xia XC, Raupp WJ (2019) Catalogue of gene symbols for wheat: 2019 supplement. In: Raupp WJ (ed) Annual Wheat Newsletter. The Wheat Genetic and Genomic Resources at Kansas State University, Manhattan, NY, pp 98–113

Mclntosh RA (1973) A catalogue of gene symbols for wheat. In: Proceedings of the 4th international wheat genetics symposium, pp 893–937

Merchan VM, Kranz J (1986) Die Wirkung des Regens auf die Entwicklung des Weizenmehltaus (*Erysiphe graminis DC. f. sp. tritici Marchal*). J Plant Dis Prot 93:262–270

Meyers BC, Kozik A, Griego A, Kuang H, Michelmore RW (2003) Genome-wide analysis of NBS-LRR-encoding genes in *Arabidopsis*. Plant Cell 15:809–834. https://doi.org/10.1105/tpc.009308

Miedaner T, Flath K (2007) Effectiveness and environmental stability of quantitative powdery mildew (*Blumeria graminis*) resistance among winter wheat cultivars. Plant Breed 126:553–558. https://doi.org/10.1111/j.1439-0523.2006.01253.x

Miranda LM, Murphy J, Marshall PD, Leath S (2006) *Pm34*: a new powdery mildew resistance gene transferred from *Aegilops tauschii* Coss. To common wheat (*Triticum aestivum* L.). Theor Appl Genet 112:1497–1504. https://doi.org/10.1007/s00122-006-0397-9

Miranda LM, Murphy JP, Marshall D, Cowger C, Leath S (2007) Chromosomal location of Pm35, a novel Aegilops tauschii derived powdery mildew resistance gene introgressed into common wheat (Triticum aestivum L.). Theor Appl Genet 114:1451–1456

Mo Y, Howell T, Vasquez-Gross H et al (2018) Mapping causal mutations by exome sequencing in a wheat TILLING population: a tall mutant case study. Mol Gen Genomics 293:463–477. https://doi.org/10.1007/s00438-017-1401-6

Mohler V, Jahoor A (1996) Allele-specific amplification of polymorphic sites for the detection of powdery mildew resistance loci in cereals. Theor Appl Genet 93:1078–1082. https://doi.org/10.1007/BF00230128

Mohler V, Bauer C, Schweizer G, Kempf H, Hartl L (2012) *Pm50*: a new powdery mildew resistance gene in common wheat derived from cultivated emmer. J Appl Genet 54:259–263. https://doi.org/10.1007/s12353-012-0158-9

Moore JW, Herrera-Foessel S, Lan C, Schnippenkoetter W, Ayliffe M, Huerta-Espino J, Lillemo M, Viccars L, Milne R, Periyannan S et al (2015) Recently evolved hexose transporter variant confers resistance to multiple pathogens in wheat. Nat Genet 47:1494–1498. https://doi.org/10.1038/ng.3439

Murphy JP, Navarro RA, Leath S (2002) Registration of NC99BGTAG11 wheat germplasm resistant to powdery mildew. Crop Sci 42:1282. https://doi.org/10.2125/cropsci2002.1282

Mwale MM, Tang XL, Chilembwe E (2017) Molecular detection of disease resistance genes to powdery mildew (*Blumeria graminis f. sp. tritici*) in wheat (*Triticum aestivum*) cultivars. Afr J Biotechnol 16:22–31. https://doi.org/10.5897/AJB2016.15720

Nsabiyera V, Baranwal D, Qureshi N, Kay P, Forrest K, Valárik M, Doležel J, Hayden MJ, Bariana HS, Bansal UK (2020) Fine mapping of *Lr49* using 90K SNP chip array and flow-sorted chromosome sequencing in wheat. Front Plant Sci 10:1787. https://doi.org/10.3389/fpls.2019.01787

Oberhaensli S, Parlange F, Buchmann JP, Jenny FH, Abbott JC, Burgis TA et al (2011) Comparative sequence analysis of wheat and barley powdery mildew fungi reveals gene colinearity, dates divergence and indicates host–pathogen co-evolution. Fungal Genet Biol 48:327–334. https://doi.org/10.1186/s12862-017-1064-2

Oerke EC, Dehne HW, Schönbeck F, Weber A (1994) Crop production and crop protection: estimated losses in major food and cash crops. p 808. https://doi.org/10.1017/S0021859600077467

Oettler G, Tams SH, Utz HF, Bauer E, Melchinger AE (2005) Prospects for hybrid breeding in winter triticale: I. Heterosis and combining ability for agronomic traits in European elite germplasm. Crop Sci 45:1476–1482. https://doi.org/10.2125/cropsci2004.0462

Patil DK, Hegde RK, Govinda HG (1969) New record of powdery mildew of wheat in Mysore state. Mysore J Agric Sci 3:238–239

Paulech C (1995) Huby múènatkotvaré (Erysiphales). Flóra Slovenska X/I, Veda, Bratislava, p 294

Persaud RR, Lipps PE, Campbell KG (1994) Identification of powdery mildew resistance genes in soft red winter wheat cultivars and Ohio breeding lines. Plant Dis 78:1072–1075. https://doi.org/10.1094/PD-78-1072

Perugini LD, Murphy JP, Marshall D, Brown-Guedira G (2008) *Pm37*, a new broadly effective powdery mildew resistance gene from *Triticum timopheevii*. Theor Appl Genet 116:417–425. https://doi.org/10.1007/s00122-007-0679-x

Peusha H, Enno T, Priilinn O (2000) Chromosomal location of powdery mildew resistance genes and cytogenetic analysis of meiosis in common wheat cultivar Meri. Hereditas 132:29–34

Piarulli L, Agata G, Giacomo M, Signorile MA, Pasquini M, Blanco A, Simeone R (2012) Molecular identification of a new powdery mildew resistance gene on chromosome 2BS from *Triticum turgidum ssp. Dicoccum*. Plant Sci 196:101–106. https://doi.org/10.17557/tjfc.83589

Pietrusińska A, Czembor JH, Czembor PC (2011) Pyramiding two genes for leaf rust and powdery mildew resistance in common wheat. Cereal Res Commun 39(4):577–588

Pietrusińska A, Żurek M, Mańkowski D (2019) The search for sources of biotic stress resistance in old varieties and landraces of wheat and triticale. Biuletyn IHAR 287:25–28

Poland JA, Nelson RJ (2011) In the eye of the beholder: the effect of rater variability and different rating scales on QTL mapping. Phytopathology 101:290–298. https://doi.org/10.1094/PHYTO-03-10-0087

Poland JA, Balint-Kurti PJ, Wisser RJ, Pratt RC, Nelson RJ (2009) Shades of gray: the world of quantitative disease resistance. Trends Plant Sci 14:21–29. https://doi.org/10.1016/j.tplants.2008.10.006

Purnhauser L, Bóna L, Láng L (2011) Occurrence of 1BL.1RS wheat-rye chromosome translocation and of *Sr36*/*Pm6* resistance gene cluster in wheat cultivars registered in Hungary. Euphytica 179:287–295. https://doi.org/10.1007/s10681-010-0312-y

Qi LL, Cao MS, Chen PD, Li WL, Liu DJ (1996) Identification, mapping, and application of polymorphic DNA associated with resistance gene *Pm21* of wheat. Genome 39:191–197. https://doi.org/10.1129/g96-025

Qi L, Friebe B, Zhang P, Gill BS (2007) Homoeologous recombination, chromosome engineering and crop improvement. Chromosom Res 15:3–19. https://doi.org/10.1007/s10577-006-1108-8

Qiu YC, Zhang SS (2004) Researches on powdery mildew resistant genes and their molecular markers in wheat. J Triticeae Crops 24:127–122

Que Q, Chilton MD, de Fontes CM, He C, Nuccio M, Zhu T, Wu Y, Chen JS, Shi L (2010) Trait stacking in transgenic crops: challenges and opportunities. GM Crops 1(4):220–229. https://doi.org/10.4161/gmcr.1.4.12439

Qureshi N, Kandiah P, Gessese MK, Nsabiyera V, Wells V, Babu P et al (2018) Development of co-dominant KASP markers co-segregating with Ug99 effective stem rust resistance gene *Sr26* in wheat. Mol Breed 38(8):97. https://doi.org/10.1007/s11032-018-0854-6

Rakszegi M, Kisgyörgy BN, Tearall K, Shewry PR, Láng L, Phillips A et al (2010) Diversity of agronomic and morphological traits in a mutant population of bread wheat studied in the Healthgrain program. Euphytica 174:409–421. https://doi.org/10.1007/s10681-010-0149-4

Rasheed A, Hao Y, Xia XC, Khan A, Xu Y, Varshney RK et al (2017) Crop breeding chips and genotyping platforms: progress, challenges and perspectives. Mol Plant 10:1047–1064. https://doi.org/10.1016/j.molp.2017.06.008

Reader SM, Miller TE (1991) The introduction into bread wheat of a major gene for resistance to powdery mildew from wild emmer wheat. Euphytica 53:57–60

Ren SX, McIntosh RA, Lu ZJ (1997) Genetic suppression of the cereal rye derived gene *Pm8* in wheat. Euphytica 93:353–360. https://doi.org/10.1023/A:1002923030266

Roelfs AP (1977) Foliar fungal diseases of wheat in the People's Republic of China. Plant Dis Rep 61:836–841

Rong JK, Millet E, Manisterski J, Feldman M (2000) A new powdery mildew resistance gene: introgression from wild emmer into common wheat and RFLP-based mapping. Euphytica 115:121–126. https://doi.org/10.1023/A:1003950431049

Saari EE, Prescott JM (1975) A scale for appraising the foliar intensity of wheat diseases. Plant Dis Rep 59:377–380

Saari EE, Wilcoxson RD (1974) Plant disease situation of high-yielding dwarf wheats in Asia and Africa. Annu Rev Phytopathol 12:49–68. https://doi.org/10.1146/annurev.py.12.090174.000405

Saintenac C, Jiang D, Akhunov ED (2011) Targeted analysis of nucleotide and copy number variation by exon capture in allotetraploid wheat genome. Genome Biol 12:R88. https://doi.org/10.1186/gb-2011-12-9-r88

Samobor V, Vukobratovic M, Jost M (2005) Effect of powdery mildew (*Erysiphe graminis* DC f. sp. *tritici* Marchal) attack on yield and physical parameters of wheat (*Triticum aestivum* spp vulgare) grain quality. Agriculture 11:30–37

Sanchez-Martin J, Keller B (2019) Contribution of recent technological advances to future resistance breeding. Theor Appl Genet 122:712–732. https://doi.org/10.1007/s00122-019-03297-1

Sánchez-Martín J, Steuernagel B, Ghosh S, Herren G, Hurni S, Adamski N (2016) Rapid gene isolation in barley and wheat by mutant chromosome sequencing. Genome Biol 17:221. https://doi.org/10.1186/s12059-016-1082-1

Sarris PF, Duxbury Z, Huh SU, Ma Y, Segonzac C, Sklenar J, Derbyshire P, Cevik V, Rallapalli G, Saucet SB (2015) A plant immune receptor detects pathogen effectors that target wrky transcription factors. Cell 161:1089–1100. https://doi.org/10.1016/j.cell.2015.04.024

Schmolke M, Mohler V, Hartl L, Zeller FJ, Hsam SLK (2011) A new powdery mildew resistance allele at the Pm4 wheat locus transferred from einkorn (Triticum monococcum). Mol Breed 29(2):449–456. https://doi.org/10.1007/s11032-011-9561-2

Shah L, Ali A, Zhu Y, Wang S, Si H, Ma C (2017) Wheat resistance to *Fusarium* head blight and possibilities of its improvement using molecular marker-assisted selection. Czech J Genet Plant Breed 53:47–54. https://doi.org/10.17221/129/2016-CJGPB

Shamanin V, Shepelev S, Pozherukova V, Gultyaeva E, Kolomiets T, Pakholkova E et al (2019) Primary hexaploid synthetics: novel sources of wheat disease resistance. Crop Prot 121:7–10. https://doi.org/10.1016/j.cropro.2019.03.003

Shaner G (1973) Evaluation of slow-mildewing resistance of Knox wheat in the field. Phytopathology 63:867–872

Shewry PR (2009) Wheat J Exp Bot 60:1537–1553. https://doi.org/10.1093/jxb/erp058

Shi AN (1997) Genetic analysis of wheat powdery mildew resistance. Ph.D. Thesis North Carolina State University

Shi AN, Leath S, Murphy JP (1998) A major gene for powdery mildew resistance transferred to common wheat from wild einkorn wheat. Phytopathology 88(2):144–147. https://doi.org/10.1094/PHYTO.1998.88.2.144

Simeone R, Piarulli L, Nigro D, Antonio Signorile M, Blanco E, Mangini G, Blanco A (2020) Mapping powdery mildew (*Blumeria graminis* f. sp. *tritici*) resistance in wild and cultivated tetraploid wheats. Int J Mol Sci 21:7910. https://doi.org/10.3390/ijms21217910

Singh DP, Sharma AK, Singh D, Rana SK, Singh KP, Srivastava K et al (2009) Resistance to powdery mildew in Indian wheat. Plant Dis Res 24:942

Singh SP, Hurni S, Ruinelli M, Brunner S, Sanchez-Martin J, Krukowski P, Peditto D, Buchmann G, Zbinden H, Keller B (2018) Evolutionary divergence of the rye *Pm17* and *Pm8* resistance genes reveals ancient diversity. Plant Mol Biol 98:249–260. https://doi.org/10.1007/s11103-018-0780-3

Srichumpa P, Brunner S, Keller B, Yahiaoui N (2005) Allelic series of four powdery mildew resistance genes at the *Pm3* locus in hexaploid bread wheat. Plant Physiol 129:885–895. https://doi.org/10.1104/pp.105.062406

Summers R, Brown J (2012) Constraints on breeding for disease resistance in commercially competitive wheat cultivars. Plant Pathol 62:115–121. https://doi.org/10.1111/ppa.12165

Sun H, Hu J, Song W et al (2018) Pm61: a recessive gene for resistance to powdery mildew in wheat landrace Xuxusanyuehuang identified by comparative genomics analysis. Theor Appl Genet 131:2085–2097. https://doi.org/10.1007/s00122-018-3135-1

Swaminathan MS, Raychaudhary SP, Joshi LM, Gera SD (1971) Incidence of black point and karnal bunt in north-western plains zone during 1969-1970. Indian Agric Res Inst New Delhi Wheat Dis Newslett 4:1–4

Talas F, Longin F, Miedaner T (2011) Sources of resistance to fusarium head blight within Syrian durum wheat landraces. Plant Breed 120:398–400. https://doi.org/10.1111/j.1439-0523.2011.01867.x

Tan C, Li G, Cowger C, Carver BF, Xu X (2018) Characterization of Pm59, a novel powdery mildew resistance gene in Afghanistan wheat landrace PI 181356. Theor Appl Genet 131(5):1145–1152. https://doi.org/10.1007/s00122-018-3067-9

Tan C, Li G, Cowger C, Carver BF, Xu X (2019) Characterization of Pm63, a powdery mildew resistance gene in Iranian landrace PI 628024. Theor Appl Genet 132(4):1137–1144. https://doi.org/10.1007/s00122-018-3265-5

Tang S, Hu Y, Zhong S, Luo P (2018) The potential role of powdery mildew-resistance gene *Pm40* in chinese wheat-breeding programs in the post-*Pm21* era. Engineering 4(4):500–506. https://doi.org/10.1016/j.eng.2018.06.004

Tanksley SD (1993) Mapping polygenes. Annu Rev Genet 27:205–233. https://doi.org/10.1146/annurev.ge.27.120193

Tao W, Liu D, Liu J, Feng Y, Chen P (2000) Genetic mapping of the powdery mildew resistance gene Pm6 in wheat by RFLP analysis. Theor Appl Genet 100:564–568. https://doi.org/10.1007/s001220050074

Tester M, Langridge P (2010) Breeding technologies to increase crop production in a changing world. Science 327(5967):818–822. https://doi.org/10.1126/science.1183700

The TT, McIntosh RA, Bennett FGA (1979) Cytogenetical studies in wheat. IX. Monosomic analysis, telocentric mapping and linkage relationship of gene Sr21, Pm4 and Mle. Aust J Biol Sci 32:115–125

Thind AK, Wicker T, Simkova H, Fossati D, Moullet O, Brabant C et al (2017) Rapid cloning of genes in hexaploid wheat using cultivar-specific long-range chromosome assembly. Nat Biotechnol 35:793–796. https://doi.org/10.1038/nbt.3877

Tosa Y, Sakai K (1990) The genetics of resistance of hexaploid wheat to the wheatgrass powdery mildew fungus. Genome 33:225–230

Tosa Y, Tsujimoto H, Ogura H (1987) A gene involved in the resistance of wheat to wheatgrass powdery mildew fungus. Genome 29:850–852

Tosa Y, Tokunaga H, Ogura H (1988) Identification of a gene for resistance to wheatgrass powdery mildew fungus in the common wheat cultivar Chinese spring. Genome 30:612–614

Tucker DM, Griffey CA, Liu S, Saghai Maroof MA (2006) Potential for effective marker-assisted selection of three quantitative trait loci conferring adult plant resistance to powdery mildew in elite wheat breeding populations. Plant Breed 125(5):430–436. https://doi.org/10.1111/j.1439-0523.2006.01233.x

Uauy C, Wulff BBH, Dubcovsky J (2017) Combining traditional mutagenesis with new High-Throughput sequencing and genome editing to reveal hidden variation in polyploid wheat. Annu Rev Genet 51:435–454. https://doi.org/10.1146/annurev-genet-120116-024533

Ueda A, Kathiresan A, Bennett J, Takabe T (2006) Comparative transcriptome analyses of barley and rice under salt stress. Theor Appl Genet 112:1286–1294. https://doi.org/10.1007/s00122-006-0231-4

Varallyay E, Giczey G, Burgyán J (2012) Virus induced gene silencing of *Mlo* genes induces powdery mildew resistance in *Triticum aestivum*. Arch Virol 157:1245–1250. https://doi.org/10.1007/s00705-012-1286-y

Vikas VK, Kumar S, Archak S et al (2020) Screening of 19,460 genotypes of wheat species for resistance to powdery mildew and identification of potential candidates using focused identification of germplasm strategy (FIGS). Crop Sci 60(6):2857–2866. https://doi.org/10.1002/csc2.20196

Voytas DF (2012) Plant genome engineering with sequence-specific nucleases. Annu Rev Plant Biol 64:327–350. https://doi.org/10.1146/annurev-arplant-042811-105552

Wallwork H (2009) The use of host plant resistance in disease control. In: Walters D (ed) Disease control in crops: biological and environmentally friendly approaches, vol 6. Wiley, West Sussex, pp 122–141

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 cultivars and lines. Plant Dis 89:457–463. https://doi.org/10.1094/PD-89-0457

Wang S, Wong D, Forrest K, Allen A, Chao S et al (2014) Characterization of polyploid wheat genomic diversity using a high density 90,000 single nucleotide polymorphism array. Plant Biotechnol J 12:787–796. https://doi.org/10.1111/pbi.12183

Ward SV, Manners JG (1974) Environmental effects on the quantity and viability of conidia produced by *Erysiphe graminis*. Trans Br Mycol Soc 62:119–128. https://doi.org/10.1016/S0007-1536(74)80012-6

Waszczak C, Akter S, Jacques S, Huang J, Messens J, Van Breusegem F (2015) Oxidative post-translational modifications of cysteine residues in plant signal transduction. J Exp Bot 66:2923–2934. https://doi.org/10.1093/jxb/erv084

Wendler N, Mascher M, Nöh C et al (2014) Unlocking the secondary gene-pool of barley with next-generation sequencing. Plant Biotechnol J 12:1122–1121. https://doi.org/10.1111/pbi.12219

Wiersma AT, Pulman JA, Brown LK, Cowger C, Olson EL (2017) Identification of Pm58 from Aegilops tauschii. Theor Appl Genet 130(6):1123–1133. https://doi.org/10.1007/s00122-017-2874-8

Wu P, Xie J, Hu J, Qiu D, Liu Z, Li J, Li M, Zhang H, Yang L, Liu H, Zhou Y, Zhang Z, Li H (2018) Development of molecular markers linked to powdery mildew resistance gene *Pm4b* by combining snp discovery from transcriptome sequencing data with bulked segregant analysis (BSR-Seq) in wheat. Front Plant Sci 9:95. https://doi.org/10.3389/fpls.2018.00095

Xiao M, Song F, Jiao J, Wang X, Xu H, Li H (2013) Identification of the gene Pm47 on chromosome 7BS conferring resistance to powdery mildew in the Chinese wheat landrace Hongyanglazi. Theor Appl Genet 126:1397–1403

Xie CJ, Sun QX, Ni ZT, Nevo E, Fahima T (2003) Chromosomal location of a Triticum dicoccoides -derived powdery mildew resistance gene in common wheat by using microsatellite markers. Theor Appl Genet 106:341

Xie J, Guo G, Wang Y, Hu T, Wang L (2020) A rare single nucleotide variant in *Pm5e* confers powdery mildew resistance in common wheat. New Phytol 228(3):1011–1026. https://doi.org/10.1111/nph.16762

Xin M, Wang X, Peng H, Yao Y, Xie C, Han Y et al (2012) Transcriptome comparison of susceptible and resistant wheat in response to powdery mildew infection. Gen Prot Bioinf 10:94–106. https://doi.org/10.1016/j.gpb.2012.05.002

Xing LP, Hu P, Liu JQ, Witek K, Zhou S, Xu JF, Zhou WH et al (2018) *Pm21* from *Haynaldia villosa* encodes a CC-NBS-LRR that confers powdery mildew resistance in wheat. Mol Plant 11:874–878. https://doi.org/10.1016/j.molp.2018.02.012

Xu HX, Yi YJ, Ma PT, Qie YM, Fu XY, Xu YF et al (2015) Molecular tagging of a new broad-spectrum powdery mildew resistance allele *Pm2c* in Chinese wheat landrace Niaomai. Theor Appl Genet 128:2077–2084. https://doi.org/10.1007/s00122-015-2568-z

Yahiaoui N, Srichumpa P, Dudler R, Keller B (2004) Genome analysis at different ploidy levels allows cloning of the powdery mildew resistance gene *Pm3b* from hexaploid wheat. Plant J 37:528–538. https://doi.org/10.1046/j.1265-312x.2003.01977.x

Yahiaoui N, Brunner S, Keller B (2006) Rapid generation of new powdery mildew resistance genes after wheat domestication. Plant J 47:85–98. https://doi.org/10.1111/j.1265-312X.2006.02772.x

Yao G, Zhang J, Yang L, Xu H, Jiang Y, Xiong L, Zhang C, Zhang Z, Ma Z, Sorrells M (2007) Genetic mapping of two powdery mildew resistance genes in einkorn (*Triticum monococcum* L.) accessions. Theor Appl Genet 114:351–358. https://doi.org/10.1007/s00122-006-0438-4

Yao F, Zhang X, Ye X, Li J, Long L, Yu C (2019) Characterization of molecular diversity and genome wide association study of stripe rust resistance at the adult plant stage in Northern Chinese wheat landraces. BMC Genet 20:38. https://doi.org/10.1186/s12863-019-0736-x

Zadoks JC, Chang TT, Konzak CF (1974) A domical code for the growth stages of cereals. Weed Res 14:415–421. https://doi.org/10.1111/j.1265-180.1974.tb01084.x

Zeller FJ, Hsam SLK (1998) Progress in breeding for resistance to powdery mildew in common wheat (*Triticum aestivum* L.). In: Slinkard AE (ed) Proceedings of the ninth international wheat genetics symposium, vol 1. University Extension Press, University of Saskatchewan, Saskatoon, pp 178–180

Zeller FJ, Lutz J, Stephan U (1993) Chromosome location of genes for resistance to powdery mildew in common wheat (Triticum aestivum L.) Mlk and other alleles at the Pm3 locus. Euphytica 68:223–229

Zeller FJ, Kong L, Hartl L, Mohler V, Hsam SLK (2002) Chromosomal location of genes for resistance to powdery mildew in common wheat (*Triticum aestivum* L. em Thell.) 7. Gene *Pm29* in line Pova. Euphytica 123:187–194. https://doi.org/10.1023/A:1014944619304

Zhan HX, Li GR, Zhang XJ, Li X, Guo HJ, Gong WP, Jia JQ, Qiao LY, Ren YK, Yang ZJ, Chang ZJ (2014) Chromosomal location and comparative genomics analysis of powdery mildew resistance gene *Pm51* in a putative wheat-*Thinopyrum ponticum* introgression line. PLoS One 9:e112455. https://doi.org/10.1271/journal.pone.0112455

Zhang ZY, Chen X, Zhang C, Xin ZY, Chen XM (2002) Selecting the pyramids of powdery mildew resistance genes *Pm4b*, *Pm12* and *Pm21* in wheat assisted by molecular marker (In Chinese). Sci Agric Sin 35:789–793

Zhang HT, Guan HY, Li JT, Zhu J, Xie CJ, Zhou YL et al (2010) Genetic and comparative genomics mapping reveals that a powdery mildew resistance gene Ml3D232 originating from wild emmer co-segregates with an NBS-LRR analog in common wheat (*Triticum aestivum* L.). Theor Appl Genet 121:1612–1621. https://doi.org/10.1007/s00122-010-1414-6

Zhang R, Sun B, Chen J, Cao A, Xing L, Feng Y, Lan C, Chen P (2016) Pm55, a developmental-stage and tissue-specific powdery mildew resistance gene introgressed from Dasypyrum villosum into common wheat. Theor Appl Genet 129:1975–1984

Zhang Y, Bai Y, Wu G, Zou S, Chen Y, Gao C, Tang D (2017) Simultaneous modification of three homoeologs of TaEDR1 by genome editing enhances powdery mildew resistance in wheat. Plant J 91(4):714–724. https://doi.org/10.1111/tpj.12599

Zhang R, Fan Y, Kong L, Wang Z, Wu J, Xing L et al (2018) Pm62, an adult-plant powdery mildew resistance gene introgressed from Dasypyrum villosum chromosome arm 2VL into wheat. Theor Appl Genet. https://doi.org/10.1007/s00122-018-3176-5

Zhang D, Zhu K, Dong L, Liang Y, Li G, Fang T et al (2019) Wheat powdery mildew resistance gene Pm64 derived from wild emmer (Triticum turgidum var. dicoccoides) is tightly linked in repulsion with stripe rust resistance gene Yr5. The Crop Journal. https://doi.org/10.1016/j.cj.2019.03.003

Zhao Z, Sun H, Song W, Lu M, Huang J, Wu LF et al (2012) Genetic analysis and detection of the gene *MlLX99* on chromosome 2BL conferring resistance to powdery mildew in the wheat cultivar Liangxing 99. Theor Appl Genet 126:3081–3089. https://doi.org/10.1007/s00122-012-2194-6

Zheng W, Li S, Zi L, Qi Z, Feng Y, Chai S (2020) Molecular marker assisted gene stacking for disease resistance and quality genes in the dwarf mutant of an elite common wheat cultivar Xiaoyan22. BMC Genet 21:45. https://doi.org/10.1186/s12863-020-00854-2

Zhou RH, Zhu ZD, Kong XY, Huo NX, Tian QZ, Li P et al (2005) Development of wheat near-isogenic lines for powdery mildew resistance. Theor Appl Genet 110:640–648. https://doi.org/10.1007/s00122-004-1889-0

Zhu Z, Zhou R, Kong X, Dong Y, Jia J (2005) Microsatellite markers linked to 2 powdery mildew resistance genes introgressed from *Triticum carthlicum* accession PS5 into common wheat. Genome 48:585–590. https://doi.org/10.1129/g05-016

Zou SH, Wang H, Li YW, Kong ZS, Tang DZ (2018) The NB-LRR gene Pm60 confers powdery mildew resistance in wheat. New Phytol 218(1):298–309. https://doi.org/10.111/nph.14964