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RESEARCHSTATUS AND PROSPECT OF GENES RELATED WITH RESISTANCE TO POWDERY MILDEW OF WHEAT

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Wheat (Triticum aestivum L.) is one of the major grain crops in the world. Wheat powdery mildew is a fungal disease caused by the infection of Blumeria graminis F. sp. tritici. It is one of the most severe wheat diseases globally, seriously affecting the yield and quality of wheat. At present, the main ways to control powdery mildew are the use of fungicides and the cultivation of disease-resistant varieties. The extensive spraying of fungicides causes pesticide residues and environmental pollution. At present, no matter wild type or artificially bred wheat powdery mildew resistant varieties are scarce, so it is urgent to cultivate resistant varieties quickly and efficiently. Traditional cross breeding has a long time and low efficiency. Still, it is a fast and effective way to get disease-resistant sorts by using modern molecular biological means to transfer disease-resistant genes into cultivated varieties. Although the cultivation of resistant varieties is the most economical and effective way to control powdery mildew in wheat, there are some limitations in the cultivation of resistant varieties by introducing resistance genes by conventional means in actual production. With the increase of disease each year, this situation will be more and more unable to meet the needs of wheat genetic improvement. It is urgent to explore a new way of breeding to improve the wheat to powdery mildew lasting broad-spectrum resistance. The disease-resistant breeding needs from cloning in plant and pathogen affinity interactions play a vital role in the study of disease genes and their mechanism of action. At present, in the wheat by manipulating disease genes make infected material gain lasting broad-spectrum resistance is less. In the case of disease genes and mutations after its disease-resistant mechanism are still not clear. So the breeding of resistant varieties need mining and utilization of resistance genes. The paper summarizes the harm and distribution of wheat powdery mildew, the genes resistance mechanism of wheat powdery mildew, and functional analysis, wheat powdery mildew resistance genes in the field of molecular biology research status, and VIGS, RNAi, such as for the prevention and control of wheat powdery mildew, explore new powdery mildew resistance genes and resistance regulation, breeding disease-resistant varieties of wheat provide the feasible scheme.

Key word: *wheat, powdery mildew of wheat, resistance genes of wheat powdery mildew, VIGS, RNAi.*
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Introduction. Wheat (*Triticum aestivum* L.), 2n = 6x = 42, AABBDD is *Gramineae*, *Triticeae*, Triticum, and is one of the earliest cultivated plants the world. It originated from the Middle East near the Mediterranean Sea and was introduced into China later. It is reported that wheat cultivation in China has a history of at least four or five thousand years (Cao, 2008). At present, wheat is also the second largest crop after rice, which is cultivated all over the world. Wheat production and output rank the first in the world, with 43 countries and more than one third of the population taking it as the staple food (Huang & Roder, 2004). Wheat has high nutritional value and can provide about one-fifth of the calories and protein of human needs (He Zhonghu et al., 2018). As the world population continues to increase, wheat will become more and more critical. In addition, wheat is the most important food for trade and international aid. According to UN COMTRADE, the world's wheat export in 2016 amounted to 148 million tons. China is the world's largest wheat production and consumption of the country, annual production accounts for about one-fifth of the global total, the world's largest output (Sun Zhilu, 2019). China's agricultural production level is constantly improving, but pests and diseases are still an essential factor to limit agricultural production. Powdery mildew is one of the wheat diseases with the most extensive range and a great influence on

yield. Wheat powdery mildew is by living nutrition obligate parasitic fungi of wheat powdery mildew caused a worldwide disease, can often result in 13 %–34 % of yield loss, on the pathogenesis of heading and filling stage, a severe loss will generate 50 % of output, in extreme infected cases can lead to dry leaves, and even plant death (Cao et al., 2011). In the past 40 years, wheat powdery mildew has spread rapidly from local areas in the southwest and southeast coastal regions to almost all wheat areas in China due to the improvement of wheat production conditions and the variation of pathogen virulence structure, causing considerable losses to China's grain production (Liu Wancai et al., 2016). The most economical and effective method to control wheat powdery mildew is to cultivate resistant varieties. The discovery of resistance genes and resistance control genes is significant for the breeding of new wheat resistant varieties.

The aim of this article is to conduct an in-depth analysis of scientific information to do about the resistance of bread wheat to powdery mildew, taking into account the use in China Gene PM46, and to determine the possibility of creating new genetic resources in breeding for immunity.

1. Research progress of wheat powdery mildew

1.1. Harm and distribution of powdery mildew in wheat. Wheat is susceptible to a variety of diseases throughout its life. These diseases are widely distributed and highly adaptable, which pose a significant threat to wheat yield (Zhao Mingyue et al., 2016). Illnesses caused by fungal pathogens alone reduce wheat yield by 15 to 20 % per year (Figueroa et al., 2018). Generating billions of dollars in damage to the global economy (Dean et al., 2012). After powdery mildew infection, wheat plants are prone to lodging. Their leaves dry and die quickly, which seriously affects the average growth and development of wheat (Dean et al., 2012; Morgounov et al., 2012). Powdery Mildew caused by Powdery wheat mildew can cause severe yield loss and grain quality deterioration in a short time (Morgounov et al., 2012). Wheat powdery mildew reduced winter wheat by 13 % and spring wheat by 20 % (Griffey et al., 1993; Conner et al., 2003; Lackermann et al., 2011).

According to statistics, wheat powdery mildew is distributed from 60 °N to 44 °N and can occur in many wheat-growing areas all year round. The crop yield loss in Russia, Brazil, and China is as high as 35 %, 62 % and 40 %, respectively. Since 2004, wheat powdery mildew has occurred over 6 million hectares every year in China. Studies have shown that it is a crucial disease mainly occurring on the leaves. In severe cases, the stalk, leaf sheath, and ear of wheat will also be infected, and even the leaves will dry up and the whole plant will die. The pathogen of wheat powdery mildew is the obligate parasite of living nutrition, causing only parasitism on living wheat (Singh et al., 2008). Wheat powdery mildew can occur in all wheat growth stages and continuously threaten wheat growth (Kang et al., 2019). When humidity is above 70 %, air temperature is 15–20 °C, nitrogen fertilizer is excessive, and the wheat planting density is high, wheat plants are green and weak, and white powder disease is likely to occur. In a dry land, with insufficient water, fertilizer, or lodging in the wheat field, the disease resistance of wheat will be weakened, powdery mildew often will be more serious (Panstruga & Lefert, 2003).

But belongs to ascomycetes subphylum fungi, conidia are elliptic, the obturator shell of pathogenic bacteria is black spherical, containing 9–30 ascus. Ascospore is round to elliptic. The ascospore shell is usually formed in the late wheat growth stage and can release ascospore after maturity (Fig. 1). Powdery mildew is widely distributed, with rapid toxicity variation and complex and changeable physiological species of pathogenic bacteria (Luo et al., 2002; Cui, 2008).

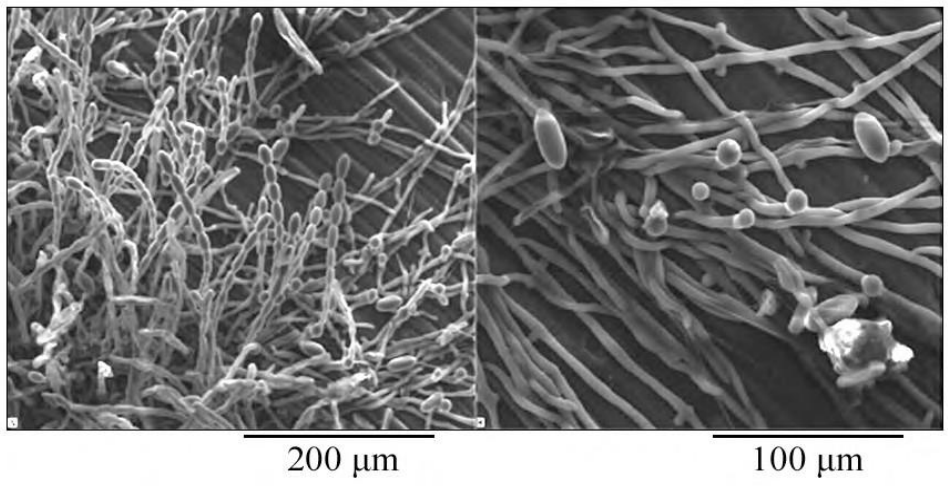

Fig. 1. The scanning electron micrograph of *B. graminis* (Luo et al., 2002).

1.2. Growth cycle of wheat Powdery mildew. Wheat powdery mildew infected wheat in a suitable environment and began to reproduce. The invasion process is as follows: first, A single conidium is blown onto the leaf, and about 1 hour later, the primary bud tube appears at one end of the obsidian (Fig. 2). The primary bud helps identify the host surface cells, attach them tightly to the leaf surface, and extract water from the host surface. Over the next few hours, a second bud tube grows from the other end of the spore, elongates toward the leaf surface, and forms an enlarged structure at its back called an aptamina, which attaches to the epidermis. After about 12 h, powdery mildew penetrates the cell wall of the host cell by invading the nail. Through the interaction between powdery mildew and host cells, about 50–70 % of the spores can successfully penetrate the cell wall, depending on the environmental conditions, the host cells, and the spores themselves (with the most significant impact).

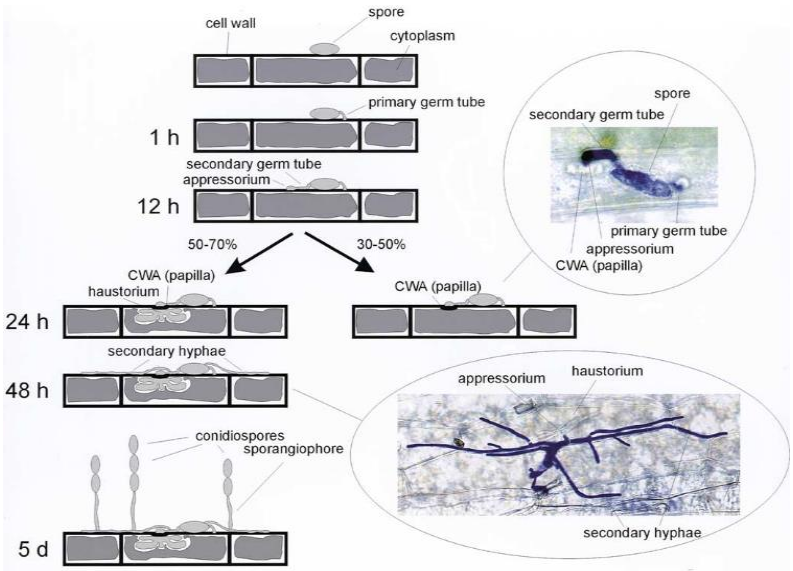

Fig. 2. Schematic diagram of powdery mildew bacteria development and scheme of the asexual life cycle of the powdery mildew fungus (Panstruga & Schulze, 2003).

The spores that successfully penetrate the cell wall, powdery mildew bacteria will form haustorium after 24 hours, which is a particular infection structure with finger-like protrusions. Houston can invade the plasma membrane of the host, also known as the organ in which the pathogen takes nutrients. After the successful establishment of the haustorium, airborne hyphae began to grow in the epidermal cells of the leaves and gradually infected other cells nearby, then formed more haustorium, and finally formed the colony of white hyphae net. After about 4–5 days of growth, hyphae will form short upright sporophytes with 5–10 conidia chains, and yet, a large number of mature conidia will be released to start the next infection cycle. This clonal propagation is the primary propagation mode of powdery mildew when the conditions are suitable (Cao et al., 2011). However, in winter, powdery mildew had sexual reproduction and existed in the closed capsule form on the leaves of the aged host (Yanrong & Geng, 2020). When released from the ascus, the ascospore behaves like conidia and begins a new round of infection (Qu Yunfeng, 2019).

2. Advances in plant disease resistance

2.1. Molecular mechanism of plant disease resistance. Plants in the natural environment in the growth and development of the whole process will be subject to various pathogenic microorganisms invasion, and pathogenic microorganisms mainly include pathogenic fungi, bacteria and viruses, etc. For a long time in the co-evolution of plants and pathogens, various defense systems have gradually evolved to inhibit the destruction of pathogens. When pathogenic bacteria invade the plant, a series of signals can be generated immediately in the plant body and transmitted to activate the plant's defense system to resist the invasion of pathogenic bacteria. Plant defense system mainly includes two immune defense line; the first line of defense is the body's immune response (Pathogen–Associated Molecular Patterns, PAMPs, PAMP–Triggered Immunity, PTI), the process is Triggered by the Pathogen Associated Molecular Patterns, when Pathogen invasion to the surface of a plant, grows on the plant cell membrane on the surface of the pattern recognition receptors can identify the Pathogen Associated Molecular Patterns PAMP, through the signal transduction, triggering an immune system response. The second line of defense is effector-triggered (ETI). Effect factors trigger this process. When pathogenic bacteria invade the surface of plants, plant disease-resistant genes secrete Effector factors that can recognize pathogenic bacteria and trigger immune system response mediated by Effector factors (Li et al., 2011).

The path of plant resistance to pathogen invasion is a very complex network. Signaling molecules play an essential role in this network. Still, the same signaling molecule can be produced in different response pathways, and the same pathogen can also stimulate other signaling molecules (Zhang & Zeng, 2019; Yang & Gao, 2016). When a pathogen enters a plant, the inside body of the plant can produce a series of signal molecules immediately, carry on transmission, excite plant oneself defense system then, make plant has the ability to resist pathogen thereby. Many signaling molecules play a role in stimulating and regulating plants' defense systems, including Ca^{2+}, salicylic acid (SA), jasmonic acid (JA), ethylene (ET), and reactive oxygen species (ROS). Ca^{2+} can not only maintain the osmotic pressure of plants but also participate in regulating the signal transduction pathway of pathogenic bacteria in plants. It is an essential second messenger molecule of plant cells. Pathogenic bacteria induce Ca^{2+} crosses the cytoplasmic membrane, resulting in intracellular Ca^{2+} As the concentration increases, the osmotic pressure increases, and the corresponding protein kinases are activated (Fan & Jiang, 2005). Salicylic acid can activate the production of some related proteases, thus making plants resistant to disease. Experiments have shown that salicylic acid content will accumulate in large quantities after plants are invaded by pathogenic bacteria (Ding Lina & Yang Guoxing, 2016). Also, salicylic acid in the process of plant resistance to pathogen invasion and H_2O_2 is closely related, and H_2O_2 can determine the host disease resistance response in plants. Simultaneously, salicylic acid may inhibit catalase activity (Grant & Lamb, 2006). Jasmonic acid and ethylene are ubiquitous in plants. They are not only the growth regulators in plants but also the signal molecules in plants in response to the invasion of pathogens. Studies have shown that salicylic acid, jasmonic acid, and ethylene have a close cross-

connection in response to pathogenic bacteria's invasion (Dong, 1998).

2.2. Research progress of genes related to plant susceptibility. Plants for pathogen resistance and disease, now most of the scholars in the fields of disease research, few people engaged in disease research, the study of disease genes is very few, but the plant disease resistance and disease of plants and pathogen interaction are equally important (Penninckx et al., 1996; Zhang Jianxia et al., 2008).

The concept of susceptibility factors was first proposed by British scholars Schulze and Vogel in 2000 (Luo Sulan & Zheng Xueqin, 2000). Subsequently, in 2002, Eckardt made a further discussion on the concept of susceptible genes, regarding them as essential factors for the successful invasion, growth, development, and reproduction of pathogenic bacteria (Schulze Lefert, & Vogel, 2000). Hy trialability gene (S gene), which can facilitate pathogen infection and facilitate affinity in plants, is currently defined as a susceptibility gene (Eckardt, 2002).

In the interaction between host and pathogen, infection genes assist pathogen invasion through the following three aspects to increase the degree of plant infection. First: When pathogens invade the host, susceptibility genes can help the host identify the pathogens and their affinity. For example, the host specialization toxin (HST) is capable of producing specialization in plants due to the interaction between the susceptibility gene and the virulent gene product of the pathogen (Liu Chao et al., 2018). Secondly, susceptibility genes can encode negative regulators with immune signals. For example, the CPR1/CPR30 gene in Arabidopsis can encode and translate F-box protein and has the ability to regulate the accumulation of SNC1 protein negatively. When the CPR1/CPR30 gene is mutated, the plant's disease resistance ability is significantly improved (Shang Ming Qing & Liu Aixin, 1998). Third: in pathogen and host mutual affinity, after invading the host, the disease gene can assist the growth and reproduction of pathogenic bacteria, for the metabolism and structure of pathogenic bacteria to provide the necessary nutrients. For example, in arabidopsis thaliana, pepper, tomato, and lettuce and other higher plants widely exist A kind of disease genes, the host of the translation initiation eIF4G and poly real viruses effect VPg, polymerase NIb and PABP (poly real A binding protein) translation initiation complex formation, combining the RNA virus m 5 'end cap structure, help complete the translation and viral RNA synthesis of viral proteins, when they had mutations, can improve the host resistance to the virus (Guo et al., 2012). Many experimental results showed that when the susceptible genes were mutated or lost, the resistance of plants to pathogen invasion was greatly enhanced. Finally, the invasion ability of pathogens was weakened.

The first gene was discovered by Vogel, an American researcher, John, an essential gene in *Arabidopsis thaliana* infection. Vogel named the gene PMR6. Deleting the PMR6 gene resulted in a mutation that showed high resistance to powdery mildew (Robaglia & Caranta, 2006). Subsequently, the disease susceptibility genes were cloned on many crops. After the $OsSSI_2$ gene was silenced in rice, the plant resistance to blast and leaf Bsr was significantly improved. After the mutation of the BSR-D1 gene, a large amount of hydrogen peroxide would accumulate in the cells, thus improving the disease resistance of rice (Vogel et al., 2002). After the interference of the GHWRKY106-1 gene with RNA interference technology in cotton, the expression of PRs, a protein related to disease course, would be significantly enhanced in cotton, thus improving the disease resistance of cotton (Jiang & Shimono, 2009). When the HVBI-1 gene was overexpressed in barley, the disease resistance of barley would be weakened, while when the HVBI-1 gene was silenced, the disease resistance of barley would be enhanced (Li & Zhu, 2017). When the TaS3 and Blufensin1 genes were silted, wheat resistance to powdery mildew and stripe rust was significantly enhanced (Li & Zhao, 2015). At present, the cloned plant susceptibility genes mainly include transcription factors, enzymes, transmembrane proteins, and other types (Eichmann & Bischof, 2010).

2.3. Research progress of powdery mildew resistance genes in wheat. In 1930, an Australian scholar reported for the first time that there was an anti-powdery mildew gene in wheat Thew, and it was dominant, thus revealing a wave of genetic research on wheat powdery mildew. The first powdery mildew gene was named Pm1 in 1950 and was located on the 7AL chromosome of wheat. So far, more than 90 powdery mildew resistance genes and their alleles, called PM1-65, have been identified (Lei Xiuyu, 2013). About half of these powdery mildew resistance genes are derived from normal wheat. Also, about one-third of the related species derived from wheat include one-grain wheat, emem-grain wheat, rough-goatgrass, and Timofeewili wheat. The remainder is derived from haretodes and haretodes rye (Chantret & Pavoine, 1999). Now, most of the resistance genes have lost resistance to powdery mildew or are very weak (Song et al., 2014; Zou et al., 2017). Only a small number of genes or alleles remain resistant to powdery mildew (Liu, 2016). In the main wheat-growing areas, Pm8 resistance was lost (Wang, 2017; Sun, 2015). The resistance of Pm2 and Pm4b was also gradually lost in the Yellow and Huai wheat region (Chi Wenjuan et al., 2007). At present, only Pm1c, Pm12, Pm21, Pm24, and Pm35 genes still have a strong resistance to powdery mildew, among which Pm21 is a rare broad-spectrum resistance gene (Zhang et al., 2004). Moreover, some disease resistance genes have been applied in wheat breeding, such as Nannong 9918 carrying powdery mildew resistance gene Pm21, Liangxing99 carrying powdery mildew resistance gene Pm52, Bannong AK58 moving powdery mildew resistance gene Pm8, etc. (Jingwei Zou et al., 2016), and achieve more significant economic benefits. Researchers study of wheat powdery mildew in the past was mainly focused on positioning and cloning of disease resistance gene mining. The current research results show that the resistance genes and powdery mildew in the evolution process, the resistance of the resistance genes out quickly, as the disease has progressed, this situation will be more and more can't meet the needs of wheat genetic improvement, is an urgent need to explore new ways of wheat powdery mildew resistance breeding to improve wheat lasting broad-spectrum resistance to powdery mildew.

3. Research progress of Pm46 gene

3.1. Gene discovery and research status. Wheat powdery mildew resistance inheritance is diverse, which is controlled by both main effect quality genes and quantitative traits of micro-effect polygenes. Since Waterhouse, an Australian scholar, first reported in 1930 that the wheat variety Thew carried a dominant powdery mildew resistance gene, scientists have identified several genes in wheat and related genera resistant to powdery mildew. These resistance genes are mainly dominant, and only a few are recessive (Li Jiao, 2019). International designation for the powdery mildew resistance gene in wheat is Pm. To solve the

damage of wheat powdery mildew, breeders transferred the powdery mildew resistance genes into wheat to cultivate the disease-resistant varieties. For example, a new variety of wheat resistant to powdery mildew (Jauhar & Chibbar, 1999) can be obtained by transferring a rye chromosome with the Pm8 gene into normal wheat (Huerta Espino & Chen, 2015; Jauhar & Chibbar, 1999). Some anti-powdery mildew genes also have a polygenic effect. For example, the resistance gene Lr34 was obtained using the mapping cloning method, which encodes a transferase subfamily protein at an ATP binding site and can also be used for rust and powdery mildew (Lillemo et al., 2008). Adult resistant genes Pm39 and Pm46 have also been proved to be "one-cause-multipotent" and resistant to wheat rust and powdery mildew (Lan et al., 2014).

In 1979, Canadian scientists Dyck and Samborski found a leaf rust resistance gene at the adult stage from Pakistani wheat. Later, they introduced the resistance gene into wheat variety Thatcher by successive backcross and obtained a resistant strain RL6077 (Thatcher*6/PI 250413) (Dyck & Samborski, 1979). Later, it was found that RL6077 was also resistant to stripe rust and stem rust (Dyck et al., 1994; Singh, 1992). In 2009, Lagudah confirmed that there was no Lr34 gene in RL6077 using molecular markers and speculated that RL6077 contained a new multi-disease resistance gene (Lagudah et al., 2009). Hiebert by observing the chromosome pairing behavior, refuted the previous views on the translocation of the Lr34 gene on 7DS to other stains (Hiebert et al., 2010). Further, genome-wide SSR molecular markers were used to analyze the osmotic chromosome fragments from donor wheat PI 250413 in RL6077, and it was found that 5 polymorphic SSR molecular markers (Xcfd71, Xbarc98, Xcfd23, Xwmc457, and Xwmc48) were associated with the leaf rust resistance genes in Thatcher/RL6077 and RL6058/RL6077 populations. Then linkage analysis using a third isolated population from RL6077 showed that the 4DL SSR marker Xcfd71 was closely linked to the resistance gene. The new gene in RL6077 was officially named Lr67 because no rust-resistant genes have been reported on the 4DL. Herera-Foessel located stripe rust resistance genes Yr46 and Lr67 in RL6077 to the same region of the 4DL chromosome. Subsequently, Herera-Foessel also found that the Lr67/Yr46 site could provide stem rust and powdery mildew resistance and presented the symptoms of tip necrosis, so it was named as the polypotent site: Lr67/Yr46/Sr55/Pm46/Ltn3. (Herera-Foessel et al., 2011; Herera-Foessel et al., 2014) The full length of the predicted Pm46 resistant protein gene consists of 1545 bases encoding 514 amino acids, contains 12 predicted transmembrane helices and is most similar to the STP13 family of H+/monosaccharide co-transporters, which promotes hexose cross-membrane transport. Their corresponding pleiotropic or tight chain gene, named Sr55, Pm46, and Ltn3, can be used to provide a broad spectrum of durable wheat resistance (Zhang, 2017). In terms of geographical distribution, the PM46 gene was found mainly in local varieties in the Punjab of India and was rarely carried in other regions. There are few reports of the PM46 gene in Wheat varieties in China. Wang Zhiwei used molecular marker CSTM4_67G to detect 42 wheat varieties and higher generations grown in Yunnan province and found that Yunmai 75, Yun15D4-15, Yimai 1, Yimai 3, Fengmai 32, and Fengmai 35 contained dual-resistant adult rust-resistant gene Lr67/Yr46/Sr55, accounting for 14.29 % of the tested materials (Wang Zhiwei et al., 2020). Both barley and wild barley themselves carry the Lr67 lineal homologous gene (HvSTP13), but neither has the G144R mutation-specific for the disease-resistant allele. Milne introduced G144R variation into HvSTP13 and obtained stable transgenic barley lines. Disease identification showed that transgenic barley showed leaf rust resistance at the seedling stage and plant stage, suggesting that the Lr67 gene mediates conservative disease resistance in barley and wheat (Milne et al. 2019).

3.2. Disease resistance mechanism of PM46 gene. The research of John, Moore with Sybil, Herera-Foessel et ales showed that Lr67res protein might reduce hexose transport by forming an inactive heterodimer protein complex that produces a dominant-negative interference mechanism. This is consistent with dominant or semi-dominant resistance phenotypes given by Pm46 genes and with phenotypic susceptibility due to deletion of this locus. Dimer-mediated dominant negative interference with transporter activity has been found in other plant sugar transport families (Dyck & Samborski, 1979). The partial resistance of Lr67res protein to different vivisection pathogens in wheat and barley may be due to the host cells' resistance to extracellular hexose detection, thus increasing the ratio of hexose/sucrose in extracellular hexose. This, in turn, induces a sugar-mediated signal response, creating an environment that is more hostile to the growth of the pathogen. The inhibition of hexose detection by Lr67res is similar to the invertase activity response induced by ubiquitous plant pathogens invading cell walls, which will change the extracellular hexose/sucrose ratio and cause hexose-mediated defense response (Jiang & Shimono, 2009).

Sugars contribute to various physiological processes and act as substrates and signaling molecules in plant defense responses (Moore & Herrera-Foessel, 2015). Activation of sucrose transport by some bacterial virulence protein-coding genes promotes host susceptibility, whereas eliminating these genes induces host resistance (Liesche et al., 2011). It remains determined whether the Pm46 gene is also detrimental to host resistance to inanimate nutritive pathogens in field-grown crops. Nevertheless, as a valuable tool for developing broad-spectrum resistance in crops, the Pm46 gene provides a favorable breeding strategy for combining different forms of broad-spectrum resistance.

4. Advances in GDSL gene research

GDSL lipase (EC 3.1.1.3) is a hydrolyzer, which can hydrolysate a variety of substrates such as thiolates, aryl esters, phospholipids, and amino acids. GDSL lipase has a unique structural characteristic with GDSL conserved amino acid sequence at the N' end of the protein, different from other lipase types with GxSxG conserved sequence. Upton and Buckley first identified the conserved domain and named it (PFAM PF00657). Subsequent studies have found that this type of lipase is widely present in prokaryotes and eukaryotes. With the development of more plant genome sequencing and bioinformatics, GDSL lipase is found to be a large gene family. At present, GDSL lipase is widely known to be involved in the average growth and development of plants, organ morphogenesis, secondary metabolism, stress, and other physiological activities, and plays an important role in the lipid metabolism of oil crop seeds (Proels & Huckelhoven, 2014). However, systematic understanding of the structure, classification, evolution, expression, and function of the family's genes is lacking.

4.1. Gene structure and species of GDSL lipase family. Plant GDSL lipase gene family is a large, Volokita for different land plants such as amino acids coded 604 GDSL lipase gene

sequence comparison and analysis, found that plant GDSL family members in the phylogenetic tree form three big family (subfamily A, B, and C), each branch contain A GDSL genes from different plants (Chen et al., 2010). Most plants GDSL family genes by four-five exons and introns (Akoh et al., 2004), Volokita for hundreds of different plants, such as GDSL lipase gene of the structure are analyzed, the results found that there are conservative, 6 introns in the six introns in three distribution different: in the family of introns 1 and 6 in the family of three are conservative, did not change, exists in most several GDSL lipase genes; Introns 5 are conserved in subfamilies A and B, while introns 2, 3, and 4 are specific in subfamilies A, B, and C, respectively. Members of the GDSL family are distributed across the chromosomes of plants, but not evenly. Some GDSL lipase genes are distributed in clusters on chromosomes. Take the arabidopsis genome as an example. Two or more GDSL lipase genes are clustered or arranged in tandem on a total of 12 chromosomal loci. Some other parts of the genes on the same chromosome, or on different chromosomes duplicate, lead to more copy phenomenon (Chepyshko et al., 2013). In addition, the gene degeneration mechanism will be the main force to drive the evolution of the GDSL family.

4.2. GDSL is involved in growth development and stress response. Pathogens can induce the expression of GDSL lipase genes in some plants, hormones such as salicylic acid, ethylene, jasmonic acid, and abiotic stress factors, indicating that they may be involved in plant resistance and stress response (Ling et al., 2006). It reported the news of salicylic acid inducing arabidopsis GDSL lipases GLIP1 disease-resistant activity, GLIP1 mutant plants of saprophytic fungi spore canola raw chain grid (*Alternaria brassicicola*) is more sensitive than the wild type, the recombinant expression GLIP1 protein with lipase activity, integrity. It can directly damage the fungal spores and inhibit its germination. Besides, the lipase can also induce the plant to produce resistance to the fungus system (Lee et al., 2009). Further studies found that excessive expression of GLIP1 in plants can enhance resistance to various pathogenic fungi and bacteria, and GLIP1 induces phylogenetic resistance of plants through ethene-mediated signaling pathways (Oh et al., 2005). Similarly, the expression of GLIP2 in *Arabidopsis thaliana* can be caused by salicylic acid, jasmonic acid, and ethylene, and has an inhibitory effect on fungal spore germination. However, GLIP2 may mediate plant disease resistance by down-regulating auxin signaling pathways (Kwon et al., 2009). It conducted a similar study on the CaGLIP1 homolog of pepper and found that its expression was induced by salicylic acid, jasmonic acid, ethylene, bacterial infection, high salt, drought, injury and other stress factors (Hong et al., 2008). Unexpectedly, capsicum plants with down-regulated CaGLIP1 expression have increased background resistance to *Xanthomonas campestris* Pv. It can be seen that, as a large plant gene family, lipases have diverse functions. Different lipases in the same species can differentiate into different functions, and the functions of homologous lipases in other species may also be different.

5. VIGS and RNAi technology

5.1. Technical principle of VIGS. VIGS is a technique that USES recombinant virus specificity reduces endogenous gene activity, based on post-transcriptional gene silencing (PTGS) (Wang Ya-ru & Yao Yun-cong., 2015). Usually VIGS viral vector can be combined with the host plants of the target gene, using PTGS as a natural antiviral defense line to fight the virus proliferation, genetic transformation mediated by agrobacterium infect plants, inserted into the part of the viral genome, its RNA degradation mechanism and the way of RNA interference are very similar, both in the virus genome to add multiple cloning sites, to their target genes into the host plant. VIGS vector inoculation in plants is usually achieved through agrobacterium tumefaciens infection by integrating t-DNA containing the viral genome into the host genome of at least one cell for standard transcription translation. This leads to the production of double-stranded RNA (dsRNA) from the viral ssRNA template, and Dicer proteins cut this viral dsRNA into short interfering RNA (siRNAs) duplicates, approximately 21–24 nucleotides in length. These siRNAs, in turn, are incorporated as single-stranded RNA molecules into RISC (RNA-induced silencing complex), which screens and destroys RNA complementary to siRNA (Yao et al., 2009; Zhang et al.,2014; Kumagai et al.,1995). In the particular case of VIGS, the viral RNA and target gene mRNA were cleaved. The virus-derived silencing signals are further amplified and spread systematically throughout the plant (Fig.3). It is assumed that siRNAs of about 21nt length mediate short-range transport, while RNA-dependent RNA polymerase 6 (RDR6) requires long-range transport, possibly amplifying the silencing signal. The systematic propagation of silencing signals occurs regardless of the successful movement of virus particles in the plant. When VIGS was applied to susceptible plants, the target gene mRNA of host plants was degraded in most plants (Baulcombe, 1999).

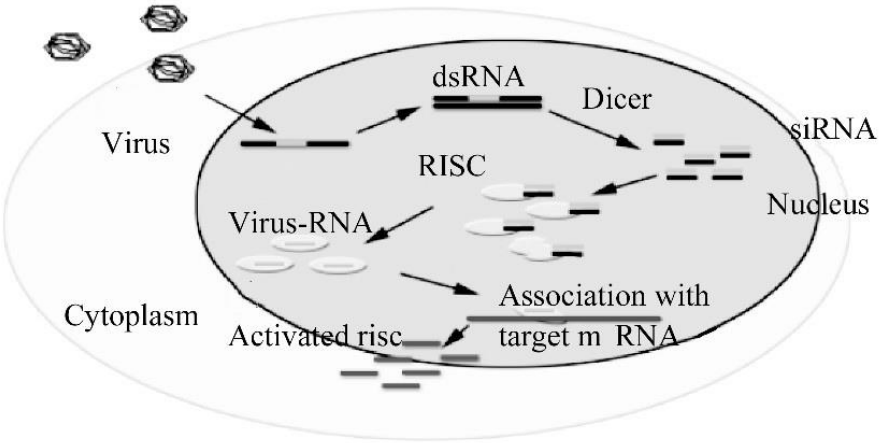

Fig. **3** The Molecular Mechanism of VIGS Technology (Baulcombe, 1999).

5.2. Application of VIGS technology. Large-scale sequencing of functional genomics in non-model plants provides primary data for studying the structural evolution of genomes or the history of repeated events in plant lineages. These new data have also contributed significantly to gene discovery and pave the way for further understanding of gene function evolution, plant-pathogen interactions, biosynthetic and developmental pathways. However, genetic tools are mainly limited to model plants such as *Arabidopsis thaliana*, rice (*Oryza sativum*), or tobacco (*Nicotiana tabacum*), and therefore methods for analyzing gene function in other non-model plants are minimal (Ratcliff et al., 2001; Baumlein et al.,1991; Turnage et al., 2002). It is particularly challenging for most plants to establish a repeatable stable genetic transformation program. As a result, VIGS, which can silence specific genes, is a powerful technique that has been successfully used in a variety of species. As the tools of functional genomics are increasingly used in plant species such as *Zea mays*, *Hordeum vulgare*, and wheat, it is tough to analyze gene function by conventional methods. VIGS technology enables the rapid study of gene function. Since almost all VIGS vectors originally used in dicotyledonous plants were derived from viruses that originally host Solanaceae, some VIGS vectors were successfully extended to other Solanaceae plants (especially tomatoes, bell peppers, and petunias) (Holzberg et al., 2002). Tobacco Brittle Virus (TRV) has a high susceptibility to a wide range of hosts and mild post-infection symptoms and is preferred as a VIGS resource for dicotyledons. More recently, VIGS are effective against rosaceae plants such as arabidopsis, peas, and cassava (Lacomme et al., 2003). TRV's experimental host range has now been extended to several species of buttercup. Recently, a new VIGS vector system was developed from Apple Latent Spherical Virus (ALSV), which can also be used in a variety of higher dicotyledonous plants, including night plants, arabidopsis, and legumes. Monocotyledons such as barley, rice, wheat, and maize are also susceptible to TRV (He Zhengbo et al., 2019). VIGS has become an essential reverse genetic tool for revealing the gene function of species that have difficulty achieving stable genetic transformation or achieving transformation.

5.3. Principles of RNAi technology. As a gene knockout technique, RNA interference (RNAi) has been widely used to analyze the gene functions of various organisms. It is a post-transcriptional gene silencing phenomenon induced by double-stranded RNA (Fire et al., 1998; Sunkar et al., 2012). Because of its high specificity and effectiveness, it has become a useful tool for gene function analysis. Detailed molecular mechanism of RNAi as shown: first of all, long dsRNA by RNase Ⅲ Dicer identify family members, and was cut into 21 nucleotides in length. When each siRNA is disbanded, one of the two strands is preferentially incorporated into the RNA-induced silencing complex (RISC). The antisense strand of siRNA was hybridized with the mRNA as a guide, and RISC cleaved the mRNA near the center (Fig. 4) (Mao et al., 2007).

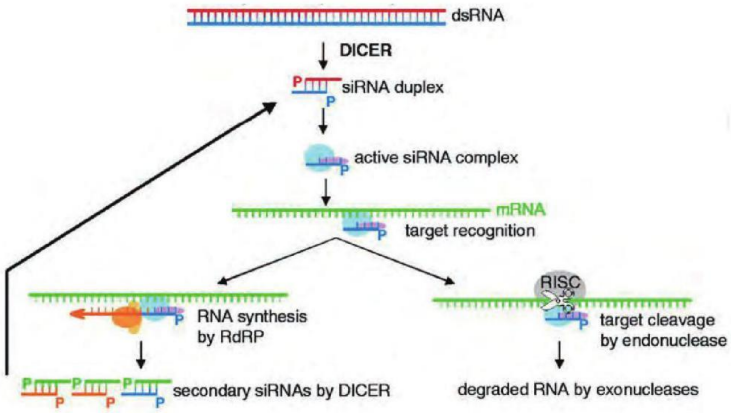

Fig. 4. The Mechanism of RNAi (Mao et al., 2007).

5.4. Application of RNAi technology. As a highly significant gene suppression technology, RNAi technology has been widely used in crop genetic improvements, such as disease resistance, quality improvement, and abiotic stress tolerance. Fire demonstrated for the first time that RNAi could be used for pest control by injecting C. elegans with bacteria that express dsRNA targeting (Wang Weiwei et al., 2017). Mao designed specific dsRNA according to CYP6AE14, the cytopigment gene of cotton bollworm, and introduced it into cotton, and obtained transgenic plants with apparent insect resistance to cotton bollworm (Zhong et al., 2016), which further confirmed the feasibility of using RNAi technology to cultivate insect-resistant crops. At the same time, Bt insecticidal protein Cry3Bb1 and DvSnf7 insecticidal resistant corn have been commercially cultivated (Mao & Zeng, 2014). Yang Xiangdong with colleagues constructed a P3 gene RNAi vector of Soybean Mosaic Virus SC-3 strains and introduced it into cultivated soybean varieties, and found that transgenic soybean plants had good resistance to multiple Soybean Mosaic Virus (SMV) strains such as SC-3, SC7, SC15, SC18 and SMV-R and Watermelon Mosaic virus under field conditions, and the resistance traits could be stably inherited (Yang et al., 2018). Zhong Xiaofang with colleagues introduced the RNA interference fragment of HG-RPS-23 gene into soybeans and obtained a new transgenic soybean material that could significantly improve the resistance to the physiological subspecies of soybean cystodes 3 (Zhong, 2004). By blocking the expression of ACC oxidase, ethylene formation in tomatoes could be significantly reduced and shelf life could be extended, while synthesis of fruit softening substances such as -mannosidase and -acetylhexanase could be inhibited, which could increase the shelf life of tomatoes (Oropeza et al., 2020). At present, RNAi transgenic crops are mainly completed by agrobacterium-mediated method, which has the ad-

vantages of simple operation and low cost. Agrobacterium contains Ti plasmids and Ri plasmids, and a section of t-DNA (transferring-DNA) is attached to the plasmids. After agrobacterium enters the cells, it can integrate this section of t-DNA into the genome of the infected plant and inherit it stably (Li Junxiang & GU Qisheng, 2020; Yang Jing, 2019). At present, RNAi technology in plant research is widely used in many fields, such as disease resistance, insect resistance, quality improvement and breeding, abiotic stress such as drought, salinity, cold tolerance in the areas of study were made certain progress, in the study of crops at various stages of crop growth and development, biological and abiotic stress response has extensive application prospect.

Conclusion. Wheat is one of the grain crops with the largest planting area, the largest yield, and the highest nutritional value in the world. During the growth and development of wheat, it is always in a struggle with the stress of adversity. Both biological anxiety and abiotic stress have a significant influence on the growth of wheat. The lesser degree of stress is manifested as slow development and reduced disease resistance. The more severe degree of stress can result in a significant reduction or even no harvest of wheat. The cultivation of resistant varieties is the most economical and effective way to control powdery mildew in wheat. Still, there are some limitations in the cultivation of resistant varieties by introducing resistance genes by conventional means in actual production. With the increase of disease each year, this situation will be more and more unable to meet the needs of wheat genetic improvement, it is urgent to explore a new way of wheat breeding resistance to powdery mildew to improve the wheat to powdery mildew lasting broad-spectrum resistance.

Multiple benefits can be gained through future critical research efforts, including the following:

1. The use of gene Pm46 in China is less, and its application in breeding is worth expecting, so the use of this gene in wheat breeding in China should be strengthened.

2. In-depth analysis of the mechanism of action of Pm46 can provide a theoretical basis for us to obtain broad-spectrum resistance in wheat by manipulating susceptibility genes or harmful resistance regulation genes.

3. Whether Pm46 is also detrimental to host resistance to non-vivisection pathogens in field-grown crops remains to be determined.

4. The effective utilization of the existing multi-effect resistance genes, identification and cloning of new genes will lay a solid foundation for the breeding of concurrent resistant and durable resistant wheat varieties in China.

If these researches can achieve breakthrough results, it will be another breakthrough direction for wheat disease resistance breeding to obtain new genetic resources.

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ДОСЛІДЖЕННЯ ТА ПЕРСПЕКТИВИ ГЕНІВ СТІЙКОСТІ ДО БОРОШНИСТОЇ РОСИ ПШЕНИЦІ

Пшениця (Triticum aestivum L.) одна з основних зернових культур у світі. Борошниста роса пшениці – грибкове захворювання, спричинене інфекцією Blumeria graminis F.sp. tritici. Це одна з найважливіших хвороб пшениці у світі, яка серйозно впливає на врожайність та якість пшениці. Наразі основними способами боротьби з борошнистою росою є використання фунгіцидів та вирощування стійких до хвороб сортів. Оброблення фунгіцидами спричиняє накопичення залишків пестицидів та забруднення навколишнього середовища. Наразі джерел резистентності до борошнистої роси дикорослих видів та штучно виведених сортів пшениці не вистачає, тому необхідно терміново та ефективно створювати стійкі сорти. Традиційне схрещування має тривалу і низьку ефективність. Тим не менше, це швидкий та ефективний спосіб отримання стійких до хвороб сорти, використовуючи сучасні молекулярно-біологічні засобів для перенесення стійких до хвороб генів у культивовані сорти. Незважаючи на те, що вирощування стійких сортів є найбільш економічним та ефек-

тивним способом контролю борошнистої роси у пшениці, існують певні обмеження при введення генів стійкості звичайними способами для вирощування стійких сортів у товарному виробництві. Зі збільшенням захворюваності з кожним роком ця ситуація буде дедалі більше нездатною задовольнити потреби генетичного вдосконалення пшениці. Необхідно терміново дослідити новий спосіб створення стійких сортів пшениці до борошнистої роси, що забезпечуватиме тривалий широкий спектр дії.

Резистентність до хвороби через селекційне клонування рослин та взаємодії спорідненості патогенів відіграють ключову роль у вивченні фітопатогенів та їх механізму дії. Наразі у пшениці шляхом маніпулювання генами хвороби інфікований матеріал отримав тривалий широкий спектр дії меншої резистентності. У випадку захворювання генів та мутацій після такої резистентості до хвороби механізм досі незрозумілий. Отже, для селекції стійких сортів потрібні добування та використання генів стійкості. У статті підсумовується шкодочинність та розповсюдження борошнистої роси пшениці, механізм стійкості генів резистентності до борошнистої роси пшениці, а також функціональний аналіз, гени стійкості до борошнистої роси пшениці у галузі досліджень молекулярної біології, а також VIGS, RNAi, агробактеріальний принцип та застосування технічних засобів, таких як профілактика та боротьба з борошнистою росою пшениці, дослідження нових генів резистентності до борошнистої роси та регулювання стійкості, селекція стійких до хвороб сортів пшениці, що забезпечують бажану схему.

Ключові слова: *пшениця, борошниста роса пшениці, гени стійкості до борошнистої роси пшениці, VIGS, RNAi.*

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