Cloning of Genes Underlying Quantitative Resistance for Plant Disease Control

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Abstract

Plant diseases pose a significant threat to global food security and sustainability, leading to substantial yield losses in various crop species. Plants employ two primary defense mechanisms to combat pathogen invasion: qualitative resistance, mediated by disease resistance (R) genes, and quantitative resistance, governed by multiple genes known as quantitative disease resistance (QDR) genes. Enhancing crop resistance to pathogens through conventional breeding, marker-assisted breeding (MAB), and transgenic development offers promising strategies to manage disease incidence and minimize yield losses. The identification of genes responsible for both qualitative and quantitative disease resistance plays a crucial role in developing effective disease management strategies.Qualitative resistance is often conferred by major resistance genes encoding cytoplasmic proteins with nucleotide-binding and leucine-rich repeat domains (NLR proteins), which detect pathogen-derived molecules called effectors. Activation of NLR protein-mediated defense responses includes a hypertensive response, rapid localized programmed cell death, ion flux, oxidative burst, lipid peroxidation, and reinforcement of cell walls. On the other hand, quantitative resistance is controlled by multiple quantitative trait loci (QTLs)/genes, providing broad-spectrum resistance that is typically more durable than race-specific resistance. Recent advancements in gene identification have facilitated the successful cloning and validation of genes responsible for quantitative disease resistance in various crop plants. Understanding the plant immune system is crucial, as it involves pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI) and effector-triggered immunity (ETI). PTI is characterized by the activation of mitogen-activated protein kinases (MAPKs), induction of reactive oxygen species (ROS), callose deposition, and upregulation of pathogenesis-related (PR) genes. Pathogens counteract PTI by delivering effector molecules into plant cells to suppress the host response and create a favorable environment for their survival. ETI, triggered by resistance (R) genes, provides strong resistance against specific pathogens but is often limited in durability due to rapid pathogen evolution. Non-host resistance (NHR) offers a more durable form of resistance and contributes to quantitative resistance. Combining known pattern recognition receptors (PRRs) and NLR R genes in cultivars holds promise for enhancing resistance. Overall, improving crop disease resistance is crucial for ensuring global food security, and a comprehensive understanding of the mechanisms underlying qualitative and quantitative resistance is essential for developing effective strategies.

1 Introduction

Plant diseases significantly diminish crop yields across various species, representing a substantial threat to global food security and sustainability. Plants have evolved defenses against pathogen invasions through two primary strategies: qualitative resistance, also known as vertical or complete resistance, which is mediated by specific disease resistance (R) genes, and quantitative resistance, or horizontal or partial resistance, which is controlled by a multitude of quantitative disease resistance (QDR) genes. Enhancing crop resistance to pathogens through traditional breeding, marker-assisted breeding (MAB), and transgenic development offers a promising approach to manage disease occurrence and reduce yield losses. To optimize this approach, it's essential to identify the genes responsible for both qualitative and quantitative disease resistance. [1]. Qualitative or complete resistance is usually provided by major resistance genes that encode cytoplasmic proteins containing nucleotide-binding and leucine-rich repeat domains (NLR proteins). These NLR proteins are capable of directly or indirectly detecting pathogen-derived molecules, also known as effectors, which are introduced into the host cell by the pathogen to facilitate infection. [2]. After recognizing the presence of effectors, an immune response mediated by NLR proteins is activated, which commonly involves a hypertensive response (HR). This response entails rapid, localized programmed cell death at the site of pathogen penetration, along with other defensive reactions such as ion flux, an oxidative burst, lipid peroxidation, and reinforcement of the cell wall. [3]. Quantitative disease resistance (QDR) is governed by multiple quantitative trait loci (QTLs) or genes, which interact with each other and the environment. Unlike major resistance genes (R genes) that confer qualitative resistance, QDR mediated by QTLs typically exhibits smaller individual effects but provides broad-spectrum or non-race-specific resistance. This makes it a promising alternative to less durable race-specific resistance for crop improvement. However, the mechanisms underlying QDR are more diverse than those responsible for qualitative disease resistance. While numerous race-specific R genes have been utilized by breeders, their limited durability is attributed to rapid pathogen evolution. In contrast, non-race-specific genes generally offer broad-spectrum resistance and are more effective at the adult plant stages, providing partial and usually more durable resistance than race-specific genes. The durability of QDR is influenced by various factors, including the biology, genetics, and evolution of the relevant pathogen. The limitations of gene-for-gene resistance traits have led to the exploration of genes underlying quantitative resistance in plants. Notably, in the last decade, a few genes responsible for QDR to various pathogens have been successfully cloned and validated in different crop plants.The insights from the search results indicate that QDR has diverse biological and molecular bases, as revealed by the cloning of QRLs and the identification of candidate genes underlying QRLs. Mapping QTLs is a powerful tool for genetic dissection of QDR, and DNA markers tightly linked to QRLs controlling QDR can be used for marker-assisted selection (MAS) to incorporate these valuable traits. Additionally, recent work has shown that the cumulative effects of pyramiding many QDR loci can result in high levels of resistance. Phenotypically, QDR exhibits a continuous distribution of resistance values that do not fit Mendelian segregation ratios, in contrast to a qualitative trait. This continuous distribution makes quantitative traits more difficult to study than qualitative traits, as they are influenced by multiple genes and environmental factors.The identification and understanding of QTLs governing QDR offer promising avenues for crop improvement and sustainable disease management. The ongoing research into the genetic basis of QDR and the successful cloning of QDR-related genes in various crop plants signify significant progress in this field. Further exploration of QDR mechanisms and the genetic

interactions underlying QTLs will contribute to the development of more resilient and disease-resistant crop varieties..[4].

2 Plant Immune Systems at a Glance

Plants possess two main innate immune responses to counteract pathogens: pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI), and effector-triggered immunity (ETI). The PTI response involves multiple processes, including the activation of mitogen-activated protein kinases (MAPKs), the production of reactive oxygen species (ROS), the deposition of a carbohydrate called callose, and the stimulation of pathogenesis-related (PR) genes. One of the early defense mechanisms against pathogen attack is a burst in ROS levels, which strengthens cell walls by cross-linking glycoproteins and prompts the activation of defense-signaling components. On the other hand, pathogens attempt to circumvent these defenses by releasing effector molecules into the plant cells, aiming to suppress the host's PTI response and create an environment within the host cell that is conducive to their survival and proliferation. [5].Plants have developed intercellular sensors encoded by resistance (R) genes containing a nucleotide binding and leucine-rich repeats (NBS-LRRs), which perceive pathogen effectors directly indirectly, leading to ETI. ETI confers strong resistance against particular pathogens, especially for a particular race, and elicit a hypersensitive response however, this is not durable, because of the rapid evolution of pathogen effector PTI is an important factor in nonhost resistance-the phenomenon whereby ants are resistant to most microbial pathogens and this contributes to quantitate e resistance [6].

3 Model Explaining Quantitative Disease Resistance

Plant defense responses include plant preformed physical or chemical barriers (e.g., rigid cell walls, presence of cuticles or trichomes, production of toxic or repellent compounds) and immune signaling responses (Table 1). The immune signaling-mediated resistance mechanism corresponds to a zig-zag model (a two-level defense system).

Table 1. Cloned quantitative ease resistance (QDR) genes and their molecular mechanisms against different host-pathogen systems.

S.No	Mechanism	Gen	Crop	Pathogen	References
	Pathogen recognition	Pbl	Rice	Rice blast; Magnaporthe oryzae	[7, 8]
		OsWAk14, OsWAK91, Os WAK92 and OsWAK112d	Rice	Rice blast; Magnaporthe oryzae	[8]
		RCGI	Maize	Anthracnose stalk rot; Colletotrichum graminicola	[9]
		Pi35	Rice	Rice blast; Magnaporthe oryzae	[10]

		RRSI, RPS4	Arabidopsis	Black rot; Xanthomonas campestris pv. campestris race 2	[11]
		ZmWAK	Maize	Head smut; Sporisorium reilianum	[12]
		Htnl	Maize	Northern leaf blight; Exserohilum turcicum	[13]
		RFO3	Arabidopsis	Wilt; Fusarium oxysporum f. sp. matthioli	[14]
	Transcription response	C3H12	Rice	Bacterial blight; Xanthomonas oryzae pv. oryzae	[15]
		OsWRKY13	Rice	Blight; anthomonas oryzae pv. oryzicola and lagr porthe grisea	[16]
	Single transd	OsMPK6	Rice	Blight rot; Xanthomonas oryzae pv . oryzicola and Magnaporthe grisea	[17]
		RKSI	Arabidopsis	Black root Xanthomonas campestris pv . compestris	[17]
		Yr36(WKSI)	Wheat	Stripe rust puccinia striiformis	[18]
		Panl	Maize	Northern leaf blight and stewarts wilt Exserohilum turcicun and Pantoea stewartii	[19]
	Defense response	Pi2I	Rice	Rice blast; magnaporthe oryzae	[20]
		Lr36	Wheat	Leaf rust, stripe rust, and pu wdery mildew; Puccinia triticina, Puccinia striifor s, and Blumeria graminis	[21]

Table 1. (Continued)

S.No	Mechanism	Gen	Crop	Pathogen	References
5.	Antimicrobial activity	Fhb	Wheat	*Fusarium head blight; Fusarium graminearum*	[22]
		OsPALA	Rice	*Bacterial blight, sheath blight, and blast;*	[23]

		OsGLP	Rice	*Xanthomonas oryzae pv. oryzae, Rhizoctonia solani, and Magnaporthe oryzae*	[24]
6.	Nutritional restriction	OsDR8	Rice	*Sheath blight and blast; Rhizoctonia solani andMagnaporthe oryzae*	[17]
		Lr67	Wheat	*Blight and blast; Xanthomonas oryzae pv. oryzicola and Magnaporthe oryzae*	[25]
		Rhg4	Soyabean	Leaf rust, stripe rust, stem rust, and powdery mildew; *Puccinia triticina, Puccinia striformis f.*	[26]
		Rhg1	soyabean	soyabean cyst;*Heterodera glycines Ichinohe*	[20]
7.	Microbial movement Hormones	ZmREM6.2	Maize	Cyst;*Heterodera glycines Ichinohe*	[27]
		ZmCCoAOMT2	Maize	Northern leaf blight; *Exserohilum turcicum*	[28]
8.	Hormones	GH3-2	Rice	Southern leaf blight; gray leaf spot, and northern leaf blight and *Exserohilum turcicum*	[28]
		GH3-8	Rice	Blight and blast ;Xanthomonas *Magnaporthe oryzae*	
9.	Others	Camalexin	Arabidopsis	*Plasmodiophora brassicae*	[25]
		POQR	Arabidopsis	*Sclerotinia sclerotiorum*	[29]

4.1 Quantitative Disease Resistance Genes in Arabidopsis

The Arabidopsis thaliana locus RESISTANCE TO POWDERY MILDEW8 (RPW8) contains two naturally polymorphic, dominant R genes—*RPW8.1* and *RPW8.2* which individually control resistance to a broad range of powdery mildew pathogens. The predicted RPW8.1 and RPW8.2 proteins are different from the previously characterized R proteins (NBS-LRR proteins); they induce localized, salicylic acid (SA)-dependent defenses similar to those induced by R genes that control specific resistance RPW8.2 is induced and specifically targeted to the extrahaustorial membrane (EHM), an enigmatic interfacial membrane believed to be derived from the host cell plasma membrane. There, *RPW8.2* anti vates an SA signaling-dependent defense strategy, which concomitantly enhances the encasement of the haustorial complex and on-site accumulation. of H202, presumably for constraining the haustorium while reducing oxidative jamage to the host cell, thus leading to broad-spectrum resistance again diverse races of powdery mildew. Natural mutations that impair either defuse aviation or EHM targeting of RPW8.2 compromise the efficacy of POW82-mediated resistance [30]. Heterologous expression of PAMP recognition systems, such as the Arabidopsis receptor kinase EFR, holds the potential to engineer broad-spectrum disease resistance to significant bacterial pathogens. In Arabidopsis, the receptor kinase FLS2 plays a crucial role in recognizing bacterial flagellin

(fg22) and activating a robust defense response. The specific interaction of the elicitor-active epitope flg22 with the FLS2 protein has been demonstrated, and the functionality of this receptor has been confirmed through heterologous expression of the Arabidopsis FLS2 gene in tomato cells. This expression conferred an additional flg22-perception system on the tomato cells, exhibiting all the properties characteristic of the perception of this elicitor in Arabidopsis.

In the context of rice, the heterologous expression of the dicotyledonous PRR efr leads to ligand-dependent activation of defense responses. Furthermore, rice plants expressing EFR or the chimeric receptor EFR::XA21, which contains the EFR ectodomain and the XA21 intracellular domain, have been shown to sense both Escherichia coli-derived and Xanthomonas oryzae pv. oryzga (s 2)-derived elfl8 peptides at subnanomolar concentrations. Treatment of BER and EFR::XA21 transgenic rice leaf tissue with elf18 leads to MAPK 2 activation, ROS production, and defense gene expression。The insights from the research sources indicate the significant potential of heterologous expression of PAMP recognition systems in conferring broad-spectrum disease resistance. The specific interactions and activation of defense responses in different plant systems underscore the versatility and applicability of this approach in enhancing plant immunity against bacterial pathogens.The ongoing research in this field, as evidenced by the successful cloning and validation of genes responsible for QDR to various pathogens in different crop plants, signifies substantial progress. This progress holds promise for the development of more resilient and disease-resistant crop varieties, contributing to global food security and sustainable agriculture. [31].

4.2 Quantitative «isease Resistance Genes in Rice

Although large numbers of QTLs for bacterial blight and blast resistance have been identified, very few have been cloned on the basis of a map-based cloning approach. All basic resistance genes (called *Pi*) encode NBS-LRR proteins, except for the *Pid2* and genes. *Pid2* encodes a b-lectin receptor-like kinase[32], and *Pid2* encodes a proline-rich protein containing a metal-binding domain. Though Pid2 confers non-race-specific, durable resistance[33], it does not affect the yield or grain quality, making pi21 a good candidate for marker-assisted selection (MAS), but unfortunately its application is limited by the close linkage of *pi21* to a gene (LOC_Os04g32890) that causes inferior grain quality [33]cloned *Pi35* through map-based cloning and identified multiple functional polymorphisms that allow effective control of the disease. Pi35 is allelic to Pish, which mediates race-specific resistance to blast and encodes a protein containing a NBS-LRR domain. Multiple functional polymorphisms cumulatively enhance resistance, and an amino acid residue in an LRR of Pi35 is strongly associated with the gene's mediation of quantitative but consistent broad-spectrum resistance to pathogen isolates in Japan, in contrast to Pish, which mediates resistance to only a single isolate. The rice *Xa21* gene confers broad and persistent resistance against X. oryzae pv. oryzae, which was isolated by positional cloning. The protein of this gene carries both a leucine-rich repeat motif and a serine-threonine kinase-like domain, which suggests a role in cell surface recognition of a pathogen ligand and subsequent activation of an intracellular defense response by phosphorylation of downstream genes.Through phosphorylation and cleavage of its intracellular kinase domain, Xa21 perceives the presence of Xoo and relays the signal to the nucleus through multistep signal cascades involving so he key proteins such as XA21 binding protein 3 (XB3), MAK5, MAPK12, ana an-scription factors (TFs) including OsWRKY62 and OsWRKY76 in the nucleus [34].

Indole-3-acetic acid (IAA), the major form of auxin in rice, helps invaders into plant cells by IAA-induced loosening of the cell wall, a natural protective barrier of plant cells against invaders. *X. oryzae pv. oryzae, X. oryzae pv. oryzicola, and M. grisea* secrete IAA, in turn inducing rice to synthesize its own IAA at the infection site. Then IAA induces the production of expansins (cell wall-loosening pro-teins) and makes rice vulnerable to pathogen entry and colonization. GH3-2, a minor resistance QTL, has been shown to be associated with variation in quantitative especially binds to messenger RNAs (mRNAs) of multiple OsPAL (*OsPALI-7)* genes and suppresses OPALI mRNA accumulation, thereby promoting OPAL turn-over. Loss of function of the Bsr-k1 gene leads to accumulation of *OsPALI-7* mRNAs in the bsr-k1 mutant and confers enhanced resistance against diverse races of M. ory-zae and Xoo. Furthermore, overexpression of OSPALI in wild-type rice TP309 confers resistance to M. oryzae, supporting the role of OsPALI in disease resistance [35] mapped the rice Pigm locus, which contains a cluster of 13 genes, including three genes encoding NLR receptors (R4, R6, and R8) that confer durable resistance to the fungus M. oryzae without a yield penalty. Among these NLR receptors, *Pigm*R (Pigm R6) confers broad-spectrum resistance agan, ta worldwide collection of M. oryzae isolates, whereas PigmS (PigmR8) attenuates *Pigm*R homodimerization to suppress resistance. The increa o expression of PigmS suppresses *Pigm*R-mediated resistance. PigmR war costively expressed at a low level in all tissues, whereas *Pigm*S was highly expressed in pollen and panicles, with only trace expression in other organs. The ep rej etic regulation of *Pigm*S fine-tunes disease resistance and the trade-off between defense and yield by high expression of *Pigm*S in pollen, which might facilitate It recitation through an unrecognized mechanism [36].

A germin-like protein gene (OsGLP) family member governs broad-spectrum disease resistance in rice (blast and sheath blight disease) and barley (powdery mildew)On chromosome 8 of rice, a cluster of 12 germin-like protein (OsGLP) gene members exhibited resistance to rice blast disease and, of the 12 OsGLPs, one clustered subfamily (OsGER4), identified by a RNA interference (RNAi) approach, contributed most to blast and sheath blight disease resistance [35] A *qBlsSa* QTL mapped on chromosome 5 confers resistance to bacterial leaf streak. [37]narrowed down the QTL and identified a gene (LOC_ Os05g01710) that encodes the gamma chain of transcription initiation factor IIA (TFIlAc), which has a nucleotide variation that cause amino acid change and further disease reaction to pathogens. The nucleotide substitutions resulted in a change of the 39th amino acid from valine (in the susceptible parent) to glutamic acid (in the resistant parent). OsPALA, a member of the phenylalanine ammonia lyase gene family located on rice chromosome 2, confers bacterial blight and sheath blight disease resis-tance. Mutation of OsPALA increased expression of the OsPAL2 gene and decreased expression of the unlinked OsPAL6 gene [38].

4.3 Quantitative Disease Resistance Genes in Wheat and Barley

Several wheat genes, including Lr34, Lr46 (not yet cloned), and Lr67, have been identified to provide partial resistance to various fungal pathogens such as leaf rust (Puccinia triticina), stripe rust (Puccinia striiformis f. sp. tritici), stem rust (Puccinia graminis f. sp. tritici), and powdery mildew (Blumeria graminis f. sp. tritici) in adult wheat plants. Among these genes, Lr34 is particularly notable for conferring durable resistance to rusts and powdery mildew disease, with a proven track record of over 100 years.

Lr34 is expressed in adult plants during the critical grain-filling stage and is most effective in the flag leaf, where it stimulates senescence-like processes in the leaf tips and edges. Initially mapped on the short arm of chromosome 7D between the markers gwm1220 and SWM10, the

Lr34 gene codes for a protein that resembles adenosine triphosphate-binding cassette transporters, specifically a putative ABC transporter. This unique protein structure is associated with its ability to confer durable resistance to multiple fungal pathogens in wheat.
The alleles of Lr34 that confer resistance or susceptibility differ by three genetic polymorphisms. One single-nucleotide polymorphism is located in the large intron 4, while the other two sequence differences are located in exons. The deletion of three base pairs (ttc) in exon 11 results in the deletion of a phenylalanine residue, and a second single-nucleotide polymorphism in exon 12 converts a tyrosine to a histidine in the resistant cultivar. Both sequence differences located in exons affect the first transmembrane domain connecting the two nucleotide-binding domains, potentially altering the structure and substrate specificity of the transporter.
The understanding of these durable resistance genes, particularly Lr34, provides valuable insights into the molecular mechanisms governing plant-fungal interactions in wheat. This knowledge is essential for the development of improved wheat varieties with enhanced resistance to multiple fungal pathogens, contributing to sustainable and resilient agricultural practices.
[39]

Similarly, *Lr67* confers quantitative resistance to rust diseases, including powdery mildew, and encodes a predicted hexose transporter. The resistant form (LR67res) differs from the susceptible form of the same protein (LR67res) by two amino acids, which are conserved in orthologous hexose transporter. LR67res may cause reduced hexose transport through a dominant-negative interference mechanism by forming inactive heteromultimerion protein complexes. The partial resistance conferred by LR67res to different bio trophic pathogens of wheat and barley could be due to the blocking of apoplectic retrieval by host cells, thereby increasing the hexose-to-sucrose ratio in the leaf apoplasm, which in turn induces a sugar-mediated signaling response these is in a more hostile environment for pathogen growth. The LR67res inhibition of hexose retrieval may mimic the ubiquitous plant response to patho an invasion of elevated cell wall invertase activity, which alters the extraco. lar apoplastic hexose-to-sucrose ratio and elicits a hexose-mediated defense response[39]. The phosphorylation of thylakoid-associated ascorbate peroxidase (tAPX) by WKS1.1 reduces the ability of the cells to detoxify ROS and contributes to cell death. This response takes several days longer than typical hypersensitive cell death responses, thus allowing the limited pathogen growth and restricted sporulation that is characteristic of the WKS1 partial resistance response to stripe rust [40].

Wheat powdery mildew is caused by B. graminis f. sp. tritici (Bgt). The *Pm21* gene, originating from Dasypyrum villosum, confers high resistance to all known Bgt races. Recently, the Pm21 gene was cloned by following integrated approaches 2 RETRACTED CHAPTER: Cloning of Genes Underlying Quantitative Resistance 35of resistance gene analog (RGA)-based cloning via comparative genomics, physical and genetic mapping, barley stripe mosaic virus-induced gene silencing (BSMV-VIGS), large-scale mutagenesis, and genetic transformation. *Pm21* encodes a typical CC-NBS-LRR protein and confers broad-spectrum resistance to wheat powdery mildew.Natural and induced loss-of-function mutations of the Mildew resistance locus o (Mlo) gene confer broad-spectrum resistance against most B. graminis f. sp. hordei (Bgh) isolates in barley. Mlo is a member of an ancient eukaryotic gene family that is conserved throughout the plant kingdom [41].

4.4 Quantitative Disease Resistance Genes in Maize

A QTL (qNLB1.02B73) on the short arm of chromosome 1, conditioning resistance to northern leaf blight (NLB), has been identified as a pleiotropic locus in maize.This locus confers resistance not only to NLB (caused by the fungus Setosphaeria turcica) but also to Stewart's wilt (caused by the bacterium Pantoea stewartia) and common rust (caused by the fungus Puccinia sorghi). A maize remorin (ZmREM6.3)—a chaperonin gene present in this QTL interval, reported to be involved in quantitative resistance against NLB-has been identified following high-resolution fine-mapping, expression analysis, and mutants in maize. Expression of ZmREM6.3 was higher in the resistant line and was downregulated upon infection with an S. turcica race 1 isolate in both the susceptible and resistant near-isogenic lines (NILs). The downregulation of ZmREM6.3 may indicate that it is an important part of the defense response and thus is targeted by the pathogen (Jamann et al. 2016). A quantitative trait locus, qMdr9.02 on chromosome 9 of maize, is associated with resistance to three important foliar maize diseases: southern leaf blight (SLB), gray leaf spot, and NLB. These QTLs were further narrow down to the gene level through fine-mapping, association analysis,expression analysis insertional mutagenesis, and a transgenic approach, and identified A gene, ZmCCoAOMT2, which encodes a caffeoyl-CoA O-methyltransferase. Thigene is associated with the phenylpropanoid pathway and lignin production, and confers quantitative resistance to both SLB and gray leaf spot. The resistance is governed by allelic variation at the level of both gene expression and the am.. acid sequence, thus resulting in differences in levels of lignin and other metalizes of the phenyl-propionic pathway, and regulation of programmed cerdeath [42] The resistance to head smut, a systemic disease in maize caused by the soil-borne fungus Sporisorium reilianum, is controlled by the quantitative resistance locus qHSR1, which contains the ZmWAK gene. ZmWAK spans the plasma membrane, potentially functioning as a receptor-like kinase to perceive and transduce extracellular signals. It is highly expressed in the mesocotyl of seedlings, where it halts the biotrophic growth of the endophytic S. reilianum. The resistance mediated by ZmWAK primarily occurs in the mesocotyl of maize seedlings, rather than in the ear or tassel, where typical symptoms occur. Impaired expression in the mesocotyl compromises ZmWAK-mediated resistance.The employment of sequence repeat markers and single-nucleotide polymorphism markers not only aids in identifying closely linked markers for the trait of interest but also facilitates the narrowing down of the QTL region. This, in turn, supports the map-based cloning of genes associated with the resistance to head smut in maize.The research on ZmWAK and its role in conferring resistance to head smut in maize provides valuable insights into the molecular mechanisms underlying plant-fungal interactions. This knowledge is crucial for the development of improved maize varieties with enhanced resistance to head smut, contributing to sustainable and resilient agricultural practices. [12].

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