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CRISPR/Cas-Mediated Genome Editing: Advancing Plant Disease Resistance through Promoter Precision

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Abstract. Infections with pathogens directly slow down the growth of crop biomass and the overall yield. Linkage drag is an issue for traditional breeding methods, while transgenic approaches raise biosafety problems because they use DNA from other organisms. The CRISPR/Cas system provides a more precise method for engineering by facilitating the specific modification of endogenous cis-regulatory regions. This review focuses on precise editing methods that are focused on promoter regions. In real experiments, changing coding sequences (CDS) directly can mess up the important jobs that proteins do in the body. On the other hand, fine-tuning promoter sequences can help the immune system and plant growth at the same time. They usually call this the growth-defense trade-off. We initially examined the principal enzymatic mechanisms involved in Cas9-mediated non-homologous end joining (NHEJ), Base Editors (BEs), and Prime Editors (PEs). After that, split the main goals that the labs are looking at into three groups. The investigation encompasses NLR receptor proteins, hormone-signaling hubs such as SlJAZ2, and susceptibility (S) genes that viruses exploit, including the SWEET family. We look at the different effects of breaking effector-binding elements (EBEs) or transcriptional repressor motifs by using phenotypic data from rice, tomato, and cassava mutants. The phenotypic results show that these mutants can give broad-spectrum resistance while keeping most of their agronomic traits with little loss. Finally, we talk about the technical problems we have right now and suggest ways to improve them, such as using AI to help design sgRNAs and new chimeric immune receptors. In short, targeted promoter mutation is a good way to make new materials that are resistant to disease without having to include genes from other organisms.

Keywords: Genome editing, CRISPR/Cas9, Promoter regulation, Susceptibility genes, Bioengineering, Crop protection

1. Introduction

During field cultivation, crops are highly susceptible to infection by pathogenic fungi, bacteria, and viruses, which undergo rapid regional adaptation to overcome plant defenses [1], severely disrupt normal physiological metabolism [2]. To combat this, conventional resistance backcrossing has been utilized for decades; however, it struggles to completely eliminate the negative effects of linkage drag. On the other hand, *Agrobacterium*-mediated transgenic approaches inevitably introduce selectable marker genes, making subsequent biosafety regulatory reviews highly

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burdensome [3]. Consequently, the field of agricultural bioengineering urgently requires a more targeted molecular operating tool that leaves no foreign DNA footprints [4].

The *Streptococcus pyogenes* CRISPR-Cas9 system has now been repurposed as a powerful genome-targeted endonuclease [5]. In practice, the Cas9 protein, guided by a sgRNA, creates a double-strand break (DSB) at the target site [6]. To repair these broken ends, host cells typically initiate the non-homologous end joining (NHEJ) pathway, generating random insertions or deletions (indels) [7]. While these frameshift mutations lead to direct gene knockout, the highly heterogeneous nature of the repair products makes it extremely difficult to use this mechanism for the fine-tuned, gradient regulation of gene transcript abundance [8].

To address this limitation, Base Editors (BEs) use the process of DNA replication to make single-base changes (C-G to T-A or A-T to G-C) without making standard DSBs [9, 10]. Prime Editors (PEs) are more difficult since they use a Cas9 nickase and a reverse transcriptase. With this new technology, researchers can avoid the generation of double-strand breaks (DSBs), and accurately introduce donor DNA into recombinant sites constructed by site-specific recombinase (SSRs), and at the same time realize accurate base modification and addition or elimination of small fragments of gene sequence [11, 12].

At present, researchers no longer simply terminate the coding sequence, but use this new technology to finely optimize the promoter, which belongs to a more rigorous research idea [13]. Site-directed mutagenesis of promoters can not only accurately regulate the transcription level of endogenous genes, but also ensure the integrity of translation products. This paper will sort out the basic principles of promoter editing, clarify the key immune targets, and promote the development of this innovative technology with specific cases.

2. Main CRISPR tools for promoter editing

The DNA sequence of the promoter directly determines the binding efficiency of transcription factors with it. At present, researchers mainly solve this problem through three types of CRISPR technology as showed in Table 1 and Figure 1 [14].

2.1. Cas9-mediated non-homologous end joining for mutations

Intracellular nonhomologous end joining (NHEJ) repair mechanism will slightly modify DNA by adding or subtracting a few bases after Cas9 cleaves DNA. If this insertion-deletion mutation happens in the transcription factor binding site, it will change the spatial structure of the target DNA in this site [15], resulting in the failure of the activation protein to normally bind to the promoter. Researchers usually use two single-directional RNAs (sgRNAs) to target large enhancer regions at the same time, but this double-cutting method is unstable and can easily lead to large-scale chromosome translocation [16].

2.2. Base editors for point mutations

The base editor (BEs) does not need to cut DNA, but converts one DNA base into another through chemical reaction, which does not produce double-strand breaks (DSBs), and is safer and less harmful to plant cells [14, 17]. Two types of base editors are mainly used in this experiment: cytosine base editor (CBEs) can convert C-G base pairs into T-A base pairs [18]; adenine base editor (ABEs) can realize the transformation from A-T base pairs to G-C base pairs, and complete the modification of other sites of the genetic code. ABE technology, represented by the modified TadA-

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8e, has been optimized recently, showing better editing performance and efficiency in the AT-rich environment [19].

Table 1. Representative applications of CRISPR-mediated promoter editing

Crop	Target Gene	Target element	Pathogen / Disease	Editing Strategy	Key outcome	Ref.
Rice (*O. sativa*)	*OsSWEET 11a*, *13*, *14*	Promoter EBEs	*Xanthomonas oryzae* pv. *oryzae* (Bacterial Blight)	CRISPR-Cas9 (NHEJ-mediated disruption)	Broad-spectrum resistance; No yield penalty; Transgene-free lines generated.	[20, 21]
	Bsr-d1	Promoter (*cis*-element repression)	*Magnaporthe oryzae* (Rice Blast)	CRISPR-Cas9 (Targeted knockout mimicking natural allele)	Enhanced blast resistance; Increased expression of peroxidase; No yield penalty.	[22]
	OsXa13	Promoter	*X. oryzae* pv. *oryzae* (Bacterial Blight)	CRISPR-Cas9 (Knockout)	Significant enhancement of bacterial blight resistance.	[23, 24]
	BRG8; *HOS59*	Promoter / Negative Regulator	*M. oryzae* & *X. oryzae*	Promoter editing (Fine-tuning expression)	Balanced immunity and growth; Prevention of autoimmunity; Broad-spectrum resistance.	[25]
	RBL1	Promoter (*cis*-regulatory regions)	Multiple pathogens	Predictive Promoter Editing (Cas9/Base Editing)	Multi-pathogen resistance; Optimized expression broke the immunity-growth trade-off.	[26]
	FLR13	Promoter	*Magnaporthe oryzae* (Rice Blast)	Promoter Editing (Releasing transcriptional repression)	Enhanced blast resistance; Simultaneous increase in yield per plant.	[27]
Cassava (*M. esculenta*)	*MeSWEET 10a*	Promoter EBE (TALE20 binding site)	*Xanthomonas axonopodis* pv. *manihotis* (CBB)	CRISPR-Cas9 (EBE disruption)	Reduced disease symptoms and bacterial proliferation; Normal plant development.	[28]
Citrus (*C. sinensis*)	*CsLOB1*	Promoter EBE (PthA4 binding site)	*Xanthomonas citri* subsp. *citri* (Citrus Canker)	CRISPR-Cas9 (EBE modification/deletion)	High degree of resistance to canker; Disrupted pathogen-induced gene expression.	[29]
Tomato (*S. lycopersicum*)	*SlJAZ2*	Gene / Regulatory Domain (Jas domain)	*Pseudomonas syringae* pv. *tomato* (Bacterial Speck)	CRISPR-Cas9 (Creating dominant repressor)	Stomatal-based resistance to bacteria; Maintained resistance to necrotrophic fungi.	[30]
	SlPAL2	Promoter	*Clavibacter michiganensis* (Bacterial Canker)	CRISPRa (Transcriptional Activation)	Upregulated *PAL2* expression; Increased lignin accumulation; Enhanced resistance.	[31]

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Table 1. (continued)

Tomato (*S. lycopersicum*)	*SlPR-1*	Promoter	*Clavibacter michiganensis* (Bacterial Canker)	CRISPRa (Transcriptional Activation)	Enhanced disease resistance; No alteration of agronomic characteristics.	[32]

2.3. Prime editors for exact sequence replacements

Prime Editor (PEs) is the most applicable CRISPR editing tool at present [12]. It uses a special guide RNA named pegRNA, which has an extra sequence at the end and can be directly used as an editing template. Reverse transcriptase can read the template information and integrate the new sequence directly into the genome. Researchers can add, delete or replace specific DNA sequences without cutting off DNA double strands or inserting foreign DNA fragments [11].

In addition to the above-mentioned structural sequence editing, CRISPRa technology opens up a new path of non-invasive editing by using the catalytically inactivated dCas system, which can realize epigenetic modification and activate the promoter through transcriptional regulation [31, 32].

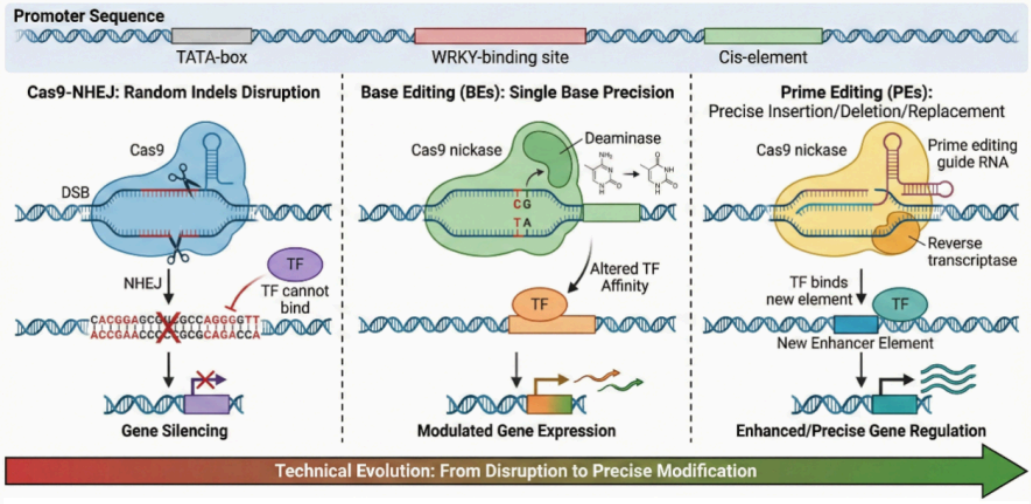


Figure 1. Diagram compares different CRISPR-based methods for editing promoters to control gene expression very precisely

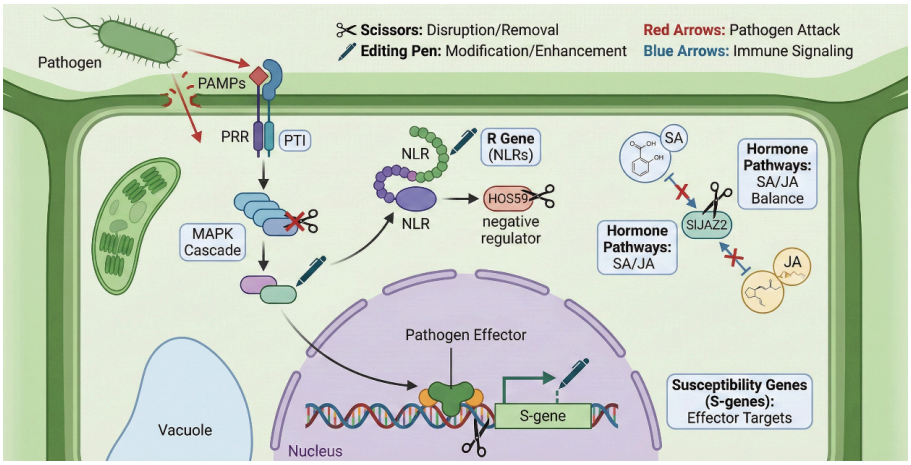


Figure 2. Strategic targeting of plant immune network switches through promoter editing

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3. Key promoter targets in plant defense networks

To build things correctly, you need to know a lot about the plant immune system, from its discovery to how it works in practice [33].

3.1. NLR immune receptor promoters

Most resistance (*R*) genes make proteins that are NLR receptors inside cells [34]. Targeted knockouts of negative regulatory features in the promoter region can greatly speed up the peak accumulation of NLR proteins during the early stages of pathogen infection [35]. In the past, this technique has worked successfully with CRISPR-induced knockout mutants of the *Prv* gene in melon [36].

3.2. Regulators of pattern-triggered immunity (PTI)

Pattern-triggered immunity (PTI) formed the tier of the plant immune system (Figure 2) [37]. It makes NLR-mediated immunity work better to build a strong defense network [38]. Through the use of editing tools to change the *cis*-regulatory sequences of key transcription factors like WRKY, including the WRKY7 positive regulator [39], engineers can artificially control the transcriptional activation of plant defensins [40].

3.3. Hormone signaling hubs

The primary hormone signaling pathways work together to coordinate essential defense mechanisms [41]. Modifying elements linked to salicylic acid (SA) signaling can enhance resistance by triggering the synthesis of lignin downstream [42]. Reducing the free protein levels of the JAZ repressor family by disrupting their promoters promptly triggers the entire JA defense cascade [43, 44].

3.4. Susceptibility (S) gene promoters

Since most *S* genes are inherently involved in the plant's normal source-sink distribution, directly knocking out their coding regions leads to stunted plant development [23]. Fine-tuning their promoters has become an operationally viable strategy to engineer disease resistance without fitness costs [24, 45, 46].

4. Applications in crop trait improvement

4.1. Mitigating growth-defense trade-offs via NLR fine-tuning

The transcription factor *HOS59* rigorously limits the protein half-life of the NLR receptor gene *BRG8* in the rice immune system. Researchers indirectly stopped the breakdown of *BRG8* by knocking out the *HOS59* promoter region using NHEJ. This stopped the cell death that usually happens when NLR is overexpressed all the time [25].

4.2. Engineering stomatal defense via phytohormone networks

Researchers used CRISPR/Cas9 to make precise changes to the *SlJAZ2* gene so that bacterial pathogens couldn't use the jasmonic acid pathway to force stomatal opening. This particularly

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inhibited the ion efflux process that responds to pathogens, but it didn't change the basal resistance at all [30].

4.3. Disrupting of effector-binding elements (ebes)

Bacterial blight pathogens use TALE domains to strongly bind to effector-binding elements (EBEs) on the promoters of *OsSWEET* sugar transporters (Figure 3) [20]. By precisely mutating the conserved bases of these EBEs, researchers rendered the pathogen's effectors completely incapable of attaching [21]. The same design method has worked well for cassava's *MeSWEET10a* [28] and the gene *CsLOB1*, which is linked to the risk of citrus canker [29].

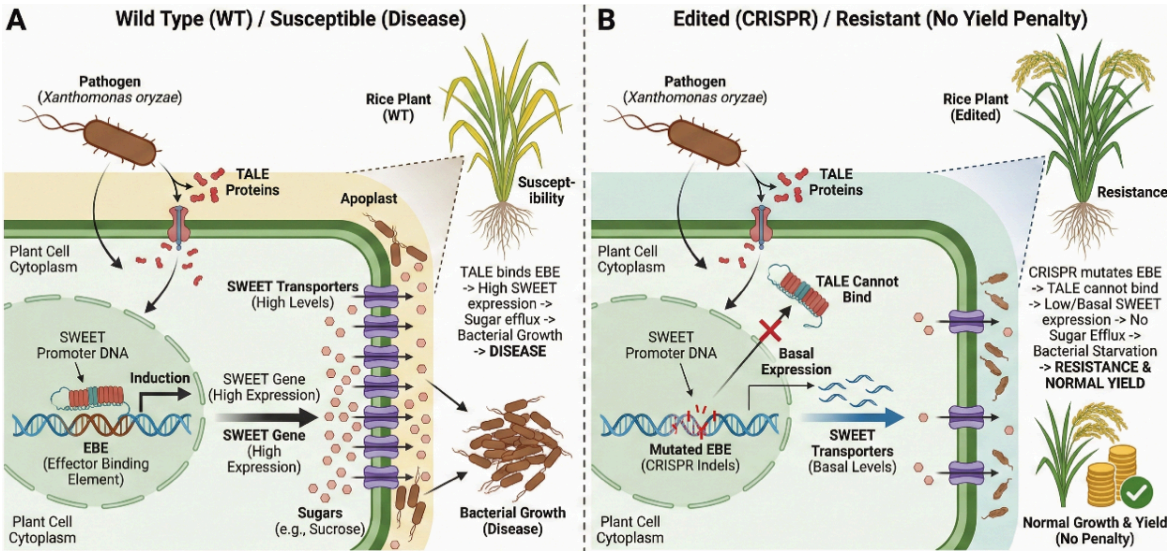


Figure 3. Diagram shows broad-spectrum resistance through changes to the susceptibility (S) gene promoters

4.4. Relieving transcriptional repression in pleiotropic genes

Rice plants' height, number of tillers, and resistance to blast are all controlled by the gene FERONIA-Like Receptor 13 (*FLR13*) [27]. When the RLK gene is overexpressed or turned off altogether, it usually leads to unfavorable developmental trade-offs. Researchers were able to employ CRISPR/Cas9 to cut out the T5T6 part of the FLR13 promoter, which was a success. It was correct that the deletion change got rid of the binding motifs, like the GAGA motif and W-box, for the transcriptional repressors *OsGBP1* and *OsWRKY53* [27, 47].

5. Challenges and perspectives

Despite its potential, promoter editing still face several practical challenges. First, off-target mitigation remain a major concern deo to that Cas9 can sometimes cut the wrong DNA sequences [48, 49]. To handle this, deep learning network models are used predict potential off-target sites before synthesizing sgRNAs, making experimental designs much safer [50-52]. Anothor bottleneck is plant tissue culture, as many important crop varieties are difficult to regenerate. This can be improved by co-expressing development-related genes (e.g., *BBM*, *WUS*) to help somatic cells to differentiated and grow into full plant more easily, bypassing genotype dependencies [53]. At the same time, engineering custom synthetic receptors, like autoactive NLRs fused with specific

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pathogen protease cleavage sites, offers a smart way to trigger rapid immune responses across species boundaries [54]. Finally, to build boader resistance, future strategies are moving toward multiplex editing and synthetic biology [22, 26].

6. Conclusion

CRISPR technology has changed how we edit *cis*-regulatory elements. Instead of knocking out complete genes, we can now make more precise changes to transcriptional regulatory areas. Variants such as *FLR13*, *SWEET*, and *SlJAZ2* have shown that it is possible to control the generation of secondary metabolites and immunological responses that are produced by plant stimulation within a certain physiological range. This improved regulation goes against the traditional idea that a solid defense slows down biomass growth. The increasing use of Prime Editors, Base Editors, and new chimeric receptor designs indicates that targeted promoter editing is poised to become a significant instrument in contemporary agricultural engineering.

References

[1] Zheng, J., et al. (2024). RXLR effector genes mediate regional adaptation of Phytophthora infestans. *Phytopathology Research, 6*(1), 63.
[2] Hamabwe, S. M., et al. (2023). Identification of quantitative trait loci for drought tolerance in Bukoba/Kijivu Andean mapping population of common bean. *Theoretical and Applied Genetics, 136*(11), 222.
[3] Entine, J., et al. (2021). Regulatory approaches for genome edited agricultural plants in select countries and jurisdictions around the world. *Transgenic Research, 30*(4), 551–584.
[4] Vidya, N., & Arun, M. (2023). Updates and applications of CRISPR/Cas technology in plants. *Journal of Plant Biology, 66*(6), 499–518.
[5] Jinek, M., et al. (2012). A programmable dual-RNA-guided DNA endonuclease in adaptive bacterial immunity. *Science, 337*(6096), 816–821.
[6] Zhang, D., et al. (2017). Perfectly matched 20-nucleotide guide RNA sequences enable robust genome editing using high-fidelity SpCas9 nucleases. *Genome Biology, 18*(1), 191.
[7] Molla, K. A., et al. (2022). Predictable NHEJ insertion and assessment of HDR editing strategies in plants. *Frontiers in Genome Editing, 4*. 2022.
[8] Wimberger, S., et al. (2023). Simultaneous inhibition of DNA-PK and PolΘ improves integration efficiency and precision of genome editing. *Nature Communications, 14*(1), 4761.
[9] Gaudelli, N. M., et al. (2017). Programmable base editing of A•T to G•C in genomic DNA without DNA cleavage. *Nature, 551*(7681), 464–471.
[10] Zong, Y., et al. (2017). Precise base editing in rice, wheat and maize with a Cas9-cytidine deaminase fusion. *Nat Biotechnol, 35*(5), 438–440.
[11] Anzalone, A. V., et al. (2019). Search-and-replace genome editing without double-strand breaks or donor DNA. *Nature, 576*(7785), 149–157.
[12] Vu, T. V., et al. (2024). Prime editing: Mechanism insight and recent applications in plants. *Plant Biotechnology Journal, 22*(1), 19–36.
[13] Gondalia, N., et al. (2025). Harnessing promoter elements to enhance gene editing in plants: Perspectives and advances. *Plant Biotechnology Journal, 23*(5), 1375–1395.
[14] Anzalone, A. V., Koblan, L. W., & Liu, D. R. (2020). Genome editing with CRISPR-Cas nucleases, base editors, transposases and prime editors. *Nat Biotechnol, 38*(7), 824–844.
[15] Kumar, J., et al. (2023). Efficient protein tagging and cis-regulatory element engineering via precise and directional oligonucleotide-based targeted insertion in plants. *Plant Cell, 35*(8), 2722–2735.
[16] Zhao, Y., et al. (2025). Precise deletion, replacement and inversion of large DNA fragments in plants using dual prime editing. *Nature Plants, 11*(2), 191–205.
[17] Wang, D., et al. (2024). Advances in base editing: A focus on base transversions. *Mutat Res Rev Mutat Res, 794*, 108515.
[18] Komor, A. C., et al. (2017). Improved base excision repair inhibition and bacteriophage Mu Gam protein yields C: G-to-T: A base editors with higher efficiency and product purity. *Sci Adv, 3*(8), eaao4774.

DOI: 10.54254/2753-8818/2026.38990

[19] Richter, M. F., et al. (2020). Phage-assisted evolution of an adenine base editor with improved Cas domain compatibility and activity. *Nature Biotechnology, 38*(7), 883–891.
[20] Oliva, R., et al. (2019). Broad-spectrum resistance to bacterial blight in rice using genome editing. *Nature Biotechnology, 37*(11), 1344–1350.
[21] Zhang, X., et al. (2024). Programmable broad-spectrum resistance to bacterial blight using targeted insertion in rice. *Cell Discovery, 10*(1), 100.
[22] Luo, H., et al. (2026). Multiplex editing of OsMads26, OsBsr-d1, OsELF3-2 and OsERF922 with CRISPR/Cas9 confers enhanced resistance to pathogens and abiotic stresses and boosts grain yield in rice (Oryza sativa). *International Journal of Molecular Sciences, 27*(2).
[23] Bishnoi, R., et al. (2023). Genome engineering of disease susceptibility genes for enhancing resistance in plants. *Functional & Integrative Genomics, 23*(3), 207.
[24] Loo, E. P. I., et al. (2025). Removal of transgenes and evaluation of yield penalties in genome edited bacterial blight resistant rice varieties. *Plant Biotechnology Journal*.
[25] Xu, S., et al. (2024). A KNOX II transcription factor suppresses the NLR immune receptor BRG8-mediated immunity in rice. *Plant Communications, 5*(10), 101001.
[26] Han, X., et al. (2025). Rational design of promoter editing confers multipathogen resistance in rice. *Genome Biology, 26*(1), 371.
[27] Guo, Y., et al. (2024). CRISPR–Cas9-mediated promoter editing of FERONIA-Like receptor 13 increases plant growth and disease resistance in rice. *The Crop Journal, 12*(6), 1597–1606.
[28] Wang, Y., et al. (2024). Editing of the MeSWEET10a promoter yields bacterial blight resistance in cassava cultivar SC8. *Molecular Plant Pathology, 25*(10), e70010.
[29] Peng, A., et al. (2017). Engineering canker-resistant plants through CRISPR/Cas9-targeted editing of the susceptibility gene CsLOB1 promoter in citrus. *Plant Biotechnology Journal, 15*(12), 1509–1519.
[30] Ortigosa, A., et al. (2019). Design of a bacterial speck resistant tomato by CRISPR/Cas9-mediated editing of SlJAZ2. *Plant Biotechnology Journal, 17*(3), 665–673.
[31] Rivera-Toro, D. M., de Folter, S., & Alvarez-Venegas, R. (2025). CRISPR/dCas12a-mediated activation of SlPAL2 enhances tomato resistance against bacterial canker disease. *PLoS One, 20*(3), e0320436.
[32] García-Murillo, L., et al. (2023). CRISPRa-mediated transcriptional activation of the SlPR-1 gene in edited tomato plants. *Plant Science, 329*, 111617.
[33] Jones, J. D. G., Staskawicz, B. J., & Dangl, J. L. (2024). The plant immune system: From discovery to deployment. *Cell, 187*(9), 2095–2116.
[34] Contreras, M. P., et al. (2023). NLR receptors in plant immunity: Making sense of the alphabet soup. *The EMBO Reports, 24*(10), EMBR202357495.
[35] Yinyan, X., Hua, Y., & Li, W. (2025). Plant immunity study: Mechanism breakthroughs and application innovations. *Chinese Bulletin of Botany, 60*(5), 693–703.
[36] Nizan, S., et al. (2023). Mutagenesis of the melon Prv gene by CRISPR/Cas9 breaks papaya ringspot virus resistance and generates an autoimmune allele with constitutive defense responses. *J Exp Bot, 74*(15), 4579–4596.
[37] Chakravarthy, S., et al. (2010). Identification of Nicotiana benthamiana genes involved in pathogen-associated molecular pattern–triggered immunity. *Molecular Plant-Microbe Interactions, 23*(6), 715-726.
[38] Ngou, B. P. M., et al. (2021). Mutual potentiation of plant immunity by cell-surface and intracellular receptors. *Nature, 592*(7852), 110-115.
[39] Wu, D., et al. (2025). Susceptibility genes in plants: From molecular mechanisms to ecological implications for disease resistance. *Biotechnol Adv, 83*, 108664.
[40] Mirzaei, L., Abbasi, S., & Fazli, M. (2025). WRKY transcription factors enhance plant defense responses against viral pathogens: A review. *Functional Plant Biology, 52*(10).
[41] Pieterse, C. M., et al. (2009). Networking by small-molecule hormones in plant immunity. *Nat Chem Biol, 5*(5), 308-16.
[42] Liu, X., et al. (2024). The miR7125-MdARF1 module enhances the resistance of apple to Colletotrichum gloeosporioides by promoting lignin synthesis in response to salicylic acid signalling. *Plant Biotechnology Journal, 22*(10), 2741-2755.
[43] Li, L., et al. (2024). A frameshift mutation in *JAZ10* resolves the growth versus defense dilemma in rice. *Proceedings of the National Academy of Sciences, 121*(52), e2413564121.
[44] Zhou, W., et al. (2019). A jasmonate signaling network activates root stem cells and promotes regeneration. *Cell, 177*(4), 942–956.e14.
[45] Thakur, S., et al. (2025). Turning susceptibility into strength: A new era of durable resistance in plants through genome editing. *Plants (Basel), 14*(19).

DOI: 10.54254/2753-8818/2026.38990

[46] Wang, D., et al. (2025). Engineering tomato disease resistance by manipulating susceptibility genes. *Front Genome Ed, 7*, 1537148.
[47] Huot, B., et al. (2014). Growth-defense tradeoffs in plants: A balancing act to optimize fitness. *Molecular Plant, 7*(8), 1267–1287.
[48] Alariqi, M., et al. (2025). Enhancing specificity, precision, accessibility, flexibility, and safety to overcome traditional CRISPR/Cas editing challenges and shape future innovations. *Adv Sci (Weinh), 12*(28), e2416331.
[49] Kleinstiver, B. P., et al. (2016). High-fidelity CRISPR–Cas9 nucleases with no detectable genome-wide off-target effects. *Nature, 529*(7587), 490–495.
[50] Kim, M.-G., et al. (2025). Revolutionizing CRISPR technology with artificial intelligence. *Experimental & Molecular Medicine, 57*(7), 1419–1431.
[51] Nakamae, K., et al. (2025). Risk prediction of RNA off-targets of CRISPR base editors in tissue-specific transcriptomes using language models. *Int J Mol Sci, 26*(4).
[52] Kim, H. K., & Kim, H. H. (2026). Evaluation and prediction of guide RNA activities in genome-editing tools. *Nature Reviews Bioengineering, 4*(1), 82–97.
[53] Maher, M. F., et al. (2020). Plant gene editing through de novo induction of meristems. *Nat Biotechnol, 38*(1), 84–89.
[54] Xiao, Y., Yu, H., & Wan, L. (2025). Plant immunity study: Mechanism breakthroughs and application innovations. *Chinese Bulletin of Botany, 60*(5), 693–703.