Agri Articles
(e-Magazine for Agricultural Articles)
Volume: 06, Issue: 03 (MAY-JUNE, 2026)
Available online at http://www.agriarticles.com
©Agri Articles, ISSN: 2582-9882

The Genetics of Plant Memory: Epigenetic Markers and Jasmonic Acid in Defense Priming Against Insect Attacks

Dr. Sunidhi Tiwari[1], *Amrisha Kaur[2], Divisha[2], Pragya Shresth[2], Khusbu Behra[2], Keshar Kumari[2], Yuvraj Pratap Singh[2] and Juhi Singh[2]
[1]Assistant Professor, Faculty of Agriculture, Jagannath University, Chaksu, Jaipur
[2]Student, B.Sc. (Hons) Agriculture, Jagannath University, Chaksu, Jaipur
*Corresponding Author's email: amrishakp@gmail.com

Plants, as sessile organisms, cannot flee from herbivore attacks. Instead, they have evolved a sophisticated immunological memory — encoded not in neurons, but in molecular marks etched onto their DNA and histone proteins. This chapter examines the genetic and epigenomic mechanisms underpinning plant defense memory, with a central focus on how jasmonic acid (JA) signaling coordinates short- and long-term priming of resistance against insect herbivores. We review how DNA methylation changes, histone modifications (H3K4me3, H3K36me3), small RNA pathways, and transposable element (TE) remodeling collectively encode a molecular record of herbivory. We further examine how these marks are inherited across generations, enabling offspring to mount faster and more robust defenses. Key model systems — principally Arabidopsis thaliana and Solanum lycopersicum — have illuminated the molecular actors: MYC2/3/4 transcription factors, RNA-directed DNA methylation (RdDM) machinery, the DNA demethylase ROS1, ARGONAUTE1 (AGO1), and DICER-LIKE proteins (DCL2, DCL3, DCL4). Together, these findings position plant epigenetic memory as a dynamic, reversible, and ecologically meaningful adaptation.

Keywords: epigenetic memory · jasmonic acid · plant immunity · herbivory priming · DNA methylation · histone modification · transgenerational resistance · Arabidopsis thaliana · small RNA · chromatin remodeling

Introduction: Rethinking Plant Intelligence

For centuries, the concept of 'memory' was considered exclusive to organisms possessing nervous systems. Plants, being sessile and brainless, were assumed to respond to stress passively. However, decades of molecular plant biology have dismantled this assumption. Plants not only respond to herbivore attacks acutely, but they encode a molecular record of prior insect encounters and use that record to prime faster and stronger defenses when attacked again — even extending this memory to the next generation.

This phenomenon, broadly called defense priming or plant immunological memory, is not mediated by neurons or immunoglobulins, but rather by a suite of epigenomic modifications: chemical tags on DNA and histone proteins that alter how genes are read, silenced, or primed for expression. Central to this process is jasmonic acid (JA), a lipid-derived phytohormone that functions as the primary alarm signal during herbivory.

Pioneering work by Rasmann, De Vos, Casteel, Tian, Halitschke, Sun, Agrawal, Felton, and Jander (2012) demonstrated that plants experiencing caterpillar herbivory in one generation could transmit enhanced insect resistance to their offspring — a finding that challenged traditional Mendelian frameworks and placed epigenetics at the center of plant defense biology. A complementary review by Holeski, Jander, and Agrawal (2012) synthesized rapidly accumulating evidence showing that DNA methylation, histone modifications, and small RNAs each contribute to this transgenerational defense induction.

This chapter traces the molecular pathway from initial herbivore perception through jasmonic acid signaling to epigenetic encoding and transgenerational inheritance of resistance, integrating findings from Arabidopsis, tomato, rice, and other systems.

The Nature of Plant Defense Memory: Definitions and Scope

Priming vs. Direct Induction

Plant defense responses fall into two broad modes. Direct induction refers to the immediate transcriptional activation of defense genes following herbivore attack — producing proteinase inhibitors, glucosinolates, alkaloids, and other anti-feedant compounds. Priming, in contrast, describes a state in which plants prepare their molecular machinery for a faster, stronger response upon subsequent attack, without paying the full metabolic cost of direct induction in the absence of threat. As described by Mahanta, Komal, Samal, Bhoi, Kumar, Mohapatra, Athulya, Majhi, and Mastinu (2025), inducible defenses rely on the detection of herbivore-associated molecular patterns (HAMPs) and damage-associated molecular patterns (DAMPs), perceived by specific receptors that initiate intracellular signaling cascades predominantly regulated by jasmonic acid (JA) and salicylic acid (SA) pathways. Priming represents a meta-level regulatory state where such cascades are pre-tuned for amplified activation.

Temporal Scales of Memory

Defense memory operates across multiple temporal scales. Within the same individual, priming can be maintained for weeks to months after a single herbivore encounter. At the generational scale, transgenerational induced resistance (TIR) has been demonstrated to persist for two or more generations in Arabidopsis. Hannan Parker, Wilkinson, and Ton (2022) note that transgenerational induced resistance is gradually reversed in the absence of stress at a rate proportional to the severity of disease experienced in previous generations — an ecologically adaptive feature that prevents the metabolic costs of constitutive resistance from accumulating when the herbivore threat subsides.

Jasmonic Acid: The Master Alarm Signal of Plant Defense

Biosynthesis and Perception

Jasmonic acid (JA) and its bioactive conjugate jasmonoyl-isoleucine (JA-Ile) are lipid-derived phytohormones biosynthesized from □-linolenic acid via the octadecanoid pathway. The pathway proceeds through the chloroplast — where 12-oxo-phytodienoic acid (OPDA) is produced — and the peroxisome, where OPDA is reduced and subsequently conjugated to isoleucine by JAR1. Herbivore attack triggers membrane damage, releasing linolenic acid and rapidly activating JA biosynthesis. JA-Ile is perceived by the SCF-COI1 receptor complex, which recruits JASMONATE ZIM-DOMAIN (JAZ) repressor proteins for degradation via the 26S proteasome. This relieves repression of MYC2, MYC3, and MYC4 transcription factors — the key activators of JA-responsive defense gene programs.

JA Signaling and Gene Activation

The critical importance of the JA pathway is demonstrated by mutant analyses. Reymond and colleagues (2007), studying Arabidopsis responses to Pieris rapae and Spodoptera littoralis, showed that larvae gained significantly more weight on coi1-1 (coronatine insensitive 1) mutant plants lacking a functional JA receptor than on wild-type plants, confirming that JA pathway integrity is essential for induced defense against both specialist and generalist herbivores.

Long-Term Effects of JA on the Epigenome

A landmark study in Nature Plants by Song and colleagues (2023) provided direct evidence that JA signaling imprints lasting epigenomic changes. Three weeks after a transient JA signaling event, Arabidopsis plants retained induced resistance against herbivory while showing increased susceptibility to pathogens — a lasting trade-off encoded in the epigenome. Transcriptome analysis revealed long-term priming and upregulation of JA-dependent defense genes alongside repression of ethylene- and salicylic acid-dependent genes. Crucially, Song et al. (2023) found that long-term JA-induced resistance required the

MYC2/3/4 transcription factors, RNA-directed DNA methylation (RdDM) machinery, the DNA demethylase ROS1, and the small RNA-binding protein AGO1 — revealing that the JA signaling pathway is directly hardwired into the epigenomic machinery.

Epigenetic Mechanisms Encoding Defense Memory

The epigenomic architecture of plant defense memory rests on three interacting pillars: DNA methylation, histone post-translational modifications, and small RNA pathways. These mechanisms operate synergistically and feed back on one another to establish and maintain the primed state.

DNA Methylation

Methylation Contexts and Machinery: Plant DNA methylation occurs in three sequence contexts: CG, CHG, and CHH (where H = A, T, or C). Each context is maintained by distinct methyltransferases — MET1 (CG), CMT3/CMT2 (CHG), and DRM2 (CHH, via RdDM). Unlike animals, plants also possess active DNA demethylation pathways mediated by the ROS1, DME, and DEMETER-LIKE (DML) family of 5-methylcytosine DNA glycosylases.

The interplay between methylation and demethylation is central to defense memory. Song et al. (2023) found that long-term JA-induced resistance required both the RdDM pathway (for de novo methylation) and ROS1 (for targeted demethylation) — indicating that defense memory is encoded not simply through hypermethylation or hypomethylation, but through a dynamic, site-specific reconfiguration of the methylome.

Transposable Elements as Epigenetic Switches: One of the most unexpected findings from methylome analyses of herbivory-primed plants is the central role of transposable elements (TEs). TEs are repetitive mobile genetic elements normally silenced by DNA methylation and heterochromatic histone marks. Stress-induced mobilization of TEs appears to have regulatory consequences for nearby defense genes. Analyses by Song et al. (2023) revealed that JA-treated Arabidopsis plants were specifically enriched with hypomethylated ATREP2 transposable elements. Hannan Parker, Wilkinson, and Ton (2022) proposed that stress-inducible epigenetic changes at TEs prime the expression of environmentally responsive genes (ERGs) with functions related to plant defence, providing individuals with adaptive resistance traits. The reduced epigenetic silencing of TEs also facilitates their mobilization and directional insertion near defense-related genes, increasing genetic and phenotypic diversification.

Histone Post-Translational Modifications

Active Marks: H3K4me3 and H3K36me3: Zander and Ecker (2026) provide a detailed account of the JA-responsive histone landscape: H3K4me3 and H3K36me3 are associated with actively transcribed genes, and during active JA signaling, H3K4me3 ChIP-seq analyses revealed increased H3K4me3 occupancy around the +1 nucleosome of JA-induced genes. H3K4me3 acts as a 'bookmarking' modification that can persist even through cell division, allowing defense-gene promoters to remain in an accessible, primed chromatin state weeks after the initial JA stimulus has dissipated — a molecular basis for within-generational defense memory. H3K36me3 also plays a role during herbivory. JA treatment and infections with necrotrophic fungi lead to increased H3K36me3 levels at JA genes in Arabidopsis and tomato (Zander and Ecker, 2026), and jasmonates acting as wound signals induce H3K36 trimethylation at JA genes during de novo root regeneration.

Repressive Marks and Polycomb Regulation: H3K27me3 is deposited by the Polycomb Repressive Complex 2 (PRC2) and is associated with gene silencing. Defense genes under normal, non-stressed conditions may be maintained in a low-expression state partly through PRC2 activity. Upon herbivore attack, relief from PRC2 repression — mediated by JA-driven recruitment of histone demethylases — allows rapid defense gene induction. Kang, Fan, Wu, Zhu, and Shen (2022) summarize that pathogen infections trigger local and global epigenetic changes that reprogram plant defense gene transcription through histone modification and chromatin remodeling factors.

Histone Acetylation (H4ac): Zander and Ecker (2026) report that a genome-wide H4ac ChIP-seq analysis demonstrated that genes transcriptionally induced by JA substantially overlap with genes enriched for JA-induced H4ac near their transcription start sites, confirming that histone acetylation is a broad epigenomic response to JA stimulation.

Small RNA Pathways

siRNAs, miRNAs, and Defense Gene Regulation: Small non-coding RNAs — including small interfering RNAs (siRNAs) and microRNAs (miRNAs) — constitute a critical layer of transcriptional and post-transcriptional gene regulation. In defense priming, siRNAs produced by DICER-LIKE (DCL) enzymes guide RNA-directed DNA methylation (RdDM) to silence TEs and regulate defense gene expression. The role of siRNAs in transgenerational defense priming is directly demonstrated by Rasmann et al. (2012), who showed that Arabidopsis mutants deficient in jasmonate perception (coronatine insensitive1) or in siRNA biogenesis (dicer-like2, dicer-like3, dicer-like4, and nuclear RNA polymerase d2a/d2b) do not exhibit inherited resistance — directly implicating the RdDM/siRNA machinery in epigenetic inheritance of defense memory.

AGO1 and the Small RNA–Epigenome Interface

Song et al. (2023) found that long-term JA-induced resistance specifically required AGO1, the primary effector of miRNA-mediated post-transcriptional gene silencing in Arabidopsis. This suggests that the miRNA pathway — not just RdDM siRNAs — is involved in maintaining the JA-primed transcriptomic state over weeks.

Transgenerational Inheritance of Defense Memory

Evidence from Model Systems

Rasmann, De Vos, Casteel, Tian, Halitschke, Sun, Agrawal, Felton, and Jander (2012) challenged Arabidopsis and tomato (Solanum lycopersicum) with caterpillar herbivory (Helicoverpa zea and Pieris rapae), methyl jasmonate application, or mechanical damage and assessed plant resistance in subsequent generations. Induced resistance caused caterpillars to grow up to 50% smaller than on control plants and persisted for two generations in Arabidopsis. The observation of inherited resistance in both Brassicaceae and Solanaceae suggested that this trait may be taxonomically widespread. Holeski, Jander, and Agrawal (2012) reviewed rapidly accumulating evidence and concluded that what was once thought an oddity of plant defense induction now appears to be a taxonomically widespread phenomenon with strong potential to impact the ecology and evolution of species interactions. Their review documented examples across diverse species and highlighted three molecular pillars: DNA methylation, histone modifications, and small RNAs.

Mechanisms of Epigenetic Inheritance

For epigenetic marks to be transmitted to offspring, they must survive gametogenesis and fertilization. In plants, methylation patterns at TEs and some gene loci are largely maintained through meiosis, enabling transmission through pollen and ovules. The key mechanism involves siRNA-mediated reinforcement of DNA methylation patterns. Rasmann et al. (2012) demonstrated that DCL2, DCL3, DCL4 and the RNA-dependent RNA polymerases NRPD2a/NRPD2b are required for transgenerational resistance, confirming that the RdDM pathway communicates defense memory from the parental methylome to offspring chromatin. Hannan Parker, Wilkinson, and Ton (2022) — analyzing Arabidopsis epigenetic recombinant inbred lines (epiRILs) — found that hypomethylated loci near TEs enhanced resistance by priming both SA-dependent and SA-independent defenses. Strikingly, TEs within epigenetic QTLs appeared to prime defense genes through trans-acting mechanisms, suggesting that TE-derived siRNAs can move between genomic loci and influence distal defense gene expression.

Ecological and Evolutionary Significance

Transgenerational resistance has direct ecological consequences. Rasmann and Agrawal (2012) argued that epiallelic variation against biotic threats should be under positive selection in populations of plants where the environment is predictable over time. In plant communities where specific herbivores recur seasonally, transgenerational induced resistance provides a Lamarckian-like adaptive advantage without requiring genetic mutation.

Hannan Parker et al. (2022) further emphasized that the reduced epigenetic silencing of TEs facilitates their mobilization and directional insertion near environmentally responsive genes, increasing the rate of genetic and phenotypic diversification — potentially accelerating adaptive evolution in response to persistent herbivore pressure.

Key Molecular Actors in Defense Memory

Table: Key molecular components of plant defense memory and their roles.

Component	Type	Role in Defense Memory	Key Reference(s)
MYC2/3/4	Transcription Factors	Master activators of JA-dependent defense genes; required for long-term priming	Song et al. (2023)
COI1 (SCF-COI1)	JA Receptor Complex	Perceives JA-Ile; triggers JAZ repressor degradation to activate defense	Reymond et al. (2007)
JAZ Repressors	Regulatory Proteins	Repress MYC2/3/4 in absence of JA; degraded upon herbivore attack	Mahanta et al. (2025)
DRM2 (RdDM)	DNA Methyltransferase	De novo CHH methylation at TEs; establishes epigenetic memory	Song et al. (2023)
ROS1	DNA Demethylase	Active DNA demethylation; required for calibrated JA memory response	Song et al. (2023)
AGO1	Small RNA Effector	Required for long-term JA-induced resistance; miRNA pathway effector	Song et al. (2023)
DCL2/3/4	DICER-LIKE Proteins	Produce 21–24 nt siRNAs required for transgenerational inheritance	Rasmann et al. (2012)
NRPD2a/NRPD2b	RNA Polymerases	Produce dsRNA substrates for DCL cleavage in RdDM pathway	Rasmann et al. (2012)
H3K4me3	Histone Mark (Active)	Bookmarks defense gene promoters for primed re-activation	Zander et al. (2026)
H3K36me3	Histone Mark (Active)	Enriched at JA genes upon herbivory and fungal infection	Zander et al. (2026)
H3K27me3 / PRC2	Repressive Mark	Silences defense genes at baseline; relieved during attack	Kang et al. (2022)
H4ac	Histone Acetylation	Genome-wide active mark at JA-induced gene TSS regions	Zander et al. (2026)
ATREP2 TEs	Transposable Elements	Hypomethylated after JA treatment; linked to epigenomic restructuring	Song et al. (2023)

Within-Generation vs. Transgenerational Memory

It is important to distinguish between within-generational (somatic) defense memory and transgenerational defense memory, as the molecular mechanisms overlap but are not identical.

Within-generational memory operates predominantly through histone modifications — H3K4me3 bookmarking, H3K36me3, and H4 acetylation — that keep defense gene chromatin in an open, primed configuration. These marks can be maintained through mitosis in somatic cells, allowing newly formed leaves to be primed for defense even when they were not present during the initial herbivore attack.

Song et al. (2023) demonstrated that within-generational JA memory persisted for three weeks and extended to newly formed leaves — tissues that had no direct contact with herbivores — demonstrating the systemic and chromatin-based nature of the primed state. Methylome analysis revealed that this within-generation memory was associated with JA-triggered TE hypomethylation rather than changes at defense gene promoters themselves.

Transgenerational memory, in contrast, requires the additional step of epigenetic marks surviving meiosis and fertilization. siRNAs produced from altered TE loci in the parent are loaded into pollen and ovules, and after fertilization, they guide RdDM in the zygote, establishing the offspring methylome in a defense-primed configuration. The memory is gradually erased over generations in the absence of herbivore pressure.

StressMemory and Priming in CropSpecies: Agricultural Implications

Meena, Shekhawat, Chand, Choudhary, Sharma, and Lekha (2023) outline how acquisition and preservation of stress memory for the progeny — through methylation, histone modification, and chromatin structure alterations — are the focus of attention for crop breeders. Priming applications place plants into a physiological state that allows them to respond more rapidly and robustly after exposure to biotic or abiotic stress.

Chemical priming agents — including □-aminobutyric acid (BABA), methyl jasmonate, and acetic acid— can mimic the epigenomic priming achieved by herbivore attack. In rice, Bertini, Proietti, Focaracci, Sabatini, and Caruso (2018) demonstrated epigenetic control of defense genes following MeJA-induced priming in Oryza sativa, showing that methyl jasmonate application triggered lasting changes in histone modifications at defense gene loci — confirming conservation of JA-epigenome interactions across monocots and dicots.

Huang and Jin (2022) of the University of California, Riverside, emphasize that plants have evolved variable phenotypic plasticity to counteract different pathogens and pests during immobile life, and that epigenetic regulation provides a key adaptive mechanism that could be exploitable in designing stress-resistant plant varieties through precision breeding or epigenome-aware agronomic interventions.

Integrative Model: The Molecular Circuit of Defense Memory

Drawing together the evidence reviewed in this chapter, we propose a five-phase integrative model for the molecular circuit of plant defense memory following insect attack:

- **Phase 1 — Perception and Signal Initiation:** Herbivore oral secretions (HAMPs) and cellular damage (DAMPs) are perceived by pattern recognition receptors (PRRs) at the plasma membrane, triggering calcium influx, MAPK cascades, and ROS bursts, culminating in activation of the octadecanoid pathway and JA biosynthesis.
- **Phase 2 — JA Signaling and Defense Activation:** JA-Ile accumulates and binds COI1, targeting JAZ repressors for proteasomal degradation. Freed MYC2/3/4 transcription factors drive expression of hundreds of defense genes, producing proteinase inhibitors, glucosinolates, alkaloids, and herbivore-deterrent volatiles.
- **Phase 3 — Epigenomic Encoding:** Concurrent with transcriptional activation, histone modification machinery deposits active marks (H3K4me3, H3K36me3, H4ac) at defense gene loci. RdDM activity guided by AGO4-loaded 24-nt siRNAs targets TE loci for CHH methylation. ROS1-mediated demethylation removes silencing marks at specific TEs, contributing to genome-wide epigenomic reprogramming.
- **Phase 4 — Memory Maintenance:** After the herbivore attack subsides and JA levels return to baseline, H3K4me3 marks persist at defense gene promoters, keeping chromatin in a primed, accessible state. Altered TE methylation patterns are stably maintained. The plant is now primed for rapid reactivation upon subsequent attack.

- **Phase 5 — Systemic Spread and Generational Transmission:** Systemic JA signaling and mobile small RNAs spread the primed state to distal tissues. In reproductive tissues, siRNAs produced from altered TE loci are loaded into pollen and ovules. After fertilization, these siRNAs guide RdDM in the zygote, establishing the offspring methylome in a defense-primed configuration — gradually erased over unstressed generations.

Open Questions and Future Directions

Despite remarkable progress, several fundamental questions remain unresolved:

Mobile signals: The identity of signals that transmit systemic priming from attacked leaves to new growth and reproductive tissues remains incompletely characterized. Small RNAs, JA itself, and peptide signals (such as SYSTEMIN in tomato) are candidates, but their relative contributions and interactions require further investigation.

TE targeting specificity: The mechanistic basis by which RdDM activity is enhanced specifically at ATREP2 and other TEs — rather than uniformly across the genome — after JA treatment is not yet understood. How does the JA pathway interface with the targeting specificity of the RdDM machinery?

Conservation across crop species: While *Arabidopsis* serves as a powerful genetic model, the conservation and divergence of defense memory mechanisms across crop species — maize, wheat, soybean, and rice — requires systematic investigation. Whether transgenerational defense priming can be exploited agronomically without deleterious fitness costs remains a key challenge.

Epigenetic evolution in natural populations: The population-level dynamics of epiallele frequency in response to recurring herbivore pressure have only begun to be modeled. Natural population epigenomics of herbivore pressure remains in its infancy, but the epiRIL populations of *Arabidopsis* provide a powerful tool for these studies.

Conclusion

The concept of plant memory, once confined to the realm of speculation, has been transformed by molecular genetics and epigenomics into a richly detailed mechanistic reality. Plants use a sophisticated molecular 'notebook' — written in DNA methylation patterns, histone modification landscapes, and small RNA populations — to record encounters with insect herbivores and pre-tune their defense systems for future attacks. Jasmonic acid sits at the center of this system, serving simultaneously as an acute alarm signal and as the initiator of long-term epigenomic remodeling. The JA–COI1–JAZ–MYC pathway, intersecting with RdDM, ROS1, AGO1, and histone modification machinery, establishes a primed epigenomic state that can persist for weeks within an individual plant and, remarkably, be transmitted to offspring through the germline. The genetics of plant memory is thus not merely an intellectual curiosity. It represents a convergence of molecular biology, ecology, and evolutionary theory that promises to reshape both our understanding of plant life and our strategies for sustainable agriculture.

References

1. Bertini, L., Proietti, S., Focaracci, F., Sabatini, B., and Caruso, C. (2018). Epigenetic control of defense genes following MeJA-induced priming in rice (*O. sativa*). *Journal of Plant Physiology*, 228, 166–177. https://doi.org/10.1016/j.jplph.2018.06.007
2. Hannan Parker, A., Wilkinson, S.W., and Ton, J. (2022). Epigenetics: a catalyst of plant immunity against pathogens. *New Phytologist*, 233(1), 66–83. https://doi.org/10.1111/nph.17699
3. Holeski, L.M., Jander, G., and Agrawal, A.A. (2012). Transgenerational defense induction and epigenetic inheritance in plants. *Trends in Ecology & Evolution*, 27(11), 618–626. https://doi.org/10.1016/j.tree.2012.07.011

4. Huang, C.-Y., and Jin, H. (2022). Coordinated epigenetic regulation in plants: a potent managerial tool to conquer biotic stress. *Frontiers in Plant Science*, 12, 795274. https://doi.org/10.3389/fpls.2021.795274
5. Kang, H., Fan, T., Wu, J., Zhu, Y., and Shen, W.-H. (2022). Histone modification and chromatin remodeling in plant response to pathogens. *Frontiers in Plant Science*, 13, 986940. https://doi.org/10.3389/fpls.2022.986940
6. Mahanta, D.K., Komal, J., Samal, I., Bhoi, T.K., Kumar, P.V.D., Mohapatra, S., Athulya, R., Majhi, P.K., and Mastinu, A. (2025). Plant defense responses to insect herbivores through molecular signaling, secondary metabolites, and associated epigenetic regulation. *Plant Environment Interactions*, e70035. https://doi.org/10.1002/pei3.70035
7. Meena, V.K., Shekhawat, H.V.S., Chand, S., Choudhary, K., Sharma, J.K., and Lekha, L. (2023). Stress memory and priming applications in plants: potential for breeders. In M.A. El-Esawi (Ed.), *Recent Trends in Plant Breeding and Genetics*. IntechOpen. https://doi.org/10.5772/intechopen.1151228
8. Rasmann, S., De Vos, M., Casteel, C.L., Tian, D., Halitschke, R., Sun, J.Y., Agrawal, A.A., Felton, G.W., and Jander, G. (2012). Herbivory in the previous generation primes plants for enhanced insect resistance. *Plant Physiology*, 158(2), 854–863. https://doi.org/10.1104/pp.111.189357
9. Rasmann, S., and Agrawal, A.A. (2012). Ecological role of transgenerational resistance against biotic threats. *Plant Signaling & Behavior*, 7(4), 447–449. https://doi.org/10.4161/psb.19525
10. Reymond, P., Bodenhausen, N., Van Poecke, R.M.P., Krishnamurthy, V., Dicke, M., and Farmer, E.E. (2007). Signaling pathways controlling induced resistance to insect herbivory in Arabidopsis. *Molecular Plant-Microbe Interactions*, 20(11), 1406–1420. https://doi.org/10.1094/MPMI-20-11-1406
11. Song, S., Wang, K., Liu, J., Peng, C., Huang, X., Feng, J., Li, Z., Xu, C., and He, Z. (2023). Long-lasting memory of jasmonic acid-dependent immunity requires DNA demethylation and ARGONAUTE1. *Nature Plants*, 9, 81–96. https://doi.org/10.1038/s41477-022-01313-9
12. Zander, M., and Ecker, J.R. (2026). The environmentally responsive plant epigenome: insights from jasmonate signaling. *New Phytologist*. https://doi.org/10.1111/nph.70865