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Insights into Epigenetic Regulation of Systemic Acquired Resistance in Plants

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ABSTRACT

Plants, as sessile organisms, depend on a multi-layered innate immune system to withstand diverse biotic stressors. Their defense begins with physical and chemical barriers, followed by inducible responses mediated through pattern-triggered (PTI) and effector-triggered immunity (ETI). These local defenses integrate with systemic mechanisms, notably Systemic Acquired Resistance (SAR) and Induced Systemic Resistance (ISR), which provide long-lasting, broad-spectrum protection. SAR operates through salicylic acid (SA)–dependent signaling involving NPR1, NPR3/4, WRKY, and TGA transcription factors, along with metabolites like N-hydroxy-pipecolic acid (NHP). Epigenetic mechanisms, including DNA methylation and histone modifications such as acetylation and methylation, fine-tune SAR by modulating chromatin accessibility and gene expression. These heritable yet reversible changes establish immune memory, strengthening both local and transgenerational resistance and offering promising strategies for durable disease management and sustainable crop protection.

KEYWORDS: Systemic acquired resistance, Gene regulation, DNA methylation, Histone modification, Salicylic acid signaling, Plant immunity

As sessile organisms, plants are constantly exposed to a wide range of biotic challenges, including pathogens, pests, and herbivores. Unlike animals, which rely on adaptive immunity, plants rely solely on innate immunity, a sophisticated and multi-layered defense system that has evolved to perceive, deter, and neutralize threats. This defense begins with constitutive barriers such as the cuticle, wax coatings, and reinforced cell walls, which serve as the first line of physical protection against invasion. These structural defenses are complemented by a diverse arsenal of antimicrobial secondary metabolites, including phenolics, alkaloids, and terpenoids, which act as chemical deterrents to potential attackers (Dangl et al., 2013; Srivastava et al., 2014; Andolfo and Ercolano, 2015; Srivastava and Ahn, 2015; Srivastava et al., 2016; Srivastava et al., 2018; Kumar et al., 2021; Srivastava and Lodhi, 2022; Srivastava et al., 2023).

Beyond these passive defenses, plants activate inducible responses upon threat detection. They rapidly synthesize defense proteins and specific phytochemicals, forming a dynamic response network that includes both broad-spectrum and pathogen-specific immunity. Central to this are two layered immune responses: pattern-triggered immunity (PTI) and effector-triggered immunity (ETI), which detect conserved microbial patterns or pathogen-derived effectors (MAMPs/PAMPs), respectively (Jones and Dangl, 2006). PTI is initiated when pattern-recognition receptors (PRRs) on the plant surface, such as FLS2 in Arabidopsis, recognize PAMPs like flg22 (Yu et al., 2024). This activates a cascade involving ion fluxes, ROS bursts, MAPK signaling, and callose deposition, creating a robust barrier to pathogen invasion. To overcome PTI, pathogens deploy effectors that suppress host defense. In response, plants have evolved intracellular receptors, mostly NB-LRR proteins, that detect these effectors and trigger ETI. This stronger immune reaction often involves localized cell death, known as the hypersensitive response, as seen in the interaction between *Pseudomonas syringae* AvrPto and the tomato resistance protein Pto (Miller et al., 2017; Lonjon et al., 2024). The coordination between PTI and ETI is regulated through phytohormones like salicylic acid (SA), jasmonic acid and ethylene. SA primarily mediates defense against biotrophic pathogens, while jasmonic acid and ethylene are more active against necrotrophs and herbivores (Vidhyasekaran, 2015; Ding et al., 2022). This hormone crosstalk allows plants to fine-tune their responses based on the nature of the threat. While PTI and ETI are local responses, they can initiate systemic defenses in uninfected tissues. One such mechanism is systemic acquired resistance (SAR), which provides long-term, broad-spectrum immunity. SAR is triggered by localized infection or chemical elicitors and involves signal generation, translocation, SA accumulation, transcriptional reprogramming, and priming of defense genes (Ryals et al., 1994; Klessig et al., 2018; Zeier, 2021).

Epigenetic regulation refers to heritable but reversible modifications in gene expression that occur without changes in the underlying DNA sequence. These modifications influence how genes are activated or silenced and are crucial for regulating plant growth,

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development, stress tolerance, and environmental adaptation. The major epigenetic mechanisms include DNA methylation, which typically represses gene transcription by altering chromatin structure; histone modifications, which change chromatin accessibility and thereby influence transcriptional activity; chromatin remodelling, which repositions nucleosomes to regulate gene accessibility; and non-coding RNAs, which modulate gene expression either post-transcriptionally or by guiding epigenetic modifications to specific genomic regions (Srivastava et al., 2014; Srivastava and Ahn, 2015; Srivastava et al., 2016; Srivastava and Lodhi, 2022; Srivastava et al., 2023). Together, these interconnected processes form a highly dynamic regulatory network that allows plants to fine-tune gene expression, establish stress memory, and even transmit adaptive responses across generations, all without altering their genetic code.

SYSTEMIC DEFENSE MECHANISMS IN PLANTS: A STRATEGIC IMMUNE RESPONSE

Plants rely on systemic defense mechanisms to survive in pathogen-rich environments, especially since they cannot escape or relocate. One of the most vital strategies is SAR, an immune response that transforms a localized infection into long-lasting and whole-plant resistance. Triggered by initial pathogen attack, SAR involves the accumulation of SA and activation of pathogenesis-related (*PR*) genes (Ryals et al., 1994; Klessig et al., 2018). This primes the entire plant to respond more effectively to future infections, even by unrelated pathogens. Unlike local defenses such as the hypersensitive response, which causes cell death at infection sites, whereas SAR is non-lethal and sustained, often lasting for weeks (Durrant and Dong, 2004). Another major systemic strategy is Induced Systemic Resistance (ISR), typically initiated by beneficial root-associated microbes. ISR relies on jasmonic acid and ethylene signaling rather than SA, and enhances defense without triggering *PR* gene expression (Van Wees et al., 2008; Pieterse et al., 2014; Yu et al., 2022). Together, SAR and ISR represent an integrated defense network that allows plants to detect, signal, and prepare distant tissues against potential threats. These mechanisms not only ensure survival but also demonstrate an efficient form of immune memory and adaptability, with growing relevance for crop protection and sustainable agriculture.

GENETIC REGULATION OF SAR PATHWAY

SAR is regulated by a complex genetic network involving biosynthetic, signaling, and transcriptional elements that collectively coordinate systemic immunity in plants. At the core of SAR lies the SA pathway, which operates through two major biosynthetic routes: the isochorismate (IC) pathway, predominant in Arabidopsis, and the phenylalanine ammonia lyase (PAL) pathway, more active in non-model plants (Wanner et al., 1995; Wildermuth et al., 2001; Huang et al., 2010). In the IC pathway, ICS1, EDS5, and PBS3 mediate SA synthesis and transport, while the PAL genes encode enzymes that convert phenylalanine into trans-cinnamic acid, a precursor of SA (Wildermuth et al., 2001; Lefevere et al., 2020). Once synthesized, SA activates the master regulator NPR1, which translocates to the nucleus and interacts with TGA transcription factors to induce *PR* genes (Cao et al., 1994; Cao et al., 1997; Fu and Dong, 2013; Agarwal et al., 2020). NPR1 stability is further modulated by NPR3 and NPR4, which act as SA receptors regulating NPR1 degradation or stabilization in a concentration-dependent manner (Fu et al., 2012; Fu and Dong, 2013; Agarwal et al., 2020). Upstream, the EDS1–PAD4 complex initiates and modulates SA accumulation and signaling, forming a foundational layer of SAR activation (Wiermer et al., 2005).

Transcriptional regulation of SAR involves multiple transcription factor families. TGA factors (TGA2, TGA5, TGA6) directly cooperate with NPR1 at SA-responsive promoters (Zhang et al., 2003; Kesarwani et al., 2007; Backer et al., 2019), while WRKY factors fine-tune defense crosstalk e.g., WRKY70 acts as a positive regulator of SA-mediated responses, whereas WRKY33 promotes jasmonic acid-dependent defenses (Li et al., 2004; Eulgem and Somssich, 2007). Additionally, SARD1 and CBP60g activate *ICS1*, the key gene of the IC pathway, reinforcing SA biosynthesis during pathogen challenge (Zhang et al., 2010).

Beyond SA, N-hydroxy-pipecolic acid (NHP) has emerged as a central SAR metabolite. Synthesized from lysine via ALD1, SARD4, and FMO1, NHP primes systemic tissues for enhanced immune readiness (Chen et al., 2018; Hartmann et al., 2018). NHP works synergistically with SA to activate NPR1-dependent transcription and induces a positive feedback loop by upregulating its own biosynthetic genes and enhancing SA accumulation (Chen et al., 2018). Other systemic signals, including glycerol-3-phosphate (G3P) and azelaic acid, further contribute to long-distance signaling, with DIR1 (Defective in Induced Resistance 1) enabling their systemic transport (Maldonado et al., 2002; Jung et al., 2009; Chanda et al., 2011; Cameron et al., 2016; Kachroo et al., 2022). To prevent overactivation, SAR is balanced by UGT76B1, a UDP-glycosyltransferase that glycosylates and inactivates both SA and NHP. Loss of UGT76B1 results in excessive SAR activation and fitness penalties,

underlining its role in immune homeostasis (von Saint Paul et al., 2011; Bauer et al., 2021). Together, this complex network, which includes TGA and WRKY regulation, NPR1-centered transcriptional control, SA and NHP metabolism, and UGT76B1-mediated fine-tuning, ensures strong defense without sacrificing development. These mechanistic discoveries also point to potential avenues for using targeted techniques to develop crops with long-lasting disease resistance.

EPIGENETIC REGULATION OF SAR PATHWAY

Epigenetic modifications, including DNA methylation, histone modifications, chromatin remodeling, and epigenetic priming, play crucial roles in plant development and defense pathways (Srivastava and Ahn, 2015; Srivastava et al., 2016; Srivastava and Lodhi, 2022; Lodhi et al., 2023; Srivastava et al., 2023). These epigenetic regulators are essential for modulating SAR by controlling defense-related gene expression (Figure 1).

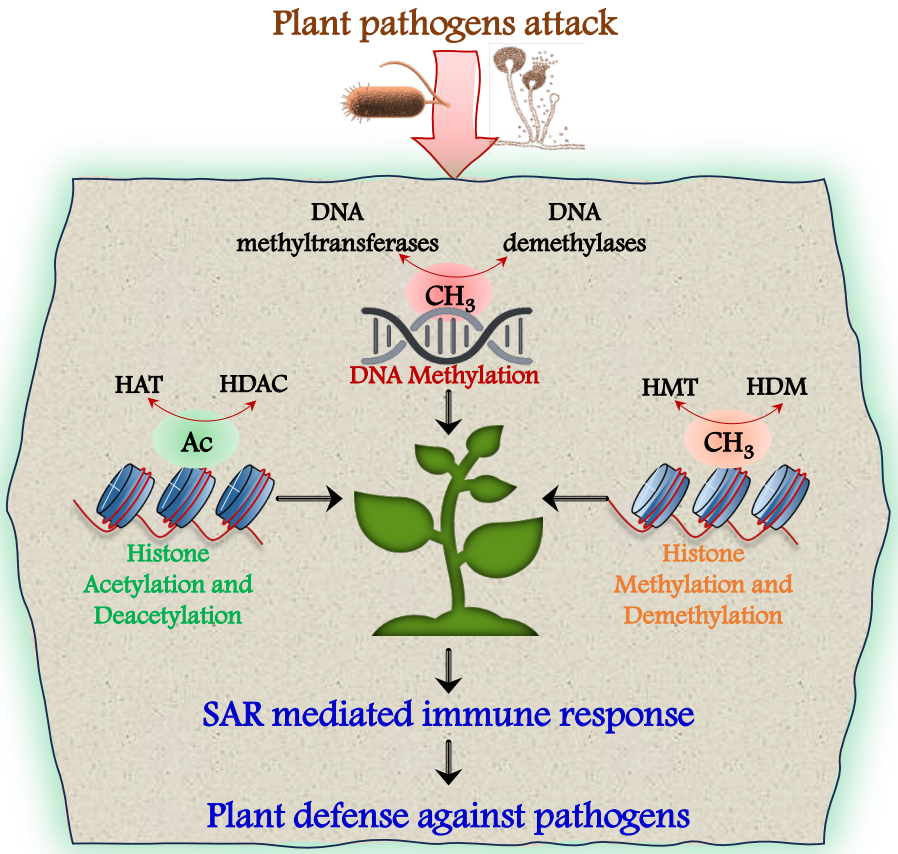

Figure 1: Epigenetic Regulation of SAR through DNA and Histone Modifications in Plants. The figure illustrates the epigenetic regulation of plant defense responses during pathogen attack. DNA methyltransferases and demethylases dynamically regulate gene expression by adding or removing methyl groups on cytosine residues, thereby influencing transcriptional silencing or activation of defense genes. Histone acetyltransferases (HATs) and deacetylases (HDACs) modify histone tails through acetylation and deacetylation, altering chromatin structure to either facilitate or restrict transcription. Likewise, histone methyltransferases (HMTs) and demethylases (HDMs) modulate histone methylation patterns, fine-tuning gene activation or repression. These epigenetic mechanisms act together to modulate SAR-mediated immune responses, enabling plants to mount a systemic defense against pathogens. Through this regulation, plants establish immune memory, allowing faster and stronger responses to future infections.

In Arabidopsis, pathogen infection reduces DNA methylation near defense genes, while the RNA-directed DNA methylation (RdDM) pathway introduces new methylation marks that influence resistance. Histone modifications further fine-tune immune gene expression, acting as either activators or repressors genes (Espinas et al., 2016; Ramirez-Prado et al., 2018; Huang and Jin, 2022). Epigenetic priming maintains defense genes in a transcriptionally poised state, enabling faster activation upon subsequent infections; in some cases, this primed

state and SAR can even be transmitted to the next generation (Luna et al., 2012). Overall, epigenetic regulation provides plants with a dynamic and heritable mechanism for immune memory, offering promising opportunities for enhancing disease resistance and promoting sustainable crop production.

HISTONE METHYLATION AND DEMETHYLATION IN THE SAR PATHWAY

Histone methylation is a key epigenetic modification regulating the expression of SAR–related genes by altering chromatin structure and accessibility. The addition or removal of methyl groups on histone tails determines whether SAR-associated genes are activated or repressed. This dynamic balance is maintained by histone methyltransferases such as ARABIDOPSIS TRITHORAX 1 (ATX1) and SET DOMAIN GROUP 8 (SDG8), and histone demethylases like JUMONJI 14 (JMJ14), which together fine-tune the transcriptional reprogramming essential for efficient SAR (Liu et al., 2019). ATX1, an H3K4 trimethyltransferase, promotes SAR by activating key defense genes. It directly catalyzes H3K4me3 deposition on immune-related genes such as transcription factor WRKY70, creating transcriptionally active chromatin and inducing the expression of defense marker PR1 (Alvarez-Venegas et al., 2007). The recruitment of ATX1 to these loci highlights an epigenetic layer of SAR regulation that enables rapid and sustained activation of defense genes upon pathogen challenge. SDG8, a key histone methyltransferase, catalyzes H3K36 methylation—a mark linked to transcriptional elongation and active chromatin. It modulates jasmonate/ethylene signaling genes such as *PDF1.2*, connecting SAR with broader defense pathways. SDG8 promotes basal and inducible expression of defense genes, including *MKK5*, *PDF1.2a*, and *VSP2*, through H3K36me3 deposition. In *sdg8-1* mutants, reduced *ERF1* and *MYC2* expression highlights its role in regulating key transcription factors. Thus, SDG8-mediated H3K36me3 establishes a permissive chromatin state that enhances defense gene activation and provides transcriptional memory supporting plant immunity (Berr et al., 2010; Kang et al., 2022). Furthermore, SDG8 enhances Arabidopsis immunity by cooperating with RNA polymerase II. The *sdg8-1* mutant shows reduced resistance to *P. syringae* and weaker activation of SA-dependent genes like *PR1* and *PR2*. SDG8 promotes H3K36me3, H3K4me3, and RNAPII loading, linking chromatin modification with strong, sustained defense gene expression (Zhang et al., 2020). In contrast, JMJ14, an H3K4 demethylase, represses defense genes in the absence of pathogens by removing activating methyl marks, conserving energy. It also represses *SNI1*, a negative regulator of SAR, by erasing H3K4me3 marks. In *jmj14* mutants, elevated *SNI1* expression and increased H3K4 methylation at PR1 promoters indicate that JMJ14 fine-tunes plant immunity by balancing positive (PR1, ALD1, and FMO1) and negative (SNI1) defense regulators (Li et al., 1999; Li et al., 2020).

HISTONE ACETYLATION AND DEACETYLATION IN THE SAR PATHWAY

Histone acetylation facilitates chromatin relaxation, enhancing transcriptional access and promoting gene expression. A study uncovers the mechanism of PR-1a transcription initiation in tobacco under SA induction. It is the first to show that a nucleosome–repressor complex over the core promoter keeps the gene inactive in the uninduced state. Upon SA or HDAC inhibitor (TSA) treatment, increased H3K9/H4K16 acetylation and H3K4me2/3 lead to nucleosome disassembly and gene activation. Meanwhile, the negative regulator SNI1 suppresses *AtPR1* in resting conditions, whereas *lsd1* mutants show up to 1000-fold higher expression. Thus, PR-1a activation depends on epigenetic remodeling that removes repressive chromatin marks following SA treatment (Lodhi et al., 2023). GCN5, a histone acetyltransferase in the SAGA complex, modulates chromatin structure by acetylating H3K14 to regulate gene expression in Arabidopsis (Srivastava et al., 2015; Kim et al., 2020). Genome-wide analyses revealed a dual role of GCN5: loss of function reduced H3K14ac at the 5′ ends of downregulated genes but increased it at the 3′ ends of upregulated ones, correlating with H3K9ac changes. Functionally, GCN5 represses SA accumulation and SA-mediated immunity, balancing biotic and abiotic stress responses. Thus, GCN5 acts as a key epigenetic regulator linking histone acetylation to plant stress adaptation (Kim et al., 2020). Another study shows that NPR1 forms a coactivator complex with CBP/p300-type histone acetyltransferases (HACs) and TGA transcription factors (Jin et al., 2018). Upon SA induction, the HAC–NPR1–TGA complex binds PR chromatin, promoting histone acetylation and transcriptional activation. Thus, SA-triggered immunity operates through NPR1–HAC–mediated epigenetic reprogramming of defense genes (Jin et al., 2018).

Conversely, histone deacetylases act as repressors of SAR. Histone deacetylase 6 (HDA6) acts as a negative regulator of pathogen defense in Arabidopsis (Wang et al., 2017). The *hda6* (*shi5)* mutant shows spontaneous defense activation, elevated *PR1* and *PR2* expression,

and increased histone acetylation at their promoters, leading to enhanced resistance to Pst DC3000. HDA6 binds these promoters under normal and infected conditions, indicating that it represses pathogen-responsive genes by maintaining low histone acetylation, thereby modulating defense activation (Wang et al., 2017). The RPD3/HDA1-class histone deacetylase HDA19 represses SA-mediated defense in Arabidopsis by deacetylating histones at PR1 and PR2 promoters. Loss of HDA19 increases SA levels, *PR* gene expression, and resistance to *P. syringae*, showing its role in preventing excessive immune activation (Choi et al., 2012). AtSRT2, a plant homolog of yeast Sir2, acts as a negative regulator of basal defense in Arabidopsis. Loss of AtSRT2 enhances resistance to *P. syringae* and upregulates *PR1*, *PAD4*, *EDS5*, and *SID2*, while overexpression increases susceptibility. Thus, AtSRT2 represses SA biosynthesis and defense gene expression, weakening immune responses (Wang et al., 2010). The histone deacetylase TaHDT701, an HD2-type enzyme, acts as a negative regulator of wheat defense against *Blumeria graminis*. The TaHDT701–TaHDA6–TaHOS15 histone deacetylase complex negatively regulates wheat defense against *B. graminis* by repressing *TaPR* and *TaWRKY45* genes. Silencing these components enhances resistance through increased histone acetylation and active chromatin at defense gene promoters (Zhi et al., 2020). Overall, the balance between histone acetylation and deacetylation enables flexible control of SAR pathway gene expression, ensuring rapid defense activation during infection while conserving energy under normal conditions.

DNA METHYLATION AND DEMETHYLATION IN THE SAR PATHWAY

DNA methylation is a stable yet reversible epigenetic modification, which is dynamicall regulated by DNA methyltransferases and DNA demethylase (Srivastava and Lodhi, 2022). In plants, methylation occurs in CG, CHG, and CHH contexts and is maintained by specific DNA methyltransferases such as MET1, CMT3, and DRM2. Equally critical is active demethylation, mediated by DNA demethylase like ROS1, DME, DML2, and DML3, which remove methyl marks to enable the transcription of immune-responsive genes (Li et al., 2018). DNA methylation dynamically regulates Arabidopsis immunity. Pathogen or SA exposure induces differentially methylated regions, especially near transposons, linked to 21-nt siRNA accumulation and altered defense gene expression (Dowen et al., 2012). Several studies revealed that regulates SAR by altering the accessibility of defense-related gene loci (Li et al., 2018). SAR provides broad-spectrum protection by activating SA-dependent defense in distal tissues. SA induces extensive gene expression and DNA demethylation at defense-related transposons, while pathogen-responsive siRNAs support long-distance and heritable immune priming. Together, these epigenetic changes likely underlie transgenerational inheritance of enhanced immunity (Yu et al., 2013). SAR can be inherited epigenetically in Arabidopsis. Offspring of PstDC3000-infected plants show enhanced SA-dependent defense and resistance to biotrophs, linked to increased H3K9 acetylation and DNA hypomethylation at defense genes. This transgenerational SAR requires NPR1, suggesting that epigenetic reprogramming primes SA-mediated immunity across generations (Luna et al., 2012). A key pathway involved in SAR regulation is RdDM, comprising components like NRPE1, NRPD2, and AGO4. RdDM primarily targets transposable elements and adjacent regulatory regions, indirectly affecting immune gene expression by modifying chromatin structure. Mutants deficient in RdDM components, such as *nrpe1* and *nrpd2*, exhibit widespread DNA hypomethylation, enhanced basal resistance, and elevated expression of SA-responsive genes like *PR1* (López et al., 2011). DNA (de)methylation influences SA-dependent defense against biotrophic pathogen *Hyaloperonospora arabidopsidis*. The *nrpe1* mutant with DNA hypomethylation shows enhanced *PR1* induction and resistance, whereas the *ros1* mutant with hypermethylation exhibits reduced *PR1* expression and susceptibility, indicating that hypomethylation primes SA-mediated defense responses (López Sánchez et al., 2016). DNA methylation influences the expression of SUPPRESSOR OF NPR1-1, CONSTITUTIVE 1 (SNC1), a constitutive repressor of PR1. In Arabidopsis, MOS1, a BAT2-domain protein, regulates *SNC1* expression at the chromatin level. Loss of MOS1 suppresses *SNC1* and associated defense responses, whereas DNA demethylation restores expression. Thus, MOS1 modulates *SNC1* transcription through DNA methylation–dependent chromatin control (Li et al., 2010). *PR1* induction was reduced in *ros1* mutants upon flg22 treatment, indicating that ROS1 functions as a positive regulator of SA-dependent defense responses during PTI (Yu et al., 2013).

CONCLUSION

SAR endows plants with long-lasting, broad-spectrum immunity through coordinated signaling involving SA, NHP, and key regulators such as NPR1, WRKY, and TGA transcription factors. Epigenetic

mechanisms, including DNA methylation, histone modifications, and chromatin remodeling, further refine SAR by regulating defense gene expression and establishing immune memory. Future research should integrate epigenomic and functional genomic approaches to unravel SAR-associated chromatin dynamics and harness epigenome editing for developing durable, broad-spectrum disease-resistant crops capable of withstanding diverse environmental challenges.

Conflicts of Interest: The author declares no conflicts of interest.

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