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REVIEW ARTICLE

Signalling cascades in plant cells under biotic stress: From herbivore detection to defence activation

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Abstract

Plants have evolved both passive and active defence mechanisms to withstand insect herbivory. Passive defences include physical barriers and toxic substances on the plant's surface, while active defences are triggered by herbivore-associated signals. Recent studies have shown that defence mechanisms, including volatile emission, molecular pattern recognition, changes in Ca^{2+} levels, shifts in plasma membrane potential, NADPH oxidase mobilization and oxygen radical formation, are triggered by interplant and intraplant signalling. Plants detect specific elicitors produced by insects during infestation, enabling them to recognize and respond to herbivory. Infested plants emit chemical signals that lead to the production of volatile terpenoids, attracting host-seeking insects. Plants deploy a wide array of defensive compounds, including cyanogenic glucosides, glucosinolates (GSLs), phenolics, alkaloids, proteinase inhibitors (PIs) and saponins, which deter feeding and impair insect digestion. Morphological features such as trichomes and thorns provide additional protection. Plant hormones such as ethylene (ET), jasmonic acid (JA) and salicylic acid (SA) mediate plant immunity. Calcium (Ca^{2+}) is crucial for regulating cellular processes and plant defence. Oxygen radical, especially hydrogen peroxide (H_2O_2), are critical for disease resistance. Nitric oxide (NO) influences H_2O_2 production, modulates the redox status and activates defence genes, thereby enhancing plant resilience against herbivory through various signalling pathways. By detailing the roles of various signalling molecules, hormones and defence compounds, the article aims to enhance understanding of plant defence strategies and the intricate signalling networks that underpin these responses.

Keywords: calcium-dependent protein kinase (CDPK); elicitor; ethylene; jasmonic acid (JA); MAPK cascade; nitric oxide (NO); reactive oxygen species (ROS)

Abbreviations

NADPH- Nicotinamide Adenine Dinucleotide Phosphate Hydrogen; HLSE- Herbivore Linked Specific Elicitor; ROS- Reactive oxygen Species; MAPK- Mitogen-activated protein kinase; OSs- Oral Secretion; FACs- Fatty –amino acid colligates; NIPs-Necrotic Inducing Proteins; VOCs- Volatile Organic Compounds; PIs- Proteinase Inhibitors; GSL- Glucosinolates; PRRs- Pattern Recognition Receptors; PAMPs- Pathogen Associated Molecular Patterns; PTI- PAMP- Triggered Immunity; ETI- Effector Triggered Immunity; SAR- Systemic Acquired Immunity; GLRs- Glutamate like Receptors; SWPs- Slow wave Potentials; l CMLs- Calmodulin like Proteins; CDPKs- Ca^{2+} Dependent Protein Kinase; ASC- Ascorbate; APX- Ascorbate Peroxidase; DHA- Dehydroascorbate; DHAR- Dehydroascorbate reductase; GSH- Glutathione ; GSSG- Glutathione disulphide; GR- Glutathione reductase; TRX- Thioredoxin; GSTs- Glutathione-S-transferases; GSNO- S-nitroso glutathione; NPR- non-expressor of PR gene; EDS- Enhanced Disease Susceptibility; PAD- Phytoalexin-Deficient; JAZ- Jasmonate Zim-Domain; MYC- Myelocytomatosis oncogenes; ETR- Ethylene Response; EIN- Ethylene Insensitive

Introduction

Plants adopt various defence mechanisms to counter insect herbivory, evolving sophisticated strategies which categorized into mechanical (thorns, trichomes, thick cuticles), chemical (secondary metabolites, volatile organic compounds) and ecological defences (mutualistic relationships, mutualistic relationships, mimicry or camouflage) over a long history of interdependent evolution with insects (1, 2). These mechanisms ensure plant survival by enabling quick and effective responses to stress signals and the preparation of defensive measures, indicating that plants have acquired both passive and active defence mechanisms throughout their evolutionary phase at multiple levels to combat pathogenic

infections. Passive resistance primarily consists of physical barriers, such as hairy or waxy surfaces and internal chemical defences that are lethal to pathogens feeding on plants, including phenols, unsaturated lactones and antimicrobial peptides (3-5). Plants develop active defence mechanisms when they detect pathogens and physical injury leads to cell wall fortification, callose deposition, phytoalexin production, systemic acquired resistance (SAR) and induced systemic resistance (ISR).

Plant defence responses originate at the cell surface, where insect herbivores cause physical damage and introduce elicitors or trigger plant-derived signalling molecules. The two most critical hormonal signalling pathways involved in induced plant defence are the salicylic acid (SA) and jasmonic acid (JA) pathways. The SA signalling route is predominantly linked to defences against sucking type of pests, bio trophic pathogens and viruses. The signalling pathway is primarily associated with defences against chewing and mites, as well as pests, necrotrophic pathogens, bacteria and nematodes (6-8). Understanding these signalling pathways and their interactions is crucial for comprehending how plants coordinate their defence mechanisms at multiple levels.

Recent findings in stress signalling have concluded that both modes of signalling - interplan signalling and intraplan signalling -are responsible for triggering defensive measures. These measures include the emission of volatiles and the recognition of molecular patterns, as well as defence effectors, at the membrane level (9-11). While at cytosolic level changes in concentration of Ca^{2+} ion causes shift in the potential between the inner and outer plasma membrane (Vm), mobilization of NADPH oxidase and formation of reactive oxygen radical (ROS), MAPK activation and phosphorylation of proteins (11-19). This review aims to compile and provide a comprehensive analysis of the signalling cascades involved in plant defence against insect herbivory. This includes the identification of elicitors, modulation of defence mechanisms, induction of signalling pathways and their crosstalk. By doing so, the article seeks to enhance the understanding of the intricate interactions between plants and insect herbivory, ultimately contributing to the development of more effective pest management strategies.

Elicitor-based identification of herbivore insect

Herbivore feeding causes physical damage to plant tissues, which is a critical factor in initiating plant defence mechanisms (20, 21). When insects feed on plants, they create wounds that expose plant cells to the environment. This mechanical injury serves as an initial signal that activates the plant's defence responses. In addition to causing physical damage, herbivores introduce various elicitors into the plant tissues. Elicitors are substances that can trigger and modulate plant defence responses, often originating from the herbivore's saliva or other excretory materials.

Plants have evolved sophisticated mechanisms to recognize and respond to herbivore-induced damage. One of the crucial processes in this recognition involves identifying specific molecular patterns associated with herbivore feeding, known as herbivore-linked specific elicitors (HLSEs). These elicitors vary in structure and include enzymes, lipids, cell wall fragments and peptides. Plants utilize receptor kinases (RKs) to detect these elicitors, which help them to distinguish between herbivore-induced damage from other forms of tissue injury. By identifying specific chemicals present in the insect's saliva (OSs), plants can distinguish between insect feeding and wounding caused by other means. This ability highlights the relationship between elicitor molecules associated with herbivory, which differ from damage incurred by plants via abiotic or mechanical means. When insects feed on a plant, they deposit substances on the wound that alter the plant cell metabolism as well as gene expression, which in turn manipulates the plant's response to the insect herbivores. HLSEs found in nature vary in structure (Fig. 1) and can take the form of enzymes, lipids, cell wall fragments and plant peptides. Examples of HLSEs include glucose oxidase and β-glucosidase, FACs (Fatty-amino acid colligates) including volicitin as well as caeliferins, pectin and oligo galacturonides and inceptin, which is basically a proteolytic shard of the chloroplast's ATP synthase subunit (Table 1). Upon recognition of HLSEs, plants initiate defensive measures.

The first well-studied HLSE was volicitin, a hydroxy FAC found inside the OSs of beet armyworms (*Spodoptera exigua*). Since then, extensive research has been conducted on herbivore oral secretions, leading to the identification of other HLSEs, such as caeliferins (20, 21). Other examples being β-glucosidase in white butterflies (*Pieris brassicae*) of *Brassica oleracea*, benzyl cyanides in *Plasmodiophora brassicae* a destructive pathogen for brassicaceae family and inceptins in the fall armyworms (*Spodoptera frugiperda*) (22-24) (Fig. 3b). However, HLSEs from sucking arthropods like spiders, aphids and others remain poorly understood. Recently, it has been found that the HLSE released from OS of aphids during cell wall digestion, like oligo galacturonides, has the potential to induce Ca^{2+}influx (25). Additionally, egg deposition may also trigger plant responses, which can be caused due to egg or egg -related elements of numerous insects. So far progress has been made only in the case of bruchid beetles, as identified chemical is long-chain lipids, which are involved (26, 27).

Transcriptome sequencing of the saliva of *Riptortus pedestris* was used to recognize HLSE. Research indicates that 200 proteins might be responsible for their role as intracellular effectors (RP191, RP246 and RP302) and apoplast-inducing effectors (RP309) have been identified as necrotic-inducing proteins (NIPs). These proteins also trigger the activation of reactive oxygen species (34).

Suppression of plant defence

Herbivorous arthropods, such as those that feed on specific plant tissues such as phloem or xylem, trigger a range of molecular and physiological changes in plants that can reduce their resistance and even impact photosynthesis (35). These responses can also involve the secretion of effector molecules that have the potential to alter host cell processes (36, 37). For instance, *Helicoverpa zea* mandibular gland releases the enzyme glucose oxidase (GOX) in its saliva, which is responsible for suppressing the host plant's defences by initiating oxidative burst (H_2O_2) in damaged leaves (38). In summary, the interaction between plants and herbivores involves complex signalling processes. Understanding how plants recognize and

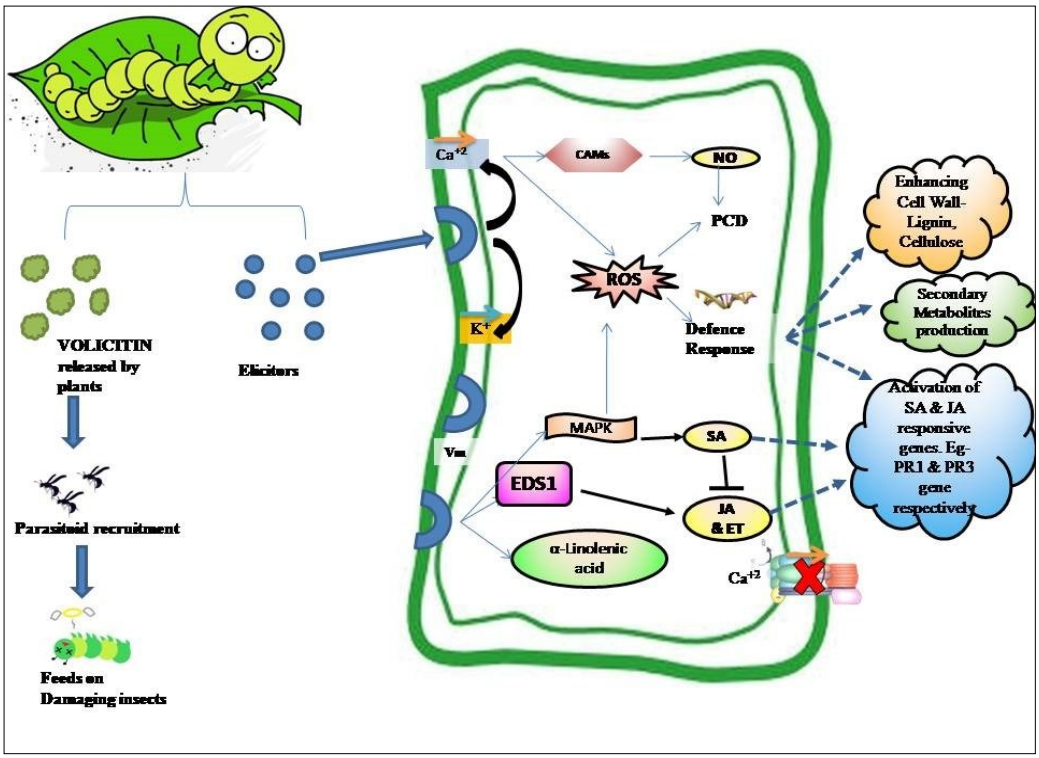

Fig. 1. A model for Plant-herbivore interaction. During the early stage of infection, the insect releases elicitor molecules as well as effector proteins. The Elicitor molecules help plants in recognizing the type and degree of damage caused by the action of herbivore insects. Receptors present on cell membranes recognize these elicitors and cause changes in membrane potential. This change in Vm causes an influx of Ca^{2+}& K^{+}, which are secondary messengers for multiple defence related transcriptional processes.

Table 1. Some prominent HLSE with their known receptor

Sl. No.	HLSE	Source	Characteristic Protein	Function	Reference
1	B-glu	*Pieri brassicae*	β-glucosidase	Provide Cotesia glomerata, parasitic wasps with enticing volatiles	(22)
2	Caeliferins	*Schistocerca americana*	Disulfooxy fatty acids	Enable corn to release volatile emissions	(21)
3	GOX	*Helicoverpa zea; European cornborer*	Glucose oxidase	Induce the tomato's JA pathway and late-responding defences	(28)
4	PLC	*Spodoptera frugiperda*	Phospholipase C	Limit the amount of weight that caterpillars gain; trigger defence reactions in Bermuda grass and maize	(29)
5	Mp10	*Myzus persicae*	Chemosensory protein	Lower tobacco's aphid fecundity	(10)
6	CathB3	*Myzus persicae*	Cysteine protease	Diminish the activity of aphids; cause the ROS burst in tobacco in an EDR1-dependent way	(30)
7	NlMLP	*Nilaparvata lugens*	Mucin-like protein	Formation of salivary sheaths; Rice and tobacco plants respond defensively	(31)
8	Te1	*Tetranychus evansi*	Tetranins	Encourage the biosynthesis of ABA, SA and JA in tobacco	(32)
9	PLP	Bacteria in *Spodoptera littoralis*	Porin-like protein	Start early defence-related processes in Arabidopsis	(33)
10	Oligogalac-turonides	Identified in the oral secretions of aphids		These substances also contribute to plant response activation	(25)

respond to herbivore-induced elicitors, as well as how herbivores suppress plant defences, provides valuable insights into plant-herbivore interactions and can inform the development of effective pest management strategies.

Despite significant advances in understanding plant defence signalling, several gaps remain in current knowledge. While numerous HLSEs have been identified, the full spectrum of elicitors released by various herbivores, especially sucking arthropods like aphids, remains underexplored. There is a need for comprehensive studies to identify and characterize these elicitors. The precise mechanisms by which plants recognize and differentiate between various HLSEs are still not fully understood. Research on receptor kinases (RKs) and pattern recognition receptors (PRRs) could provide deeper insights into the recognition processes. Although the antagonist relationship between SA and JA pathways is acknowledged, the detailed interactions and integration of these pathways with other signalling events, such as Ca^{2+} influx and ROS production, need further investigation. The mechanisms exploited by herbivores to suppress plant defences are not yet fully elucidated, particularly for different types of herbivores and their associated effector molecules. The dynamic interactions between plants and herbivores, including temporal and spatial variations in signalling responses, warrant further investigation.

Signalling pathways mediated by plant hormones

Plant defence against insect herbivory is governed by hormonal signalling pathways. Most of these pathways are mediated by SA, JA and ethylene. These pathways activate a set of defence-related genes in response to insect feeding (Fig. 2).

SA-induced disease resistance cascade

Salicylic acid (SA) is crucial in various plant physiological processes, such as growth, development, as well as stress resistance (39, 40). It is essential for both localized resistances in infected tissues and systemic acquired resistance (SAR) in uninfected areas. The expression of pathogenesis-related (PR) genes in uninfected tissues is induced by enhancing SA levels, leading to disease resistance throughout the plant. This increase in SA is triggered by elevated Ca^{2+} levels and the formation of reactive oxygen species (ROS) such as H_2O_2, which initiate SA biosynthesis (41).

There are some key components and functions of the SA signalling pathway. SA inhibits catalase and ascorbate peroxidase, enzymes that typically break down H_2O_2 in cells, leading to a surge in ROS levels. This rapid, regenerative feedback process, known as the oxidative burst, alters the cell's reduction-oxidation potential, thereby activating downstream defence mechanisms (42). NPR1 is a transcriptional co-activator activated by changes in cellular H_2O_2 levels. It has a key role in initiating the expression of defence-related genes in the SAR pathway, leading to enhanced disease resistance throughout the plant (2, 43). EDS1 (Enhanced Disease Susceptibility 1) and PAD4 (Phytoalexin Deficient 4) are proteins that maintain SA accumulation in response to herbivory. EDS1 collaborates closely with SA in signalling pathways involving coiled-coil nucleotide-binding site-leucine-rich repeat (CC-NB-LRR) proteins, which are essential for recognizing specific pathogens and activating immune responses (44). In Arabidopsis, the NDR1 (Non-Race-Specific Disease Resistance 1) gene enhances SA accumulation and supports R gene-mediated signalling. This gene plays a crucial role in amplifying SA-mediated defence responses, ensuring a robust immune reaction against pathogens (45, 46).

SA signalling reacts to different threats. Following are some examples. SA is associated with plant defence against sucking herbivores (eg. aphids) by mediating the synthesis of ROS and PR proteins. These responses strengthen the plant's physical and chemical barriers, making it less susceptible to herbivore attack (47). SA is essential for defending against biotrophic pathogens, which rely on living host tissue for nutrients. The SA pathway activates localized cell death and systemic acquired resistance (SAR), limiting pathogen spread and ensuring systemic resistance (48). SA signalling is important in mounting defence responses against viruses. The activation of PR genes and the accumulation of SA help inhibit viral replication and movement within the plant, reducing disease severity.

Jasmonic acid-modulated disease resistant cascade

Jasmonic acid (JA) is a phytohormone that triggers resistance mechanisms in response to herbivory and pathogen attack. JA-dependent induced resistance (IR) effectively deters chewing insect herbivores. The octadecanoid pathway produces various oxy lipids which modulate defence and wound anticipation. Additionally, JA can initiate production of alkaloids and certain volatiles, as well as emergence of defence related complexes that contribute to biotic stress resistance in plants. Research indicates that JA signal transduction involves four interrelated components: the JA signal itself, the SCFCOI1 E3 ubiquitin ligase, JAZ repressor proteins which are marked for degradation by SCFCOI1 via the ubiquitin/26S proteasome pathway, as well as transcriptional factors like MYC2 which enhance the elucidation expression of JA-responsive genes (18).

The JAZ protein, which is part of the jasmonate ZIM domain, functions as a deterrent of the JA signalling cascade (Fig. 3). It does this by suppressing the expression of JA responsive genes when it interacts with MYC2. When JA signals are present, they can cause the JAZ protein to interact with SCFCOI1 ubiquitin ligase, which leads to the JAZ protein being ubiquitinated and subsequently reduced by the 26S proteasome (49). Following JAZ dissociation, transcription factors such as MYC2 are activated, which then go on to activate JA response genes (50-52). JA induces the production

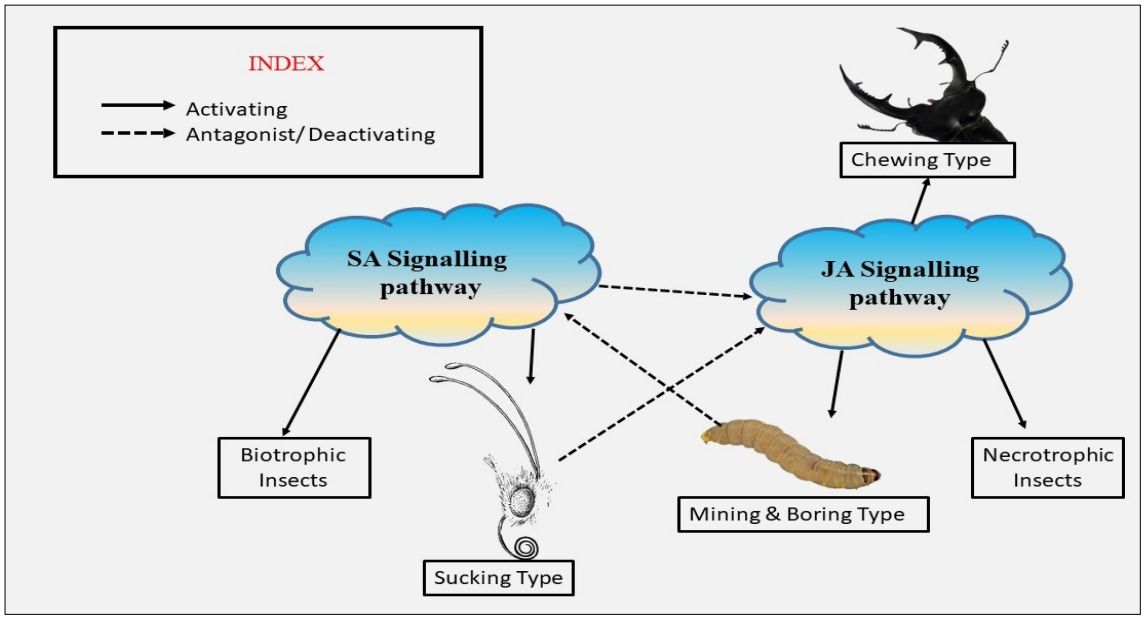

Fig. 2. Diagrammatic representation of feeding type and signalling pathway activated.

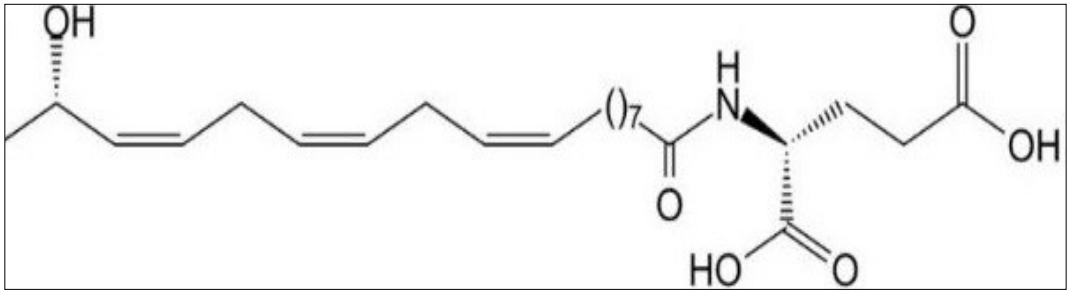

Fig. 3. a) Volicitin; b) Inceptins-Peptides derived from chloroplast ATP synthase subunits, found in the oral secretions of all armyworms (*Spodoptera frugiperda*), that play a role in plant defence activation (24).

of defensive proteins namely polyphenol oxidase (PPO), protease inhibitors, peroxidase (POX) and lipoxygenase. These proteins deter herbivores by changing chemical composition of the plant tissues and making it less palatable or more toxic. JA also triggers the production of alkaloids and VOCs, which as herbivores repellent or attract parasitoids. It plays a pivotal role in defending against necrotrophic pathogens that kill host tissue to feed on it (Fig. 4). The activation of JA-responsive genes leads to the production of phytoalexins and other antimicrobial compounds that inhibit pathogen growth. JA signalling contributes to the synthesis of stress resistance metabolites and defence-related complexes, enhancing the plant's resistance towards bacterial and nematode attacks. Genes like Vegetative Storage Protein2, Plant defensin and Hevein-Like protein are indicators of JA-triggered resistance

Plants often use the concentration of anti-digestive as well as resistive proteins as a protective mechanism in anticipation of caterpillar herbivory. Phytoalexins, for instance, is a JA- reliant proteins which hinder dietary proteases inside the insect viscera, thereby checking the amino acid nutrients accessibility necessary for their survival (53). The JA and SA pathways are often antagonistic, allowing plants to prioritize their responses based on the type of threat (54, 55). This antagonism enables plants to fine-tune their defence mechanisms when facing multiple attackers. Some herbivores, like the caterpillar *Spodoptera exigua*, exploit this antagonism by using effectors in their saliva to activate the SA pathway, which suppresses JA-mediated defences, thus aiding their survival. However, some attackers are able to shift this particular antagonism to their advantage. Caterpillars, like as *S. exigua*, use effectors in their labial saliva to activate SA-facilitated SAR pathway, which suppresses JA- facilitated defences (56, 57).

ET- modulated disease resistant cascade

Numerous factors, including environmental (light, high temperature, drought and waterlogging) and developmental ones such as fruit ripening and senescence, influence the regulation of ET (Ethylene) biosynthesis. Arabidopsis possesses five membrane binding receptors for Ethylene that can send signals to downstream effectors: ETR1, ETR2, ERS1, ERS2 and EIN4. In Arabidopsis, *Fusarium oxysporum* infection enhances susceptibility via ETR1-dependent ET signalling (5). Above mentioned ET receptor triggers CTR1 (serine/threonine protein kinase), that mainly inhibits the downstream pathway in order for ET response in the absence of an ET signal. CTR1, which inhibits the downstream ET response mechanism. EIN2 is one of the positive regulators of the ET cascade when the receptor is inactivated after binding to ET, which also inactivates CTR1. The transcription factor EIN3, which is found in the nucleus, receives a signal from EIN2 and binds to the ET response element in the promoter of ET response factor 1 (ERF1). To initiate the downstream ET response, ERF1 can bind to the GCC box of the target gene promoter (58). In order to activate genes that relate to defence like PR-3, 4, 12, that encode antimicrobial peptides implicated in JA/ET response, ERF1 and MYC2 help in the integration of signals from the JA/ET signalling cascade. In the jar1 mutant, ET can also induce systemic resistance, revealing that components for ET response cause downstream signalling for inducible systemic resistance (59).

The accumulation of SA-responsive gene transcripts in Arabidopsis which has been attacked by the phloem-feeder silver leaf whiteflies (*Bemisia tabaci*) occurred more localized and systemically, while the expression of JA was either subdued or unchanged (60). The initiation of SA and JA signalling pathways associated with plant defence responses was suppressed in tomato plants infested by *Tetranychus evansi* (61). Aphids mainly introduce effectors inside the plant hosts to manipulate cellular processes and ensure successful

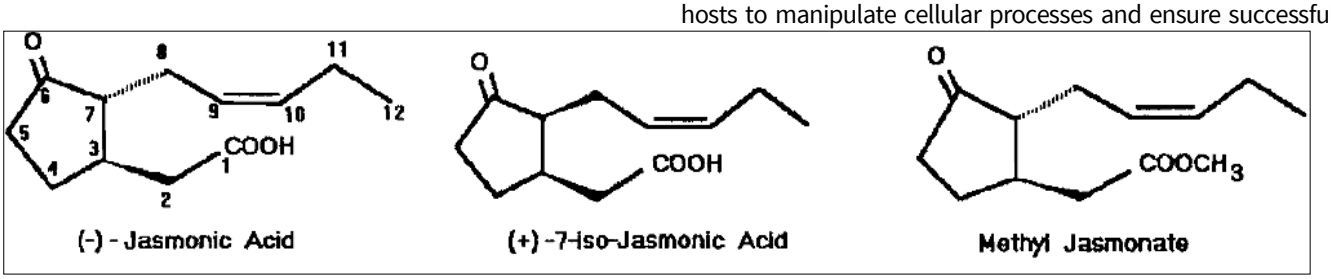

Fig. 4. Methyl jasmonate, a highly volatile chemical, is exploited by plants for Interplant signalling (62).

infestation (10).

Inter-plant signalling

Chemicals that facilitate communication between and among various species, or among members of the same species, are referred to as semiochemicals, which derive their name from the Greek term "semeion," meaning mark or signal. One such semio chemical is methyl jasmonate, which is highly volatile and can be easily transmitted via air (63). This has been isolated from the fragrance causing essential oil in *Jasminum grandiflorum*. In a recent study conducted at Washington State University, Pullman, USA, it was demonstrated that methyl jasmonate released by one plant when dispersed by air causes the synthesis of an inhibitor of the proteinase enzyme in uninfected plants at a distance. *Artemisia tridentata*, a member of the Asteraceae family, is able to produce methyl jasmonate naturally. Intercropping of *Artemisia tridentata*in tomato fields has effectively been found to induce proteinase inhibitors in tomato plants (62). Volatile semiochemicals attract parasitoids/ predators, indirectly defending plants via tritrophic interactions (Fig. 5) (9).

Certain research has focused on the plant intertalk through the release of volatile chemicals. Several findings have shown that wheat seedlings that herbivores have not damaged are capable of enticing aphids, while high aphid density can repel further aphid infestation (64). In response to insect damage, volatile terpenoids are released from leaves, which allow insect predators, like parasitic wasps, to differentiate between herbivore damaged plants and healthy plants. This helps them locate hosts or prey. Host-seeking insects possess the capacity to detect and react to specific chemical signals (eg. volicitin) emitted by plants under insect infestation. This ability implies that infested plants release volatile compounds that are distinguishable from those produced in reaction to alternative forms of damage or by healthy, undisturbed plants (9). For example, spider mite-infested apple plants and lima beans emit volatiles that lure predatory mites (65). In contrast, cotton plants produce volatiles that entice hymenopterous parasitic insects that target several larvae of Lepidoptera species (66). It has been recently found that volicitin, derived from insect OSs containing N-(17-hydroxylinolenoyl)-L glutamine, can stimulate the production of volatile terpenoid compounds. Volicitin is a result of crosstalk between a plant and its herbivore (Fig. 3a). The below mentioned sequence of events is believed to contribute to the indirect defence potential in plants (62).

Intra-plant signalling

Plant responses to intra-plant signalling in the context of biotic stress have been generally categorized into two primary groups: direct and indirect mechanisms. In the event of herbivory, plants typically employ indirect defences, like the emission of VOCs (Volatile Organic Compounds) that serve to entice natural predators and parasites to target the harmful herbivore insect (Fig. 6).

Direct defence mechanisms, such as the presence of trichomes, hairs and thorns, as well as the production of chemical compounds like cyanogenic glucosides, phenolics, alkaloids, PIs, GSL and saponins, serve as a plant's foremost defensive strategy against herbivorous insects (2). These act as dissuasive compounds against feeding, possessing properties that hinder digestion, or exhibiting toxicity towards herbivores which aid in safeguarding plants from these herbivores. (67, 68). For instance, multiple species of adult female moths and butterflies sense the presence of nicotine in glandular trichomes of tobacco plants, which serves as a signal prompting them to steer clear of these plants (69). Nevertheless, the creation of these inherent defences imposes a significant cost on the plant, in both possible ways, i.e., in terms of the chemical resources required for compound production and the development of specialized structures for their containment (53, 70). Keeping track of these incurred costs plants must carefully maintain a balance between survival and growth as well as resource allocation. Researchers have examined two significant patho systems for intra-plant signalling (5).

A series of signals is triggered by the interactions

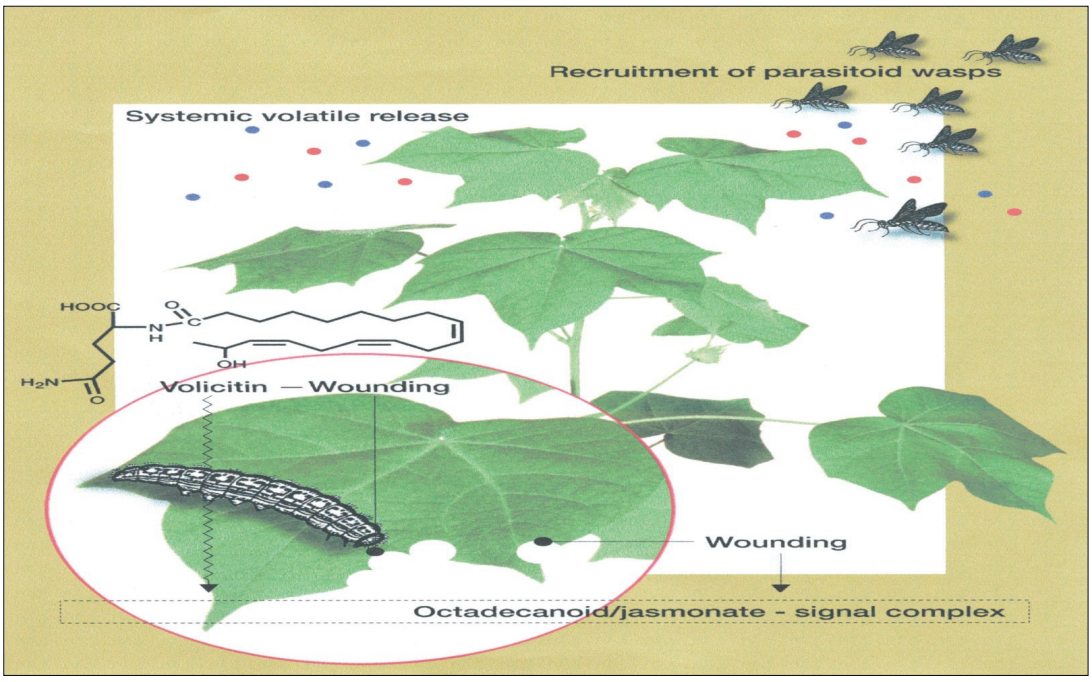

Fig. 5. Interlinked complex system of volatile compound secretion and parasitoid recruitment (9).

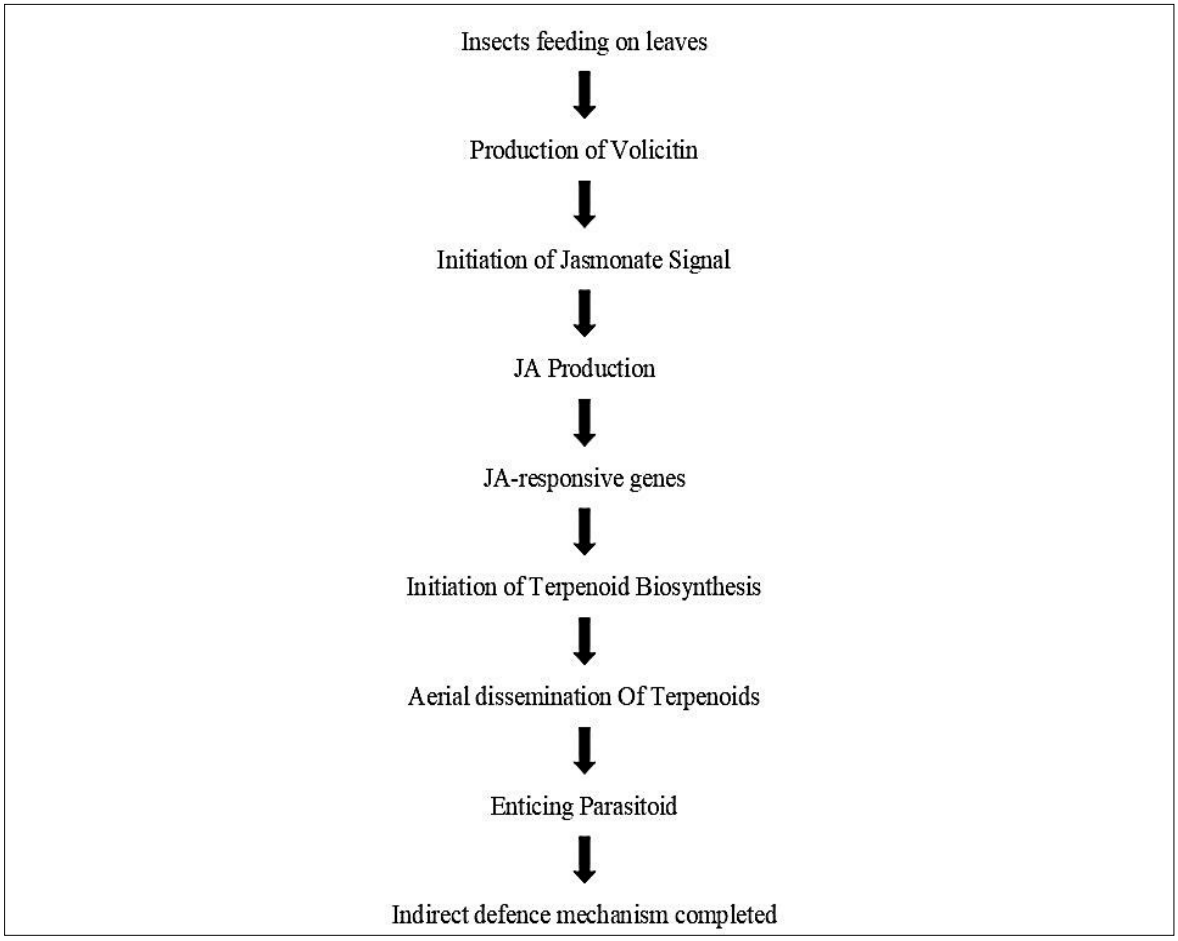

Fig. 6. Sequence of events that contribute to the indirect defence potential in plants.

between plants and pathogens that prompt plant defence responses. These signal pathways are intricate and varied and they connect with one another to form intricate signal transduction networks within plants. SA, JA and ET are central plant hormone regulation of both PTI and ETI (Fig. 7 & 8).

Secondary messenger molecule- Mediated cascade in plants

Ca^{2+} mediated cascade

Calcium is a vital signal molecule involved in various cellular functions, including regulating oxygen radical production, gene expression, signal transduction and apoptosis. It plays a crucial role in both abiotic and biotic stress responses (15, 79, 80). Calcium (Ca^{2+}), a ubiquitous signal molecule, regulates a broad array of cellular metabolic functions, including control of oxygen radical production, gene elucidation, signal transduction and many apoptotic stages. It is crucial in regulating various stress-related genes involved in defence mechanism (81). One of the early events of this signalling pathway is the depolarisation of membrane. Herbivore-induced damage causes membrane depolarization, which is dependent on Ca^{2+}-regulated potassium ion (K^{+}) channels (16, 82). Depolarization is prolonged in herbivore damage due to Ca^{2+} waves and wave potentials influenced by Glutamate-like Receptors (GLRs) and P-type H^{+}-ATPase-AHA1.The herbivore's OSs inhibits the H^{+}-ATPase activity which leads to alkalinization of the extracellular space, which in turn causes potential difference of the membrane. The initiation of GLRs, in conjunction with the production of Glutamate from the damaged phloem, leads to depolarisation of the plasma membrane potential in the form of SWPs. This depolarization, coupled with an increase in (Ca^{2+}) cyt, generates the systemic wound signalling (15, 83).

Intracellular calcium signals are transmitted to receptor proteins. Calcium receptor proteins has been classified under two broad categories: - Calmodulins (CaMs) and Ca^{2+}-dependent protein kinase (CDPK). The test plant *Arabidopsis* contains a large family of Ca^{2+}sensing receptor proteins, including CaMs and CMLs proteins, which transmit and interpret Ca^{2+} signals in many signal transduction processes (12). Recent investigations on CML proteins, shows that several CML such as CML8 as well as CML13, plays role in plant defence mechanisms against plant pathogens like *Pseudomonas syringae* and *Ralstonia solanacearum*. Additionally, upregulation of these proteins regulates the expression of pathogenesis-related (PR) genes and multiple genes linked to signal transduction and stress responses (84). Evidence supporting contribution of CDPKs towards plant defence responses has accumulated over time. For instance, research has shown that CPK5, a CDPK in *A. thaliana*, boosts the SA-mediated resistance of the plant against the bacterial pathogen *P. syringae* pv. tomato strain DC3000. Research indicates the variation in expression of defence genes within the plant and triggers the formation of O_2radicals (ROS) (85). As a crucial intracellular signalling molecule, calcium (Ca^{2+}) is pivotal in modulating the plant's defence mechanisms gene expression and plant innate immunity. Despite its importance, the specific mechanisms by which Ca^{2+} regulates gene expression at the molecular level remain unclear.

ROS-mediated disease resistance signalling pathway

ROS, including H_2O_2, are crucial for plant resistance against mechanical wounding and herbivore feeding. NADPH oxidase

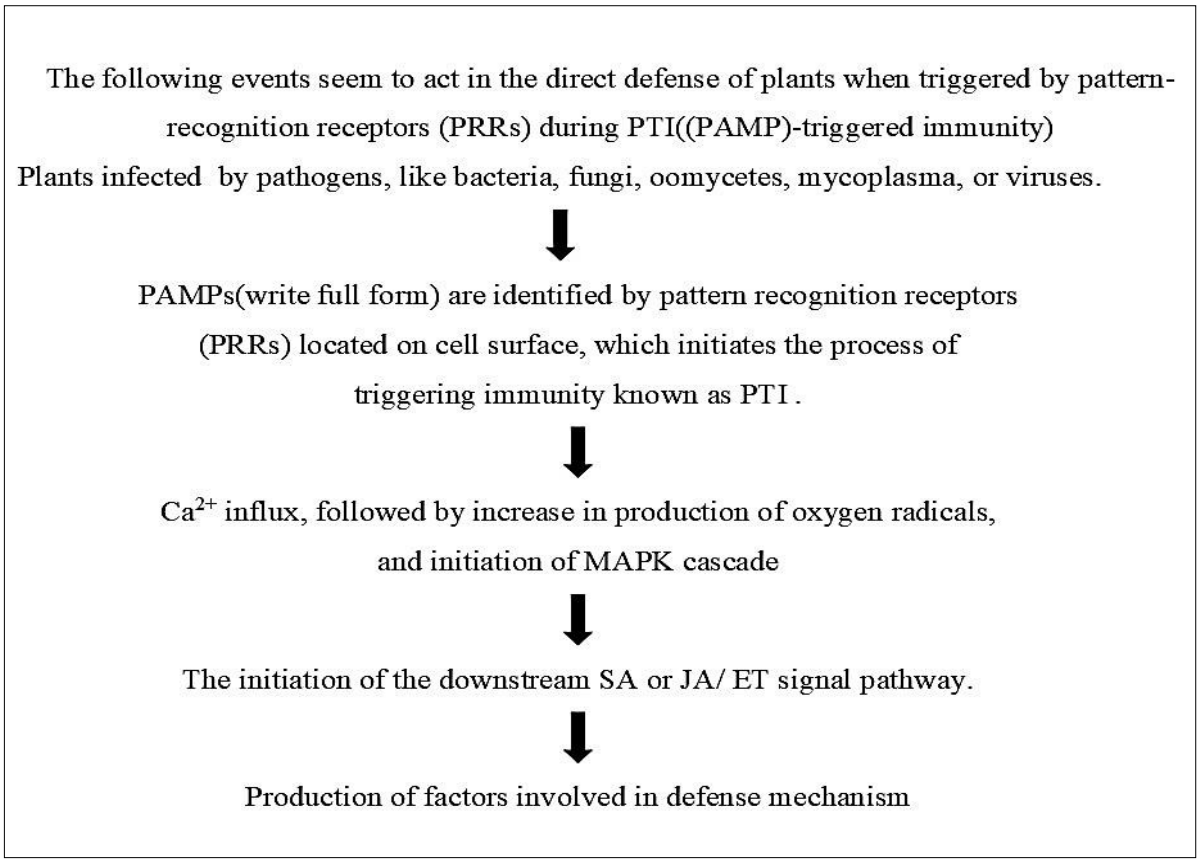

Fig. 7. Direct defence under PRRs.

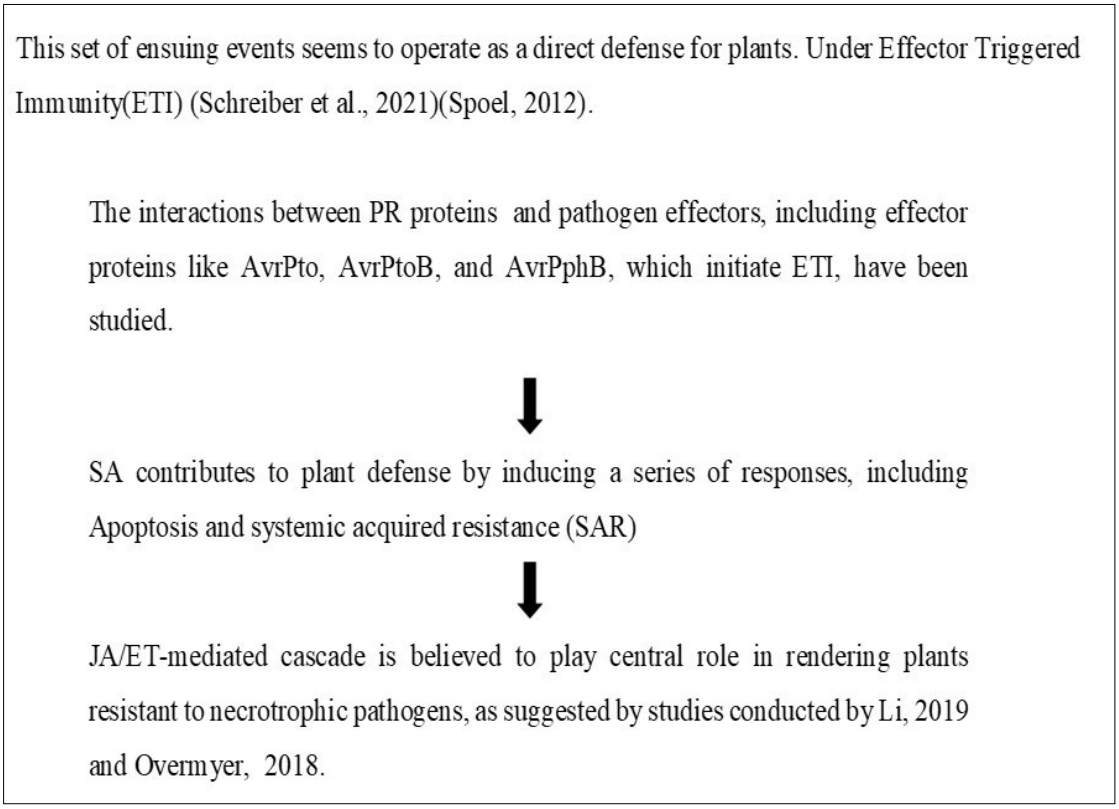

Fig. 8. Direct defence under ETI.

is activated by CDPKs, leading to H_2O_2 production in wounded tissues (2, 86). Enzymes like superoxide dismutase, peroxidases and catalases regulate ROS levels and helps in detoxification of ROS produced during stress. The ascorbate-glutathione cycle tightly controls H_2O_2 levels to minimize damage (11). When H_2O_2 levels rise due to oxidative stress, APX (Ascorbate Peroxidase) converts H_2O_2 into water and oxidizes ASC (Ascorbate) to DHA (Dehydroascorbate). DHAR (DHA reductase) then reduces DHA into ASC, using GSH to generate oxidized GSSG. GR subsequently reduces GSSG into GSH, producing NADPH from NADP+ (Fig. 9). This tight control of ROS production minimizes the occurrence of detrimental reactions. ASC and GSH, a small thiol tripeptide, are essential antioxidants as well as regulators of the ROS (87) (Fig. 10).

Glutathione also plays its part in the signalling cascade by either direct or indirect modification of proteins via post-

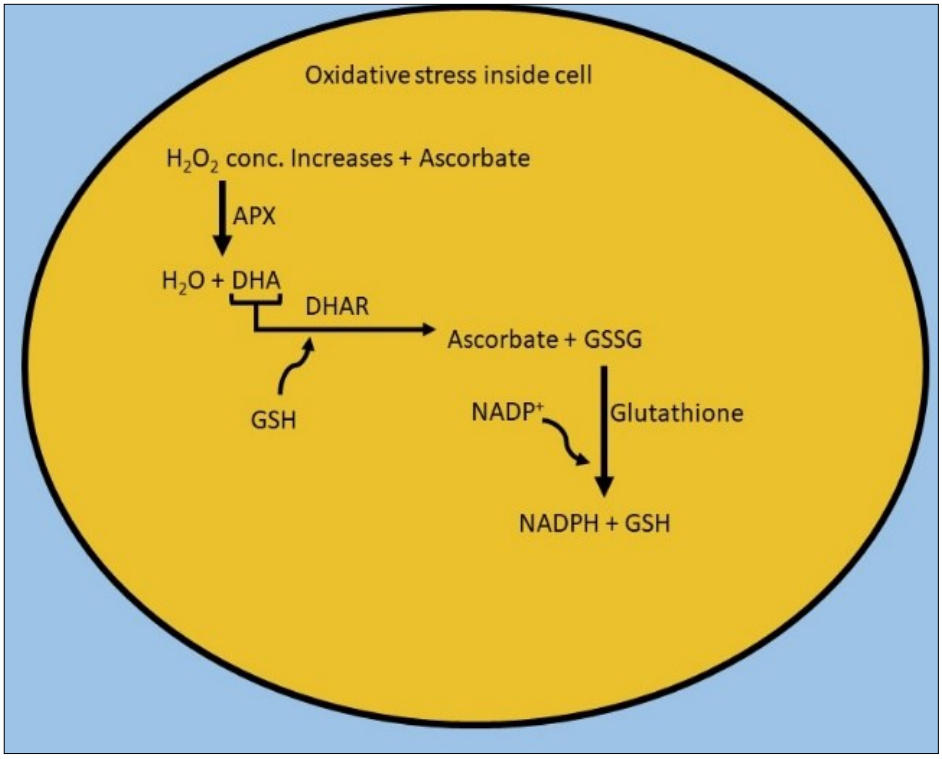

Fig. 9. Oxidative Stress enticing Oxygen radical pathways [Ascorbate peroxidase (APX), Dehydroascorbate (DHA), DHAreductase (DHAR), Glutathione (GSH), Glutathione reductase (GR)].

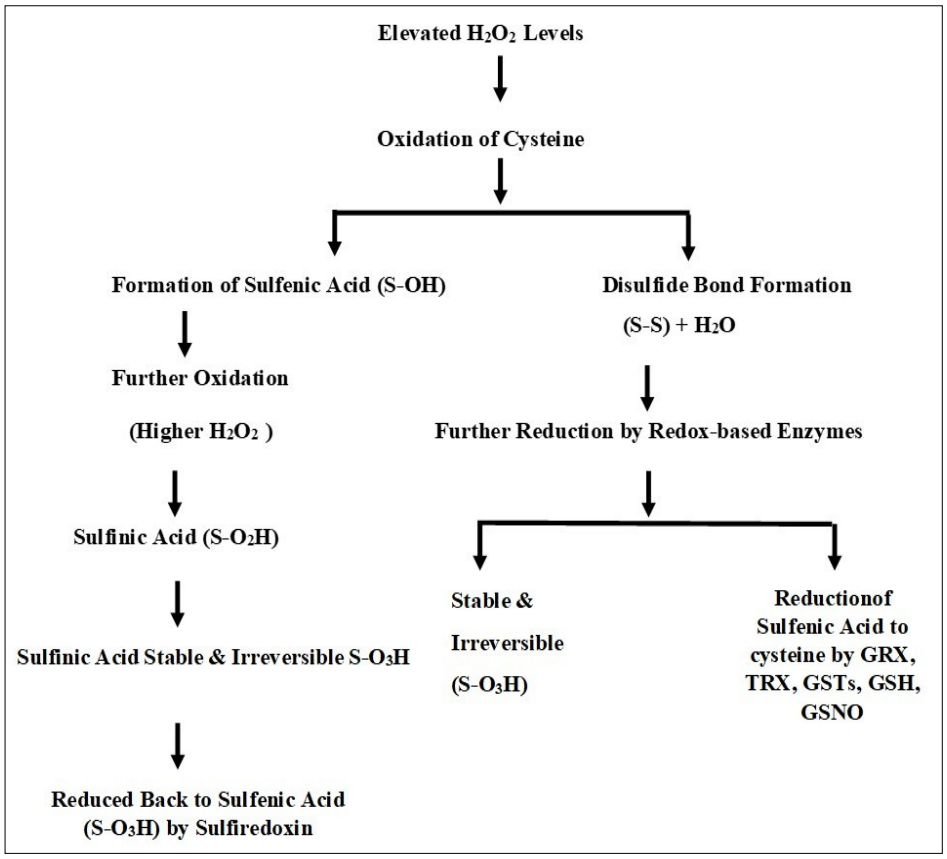

Fig. 10. ROS-mediated disease resistance signalling pathway.

translational modification processes. The modification of proteins induces shifts in redox potential, influenced by the H_2O_2 produced during herbivory. Because of the mutation in the GSH1 gene, Arabidopsis pad2.1 has low constitutive levels of glutathione, which makes it more prone to caterpillar attack as compared to wild-variety plants (88). H_2O_2 has a key role in modulating signalling cascade by altering protein activities. H_2O_2can modulate protein cysteine residues through reversible post-translational modifications, leading to the establishment of disulfide bonds in cellular contexts.

As H_2O_2 levels rise, it causes formation of sulfenic acid (S -OH) due to oxidation of cysteine residues. These sulfenic acids can interact with one another to create disulfide bonds, releasing water in the process. When exposed to even higher concentrations of H_2O_2, sulfenic acid may be further oxidized to

sulfinic acid (SO_2H) or sulfonic acid (SO_3H). Sulfonic acid is stable and remains unchanged, whereas sulfenic acid could be achieved back through enzymatic reduction of sulfinic acid by sulfiredoxin. Furthermore, sulfenic acid could be reduced further more via numerous redox-based enzymes, including glutathione, glutathione-S-transferases (GSTs) and S-nitroso glutathione, thioredoxin (TRX), glutaredoxin (GRX). H_2O_2 modulates protein cysteine residues through reversible post-translational modifications, leading to the formation of disulfide bonds (89, 90). The redox state of proteins is influenced by glutathione and other redox-based enzymes. H_2O_2 acts as a signalling molecule, modifying proteins directly or indirectly to initiate defence pathways.

NO-induced disease resistance signalling pathway

NO is a signalling molecule involved in redox reactions and physiological processes through post-translational modifications (PTMs) Under pathogen stimulation, the rate of NO synthesis increases, leading to the buildup of phytoalexins, having an impact on accumulation of ROS, inducing the formation of H_2O_2, adjusting host's oxidation-reduction state and activating MAPK defence genes and PR proteins (91). Research indicates that NO governs hypersensitive response and apoptosis and collaborates with oxygen radicals to activate the plant's expression of disease resistance and activation of related genes for defensive measures. In compatible interactions, such as those involving tobacco and *Botrytis cinerea* (a necrotrophic pathogen), NO synthesis increases in response to pathogen attack, leading to ROS accumulation and the activation of defence genes. NO and ROS work together to mediate defence responses, including hypersensitive response and apoptosis (92, 93).

Heterotrimeric G Protein-facilitated defence Pathway

Heterotrimeric G Proteins translate extracellular signals into intracellular responses and amplify these signals through effector enzymes, such as adenylate cyclase and phospholipase C (94). These proteins not only translate intracellular signals but also amplify them and initiate a range of imminent effector enzymes, like adenylate cyclase (AC1) in case of animals as well as phospholipase C in case of plants. The accumulation of these proteins results in the formation of effector enzymes, which ultimately cause the production of multiple secondary messengers, namely cAMP, that has the potential to boost signals via various signal transduction cascades (95).

The ability of Arabidopsis to resist necrotizing pathogenic fungi, such as *A. brassicola* and *F. oxysporum,* is dependent primarily on a G protein-facilitated signalling cascade. The MAPK cascade serves as a precursor for G protein signal transduction. RACK1, a well-studied plant MAPK protein, plays a key role in interlinking heterotrimeric G proteins to the MAPK signalling cascade to initiate a distinctive signalling cascade for plant immunity.

Protein phosphorylation signalling: MAPK

Protein phosphorylation signalling involves the MAPK Cascade. Protein kinase cascades, including MAPKs, MAPKKs and MAPKKKs, regulate various functions like photosynthesis, self-incompatibility and stress responses (Fig. 11). More than 30 protein kinases have been found in plants and they affect a variety of functions like photosynthesis, self-incompatibility, cold and light tolerance and cell division (96). MAPK is a distinctive Ser/Thr-based protein kinase with three primary types: MAPKs, MAPKKs and MAPKKKs (97). MAPKKKs are positioned upstream in the signalling hierarchy and connect interstetial signals to preceeding regulators via phosphorylation (98). MAPK cascades are involved in both PTI and ETI responses (Table 2). Transcription factors like WRKY67 enhance resistance to bacterial blight in rice (5). In *Nicotiana attenuata*, MAPKs regulate phytohormone production and secondary metabolites in response to herbivory, as well as those that oppose herbivory, which in turn control responses triggered by wounding and herbivory. In reciprocation to attack of *Helicoverpa armigera*in chick pea, multiple MAPK genes have been enumerated in chickpea (*Cicer arietinum*),

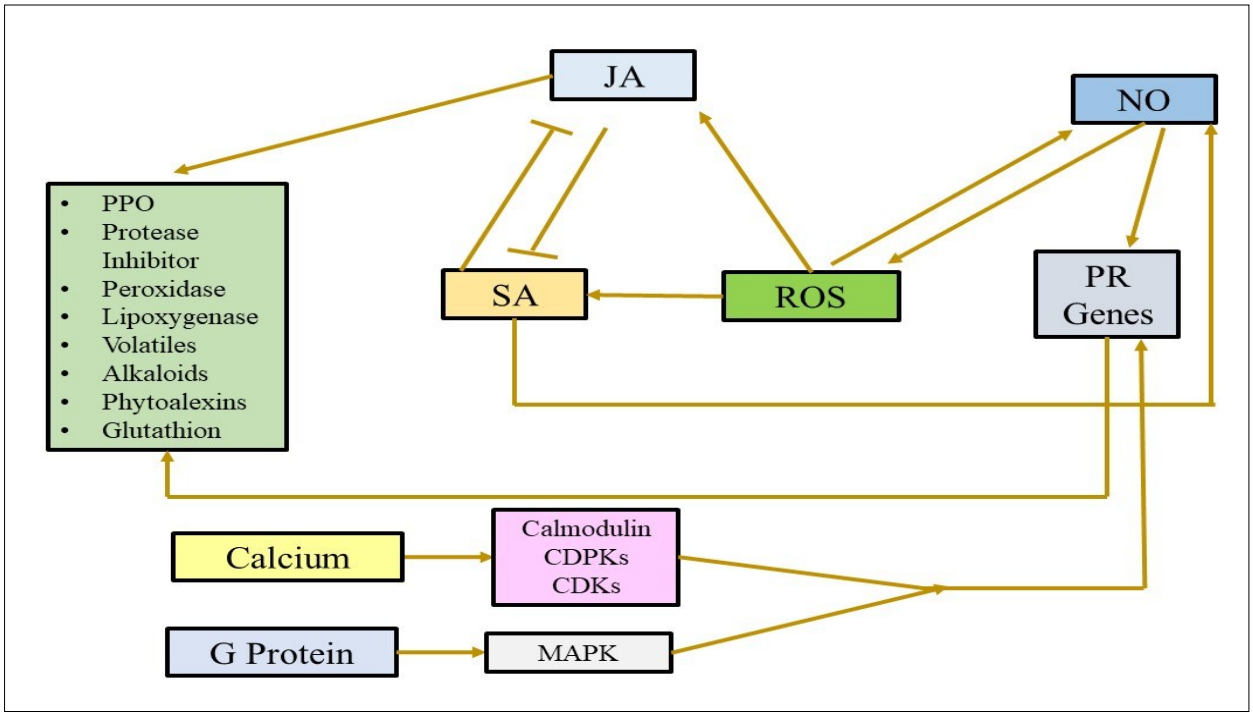

Fig. 11. Representation of interconnected defence mechanism.

Table 2. Some prominent Effector proteins involved in ETI pathway

Sl. No.	Effector	Source	Characteristic protein	Function	Reference
1	GOX	*Helicoverpa zea*	Glucose oxidase	Prevent tobacco from producing nicotine	(38)
2	HARP1	*Helicoverpa armigera*	Venom R-like protein	Boost the feeding efficiency of cotton bollworms; interact with JAZ in Arabidopsis to block the JA pathway	(71)
3	HAS1	*Helicoverpa armigera*	Venom R-like protein	Improve the feeding efficiency of cotton bollworms; inhibit the JA pathway in Arabidopsis by interacting with MYC3/MYC4	(71)
4	Me10	*Macrosiphum euphorbiae*	Salivary glands-abundant secretory protein	Increase the fertility of aphids, suppress defences and interact with tomato TFT7	(72, 73)
5	NlSEF1	*Nilaparvata lugens*	EF-hand calcium-binding protein	Reduce the amount of Ca^{2+} and H_2O_2 that rice produces	(74)
6	NlEG1	*Nilaparvata lugens.*	Endo-b-1,4-Glucanase	Allow BPH feeding; break down rice celluloses	(75)
7	CaM	*Nilaparvata lugens; Laodelphax striatellus*	Calmodulin binding protein	Allow BPH fecundity; inhibit callose deposition and H_2O_2 build-up in rice	(76)
8	VgC	*Laodelphax striatellus*	C-terminal peptide of vitellogenin	Reduce H_2O_2 build-up in rice by focusing on OsWRKY71	(77)
9	C2	Tomato yellow leaf curl virus in Bemisia tabaci	Virus protein	Enhance the survival and procreation of whiteflies; suppress plant defences through interactions with plant ubiquitin	(78)

while two MAPKs have been enumerated in soybean (*Glycine max*) that have been infested by the green stink bug (*Nezara viridula*) (99).

Conclusion

This manuscript highlights the intricate signalling mechanisms that plants employ to defend against herbivore attacks, beginning with the recognition of early elicitors and progressing to the activation of complex biochemical pathways. While plants have developed advanced defence systems, herbivores counteract with effector molecules, resulting in a dynamic interplay of defence and attack. Enhancing our understanding of this interplay and utilizing genetic engineering could lead to the development of resilient crops. However, key knowledge gaps remain, particularly in understanding the initial stages of signal perception and examining various plant systems beyond model organism. Future research addressing these gaps will improve our ability to combat biotic stresses in agriculture.

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Authors' contributions

YB conceptualized the article, contributed to data interpretation, and prepared the initial draft of the manuscript. HS developed the research concept and provided intellectual input during the conceptualization stage. JP co-conceptualized the article, assisted with data interpretation, and contributed to manuscript preparation. RA collected research data and contributed insights during the interpretation of results. RR assisted in data collection and supported the interpretation of findings. KP participated in data collection, contributed to result interpretation, and helped with final language editing. GK assisted in writing and organizing the manuscript content. AJ reviewed and edited the manuscript for language clarity and grammar. AK contributed to improving the language and coherence of the final manuscript. A performed the final language check and assisted in correcting, formatting and technical issues. All authors read and approved the final version of the manuscript.

Compliance with ethical standards

Conflict of interest: The authors declared no conflict of interest

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