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Molecular Response Mechanism of Cotton to Verticillium Wilt and Fusarium Wilt

Xiaojing Yang, Tianze Zhang
Modern Agriculture Research Center, Cuixi Academy of Biotechnology, Zhuji, 311800, Zhejiang, China
Corresponding email: tianze.zhang@cuixi.org
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Abstract Cotton is an important fiber crop worldwide and is threatened by soil-borne fungal pathogens such as *Verticillium dahliae* and *Fusarium oxysporum*, which cause Verticillium wilt and Fusarium wilt, respectively. This study systematically explored the molecular responses of cotton to these two devastating diseases, aiming to lay the foundation for improved disease resistance strategies. We first outlined the biological characteristics, infection mechanisms, and global distribution of the pathogens, and then discussed in detail the innate immune response of cotton, including pattern recognition receptors, phytohormone-mediated pathways, and effector-triggered immune responses. We further emphasized the changes in the transcriptome and proteome during infection, as well as the functional roles of resistance genes, transcription factors, and secondary metabolites. We also discussed the recent progress in functional genomics and gene editing tools such as CRISPR/Cas in the discovery and validation of resistance genes, as well as the overlapping molecular responses triggered by the two pathogens. Using disease-resistant Xinjiang cotton varieties as an example, this study will provide a practical reference for regional breeding programs. This comprehensive study highlights the complexity of cotton-pathogen interactions and anticipates that integrating multi-omics data will be key to cultivating durable resistance through molecular breeding and precision agriculture.
Keywords Cotton; Verticillium wilt; Fusarium wilt; Molecular resistance mechanism; Functional genomics

1 Introduction

Cotton (*Gossypium* spp.) is the most basic raw material in the global textile industry. It is also an important cash crop in many countries, especially developing countries. Cotton plays a major role in the production of natural fibers and oilseeds (Shaban et al., 2018). However, cotton cultivation often encounters serious disease problems, especially Verticillium wilt caused by *Verticillium dahliae* and Fusarium wilt caused by *Fusarium oxysporum*. These two soil-borne fungal diseases are among the most serious biological factors that harm cotton worldwide (Man et al., 2022). These diseases not only reduce yields, but also affect fiber quality, putting considerable pressure on the sustainable development of cotton (Wang and Zhang, 2024).

At present, breeding experts generally believe that breeding disease-resistant cotton varieties is the most effective, economical and environmentally friendly way to control Verticillium wilt and Fusarium wilt (Billah et al., 2021). But the problem is that there are relatively few disease-resistant genes available in cotton varieties, which makes breeding work very difficult (Liu et al., 2023). In order to promote the breeding of disease-resistant varieties and reduce dependence on fungicides, we need to have a deeper understanding of cotton's own disease resistance mechanisms, such as which genes are activated, which signaling pathways are involved, and which antifungal substances cotton produces (Liu et al., 2017). Only by mastering these molecular-level response mechanisms can we better apply genetic engineering technology and marker-assisted breeding methods to ultimately breed cotton varieties that can resist a wide range of diseases and have long-lasting resistance (Abdelraheem et al., 2019).

This study will focus on the molecular response mechanisms of cotton to Verticillium wilt and Fusarium wilt. We will talk about which key disease resistance genes, signaling pathways, and physiological responses have been discovered and studied. This study will also analyze the challenges and possible breakthroughs encountered in

using these molecular research results to breed disease-resistant cotton. In addition, we will also explore the future direction of achieving sustainable cotton disease control in the context of changing environments such as climate change.

2 Overview of Verticillium Wilt and Fusarium Wilt in Cotton

2.1 Pathogen characteristics and infection biology of *Verticillium dahliae* and *Fusarium oxysporum*

Verticillium wilt is caused by a soil-dwelling fungus called Verticillium wilt. Fusarium wilt is caused by a specialized fungus called Vasinfectum (Abdelraheem et al., 2019). Both pathogens enter the cotton plant through the roots and eventually grow in the plant's vascular tissue, interfering with the transport of water and nutrients. There are many different types of Verticillium wilt, some of which cause leaf drop and some do not, and they can survive in the soil for many years as microsclerotia (Wagner et al., 2020). Fusarium wilt also has many different types and multiple physiological subspecies. It also often occurs with root-knot nematodes, which can make the disease more severe. Both fungi are highly adaptable and can infect cotton from seedling to mature stage, which makes disease prevention and control very difficult.

2.2 Comparative symptoms and disease progression in cotton plants

Both diseases cause cotton leaves to turn yellow, wilt, and in severe cases, necrosis, but their development process and symptoms are somewhat different. Verticillium wilt often causes leaves to turn yellow and lose water, with the lower leaves becoming infected first, and the vascular bundles turning brown. If infected with a deciduous strain, the plant will also lose leaves (Bhandari et al., 2020). Fusarium wilt often causes only one side of the leaves to turn yellow, the plant grows slower, and the vascular bundles change color. Sometimes the disease progresses to the death of the entire plant (Figure 1). If it occurs at the same time as nematodes, it will make the symptoms more severe. The severity and development speed of the disease are related to the virulence of the fungus itself, the number of infections, environmental conditions, and the disease resistance of the cotton variety.

2.3 Economic impact and geographical distribution of both diseases

Verticillium wilt and Fusarium wilt are the two most damaging diseases to cotton worldwide, which can significantly reduce cotton yield and quality (Li et al., 2017b). In the United States, yield losses caused by Verticillium wilt ranged from 0.75% to 2.8% over the past 20 years. In China, Spain, and Uzbekistan, losses can reach 30% to 50%. Fusarium wilt has spread to all major cotton-producing areas and has recently been found in Australia. In 2001, it reduced average cotton yields in the United States by about 0.5%. The spread and severity of these two diseases are affected by the variety planted, crop rotation, and local climate. Long-term surveys in China have shown that these factors affect the occurrence of the disease. In major cotton-producing areas such as Xinjiang, China, these two pathogens often appear at the same time, making prevention and control more difficult and increasing the challenges of breeding disease-resistant varieties.

3 Cotton Innate Immune System and Defense Mechanisms

3.1 Pattern recognition receptors (PRRs) and PAMP-triggered immunity (PTI)

Cotton detects fungi such as *Verticillium dahliae* and *Fusarium oxysporum* through pattern recognition receptors (PRRs) on the cell surface. These receptors recognize specific molecules carried by pathogens, also known as PAMPs (Liu et al., 2023). Once these molecules are recognized, cotton initiates PAMP-triggered immunity (PTI), which produces early defense responses. These responses include activation of MAPK cascade signaling pathways, production of reactive oxygen species (ROS), and initiation of expression of some defense-related genes (Zhou et al., 2020). For example, GauSR45a is a serine- and arginine-rich RNA-binding protein that regulates the alternative splicing of PTI-related immune genes (such as BAK1 and CERK1), thereby enhancing resistance to Verticillium wilt. In addition, somatic embryo receptor kinase (SERK) plays an auxiliary role in PTI signaling, which can interact with regulatory proteins such as GRF7 to further enhance cotton's immune response.

3.2 Effector-triggered immunity (ETI) and hypersensitive response

When pathogens release effectors to interfere with PTI, cotton responds with a class of proteins inside the cell. These proteins are called NLRs, which are proteins containing nucleotide binding sites and leucine repeat

structures (Zhu et al., 2023). NLR proteins can recognize these effectors and then initiate ETI (effector-triggered immunity). ETI often triggers local cell death, a reaction called hypersensitive response (HR), which can prevent the further spread of pathogens. GRF7 is involved in regulating the expression of NLR genes (such as ADR1 and NRG1), which enhances ETI levels and improves cotton's resistance to Verticillium wilt. In addition, during the ETI process, many immune genes also undergo alternative splicing. In disease-resistant cotton varieties, such splicing events are more common, indicating that they play an important role in defense responses (Li et al., 2024).

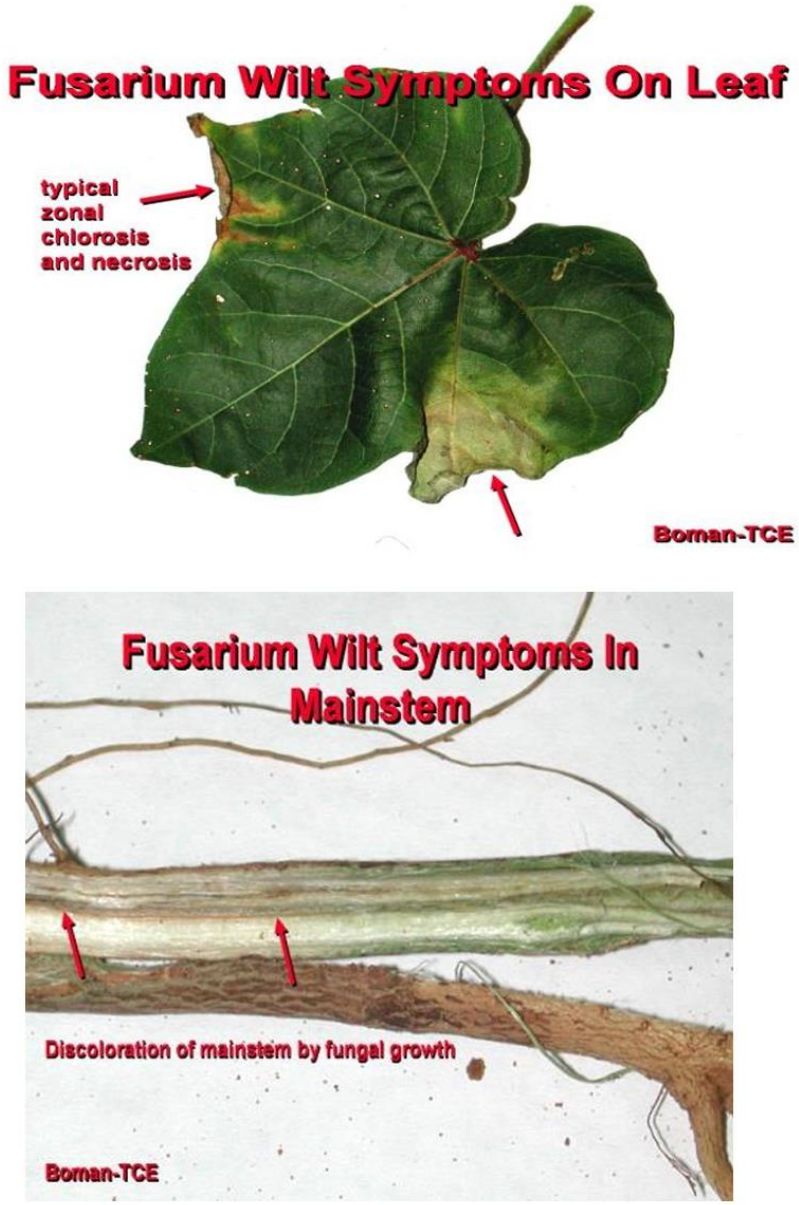

Figure 1 Chlorosis, necrosis and vascular discoloration of cotton by FOV (Adopted from Bhandari et al., 2020)

3.3 Role of phytohormones (SA, JA, ET) in mediating disease resistance

Plant hormones play a central role in the fight against wilt fungi. Salicylic acid (SA) is very important in activating systemic acquired resistance. In disease-resistant cotton varieties, SA content is increased, which can promote the accumulation of ROS and the expression of defense genes (Zhang et al., 2016). Jasmonic acid (JA) is also critical. Genes such as GhOPR9 and GhRFS6 enhance the JA signaling pathway, thereby improving cotton resistance to *Verticillium dahliae* (Chang et al., 2023). In addition, ethylene (ET) also participates in regulating defense responses together with SA and JA. Together, they form a complex signaling network that regulates the

expression of defense-related genes and physiological changes in plants (Xu et al., 2014). The interaction between these hormones determines the strength and specific mode of the immune response. When pathogens invade, both SA and JA signaling pathways are usually activated. Overall, cotton's innate immune system coordinates PRR-mediated PTI, NLR-triggered ETI, and various hormone signals to form a complete defense system to help cotton resist Verticillium wilt and Fusarium wilt, which also provides a basis for the breeding of disease-resistant varieties in the future.

4 Transcriptomic and Proteomic Responses to Infection

4.1 Gene expression changes during early and late stages of infection

Studies have found that after cotton is infected with *Verticillium dahliae* or *Fusarium oxysporum*, its gene expression changes significantly. Transcriptomic analysis shows that many genes related to disease resistance change significantly in the early stage of infection (generally within 24 to 48 hours). These genes are mainly involved in the synthesis of flavonoids, hormone signaling, and the interaction between plants and pathogens (Zhang et al., 2020; Li et al., 2024). Thousands of differentially expressed genes (DEGs) can be seen in both resistant and susceptible varieties. However, the response of susceptible varieties is often more obvious (Zhang et al., 2021a). As the infection continues, some genes involved in lignin synthesis, cell wall changes, and immune signaling remain active. This shows that cotton continues to adjust its defense and adaptation mechanisms.

4.2 Differential protein expression and post-translational modifications

Proteomics studies have shown that the expression levels of many proteins in infected cotton have changed. Most of these proteins are related to stimulus response, secondary metabolism and plant hormone signaling pathways (Gao et al., 2013). In disease-resistant cotton, key proteins such as peroxidase, polyphenol oxidase and cottonpol biosynthesis enzymes increase, which can help cotton improve resistance (Li et al., 2019). In addition, many proteins have undergone post-translational modifications, such as those related to phenylpropanoid pathways and hormone regulation, which can also enhance or change their functions (Gao et al., 2013). Some important proteins, such as GbCAD1, which is involved in cottonpol synthesis, will reduce cotton resistance if they are inhibited, which also shows that these proteins play a key role in defense.

4.3 Functional enrichment of defense-related pathways

Many functional enrichment analysis results show that cotton activates a series of defense pathways when infected with pathogens. For example, in reactive oxygen species (ROS) metabolism, some genes and proteins involved in ROS production and removal are upregulated, which helps plants send out rapid response signals. Lignin synthesis and phenylpropanoid metabolic pathways are also enhanced, making cell walls stronger and limiting the spread of pathogens (Sun et al., 2013). In addition, glutathione metabolism is also important. Studies have found that the detoxification role of glutathione S-transferase in the disease resistance process cannot be ignored (Xing et al., 2024). For example, in terms of hormone signal transduction, related pathways such as salicylic acid, jasmonic acid, ethylene and brassinosteroids are also activated. These signals can cooperate with each other to jointly regulate immune responses (Zhang et al., 2013). These "omics" studies at different levels have given us a more comprehensive understanding of the molecular mechanisms of cotton's resistance to Verticillium wilt and Fusarium wilt, and have also helped us find many disease-resistant genes and key pathways worthy of attention, providing important clues for breeding.

5 Key Molecular Players in Resistance

5.1 Resistance (R) genes and their functional classification

The reason why cotton can resist Verticillium wilt and Fusarium wilt is closely related to whether it has disease resistance genes and whether these genes are active. In particular, those genes encoding NBS-LRR proteins (which have nucleotide binding sites and leucine repeat structures) (Abdelraheem et al., 2019). Through genome-wide association analysis (GWAS) and QTL mapping, researchers have found many NBS-LRR genes on multiple chromosomes. Some of these genes can also resist two diseases at the same time (Li et al., 2017b). For example, a candidate gene called CG02, which contains a TIR-NBS-LRR domain, is more highly expressed in disease-resistant cotton. If it is silenced, cotton will be less resistant to *Verticillium dahliae*. Some R genes are also

critical, such as *GhAMT2*, a high-affinity ammonium transporter that can link immune signals with cell wall reinforcement and systemic defense. In addition, through GWAS and transcriptome studies, genes such as Ghir_A01G006660 and Ghir_A02G008980 were also found to be related to disease resistance (Ayyaz et al., 2025).

5.2 Transcription factors (WRKY, MYB, NAC) involved in defense signaling

Transcription factors are the core "switches" that regulate the plant defense system. When cotton is infected with pathogens, transcription factors from the WRKY, MYB and NAC families are rapidly activated. They can regulate many downstream defense genes and signal pathways. For example, they control the expression of pathogenesis-related proteins, hormone signaling elements, and some secondary metabolism-related genes. These regulations can enhance cotton's ability to respond to pathogens (Zhang et al., 2025). These transcription factors work together to help cotton achieve a balance between growth and defense. A typical example is *GhSTR1*, which plays an important role in regulating this balance (Cheng et al., 2025).

5.3 Secondary metabolites and antimicrobial peptides

In addition to genes and transcription factors, some secondary metabolites and antimicrobial peptides are also important. They can directly inhibit the growth of pathogens and make cotton cell walls stronger. Proteins like PGIP (also called polygalacturonase inhibitory protein), such as GhPGIP1, can specifically inhibit the enzymes used by pathogens to break down plant cell walls. If this protein is expressed more in cotton, it can improve cotton's resistance to Verticillium wilt and Fusarium wilt (Liu et al., 2017). There are also germ-like proteins (GLPs), such as GhGLP2, which have the function of superoxide dismutase and can inhibit spore germination. It can also promote callose deposition and lignification, making the defense of the infected site stronger and improving antioxidant capacity (Pei et al., 2020). Lipid metabolism also plays a role, such as the GhSSI2 isoform, which can regulate oleic acid levels, activate defense pathways related to or unrelated to salicylic acid, and participate in nitric oxide signaling (Mo et al., 2021). In addition, proteins such as the ABCG transporter *GhSTR1* and some proteins involved in ROS metabolism and cell wall modification are also very key molecules in cotton disease resistance.

6 Functional Genomics and Gene Editing Approaches

6.1 RNAi and CRISPR/Cas-mediated studies identifying key resistance genes

Researchers often use RNA interference (RNAi) and virus-induced gene silencing (VIGS) to study the function of disease resistance genes in cotton. By silencing some key genes, such as *GhAMT2*, *GhNDR1*, *GhMKK2*, *GhIQD1*, *GhEB1C*, *GbCRK18* and *GbCNL130*, cotton's resistance to Verticillium wilt will decrease, indicating that these genes are important in the defense process (Gao et al., 2011; Li et al., 2018). These experimental results show that "turning off" the function of specific genes can help us determine their role in disease resistance, and also lay the foundation for future gene editing using CRISPR/Cas technology.

6.2 Overexpression and knockdown studies validating gene function

In addition to gene silencing, expressing genes more (also called "overexpression") is also a common method. For example, overexpression of *GhAMT2* in *Arabidopsis*, or overexpression of *GhIQD1* and *GhEB1C* in tobacco, can enhance plant resistance to *Verticillium dahliae*. If these genes are knocked down in cotton (that is, their expression is reduced), cotton will become more susceptible to pathogens (Xu et al., 2024b). In addition, studies have found that overexpression of *GbCNL130* in *Arabidopsis* can enhance disease resistance, but if its expression is reduced in cotton, resistance will decrease (Li et al., 2021). These results show that functional verification is effective whether through overexpression or knockdown. Such resistance genes usually affect key defense processes such as hormone signaling, regulation of reactive oxygen species (ROS), and enhancement of cell walls (Xu et al., 2024a).

6.3 Use of genome-wide association studies (GWAS) and QTL mapping

Genome-wide association analysis (GWAS) and quantitative trait loci (QTL) mapping techniques are also often used to identify key genes and regions related to resistance to Verticillium wilt (Zhu and Luo, 2024). For example, GWAS found a major QTL on chromosome A01, which contains the important gene *GhAMT2*. Another major

QTL was found on chromosome A10, which contains the CG02 gene. Both genes were later experimentally verified to be related to resistance. In a larger GWAS study, scientists also found a key region on chromosome Dt11. This region contains multiple excellent alleles and gene clusters that are closely related to disease resistance, including L-type lectin domain receptor kinase (Zhang et al., 2021b). Therefore, QTL mapping and GWAS can provide reliable targets for cotton breeding and are also suitable for marker-assisted selection and genetic engineering (Li et al., 2017a). It can be said that functional genomics and gene editing tools allow us to find important genes related to disease resistance more quickly, and also provide a powerful means for breeding disease-resistant cotton varieties.

7 Crosstalk and Shared Mechanisms in Response to Verticillium and Fusarium

7.1 Common signaling pathways activated by both pathogens

When cotton is infected by *Verticillium dahliae* or *Fusarium oxysporum*, it activates several of the same signaling pathways to defend itself. A major mechanism is that cotton uses receptor kinases (LYKs) with lysozyme structures and chitin-sensing receptor kinase 1 (CERK1) to recognize chitin in the cell wall of pathogens. Chitin is an important component of the fungal cell wall. The cell wall-associated kinase *GhWAK7A* can interact with the sensing complex composed of GhCERK1-GhLYK5, promote their binding and phosphorylation, and then activate subsequent defense responses (Wang et al., 2020). In addition, some common signaling pathways are also activated, such as the MAPK kinase pathway, hormone signals such as salicylic acid (SA), jasmonic acid (JA), abscisic acid (ABA), and the regulatory system of reactive oxygen species (ROS) metabolism (Yi et al., 2023).

7.2 Shared downstream effectors and defense genes

Whether it is Verticillium or Fusarium invasion, cotton will be induced to activate some of the same defense-related genes and proteins. Such genes include NBS-LRR type resistance genes, PR (pathogenesis-related) proteins, enzymes that regulate ROS production, and factors related to strengthening the cell wall (Mo et al., 2021). For example, the fatty acid transporter *GhSTR1*, which is expressed more after infection by both pathogens, can play a balancing role between defense and growth and plays a significant role in broad-spectrum resistance. In addition, some genes that regulate phenylpropanoid metabolism and lignin synthesis are also activated at the same time. These genes help strengthen the cell wall and produce antibacterial substances (Billah et al., 2021). In particular, salicylic acid-dependent defense pathways are critical in both diseases, such as the upregulation of genes such as *GbCNL130* (Li et al., 2021).

7.3 Potential for cross-resistance breeding strategies

Some QTL regions and candidate genes that are effective against both Verticillium wilt and Fusarium wilt have been discovered, which shows that breeding through cross-resistance strategies is feasible. GWAS studies have found that there are some overlapping QTL regions related to both diseases on chromosomes c16 and c19, which are rich in NBS-LRR gene clusters (Abdelraheem et al., 2019). Genes such as *GhWAK7A* and *GhSTR1* play a role in resisting both pathogens. After verification, these genes have further proved the possibility of breeding broad-spectrum disease-resistant varieties by "stacking" such common resistance genes (Cheng et al., 2025). If these universal molecular mechanisms are rationally applied to breeding, it is expected to improve cotton's long-term resistance to multiple wilt diseases and reduce the impact of diseases on cotton yield.

8 Case Study Molecular Resistance in Xinjiang Cotton Varieties

8.1 Selection of resistant cotton cultivars and local pathogen strains

In Xinjiang, Verticillium wilt and Fusarium wilt have a great impact on cotton yield. Therefore, finding disease-resistant cotton varieties has always been one of the key tasks. Through field trials and nursery observations, researchers found that JK1775 is a disease-resistant variety, while Z8 is susceptible to Verticillium wilt (Figure 2). These two varieties are often used in related research. In terms of Fusarium wilt, disease-resistant sea island cotton varieties such as 06-146 are used to study their genetic resistance mechanisms. In addition, transgenic colored cotton with GAFP (Gastrodia antifungal protein) genes has also been tested in Xinjiang. These lines have shown resistance to Verticillium wilt and Fusarium wilt in actual planting. For example, the two lines LB5007 and ZB5020 are particularly outstanding in disease prevention (Liu et al., 2004). However, the survey

found that most cotton varieties planted in Xinjiang only have a certain degree of disease resistance, not real high resistance, which shows that we still need to develop more new disease-resistant materials (Liu et al., 2012).

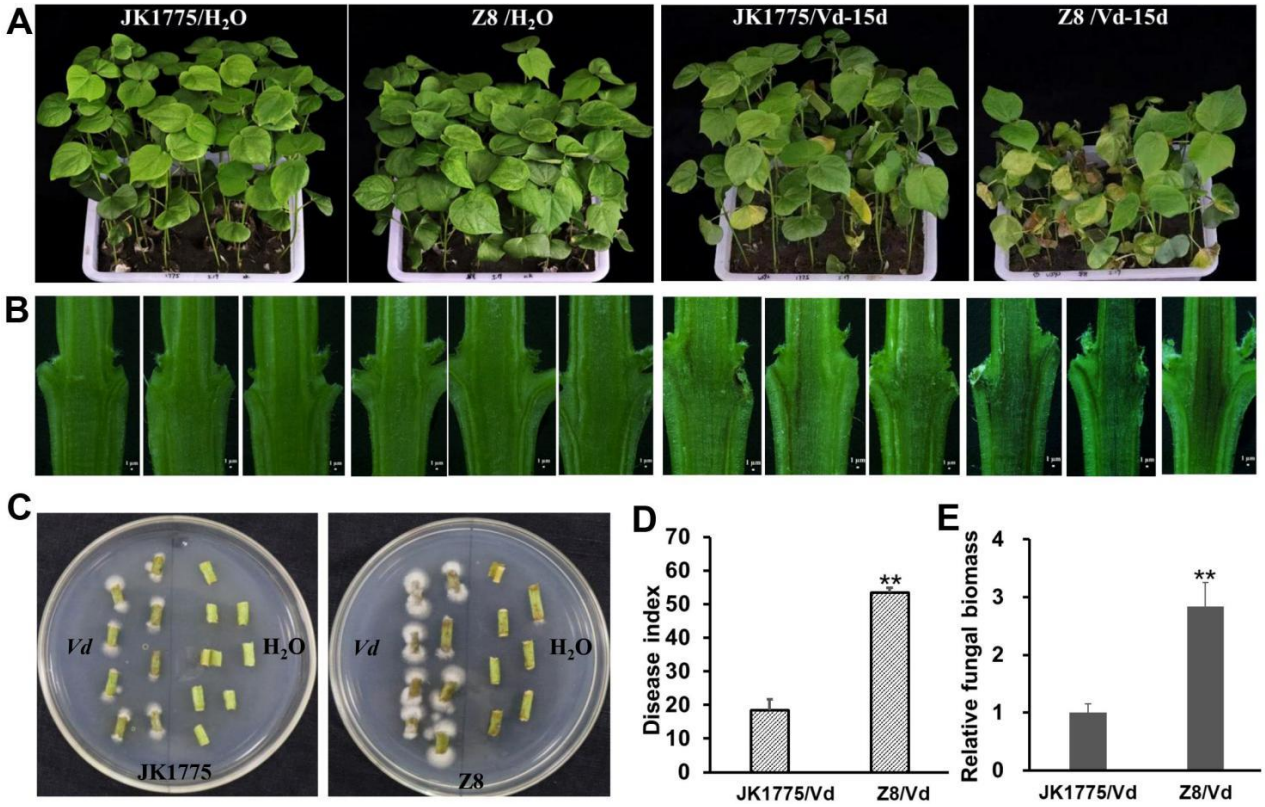

Figure 2 Different responses of JK1775 (R) and Z8 (S) to V592 at 15 dpi. (A) Disease symptoms of JK1775 and Z8 after inoculation with V592. (B) Observation of vascular bundle browning in longitudinal stem sections of JK1775 and Z8 plants cut lengthwise by hand. (C) Fungal recovery assay of the stems of JK1775 and Z8. (D) The disease indices of JK1775 and Z8. (E) Fungal biomass in the stems of JK1775 and Z8 plants was measured by qRT-PCR. Significance was determined using Duncan's multiple range test, indicated by ** $p \leq 0.01$ (Adopted from Zhang et al., 2024)

8.2 Transcriptomic profiling reveals key resistance-associated genes

Transcriptome analysis of Xinjiang cotton varieties found that disease-resistant varieties had more genes undergoing significant expression changes after infection with *Verticillium dahliae*. In contrast, susceptible varieties had fewer changes. These differentially expressed genes are mainly concentrated in the MAPK signaling pathway and phenylpropanoid metabolism, especially those that regulate lignin and coumarin synthesis, which are related to cell wall thickening and disease resistance (Zhang et al., 2024). In terms of Fusarium disease, the GbC4H gene in Sea Island cotton can regulate the synthesis of flavonoids. If this gene is silenced, cotton resistance will weaken, indicating that the accumulation of flavonoids is important for disease resistance (Qu et al., 2023). This type of response is also regulated by the methyl jasmonic acid (MeJA) and salicylic acid (SA) signaling pathways. Through GWAS and transcriptome combined analysis, the researchers found several genes related to resistance, such as *GhAMT2*, Ghir_A01G006660 and Ghir_A02G008980. These genes are involved in lignin synthesis, ROS homeostasis maintenance, and immune signaling (Ayyaz et al., 2025). Further experiments, including gene silencing and overexpression, have demonstrated that these genes play an important role in disease resistance.

8.3 Outcomes for breeding programs and regional disease management

These molecular information in Xinjiang cotton varieties provide a lot of useful genetic resources for breeding. The location of disease-resistant genes and related QTLs helps researchers better conduct marker-assisted breeding and allows them to breed more disease-resistant cotton varieties (Li et al., 2017a). In terms of breeding methods, transgenic methods have also played a positive role. For example, after the introduction of the GAFP

gene, a line that can resist both Verticillium wilt and Fusarium wilt was bred. This double-resistant cotton provides a new solution for the integrated prevention and control of cotton diseases in Xinjiang. These research results not only help solve the problem of insufficient local high-resistant varieties, but also make Xinjiang's cotton production more stable and sustainable (Liu et al., 2006).

9 Challenges and Future Directions

The "war" between cotton and Fusarium wilt pathogens continues. *Verticillium dahliae* and *Fusarium oxysporum* evolve very quickly and have many genetic changes, which brings a lot of trouble to prevention and control. These pathogens will use various methods to deal with cotton, such as interfering with the plant's immune response, adapting to poor soil environments, and secreting toxins to harm plants. These mechanisms make it difficult for cotton to maintain long-term disease resistance. There is a complex interaction between the virulence factors of pathogens and the defense system of cotton. Links such as ROS (reactive oxygen species) homeostasis, hormone signaling, and immune receptor activation have not been fully understood. This also makes disease management more challenging.

Although we have achieved some results in gene mapping, QTL (quantitative trait loci) discovery, and molecular marker-assisted breeding, cotton itself does not have enough disease resistance gene resources, and many resistance genes are not strong enough. This limits the effectiveness of current molecular breeding. Most of the disease resistance genes that have been discovered have only moderate resistance effects, and these resistances are often controlled by multiple genes and are also affected by the environment, which makes breeding more complicated. When introducing resistance genes into superior varieties, there may also be a problem of "linkage drag", that is, when introducing resistance, undesirable agronomic traits are brought in together, which will affect yield or other traits. In addition, we do not have a deep enough understanding of the entire network that regulates these resistance responses, and there is a lack of highly resistant germplasm resources. These problems also limit the further advancement of research.

Future research needs to combine multiple "omics", such as genomics, transcriptomics, proteomics and metabolomics, so that we can have a clearer understanding of how resistance is regulated. Recently, some studies have used methods such as GWAS (genome-wide association analysis), TWAS (transcriptome-wide association study) and eQTL (expression quantitative trait loci) to find key gene modules and regulatory hotspots related to ROS homeostasis and immune response. These results provide new directions for precision breeding. In addition, systems biology and high-throughput phenotyping techniques (such as disease identification combined with machine learning) may also accelerate the discovery of resistance genes and help us build better disease resistance prediction models. Combining these approaches is expected to improve breeding efficiency and accuracy, and ultimately select new cotton varieties with durable and broad-spectrum resistance to both Verticillium wilt and Fusarium wilt.

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Conflict of Interest Disclosure

The authors affirm that this research was conducted without any commercial or financial relationships that could be construed as a potential conflict of interest.

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