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Biosynthesis and Regulation of Herbivore-Induced Plant Volatile Emission

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Abstract | Volatile phytochemicals function as air-borne communication signals between plants and insects. Plants emit a bouquet of volatile organic compounds not only from flowers and fruits but also from vegetative tissues. Herbivore-induced plant volatiles (HIPVs) are released *de novo* in response to tissue damage by insects. They act as an indirect defense by attracting natural enemies of the herbivore, which sense them as sources of food and/or reproduction. Additionally, HIPVs can inhibit pathogen growth and mediate inter- and intra–plant communication. HIPV emission varies with ontogeny, type of herbivore as well as environmental conditions. Plant hormones, particularly jasmonates, salicylic acid and ethylene, are implicated to play pivotal roles in the regulation of HIPV emission. Crosstalk among phytohormone signaling pathways enables fine-tuning of plant responses. This review presents biosynthesis of HIPVs and regulation by phytohormones.

Keywords: *herbivory, jasmonates, phytohormones, plant volatile emission, terpenoids*

1 Introduction

Plants, being limited by their sessile nature, have evolved multiple ways to defend themselves against enemies. Direct defence such as hard waxes, thorns or toxic chemicals which act as repellents, deterrents, or as anti-nutrients/anti-digestive compounds constitutes one way by which plants escape herbivory. In addition, plants also exhibit indirect defence mechanisms which help in recruiting predatory insects that attack herbivores.[1,2] Volatile emission is one such indirect defence strategy which attracts enemies of herbivores to damaged plants. By this emission, plants send out a distress signal to predatory insects that interpret it as an indication of an oviposition or prey zone. These insects may be parasitoids that oviposit into the herbivorous prey or true predators that feed on the prey. Additionally, volatile emission also serves a role in a range of ecological functions including pollinator attraction, plant–pathogen and plant–plant interactions.[3–5] Volatiles also function as direct defences by acting as feeding deterrents.[6] Although direct defence is important in plant resistance, indirect defence confers phenotypic plasticity and is a useful strategy for pest control in agriculture.[7,8] Plant volatiles are a complex mixture of organic compounds constituting about 1% of plant secondary metabolites with molecular weight lower than 300 Da.[7] Over 2000 volatile compounds have been characterized from different plant families.[9] Plant volatiles can be emitted from leaves, fruits, flowers and also from roots. Here, I focus on herbivore-induced plant volatiles (HIPVs) emitted *de novo* from tissues after herbivory. HIPVs are usually a blend of several organic compounds predominantly composed of terpenes and derivatives of fatty acids or amino acids. The composition of HIPVs is often plant- and herbivore-specific and also depends on various environmental factors.[7,9,10] Plants need to avoid being apparent to herbivores but at the same time attract pollinators and natural enemies of herbivores using air-borne signals. For an insect on the other hand, a strong selection pressure operates to evolve sensitive detection ability to locate resources for food and reproduction. Hence, it is reasonable to expect

Herbivore-induced plant volatiles: these compounds are biosynthesized in larger amounts upon herbivore damage. In the absence of herbivory or other tissue damage these compounds are either not produced or made in low levels.

Plant–plant interactions: plants can warn or detect warning from their neighboring plants via volatiles or light signaling. This is termed as plant–plant interaction.

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that not all individual components of the volatile bouquet are perceived by insects and that their behavioral responses to plant odors are plastic.[11] Constituents of the HIPV blend originate from different biosynthetic routes and are emitted at the site of attack as well from distal plant parts via systemic signaling.[12,13] They are usually released from membranes on the epidermal tissues or from other structures such as glandular trichomes or special ducts and laticifers.[14]

Plant growth and development are orchestrated by a group of structurally unrelated small molecules, namely phytohormones. These molecules work at low concentrations (pmol range) and act as a relay integrating external cues such as environmental changes and biotic stress into endogenous developmental responses. In addition to the classical growth hormones such as cytokinin, auxin, gibberellin, ethylene and abscisic acid, new hormones such as jasmonate (JA), salicylic acid (SA), nitric oxide, strigolactone and brassinosteroids have been identified. Decades of research have led to the discovery of receptors of all the major classes of phytohormones.[15,16] The phytohormones JA, SA and ethylene are largely implicated in the regulation of HIPVs, although there is emerging evidence involving other hormones.[17] Moreover, some phytohormones such as ethylene are by themselves volatile or are converted into volatile products, such as methyl jasmonate (MeJA) or methyl salicylate (MeSA). Thus, these molecules play a vital role as internal hormones and as volatile signals during plant–insect interactions. Here, biosynthesis of HIPVs and the role of phytohormones in their regulation is presented.

2 Biosynthesis of Plant Volatiles (HIPVs)

2.1 *Terpenoids*

A major portion of HIPVs is of isoprenoid origin and there is much interest in their emission due to their impact on atmospheric chemistry and ozone formation.[18] All isoprenoids originate from two basic C5 units, isopentenyl diphosphate (IDP) and its isomer dimethylallyl diphosphate (DMADP). Based on the carbon length they are termed as hemi- (C_5), mono- (C_{10}), sesqui- (C_{15}), tri- (C_{30}) or tetra- (C_{40}) terpenoids. Volatile terpenoids belong to the class of hemi-, mono- or sesquiterpenes because of their higher vapor pressure. The biosynthesis of terpenoids involves two separate metabolic pathways in plants: the cytosolic mevalonic acid (MVA) pathway and the plastidic 2-C-methyl-erythritol 4-phosphate (MEP) pathway. In the MVA pathway, IDP is synthesized from acetyl-CoA, while in the MEP pathway, IDP is derived from pyruvate and glyceraldehyde-3-phosphate. Although these two pathways are compartmentalized in the cell operating independently, crosstalk among them is known.[19,20] After the formation of IDP and DMADP, they are condensed by prenyltransferases also known as isoprenyl diphosphate synthases to produce prenyl diphosphates such as farnesyl diphosphate (FDP) in the cytosol, as well as geranyl diphosphate (GDP) and geranylgeranyl diphosphate (GGDP) in the plastids. A diverse range of terpenoids are synthesized by a large family of enzymes known as terpene synthases (TPS) using DMADP, GDP, FDP and GGDP as substrates. A unique feature of these enzymes is the ability to generate multiple structures owing to the generation of carbocation intermediates that can undergo a variety of reactions (cyclization, rearrangement, and hydride shifts).[21] In *Arabidopsis thaliana*, 14 TPS genes have been characterized, whose expression were tissue specific.[22] Herbivory-induced induction of *TPS* gene expression subsequently leads to HIPV emission.[22,23] Engineering of *TPS* provides opportunities to alter specific volatile compositions, which can be useful in pest management. For example, *Zea mays* (maize) plants emit (*E*)-β-caryophyllene when attacked by *Spodoptera littoralis* and the root herbivore *Diabrotica virgifera*. This sesquiterpene is synthesized by *TPS23*, a gene active in teosinte and European lines but not in North American lines.[24] By transforming the non-emitting maize plants with oregano *TPS*, it was shown to suffer less root damage in field trials since the entomopathogenic nematode *Heterorhabditis megidis* was strongly attracted to the (*E*)-β-caryophyllene emitted by damaged maize roots.[25]

Carbocation: a molecule bearing positively-charged carbon atom with three bonds. The general order of stability is tertiary > secondary > primary. Terpene synthases generate diverse terpene structures from simple precursors through multistep-carbocation rearrangements.

2.2 *Phenylpropanoids/benzenoids*

Phenylpropanoids are involved in many aspects of plant responses to biotic and abiotic stress factors as well as being components of structural plant polymers such as lignin and suberin.[26,27] They are synthesized via the shikimate pathway in plants that is localized in the plastids. In the first step *L*-phenylalanine is converted into *trans*-cinnamic acid by the enzyme *L*-phenylalanine ammonia-lyase (PAL). The downstream steps are shared with lignin biosynthesis leading to phenylpropanoid monomers such as coumaric acid, ferulic acid and sinapic acid.[26] After this, through hydroxylation and methylation reactions, a variety of volatile cinnamic acid derivatives are formed. In comparison, the formation of phenolic compounds with C_1–C_6 chains such as benzoic acid is not well known. The synthesis of such compounds starts as a branch from *trans*-

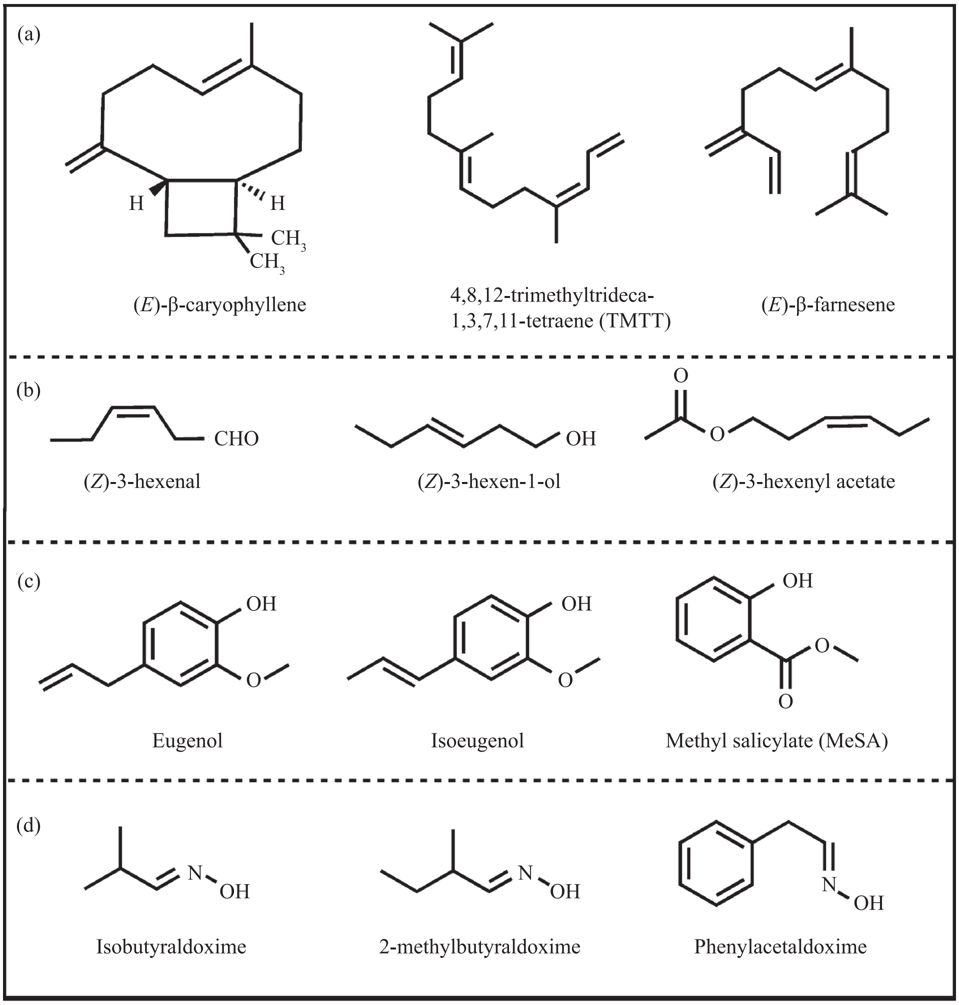

Figure 1: Chemical structures of important HIPVs discussed in this section. (a) Terpenoids (b) Green leaf volatiles (c) Phenylpropanoids and (d) Amino acid derived volatiles.

cinnamic acid and proceeds either towards oxidative or non-oxidative pathways. In *Petunia hybrida* petals it was shown by deuterium-feeding experiments that the flux through CoA-independent non-oxidative path from cinnamate to benzoic acid was about twice as that of the oxidative pathway involving benzoyl CoA.[28] From benzoic acid, it was believed that its 2-hydroxy derivative, SA, is formed by a cytochrome P450 monooxygenase identified first in tobacco.[29] However, evidence indicates that SA is derived from chorismate because the *Arabidopsis thaliana sid2* mutant encoding isochorismate synthase showed low levels of SA.[30,31] Methylation of SA converts it into a mobile signal MeSA. This O-methylation is catalyzed by the SABATH family of methyltransferases implicated in phenylpropanoid volatile formation as well as SA homeostasis.[32,33] In addition, phenylpropenes also function as antimicrobials.[34] For instance, phenylpropenes such as eugenol or isoeugenol are formed from coniferyl acetate in a reaction catalyzed by eugenol or isoeugenol synthase in *Petunia* and basil.[35,36] Eugenol can induce cell lysis by leakage of proteins and lipid contents, and is thus effective as an antibacterial agent.[37]

2.3 *Fatty acid derivatives*

Volatile fatty acid derivatives such as hexenal, hexenol and hexenyl acetate are six-carbon volatiles commonly known as ‘green leaf volatiles’ (GLVs) (see Fig. 1). GLVs are ubiquitously made by all green plants and are emitted immediately upon tissue damage.[38] These compounds originate from linolenic or linoleic acid by the action of lipoxygenase (LOX) and hydroperoxide lyase (HPL) as a side-branch of oxylipin synthesis.[39] Cleavage of free unsaturated fatty acids from membrane lipids is the rate limiting step in their synthesis but the specific lipases involved

Antimicrobials: compounds that can kill or inhibit the growth of microorganisms.

Oxylipins: cyclic or acyclic products of polyunsaturated fatty acid oxidation.

in GLV biosynthesis are still unknown.[40] Lipase activity in insect (grasshopper) oral secretion was shown to play an important role in eliciting plant defence responses in *Arabidopsis*.[41] Thus, HPL is an important enzyme for GLV synthesis as shown by studies manipulating its activity. *Arabidopsis thaliana* expressing *HPL* ectopically showed significant increase in GLVs upon *Pieris rapae* caterpillar damage subsequently attracting higher numbers of the parasitic wasp *Cotesia glomerata*.[42] *Nicotiana attenuata* plants with reduced expression of HPL (*as-HPL*) were less attractive to the predator *Geocoris* sp.[43] In addition to being a part of the volatile bouquet released upon herbivory, GLVs play an prominent role in 'priming', a state which prepares a plant to respond to stress in an accelerated manner.[40] (*Z*)-3-hexen-1-ol, (*Z*)-3-hexenyl acetate and (Z)-3-hexenal are known to prime defence responses such as higher JA concentrations in maize[44] and poplar.[45,46] Further, exposure to (*E*)-2-hexenal induced another anti-herbivore defence, trypsin proteinase inhibitors (TPI), in *N. attenuata* plants upon feeding by the tobacco worm *Manduca sexta*, suggesting a role of this GLV in priming.[47] Interestingly, isomerization of GLVs is known to be triggered by herbivory.[48] In the same study, it was shown that the predatory insect could clearly distinguish between the (*Z*) and (*E*) isomers of GLVs[48] underlining the specificity of tri-trophic interactions mediated by volatiles. (*Z*)-3-hexenyl acetate was identified as the specific VOC capable of priming another indirect defence, extrafloral nectar secretion in wild lima bean plants.[49,50]

Indirect plant defence: a plant defence response that benefits the plant indirectly. For example, carnivorous predators attracted to plants emitting specific volatiles aid by relieving the herbivore-pressure.

Priming: when plants prepare their defences after sensing a signal or due to a previous challenge and respond with increased vigor, they are said to be in a primed state.

2.4 *Amino acid-derived volatiles*

Amino acid (AA)-derived volatiles are widely reported in many fruit aroma bouquets such as melon, banana, tomato and apple.[51] The biosynthetic pathway of amino acid catabolism is well known in microorganisms where 2-keto acids serve as substrates for downstream reactions and plants are thought to possess similar pathways.[52] These reactions followed by decarboxylation, reductions and esterification form aldehydes, acids, alcohols and esters. The esterification by alcohol dehydrogenase and acetyltransferase has been extensively investigated in melon and other fruits. AA-derived volatiles such as aldoximes constitute a minor portion of the HIPV blend. Eight different aldoximes were released from *Populus trichocarpa* when gypsy moth caterpillars fed on them.[53] They are also reported to be produced upon spider mite infestation on various plant families.[54] The biochemistry and function of aldoximes is not clearly understood and warrants further study.

3 Hormonal Regulation of HIPVs

HIPV emission is controlled by activation of different phytohormone signaling pathways, of which three are most important: 1. Octadecanoid pathway (JA biosynthesis), 2. Shikimate pathway (SA biosynthesis), and 3. Ethylene (ET) pathway. Individual herbivores are known to activate different combinations of phytohormones and interplay between hormones alter plant responses.[7,17,55] When a herbivore chews on a leaf, the elicitors present in its oral secretions interact with the plant cells at the site of damage and lead to strong Ca^{2+}-mediated depolarization and activate the mitogen-activated protein kinase (MAPK) cascade. These kinases regulate the herbivore-induced levels of JA, SA and ET.[56]

3.1 *Jasmonate (JA)*

JA modulates plant defences against herbivorous insects and necrotrophic pathogens while SA is known to regulate defences against biotrophic pathogens and piercing/sucking herbivores.[57] The levels of these phytohormones are induced within few hours of herbivore attack. JA is synthesized via the oxylipin or octadecanoid pathway, which is by far the most important signaling cascade for HIPV emission. As mentioned earlier, the biosynthesis of these hormones is initiated by LOX, which catalyzes stereoselective dioxygenation of polyunsaturated fatty acids. After the action of the enzymes allene oxide synthase (AOS) and allene oxide cyclase (AOC), JA is finally formed from its precursor, 12-oxo-phytodienoic acid (OPDA), after 2–3 rounds of β-oxidation.[58,59] JA is converted into its bioactive form, JA-Ile, by the jasmonic acid-amido synthetase JAR1.[60] JA-Ile binds to the SCF^{COI1} complex (Skip-Cullin-F-box protein, CORONATINE INSENSITIVE1) leading to the proteosomal degradation of JASMONATE ZIM-domain (JAZ) transcriptional repressor proteins culminating in the activation of several defence-related genes[61] (Fig. 2). Recently, *JAV1* has been identified as a key gene in the JA pathway serving as a negative regulator of plant defences against herbivores and pathogens.[62] Exogenous application of JA induces a volatile blend in lima bean plants which is similar to the blend induced by the attack of the herbivorous spider mite, *Tetranychus urticae*, with only slight differences such as MeSA and 4,8,12-trimethyl-1,3-(*E*),7(*E*),11-tridecatetraene (TMTT) being detected only upon the real herbivore attack.[63] Additionally, JA-induced plants were visited by more carnivorous insects than non-induced plants[64] although no change in pollinator preferences were reported[65] suggesting that JA treatment may be used effectively in pest

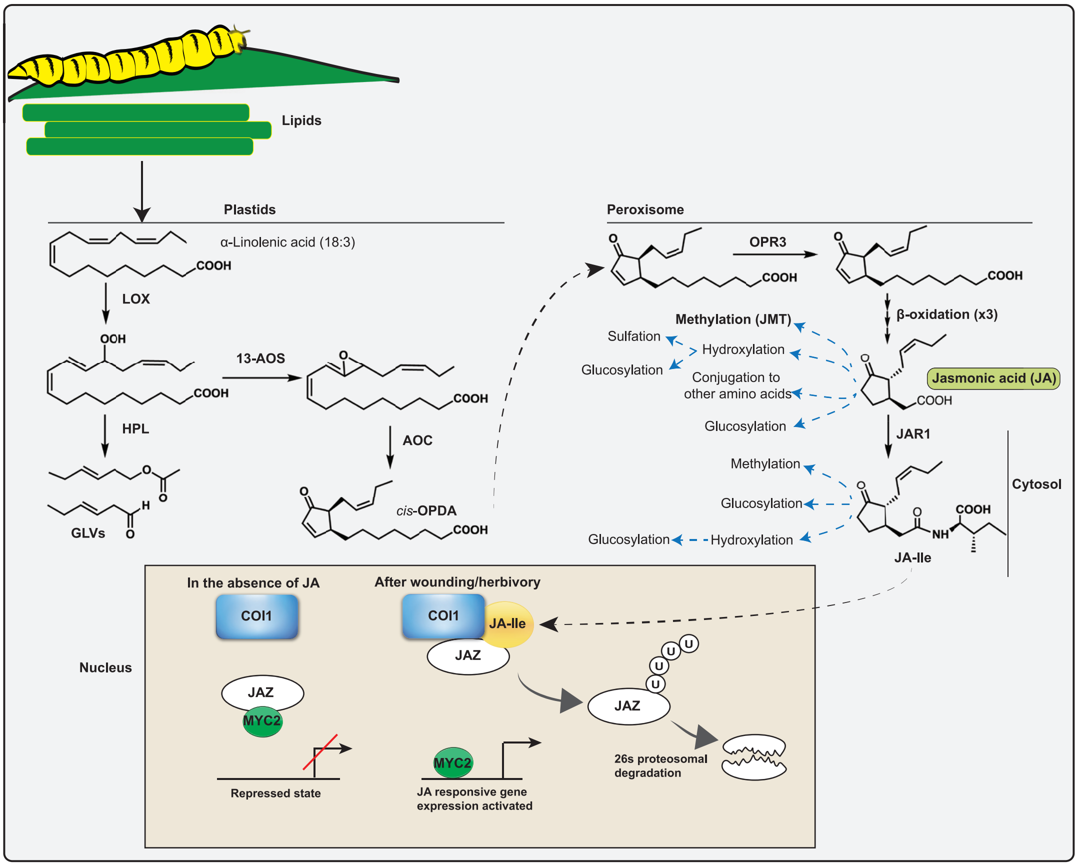

Figure 2: JA biosynthesis and signaling pathway. JA biosynthesis starts in the plastids, the product cis-OPDA is transported to the peroxisome by an ABC transporter protein, where it is reduced to JA. Blue arrows represent the chemical modifications of JA and JA-Ile. Methylation of JA converts it into MeJA which is an important volatile hormone. The hydroperoxide lyases (HPLs) form a branch of the oxylipin pathway leading to GLVs. Upon herbivory, JA biosynthesis is triggered which relieves the JAZ repression culminating in the activation of JA-responsive genes for plant defences.

management. Even plants grown from seeds treated with JA showed increased resistance against herbivory and the necrotrophic fungal pathogen, *Botrytis cinerea*.[66] Numerous mutants impaired in JA synthesis or response have been characterized which clearly emphasized the role of JA in plant defence.[67–69] JA also plays a major role in plant reproductive development as shown by several *Arabidopsis* JA mutants being male sterile.[70] Interestingly, JA interacts with other phytohormones. Microarray analysis of *Arabidopsis* WT and *coi1* mutant plants after herbivory revealed that of the 41 JA-related genes, three are involved in ethylene, auxin and SA pathways confirming the crosstalk between phytohormones.[71] For instance, the *Arabidopsis* mutant *constitutive expression of vegetative storage protein* (*cev1*) has a dwarf phenotype that constitutively produces JA and ethylene, and treating this mutant with SA suppresses *PDF1.2* (JA-responsive gene).[72] This and several other reports clearly established that JA and SA act antagonistically[73] while JA and ethylene interact in a synergistic fashion.[74] JA–SA crosstalk also affects host preference and oviposition-site selection as shown in lima bean–spider mite interaction.[75]

3.2 *Salicylic acid (SA)*

SA is synthesized via two pathways both of which require chorismate.[31] One of them is catalyzed by phenylalanine ammonia lyase (PAL), whereas in the other pathway SA is formed from isochorismate by the action of isochorismate synthase (ICS).[31] MeSA is a volatile ester, commonly not present in plants but induced upon pathogen infection or herbivory. MeSA is reported in the headspace volatiles of many plants such as lima bean,[75] *Arabidopsis*, tomato and soybean[77] within hours of damage. Also, MeSA is attractive to insect predators singly and in combination with other

Chemoreceptors: these help insects in detecting the chemical composition of their internal and external environment in addition to identifying oviposition sites and suitable mates. The chemosensory neurons are present in special sensory structures called sensilla.

HIPVs[78,79] since the chemoreceptors of insects are sensitive to MeSA.[80,81] An important role of SA in anti-herbivore defence stems from the fact that it interacts antagonistically with JA signalling.[73,75] However, there is contrasting evidence; for example, treatment of lima bean plants with MeSA and JA induced a volatile blend closely resembling that of *T. urticae*-induced volatiles rather than JA treatment alone which suggested that the JA and SA signaling pathways are synergistically involved in HIPV induction.[76] In another study, it was shown that caterpillar-infested *Arabidopsis NahG* plants (that do not accumulate SA) were less attractive to parasitoids, which could be a cumulative effect of SA–JA antagonism.[82] It is interesting to note that in the same study, exogenous application of MeSA failed to attract parasitoid wasps.[82]

3.3 *Ethylene*

Ethylene modulates pathogen responses, seed germination, root hair growth, nodulation, flower senescence and fruit ripening.[83–85] The first committed step in the biosynthesis of ethylene is the conversion of *S*-adenosyl-methionine to 1-aminocyclopropane-1-carboxylic acid (ACC) by ACC synthase[83,86] ACC is then oxidized to ethylene by ACC oxidase, the byproducts being CO_2 and cyanide, which is subsequently detoxified. Induction of JA upon wounding also induces an ethylene burst via increase in ACC synthase activity. Wound-induced JA accumulation on the other hand is known to reduce to 20–30% when plants are treated with ethylene biosynthesis inhibitors.[84] In *Zea mays*, pre-treatment with 1-methylcyclopropene (1-MCP), an inhibitor of ethylene perception, did not lead to altered JA levels following feeding by *Spodoptera* caterpillars but reduced the emission of sesquiterpene and indole volatiles.[87] Exogenous treatment with JA in combination with ACC enhanced the production of (*E*)-β-ocimene and (*Z*)-3-hexenyl acetate as well as attractiveness of lima bean plants to the predatory mite *Phytoseiulus persimilis*, a natural enemy of *T. urticae*.[88] Further, plant–plant signaling mediated by (*Z*)-3-hexen-1-ol was enhanced when the receiver plants were exposed to ethylene.[89]

4 Future Perspectives

Much evidence has accumulated in the past few decades supporting the role of HIPVs as an effective anti-herbivore defence mechanism. Still, it is unclear how a plant would respond to multiple attacks from different types of herbivores, a condition which can be easily expected in nature. Below-ground herbivory is known to influence above-ground volatile emission[90] and it was shown that phloem-feeding insects interfere with the attraction of the parasitoid *Diadegma semiclausam* to *A. thaliana* plants.[91] These examples clearly illustrate the need to understand volatile emission from a community perspective; yet, very few studies addressing multiple infestations are known.[9] Further, plant growth hormones such as cytokinin, gibberellins and abscisic acid are emerging as important modulators of the JA pathway suggesting that plant responses to different attacks are fine-tuned by many interacting phytohormones.[17,92] The complexity of HIPV emission and the underlying network of various phytohormones provides a challenge in relating biochemistry with ecological consequences. However, using molecular tools such as silencing specific genes in plants and manipulative field studies could be useful in the study of community dynamics shaped by these indirect plant defences.

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