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Jasmonate-ethylene (JA/ET) signaling – metabolic defense system in the integrative model of plant immunity against phytophagous insects

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Every year, humanity loses up to 40% of crop yields due to pests and plant diseases. This accounts for over USD 220 billion in economic losses, with approximately USD 70 billion caused by invasive insects. Plant responses to damage by arthropod herbivores represent a complex form of biotic stress that combines mechanical injury, danger signals, and hormonal interactions. This article proposes a systemic concept that explains how the JA/ET-dominant immune mode is integrated into a multistable plant immune defense network. Local tissue damage activates DAMP and HAMP signaling, which triggers Ca^{2+}–ROS–MAPK cascades and the synthesis of jasmonic acid and ethylene. These pathways interact with autophagy, ROS/NO homeostasis, and the antioxidant system, forming feedback-regulated loops that coordinate programmed cell death (PCD) with systemic tolerance. The JA/ET branch is viewed as an adaptive configuration of plant immunity that provides energy-efficient, non-specific resistance of the ISR (induced systemic resistance) type. The integration of JA/ET signaling with autophagic and redox-regulatory circuits explains how a plant converts local damage into systemic resilience. Damage to plants caused by phytophagous insects integrates both local and systemic levels of immunity into a unified, dynamic defense system represented by the JA/ET-dominant immune mode. This mode combines molecular signaling, metabolic control, and holistic plant adaptation to herbivores. The integrative multistate model of plant immunity introduces a new level of understanding of natural defense mechanisms and provides a foundation for breeding highly resistant crop varieties of the future. It also forms a scientific basis for the development of fourth-generation bioproducts that activate the plant's natural immunity (either independently or in combination with pesticides). The use of such integrated immune strategies has the potential to reduce crop losses from pests and diseases by 10–20%, which is equivalent to a global economic savings of USD 7–14 billion annually. This approach lowers the risk of resistance development in harmful organisms and promotes ecological sustainability in agroecosystems.

Keywords: JA/ET signaling; autophagy; ROS homeostasis; ISR; systemic plant immunity; herbivore-induced damage.

Introduction

According to estimates by the Food and Agriculture Organization (FAO), up to 40% of global agricultural crop yields are lost annually due to pests and diseases, costing the global economy over USD 220 billion, with invasive insects alone accounting for at least USD 70 billion (Bradshaw et al., 2016).

Arthropod herbivores are among the most widespread and dangerous biotic stressors in agroecosystems. Their harmful impact is not limited to direct mechanical tissue damage, but also includes the induction of complex metabolic and signaling alterations in the plant. Damage to the cellular apoplast, loss of turgor, and release of cellular fragments (DAMPs) trigger local reactive oxygen species (ROS) cascades, changes in Ca^{2+} levels, and activation of hormonal signaling circuits, particularly those involving jasmonic acid (JA) and ethylene (ET). These immune pathways are considered key in the development of natural plant resistance to herbivores and are distinct from the responses to pathogenic microorganisms (Santamaria et al., 2018; Sperotto et al., 2019).

Traditionally, plant immunity is classified into three levels: PTI (Pattern-Triggered Immunity) – immunity triggered by the recognition of pathogen-associated molecular patterns (PAMPs/MAMPs); ETI (Effector-Triggered Immunity) – a specific response to pathogen effectors that activates a hypersensitive reaction (HR/PCD); and ISR (Induced Systemic Resistance) – systemic resistance typically activated by non-pathogenic microorganisms or tissue damage and mediated by JA/ET signaling (Ding et al., 2022; Yu et al., 2022; Wilson et al., 2023; Yu et al., 2024).

Despite the usefulness of this classification, classical immunity does not fully explain the immune responses associated with traumatic damage caused by herbivorous insects. In particular, the mechanical sensing of damage, the connection between the ROS burst, hormonal regulation (JA/ET), and autophagic processes – which determine the balance between cell death and systemic recovery – remain insufficiently studied (Coutand, 2020; Zou et al., 2023).

Recent research has led to the development of an integrative model of plant immunity and stress tolerance, in which the immune system is viewed as a dynamic, multi-level, and multistable signal–metabolic network. Unlike the classical PTI/ETI concept, this model describes immune states as flexible regulatory modes (SA-, JA/ET-, ABA-dominant, and growth-regulatory), which arise depending on the type of pathogen, the nature of cellular damage, and the combined impact of biotic and abiotic stresses. This approach ensures a balance between local damage responses and the preservation of the organism's functional integrity, optimizes energy flows, and promotes the development of adaptive plant tolerance to combined stresses (Patyka et al., 2025a).

The model demonstrates the stepwise integration of immune processes – from the molecular level to organismal and ecological levels – within which the immune response becomes systemic through the coordinated action of tissues, organs, and the microbiome. It provides a scientific foundation for more effective breeding of highly resistant varieties and hybrids, as well as for the development of fourth-generation plant protection products capable of activating natural immunity either independently or in combination with pesticides. At the same time, this approach reduces the risk of resistance development and decreases the need for frequent chemical treatments.

At the center of the new paradigm lies autophagy – a process of cellular reboot that, within the integrative model, is interpreted not only as a mechanism for the removal of damaged components but also as a key regulatory hub of the immune network. Autophagic loops integrate ROS homeostasis, hormonal signaling (JA/ET, ABA, SA), and energy sensors (SnRK1, TOR), thereby creating a regulatory platform for the establishment of tolerance and immune "memory." It has been demonstrated that moderate activation of autophagy induces a state of autophagic priming – a semi-active mode of cellular readiness that enables faster and more energy-efficient responses to attacks by herbivores or pathogens.

Within this model, autophagy becomes a central element of the JA/ET-dominant immune branch, linking local damage responses (PCD, ROS burst) with systemic tolerance of the ISR type (Sertsuvalkul et al., 2022; Zou et al., 2023). ISR represents a JA/ET-dependent systemic physiological and metabolic adaptation that enhances overall resistance to diverse stresses without establishing long-term immune memory (Table 1) (Zhou et al., 2014). In contrast, SAR is an SA-dependent systemic response that develops following local infection and provides long-lasting, specific resistance (Fig. 1).

Table 1
Comparison of SAR and ISR in plants

Characteristic	SAR – systemic acquired resistance	ISR – induced systemic resistance
Type of elicitor	triggered by biotrophic pathogens (viruses, bacteria, fungi that feed on living cells)	induced by necrotrophs, insects, PGPR (plant growth-promoting rhizobacteria), and endophytes.
Main signaling pathway	SA-dominant (salicylic acid)	JA/ET-dominant (jasmonate and ethylene)
Mechanism of action	formation of long-term immune memory following local infection; activation of PR proteins, NPR1, and SAR-related transcription factors	enhancement of tissue "readiness" for stress; activation of antioxidant enzymes, autophagy, and ROS homeostasis without direct involvement of PR proteins
Type of response	specific and immune (analogous to secondary immunity in animals)	non-specific, physiological–metabolic (energy-regulated tolerance)
Duration of effect	long-lasting (weeks, sometimes the entire season)	flexible and reversible (activated only upon repeated stress)
Type of cellular response	local apoptosis (HR) with the formation of immune memory	limited PCD with enhanced antioxidant and detoxification defenses
Molecular markers	PR1, PR2, NPR1, ICS1.	PDF1.2, VSP, LOX, ERF1.
Energy balance	energy-intensive, but provides stable immune memory	energy-efficient, aimed at recovery and tolerance
Biological significance	"immune memory" after infection: the plant "remembers" the pathogen	"systemic tolerance": the plant "trains" its resistance through metabolic readiness

This conceptual article aims to integrate mite and insect herbivory into the plant immune network within the framework of a new integrative model, with a focus on JA/ET-mediated defense as an adaptive response system to herbivores, in interaction with other immune states.

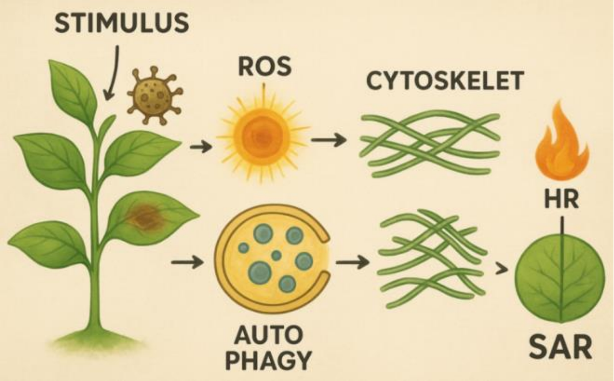

Fig. 1. Model of systemic acquired resistance (SAR) development in plants

Biotic stress from arthropod herbivores: sensing and primary signaling

Damage caused by herbivorous arthropods activates the JA/ET-dominant state of the plant's multistable immune network, which is coordinated by autophagy, the ROS-antioxidant system, the cytoskeleton, and hormonal signaling (Zhao & Wang, 2024; Haghpanah et al., 2025). This response belongs to a non-specific immune management strategy aimed at maintaining homeostasis through controlled programmed cell death (PCD) and the activation of systemic resistance (Zhang et al., 2017).

This protective state corresponds to Induced Systemic Resistance – an energy-regulated systemic resistance that integrates local PCD programs with mechanisms of recovery and tolerance. In response to necrotrophic threats, JA/ET signaling closely interacts with autophagic processes and the ROS system, ensuring the preservation of the plant's functional integrity at the systemic level (Zhao & Wang, 2024; Haghpanah et al., 2025).

The response to herbivores represents one of the most complex forms of biotic stress. It combines mechanical damage, the release of danger-associated molecular patterns (DAMPs), the action of herbivore-associated molecular patterns (HAMPs) such as salivary components, and the activation of cellular mechanosensors. The plant perceives the attack as a sequence of micro-signals – local cell disruption, osmotic shifts, and cell wall degradation are transformed into signaling impulses that initiate a systemic immune response (Santamaria et al., 2018; Abdul et al., 2020).

Among the key DAMPs generated by mechanical cell damage are:

– oligogalacturonides (OGs), fragments of the pectin matrix that activate WAK receptors;

– extracellular ATP, which triggers Ca^{2+} signaling via P2K1/P2K2 receptors;

– peptide signals (Pep1, Pep3), which stimulate PEPR1/2 receptors, promoting a ROS burst and activation of JA/ET-responsive genes (Abdul et al., 2020; Snoeck et al., 2022).

In parallel, the saliva and secretions of herbivores contain HAMPs (herbivore-associated molecular patterns) – low-molecular-weight compounds that modulate the plant immune response. These include enzymes (such as glucosidases, lipases, oxidases), peptides like volicitin and inceptin, fatty acids and their derivatives, which interact with plasma membrane receptors and induce jasmonate synthesis.

Unlike DAMPs, HAMPs not only trigger defense responses but can also manipulate plant signaling pathways (for example, by suppressing salicylic acid (SA) or abscisic acid (ABA) levels) temporarily giving the herbivore a physiological advantage (Snoeck et al., 2022).

The first level of mechanical stress perception is mechanosensing. Injury to epidermal cells and intercellular junctions activates mechanosensitive ion channel receptors such as PIEZO and MCA, as well as wall-associated receptor-like kinases (RLKs). These include:

– WAKs (Wall-Associated Kinases), which recognize DAMP fragments of pectin;

– THESEUS1 and FERONIA (FER), which regulate the cell wall's response to mechanical strain and help maintain the balance between growth and defense;

– LRXs (leucine-rich extensins), which contribute to stabilizing the cell wall during injury.

Activation of these sensors results in a rapid influx of Ca^{2+} into the cytoplasm, serving as a universal primary damage signal (Haghpanah et al., 2025).

An increase in cytosolic Ca^{2+} levels activates calcium-dependent protein kinases (CDPKs) and NADPH oxidases (RBOHs), which catalyze the production of reactive oxygen species (ROS) at the plasma membrane. The ROS burst plays a dual role: as a secondary wave signal propagating between cells, and as a regulator of local programmed cell death (PCD), restricting nutrient access for herbivores. ROS and Ca^{2+} positively reinforce each other, forming a Ca^{2+}–ROS feedback loop that subsequently activates MAPK cascades (MPK3, MPK6, MPK4). These MAPK modules transmit the signal to the nucleus, stimulating transcription of COI1–JAZ–MYC, ERF1/ORA59, PR6, protease inhibitors, and other components of the JA/ET-mediated response (Zhang et al., 2017).

The cell wall functions not only as a passive mechanical barrier but also as a sensory platform – a mechanism known as Cell Wall Integrity (CWI) signaling – capable of responding to the loss of structural integrity. Disruptions in the cellulose–pectin network are detected by receptors such as WAK, THESEUS1, and FER, which initiate signaling cascades involving the activation of ROS, jasmonic acid (JA), and ethylene (ET) (Abdul et al., 2012; Santamaria et al., 2018). Thus, cell wall damage becomes a "language of danger" that the plant translates into a hormonal immune response.

In grasses (e.g., wheat), CWI signaling is more closely linked to ROS cascades and local responses, whereas in dicots (e.g., sunflower), it is more associated with JA/ET regulation and systemic redistribution of metabolites, which determines the distinction between local and systemic types of resistance. Wheat exhibits a JA/ET-dominant, rapid-response type aimed at damage localization, ROS burst, and programmed cell death (PCD). In contrast, sunflower develops a stable JA/ET-based tolerance system, where autophagy, antioxidant mechanisms, and ISR form a systemic homeostatic loop.

This reflects the evolutionary divergence between the reactive (grass-type) and tolerant (oilseed-type) models of JA/ET-mediated immunity. JA/ET-mediated immunity in wheat and sunflower illustrates evolutionarily distinct adaptation strategies: in grasses, via reactive local defense, and in dicots, via systemic regulation of tolerance (Haghpanah et al., 2025). Both models represent functional components of the integrative plant immune network (Table 2).

Table 2
Comparative characteristics of JA/ET-dominant immunity in wheat (monocots) and sunflower (dicots)

Level/module	Winter wheat (monocots)	Sunflower (dicots)	Main differences/features
Sensing and primary signals	RLK and WAK receptors dominate (WAK, FERONIA, LRR-RLK), along with mechanosensors; rapid Ca^{2+} response and activation of DAMP pathways	a well-developed RLK and RLP system (FERONIA, PEPR, THESEUS1), efficient recognition of DAMPs and HAMPs, and pronounced mechanosensitivity	sunflower has a broader set of HAMP receptors, whereas wheat sensing is more mechanosensor-driven
ROS/NO system	short-term local ROS burst (RBOHD/F), activation of SOD, CAT, APX; limited systemic wave	prolonged ROS response and antioxidant control (APX, CAT, ascorbate–glutathione cycle). NO modulates systemic resistance	wheat shows an acute "stress-response" reaction, whereas sunflower exhibits controlled ROS homeostasis and systemic tolerance
JA/ET signaling module	key genes: *TaCOI1*, *TaJAZ1*, *TaMYC2*; lower ethylene sensitivity	JAZ–MYC and *ERF* circuits are activated (homologs of *COI1*, *MYC2*, *ORA*59 have been identified); strong JA/ET co-activation	in sunflower, JA and ET act synergistically, whereas in wheat, they function partially independently
Hormonal crosstalk	JA ↔ SA antagonism predominates; weak interaction with ABA	balance between JA/ET ↔ SA (more often antagonistic) and cooperation with ABA in tolerance formation	sunflower exhibits more flexible hormonal circuits, whereas wheat shows more segregated ones
Autophagy (ATG module)	activated under severe damage; dampens ROS and limits PCD	systemic in nature, supports ISR, regulated by SnRK1/TOR; potentially establishes a primed state	in sunflower, autophagy acts as a tolerance stabilizer, whereas in wheat, it is more local and defense-oriented
ISR (induced systemic resistance)	short-term and localized near the damage site; weak involvement of PGPR	well-pronounced and long-lasting, supported by PGPR and endophytes; establishes systemic JA/ET-mediated tolerance	in sunflower, ISR is a key mechanism of the JA/ET branch, whereas in wheat, it plays a secondary role
Secondary metabolites and volatile organic compounds (VOCs)	synthesis of oxylipins (12-OPDA), phenolic compounds, and protease inhibitors; weak VOC response	active synthesis of terpenes, phenylpropanoids, GLVs, and MeJA; volatile signals coordinate inter-plant communication	sunflower establishes chemical communication and defensive volatile signaling, whereas wheat relies mainly on local defense
Epigenetic control and memory	short-term transcriptional memory (methylation of TaMYC2); weak seed transmission	potential involvement of miR319 and miR396; methylome-based memory of JA/ET genes and possible vegetative inheritance	sunflower shows higher epigenetic stability, whereas wheat exhibits more transient adaptation
Energy strategy and tolerance	high energy costs; rapid response accompanied by PCD	energy-saving strategy with autophagy, ROS, and JA/ET in balance	wheat operates in a reactive "fight mode", whereas sunflower adopts an adaptive "tolerance mode"
Overall type of JA/ET-dominant response	local defensive response (PCD, barrier resistance)	systemic adaptive response (ISR, tolerance, priming)	wheat relies on local defense, whereas sunflower exhibits systemic regulation

The morphological and physiological organization of grasses and dicots determines the type of JA/ET-mediated immunity: Wheat follows a localized defense strategy characterized by high mechanical resistance, while sunflower exhibits a systemic tolerant response involving the microbiome and autophagy. Wheat has evolved a structural-mechanical type of resistance: rigid cell walls, a thick cuticle, and a short life cycle. JA/ET functions rapidly and locally. Sunflower, on the other hand, employs an energy-based systemic resistance strategy: expanded leaf surface, microbial associations, trichomes, and a prolonged life cycle — JA/ET promotes long-term ISR-based tolerance. These morpho-physiological differences explain the evolutionary divergence between the local (wheat) and systemic (sunflower) modes of JA/ET-mediated immunity (Table 3).

The JA/ET-dominant mode as a branch of the integrative immunity model

Jasmonic acid (JA) and ethylene (ET) are central regulators of necrotroph and insect-associated immune responses. They form an interconnected signaling branch that coordinates plant responses to mechanical damage and necrotrophic pathogens, ensuring a switch of cellular programs from growth to defense (Wasternack & Strnad, 2016) (Fig. 2).

Under normal conditions, JA and ET levels are low; however, under stress, their biosynthesis is activated: JA via the linolenic acid pathway (LOX–AOS–OPR3) and ET via the methionine pathway (SAM–ACC–ACO). Their synergistic action stimulates the expression of defense-related genes, in particular ERF1 and ORA59, which activate chitinases, protease inhibitors, lipoxygenases, and enzymes of the phenylpropanoid pathway. As a result, a characteristic response is established that limits herbivore feeding, promotes regeneration of damaged tissues, and enhances systemic preparedness (War et al., 2012).

Jasmonic acid and ethylene are central regulators of necrotroph- and insect-associated immune responses. They form an interconnected signaling branch that coordinates plant responses to mechanical damage and necrotrophic pathogens, ensuring a transition of cells from a growth mode to a defense mode (Wasternack & Strnad, 2016) (Fig. 2).

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Table 3
Morphological and physiological basis of JA/ET-mediated immunity differentiation in winter wheat (*Triticum aestivum*) and sunflower (*Helianthus annuus*)

Aspect / direction	Morphological and physiological features of winter wheat (monocot)	Morphological and physiological features of sunflower (dicot)	Impact on JA/ET-mediated immunity
Leaf anatomical structure	narrow leaf blade, thick cuticle, fewer stomata, sparse trichomes	large leaf blade, thinner cuticle, numerous stomata, predominantly secretory trichomes	sunflower provides more sensory surfaces for HAMP/DAMP perception, whereas wheat more effectively restricts infection spread
Cell wall structure	rich in silicon (variety-dependent), β-glucans, and arabinoxylans; characterized by high mechanical strength	high content of pectins, phenolics, and lignin; activation of a phenolic-based chemical defense response	wheat primarily employs a mechanical barrier, whereas sunflower predominantly initiates a chemical phenolic response
Stomata and transpiration	amphistomatic stomata; ABA-dependent regulation of opening	stomata predominantly on the abaxial surface; rapid ABA-induced closure, with JA/ET accompanying chronic adaptation; VOC emission	in wheat, JA/ET is mainly linked to osmoregulation, whereas in sunflower, it is associated with the regulation of defense proteins and volatile communication signals
Photosynthetic type and metabolism	C3 type, high photosensitivity, limited recovery under stress	C3 type with increased photosystem plasticity, able to maintain JA/ET-mediated immune responses under stress without major photosynthetic losses	sunflower can sustain a stable JA/ET response, whereas wheat shows a short-term reaction
Root system	fibrous, shallow, with weak interaction with PGPR	taproot system, deep, with associations with PGPR and endophytes depending on soil conditions	sunflower establishes a more systemic ISR via root-associated JA/ET signaling, whereas wheat exhibits a more local response
Senescence and lifespan	short life cycle, programmed senescence after heading	longer vegetative period, gradual senescence, high plasticity	sunflower maintains gradual JA/ET-mediated immune activity, whereas JA/ET activation in wheat is short-lived
Adaptation to abiotic stresses	high frost and drought tolerance; ABA-dominant control	moderate drought tolerance; JA/ET accompanies the induction of defense proteins under water stress	wheat relies on ABA-oriented stress resistance, whereas sunflower shows JA/ET-supported adaptation without major energy costs
Phytohormonal profile	ABA dominance, weak JA/ET ↔ SA crosstalk	balanced JA/ET ↔ SA profile; active interaction with ET and induction of defense proteins	this determines the nature and duration of the JA/ET response: in wheat, a short "fight mode", and in sunflower, a stable "tolerance mode."

The JA/ET branch operates in interaction with other hormonal pathways. SA (salicylic acid) is active against biotrophs and can suppress JA/ET signaling via NPR1 and TGA factors. ABA (abscisic acid) coordinates abiotic stress resistance and may attenuate the JA/ET response under conditions of energy limitation. The balance between JA/ET ↔ SA ↔ ABA determines the plant's adaptive strategy – local, systemic, or tolerance-oriented (Table 4).

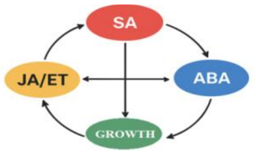

Fig. 2. Distinct defensive states of plant immunity (SA-, JA/ET-, ABA-dominant, and growth-related regulatory immune modes)

Table 4
Comparison of plant immune regimes in an integrative model of defense and stress tolerance

Parameter	SA-dominant mode	JA/ET-dominant mode	ABA-dominant mode	Growth mode
Primary stimulus	biotrophic pathogens (viruses, bacteria)	necrotrophic pathogens, insects, and mites	abiotic stresses (drought, cold, salinity)	optimal growth conditions, minimal stress
Type of immune response	specific (ETI), systemic activation	non-specific, systemic, ISR-mediated	non-specific, ABA-dependent, both local and systemic	minimal immune activation, with resources directed toward growth
Key signaling hormones	SA (salicylic acid)	JA (jasmonates), ET (ethylene)	ABA (abscisic acid)	gibberellins, cytokinins, auxins
Key mechanisms	activation of PR proteins, systemic activation	autophagy, ROS homeostasis, local PCD, VOCs	stomatal closure, osmoregulation, antioxidant systems	synthesis of growth-related proteins, cell division, and expansion
Local vs. systemic nature	systemic (SAR)	local + systemic (ISR)	local + partially systemic	local, growth-oriented
Energy costs	moderate, oriented toward long-term resistance	high during active attack	moderate; resources are directed toward survival	low; resources are allocated to growth and accumulation
Interaction with other modes	may inhibit JA/ET; SA ↔ JA crosstalk	often antagonistic JA/ET ↔ SA; cooperation with ABA under stress	ABA may enhance JA/ET under combined stresses	usually suppresses SA and JA/ET to maximize growth
Effect on stress resistance	high against biotrophs; limited against necrotrophs	high against necrotrophs and herbivores	high against abiotic stresses	supports growth and productivity in the absence of stress

In this model, the JA/ET mode represents one of the multistable states of the regulatory network (Berens et al., 2019). It is activated in response to herbivores and necrotrophs and integrates local reactions (PCD, ROS burst) with systemic ISR and autophagic regulation (Wasternack & Strnad, 2016).

JA/ET signaling is mediated through the COI1–JAZ–MYC2 and EIN2–EIN3–ERF1/ORA59 cascades. Their overall architecture is conserved across plant species but exhibits species-specific features. In wheat (*Triticum aestivum*), these genes occur in multicopy forms (e.g., TaCOI1-A/B/D), enabling a flexible but short-lived response.

In sunflower (*Helianthus annuus*), they are more clustered, with duplicated JAZ and ERF copies, which support a stable ISR response and energy balance (Table 5).

These genomic features underlie evolutionary differences: in wheat, a reactive, local JA/ET model predominates, oriented toward a short-term response; in sunflower, a systemic, adaptive model with long-term resilience is established. Accordingly, the SnRK1–TOR–ATG balance in wheat favors a "fight mode," whereas in sunflower it supports a "tolerance mode" (Table 6).

Thus, the JA/ET-dominant regime coordinates local damage, hormonal crosstalk, and systemic immune tolerance through autophagy-energy integration. It acts as a key adaptive hub, transforming threat signals into a systemic survival response (Song et al., 2022).

Table 5
Key genes and regulatory modules of the JA/ET branch

Gene	Function	Level of regulation	Role in response to herbivores
COI1 (Coronatine Insensitive 1)	JA-Ile receptor, component of the SCF complex	post-translational control	perception of jasmonates and degradation of JAZ repressors
JAZ (Jasmonate ZIM-domain proteins)	repressors of JA-dependent genes	transcriptional control	repression/activation of MYC transcription factors in response to JA
MYC2	bHLH transcription factor	nuclear transcription	coordinates the expression of protease inhibitors and ROS-related genes
ERF1 / ORA59	JA–ET synergy transcription factors	integration of JA/ET signals	regulate anti-insect proteins, phenylpropanoids, and terpene pathways
EIN2 / EIN3	ethylene sensors	membrane-to-nucleus signal transduction	enhance ERF activation and stabilize systemic tolerance

Table 6
Genomic organization of JA/ET signaling modules in grasses (winter wheat, *Triticum aestivum*) and dicot crops (sunflower, *Helianthus annuus*)

Component/module of the JA/ET pathway	Winter wheat (*Triticum aestivum*) – monocots (Poaceae)	Sunflower (*Helianthus annuus*) – dicots (Asteraceae)	Evolutionary and functional interpretation
COI1 (core subunit of the COI1–JAZ receptor complex)	multicopy organization (*TaCOI1-A1*, *TaCOI1-B1*, *TaCOI1-D1*) across three subgenomes. High expression plasticity.	a single major homolog (*HanXRQrCOI1*) with stable expression during damage.	in wheat – polygenic adaptation to variable conditions; in sunflower – stable JA/ET coordination.
JAZ (repressors of JA signaling)	up to 13 homologs (*TaJAZ1–13*) are distributed across subgenomes; rapid degradation under stress	clustered (physical and transcriptional) organization of *HaJAZ* genes with duplications; a high level of interaction	in sunflower – stable coordination of JAZ regulation (supporting ISR); in wheat – a short defensive wave
MYC2 (transcriptional activator of the JA response)	several paralogs (*TaMYC2-A/B/D*) were activated transiently after damage	a single major regulator, *HaMYC2*, coordinates JA/ET-dependent transcriptional cascades	wheat: narrowly specialized regulation; sunflower: integrated, prolonged JA/ET activation
ERF/ORA59 (ethylene-dependent transcription factors)	limited representation of ERF groups and a weak JA↔ET connection	duplicates of *HaERF1* and *HaORA59;* enhanced JA/ET synergy and ISR formation.	sunflower exhibits strong JA/ET convergence, whereas wheat shows a predominantly JA-dominant response
SnRK1–TOR–ATG circuit (energy regulation and autophagy)	an active SnRK1–TOR balance; autophagy is rapidly induced and performs a regenerative function	a shift toward an SnRK1-dominant regime; TOR is partially suppressed, stimulating autophagic tolerance	wheat – reactive "local defense" strategy; sunflower – systemic tolerant "energy conservation" strategy
Genomic architecture	hexaploid (AABBDD), multicopy COI1, JAZ, and MYC2 genes; high genetic plasticity	diploid with segmental duplications of JA/ET clusters; stable epigenetic regulators	these differences reflect an evolutionary divergence between a reactive (grasses) and an adaptive–systemic (asterids) model of immunity
Type of JA/ET-dominant response	local, short-term, energy-dependent defensive response of the "reactive immunity" type	systemic, long-term, autophagy-regulated tolerance of the "systemic immunity" type	wheat– a rapid "fight response"; sunflower—a stable "tolerance configuration."

The role of autophagy as an integrator of the immune response

Autophagy is a key mechanism of cellular self-regulation that ensures the removal of damaged organelles, protein aggregates, and the maintenance of energy homeostasis. According to our proposed integrative model of plant defense against pathogens and stresses, within JA/ET-dominant immunity, autophagy performs not only a degradative function but also acts as a systemic integrator of ROS signals, hormonal cascades, and programmed cell death (PCD), thereby determining cell fate – death or recovery.

Following herbivore-induced damage, local autophagy is activated in regions with elevated ROS levels. Autophagosomes engulf damaged chloroplasts and mitochondria and transport them to the vacuole, where nutrient recycling – including amino acids, lipids, and sugars – takes place. This process keeps the cell within an adaptive response window, preventing uncontrolled PCD while sustaining basal metabolism.

As JA/ET signaling intensifies, the autophagic flux spreads systemically, supporting long-term tolerance at the whole-plant level, analogous to Induced Systemic Resistance (ISR). This transformation from local recycling to a signaling module involves ATG8, ATG18, and ATG5 proteins, which interact with the COI1–JAZ–MYC and EIN2–ERF cascades, as well as with the energy sensors SnRK1 and TOR.

ROS and NO signals modulate autophagic activity: the former initiate the process, while the latter fine-tune its intensity via S-nitrosylation of ATG proteins. Under energy deficiency, SnRK1 activates autophagy, whereas TOR suppresses it. In a moderate regime, autophagy reduces ROS to sublethal levels, thereby shifting the response from cell death toward recovery. Thus, ROS ↔ autophagy ↔ SnRK1/TOR ↔ PCD form an interconnected regulatory loop that coordinates the cellular stress response.

The JA/ET-dominant immune configuration implements a spatially segregated strategy: PCD predominates at the damage site, whereas autophagy-mediated tolerance is activated in peripheral tissues. In wheat, this process is localized and short-lived, while in sunflower it is systemic and long-lasting, consistent with an evolutionarily established ISR-based JA/ET immunity model (Baena-González & Hanson, 2017; Marshall & Vierstra, 2018; Soto-Burgos et al., 2018).

Thus, autophagy acts as a molecular interface between local cellular sacrifice and systemic tolerance, converting destructive signals into a controlled survival program.

ROS homeostasis and antioxidant control

Reactive oxygen species (ROS) are among the earliest and most important secondary messengers in the JA/ET-dominant immune response. They play a dual role: on the one hand, they act as damage signals that initiate local defense reactions; on the other hand, they function as regulators of tolerance, contributing to the establishment of systemic adaptation and stress "memory".

In response to herbivore damage, a rapid local ROS burst is triggered, generated by membrane-bound NADPH oxidases of the RBOH (Respiratory Burst Oxidase Homolog) family. The activity of RBOHD/F proteins leads to the production of superoxide (O_2^-), which is rapidly converted into hydrogen peroxide (H_2O_2) – a key diffusible signal transmitted to neighboring cells. In wheat, the ROS burst is impulsive and localized, whereas in sunflower, a systemic ROS wave is observed, consistent with its JA/ET-oriented ISR response.

The ROS wave propagates through plasmodesmata and the apoplast, accompanied by the activation of Ca^{2+} channels and the initiation of MAPK cascades (MPK3, MPK6, MPK4), which directly regulate the transcription of JA/ET-dependent genes (COI1, JAZ, MYC2, ERF1) (Liu et al., 2016; Hu et al., 2020).

To avoid the toxic effects of excessive ROS, plant cells activate an antioxidant network that maintains a homeostatic balance between signaling and damaging ROS levels. This network includes the following enzymes: SOD (Superoxide Dismutase), which converts superoxide into H_2O_2; CAT (Catalase), which decomposes H_2O_2 in peroxisomes; and APX (Ascorbate Peroxidase), which reduces H_2O_2 levels in chloroplasts and the cytosol (Lee, 2020).

JA/ET signaling regulates the expression of these enzymes via MYC2 and ERF1/ORA59, establishing a feedback loop between hormonal activation and redox homeostasis. In wheat, the antioxidant system responds rapidly but transiently, whereas in sunflower it is maintained for longer periods, ensuring systemic ROS stabilization, a prerequisite for ISR formation.

Alongside ROS, nitric oxide (NO) acts as an important signaling coregulator of the JA/ET response. NO participates in the modification of ATG proteins via S-nitrosylation, thereby regulating the intensity of autophagy; in interactions with H_2O_2, leading to the formation of peroxynitrite ($ONOO^-$), which affects antioxidant enzyme activity; and in the regulation of PCD through nitrosylation of caspase-like proteases. Nitric oxide (NO) acts as a “fine boundary regulator” between cellular protection and self-destruction. In JA/ET-dominant states, it enables flexible control of the autophagic loop, keeping cells in a mode of adaptive tolerance rather than complete death.

Reactive oxygen species (ROS) play a key role in the formation of plant immune memory (stress memory). Repeated damage or infection leads to faster and stronger activation of antioxidant enzymes and JA/ET genes – a phenomenon known as priming. At the molecular level, this is associated with epigenetic modifications of the promoters of antioxidant genes (CAT, APX) and JA/ET regulators (MYC2, ERF1), which maintain chromatin in an “open” state (Rao et al., 2025).

Thus, ROS not only initiates the primary response but also ensures adaptive stability and epigenetic readiness for future stresses.

Effector level: secondary metabolites and volatile signals

At the effector level, the JA/ET-dominant immune response is transformed into biochemical mechanisms of stress containment and compensation. This involves activation of the synthesis of secondary metabolites, antimicrobial and anti-insect compounds, protease inhibitors, chitinases, phenylpropanoids, and volatile organic compounds (VOCs), which provide both local defense and systemic intercellular communication (Barbaś et al., 2022).

JA/ET signaling activates enzyme cascades, including PAL–C4H–4CL–CHS, which regulate the biosynthesis of phytoalexins and antioxidants. Protease inhibitors (PR6, PIN2) suppress insect digestive enzymes; chitinases and β-1,3-glucanases degrade the outer structures of pathogens; and phenylpropanoids reinforce cell walls and reduce oxidative stress. Terpenoids and GLVs (green leaf volatiles), under the control of MYC2 and lipoxygenases (LOX), function as alarm signals and chemical repellents. Methyl jasmonate (MeJA), a volatile analog of JA, spreads through the air or via the phloem, activating defense responses in distant organs.

VOCs also play a role in inter-plant communication, inducing priming and increased sensitivity to pathogens in neighboring plants. Some of these compounds (terpenes, phenols) attract natural enemies of phytophagous insects – parasitic wasps, mites, and beetles – thereby forming an ecological feedback mechanism (Kutty & Mishra, 2023).

This type of signaling interaction is a key element of the systemic level of JA/ET immunity, which provides protection not only to individual cells but to the entire phytocoenosis (Table 7).

Table 7
Comparison of local (PCD) and systemic (ISR) effects

Response level	Biological nature	Main manifestations	Energy profile
Local response (PCD)	programmed cell death in the damaged area	ROS burst, membrane depolarization, accumulation of phenolics, and PIs	high energy costs, short-term action
Systemic response (ISR)	JA/ET-dependent tolerance and metabolic stabilization	activation of antioxidants, autophagy, secondary metabolites, and VOCs	low energy costs, long-term action

Thus, the JA/ET-dominant immune system coordinates the coexistence of two response levels: local programmed cell death (PCD) to limit damage, and systemic induced systemic resistance (ISR) to maintain organismal viability. In wheat, a rapid local type of effector response predominates, whereas sunflower exhibits sustained systemic production of terpenes and phenylpropanoids, characteristic of prolonged JA/ET activation.

Systemic integration and the role of the microbiome

The immune response of plants is not limited to local reactions in the damaged area. At the whole-organism level, it transforms into systemic resistance, which encompasses all organs, tissues, and even the phytosphere – the community of microorganisms that coexist with the plant. In the JA/ET-dominant mode, Induced Systemic Resistance (ISR) is the main manifestation of long-term adaptation, with the microbiome acting as a key modulating factor in this process.

Induced Systemic Resistance (ISR) refers to a state of a “primed” immune system that develops in response to mild or localized stimuli, such as herbivore attacks or the action of root symbionts. Unlike Effector-Triggered Immunity (ETI), ISR is not accompanied by a hypersensitive response or massive cell death. Instead, it is based on metabolic reprogramming, hormonal adjustments, and reduced energy expenditure for defense.

JA/ET signaling in this case promotes the transition of cells into a “metabolically primed” state, in which defense genes (PR, PI, LOX, PAL) are activated much more rapidly upon recurring stress. This mode allows plants to effectively endure combined biotic and abiotic challenges – such as drought, heat, and pest damage – without depleting their energy reserves. Such reprogramming has also been observed during pathogenic infections, where JA/ET homeostasis becomes a key factor in establishing systemic resistance (Guliaeva, 2024).

The rhizosphere and phyllosphere microbiomes act as active partners of the plant immune system. Among their components, Plant Growth-Promoting Rhizobacteria (PGPR) and endophytes are of particular importance, as they can induce JA/ET-dependent defense responses without direct pathogenicity. Microorganisms such as *Pseudomonas fluorescens*, *Bacillus subtilis*, and *Trichoderma harzianum* release volatile compounds (e.g., acetoin, 2,3-butanediol), peptides, and lipopeptides that trigger JA/ET signaling pathways in root cells (Patyka et al., 2025b).

These signals activate transcription factors such as MYC2, ERF1, and ORA59, enhance the expression of JA/ET-induced genes (e.g., LOX, AOS, PDF1.2, PR6), and increase autophagy activity and ROS homeostasis in root tissues, thereby creating a metabolic “reserve” for future responses.

Thus, the microbiome functions as a biological primer, preparing the JA/ET system for a rapid and well-balanced response to herbivores. ISR represents a multi-organ form of immune coordination, in which signals originating in the roots are transmitted to aboveground organs. This process is mediated by: transport of JA, MeJA, and ET hormones via the phloem; electrical signals and ROS waves spreading between cells; and volatile compounds that convey information through the air or between leaves.

Thanks to this interaction, leaf damage can activate root symbionts, while an active microbiome can enhance the resistance of aerial parts. As a result, the JA/ET–ISR network forms an integrated regulatory system: "microbiome ↔ JA/ET ↔ ISR ↔ tolerance", which maintains homeostasis even under stress conditions.

Epigenetic control and immune response memory

The JA/ET-dominant branch of the immune network not only provides a short-term defense response to herbivore attack, but also forms a long-term "stress memory" – the plant's ability to respond more rapidly and robustly to repeated damage. This effect is based on epigenetic regulation, which includes DNA methylation, histone modifications, and non-coding RNAs that stably alter the expression of immune genes without changing the DNA sequence. This leads to the formation of epigenetic priming, which ensures the metabolic preparedness of cells for recurrent stress. This concept aligns with the findings of Parker (2022), who highlighted the role of epigenetic mechanisms in shaping plant immune memory.

During the induction of JA/ET immunity, chromatin reprogramming occurs in the regulatory regions of key genes such as COI1, JAZ, MYC2, ERF1, and ORA59. The main mechanisms involved in this process are as follows: Demethylation of JA/ET-regulated gene promoters by enzymes such as ROS1 and DML2, which facilitates the binding of transcription factors MYC2 and ERF1; Histone acetylation (e.g., H3K9ac, H3K27ac), which promotes chromatin opening and maintains gene activity even after the stressor is removed ; Regulation by non-coding RNAs (miRNAs, siRNAs) that modulate JA/ET signaling at the post-transcriptional level (Banihashemian et al., 2025). For example, miR319 and miR396 repress the expression of negative regulators in the JA pathway, while miR393 is involved in balancing JA/SA signaling, determining the switch between anti-pathogenic and anti-insect types of responses. Thus, JA/ET priming represents a molecular form of memory that ensures effective immune responses even weeks after the initial attack (Reimer-Michalski & Conrath, 2016).

During repeated herbivore attacks, plants exhibit an enhanced and accelerated JA/ET response, driven by stable epigenetic marks. This phenomenon is known as stress memory or "immune memory". Key manifestations of this state include: a faster accumulation of ROS and NO upon subsequent stress, increased expression of LOX, AOS, JAZ, and ERF1 compared to the primary damage, and the active production of protease inhibitors and secondary metabolites, even in the absence of a direct herbivore threat (Lämke & Bäurle, 2017). At the cellular level, this memory is supported by persistent autophagy priming: after the initial attack, cells retain part of the ATG complexes in a semi-active state, allowing the immediate activation of autophagy in response to subsequent signals. Therefore, epigenetic and autophagic mechanisms are interconnected, forming a multi-layered stability circuit that maintains a robust JA/ET immune response.

Recent studies have demonstrated that epigenetic changes in the JA/ET system can be transmitted across generations. In wheat, sunflower, and Arabidopsis, a phenomenon known as "transgenerational inheritance of resistance" has been observed: the offspring of plants exposed to herbivores show elevated expression of JA/ET-related genes and enhanced tolerance (Wang et al., 2023). The main mechanisms underlying this inheritance include: Methylation of promoters of JA/ET regulators (COI1, MYC2, ERF1) in seeds; Persistence of siRNAs in embryo cells that retain stress-related information; Vegetative transmission of methylation patterns in cloned or vegetatively propagated forms (e.g., in sunflower via cuttings or meristem tissue). Thus, the JA/ET-dominant immune system not only coordinates short-term responses but also establishes a heritable memory of stress that enhances evolutionary resilience of plant populations and promotes the development of adaptive tolerance phenotypes.

The concept of a multistable immune-stress network in the integrative model of plant immunity

The general concept of the integrative model of plant immunity is based on the idea of a multistable signaling-metabolic network that coordinates plant responses to arthropod herbivores, pathogens, and abiotic factors. The immune and stress architecture of the plant is a dynamic system, capable of existing in multiple stable states that define the balance or trade-offs between growth and defense. These states can be broadly classified into the following categories: Growth-dominant mode, SA-dominant state (activation of systemic acquired resistance), JA/ET-dominant state (induction of resistance associated with the microbiome and necrotrophs), and ABA-dominant state, which coordinates responses to abiotic stresses. The signaling-metabolic network integrates local and systemic signals, linking biotic and abiotic inputs with the cellular metabolic state, resulting in an adaptive, self-regulating, and flexible system. Functional modules (e.g., ROS signaling, hormone cascades, autophagy) act as autonomous blocks that interact through integration hubs, which direct the flow of information. Interaction between modules and hubs occurs through signaling cascades and secondary messengers, forming a dynamic multistable network with nonlinear regulatory logic.

The central core and switchboard of the system is autophagy, which acts as a crossroad for signals between functional modules – including ROS/antioxidant systems, the cytoskeleton, the cell wall, PRR signaling, and hormonal pathways. Autophagy is also connected to integration hubs such as the TOR pathway and the "outer regulatory ring", which encompasses epigenetic mechanisms, metabolic status, the microbiome, photoperiod, and endoplasmic reticulum (ER) stress.

Threshold logic determines the activation conditions of individual network hubs based on the intensity of incoming signals. When a signal exceeds a certain threshold, the system switches into a corresponding stable state. Thus, the immune-stress network functions not linearly, but contextually – responding to a combination of external stimuli and the cell's internal physiological state. This concept demonstrates that plant adaptation to stress is the result of dynamic multistability within the network, which enables flexible balancing between growth, defense, and metabolic stability.

The activation of key hubs and modules in the immune-stress network follows threshold-based principles, which determine the transition point of the cell between alternative stable states. Depending on the intensity and duration of the signal, the network responds via different pathways. An energy deficit (↑SnRK1/↓TOR) triggers autophagy and the redistribution of metabolic resources. Sustained accumulation of NO and ROS leads to the activation of the hypersensitive response (HR) or programmed cell death (PCD).

Increased aquaporin (AQP) activity promotes the systemic transmission of ROS signals between cells. Cell wall integrity disruption initiates DAMP-dependent activation of the PTI pathway. Circadian fluctuations, especially during evening phases, shift the balance between SA and JA activity. The presence of the microbiome (ISR) results in JA priming and a reduction in energy costs required for maintaining defense responses (Table 8).

This "threshold" or "switch" logic integrates signals of various origins, activating only those modules whose intensity or energy level exceeds a critical threshold. Thanks to this mechanism, the network maintains adaptive multistability – the ability to switch between growth, survival, and defense states: Mild stress → activation of PRRs, CWI, and a moderate ROS response → survival and immune system priming. Moderate stress → engagement of the SnRK1–TOR pathway, autophagy, and hormonal regulation → balance between growth and defense. Severe stress or infection → sustained ↑ROS + ↑NO, JA/ET dominance → HR/PCD or mega-autophagy. Threshold logic ensures the nonlinear behavior of the immune network, where the outcome is determined not only by signal strength, but also by the metabolic and epigenetic context of the cell.

TOR kinase functions as the central sensor of the cell's energy and nutrient status. Under optimal conditions, it remains active, promoting cell growth, biosynthetic processes, and suppressing autophagy. In response to stress or resource deficiency – particularly during infection, drought, or nitrogen and carbohydrate shortages – TOR becomes inactivated. At this point, the SnRK1 and GCN2 pathways are activated, signaling energy depletion. This releases the inhibition of autophagy and shifts the cell into an energy-conservation mode. Thus, TOR serves as the key molecular switch between growth programs and the defense response.

Table 8
Threshold logic (“Switch Logic”) of immune-stress node activation in plant cells

№	Trigger / Signal	Activation condition (threshold)	System response / outcome
1	↑SnRK1 / ↓TOR	energy deficit, decreased metabolic energy (ATP)	activation of autophagy, redistribution of resources
2	↑NO + ↑ROS	prolonged accumulation of reactive nitrogen and oxygen species	initiation of HR (hypersensitive response) or PCD (programmed cell death)
3	↑AQP	increased aquaporin activity, enhanced water and signal conductivity	systemic transmission of ROS signals between cells
4	CWI defect	cell wall damage, release of DAMP signals	activation of PTI (PAMP/DAMP-triggered immunity)
5	Circadian phase (evening)	diurnal rhythm, fluctuations in transcriptional activity	shift in SA/JA balance; activation of JA-dependent pathways
6	ISR	presence of beneficial microbiome, low energy expenditure	JA priming and formation of systemic tolerance

The outer regulatory ring includes epigenetic mechanisms, metabolic pathways, the microbiome, photoperiodic and circadian rhythms, endoplasmic reticulum (ER) stress, and other abiotic environmental factors (see Fig. 3). Epigenetic regulation (DNA methylation, histone modifications, and the action of non-coding RNAs – miRNA, siRNA) determines the expression level of immune-related genes, forms stress memory, and enables faster and stronger responses upon repeated infection. It also controls the transcription of autophagy-related ATG genes, PRR receptors, and antioxidant enzymes.

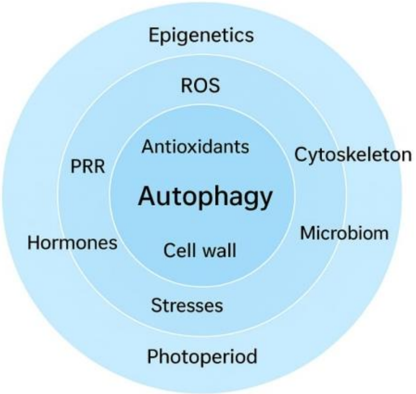

Fig. 3. Plant immunity is represented as a concentric model of multilevel regulation of cellular defense and survival: the central core includes autophagy as a key mechanism for maintaining homeostasis and integrating stress signals; the middle ring illustrates major intracellular signaling and structural components: ROS, antioxidants, cytoskeleton, cell wall, hormones, and PRRs; the outer ring reflects systemic and external regulators: epigenetics, metabolism, the TOR pathway, microbiome, photoperiod, endoplasmic reticulum (ER), and other stress-related factors

Metabolic regulation is closely linked to the activity of the TOR/SnRK1 complexes and determines the allocation of carbon and nitrogen resources between growth and defense processes. It includes the synthesis of phenylpropanoids, flavonoids, lignin, and other secondary metabolites involved in plant defense. Metabolic sensors SnRK1 and GCN2 respond to energy deficits by activating autophagy, which recycles damaged proteins and organelles.

The microbiome (especially PGPR and mycorrhizal fungi) functions as an external module of the plant immune system, triggering Induced Systemic Resistance (ISR) through JA/ET signaling, ROS metabolism, and antioxidant pathways. The microbiota can enhance or modulate resistance, adapting it to specific soil environment conditions.

Photoperiodic and circadian rhythms regulate the daily dynamics of immune activity: a significant portion of genes encoding PRR receptors, ROS-metabolism enzymes, and antioxidant systems are under circadian control. During the dark phase, SA-dependent immunity dominates, which is effective against biotrophic pathogens, while in the light phase, JA/ET pathways are activated, targeting necrotrophs. This daily coordination optimizes energy expenditure between growth and defense. Endoplasmic reticulum (ER) stress arises during infections or abiotic damage as a result of the accumulation of misfolded proteins. In response, the unfolded protein response (UPR) is activated – an adaptive ER mechanism aimed at reducing protein load and restoring protein homeostasis. If this response is insufficient, autophagy or programmed cell death (PCD) is initiated. Because the ER is responsible for the synthesis and maturation of PRR receptors and numerous defense-related proteins, its functional state directly affects the efficiency of immune responses. Additionally, environmental factors such as temperature, humidity, osmotic stress, and mechanical damage influence immunity through hormonal and metabolic signaling pathways.

Taken together, the outer regulatory ring functions as a fine-tuning layer over the core immune network, adapting it to specific environmental conditions. Among these factors, TOR kinase and ER stress exert the most direct control over autophagy and PCD, whereas other factors act primarily through indirect regulatory mechanisms (Table 9).

The JA/ET module is the basis of the systemic immune response of plants to herbivores

The levels of plant immune response to herbivore damage discussed above indicate that the plant's defense system functions not as a set of isolated reactions, but as a unified, multi-level signal-metabolic network with various defensive states. Within this network, the JA/ET-dominant branch serves as a non-specific adaptive mode that integrates local stress signals, hormonal coordination, autophagy, and epigenetic memory into a cohesive mechanism of energy-efficient immunity.

The immune response unfolds according to a universal scheme that includes six interconnected levels:

– damage sensing (DAMPs, HAMPs, mechanoreceptors) → Detection of cell wall damage, activation of WAK, FERONIA, PIEZO, changes in membrane potential;

– ROS/NO signaling → Initiation of local ROS burst (RBOHD/F), activation of MAPK cascades, involvement of NO in autophagy modulation;

– JA/ET hormonal axis → Activation of the COI1–JAZ–MYC2 and ERF1/ORA59 circuits; formation of an “insect mode” in a multistable immune system;

– autophagic regulation → Control of energy flows, degradation of damaged organelles, balancing PCD and tolerance;

– ISR (Induced Systemic Resistance) → Development of systemic tolerance, involvement of the microbiome, stabilization of antioxidant and hormonal balance;

– epigenetic memory → Methylation, acetylation, and miRNA-based regulation of JA/ET pathways; formation of “stress memory” and its transmission through seeds (Table 10).

These levels function in an interconnected manner, forming a closed system of positive and negative feedback loops that maintain the plant’s immune network in a state of functional stability, even under multifactorial environmental pressure. The JA/ET-dominant configuration represents an energy-optimized type of immune response, aimed not at eliminating the herbivore at any cost, but at preserving the structural integrity and homeostasis of the plant. In contrast to the SA-dominant (hypersensitive) mode, the JA/ET branch minimizes cell loss and ensures metabolic tolerance through: maintaining ROS homeostasis and antioxidant control; an autophagic buffer that prevents energy collapse; an ISR module that stabilizes systemic readiness; and epigenetic memory that enhances intergenerational adaptation.

Thus, the JA/ET-dominant system represents a higher level of plant immune evolution, combining rapid local response, systemic regulation, and long-term stability over time.

Table 9
The TOR pathway and the "outer ring" of plant immune response regulators

Regulator	Mechanism of action	Impact on immunity	Key molecules / examples	Type of stress	Link to autophagy / PCD
TOR pathway	TOR kinase is active under favorable conditions: it promotes growth, protein biosynthesis, and inhibits autophagy. Under energy or nutrient deficiency, TOR becomes inactivated, while SnRK1 and GCN2 are activated.	it switches the cell between growth and a protective, energy-saving mode	*TOR*, *SnRK1*, *GCN2*, *ATG* genes	energy deficiency, nitrogen starvation, infection, drought	direct regulation of autophagy: TOR inhibition → autophagy activation
Epigenetics	DNA methylation, histone modifications, and non-coding RNAs regulate the expression of immune genes and the ATG complex	they contribute to stress memory formation and enable a faster secondary response	key components: DNA methyltransferases, H3K9me2, miR393	biotic and abiotic stresses	epigenetic control of autophagy and PCD, influence on the duration and intensity of the response
Metabolism	balances carbon and nitrogen fluxes; activates the synthesis of phenylpropanoids, flavonoids, and lignin. SnRK1 and GCN2 initiate autophagy under energy-deficient conditions	enhances defense responses and promotes the accumulation of secondary metabolites	PAL, SnRK1, GCN2	energy or nutrient deficiency	autophagy is activated to degrade damaged proteins and organelles
Microbiome	PGPR and mycorrhizal fungi activate induced systemic resistance (ISR) through JA/ET and antioxidant pathways	enhances pathogen resistance and reduces the energy cost of the immune response	*Pseudomonas*, *Bacillus*, *Glomus*	biotic interactions	indirect induction of autophagy via JA/ET and ROS signaling
Photoperiod and circadian rhythms	diurnal rhythms regulate the activity of SA and JA/ET pathways: SA dominates at night, JA/ET during the day	aligns immune responses with daily phases and environmental conditions	CCA1, LHY, TOC1	light changes and seasonality	circadian regulation of ATG genes, ROS-dependent PCD
Endoplasmic Reticulum (ER) Stress	accumulation of misfolded proteins activates the UPR; under severe stress, triggers autophagy or PCD	determines the functionality of PRR receptors and defense-related proteins	BiP, bZIP60, IRE1	infection, heat or osmotic stress	directly triggers autophagy and PCD in cases of irreversible damage.
Other environmental factors	temperature, humidity, and mechanical damage are integrated through hormonal and metabolic cascades	modulate the strength of the immune response and ROS homeostasis	key components: HSP70 (heat stress), RD29A (osmotic stress)	abiotic stresses	through ROS and hormonal signaling, they may induce autophagy or PCD

Table 10
Levels and modules of JA/ET-mediated defense in the integrative model of immunity under herbivore damage

Level/model module	Plant defense mechanisms against mites and insects	How exactly are they integrated into the model of plant immunity
Sensing and primary signals	Mechanical damage from insect bites activates mechanosensors (WAK, THESEUS1, PIEZO), leading to the formation of DAMP and HAMP signals (insect peptides, enzymes, fatty acids), which are recognized by PRR receptors	This corresponds to the primary Ca^{2+}–ROS–MAPK signaling modules. Bites are perceived as biotic DAMPs, which instantly trigger immune signaling
ROS/NO and the antioxidant system	Damage stimulates a ROS burst (via NADPH oxidase RBOHD/F) and NO signaling, which coordinates local oxidative waves. Enzymes such as SOD, APX, CAT, and GPX are activated to limit excessive ROS accumulation	This represents a direct analogue of the antioxidant module in the model – ROS act as danger signals, but autophagy and antioxidant responses stabilize redox homeostasis, preventing necrosis
Autophagy and energy sensors (SnRK1/TOR)	In the damaged area, selective and macroautophagy are induced – damaged organelles are recycled, and energy balance is maintained	This corresponds to the central integrator of the model – autophagy acts as a stress buffer: it coordinates JA/ET and ROS responses, preventing excessive cell death
Hormonal pathways (SA, JA/ET, ABA)	Insect bites stimulate jasmonic acid (JA) and ethylene (ET), which activate genes encoding proteinase inhibitors (PIs), chitinases, and phenylpropanoids. In parallel, abscisic acid (ABA) modulates stomatal immunity	This reflects a JA/ET-dominant state of a multistable system – a typical response to necrotrophic and insect-derived stress. The system shifts into a defensive mode with enhanced synthesis of secondary metabolites
Cytoskeleton and cell wall (CWI Sensors)	Reorganization of actin filaments and microtubules, enhanced lignification, and callose deposition at the bite sites occur. A physical barrier is formed to prevent further damage	This fully corresponds to the cytoskeleton–barrier module of your model: mechanical reinforcement of the cell wall is integrated with CWI signaling (WAK/FERONIA)
Metabolic adaptation (Secondary Compounds)	Induction of the phenylpropanoid and terpenoid pathways leads to the synthesis of volatile GLVs, MeJA, and terpenes, which repel herbivores and attract their natural enemies	This corresponds to the effector level of the model – chemical defense and interplant signaling (VOCs), analogous to PR proteins in PTI/ETI
Epigenetics and memory	Repeated attacks induce epigenetic priming of JA/ET pathways (promoter methylation, activation of miRNAs and siRNAs)	This represents the epigenetic module of the model – immunological memory (ISR) against herbivores is formed, enabling a faster response to recurring stress
Organismal-systemic level (SAR/ISR, microbiome)	Volatile compounds (MeJA, GLVs) and signals from the root microbiome activate Induced Systemic Resistance in distal tissues	This corresponds to the systemic integration level of the model: JA/ET-dependent ISR represents the insect-related form of systemic resistance, coordinated with microbiome activity and hormonal networks

Overall, plant damage caused by phytophagous organisms is integrated into the proposed model as a JA/ET-dominant mode of immune regulation, characteristic of responses to necrotrophic and wounding stressors. Primary cell wall damage during herbivore feeding activates mechanosensors (WAK, THESEUS1, FERONIA) and generates DAMP and HAMP signals (herbivore-associated molecular patterns), which are recognized by pattern recognition receptors (PRRs). This triggers a rapid Ca^{2+}–ROS–MAPK cascade that initiates a local oxidative burst and the synthesis of jasmonic acid (JA) and ethylene (ET) hormones.

Subsequent response coordination is mediated through autophagy, which serves as a central integrator and regulator of redox homeostasis, preventing excessive cell death and ensuring the removal of damaged organelles. Concurrently, the ROS/NO system generates wave-like signals that coordinate the activation of antioxidant enzymes (SOD, APX, CAT) and protective metabolites. At the effector level, the biosynthesis of secondary compounds – phenylpropanoids, terpenes, volatile organic compounds (GLV, MeJA), protease inhibitors, and chitinases – is enhanced, serving barrier, signaling, and anti-feeding functions.

This branch is responsible for non-specific, systemic, and energy-regulated resistance of the ISR (Induced Systemic Resistance) type, within which local programmed cell death (PCD) programs are combined with systemic tolerance, tissue recovery, and regeneration. The interaction of JA/ET signaling pathways with the autophagic loop and the ROS-antioxidant system enables flexible balancing between localized cell loss and the maintenance of whole-organism homeostasis.

Thus, insect-induced damage is viewed as an integrated component of a multistable signaling-metabolic network, where hormonal, autophagic, and oxidative mechanisms act in concert to preserve plant integrity and activate systemic immune potential.

Prospects and applications

The JA/ET-dominant mode of immune regulation in the integrative model of plant immunity opens new horizons for developing resilient, energy-efficient, and ecologically balanced strategies for crop protection. Its systemic nature not only allows a deeper understanding of the fundamental principles of biological resistance but also provides tools for targeted management of natural plant defenses through biotechnological, breeding, and agroecological approaches. The JA/ET signaling branch and Induced Systemic Resistance (ISR) can serve as the foundation for a new generation of strategies to enhance crop resistance against mites and insects.

Key directions for practical application include: breeding of cultivars and hybrids with enhanced JA/ET activity; identification of alleles of genes such as COI1, JAZ, MYC2, ERF1, and ORA59, and their use as genetic markers of natural insect tolerance in wheat, sunflower, soybean, and canola ; next-generation phytohormonal elicitors ; application of low doses of methyl jasmonate (MeJA), ethylene precursors, and synthetic ISR activators to prime the immune system without phytotoxic effects; integration with agroecological technologies. JA/ET-based protection can serve as the basis for biocontrol systems, combining natural microbial inoculants, tolerance inducers, and reduced pesticide use.

Microbial symbionts of the rhizosphere, phyllosphere, and endosphere act as "natural regulators" of JA/ET pathways, contributing to the resilience and ecological stability of agroecosystems. PGPR (*Pseudomonas*, *Bacillus*, *Azospirillum*), mycorrhizal fungi (*Rhizophagus*, *Trichoderma*), and yeasts (*Metschnikowia*, *Hanseniaspora*) stimulate the JA/ET response and enhance Induced Systemic Resistance. This opens up several promising avenues for application: development of biocontrol consortia that not only suppress pests but also induce natural resistance in crops; implementation of ecological farming technologies, where the microbiome functions as an integral part of the plant immune system; creation of "living bio-elicitors", capable of forming a stable JA/ET immune memory in field-grown crops.

The further development of the JA/ET-dominant immunity concept is closely linked to the application of modern multi-omics and systems biology approaches, enabling the integration of knowledge from the molecular to the population level. Key research directions include: identification of complete JA/ET-dependent signatures in wheat, sunflower, and other crops under different types of herbivore damage; analysis of genes such as ATG8, TOR, SnRK1, DCL, and AGO as potential markers of long-term tolerance and systemic stability; development of dynamic models describing the multistable states of the plant immune system, including energy flows, ROS homeostasis, and microbiome interactions; investigation of the evolution of COI1–JAZ–MYC and ERF modules in grasses and oilseed species to identify conserved and adaptive elements of insect resistance.

Understanding the JA/ET-dominant branch as a natural adaptive immune mode in plants lays the foundation for: targeted breeding of tolerant cultivars, optimization of nutrition and protection through endophytic consortia, reduction in pesticide dependence, and increased resilience of agroecosystems to climate change.

Thus, the integration of JA/ET mechanisms, autophagy, ROS homeostasis, and ISR into modern agricultural practices may represent a new paradigm of sustainable plant protection – one that relies on natural resistance mechanisms rather than chemical intervention. This approach opens new opportunities for bioengineering and the development of resilient agriculture in the face of a changing climate.

Conclusions

JA/ET-mediated immunity represents a key adaptive branch of the integrative model of the plant immune network, coordinating responses to phytophagous damage through the integration of local signaling, systemic tolerance, and epigenetic memory. Its architecture combines sensory, hormonal, autophagic, antioxidant, and metabolic modules that function as a single dynamic system for maintaining homeostasis. The JA/ET-dominant mode provides an energetically balanced defense in which the primary goal is not the elimination of the phytophage but the preservation of the plant's functional integrity. Induced systemic resistance (ISR) and the microbiome act as important regulators of this process, transforming local damage into systemic adaptation. Understanding the principles underlying the JA/ET model opens prospects for developing cultivars with natural tolerance, applying biocontrol microorganisms, advancing ecological agriculture, and shaping a new bioenergetic paradigm of plant immunity.

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