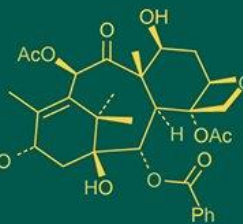
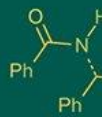


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Molecular defenses: Unraveling plant resistance mechanisms against insect herbivory

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Abstract

As immobile organisms, plants are persistently exposed to a wide array of biotic and abiotic stressors throughout their life cycle. Among biotic challenges, insect herbivory poses a significant threat, contributing to the loss of approximately 20% of global agricultural productivity each year. Insect herbivores display a broad spectrum of feeding strategies, including leaf defoliation, sap-sucking, and phloem-feeding each triggering distinct and often complex defensive responses in plants. These interactions have driven the evolution of sophisticated plant defense mechanisms over millions of years. Herbivore associated elicitors (HAE) are molecules produced by herbivore as Oral secretions. A key aspect of plant defense involves the recognition of herbivore attack through conserved molecules, termed herbivore-associated molecular patterns (HAMPs) and damage-associated molecular patterns (DAMPs). These molecules are detected by plant pattern recognition receptors (PRRs), which activate PRR-triggered immunity (PTI), a defense pathway analogous to pathogen-associated molecular pattern (PAMP)-triggered immunity. The recent discovery of DAMP receptors has provided new insights into the distinct and overlapping roles of DAMPs and HAMPs in mediating plant-herbivore interactions. Despite advances in understanding pathogen-induced responses, a unified conceptual framework, such as the zigzag model established for plant-pathogen interactions, is lacking for plant-insect systems. This review consolidates current insights into insect herbivore classification, plant perception of herbivory, and the spatial and temporal dynamics of induced plant defenses. It further underscores the need for a comprehensive model to elucidate the molecular underpinnings of plant-insect interactions and guide future research in plant defense biology.

Keywords: Insect herbivory, defense signaling, HAMPs, DAMPs, EFN, HAE, PRR, ovipositional fluids, direct defense

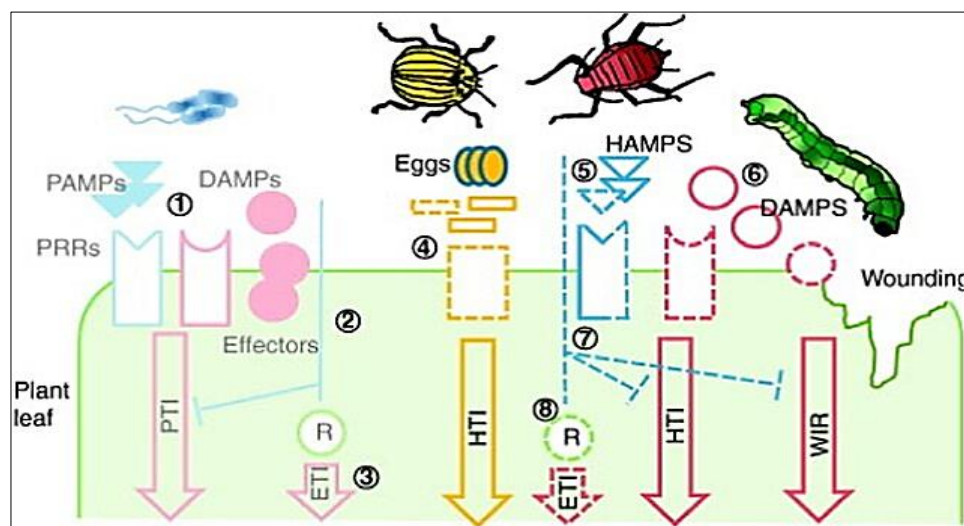
1. Introduction

All living organisms inevitably encounter a range of biotic and environmental challenges throughout their lives. However, due to their sessile nature, plants are uniquely and continuously exposed to a diverse array of biotic and abiotic stressors in their habitats, including attacks by herbivores, drought, salt, UV- radiation, extremely high or low temperatures, and nutritional imbalances in their natural habitats ^[1-3]. Insect herbivory refers to the process by which insects feed on plant- parts, leaves, stems, roots or seeds. Based on their feeding behavior insect herbivores can be: Leaf defoliator (Cause significant damage to plant by chewing or tearing), Rasping and sucking (Suck the liquid content (Sap) from epidermal cells) and Phloem-suckers (Insert stylet between mesophyll cells and into the phloem). Insect herbivores are broadly categorized into generalists (Polyphagous) which feed on multiple plant families of various hosts, or specialists (Monophagous and oligophagous), that feed on one or more plant types belonging to same family. The generalists have ability to tolerate a wide range of defences present in most of the plants, but they are unable to feed on some plants that have evolved more distinctive defence systems. Whereas, specialists depend on a specific variety of host plants that release defence compounds that may also act as ovipositioning cues and feeding stimulants ^[4-6]. It is estimated that each year, phytophagous herbivores destroy one-fifth of the world's total agricultural yield ^[7]. Insect herbivores have exerted selection pressure over millions of years, leading to the development of complex plant defences. Numerous studies have been conducted on the ecology and evolution of plant-insect interactions ^[8]. A zigzag model is frequently used to demonstrate plant immunity to several phyto-pathogens ^[9].

But such a model is not yet fully developed for plant-insect interactions, and it is unclear how applicable it is to them. According to the canonical model of plant herbivore perception, plants perceive herbivory through the binding of herbivore-associated molecular patterns (HAMPs and DAMPs) to pattern recognition receptors (PRRs). Similar to pathogen-associated molecular patterns (PAMPs), insect herbivory frequently produces conserved molecules called herbivore-associated molecular patterns (HAMPs) or damage-

associate molecular patterns (DAMPs) (Fig 1). The pattern recognition receptors (PRRs) in plants are able to recognise these compounds and cause PTI (PRR-triggered immunity), which is analogous to PAMP-triggered immunity. Additionally, the temporal dynamics of plant defence responses against insects are shown, with some defence responses activating within minutes of insect herbivory and others taking days to activate ^[10].

How plant recognizes insect herbivore?



Phyto-hormone signaling and induced resistance

Fig 1: Mechanism of Molecular recognition pathways in plant defense system against pathogens and herbivores

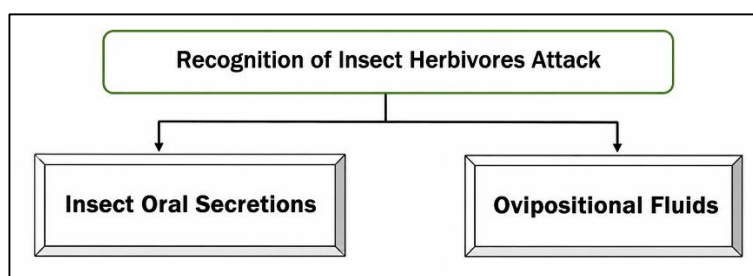
1. PAMPs (Pathogen associated molecular pattern) and DAMPs (Damage associated molecular pattern). These molecular patterns are recognized by PRRs (Pattern recognition receptors) and leads to PTI (PAMP-triggered immunity)
2. Pathogen effectors suppressed by PTI
3. The products of resistance gene recognize effectors leading to ETI (Effector triggered immunity)
4. Oviposition-related compounds are recognized by unknown receptors and trigger defensive responses
5. Likewise HAMPs (Herbivore-associated molecular patterns) are recognized by receptors and lead to HTI (Herbivore triggered immunity)
6. Wounding leads to the release of DAMPs and to WIR (Wound induced resistance)
7. Effector like molecules from insects can suppress HTI and WIR **Broken Lines indicates uncharacterized elements.

Plants rely on both chemical and mechanical cues to recognize herbivores. Early detection is quite crucial for activating an effective defense response against biological threats. Pattern recognition receptors so called PRRs that are located on the plant cell surface detect conserved microbial signatures popularly known as MAMPs/PAMPs (Microbe or Pathogen-associated molecular patterns). Upon recognition of these patterns, the plant activates a defense response known as PAMP-triggered immunity (PTI) as shown in figure 1. In order to initiate effective defense responses, PRRs also recognize damage-associated molecular patterns (DAMPs) which are endogenous molecules released by the plant during infection or tissue damage ^[11]. Pathogens can avoid this innate immune response by delivering effector

proteins that interfere with PAMP-triggered immunity (PTI) upon entering the host cell. In response, certain plant genotypes possess disease-resistance (R) proteins that specifically recognize these pathogen effectors, activating a stronger defense known as effector-triggered immunity (ETI) ^[12]. Though it is believed that PTI/ETI model is too simplistic to understand ^[13]. Now it is utterly possible to make conclusion on the specificity of recognition in plant-pathogen interactions due to presence of specific ligands and receptors involved. Thus it is easy to understand how plants recognize different threats. PTI often triggered on the non-specific recognition of very common microbial chemicals, whereas ETI gets activated by substances which are quite pathogen specific ^[14]. This raises an important question: to what extent can the PTI/ETI model guide researchers on the specificity of plant-herbivore recognition? Unlike pathogens, insects are multicellular and highly complex organisms with diverse life strategies and behaviors. Plants may depend on various cues derived from these behaviors to recognize herbivory and initiate suitable defense responses ^[15] (Figure 1). The earliest stage of plant-herbivore interaction often starts from the moment an insect's tarsi touch the leaf surface. Walking or landing causes mechanical pressure, breaks trichomes, and deposits chemicals from the insect's tarsal pads onto the leaf ^[15]. In order to perceive pressure, plants have developed mechanism. In case of Venus fly trap (*Dionaea muscipula*), for example, when insects trigger the sensory hairs it instantly closes ^[16]. Moreover, non-carnivorous plants are utterly sensitive to touch ^[17]. In certain instances, mechano-stimulation through repeated contact is adequate for stimulating the accumulation of jasmonic acid (JA) ^[18], the precursor of the defense hormone jasmonoyl-L-isoleucine

(JA-Ile). When caterpillars or adult moth break the trichomes on tomato (*Solanum lycopersicum*) leaves, hydrogen peroxide (H_2O_2) is produced, and defence proteinase inhibitors are expressed^[19]. The observed effects

may primarily be attributable to DAMP-like effects, and there is presently no clear cut proof that this kind of "early warning" response is quite unique to any one insect species.



1.1 Insect Oral Secretions

1.1.1 Herbivore-associated elicitors

Herbivore-Associated Elicitors (HAEs) are molecules found in herbivore oral secretions that trigger a range of plant

defense responses. These include the synthesis of secondary metabolites, proteinase inhibitors, and volatile organic compounds that either deter herbivores or attract their natural predators.

Table 1: Herbivore-Associated Elicitors (HAEs), Insect Species, and Host Plant Species

HAEs	Insect species	Plant species
Glucose Oxidase System (GOS)	<i>Helicoverpa armigera</i>	Tomato
β -Glucosidase	<i>Pieris brassicae</i>	Cabbage
Caeliferins	<i>Schistocerca americana</i>	Maize <i>Arabidopsis thaliana</i>
Inceptin	Produced by degradation of a plant ATP synthase during folivory by <i>Spodoptera frugiperda</i>	<i>Vigna unguiculata</i> (Cowpea)
Oligouronides	Produced by degradation of plant cell walls during insect folivory	Tomato

(Bonaventure *et al.* 2011)^[20].

Defense signaling cascades triggered by Elicitors

In plants induced resistance is one of the most crucial defense mechanism that enhances a plant's capability to protect itself against a broad range of pathogens as well as insect pests following the perception of specific stimuli.

Such extend level of resistance, triggered by mostly exposure to infectious agents, is commonly termed as Induced Systemic Resistance (ISR) or we call as Systemic Acquired Resistance (SAR)^[21-22].

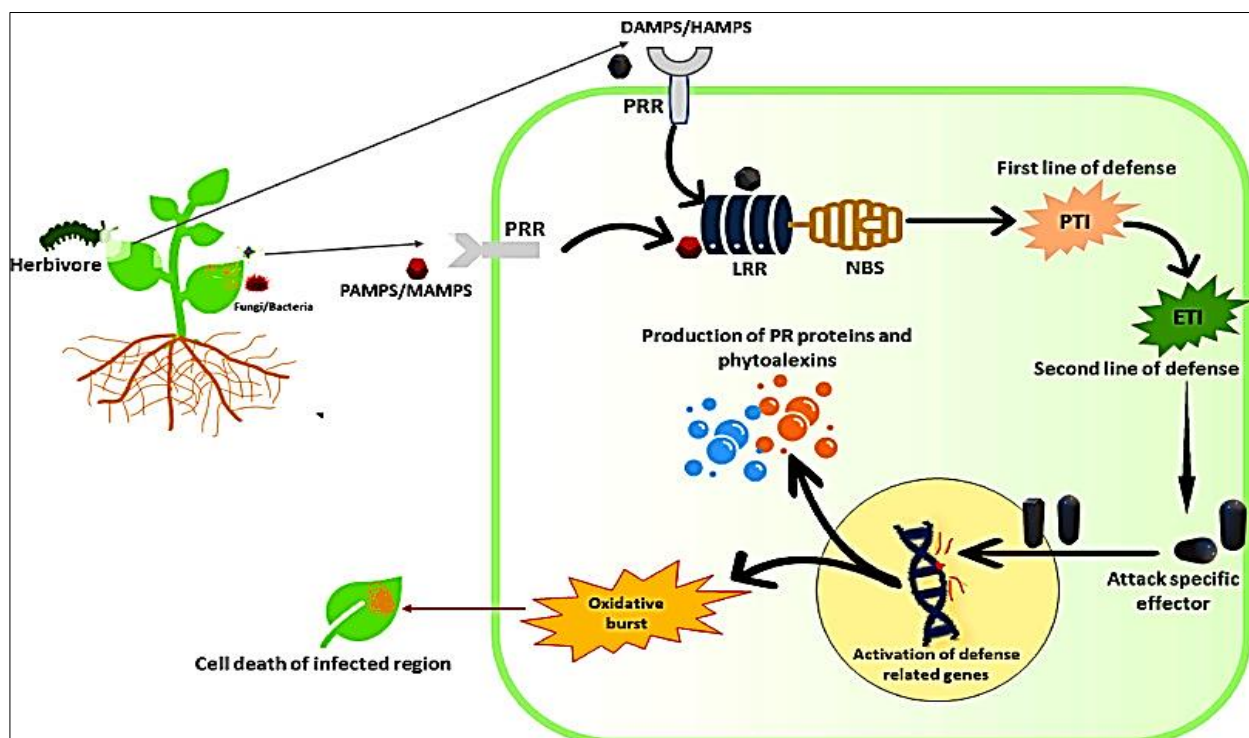


Fig 2: A schematic representation of the elicitor-based defense mechanism shows how plant receptors detect chemical signals released during herbivore attack

Upon perception of these elicitors, receptor molecules activate intracellular signaling cascades, ultimately triggering various defense responses. *PTI: Pattern-triggered immunity * ETI: Effector-triggered immunity *PRR: Pattern-recognition receptor *LRR: Leucine-rich repeat *NBS: Nucleotide-binding site * PAMPs: Pathogen-associated molecular patterns *MAMPs: Microbe-associated molecular patterns *DAMPs: Damage-associated molecular patterns * HAMPs: Herbivore-associated molecular patterns [23].

During plant-microorganism interactions, plant roots release a diverse array of specialized chemical compounds into the soil. These exudates play a key role in attracting microbes toward the roots, facilitating the establishment of a complex and beneficial network between plants and microorganisms [23]. Certain microorganisms enhance plant resistance to a wide range of infectious diseases not by directly antagonizing pathogens, but by activating the plant's own defense mechanisms. These beneficial microbes stimulate key physiological processes that trigger Induced Systemic Resistance (ISR) against various pathogenic species [24]. Plant-induced immunity is triggered by signaling molecules released by microorganisms, which activate the plant's defense responses to protect against biotic stress and pathogen attack [25]. Plant-specific receptor molecules recognize chemical signals released during pathogen invasion. Subsequent to activation, these receptors mediate the initiation of a signaling cascade leading to the activation of defense responses within the plant [26] (Figure 2). A key strategy in plant defense signaling involves altering the ion permeability of the cell membrane. This process begins with receptor activation, which triggers the opening of ion channels across the membrane. This process leads to an influx of calcium ions into plant cells, which subsequently triggers the production of reactive oxygen species (ROS). The accumulation of ROS is a well-documented response in plants during bacterial and fungal pathogen attacks [27]. The accumulation of reactive oxygen species (ROS) triggers an oxidative burst, serving either as a direct defense mechanism or as a signal that activates additional defense responses [28]. This initial oxidative burst plays a crucial role in launching the signaling network required to initiate comprehensive plant defense mechanisms [29]. Programmed cell death (PCD) represents another systemic defense strategy, involving the expression of various genes that contribute to the rapid accumulation of toxic compounds and ROS, ultimately leading to localized cell death [30-31]. Upon pathogen infection, plants synthesize a group of specialized proteins known as pathogenesis-related (PR) proteins. These include hydrolytic enzymes such as chitinases, glucanases, and other hydrolases, which are particularly induced during fungal invasions. In response to herbivorous insect attacks, plants also produce insecticidal proteins that interfere with the insect's digestive processes. For instance, plants like legumes and tomatoes synthesize enzyme inhibitors such as α -amylase and protease inhibitors, which suppress the activity of these enzymes,

thereby hindering the growth and development of herbivorous insects [32-33]. These defense strategies are often outcomes of systemic acquired resistance (SAR). Another key chemical defense is the synthesis of phytoalexins, secondary metabolites of phenolic nature produced in response to elicitors that trigger their biosynthesis [34]. Moreover, certain microorganisms can secrete plant hormone-like compounds such as auxins and cytokinins. These hormones are recognized by plant hormone receptors (HRs), leading to alterations in root morphology and physiology [35-38]. Effector-triggered immunity (ETI) represents a more specialized layer of plant defense. It involves the recognition of pathogen-derived effector molecules, essentially virulence factors secreted into the host plant, by plant resistance (R) proteins. This recognition initiates a strong immune response aimed at neutralizing the invading pathogen [39]. Following the recognition of specific pathogen effectors by resistance (R) proteins, plants rapidly trigger immune responses through the activation of effector-triggered immunity (ETI). In tomatoes, the synthesis of prosystemin, a precursor protein is rapidly induced upon insect-induced wounding. Prosystemin undergoes proteolytic cleavage to form systemin, which then activates defense signaling by binding to its receptor on the plasma membrane [40].

2. Ovipositional Fluids

Plants have ability to sense a wide range of herbivore-associated signals, there is strong evidence that herbivores, in turn, release effector molecules that suppress plant defense responses. Oviposition (the process of egg-laying) presents an additional opportunity for plants to detect the presence of insect herbivores. In response, some plants form necrotic zones at the site of egg deposition, a phenomenon observed in black mustard (*Brassica nigra*) and certain *Solanum spp.* including potato clones [41-42]. In pea plants (*Pisum sativum*), long-chain alpha, omega-diols known as bruchins, secreted during oviposition by pea weevils (*Bruchus pisorum*) induce the formation of undifferentiated cells beneath the deposited eggs. This localized response enhances plant resistance by creating a physical barrier that impedes larval penetration into the pods [43]. Oviposition fluids have been shown to activate the salicylic acid (SA) signaling pathway in Arabidopsis, which, paradoxically, enhances the growth of Egyptian cotton leafworm (*Spodoptera littoralis*) larvae [44-45]. Moreover, oviposition can reduce the release of herbivore-induced plant volatiles (HIPVs) in maize, thereby affecting the indirect plant defenses [46]. While feeding, most of the insect herbivores secrete effector-like molecules that actively reduced plant immune responses. One of the most widely cited examples is glucose oxidase, an enzyme secreted by the salivary glands of many lepidopteran species, which interferes with and suppresses plant defense signaling [47]. Similarly, many aphid species also secrete effector-like compounds that manipulate host immunity to facilitate colonization

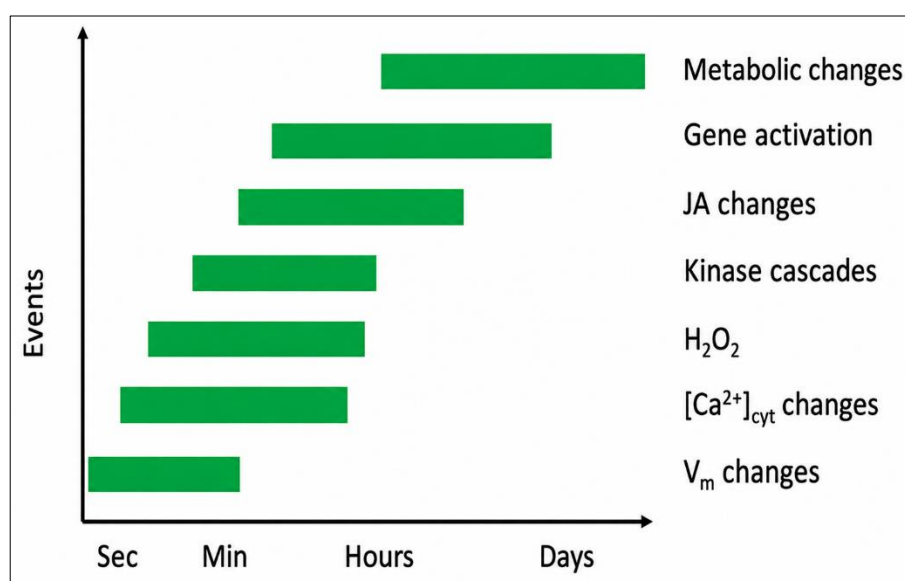
Table 2: Oviposition-induced plant responses acting against the herbivores

Plant (Direct Defense)	Herbivore	Effect of egg deposition	Induction by	Elicitors
Pea	Bruchids	Neoplasm growth (local growth only)	Egg cement No (Ovipositional) wounding	3-Hydroxy-propanoates
Rice	Plant hoppers	Ovicidal substances (Local response)	Egg on watery oviposition lesion Ovipositional wounding	Unknown
Potato	Colorado potato leaf beetle	Hypersensitive response (Local response only)	Egg shell or egg cement No ovipositional wound	Unknown
Cabbage	Cabbage butterfly	Oviposition deterrence (Local response, systemic response indicated)	Egg on undamaged surface	Unknown

Early events in the Plant defense

The early signaling events at the plant-insect interface occur rapidly, long before any observable changes in gene expression or defense-related metabolism. Upon herbivore attack, the first detectable response in the plant is an immediate alteration in transmembrane potential (V_m), followed by a rapid increase in intracellular calcium ion (Ca^{2+}) concentrations and the production of hydrogen

peroxide (H_2O_2). These early signals initiate downstream pathways, including the activation of kinases and the accumulation of the phyto-hormone Jasmonic acid (JA), both of which can be detected within minutes of herbivory. Approximately 1 hour after the initial damage, these signals lead to gene activation and subsequent metabolic reprogramming, as illustrated in Figure 3 [48].

**Fig 3:** Early events in plant defense [49]

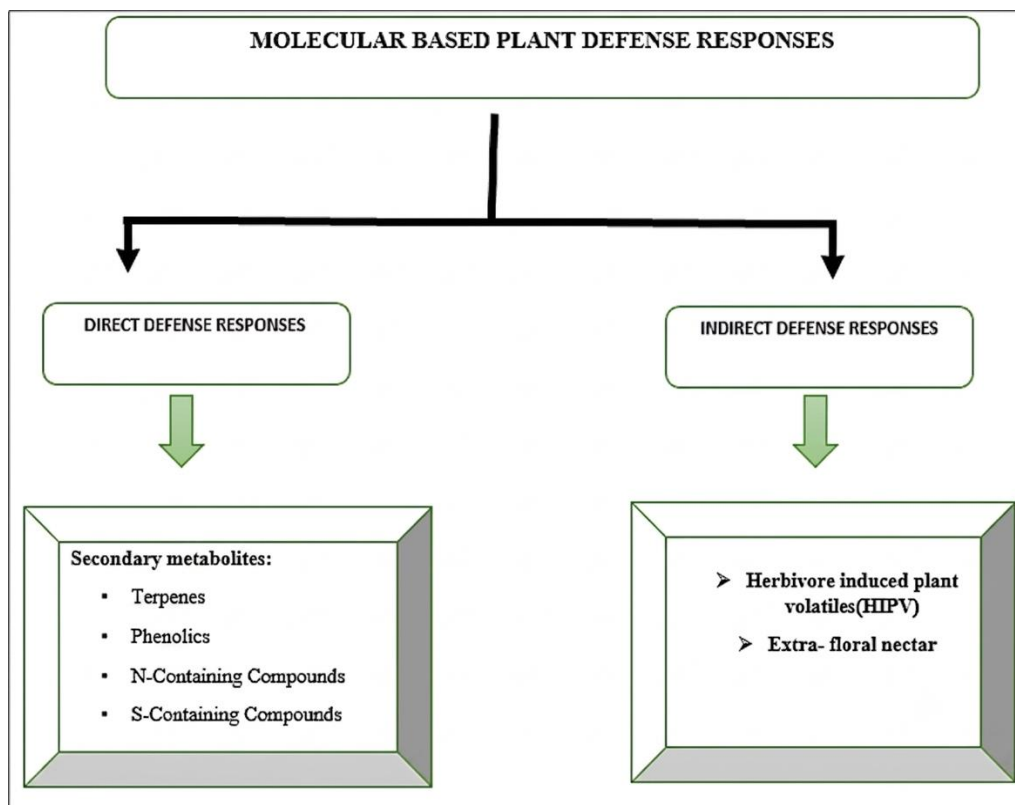
Recognition of Insect Herbivore Attack

Plants possess a remarkable ability to distinguish true herbivory from mere mechanical damage caused by wind, hail, or other physical factors, as well as to detect insect oviposition. This is important because strong defense responses use a lot of energy, so plants benefit by activating them only when a real herbivore threat is present [49].

Feeding Guilds

Over one million herbivorous insect species have been identified till date, each with unique feeding strategies that cause varying levels of mechanical injury to plants.

Approximately two-thirds of these herbivores are leaf-eating beetles and weevils (Coleoptera) and butterfly and moth (Lepidoptera), which inflict damage using specialized chewing mouthparts [50]. Leaf miners consume the soft internal leaf tissues or epidermal cells, whereas rasping and sucking insects such as thrips and spider mites scrape the leaf surface and extract sap. Meanwhile, phloem feeders including aphids, whiteflies, and other Hemipterans, use needle-like stylets to penetrate plant cells and withdraw phloem contents. Though less extensively studied, root-feeding insect herbivores mainly consist of root-chewers, with some root borers and piercers also reported [51].



Molecular Advances in Direct Defense

The term 'direct defense' refers to the production of physical barriers by plants or the synthesis of compounds that act as repellent, reduce nutritional value, or exhibit toxic effects

against insect herbivores. Virtually any plant trait can be adapted for direct defense; however, most research has concentrated on the wide array of secondary metabolites that act as toxins, digestibility inhibitors, and repellents [52].

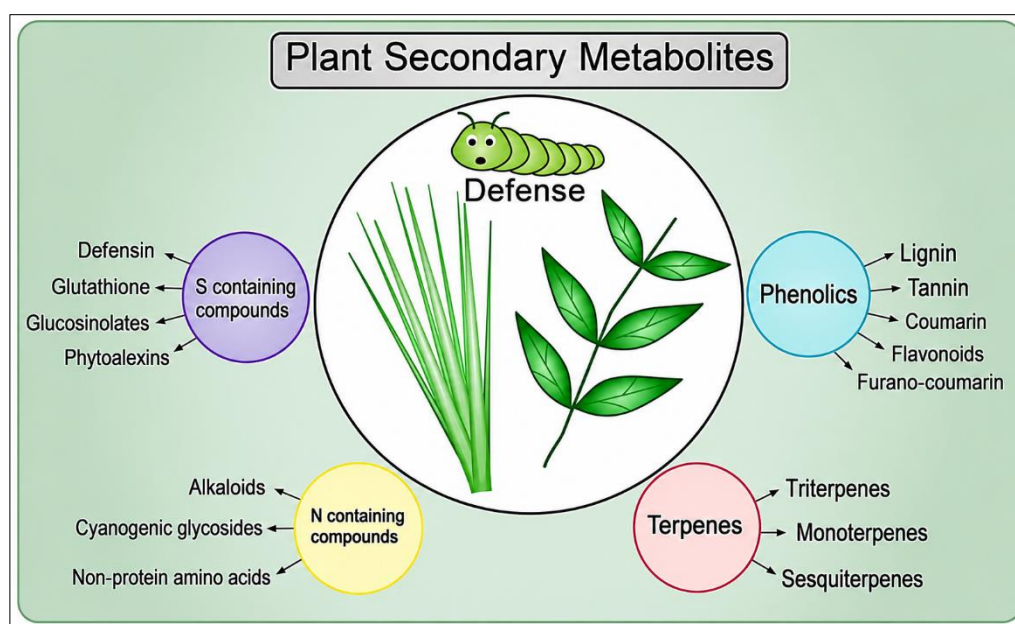


Fig 3: The main plant secondary metabolites (SMs) involved in plant defenses against herbivores, broadly classified as phenolics, terpenes, and sulfur (S) and nitrogen (N) containing compounds [53].

Table 4: Chemical derived substances involved in plant resistances against insect herbivory

Class/Sub-Class	Function	Reference
Alkanes, Aldehydes, Ketones, waxy cuticle	Protective layer	Panda and Khush (1995) [54]
Lignins, Tannins	Mechanical- barrier, unpalatability	De Bruxelles and Roberts (2001) [55]
Terpenoids	Toxicants, feeding deterrents, oviposition deterrents	Nishida (2002) [56]
Phenolics	Toxicants and ovicidal	Nishida (2002) [56]
Flavonoids	Anti-feedant, Phytoalexins	Ananthakrishnan (1999) [57]
Glucosinolates	Repellent, toxicant, irritant, antibiotic	Kliebenstein <i>et al.</i> (2001) [58]

Table 4: Protein derived substances involved in plant resistance to insect herbivory

Class/Sub-Class	Function	Reference
Lectins	Interfere with the absorption of nutrients, increase the absorption of toxic substances	Falco. <i>et al.</i> (2001) ^[59]
Chitinase	Damages the insect midgut	Falco <i>et al.</i> (2001) ^[59]
Alpha- amylase inhibitors	Digestive enzyme inhibitor	Silva <i>et al.</i> (2001) ^[60]
Protease inhibitors	Digestive enzyme inhibitor	Pompermayer <i>et al.</i> (2001) ^[61]
Polyphenol oxidase (PPO)	Reduction of the nutritive value of protein	Falco <i>et al.</i> (2001) ^[59]
Lipoxygenase (LOX)	JA biosynthesis	Arimura <i>et al.</i> (2000) ^[62]

Molecular Advances in Indirect Defense

The term “Indirect defense” refers to strategies where plants attract, support, and provide shelter for other organisms that help reduce herbivore pressure. This includes:

- Herbivore- induced plant volatile (HIPV)
- Extra floral nectar (EFN)

Herbivore- induced plant volatile (HIPV):

HIPVs stands for Herbivore-induced plant volatiles. HIPVs are basically specific mixture of volatile compounds released by plants in return back to herbivore attack. These volatile compounds play a utmost significant role in plant defense by either attracting natural enemies of the herbivores or deterring herbivore feeding and oviposition ^[63-64]. HIPVs are fat loving compounds (lipophilic) with high vapour pressure mainly released from the fruits, flowers, and also from leaves into the surroundings, also even from the roots into the soil, in response to herbivore attack ^[63]. The specific blend of HIPVs produced vary depending on the plant and herbivore species, also the developmental stage and condition of both the plant and the herbivore ^[65]. The volatile mixture released by plants during insect attacks is so unique to each insect-plant interaction, affecting both natural enemies and nearby plants ^[66]. HIPVs mediate complex interactions among plants, arthropods, microorganisms, undamaged neighboring plants, and even intra-plant signaling, which warns unaffected areas of the plant to the threat ^[67]. The specific volatile compounds produced are influenced by the feeding behavior of the insect pests, which activates different defense signaling pathways ^[68]. HIPVs include Green leafy volatiles (GLVs), Ethylene, Terpenes, Methyl salicylate, and many other volatile organic compounds (VOCs). One well-known type of HIPVs are GLVs, which are produced via hydro-peroxide lyase (HPL) branch of the oxylipin pathway. Green leaf volatiles (GLVs) function as reactive electrophilic species that are central to mediating stress responses and helps in activating plant defense signaling pathways. GLVs include C₆-aldehydes such as (Z)-3-hexenal and n-hexanal, along with their derivatives, including (Z)-3-hexenol, (Z)-3-hexen-1-yl acetate, and their corresponding E-isomers ^[69]. For better understand the functions of these C₆-aldehydes as well as their derivatives in plant defense, researchers have changed GLV levels through the application of elicitors or by genetically manipulating the expression of hydro peroxide lyase (HPL) in plants. GLVs are crucial in plant defense by attracting natural enemies of herbivores ^[70]. Moreover, plant volatiles such as C₁₆-homoterpene (E, E)-TMTT—4, 8, 12-trimethyl-1, 3(E), 7(E), methyl salicylate (MeSA), 11-tridecatetraene have been evidence to attract predatory mites ^[71]. Methyl salicylate, one of the most common components of herbivore-induced plant volatiles (HIPVs), has been detected in the headspace of several insect-infested plants, including lima bean and Arabidopsis.

MeSA has been linked to the inhibition of oviposition by cabbage moths (*Mamestra brassicae*) during infestation, suggesting that it can also be detected by the attacking herbivores. Methyl benzoate (MeBA), structurally similar to MeSA, has also been identified in insect-infested plants ^[72]. In rice, fall armyworm (*Spodoptera frugiperda*) infestation induces the emission of about 30 volatiles, including MeSA and MeBA, both of which are highly attractive to natural enemies like *Cotesia marginiventris* ^[73]. However, there is an ecological cost to utilizing HIPVs to attract natural enemies, as these volatiles can also lure crop pests. For example, *Leptinotarsa decemlineata* (Colorado potato beetle) gets attracted to volatiles blends consisting of cis-3-hexenyl acetate, linalool, and MeSA ^[74]. In addition to volatiles released from the aerial parts of plants, roots also emit a variety of compounds that help defend against below-ground insect pests. These root-emitted volatiles serve antimicrobial and anti-herbivore functions, and some even attract the natural enemies of root-feeding pests. For instance, Root-feeding insect like *Diuraphis noxia* triggers the emission of 1, 8-cineole, a monoterpene volatile that is toxic and repellent to certain insects ^[75]. However, root-emitted volatiles like 1, 8-cineole can inhibit the growth of *Brassica campestris* seedlings by hindering cell proliferation more than cell elongation. This is because root growth relies on both cell elongation and proliferation, and the compound interferes with nuclear and DNA synthesis that take place in root apical meristem, as well as altering the composition of root phospholipids and sterols ^[76-77].

Extra floral Nectar

Extra floral nectaries (EFNs) are specialized glands found on the leaves and shoots of plants, secreting nectar that provides indirect defense by attracting natural enemies that act as predators of herbivorous pests. Recent studies have begun to explore the use of EFNs in biological control within agricultural systems, aiming to enhance crop success while minimizing environmental impacts. Research has shown that plants with EFNs, when integrated into orchards or herbaceous crops, can support natural enemies and boost biological control. For example, wild cotton varieties have been found to produce more nectar per leaf than cultivated varieties ^[78]. EFNs are primarily composed of monosaccharides, disaccharides, amino acids, lipids, carbohydrates, and proteins. Their production is increased by herbivore attack and decrease in the absence of herbivory. Crop species known to produce extrafloral nectaries (EFNs) include *Manihot esculenta* (cassava), *Passiflora* spp. (passion flowers), *Gossypium herbaceum* (cotton), *Anacardium occidentale* (cashew), *Ricinus communis* (castor), *Prunus* spp. (almond, cherry, peach, and plum), as well as numerous members of the Leguminosae family ^[79]. EFNs consist primarily of sugars (about 90%) and also contain amino acids, lipids, proteins, antioxidants, mineral nutrients, and various bioactive compounds such as

alkaloids, phenolics, and volatile organic compounds (VOCs) ^[80-81]. However, the composition of EFNs varies widely across species and even among different types of nectars produced within the same plant species ^[82]. Although EFNs contain various bioactive compounds, they are not always toxic in nature ^[83], a compound that is harmful to one insect species may not necessarily affect others ^[84]. EFN secretion increases when plants experience herbivore attack and decreases when the threat is removed ^[85-86]. EFN production is also triggered by volatile organic compounds released from herbivore-damaged plants, as shown in *Phaseolus lunatus*, and by the application of exogenous jasmonic acid (JA) in *Macaranga tanarius* (Parasol leaf tree) ^[87-88]. A major focus of EFN research has been on protective ants, as their efficient foraging behavior, strong recruitment ability, and aggressive defense of food sources make them highly effective bodyguards for plants. A well-known example is Parasol leaf tree, where leaf damage significantly increases EFN secretions thereby attracting more protective ants and thus reducing herbivore pressure ^[88]. The Euphorbiaceae family is the largest group of plants with visible EFNs, which typically attract ants. EFNs are found in various shapes, including button-shaped, cup-shaped, stalk-shaped, pit-shaped, and pore-shaped structures ^[89]. Despite the benefits of “top-down” pest control through natural enemies, this strategy is often overlooked in plant breeding, and in many cases, beneficial interactions with third trophic-level organisms have been diminished due to breeding practices ^[90].

Conclusion

- Plants recognize and respond to damage-associated molecular patterns (DAMPs) and herbivore-associated molecular patterns (HAMPs) by initiating a suite of defense responses.
- The recent discovery of DAMP receptors has provided new insights into the distinct and overlapping roles of DAMPs and HAMPs in mediating plant-herbivore interactions.
- Early signaling events in plant defense involve key molecular components such as calcium ions (Ca²⁺), reactive oxygen species (ROS), and mitogen-activated protein kinase (MAPK) cascades, each playing a critical role in orchestrating downstream defense pathways.
- Jasmonic acid (JA) and salicylic acid (SA) are the two primary phytohormones regulating plant immune responses.
- Generally, chewing herbivores (e.g., caterpillars) activate the JA signaling pathway, whereas phloem-feeding insects (e.g., aphids) predominantly stimulate the SA pathway.
- Advances in molecular manipulation of plant defense mechanisms have enabled deeper exploration of plant-herbivore interactions, revealing complex cross-talk between plant immune systems and herbivore defense/immunity networks.

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