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# Integrative model of plant immunity to pathogens and stresses: A multilevel signal-metabolic network

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The study presents an innovative integrative model of plant immune architecture, based on the utilization of autophagy as a key regulator of multilevel immune cascades. The proposed approach establishes the foundation for a new practical paradigm in crop science – "immune management," which enables targeted regulation of the balance between autophagy, reactive oxygen species (ROS), hormonal signaling, and regenerative processes. The concept of autophagic priming provides the basis for preventive immunity, substantially enhancing both the speed and efficiency of crop adaptation to diverse pathogens and stress factors. The practical significance of this model lies in its potential for the development of immunomodulatory bioproducts, the breeding of cultivars with broad-spectrum stress resistance, and the design of flexible agro-technological schemes aimed at yield stability and reduced reliance on chemical crop protection. The integration of biological inducers, microbiome-based products, and precision farming technologies paves the way for next-generation agricultural systems capable of ensuring sustainable production under variable climatic conditions. The cumulative economic effect of implementing autophagic priming and fourth-generation bioproducts worldwide is estimated at 15–25 billion USD annually, highlighting their potential as a key driver of global transformation in the crop protection market.

*Keywords*: autophagy; immune management; autophagic priming; plant stress resistance; bioproducts; microbiome; integrative model.

## Introduction

Modern agriculture faces a dual challenge: population growth demands an increase in food production, while biotic and abiotic factors (plant diseases, climate change) exert mounting pressure on crop yields. Therefore, a deeper understanding of plant immunity mechanisms has both fundamental and applied significance for the development of sustainable strategies in agriculture (Son & Park, 2022; Jones et al., 2024; He et al., 2024).

Plants, being sessile organisms, cannot escape the impact of biotic agents (bacteria, fungi, viruses, parasites) and abiotic stresses (drought, extreme temperatures, salinity). Their ability to cope with these challenges relies on a sophisticated immune system that integrates receptor-mediated signal perception (PRRs, NLRs, etc.) with multilayered intracellular adaptive programs (Ngou et al., 2022; Abdul Aziz et al., 2025; Kashtoh et al., 2025).

The classical model encompasses two tiers of plant immune responses: PTI (pattern-triggered immunity, initiated by the recognition of MAMPs/DAMPs) and ETI (effector-triggered immunity, activated by pathogen effectors). However, these mechanisms are not isolated – instead, they are interconnected through complex signaling crosstalk, which enhances the overall efficiency of defense responses (Yuan et al., 2021; 2023).

Traditional immunity models – particularly the PTI (PAMP-triggered immunity) and ETI (effector-triggered immunity) concepts – capture the fundamental principles of pathogen recognition and defense activation but overlook the integrative nature of the interactions among numerous cellular and systemic mechanisms operating at different levels of plant organization. Intracellular processes play a pivotal role: ROS burst, $Ca^{2+}$ signaling, MAPK cascades, and hormonal regulation collectively constitute the core internal circuitry of plant defense (Ali et al., 2024; Galaud et al., 2025; Khablak et al., 2025).

Over the past decade, research has confirmed the pivotal role of autophagy not only in the removal of damaged cellular components but also in the regulation of ROS homeostasis, programmed cell death (PCD), and infection-related responses (Wang et al., 2021; Li et al., 2023; Yagyu et al., 2024; Haghpanah et al., 2025). Complementary functions of the cytoskeleton – including autophagosome trafficking and cell wall remodeling – together with the dynamic nature of the wall itself, modified through callose deposition, lignification, and ROS-mediated cross-linking, further contribute to plant defense (Dong et al., 2024; Khablak et al., 2025; Patyka et al., 2025). Phytohormones (SA, JA/ET, ABA, GA, cytokinins) orchestrate these processes, determining the strategic balance between local responses such as the hypersensitive response (HR) and systemic acquired resistance (SAR) (Zhao & Li, 2021; Movahedi et al., 2022; Leisner et al., 2023). Overall, the classical PTI/ETI framework does not adequately explain the remarkable plasticity of plant immune strategies. An integrative concept that encompasses PRR signaling, autophagy, the cytoskeleton, the cell wall, and hormonal coordination is still lacking.

The aim of this conceptual work is to address this gap by presenting a model that explains the coordination of local and systemic plant responses to stress, with a clear perspective for practical applications.

## Cytoskeleton, cell wall, autophagy, antioxidant systems, and hormonal regulation: An integrative model of plant defense against pathogens and stresses

Based on an analysis of current literature and our own research, we have formulated a conceptual integrative model that describes the interplay of autophagy, the cytoskeleton, the cell wall, antioxidant systems, and hormonal regulation in shaping multilevel plant defense strategies against biotic and abiotic factors. In the proposed model, these mechanisms do not operate in isolation but function as intercon-

nected modules within a unified defense network. A critical decision point in this system is the choice between cell survival and programmed cell death, determined by the balance of signaling pathways and the metabolic state. Table 1 summarizes the principal modules of the integrative model, their functions, and key signaling pathways, serving as a foundational "map" of the entire system.

**Table 1**
Core modules of the integrative model of plant immunity

| Module | Key functions | Major signaling pathways / components |
|---|---|---|
| Sensing (PRR, DAMP, PAMP) | primary recognition of pathogens and damage | PRRs (FLS2, EFR), PEP receptors, mechanosensors |
| Ion signaling | rapid signal transmission, kinase activation | $Ca^{2+}$ channels, CDPK, CNGC, TPC1 |
| ROS/NO system | secondary messengers, HR induction, wave signaling | RBOHD/F, NADPH oxidase, NO synthase-like activity |
| CWI sensors (cell wall integrity) | monitoring integrity, immune activation | WAK, THESEUS1, FERONIA |
| Autophagy | survival/PCD balance, degradation of pathogenic effectors | ATG proteins, selective and bulk autophagy |
| Antioxidant systems | ROS detoxification, maintenance of redox homeostasis | APX, CAT, SOD, GPX, glutathione |
| Energy sensors | coordination of metabolism and stress responses | SnRK1, TOR, GCN2 |
| Proteostasis | protein degradation, quality control | UPS (26S proteasome), ERAD, UPR |
| Cytoskeleton | autophagosome trafficking, organelle distribution, secretion | microtubules, actin filaments, motor proteins |
| Cell wall and trafficking | barrier reinforcement, secretion of PR proteins | callose deposition, lignification, vesicular transport |
| Lipid signaling | secondary messenger generation, regulation of autophagy | phosphoinositides, oxylipins, phospholipases |
| Stomatal immunity | control of pathogen entry | ABA, OST1 kinase, ROS/NO signaling |
| Systemic signals | SAR/ISR transmission, intercellular communication | ROS and $Ca^{2+}$ waves, plasmodesmata, NO |
| SAR/ISR | long-term systemic resistance | SA (NPR1, TGA), JA/ET (MYC2, ERF) |
| Microbiome | immunity support, ISR | Rhizobacteria, PGPR, signaling metabolites |
| Volatile organic compounds (VOCs) | plant-to-plant communication, systemic signaling | methyl salicylate, GLVs, terpenes |
| Hormonal networks | modulation of defense and growth | SA, JA, ET, ABA, IAA, CK, BR, GA |
| Epigenetics and RNA | "immune memory," transcriptional control | DNA methylation, H3K4/H3K27 modifications, siRNA, miRNA |
| Circadian rhythms | temporal coordination of sensitivity | CCA1, LHY, TOC1 |
| Final outcomes | HR/PCD, tolerance, SAR, regeneration | integration of all preceding pathways |

**Description of the modules of the integrative model**

The central module – autophagy and cell survival. The autophagy module is considered a key integrator of intracellular and extracellular stress signals. It mediates the degradation of damaged proteins and organelles, maintaining cellular homeostasis and energy balance. Depending on the severity of cellular damage, autophagy determines cell fate – either sustaining cellular function or triggering programmed cell death (PCD) to preserve the integrity of the organism.

*Intracellular signaling and ROS homeostasis module.* This module encompasses ROS, antioxidant systems, calcium fluxes, and key signaling kinases. ROS serve a dual function: acting as secondary messengers that coordinate immune responses and as localized cytotoxic agents that restrict pathogen spread. Antioxidant enzymes regulate ROS levels, preventing oxidative damage to cellular components.

*Cytoskeleton and cell wall module.* The cytoskeleton mediates targeted trafficking of autophagosomes, organelles, and defense molecules to infection sites. Concurrently, cell wall modifications – including lignification, callose deposition, and structural remodeling of pectins – reinforce the physical barrier, restricting pathogen spread.

*Hormonal regulation module.* Hormonal networks coordinate both local and systemic responses: SA is primarily associated with defense against biotrophic pathogens, JA/ET with resistance to necrotrophs and insect herbivores, while ABA integrates abiotic stress signals into the overall immune response. These hormonal pathways establish a flexible "balancing" system between growth and defense.

*Epigenetic regulation and metabolic adaptation module.* Epigenetic mechanisms – including DNA methylation, histone modifications, and non-coding RNAs – establish an "immune memory" that enables faster and stronger responses to recurring infections. Metabolic pathways for secondary metabolite synthesis provide an additional chemical barrier against pathogens and support the energetic efficiency of cellular responses.

*Systemic coordination and intercellular signaling module.* This module integrates local and systemic responses through mobile signals such as ROS, $Ca^{2+}$ waves, hormones, and volatile organic compounds. It mediates the activation of systemic acquired resistance (SAR) and induced systemic resistance (ISR), establishing a unified, multilevel defense network.

In summary, the proposed integrative model conceptualizes plant immunity as a dynamic signal–metabolic defense network organized into interacting functional modules. Its multilevel architecture balances local cell losses with the preservation of overall organismal integrity, optimizes the use of energetic resources, and enhances plant survival under complex biotic and abiotic challenges.

**Logical hierarchy of the plant immune system: Levels of integration**

Plant immunity is organized into a complex multilevel hierarchy that ensures effective coordination of signals from primary sensing to systemic responses and interactions with the environmental context. To explain this hierarchy, a structural approach is proposed, reflecting the gradual integration of processes from fundamental molecular mechanisms to the organismal and ecological scale.

In this model, autophagy is considered not merely as a catabolic mechanism for the removal of damaged cellular structures, but as a central integrator and coordinator linking immune responses with stress adaptation. It clears the cell of damaged organelles and proteins, regulates reactive oxygen species (ROS) levels in close interplay with antioxidant systems, and orchestrates the activation of programmed cell death (PCD) or tolerance mechanisms. Thus, autophagy emerges as a universal control hub for homeostasis, stress resilience, and the strategic decision of "survive or sacrifice the cell".

Other key components operate in close coordination with autophagy. The plant immune system is conceptualized as a multilevel, integrated network protecting the plant from pathogens and stressors, encompassing molecules, organelles, cells, tissues, organs, the whole organism, environmental interactions, temporal dynamics, and metabolism, functioning as a dynamic multistable system. Plant immunity acts as a dynamic, multicomponent stress-response network, in which autophagy, ROS–antioxidant systems, the cytoskeleton, the cell wall, and hormonal cascades interact in an integrated defense mode. The choice between cell survival and sacrifice is not random but follows the system's logic, aimed at ensuring the survival of the entire plant.

Within the proposed model, the plant immune response is described as a multilevel signal–metabolic defense network operating on the principle of hierarchical modularity. Autophagy serves as the central integrator, coordinating the interactions of sensory, signaling, and effector systems, while maintaining the balance between cell survival and programmed cell death.

To better understand the functioning of the integrative model of plant defense against biotic and abiotic stresses, the workflow of its key modules across different organizational levels of the plant is briefly outlined below. At the core of this network lies a multilevel defense structure, in which each module performs a specific role while

simultaneously adhering to the overarching logic of "ensuring the survival of the entire plant, even at the expense of individual cells".

*1. Sensing and primary signaling.* The first level involves the pathogen- and damage-recognition system. At this level, early detection of pathogens and stresses is mediated by pattern-recognition receptors (PRRs), which respond to microbe-associated molecular patterns (MAMPs) or damage-associated molecular patterns (DAMPs) from the plant's own cells. Activation of PRRs triggers rapid $Ca^{2+}$–ROS–MAPK signaling, initiating local defense mechanisms. Additional primary sensing is provided by cell wall integrity sensors (CWI, mechanosensors, PIEZO) and local ionic and peptide signals (PEP1–PEP7, $K^+/Cl^-$ fluxes, pH changes), integrating information on the physiological state of the cell and the external environment.

PRR signaling (FLS2, EFR, CERK1) initiates PTI by activating $Ca^{2+}$ fluctuations, ROS bursts, and MAPK cascades. $Ca^{2+}$ signaling is mediated by plasma membrane and tonoplast channels (CNGC, TPC1) and coordinates the activation of $Ca^{2+}$-dependent protein kinases. The ROS and NO system functions as a secondary messenger, triggering both local and systemic defense waves. CWI sensors (WAK, THESEUS1, MIK2) and mechanosensors monitor cell wall integrity and respond to DAMPs. Peptide signals (PEPs) and their receptors activate early immune cascades. Aquaporins regulate $H_2O_2$ transport, facilitating signal propagation. PTI–ETI integration is achieved through resistosome formation and activation of NLR proteins.

*2. Cellular response integrators.* The second level encompasses intracellular mechanisms that coordinate rapid and localized stress responses. Autophagy plays a central role by regulating the degradation of damaged proteins and organelles and maintaining energy homeostasis. The cytoskeleton serves as a transport and structural system for delivering signals and effectors to specific cellular sites. Proteostasis mechanisms (UPS/ERAD, UPR) ensure proper protein conformation, while energy sensors (SnRK1–TOR–GCN2) enable metabolic adaptation under resource-limited conditions.

This level determines cell fate depending on the intensity and duration of the stress signal. Autophagy acts as a key regulator, performing selective degradation of effectors or bulk removal of cellular structures during the hypersensitive response (HR). Antioxidant systems (SOD, APX, CAT, GPX, glutathione) maintain redox homeostasis. Energy sensors (SnRK1, TOR, GCN2) coordinate the balance between anabolism, catabolism, and defense. Proteostasis is maintained by UPS, ERAD, and UPR, which regulate protein degradation and proteome quality. The cytoskeleton (actin, microtubules) orchestrates autophagosome and vesicle trafficking, ensuring redistribution of resources. Osmotic–ionic and metabolic switches ($K^+$, $Cl^-$, proline, melatonin, GABA) enhance stress tolerance. The multistability of the network is described as a "state landscape," allowing transitions between SA-, JA/ET-, and ABA-dominant modes.

*3. Cellular-level effectors.* At this level, the direct response against pathogens is executed, encompassing physical, chemical, and biochemical defense mechanisms. Cell wall modifications create mechanical barriers, while antioxidant systems and the RNS/NO module regulate local oxidative processes. Membrane trafficking, including ESCRT pathways, and lipid signaling ensure proper localization of effector proteins. Secondary metabolites, such as phenylpropanoids and volatile terpenoids (VOCs), function as antibiotics and signaling molecules between cells and organs.

The cell wall is reinforced through the deposition of callose, lignin, and suberin. Membrane trafficking and ESCRT systems mediate PR protein secretion and vesicle-based immune responses. Lipid signaling (oxylipins, phosphatidic acid, sphingolipids) coordinates stress-induced autophagy. Stomatal immunity regulates pathogen entry via ABA- and ROS/NO-dependent mechanisms.

*4. Systemic integration: organismal and ecological level.* This level integrates local responses across the whole organism. Hormonal networks (SA, JA/ET, ABA, IAA, BR, GA, CK) coordinate intercellular and inter-organ signaling. $Ca^{2+}$ waves and electro-/hydraulic signals enable rapid long-distance information transfer. Plasmodesmata and intercellular communication synchronize defense responses, while stomatal immunity mechanisms regulate gas exchange permeability and limit infection at the leaf surface.

At this level, coordination of primary and secondary immunity occurs, encompassing PTI–ETI synergy, resistosome formation, and epigenetic priming. The microbiome, ISR, and organelles (chloroplasts, mitochondria) influence systemic immune responses through retrograde signaling. Tissue repair and regeneration restore damaged regions, while integration with development, senescence, and the reproductive phase optimizes defensive resource allocation according to the plant's life cycle.

Following the acute immune response (PTI/ETI, HR, PCD), the plant transitions into a repair phase. Programs for cell wall regeneration are activated, with enhanced biosynthesis of cellulose, hemicellulose, and lignin. Meristematic cells receive signals from damaged tissues via hormones (cytokinins, auxins) and ROS/NO waves, triggering cell division and tissue restoration.

The immune network is integrated with the regulation of flowering and the formation of reproductive structures. Stress signals (SA, JA, ABA, ROS) can either accelerate flowering (escape strategy) or delay it (conservation strategy). For instance, pathogen infection may trigger early induced flowering via FT and SOC1, whereas abiotic stress mediated by ABA can suppress pollen fertility.

Organelles act as critical stress sensors and sources of ROS. In chloroplasts, ROS are generated via the Mehler reaction, while mitochondria produce ROS through complexes I and III. These signals are transmitted to the nucleus (retrograde signaling), modulating transcriptional programs. GUN1 and ANAC017 coordinate the integration of photosynthetic and respiratory signals with the immune network.

The ultimate effector layer of plant immunity involves the synthesis of secondary metabolites. Phenylpropanoids (lignin, coumarins), terpenoid volatiles, and phytoalexins (e.g., camalexin) directly limit pathogens and herbivorous insects. These molecules also function as signals to neighboring cells and even to other organisms (microbiome, antagonist insects).

The immune response varies depending on cell and tissue type. In the epidermis, guard cells play a key role (ROS/NO-mediated regulation of stomatal closure). In the xylem and phloem, lignification occurs alongside the transport of systemic signals. In meristems, the immune response is attenuated to preserve stem cells. Thus, the immune network exhibits spatially distributed dynamics.

Immunity is intertwined with senescence programs. SA- and JA-signaling activate aging as a form of controlled PCD, enhancing defense against biotrophs and preparing tissues for death. Senescing leaves exhibit elevated basal resistance ("age-related resistance"). The plant immune network varies among species, ecotypes, and cultivars. Natural alleles of PRR/NLR genes, as well as variations in hormonal regulators, determine different levels of resistance. This creates an evolutionary landscape in which the immune network is tuned to specific ecological niches.

The defensive response extends beyond individual cells, encompassing tissues and the entire organism. Plasmodesmata mediate intercellular signal transduction, while ROS and $Ca^{2+}$ waves serve as key systemic messengers. Systemic acquired resistance (SAR) and induced systemic resistance (ISR) confer long-lasting protection. Local responses at the infection site (HR, PCD) are invariably integrated with systemic programs (SAR and ISR) established in distal tissues. This integration is mediated by the translocation of mobile signals, including methyl salicylate (MeSA), methyl jasmonate (MeJA), peptide signals (Pep, CLE), and mobile miRNAs.

A critical integrative conduit is the root↔shoot axis: stress perception in roots (e.g., salinity, drought) reprograms immune responses in aerial organs. This level of organization integrates local and systemic processes within both organismal and environmental contexts. Local-systemic coordination among roots, leaves, and meristems orchestrates adaptive modifications. Photoreceptors and circadian rhythms regulate the temporal activation of defense mechanisms, with SA signaling peaking in the morning and JA/ET signaling in the evening.

Memory mechanisms, including priming, epigenetic modifications, and even intergenerational signal translocation, establish transgenerational tolerance. The plant microbiome and plant growth-promoting rhizobacteria (PGPR) produce signaling molecules that enhance immunity. At the organismal level, integration coordinates signaling between roots, leaves, and meristems. Ecological context shapes cross-tolerance to combined biotic and abiotic stresses. Osmotic-ionic and metabolic regulation, tissue specificity (epidermis, xylem, meristem), and eco-evolutionary context collectively determine cross-tolerance and the adaptive potential of plants under multifactorial stress conditions.

At the whole-organism level and in the context of environmental interactions, plant immune responses acquire a systemic character. Local cellular signals are translated into organism-wide reactions through systemic integration, which coordinates communication among different organs, tissues, and the plant's immediate microenvironment.

Systemic activation of immunity, or Systemic Acquired Resistance (SAR), is mediated by the dissemination of signals from locally infected tissues to distal organs. SAR confers long-lasting protection against pathogens and induces the expression of pathogenesis-related (PR) proteins, antioxidant enzymes, and repair factors. Key molecules, including methyl salicylate, azelaic acid, and peptide signals, transmit information about local stress to systemic tissues, thereby establishing organism-level immune memory.

The plant immune network operates within an ecological context rather than in isolation. The rhizosphere and phyllosphere microbiomes provide the foundational basis for Induced Systemic Resistance (ISR). Secondary metabolites, including phytoalexins, phenylpropanoids, and volatile terpenes, function not only as direct antimicrobial agents but also modulate neighboring plants and microbial communities. ISR is activated by commensal or symbiotic microorganisms interacting with the root system, enhancing the plant's preparedness against pathogen attacks by stimulating JA/ET hormonal pathways and mobilizing ROS/NO in distal tissues. Unlike SAR, ISR is often not associated with the accumulation of PR proteins but confers a rapid and energetically efficient defense response.

Given the high energetic cost of stress responses, a trade-off between growth and defense is continuously maintained. Key metabolic and energy sensors, including SnRK1–TOR and GCN2, along with metabolites such as sugars, amino acids, polyamines, melatonin, and GABA, regulate the allocation of resources. Chloroplasts act simultaneously as energy sources and generators of ROS signals, integrating photosynthetic activity with immune responses.

Cross-tolerance is a well-documented phenomenon in plants, whereby prior mild stress–such as heat or oxidative priming-enhances resistance to an entirely different stress, including pathogen attack or drought. Cross-tolerance reflects the plant's capacity to integrate diverse biotic and abiotic stress signals to establish adaptive resilience. Mechanisms underlying cross-tolerance involve the crosstalk of hormonal networks (SA, JA, ABA), ROS/NO signaling waves, autophagy, and secondary metabolism, enabling an optimized balance between growth, defense, and reproductive functions. The multistable nature of the immune system allows rapid switching between different dominant immune states depending on the combination of stress factors encountered.

The microbiome acts as a key modulatory ecological factor in systemic immunity. Symbiotic bacteria and fungi form a protective barrier against pathogens, stimulate ISR, improve nutrient and water status, and modulate hormonal and metabolic pathways. Interaction with the microbiome enables plants to adapt to fluctuating environmental conditions, enhancing cross-tolerance and promoting long-term survival at the ecosystem level.

Thus, the integration of cellular, tissue-specific, and systemic signals establishes an organismal-ecological immune network that coordinates local and systemic responses, optimizes the trade-off between defense and growth, and incorporates environmental context and microbiome interactions to sustain long-term plant survival.

*5. Regulatory layers, dynamics, system states, and multistability.* This level defines the dynamics and duration of immune responses. The plant immune network functions as a dynamic, multistable system capable of rapidly shifting dominant states depending on the type, intensity, and duration of stress. Epigenetic mechanisms (DNA methylation, histone modifications, small RNAs) establish immune "memory." Photoreceptors and circadian rhythms (CCA1, LHY, TOC1) regulate temporal sensitivity.

A central element of this dynamic behavior is the interplay among key hormonal networks (SA, JA/ET, and ABA) with growth regulators and metabolic signals. The dynamics of the immune system can be conceptualized as a multistable network for plant defense against pathogens and stress. The "landscape of states" reflects the predominance of particular signals or hormonal pathways: SA-dominant, JA/ET-dominant, ABA-dominant, and growth states.

The SA-dominant state promotes cell survival under biotrophic attack and moderate abiotic stress. The JA/ET-dominant state induces PCD and extensive autophagy in response to necrotrophs or massive cellular damage. Hemibiotrophs trigger a phase transition between SA- and JA/ET-regimes, which is sensitive to abiotic factors. The ABA state is activated by abiotic stress, leading to stomatal closure and antioxidant protection. GA, IAA, BR, and CK coordinate the balance between growth, tissue recovery, and abiotic stress tolerance (supporting growth, regeneration, and trade-offs with defense).

The SA-dominant state is typically triggered by biotrophic pathogens and localized damage, initiating PR protein accumulation, ROS elevation, and systemic activation of SAR. This state is characterized by high defensive readiness but is accompanied by temporary suppression of growth processes due to energetic resource reallocation.

The JA/ET-dominant state primarily arises in response to necrotrophic pathogens or herbivorous insects. It promotes the synthesis of defensive secondary metabolites, activates antioxidant systems and lipid signaling, and establishes ISR in distal tissues. JA/ET signaling is often antagonistic to SA, enabling the plant to selectively enhance the appropriate defense pathway according to pathogen type.

The ABA-dominant state responds to abiotic stresses such as drought, osmotic stress, or temperature extremes, activating osmo-ionic regulation, stomatal closure, and mobilization of antioxidant defenses. ABA also interacts with SA and JA/ET, forming a flexible, multistable network that optimizes the trade-off between survival and growth.

The growth state is activated in the absence of stress or following its alleviation. In this state, growth hormones (IAA, GA, BR, CK) predominate, promoting cell division and differentiation, while defense mechanisms are temporarily downregulated to conserve energy.

Rapid phase transitions occur between these states under the influence of local and systemic signals (ROS/NO, $Ca^{2+}$ waves, peptide triggers) and are integrated at the organismal and tissue levels. The system exhibits multistability, whereby multiple states can potentially coexist, with the dominant state determined by stress specificity and ecological context. Stress signals can swiftly shift the system from one state to another, providing adaptive flexibility and balancing growth, recovery, and defense.

Thus, the dynamics of SA ↔ JA/ET ↔ ABA ↔ growth states confer adaptive plasticity, allowing the plant to rapidly switch between defense and growth, integrate signals of diverse origin, and maintain resilience under multifactorial stress conditions.

*6. Integration of modules into a unified network and final outcomes.* The plant immune response emerges from the coordinated interplay of numerous molecular and cellular modules, which operate not in isolation but as a single integrated defense network. Central coordinators of this system are the hormonal signaling networks, ROS/NO activity, and autophagy mechanisms, collectively ensuring an adaptive response even under extensive cellular damage.

Hormonal integration is achieved through complex interactions among classical phytohormones such as SA, JA/ET, ABA, IAA, BR, GA, and CK. These signals orchestrate both local and systemic defense reactions, modulate PRR- and NLR-dependent immunity, and determine the balance between growth and defense functions. While SA and JA/ET are largely antagonistic, they can form synergistic interactions in specific contexts, enhancing the PTI–ETI response. ABA and

CK contribute to stress tolerance and water balance regulation, simultaneously fine-tuning the effectiveness of the immune response.

ROS/NO, as signaling molecules, function as rapid secondary messengers that integrate sensory inputs and transmit them to cellular effectors. The generation of local and systemic ROS waves and NO bursts coordinates antioxidant system activity, modulates the cytoskeleton, regulates membrane trafficking, and mobilizes secondary metabolites. ROS/NO act not only as local damage effectors but also as central nodes synchronizing hormonal signaling pathways with repair and autophagy mechanisms.

Autophagy integrates intracellular processes that maintain homeostasis and energy stability. Through selective degradation of damaged proteins and organelles, autophagy ensures efficient resource utilization while modulating ROS/NO signaling. It interacts with hormonal pathways (SA, ABA, TOR/SnRK1), coordinating the balance between cell survival and programmed cell death (PCD). Furthermore, autophagy contributes to the regulation of local and systemic immune responses by interfacing with PTI–ETI synergy and epigenetic priming.

A unified integrated network emerges through continuous crosstalk between hormonal signals, ROS/NO, and autophagy. This network enables the plant to rapidly adapt to combined stresses, coordinate local and systemic responses, support tissue repair, and maintain viability even under extensive cellular damage. Such integration creates a multistable system capable of switching between SA-, JA/ET-, or ABA-dominant states, optimizing the balance between defense, growth, and reproductive processes.

Signals and the internal metabolic state of the cell coordinate the activity and interaction of all modules, determining whether a cell survives or undergoes PCD depending on pathogen type or abiotic stress. Under biotrophic infection or mild abiotic stress, cells tend to survive. Under necrotrophic attack or severe damage, cells undergo PCD, but the plant as a whole survives. This explains why the same plant can either preserve a cell (biotrophs, low-intensity abiotic stress) or deliberately sacrifice it (necrotrophs/severe damage) while remaining viable as an organism.

Depending on signal integration, alternative outcomes are possible: 1. Localized cell death (HR/PCD) to restrict pathogen spread; 2. Tolerance via homeostatic mechanisms; 3. Systemic resistance (SAR/ISR) for long-term protection; 4. Tissue regeneration following stress; 5. Regulation of flowering and senescence as adaptive strategies.

*7. Generalized decision-making framework.* At low damage signals (low DAMPs, moderate ROS), SA-dependent pathways are activated, and selective autophagy promotes cell survival and tolerance formation. Under severe damage with high DAMPs and MAMPs (JA/ET dominance, ROS burst), massive autophagy and HR trigger local pathogen containment. During hemibiotrophic infections, a phase transition between SA ↔ JA/ET occurs. Systemic signals (ROS, SA, VOCs) induce SAR/ISR in distant tissues. Growth hormones (auxins, GA, BR) facilitate post-stress recovery and regeneration. Thus, the plant cell integrates MAMP/DAMP signals, hormonal status, and abiotic factors, coordinating autophagy, the cytoskeleton, cell wall remodeling, and HR/PCD to achieve an optimal defense strategy.

The state landscape of this integrative model of plant immune and stress responses has several practical applications. First, it allows prediction of plant responses to pathogens or abiotic stress. By identifying the type of stress (biotrophic, necrotrophic, or, for example, drought) one can predict which hormonal state will dominate (SA, JA/ET, or ABA). This enables agronomists and researchers to anticipate defensive protein activity, ROS production, and secondary metabolite synthesis.

The second application concerns optimizing the use of biostimulants and bioprotectants. Activating systemic resistance inducers (ISR), associated with JA/ET and the microbiome, or systemic acquired resistance (SAR) inducers based on SA analogs at the appropriate time increases their effectiveness. For instance, during biotrophic infection, activating SA signaling is advantageous, whereas necrotrophic attacks call for JA/ET pathways.

The third aspect is planning agronomic practices. Understanding phase transitions between different states allows for regulating irrigation, fertilization, pruning, or other interventions without suppressing defense mechanisms during periods of greatest pathogen vulnerability.

The fourth application relates to breeding cultivars that combine high productivity with enhanced stress tolerance. Selecting genotypes capable of efficiently switching between SA-, JA/ET-, ABA-, and growth-dominant states ensures better adaptive plasticity and resilience to multifactorial stress.

Finally, the model offers broad opportunities for scientific modeling and visualization. Using the state landscape (SA ↔ JA/ET ↔ ABA ↔ growth) provides a clear representation of multistability and phase transitions in publications, presentations, or research, illustrating the dynamic behavior of the immune network and plant responses to various stress signals.

Deepening the theory of plant immunity to pathogens requires a detailed understanding of the roles of autophagy, the cytoskeleton, cell wall dynamics, and hormonal regulation in shaping defense responses and nonspecific resistance, as well as studying cell-associated mechanisms of resilience and elucidating why these processes fail during infections caused by specific pathogenic microorganisms and viruses. This approach is fundamentally important for modern breeding and the development of novel plant protection strategies, including the creation of cultivars and hybrids with enhanced resistance, as well as fourth-generation protective agents. These latest-generation agents are capable of inducing plant immunity both independently and in combination with conventional fungicides, thereby preventing the rapid emergence of resistance to systemic treatments.

In this context, we propose an integrative model of plant immunity to abiotic and biotic stresses that encompasses key components: autophagy, antioxidant systems, ROS homeostasis, cytoskeletal dynamics, cell wall modification, hormonal regulation, epigenetic mechanisms, and metabolic reprogramming. This approach not only clarifies the complexity of biotic–abiotic signal interactions but also identifies potential regulatory nodes for enhancing plant stress resilience. The aim of the model is to explain how a plant selects the optimal strategy: to preserve a cell or sacrifice it for the integrity of the organism, depending on the intensity of abiotic and biotic stressors and the degree of cellular damage.

The central concept of the integrative model is that PRR signaling instantaneously activates the $Ca^{2+}$–ROS–MAPK module as the first line of defense. Within this system, autophagy functions as a "damping" mechanism, limiting excessive reactivity, preventing toxic ROS accumulation, and simultaneously guiding the cell toward an optimal protective strategy. An additional critical regulatory layer is provided by antioxidant systems–both enzymatic (SOD, CAT, APX) and non-enzymatic (ascorbate, glutathione)–which finely tune ROS signaling waves, preventing their destructive effects. Autophagy removes sources of excessive ROS (damaged mitochondria, peroxisomes), while the antioxidant system neutralizes ROS that have already formed, both enzymatically and non-enzymatically. Together, these mechanisms create a dual-layered control system: "prevention plus removal." Low ROS concentrations act as signaling molecules that enhance $Ca^{2+}$-dependent cascades and activate SA/JA/ET pathways, whereas high ROS levels trigger oxidative stress and initiate PCD. Here, the antioxidant system determines whether ROS function as survival signals or death signals.

Intracellular logistics are mediated by the cytoskeleton, while the cell wall serves as a physical barrier against pathogens. Hormonal signaling networks act as "steering mechanisms," determining the fate of the cell according to the type of stress. SA-dominance promotes cellular viability during biotrophic infections and under moderate abiotic stress. SA can both stimulate ROS bursts and activate antioxidant enzymes, balancing HR and systemic resistance. In contrast, JA/ET-dominance is associated with programmed cell death and massive autophagy, typical for responses to necrotrophs or extensive cellular damage. JA/ET signaling also enhances antioxidant defenses during necrotrophic attacks. In the case of hemibiotrophic pathogens, a phase transition occurs between SA- and JA/ET-dominant modes, with the direction of this shift being strongly influenced by environmental abiotic factors.

Additional regulation is provided by ABA, GA, IAA, BR, and cytokinins, which maintain the balance between growth, recovery, and tolerance to abiotic stresses, forming a flexible, multilevel control system that integrates cell survival with the adaptive potential of the entire plant. Hormonal signaling pathways finely adjust the trade-off between growth and defense. Abscisic acid (ABA) rapidly induces stomatal closure, reducing water loss and enhancing tolerance to drought and salinity, while also modulating the expression of antioxidant enzymes. ABA activates antioxidant defenses under drought or salinity stress. Gibberellins (GA) regulate the transition from stress response to post-stress growth recovery, promoting cell elongation and tissue restoration. Auxins (IAA) coordinate cytoskeletal remodeling and stimulate root system regeneration, enhancing the plant's adaptive capacity. Brassinosteroids (BR) strengthen cell walls, boost antioxidant protection, and simultaneously support growth even under stress conditions. Cytokinins (CK) activate cell division in meristems and ensure the recovery of vegetative organs after damage.

Collectively, these regulators form a "second layer" of the integrative model, providing plasticity and resilience of cellular networks under combined biotic and abiotic stresses. Overall, these elements constitute a multilevel integrative model of plant immunity and stress tolerance, in which rapid signals ($Ca^{2+}$, ROS), regulatory circuits (autophagy, cytoskeleton, cell wall), and hormonal-antioxidant control work coherently according to a unified logic: preserving the integrity of the whole plant, even at the expense of individual cells.

The integrative model of plant defense against pathogens and stresses is based on the concept of a multilevel system of interconnected modules that collectively determine the nature and efficiency of cellular defense responses and establish specific types of immune reactions in the form of a hierarchical signal-metabolic network. The central integrator is autophagy, which coordinates the exchange of information among cellular sensors, hormonal networks, cytoskeletal structures, and the barrier mechanisms of the cell wall. The model is structured as a multilevel plant defense network with autophagy at its core, where all other modules (PRR signaling, ROS-antioxidant systems, cytoskeleton, cell wall, hormones, organelles, microbiome, and epigenetic mechanisms) are integrated into a unified functional system.

The proposed plant immunity model aids in understanding diverse cellular strategies for responding to pathogens and stress. It conceptualizes the defense response to biotic and abiotic stressors not as a linear reaction but as a multilevel signal–metabolic network combining autophagy, antioxidant systems, ROS homeostasis, cytoskeletal dynamics, cell wall modification, hormonal regulation, epigenetic mechanisms, and metabolic remodeling. The character of the plant's signal–metabolic response to biotic and abiotic stress depends on the combination of external signals and the internal cellular state. Autophagy serves as the central integrator of the network, coordinating interactions among sensors of external and internal signals, determining the balance between cell survival and programmed cell death, and facilitating the adaptive integration of local and systemic resistance.

This approach provides a novel explanation for how a plant "decides" on its defense strategy, taking into account the nature and intensity of stress. The model illustrates how a plant cell integrates external and internal cues to select the optimal strategy–either preserving the cell or initiating its programmed death for the survival of the whole organism. It allows for flexible strategic choices: localized cell death (HR), enhanced tolerance, systemic resistance (SAR/ISR), or tissue recovery and regeneration. Through this multilevel integration, the plant maintains an optimal balance between defense, growth, and reproductive development.

**General logic of the model**

According to the model, the plant immune system is structured and functions based on the principle of prioritizing the survival of the whole organism over individual cells. This means that cells can sacrifice their own viability to localize infection, preserve tissues, or maintain overall homeostasis. The plant immune response model is based on several key principles: a modular hierarchy of mechanisms, the "partial sacrifice" principle, systemic coordination, energetic efficiency, and the balance between cell life and death.

Central mechanisms such as autophagy, apoptosis-like programmed cell death, and the hypersensitive response (HR) serve as the first line of local control, isolating or eliminating infected cells to prevent pathogen spread. This process embodies the principle of "partial sacrifice": the plant loses a small number of cells at the site of infection, but maintains the integrity and viability of the entire organism. Local signals (including reactive oxygen species (ROS), calcium pulses, and hormonal cascades) are rapidly transmitted to neighboring cells and organs, establishing systemic resistance (SAR, ISR) and enhancing nonspecific immunity at the whole-plant level.

From an energetic perspective, this strategy is advantageous: the cost of losing individual cells or a portion of tissue is far lower than the potential losses from uncontrolled pathogen spread, while the plant's reproductive capacity and genetic inheritance remain intact even at the expense of local damage. At the same time, excessive cell death can lead to loss of functional tissue, whereas insufficient death can result in generalized infection, requiring precise regulation. Therefore, the plant immune system maintains a dynamic balance by integrating signaling networks mediated by phytohormones (SA, JA, ET, ABA) to finely tune the response.

In conclusion, this plant immunity model operates according to an evolutionarily advantageous principle: it is better to sacrifice individual cells or a local tissue area than to allow the loss of the entire organism.

The general logic of the plant immune model provides a foundation for new experimental studies and potential biotechnological applications aimed at enhancing crop stress tolerance and supports the development of novel strategies for crop protection. Its practical significance lies in identifying new targets for biotechnology (regulators of autophagy, SnRK1/TOR signaling, ROS/NO homeostasis, and epigenetic factors) which can be leveraged to improve the resilience of agricultural crops.

The proposed integrative model of plant immunity represents a multi-level signal-metabolic defense network with autophagy as the central integrator. Its key advantage is the ability to explain the balance between cell survival, local cell death, and systemic resistance, which has both fundamental and applied relevance for modern biology and agriculture. The integrative model overcomes the fragmentation of previous PTI/ETI concepts and unifies cellular, organellar, hormonal, and systemic levels of defense. In this model, the plant immune response is not seen as a collection of isolated reactions but is interpreted as a single signal–metabolic defense network, where individual components (sensors, signaling cascades, effectors, hormonal regulators, and epigenetic mechanisms) function in close interaction.

Autophagy serves as the central integrator, responding to ROS signals, energy sensors, and hormonal cues to determine cell fate: maintaining tolerance, activating HR/PCD, or initiating systemic resistance.

**Description of the model's operation across different levels of plant organization**

Current understanding of plant resistance mechanisms to biotic and abiotic stressors emphasizes viewing the defensive response not as a collection of isolated reactions, but as a unified integrative defense network, in which various signaling and effector modules operate in close coordination and mutual regulation. The central regulatory hub of this network is autophagy, which, on one hand, functions as an intracellular homeostatic mechanism through the degradation of damaged organelles and protein aggregates, and on the other hand, provides a critical decision-making point between cellular survival and programmed cell death (PCD), depending on the nature and intensity of the stress stimulus.

At the initial stage of the defense response, pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs) are recognized via membrane-bound pattern recognition receptors (PRRs) and mechanosensory proteins, initiating a cascade of early signaling events. These include ion fluxes ($Ca^{2+}$, $K^{+}$, $H^{+}$),

generation of reactive oxygen species (ROS), and activation of MAP kinase cascades, collectively triggering transcriptional defense programs.

The subsequent execution of local defense involves cell wall reinforcement, which serves not only as a physical barrier but also as a signaling structure. Remodeling of the cell wall is accompanied by the deposition of callose, lignin, and suberin, along with the release of DAMP signals that amplify PRR-dependent pathways. Cytoskeletal structures ensure spatiotemporal coordination of the transport of autophagosomes, enzymes, vesicles containing structural components, and regulatory molecules to infection sites, thereby establishing the "intracellular logistics" of cellular defense.

A key stage of integration is autophagic control, which interprets signals from ROS, antioxidant systems, and hormonal regulators to modulate the intensity of catabolic processes. Autophagy determines whether a cell will maintain viability via selective degradation of damaged components or initiate a localized programmed cell death program, such as the hypersensitive response (HR), to limit pathogen spread.

Hormonal networks act as key coordinators of this process. Salicylic acid (SA) is associated with the induction of systemic acquired resistance (SAR) and promotes the maintenance of cellular tolerance through the activation of selective autophagy. In contrast, jasmonic acid (JA) and ethylene (ET) predominantly stimulate the development of bulk autophagy and the hypersensitive response (HR), corresponding to a local sacrifice strategy. Abscisic acid (ABA) integrates abiotic signals (drought, salinity, cold) and enhances antioxidant protection. Auxins, cytokinins, brassinosteroids, and gibberellins participate in regulating the balance between growth and defense processes, determining the potential for tissue regeneration and recovery following stress.

At the level of systemic organization, defensive signals propagate through ROS and NO waves, mobile peptide factors, retrograde signaling from chloroplasts and mitochondria, as well as interactions with the root and leaf microbiome, collectively contributing to the formation of induced systemic resistance (ISR). Additionally, volatile organic compounds (VOCs) are integrated into the response, mediating inter-organismal communication and priming neighboring plants.

Thus, local responses are coupled with systemic ones: ROS and NO waves, mobile peptides, and chloroplast- and mitochondria-derived signals trigger defense activation in neighboring cells and distant organs. This coordination establishes systemic acquired resistance (SAR) and induced systemic resistance (ISR) involving both the microbiome and volatile organic compounds.

Protective states are reinforced through epigenetic mechanisms–including DNA methylation, histone modifications, and the activity of non-coding RNAs, which establish priming and even transgenerational memory, enhancing the efficiency of responses in the progeny. Epigenetic regulation plays a particularly important role, as it enables priming and potentially transgenerational memory, allowing offspring to exhibit an elevated readiness for subsequent stress exposures.

In summary, the integrative plant defense model represents a multilevel signal-metabolic network of immune responses, with autophagy functioning as the central integrator. All other components (PRR signaling, the ROS-antioxidant system, the cytoskeleton, the cell wall, hormones, organellar sensors, the microbiome, and epigenetic mechanisms) interact continuously, providing the plant with a flexible choice between local cell death, enhanced tolerance, systemic resistance, and regenerative processes. The model is structured as a multilevel defense network with autophagy at its core, where all modules (PRR signaling, ROS–antioxidant systems, cytoskeleton, cell wall, hormones, organelles, microbiome, and epigenetic regulators) are integrated into a unified functional immune system. Such multilevel integration enables the plant to maintain a dynamic balance between defense, growth, and reproductive functions, which is critical for survival under variable environmental conditions. The model allows a flexible strategic choice: local cell death (HR), enhanced tolerance, systemic resistance, or recovery and regeneration. Through this multilevel integration, the plant maintains an optimal balance between protection, growth, and reproductive development.

Thus, plant immunity functions as a dynamic, multicomponent defense network against stress factors, where autophagy, ROS-antioxidant systems, the cytoskeleton, the cell wall, and hormonal cascades interact in an integrated defense mode. The choice between cell survival and sacrifice is not random but follows a system-level logic aimed at preserving the survival of the entire plant. This model opens opportunities for experimental identification of regulatory nodes and practical applications in biotechnology to enhance crop stress tolerance.

The plant immune system can be conceptualized as a multilevel integrated defense network encompassing molecules, organelles, cells, tissues, organs, the entire organism, interactions with the environment, temporal dynamics, and metabolism, functioning as a dynamic, multistable system. Plant immunity can thus be regarded as a multilevel signal-metabolic network that organizes immune responses into functional modules, each responsible for a specific aspect of protection and survival. Plant defense is organized into a complex hierarchical structure that ensures efficient coordination of signals from primary sensing to systemic responses and interactions with the ecological environment. This structural approach reflects the progressive integration of processes from basic molecular mechanisms of plant immunity to organismal and ecological contexts.

The plant immune response arises from the close interaction of numerous molecular and cellular modules, which operate not in isolation but as a unified integrated network. Central coordinators of this system are hormonal signaling networks, ROS/NO activity, and autophagy mechanisms, which collectively ensure an adaptive response even under extensive cellular damage.

Autophagy integrates intracellular processes that maintain homeostasis and energy stability. Through selective degradation of damaged proteins and organelles, autophagy ensures efficient resource utilization while simultaneously modulating ROS/NO signaling. It interacts with hormonal pathways (SA, ABA, TOR/SnRK1), coordinating the balance between cell survival and programmed cell death (PCD). Furthermore, autophagy contributes to the regulation of local and systemic immune responses by interfacing with PTI–ETI synergy and epigenetic priming.

A unified integrated network emerges through continuous crosstalk between hormonal signals, ROS/NO, and autophagy. This network allows the plant to rapidly adapt to combined stresses, coordinate local and systemic responses, promote tissue repair, and maintain viability even under substantial cellular damage. Such integration creates a multistable system capable of swiftly switching between SA-, JA/ET-, or ABA-dominant states, optimizing the balance between defense, growth, and reproductive processes.

At the whole-plant level, including its interactions with the environment, the immune response acquires a systemic character. Local cellular signals are translated into organismal responses through systemic integration, ensuring coordination among different organs, tissues, and the plant's microenvironment. Key processes include systemic acquired resistance (SAR), induced systemic resistance (ISR), cross-tolerance, and the microbiome as a modulating factor of systemic immunity. The integration of cellular, tissue, and systemic signals forms an organismal-ecological immune network, coordinating local and systemic responses, optimizing the defense–growth balance, and incorporating environmental context and microbiome interactions to support long-term plant survival.

The plant immune network represents a dynamic and multistable system, capable of rapidly shifting dominant states depending on the type, intensity, and duration of stress. A central element of this dynamic behavior is the interaction among key hormonal networks (SA, JA/ET, and ABA) with growth regulators and metabolic signals. Transitions between states (SA-dominant, JA/ET-dominant, ABA-dominant, growth state) occur rapidly, driven by local and systemic signals (ROS/NO, $Ca^{2+}$ waves, peptide triggers) and are integrated at the tissue and whole-organism levels. The system exhibits multistability, wherein multiple states may be simultaneously potentially active, and the selection of a dominant state is determined by stress specificity and the environmental context. The dynamic interplay of SA ↔ JA/ET ↔ ABA ↔ growth states provides adaptive plasticity, ena-

bling the plant to switch efficiently between defense and growth, integrate signals from diverse origins, and maintain resilience under multifactorial stress conditions.

This logic explains why the same plant can adopt diametrically opposite strategies (cell survival or programmed sacrifice) while still preserving organismal integrity. The model elucidates the plasticity of the immune response: the same plant can respond differently depending on pathogen type and context (necrotroph, biotroph, mild abiotic stress, severe drought). Table 2 consolidates the principal modules of the integrative plant immunity and stress-resilience model into a single, unified defense network against biotic and abiotic factors. It includes modules, functional nodes, representative genes/proteins, and the outcomes of activation. The table highlights the threshold-based ("switch") logic and multistable nature of the network.

**Table 2**

Integrative model of plant immune architecture and stress resilience, linking central modules, functional nodes, and threshold-based logic to generate a unified, multistable immune and stress-response network for protection against pathogens and environmental stresses

| Module / node | Primary role | Mechanism of action | Gene / protein examples | Outcome / effect | Threshold / switch logic |
|---|---|---|---|---|---|
| PRR Signaling | defense initiation | recognition of MAMPs/DAMPs → ↑$Ca^{2+}$ → activation of $Ca^{2+}$-dependent kinases → ROS via NADPH oxidases → MAPK → transcriptional activation | FLS2, EFR, CERK1 | rapid cellular mobilization; primary "alarm signal" | N/A |
| Autophagy | regulatory "brake" system | selective/mass degradation of damaged organelles; modulation of hormonal sensitivity | ATG5, ATG8, ATG12 | mitigation of ROS damage; maintains growth ↔ defense balance | ↑SnRK1 / ↓TOR → autophagy ↑ |
| Cytoskeleton (AF, MT) | defense logistics | vesicle, enzyme, autophagosome transport; coordination with autophagy | ACT7, TUB6, MAP65 | rapid papilla/callose deposition; delivery of lignin and enzymes | N/A |
| Cell Wall (CWI sensing) | barrier and sensor | callose deposition, lignification; degradation products → DAMPs → PTI | WAK1, THE1, PME | physical barrier and sensory signaling | defect → DAMPs → PTI |
| Hormones | defense scenario coordinators | SA: restrained ROS, selective autophagy; JA/ET: massive PCD; ABA: abiotic integration, SA suppression; IAA/BR/GA/cytokinins: growth ↔ defense balance | NPR1, COI1, ABI1, DELLA | strategy selection: survival vs. PCD | circadian rhythms: evening → SA shift / JA activation |
| Antioxidant Systems | ROS buffer | SOD, CAT, APX, GPX; non-enzymatic – ascorbate, tocopherols | SOD1, CAT2, APX1 | prevent oxidative damage, preserve ROS signaling | N/A |
| RNS / NO | oxidative signaling, HR/PCD | NO + ROS → protein S-nitrosylation; PCD activation | NR, NOS1 | sustained accumulation → HR / PCD | ↑NO + ↑ROS → HR/PCD |
| SnRK1–TOR | energy control | energy status sensor; regulates autophagy and growth | SnRK1, TOR | growth ↔ defense balance | energy deficit → autophagy ↑ |
| UPS / ERAD – UPR | proteostasis | protein quality control under stress; degradation of damaged proteins | HSP70, BiP, PDI | prevents accumulation of defective proteins | N/A |
| Vesicular Trafficking / ESCRT | vesicle transport | transport of autophagosomes and receptor vesicles | VPS4, CHMP1 | coordination with autophagy | N/A |
| $Ca^{2+}$ Waves | secondary messenger | systemic $Ca^{2+}$ signal propagation → activation of $Ca^{2+}$-dependent proteins | CNGC2, CAMTA3 | systemic defense mobilization | N/A |
| AQP–$H_2O_2$ | water and ROS transport | AQP mediates $H_2O_2$ transport, enabling systemic signaling | PIP1;2, PIP2;1 | systemic response to local stress | ↑AQP → systemic ROS |
| Lipid Signaling | phospholipids as signals | PA, DAG → activation of NADPH oxidases, MAPK | PLDα1, DGK5 | defense cascade mobilization | N/A |
| Stomatal Immunity | gas exchange regulation | ABA, JA, ROS → stomatal closure/opening | OST1, SLAC1 | transpiration and gas exchange control | N/A |
| Plasmodesmata (PD) | intercellular communication | Transmission of SA, JA, ROS, peptides | PDLP5, Callose synthase | coordination of systemic response | N/A |
| PTI–ETI Integration | surface and intracellular immune response | PRR + NLR → ROS, PCD, autophagy activation | RPM1, RPS2, RPS5 | synergy of local and systemic defense | N/A |
| Epigenetic Memory | priming, transgenerational tolerance | DNA methylation, histone modifications, H3K4me3 → sustained readiness | DRM2, MET1, HDA6 | accelerated/enhanced response to repeated stress | N/A |
| Microbiome / ISR | induced systemic resistance | beneficial microbes → JA/ET priming, ROS balance | NPR1, MYC2 | resource-efficient, rapid defense | ISR + low resources → JA priming |
| Light / Circadian | diurnal rhythms | SA in morning, JA/ET in evening; coordinates with PCD | CCA1, TOC1 | temporal defense adaptation | Evening → SA shift / JA activation |
| Osmolytes | drought/salinity protection | proline, sucrose, trehalose → osmotic balance | P5CS1, TPS1 | turgor maintenance, ROS buffering | N/A |

**Hormonal crosstalk**

The model posits a complex network of feedback loops that ensures flexible and balanced functioning of the plant immune network, coordinating the speed of activation with control of excessive reactivity. SA, JA, and ET determine the type of defense scenario against pathogens, while ABA, IAA, BR, GA, and cytokinins modulate adaptation to abiotic stresses and balance growth versus defense. Together, these hormones form a feedback network (hormonal crosstalk) that regulates autophagy, ROS, the cytoskeleton, and the cell wall.

Salicylic acid (SA) and jasmonic acid (JA) with ethylene (ET) act antagonistically (SA → biotroph defense; JA/ET → necrotroph defense), whereas JA and ET function synergistically. ABA plays a central role under drought, salinity, and temperature stress, enhancing JA/ET responses while suppressing SA-dependent pathways. GA and IAA direct resources toward growth, attenuating defense programs. Brassinosteroids (BR) and cytokinins (CK) act as auxiliary regulators, enhancing PTI signaling and tissue recovery.

Positive feedback loops, such as PRR signaling via the $Ca^{2+}$ → ROS → $Ca^{2+}$ cascade, amplify the strength and dynamics of defense responses: each $Ca^{2+}$ wave triggers a ROS burst, which in turn induc-

es a new $Ca^{2+}$ influx, generating a rapid and effective "alarm signal." A similar amplification occurs during cell wall modifications: DAMPs released from damaged cell walls activate PRRs, locally reinforcing defense mechanisms.

At the same time, the system incorporates negative regulation, with autophagy acting as a buffer that suppresses excessive ROS accumulation. This prevents oxidative stress and uncontrolled PCD, thereby preserving cell viability.

Hormonal antagonisms, particularly the SA–JA/ET interplay, serve as a key regulatory block, guiding strategy selection: SA dominance promotes cell survival under biotrophic infections or moderate abiotic stress, whereas JA/ET dominance triggers PCD and massive autophagy in response to necrotrophic pathogens or severe damage. Additional integration arises from mutual reinforcement between the cytoskeleton and autophagy: microtubules and actin filaments direct autophagosome trafficking, while autophagy maintains cytoskeletal stability, enhancing the efficiency of defense mobilization.

Together, these mechanisms enable the model to function as a flexible, adaptive system: it rapidly activates defense responses, prevents excessive tissue damage, and allows the plant to optimize its response strategy according to the nature and intensity of the threat.

**Multistable immune and stress defense network in plants**

The activity of functional nodes and the interactions among modules in the integrative model of plant immunity and stress tolerance form a multistable network that protects the plant from abiotic and biotic stress factors. Each module–from PRR signaling and autophagy to hormonal regulation, antioxidant systems, and phase-dependent response dynamics–not only performs its specific function but also integrates into a broad system of intercellular and intracellular signaling.

Table 3, which describes the functional nodes of the integrative model of the multistable immune and stress defense network, details the key signaling and metabolic pathways, including examples of genes and proteins responsible for each module, and outlines the consequences of their activation for the cell. This approach illustrates the logic of intermodular integration, where changes in one node of the cellular defense network can influence the state of other modules, thereby establishing adaptive multistability within the system.

**Table 3**
Functional nodes and key proteins/genes demonstrating how diverse signals are integrated into the plant defense network, enabling dynamic cellular adaptation to biotic and abiotic stresses

| Node / Signal | Function | Example Genes / Proteins | Intermodular Integration |
|---|---|---|---|
| RNS / NO | oxidative signal, induction of HR / PCD | NOA1, NR, RBOH | links with ROS, HR, autophagy |
| SnRK1–TOR | energy control, growth ↔ defense balance | SnRK1, TOR, RAPTOR | regulates autophagy, JA/SA shift |
| UPS / ERAD – UPR | proteostasis, protein quality control | BiP, PDI, ATF6, IRE1 | supports PTI/ETI and stress priming |
| Trafficking / ESCRT | vesicular transport, autophagic vesicles | VPS4, CHMP4 | directs ROS and peptide signals |
| $Ca^{2+}$ waves | secondary messenger | CNGC, GLR, Calmodulin | activates PTI, HR, systemic signaling |
| AQP–$H_2O_2$ | water and ROS transport | PIP1;2, PIP2;1 | systemic ROS signaling, ABA regulation |
| CWI sensing | detection of cell wall defects | WAK1, THE1 | DAMPs → PTI, links with JA/SA |
| Lipid signaling | phospholipids, PA, DAG in stress signaling | PLDα, DGK, PA-binding proteins | modulates ROS, $Ca^{2+}$, HR |
| Stomatal immunity | regulation of gas exchange & ABA / JA / ROS | OST1, SLAC1, MYB60 | coordinates ABA, JA, SA, osmoregulation |
| Plasmodesmata (PD) | intercellular signal communication | PDLP1, Callose synthase | translocation of ROS, SA, peptides, RNA |
| PTI–ETI | integration of surface and intracellular immunity | FLS2, BAK1, NLRs (CNL/TNL), EDS1, PAD4 | links with HR, ROS, JA/SA, autophagy |
| Epigenetic memory | priming, transgenerational tolerance | HDA6, MET1, DRM2 | modulates SA/JA sensitivity, ISR |
| Microbiome / ISR | local and systemic induced resistance | NPR1, MYC2, JA/ET signaling | JA priming, cross-stress tolerance |
| Light / Circadian | diurnal rhythms, SA/JA shift | CCA1, LHY, TOC1 | influences HR, PTI, SA/JA balance |
| Osmolytes | protection under drought / salinity | P5CS, LEA, ProDH | coordinates ABA, AQP, ROS |

*Note*: all nodes interact within a multistable network; the "growth", "SA-dominant", "JA/ET-dominant", or "ABA/abiotic-dominant" state is determined by the combination of internal and external signals and activation thresholds.

The following Table 4 summarizes the key triggers and threshold conditions that determine when a cell switches between different states: survival, mass defense, HR/PCD, or priming. For example, energy deficit through SnRK1 activation and TOR inhibition stimulates autophagy; prolonged accumulation of NO and ROS triggers HR/PCD; and increased AQP activity ensures systemic ROS signal propagation. These threshold rules highlight the multistability of the network, demonstrating that the plant's response to biotic and abiotic stresses is non-linear and depends on the combination of signals and the internal cellular state. Thus, the sequence of tables (from a general overview of modules to functional nodes and threshold mechanisms) forms a coherent, scientifically grounded model of plant immune and stress architecture. It integrates cellular, tissue, and systemic levels, reflects connections with metabolic and hormonal contexts, and accounts for network dynamics and multistability. This demonstrates how local, regulatory, systemic, and effector nodes are interconnected through threshold logic, providing flexibility and resilience to the plant's immune architecture.

**Table 4**
Threshold logic ("switch logic")

| Trigger | Threshold / Condition | Outcome |
|---|---|---|
| Energy deficit (SnRK1↑ / TOR↓) | low resources | autophagy ↑, metabolite redistribution |
| NO↑ + ROS↑ (sustained) | accumulation | HR / PCD, local cell death |
| AQP↑ | increased water conductivity | systemic ROS signal, ABA coordination |
| CWI defect | cell wall damage | DAMPs → PTI, JA/SA activation |
| Evening circadian phases | daily rhythms | SA shift / JA activation |
| ISR | presence of beneficial microbiome | JA priming, reduced energy expenditure |

*Note*: these thresholds establish dynamic multistability, where the plant can occupy different states depending on the combination of stress signals, resource availability, and circadian phases.

**Modules, nodes, and switch logic in the integrative model of plant immune and stress responses**

In the context of the integrative model of plant immune and stress responses, the concepts of module, node, and switch (threshold) logic are central for describing the organization of signaling networks. A module represents a functional part of the system that performs a relatively autonomous role. It consists of a group of genes, proteins, or cellular structures that closely interact with each other to implement a specific function. Examples of such modules include the reactive oxygen species (ROS) signaling module and the hormonal response module, which encompasses signaling pathways of salicylic acid (SA), jasmonic acid (JA), and abscisic acid (ABA).

A node is a key point of integration or control that coordinates the activity of different modules and directs their signals appropriately. It functions as a "decision-making center," transforming incoming

stimuli into specific output responses. Typical nodes in biological systems include pattern-recognition receptors (PRRs) that sense MAMP and DAMP signals, as well as MAPK cascades, which transmit signals from receptors to effector responses.

Threshold logic defines the rules for node activation depending on the intensity of incoming signals. When the signal reaches or exceeds a certain threshold, the corresponding node is activated. This logic can take several forms: threshold, where the response occurs after the signal reaches a critical level; integrative, where multiple signals are summed to reach the activation threshold; and binary (On/Off), where the response is discrete ("on" or "off"). For example, the activation of the salicylic acid signaling pathway occurs when ROS levels exceed a critical threshold, triggering plant defense responses.

Thus, modules provide specialized functions, nodes integrate and coordinate information flows, and threshold logic regulates the conditions and nature of activation of protective mechanisms.

**General concept of the multistable plant immune and stress defense network**

The plant immune and stress architecture network is dynamic and multistable, capable of existing in multiple stable states that prioritize either growth or defense (growth-dominant, SA-dominant, JA/ET-dominant, ABA/abiotic-dominant). It integrates local and systemic signals, combines biotic and abiotic stress inputs, and incorporates the cell's metabolic status, forming an adaptive and flexible system.

Modules act as autonomous functional units, such as ROS signaling, hormonal cascades, and autophagy, while nodes serve as integration centers that direct information flow. Modules and nodes interact via signaling cascades and secondary messengers, creating a dynamic multistable network.

At the core of the network is autophagy, which functions as a central hub and switch, interconnected with other modules (ROS/antioxidant systems, cytoskeleton, cell wall, PRR signaling, and hormonal regulation) as well as with key nodes like the TOR pathway and the "outer ring" of modulators: epigenetics, metabolism, microbiome, photoperiod, and ER stress (endoplasmic reticulum).

This architecture allows plants to flexibly adjust their defense and growth strategies depending on the combination of internal and external cues, ensuring survival and optimal performance under multifactorial stress conditions.

*TOR pathway (target of rapamycin).* The TOR kinase acts as a central sensor of the cell's energy and nutrient status. Under favorable conditions, TOR is active, promoting growth, protein biosynthesis, and suppressing autophagy. Under stress or resource limitation (e.g., pathogen attack, drought, nitrogen or sugar deficiency), TOR is inactivated, while SnRK1 and GCN2 signal energy deficiency. This lifts the inhibition on autophagy and shifts the cell into a resource-conservation mode.

Thus, TOR functions as a switch between growth and defense programs, coordinating the balance between cellular proliferation and survival mechanisms under varying environmental and metabolic conditions.

*"External regulatory ring".* The external regulatory ring comprises epigenetic mechanisms, metabolism, the microbiome, photoperiodic and circadian rhythms, endoplasmic reticulum (ER) stress, and other environmental factors. Epigenetic regulation (including DNA methylation, histone modifications, and non-coding RNAs (miRNAs, siRNAs)) modulates the expression of immunity-related genes, establishes stress memory, and enables faster and stronger responses upon repeated challenges. These mechanisms influence the transcription of ATG autophagy genes, PRR receptors, and antioxidant enzymes.

Metabolism is tightly linked to the TOR/SnRK1 pathways and determines the allocation of carbon and nitrogen resources between growth and defense. It encompasses the biosynthesis of phenylpropanoids, flavonoids, lignin, and other defense-related metabolites. Metabolic sensors such as SnRK1 and GCN2 can trigger autophagy to recycle damaged proteins and organelles.

The microbiome, particularly root-associated PGPR and mycorrhizal fungi, induces systemic resistance (ISR) predominantly via JA/ET signaling pathways and antioxidant systems, acting as an "external module" of the plant immune system that can either enhance or attenuate plant resilience.

Photoperiod and circadian rhythms influence pathogen sensitivity over the diurnal cycle. Many immunity-related genes, including PRRs and ROS-metabolizing enzymes, are under circadian control. During the dark phase, SA-dependent pathways predominate, providing effective defense against biotrophs, whereas during the light phase JA/ET pathways are more active, conferring protection against necrotrophs, thus optimizing defense relative to environmental conditions.

ER stress arises during infection or abiotic damage due to the accumulation of misfolded proteins. The unfolded protein response (UPR) is activated to alleviate ER burden; if insufficient, autophagy or programmed cell death (PCD) is triggered. Because PRRs and numerous defense proteins traffic through the ER, its functional status critically influences immunity.

Additional environmental factors, including temperature, humidity, osmotic stress, and mechanical damage, modulate immune responses indirectly via hormonal and metabolic pathways. Collectively, the external ring acts as a higher-level regulatory layer, fine-tuning the core immune network according to environmental conditions. TOR signaling and ER stress exert the most direct control over autophagy and PCD, whereas other factors act more indirectly (Table 5).

The immune and stress-protection architecture network in plants consists of multiple functional modules and nodes:

1. Trigger signals – PRR signaling recognizes MAMPs/DAMPs and activates $Ca^{2+}$-dependent kinases, ROS, and MAPK cascades, enabling rapid mobilization of defense mechanisms.

2. Logistics and damage control – autophagy degrades damaged organelles and proteins, regulates hormonal pathways (SA, JA, ET, ABA), and is activated under energy deficit conditions (SnRK1↑ / TOR↓), mitigating ROS-induced damage and maintaining the growth-defense balance. The cytoskeleton (actin filaments, microtubules) facilitates vesicle, enzyme, and autophagosome transport, coordinating papillae formation, callose deposition, and lignin delivery.

3. Sensor nodes and barriers – The cell wall (CWI sensing) detects defects and generates DAMPs, activating PRR-PTI signaling (CWI defect → DAMPs → PTI). Stomatal immunity regulates gas exchange via ABA, JA, and ROS to control transpiration.

4. Hormonal regulators – SA, JA, ET, ABA, IAA, BR, GA, and cytokinins coordinate cellular defense strategies by integrating biotic and abiotic stress signals. Circadian rhythms modulate SA/JA activity, particularly during evening phases.

5. Oxidative and chemical signals – RNS/NO, when accumulated for prolonged periods in combination with ROS, induce HR/PCD. Antioxidant systems (SOD, CAT, APX, GPX, ascorbate, tocopherols) buffer ROS, serving as a "dampener" between signaling and damage. AQP–$H_2O_2$ mediates systemic ROS and water transport, coordinating intercellular signaling.

6. Metabolic and energy nodes – SnRK1–TOR regulates the growth-defense balance, determining autophagy intensity. UPS/ERAD and UPR pathways ensure protein quality control and maintain proteostasis.

7. Systemic integration and communication – vesicular trafficking (traffic/ESCRT) delivers autophagosomes and receptor vesicles. Plasmodesmata facilitate intercellular transmission of SA, JA, ROS, and peptides. PTI–ETI integrates surface and intracellular immune responses, while the microbiome (ISR) enhances JA priming under limited resources. Light and circadian cues synchronize hormonal and defense responses over time. Osmolytes maintain turgor and antioxidant protection during drought and salinity stress.

8. Epigenetic memory – priming and transgenerational tolerance through chromatin modifications accelerate responses upon repeated stress exposure.

**Threshold logic ("switch logic")**

The plant defense network operates according to a threshold-based switching principle, where node activation depends on specific

signals and the cell's resource status. This network does not function in an "all-or-none" manner. It is multistable: the cell can adapt by activating only a subset of modules depending on the context (stress type, intensity, time of day, resource availability). This confers dynamic adaptability and resilience to the plant. Such logic allows the network to respond specifically to the current context, activating only the necessary modules, and ensures dynamic multistability: cells can maintain viability under mild stress or induce PCD/mega-autophagy under severe pathogen or abiotic pressure. Table 6 systematizes these conditions, demonstrating that the plant defense network does not operate linearly but responds according to the combination of external signals and the internal state of the cell. This underlies its dynamic multistability and flexible adaptation to stress.

**Table 5**
TOR pathway and the "external regulatory ring of plant immune responses"

| Regulator | Main Mechanisms | Consequences for Immunity | Key molecules / examples | Type of stress | Connection with autophagy / PCD |
|---|---|---|---|---|---|
| TOR pathway | TOR kinase is active under sufficient resources: promotes growth, protein biosynthesis, and suppresses autophagy. Under nutrient deficiency, TOR is inactivated, SnRK1 and GCN2 are activated. | switches the cell between growth and defense/resource conservation. | TOR, SnRK1, GCN2, ATG genes | energy, nitrogen, sugar deficiency; infection, drought | direct control of autophagy; TOR inhibition → autophagy activation |
| Epigenetics | DNA methylation, histone modifications, non-coding RNAs regulate immune gene expression and establish stress memory. | faster and stronger response upon re-infection. | H3K9me2, DNA methyltransferases, miR393 | biotic and abiotic stress | regulates ATG and PCD genes, influencing duration and strength of autophagy |
| Metabolism | resource redistribution; synthesis of defensive metabolites. SnRK1 and GCN2 sensors trigger autophagy. | enhanced defense; accumulation of flavonoids, lignin. | PAL, SnRK1, GCN2 | nutrient deficiency, energy stress | autophagy recycles damaged proteins and organelles |
| Microbiome | PGPR and mycorrhizae activate systemic resistance via JA/ET and antioxidant pathways. | strengthens immunity. | *Pseudomonas*, *Bacillus*, *Glomus* | biotic interactions | indirect connection – some microbial signals can induce autophagy |
| Photoperiod & Circadian Rhythms | regulate daily sensitivity to pathogens; SA pathways active at night, JA/ET during the day. | optimize immune responses according to the time of day. | CCA1, LHY, TOC1 | light/dark cycles, seasonality | circadian regulation of ATG genes and ROS-dependent PCD |
| ER Stress | accumulation of misfolded proteins → UPR; under excessive stress – autophagy and/or PCD. | determines functionality of PRRs and defense proteins. | BiP, bZIP60, IRE1 | infection, heat, osmotic stress | direct induction of autophagy and PCD under irreversible stress |
| Other Factors | temperature, humidity, mechanical damage integrated via hormonal and metabolic pathways. | modulate immune response levels. | HSP70 (heat stress), RD29A (osmotic stress) | abiotic stresses | can trigger PCD or autophagy via ROS and hormonal pathways |

**Table 6**
Threshold logic ("switch logic")

| Trigger | Threshold / Condition | Outcome |
|---|---|---|
| ↑SnRK1 / ↓TOR | energy deficit | ↑ Autophagy |
| ↑NO + ↑ROS | prolonged accumulation | HR / PCD |
| ↑AQP | enhanced water conductivity | Systemic ROS signaling |
| CWI defect | cell wall damage | DAMPs → PTI |
| Evening circadian phases | daily rhythms | SA bias / JA activation |
| ISR | presence of beneficial microbiome | JA priming under limited resources |

Based on the integrative model of the plant immune and stress-protection network, all functional modules and nodes can be categorized as follows:

1. Initiation nodes (PRR, CWI, RNS/NO, $Ca^{2+}$ waves) generate local stress signals and activate primary ROS, MAPK, and $Ca^{2+}$ cascades.

2. Regulatory nodes (SnRK1–TOR, autophagy, hormones, UPS/ERAD, cytoskeleton) coordinate the balance between growth and defense, maintaining proteostasis and managing the logistics of defense components.

3. Systemic nodes (AQP–$H_2O_2$, plasmodesmata, microbiome/ ISR, light/circadian rhythms) ensure intercellular and inter-tissue signal coordination and hormonal modulation.

4. Effector nodes (stomatal immunity, PTI–ETI, osmo-metabolites, phase dynamics, epigenetic memory) execute physical, chemical, and hormonal defense strategies, determining survival or PCD as required.

Node activation follows threshold-based rules that determine when a cell switches between different states:

– energy deficit (↑SnRK1 / ↓TOR) → induction of autophagy and resource redistribution;

– prolonged accumulation of NO and ROS → HR/PCD;

– increased AQP activity → systemic ROS signal propagation;

– cell wall defects → DAMP-dependent activation of PTI;

– evening phases of the circadian cycle → shift in SA/JA activity;

– presence of the microbiome (ISR) → JA priming and reduced energy expenditure.

Threshold logic ("switch logic") integrates these nodes. Only modules and nodes that exceed critical signal or resource thresholds are activated, ensuring adaptive multistability:

– mild stress → activation of PRR, CWI, moderate ROS → survival and priming;

– moderate stress → additional engagement of SnRK1–TOR, autophagy, hormonal modulation → balance between growth and defense;

– severe stress or pathogen attack → prolonged ↑ROS + ↑NO, phase-specific JA/ET dominance → HR/PCD or mega-autophagy.

Thus, grouping modules and nodes into functional categories reflects the dynamic nature of the plant stress-response network with multiple stable states, where each node interacts with others and state transitions are governed by both stress-derived signals and the energetic and hormonal balance. This framework allows simultaneous visualization of initiating signals, regulatory nodes, systemic effects, and threshold logic, highlighting the integration of biotic and abiotic factors into a multistable adaptive defense network.

The integration of modules, functional nodes, and switch logic enables their consolidation into a unified multistable immune and stress-response network. In this model, initiating nodes (PRR, CWI sensing, $Ca^{2+}$ signals, RNS/NO) trigger local signaling cascades, including ROS, MAPKs, and secondary messengers. Regulatory nodes (SnRK1–TOR, autophagy, hormonal modules, UPS/ERAD, cytoskeleton) coordinate the balance between growth and defense, maintaining proteostasis and energetic homeostasis. Systemic nodes (AQP–$H_2O_2$, plasmodesmata, microbiome/ISR, photoperiod and

circadian rhythms) ensure intercellular and tissue-level integration of signals. Finally, effector nodes (stomatal immunity, PTI–ETI, osmometabolites, epigenetic memory) execute protective responses and define long-term adaptation.

Threshold logic ("switch logic") determines the operational mode of the system (from survival and growth to extensive defense or programmed cell death). This provides a comprehensive, integrative view of the plant multistable defense network, where local, regulatory, systemic, and effector nodes are interconnected through threshold mechanisms, ensuring flexibility and resilience of the plant immune architecture.

Overall, this conceptual framework constitutes a fully developed system-level map of plant immunity, in which each component (autophagy, cytoskeleton, cell wall, ROS/antioxidant system, hormones, epigenetics, and metabolism) functions not in isolation but as part of a cohesive network.

**Scientific and practical impact of studying autophagy as an integrator of stress responses in plants**

*Scientific significance of autophagy in the regulation of plant stress responses.* Investigating autophagy within the context of plant stress physiology holds substantial potential for shaping new conceptual frameworks. Traditionally, autophagy has been considered a "cellular recycling" mechanism, responsible for the degradation of damaged organelles, proteins, and other macromolecules (Petersen et al., 2024; Gross et al., 2025; Feng et al., 2025). Our approach expands this view by proposing autophagy as a universal integrator of plant immunity and stress resilience. This implies that autophagy not only performs degradative functions but also coordinates antioxidant systems, hormonal regulation, and the cytoskeleton, thereby ensuring systemic cellular adaptation under stress conditions. Consequently, the scientific impact lies not merely in refining the functional understanding of autophagy, but in shifting the paradigm from perceiving autophagy as a local mechanism to recognizing it as a global integrator of protective and adaptive processes in plants.

The novel integrative model of plant defense against pathogens and stresses is based on the concept of a multi-level system of interconnected modules that collectively determine the nature and efficiency of plant immune responses, forming a dynamic, multi-tiered signaling-metabolic network. Autophagy serves as the central integrator of this model, coordinating information flow between cellular sensors, hormonal networks, cytoskeletal structures, and cell wall barrier mechanisms. Within this integrative framework, plant defense is viewed not as a set of isolated reactions, but as a dynamic, multilevel signaling–metabolic network that links different organizational levels through a central regulatory module (autophagy) which balances cellular survival and programmed cell death.

Analysis of interactions between autophagy, ROS signaling, hormonal networks (salicylic acid, jasmonates, ethylene, abscisic acid, etc.), and the cytoskeleton allowed us to develop an integrative model of cross-talk. This model explains how plant cells establish a hierarchical response to various stresses (biotic and abiotic) and which signaling cascades dominate under specific conditions. It opens a pathway toward a systems biology perspective of autophagy, wherein autophagy is considered a central module of the regulatory network.

Investigation of the effects of mega-autophagy on SAR activation led us to identify a novel concept–"autophagic priming." In plant physiology, priming is known as a phenomenon in which a prior mild stress stimulus enhances tolerance to subsequent challenges (Hilker & Schmülling, 2019). However, prior to our study, the literature had not described the effect of moderate autophagy activation as a priming mechanism. Thus, the term "autophagic priming" denotes a new concept in which moderate autophagy activation increases the plant's preparedness for stress. Unlike classical induced resistance, where key biochemical reactions are activated only after a stress signal appears, autophagic priming allows cellular networks to be pre-configured for a rapid and controlled response.

Autophagic priming defines a cellular state in which moderate activation of autophagy prepares the cell for a more effective response to future stress challenges. In contrast to classical induced resistance, where defense mechanisms are triggered only post-stimulus, autophagic priming enables pre-conditioning of cellular networks, providing a faster and more controlled response. Introducing this concept highlights a fundamentally new approach that integrates cell biology, stress physiology, and biotechnology.

Mechanistically, autophagic priming involves moderate activation of ATG cascades, forming a basal pool of autophagosomes without inducing programmed cell death. This ensures pre-allocation of resources and degradation of damaged components, reducing the risk of oxidative and abiotic stress. Controlled ROS levels serve a signaling role without causing toxicity, while the integration of autophagy with the cytoskeleton and hormonal networks (SA, JA, ET) enhances autophagosome trafficking and coordination of immune responses.

As a result, autophagic priming increases cellular readiness against both biotic (pathogens, parasites) and abiotic (drought, temperature stress) factors, providing a basis for breeding stress-tolerant cultivars with specific markers (e.g., ATG8 flux, ROS profiles) and opening prospects for biotechnological applications, including autophagy regulators. This approach establishes a new integrative direction at the interface of cell biology, stress physiology, and biotechnology, paving the way toward systems-level understanding of preparatory cellular mechanisms and strategies for pre-conditioning plants to enhance stress resilience.

**Applied aspects of autophagy research in the context of plant stress resilience**

The developed integrative model of plant resistance to pathogens and environmental stresses not only elucidates fundamental mechanisms but also serves as a platform for practical applications, including the development of bioprotectants, breeding of stress-tolerant cultivars, and precise regulation of "immune states" in crop production. It provides deeper insights into the role of cell-associated mechanisms in shaping defense responses and non-specific resistance, and explains why these processes may be insufficiently effective against certain microorganisms and viruses.

This scientific framework is highly relevant for modern plant breeding and for devising new crop protection strategies–from developing cultivars and hybrids with enhanced resistance to implementing next-generation (fourth-generation) plant protection products. Such agents can activate plant immune responses either independently or in combination with conventional fungicides, reducing the risk of rapid resistance development to systemic treatments.

**Next-generation agrotechnologies: Field applications of the integrative model**

The integrative model of plant immune architecture provides a tool for adaptive immune management in crops. This means that farmers or agronomists can not only activate plant immunity but also direct it toward specific scenarios (biotrophic, necrotrophic, viral, or abiotic stress). This approach paves the way for intelligent bioprotectants that function as "immunomodulators" rather than conventional protective agents. Instead of the classical "pesticide → pathogen" approach, the concept of immunomodulators emerges–bioproducts that fine-tune the balance between ROS, autophagy, hormonal circuits, and regeneration.

The use of autophagy inducers accelerates adaptive responses, ensuring rapid formation of defense mechanisms before stress onset. This enables the development of innovative crop treatment schemes, in which plant preparation for stress is performed preventively rather than post facto. A modular approach allows the combination of different inducers to achieve an optimal balance between ROS, autophagy, and immune responses.

Key principles of autophagic priming and innovative strategies for enhancing plant stress tolerance include preventiveness, modularity, use of live microbial preparations, and integration with modern

AgriTech technologies. Preventiveness implies establishing an immune-ready state before pathogen attack or stress exposure, ensuring a rapid and controlled response. Modularity enables combining various inducers to precisely balance major hormonal regulators (salicylic acid, jasmonates, ethylene, abscisic acid, auxins, brassinosteroids, and cytokinins). Live microbiome-based products act as "bio-inducers" of immune cascades, enhancing natural resistance mechanisms. Finally, integration with AgriTech involves the use of sensors and predictive models for timely application of bioproducts, allowing personalized immune responses and improving the efficacy of biological crop protection.

In practical applications, autophagic priming enables the formation of preventive immunity across different plant groups, adapting interventions to stress type and crop characteristics. Cereal crops (wheat, barley, rice) face key stresses such as powdery mildew (biotrophs), septoria (hemibiotrophs), and fusarium (necrotrophs). SA inducers, such as salicylates and BTH, activate the SA module, stimulate selective autophagy, and suppress biotrophic pathogens. DAMP elicitors, including chitosan and β-glucans, promote a shift toward JA/ET responses, induce spike lignification, and limit necrotroph development. ROS primers in microdoses (e.g., $H_2O_2$) enhance immune system readiness without inducing stress overload, while brassinosteroids support root growth and recovery post-infection.

Vegetable crops (tomato, cucumber, cabbage) commonly encounter late blight (hemibiotroph), alternaria (necrotroph), and viral infections. Viral threats are managed through SA-dominant strategies combined with polyphenol-based bioprotectants that activate selective autophagy for the elimination of viral complexes. Necrotrophic infections, such as alternaria, are controlled via JA/ET priming using methyl jasmonate, ethylene releasers, or laminarin, inducing ROS bursts and lignin-mediated wound closure. In cases of mixed infections or abiotic stress, combined microdoses of SA and JA balance response scenarios, while cytokinins stimulate cell division in growth zones and tissue recovery.

In horticultural systems such as vineyards, apple orchards, and berry plantations, biotrophic pathogens like powdery mildew (*Oidium* spp.) are managed through early applications of SA inducers. Necrotrophic pathogens, including *Botrytis cinerea*, are controlled by post-injury treatments with chitosan or laminarin. Microbial bioproducts (e.g., *Trichoderma*, *Bacillus* spp.) perform dual functions, acting both as PTI elicitors and as modulators of ROS via autophagy, while IAA-based treatments accelerate wound healing and restore cell wall integrity after mechanical damage or hail.

Under abiotic stresses such as drought, salinity, or frost, mild autophagy inducers (flavonoids, *Trichoderma* metabolites) are applied to facilitate the degradation of damaged organelles and regulate ROS. ROS priming with low doses of $H_2O_2$ or $ZnO/SiO_2$ nanoparticles prepares the antioxidant system, ABA stimulation promotes stomatal closure and enhances salt tolerance, and bacterial exopolysaccharides stabilize the cytoskeleton and accelerate cell wall recovery.

In field conditions, biotic and abiotic stresses often co-occur, for example, drought combined with fusarium infection. The integrative model enables the creation of a treatment matrix, where SA is applied against biotrophs, JA/ET against necrotrophs, and ABA for abiotic stress, allowing microdose combinations tailored to complex scenarios. Digital sensors and predictive models monitoring humidity, temperature, and ROS signals enable precise and timely application of immunomodulators. This integrative, preventive, and modular strategy exemplifies next-generation agrotechnology, leveraging autophagic priming and fine hormonal tuning to optimize plant stress resilience across horticultural crops.

In summary, the integrative model shifts plant protection from reactive to preventive, where bioproducts and microbiomes function as precise regulators of the balance between growth and defense mechanisms.

**Prospects for next-generation autophagy inducers to enhance systemic crop resilience**

Understanding the interplay between the cytoskeleton, autophagy, and the cell wall in plant immunity opens avenues for the development of autophagy-inducing bioproducts. Such agents have the potential to enhance crop resilience against combined biotic and abiotic stresses, optimize ROS homeostasis, and strengthen local cell wall structures, thereby supporting the selection of more tolerant cultivars. The contemporary concept of integrative plant immune architecture provides the basis for "fourth-generation bioproducts," which function not as broad-spectrum stimulants but as precise regulators of intracellular balances. Traditional systemic acquired resistance (SAR) inducers, such as salicylic acid, activate plant defenses via general immune pathways, often without fine control over cellular processes. In contrast, next-generation strategies aim for targeted activation of autophagy and ROS networks, preparing cells in a controlled manner for impending stress challenges. This approach enhances crop resistance to biotic factors (pathogens, parasites) and abiotic factors (drought, temperature fluctuations).

The concept of intelligent bioproducts envisions agents that simultaneously regulate autophagy, modulate ROS profiles, and maintain cytoskeletal stability – critical for autophagosome transport and defense protein trafficking. Mild autophagy inducers (natural polyphenols, low-dose rapamycin, microbial/fungal metabolites) facilitate the removal of damaged organelles and excessive ROS, preventing necrosis. Controlled ROS priming (e.g., low doses of $H_2O_2$ or metal oxide nanoparticles in microconcentrations) delivers a signal pulse that activates the immune network without causing excessive oxidative damage. Cell wall elicitors (chitosan, β-glucans, oligogalacturonides) trigger DAMP-signaling cascades, reinforcing physical barrier defenses.

In combination, these interventions allow controlled switching of defense modes: SA-dominant for protection against biotrophs and viruses, JA/ET-dominant against necrotrophs and pests, and a balanced response under abiotic stress (drought, salinity). Such bioproducts can be regarded as biotechnological tools for stress priming, enhancing yield and resilience without imposing chemical loads on crops.

Unlike conventional agents that mainly activate broad SAR or ISR pathways, next-generation autophagy inducers precisely modulate autophagic processes, balancing defense activation with the risk of cellular self-damage. This enables plants to optimally allocate their own defense resources, increasing resistance to diverse pathogens and abiotic stressors while maintaining growth and metabolic efficiency.

The proposed approach is expected to lead to the development of novel, targeted, and controllable bioproducts for agriculture, capable of enhancing crop stress tolerance and productivity. Implementation of this strategy establishes a methodology for preventive preparation of plants for stress conditions and provides a foundation for further research on autophagy as a central regulator of plant viability and yield.

A key feature of this concept is the focus on next-generation bioproducts that combine activation of systemic and local immunity with precise regulation of intracellular processes, particularly autophagy. This regulation allows plants to respond flexibly to challenges–surviving moderate pathogen attacks while initiating programmed cell death (PCD) only under critical damage. The integration of synthetic compounds and nanomaterials enables targeted modulation of ATG cascades, which previously could be controlled only under laboratory conditions.

Additionally, the use of inducers of secondary metabolites and oligosaccharides provides environmentally safe control of fungal and bacterial pathogens, minimizing additional stress on the plant. The result is a multilevel strategy that combines modern biotechnological approaches with natural regulatory mechanisms, opening avenues for a new generation of precise and controllable immune modulators.

From an economic perspective, the global crop protection market exceeds USD 70 billion per year, with bioproducts demonstrating stable annual growth of 10–12%, projected to reach USD 15–20 billion by 2030. In Ukraine, the agribusiness sector spends approximately USD 1.2–1.5 billion annually on pesticides. Fourth-generation bioproducts can induce plant immunity independently or in combination with conventional fungicides, preventing rapid development

of resistance to systemic agents. If their application reduces protection costs by 20–30%, this would result in savings of USD 250–450 million per year. Moreover, reduced losses from diseases and stress factors could increase crop yields by 5–10%, potentially adding USD 1–2 billion to agro-export revenues (currently around USD 20 billion annually).

Overall, the total economic effect of implementing fourth-generation bioproducts in Ukraine could reach USD 1.3–2.5 billion annually through combined cost savings and increased revenues. Globally, this figure may rise to USD 15–25 billion per year, highlighting their potential as a key driver of transformation in the global crop protection market. According to our projections, the concept of autophagy priming enables farmers not only to reduce protection costs but also to ensure yield stability under challenging climatic conditions.

## Breeding potential of autophagy and stress-resistance markers in plants

The breeding relevance of autophagy research in controlling plant stress responses lies in the possibility of using molecular markers to accelerate the selection of stress-tolerant crops. One of the key markers is ATG8 flux, which reflects the level of autophagy activity: elevated values indicate cellular readiness for stress conditions and the plant's ability to rapidly mobilize defense mechanisms against pathogens or abiotic factors.

Another important predictor is the ROS profile, based on the ratio of $H_2O_2$ to antioxidant enzyme activity (SOD, CAT, APX). An optimal balance of these indicators reflects a controlled stress response, where ROS function as signaling molecules without causing excessive toxicity. Additionally, cytoskeleton stability (particularly the organization of actin filaments and microtubules) serves as a critical marker, ensuring efficient transport of autophagosomes and defense proteins to vacuoles and the cell wall.

The use of such molecular markers forms the basis for marker-assisted selection (MAS). Incorporating ATG genes and related markers into breeding programs allows significant acceleration of the selection process, increasing precision and reducing the number of generations required to obtain cultivars with high stress tolerance.

Based on these approaches, new breeding programs are emerging that focus on multi-stress resistance. These programs combine tolerance to abiotic factors (drought, temperature fluctuations), biotic threats (parasites, fungal infections), and oxidative stress induced by ROS. Integration of molecular markers into these programs enables the combination of different defense mechanisms, enhancing the efficiency of developing new stress-resilient cultivars.

## Conclusions

The proposed integrative model of plant immunity represents a multilevel signal-metabolic defense network composed of interconnected modules that collectively determine the nature and effectiveness of plant defense responses, with autophagy serving as the central integrator. Its key advantage lies in its ability to explain the balance between cell survival, localized cell death, and systemic resistance, which has both fundamental and applied significance for modern biology and agriculture.

The integrative model overcomes the fragmentation of previous PTI/ETI concepts and unifies cellular, organellar, hormonal, and systemic levels of defense into a single immune system. Its practical relevance lies in identifying novel biotechnological targets, including regulators of autophagy, SnRK1/TOR signaling, ROS/NO homeostasis, and epigenetic factors, which can be exploited to enhance stress tolerance in crop plants.

This model establishes a new conceptual understanding of cellular defense responses in plant immunity. Here, the immune response is not viewed as a set of discrete reactions but as a unified signal-metabolic defense network, where sensors, signaling cascades, effectors, hormonal regulators, and epigenetic mechanisms function in close coordination. Autophagy serves as the central integrator, responding to ROS signals, energy sensors, and hormones to determine cell fate: maintaining tolerance, activating HR/PCD, or initiating systemic resistance.

Autophagy acts as a key regulator coordinating plant responses to various stresses – both biotic (pathogens, pests) and abiotic (drought, salinity, temperature fluctuations). Autophagy, the cytoskeleton, the cell wall, and hormonal regulation form a unified integrative defense network. Autophagy maintains the balance between the activation of rapid defense waves (ROS, MAPK) and the protection of cells from self-damage, regulating the choice between cell survival and programmed cell death (PCD). Thus, autophagy integrates signals from different immune modules, acting as a "central hub" in the defense network. Actin filaments and microtubules facilitate the transport of vesicles, autophagosomes, and enzymes to pathogen attack sites, while the cell wall functions as both a physical barrier and a sensor through DAMPs. Together, these components form the implementation arm of the integrative model, translating PRR and hormonal signals into concrete defense structures, such as callose deposition or lignification.

Constructing a systemic model of immune architecture and plant stress resilience that integrates PRR signaling, autophagy, the cytoskeleton, the cell wall, and "hormonal steering" (SA/JA/ET and others) provides a robust foundation for developing fourth-generation biopreparations – autophagy inducers that enhance systemic crop resistance to abiotic and biotic stresses. Such agents can specifically activate SAR/ISR and modulate autophagy, improving crop resilience without harming the plant.

Observed patterns in selective mega-autophagy open a novel avenue in stress biology – autophagy priming, i.e., preconditioning plants with signals or treatments that prepare the immune system for an effective response to future attacks. This strategy enhances crop productivity and resilience by optimizing resource allocation and reducing losses from pathogens and abiotic factors.

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