



Epigenetic Modulation of Plant Disease Resistance

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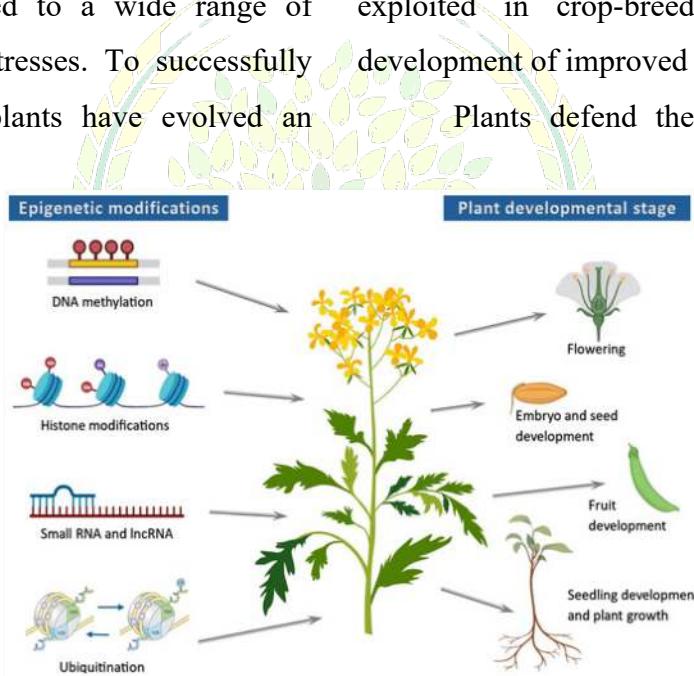
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Plants, being sessile organisms, are continuously exposed to a wide range of biotic and abiotic stresses. To successfully repel invading pathogens, plants have evolved an intricate and highly regulated immune system that enables rapid and need-based activation of defence genes. Epigenetics refers to heritable changes in gene function that occur without alterations in the underlying DNA sequence and can be transmitted mitotically and/or meiotically. Epigenetic regulation—mediated through DNA methylation, histone modifications, and small RNAs—plays a critical role in controlling gene expression during stress responses. With advances in molecular tools, epigenome editing has recently emerged as a promising strategy for enhancing resistance against various plant stresses. Stable and heritable epigenetic regulatory mechanisms

associated with disease resistance can be effectively exploited in crop-breeding programmes for the development of improved and resilient varieties.



Plants defend themselves against pathogens through sophisticated defence mechanisms that have evolved over time. Pattern-triggered immunity (PTI) is activated when pattern recognition receptors (PRRs) recognize pathogen-associated molecular patterns

(PAMPs) or damage-associated molecular patterns (DAMPs). In contrast, effector-triggered immunity (ETI) is initiated when plant resistance (R) proteins detect pathogen-secreted effector molecules (Jones et al., 2016). Both PTI and ETI involve extensive transcriptional reprogramming of defence-related genes; however, they differ in the magnitude and duration of downstream responses.

Many defence-associated genes are located in heterochromatic regions and are transcriptionally inaccessible under normal conditions. Activation of these genes requires coordinated action of epigenetic regulators that modulate DNA methylation patterns, chromatin structure, and histone modifications (Qi et al., 2023).

Types of Epigenetic Mechanisms

Epigenetic mechanisms can be broadly classified into three major categories: DNA methylation/demethylation, histone modifications, and non-coding RNAs.

DNA Methylation

DNA methylation involves the addition of a methyl group to the 5-position of cytosine residues, catalysed by DNA methyltransferases (MTases). It is generally associated with transcriptional repression, particularly when present in promoter regions, and results in heritable methylation patterns transmitted during mitosis and meiosis. DNA methylation is regulated through both methylation (maintenance and de novo) and demethylation (active and passive) processes. Maintenance methylation preserves existing methylation patterns during DNA replication, whereas de novo methylation establishes new methylation marks on previously unmethylated regions, primarily via the RNA-directed DNA methylation (RdDM) pathway. In plants, DNA methylation is controlled by three major families of methyltransferases: maintenance methyltransferases (METs), chromomethylases (CMTs), and de novo DNA methyltransferases (DRMs).

Histone Modifications

Histones are basic proteins rich in lysine and arginine residues, which impart a positive charge and facilitate interaction with negatively charged DNA. The N-terminal tails of histones are subject to diverse post-translational modifications, including methylation, acetylation, ubiquitination, and phosphorylation. Histone methylation is among the most extensively studied modifications and can either activate or repress gene expression depending on the specific residue modified; for example, H3K4 methylation is associated with transcriptional activation, whereas H3K27 methylation is linked to gene repression. Histone acetylation neutralizes the positive charge on lysine residues, reducing histone–DNA affinity and thereby enhancing accessibility of transcriptional machinery such as transcription factors and RNA polymerase II.

Non-coding RNAs

Non-coding RNAs are RNA molecules that do not encode proteins but play crucial roles in gene regulation and genome stability. Major classes include microRNAs (miRNAs) and small interfering RNAs (siRNAs), which mediate RNA interference pathways by promoting mRNA degradation or inhibiting translation, thereby regulating gene expression during plant–pathogen interactions.

Applications in Plant Disease Resistance

Both naturally occurring epigenetic variation and artificially induced epigenetic modifications can significantly influence plant responses to pathogens. These mechanisms offer considerable potential for epigenetic breeding approaches aimed at improving

disease resistance. Selected examples of epigenetic regulators implicated in plant disease resistance are summarized in Table 1.

Table 1. Reported use of epigenetic regulators in plant disease resistance

Gene name	Gene function	Role in host-pathogen interaction	Reference
AeDRM2	De novo DNA methyltransferase	Knockdown enhanced resistance of <i>Aegilops tauschii</i> to <i>Blumeria graminis</i> f. sp. <i>tritici</i>	Geng et al., 2019
Xa21G	Resistance to <i>Xanthomonas oryzae</i> pv. <i>oryzae</i>	Demethylation using 5-azadeoxycytidine improved disease resistance	Akimoto et al., 2007
IR-specific siRNA (24 nt)	Transcriptional gene silencing	Increased methylation of MYMIV intergenic region conferred resistance in soybean	Yadav & Chattopadhyay, 2011
MeSWEET10a	Susceptibility gene in cassava	Targeted methylation of EBE site conferred resistance to bacterial blight	Veley et al., 2023
SIPR1	Defence-related gene	H3K4me3 enrichment enhanced resistance to <i>Clavibacter michiganensis</i> ssp. <i>michiganensis</i>	García-Murillo et al., 2023

Conclusion

Epigenetic modification represents a promising alternative strategy for developing disease-resistant crop varieties through epi-breeding. Epigenomic variations associated with disease resistance traits can serve as molecular epigenetic markers to support selection and evaluation in breeding programmes. Furthermore, epigenetic modelling may help predict the effects of epigenomic variation on disease resistance and guide targeted epigenome engineering. Future research should focus on elucidating the precise regulatory mechanisms by which epigenetic factors are recruited to defence genes during plant immune responses.

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