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Volatile dialogues between plants and microorganisms

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Volatile organic compounds (VOCs) are key mediators of long-distance communication in biological systems. While their roles in plant–insect interactions are well established, emerging evidence highlights their importance in plant–microbe interactions. In this highlight, we discuss the biosynthesis and ecological functions of plant VOCs (pVOCs) and their impact on microbiome assembly and function. We examine how constitutive and stress-induced pVOCs shape microbial community composition and how microbial VOCs (mVOCs) influence plant growth and defense by modulating hormonal and metabolic pathways. We further address the bidirectional nature of volatile-mediated interactions and the challenges associated with studying complex VOC blends in natural environments. Understanding these dynamic volatile dialogues provides new opportunities for microbiome engineering and sustainable crop production.

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1. Introduction
2. The biosynthesis of plant-emitted volatile organic compounds (pVOCs)
3. Constitutive pVOC emission in plant–microbe interactions
4. Stress-induced pVOCs and their role in plant–microbe interactions
5. Impact of microbial VOCs on plant growth and defense
6. Experimental challenges and future perspectives
7. Author contributions
8. Conflicts of interest
9. Data availability
10. Acknowledgements
11. References

1. Introduction

Living organisms communicate *via* a sophisticated chemical language composed of structurally diverse compounds. Among these, volatile organic compounds (VOCs) represent a dynamic class of small, low-molecular-weight metabolites with high vapor pressure and the ability to diffuse over relatively long distances.[1] VOCs are produced by plants, microbes, insects and animals and function as mediators of ecological interactions across spatial and temporal scales.[2] Unlike non-volatile metabolites that typically act locally, VOCs enable organisms to exchange information without direct physical contact, making them powerful long-distance communication signals in natural and man-made ecosystems. Historically, research on VOC-mediated communication was elegantly demonstrated in studies of plant–insect interactions. Foundational work demonstrated that plants emit insect-induced volatile blends that attract parasitoids of these herbivores, thereby mediating indirect plant defense.[3] These exciting discoveries shed light on the role of plant volatiles as central players of the so-called "cry-for-help" phenomenon and laid the groundwork for chemical ecology of VOCs in multitrophic interactions. Over time, the scope of VOC-mediated interactions has broadened substantially. It is now evident that VOCs also act in plant–microbe and microbe–microbe interactions, particularly in the plant phyllosphere, anthosphere and rhizosphere where they mediate cross-kingdom communication without physical contact.[4–8] Increasing evidence also suggests that this "cry-for-help" phenomenon extends to belowground interactions, where plant roots exude specific metabolites to recruit beneficial microorganisms for protection against biotic (*e.g.* fungal root diseases) and abiotic stresses (*e.g.* drought).[9,10] Non-volatile root exudates have long been recognized as mediators of plant microbiome assembly, but volatiles may function as complementary

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communication signals capable of shaping soil microbial community composition and activity from a distance.[10,11] Several intrinsic properties make VOCs uniquely suited for plant–microbe communication both aboveground and belowground. VOCs generally have a low molecular weight (<300 Da) and are hydrophobic, allowing for rapid diffusion not only in the atmosphere, but also in gas and aqueous phases in soil pores.[12] The rapid inducibility of plant VOCs enables dynamic responses to biotic and abiotic stresses, directly linking metabolic changes to the emission of signals that influence the surrounding environment.[13] VOCs are typically emitted as complex blends, with qualitative or quantitative shifts in VOC profiles as a signature of context-dependent information.

In this Highlight, we discuss the emerging role of plant VOCs (pVOCs) as prominent communication signals in plant defense. We focus on how constitutive and stress-induced pVOCs influence microbiome assembly, both directly and indirectly *via* ecological interactions and signaling cascades. Furthermore, we examine microbial perception of pVOCs, including the induction of microbial VOCs (mVOC) and their reciprocal effects on plant physiology, stress resilience, and microbiome dynamics. We highlight that fundamental knowledge of the intricate bidirectional volatile dialogues between plants and microorganisms can give new directions to management of plant health and sustainable crop production.

2. The biosynthesis of plant-emitted volatile organic compounds (pVOCs)

pVOCs are the products of diverse primary metabolic pathways (Fig. 1) and are emitted from various plant organs, including leaves, flowers, and roots, where they mediate a range of physiological and ecological functions.[5,7] Based on their chemical structure and biosynthetic origin, pVOCs can be broadly grouped into terpenoids, fatty acid derivatives, phenylpropanoid/benzenoid compounds, sulfur- or nitrogen-containing volatiles.[14,15] Each pVOC class arises from distinct metabolic routes and contributes differently to plant development, environmental adaptation, and biotic interactions.

Terpenoids represent one of the largest and structurally most diverse pVOCs.[16] They are synthesized from the universal five-carbon precursors isopentenyl diphosphate (IPP) and dimethylallyl diphosphate (DMAPP), which are produced *via* two independent pathways: the mevalonic acid (MVA) pathway and the deoxyxylulose 5-phosphate (DOX) pathway (Fig. 1).[16,17] The MVA pathway occurs in fungi, some bacteria and in the cytosol of plants, while the DOX pathway is more common in bacteria and in plastids of plants, reflecting the evolutionary connection between chloroplasts from bacteria (based on the endosymbiont hypothesis). Both pathways make use of intermediates and/or final products of glycolysis: in a first step, the DOX pathway fuses D-glyceraldehyde 3-phosphate and pyruvate to the eponymous intermediate deoxyxylulose 5-phosphate.[18] Along the first transformations in the MVA pathway, three units of acetyl-CoA are assembled to yield mevalonic acid. Despite their independent evolution, both pathways end at IPP, with simultaneous production of its isomer DMAPP in case of the DOX pathway only. An isopentenyl diphosphate isomerase (IDI) can mediate between these two C_5 monomers and is strictly required for organisms relying on the MVA pathway, but optional for organisms using the DOX pathway.[19] In general, the MVA pathway predominantly supplies precursors for sesquiterpenes, whereas the DOX pathway contributes to hemiterpene, monoterpene, and diterpene biosynthesis. Because of their fundamentally different reactivity, the allyl diphosphate DMAPP is an electrophile and the homoallyl diphosphate IPP reacts as a nucleophile, these monomers can be coupled through catalysis by a prenyltransferase to yield geranyl diphosphate (GPP, the precursor to monoterpenes C_{10}).[20] The product itself is an allyl diphosphate, allowing for repeated nucleophilic substitutions with IPP units to sequentially form farnesyl diphosphate (FPP, sesquiterpene precursor, C_{15}) and geranylgeranyl diphosphate (GGPP, diterpene precursor, C_{20}). Even higher oligoprenyl diphosphates may be formed, but they are, as a result of the high molecular weight of terpenes derived from them, not considered as precursors to VOCs and will not be further discussed in this article. The shorter intermediates up to C_{20} serve as precursors to volatile terpenes that may be generated by terpene synthases (TPSs), a highly diverse enzyme class that produces a plethora of structurally distinct terpene hydrocarbons and alcohols. The complex cascades involve typical elementary steps such as cyclizations, Wagner-Meerwein rearrangements, hydride and proton transfers.[21] Isoprene is among the most abundant terpenes, largely due to its extensive production by terrestrial plants and its substantial emission into the atmosphere, where it plays a central role in atmospheric chemistry and secondary organic aerosol formation.[22] Alongside isoprene, monoterpenes constitute another major and structurally diverse class of biogenic volatile organic compounds (bVOCs) emitted by plants, with significant contributions to plant physiology and adaptation to environmental stress.[23]

Plants also emit aromatic volatiles derived primarily from the shikimate pathway.[14,24] The shikimate pathway is rooted in D-erythrose 4-phosphate formed from glucose through the pentose phosphate cycle and phosphoenolpyruvate, and proceeds towards aromatic amino acids including phenylalanine. Aromatic compounds are typically classified based on side chain length, with phenylpropanoids possessing an Ar–C_3 backbone (aryl substituent at a C_3 chain). Phenylacetate derivatives exhibit an Ar–C_2 skeleton and benzoate derivatives a shorter Ar–C_1 structure (Fig. 2). Transformation of the C_3 substituent at the benzene ring leads to phenylpropanoids (Ar–C_3) and is typically initiated by L-phenylalanine ammonia-lyase (PAL), which converts phenylalanine into *trans*-cinnamic acid. This intermediate is subsequently transformed through cinnamoyl-CoA into *para*-coumaroyl-CoA, a key metabolic hub for numerous plant metabolites.[25] A famous downstream product is the widespread compound eugenol, one of the main flavour compounds in cloves. Side chain degradation by two carbons gives access to benzoate derivatives (Ar–C_1). This compound class can be reached from cinnamoyl-CoA through side chain degradation by two carbons involving hydration and

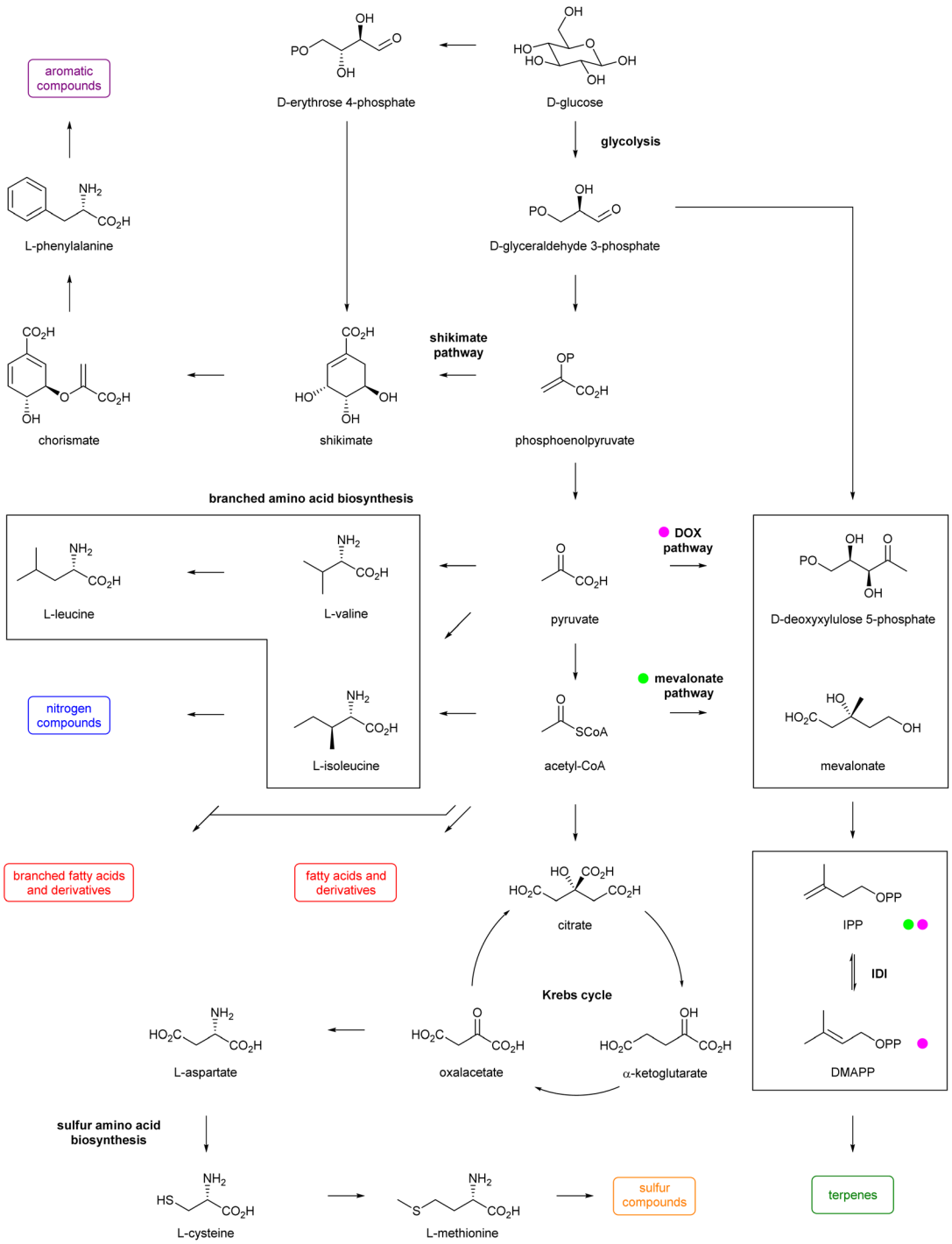

Fig. 1 Primary metabolic pathways and their connection to the biosynthesis of VOCs.

cleavage to benzaldehyde and acetyl-CoA, followed by oxidation to benzoic acid. Benzoate derivatives constitute a large family of natural products including methyl benzoate, benzaldehyde, benzyl alcohol and methyl salicylate.[26] A degradation by one carbon is also possible, resulting in Ar–C_2 compounds (phenylacetate derivatives). They can be formed from phenylalanine by transamination to phenylpyruvate and oxidative decarboxylation to phenylacetyl-CoA. Widespread representatives are phenylacetic acid, an important plant hormone,[27] phenylethanol, esters derived from it, and phenylacetaldehyde. Overall, aromatic compounds constitute a significant proportion of pVOCs and typically exhibit pronounced floral or spicy scents.[14,24]

Fatty acid-derived volatiles represent another major class of pVOCs and are often rapidly generated in response to tissue disruption or environmental stress. These compounds originate from membrane lipids and are primarily synthesized through the lipoxygenase (LOX) pathway (Fig. 3).[28] Fatty acid

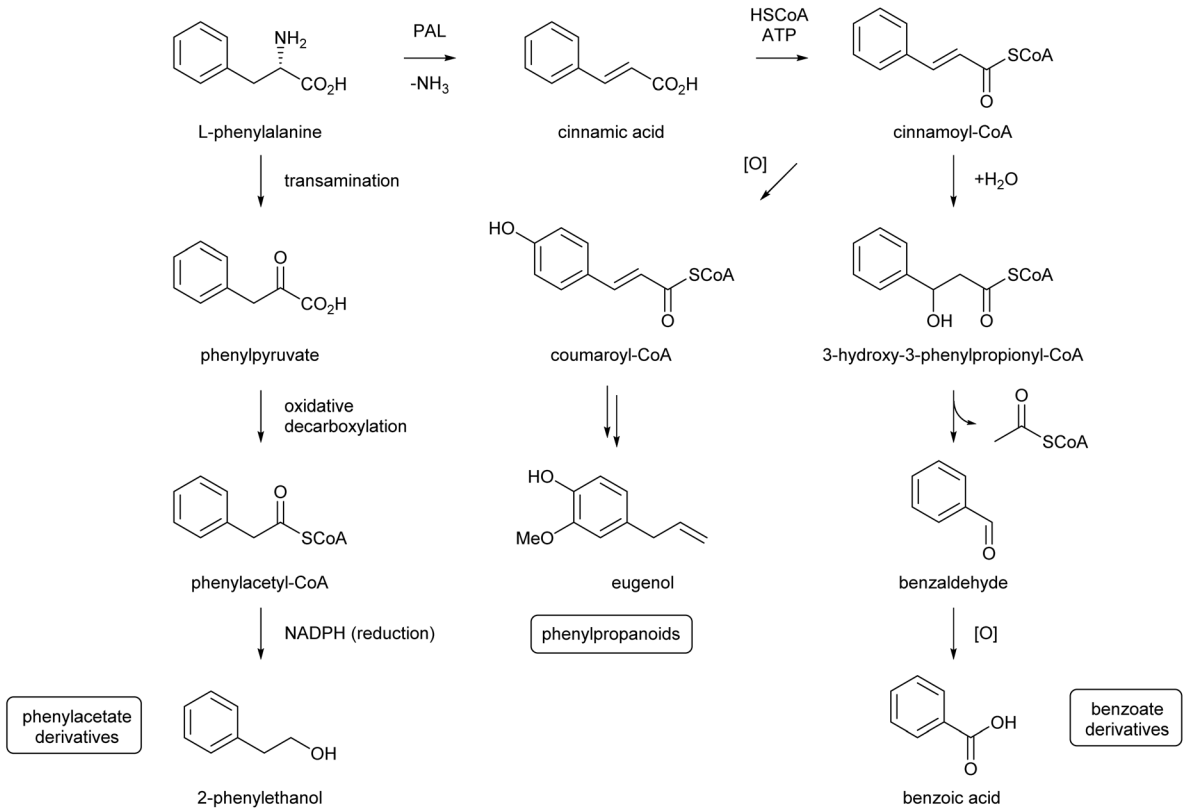

Fig. 2 Primary metabolic pathways and their connection to the biosynthesis of VOCs.

biosynthesis is an iterative process in which decarboxylative Claisen condensations and reductions of the 3-oxo group convert the starter unit acetyl-CoA and 8 elongation units of malonyl-CoA ultimately form stearic acid. The ubiquitous Δ^9 desaturase introduces a *Z* double bond at C9 to yield oleic acid.[29] Additional double bonds are introduced towards the methyl terminus, leading to linolic acid and α-linolenic acid by the Δ^{12} and Δ^{15} desaturases, respectively.[30] Upon mechanical damage or herbivory, linolenic acid is oxidized at C13 by a lipoxygenase (LOX) to 13-HPOT and further processed by a hydroperoxide lyase (HPL) to yield (*Z*)-3-hexenal (C_6).[31,32] Analogous reactions with oxidation at C10 can lead to corresponding C_9 compounds.[33] Downstream modifications produce a range of C_6 and C_9 aldehydes, alcohols, and esters collectively known as green leaf volatiles (GLVs).[34] These GLVs, including (*E*)-2-hexenal, (*Z*)-3-hexenal, (*Z*)-3-hexen-1-ol and (*Z*)-3-hexenyl acetate are among the fastest-emitted plant VOCs and often function as early chemical signals associated with physical wounding and herbivore attack.[35] In addition to these major classes, plants also produce sulfur- and nitrogen-containing VOCs that arise from distinct metabolic origins. Sulfur-containing volatiles are often derived from sulfur-rich secondary metabolites such as glucosinolates, which are enzymatically converted into VOCs such as allyl isothiocyanate, dimethyl disulfide, and dimethyl trisulfide.[36] Nitrogen-containing VOCs typically originate from amino acid metabolism and include volatile compounds such as indoles[14] and pyrazines.[37,38] Compared to other VOC classes, these compounds are often produced in smaller quantities but can contribute substantially to the overall diversity of pVOC blends. The biosynthesis of sulfur and nitrogen-containing VOCs is frequently associated with stress-responsive pathways,[39,40] reflecting the integration of primary and secondary metabolism in shaping pVOC emissions.

3. Constitutive pVOC emission in plant–microbe interactions

Under non-stress conditions, plants constitutively emit pVOCs that are often linked to developmental processes such as growth, floral scent and fruit ripening.[24,41,42] Constitutive pVOC emissions represent baseline chemical landscapes that drive the initial assembly and establishment of microbiota on plant surfaces (Fig. 5). Continuous pVOC emissions from leaves, flowers, and roots can influence microbial metabolism and competitive interactions, thereby contributing to the formation of a stable microbiome composition. For example, constitutive emission of methanol released by plant leaves during cell wall expansion can serve as a carbon source for methylotrophic phyllosphere bacteria such as *Methylobacterium extorquens*. Such study further showed that methanol emissions from axenic (microbe-free) leaves of *Nicotiana tabacum* were higher than from tissues colonized by these bacteria, suggesting active microbial consumption of pVOCs. Similarly, isoprene (**1**) (Fig. 4) emissions from oil palm trees coincided with the enrichment of

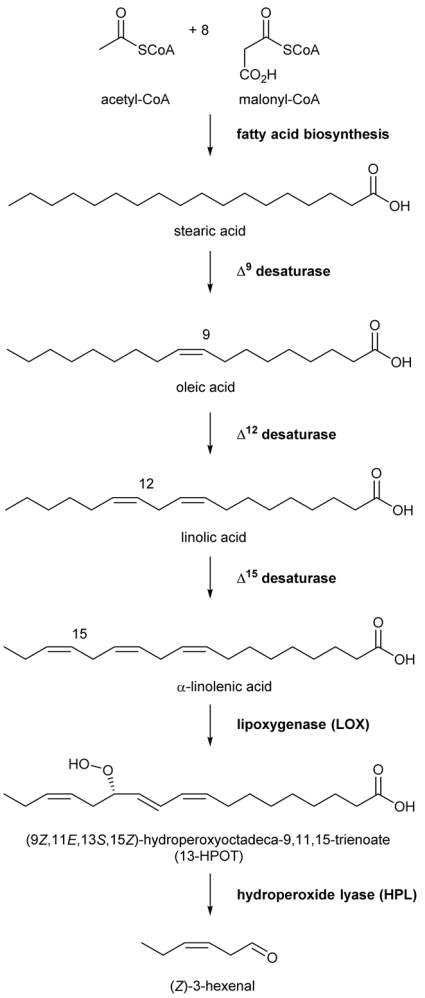

Fig. 3 Biosynthesis of fatty acid derived volatile compounds.

isoprene-degrading bacteria in the phyllosphere, including members of the genera *Novosphingobium*, *Pelomonas*, *Rhodoblastus*, *Sphingomonas*, and *Zoogloea*.[43] Constitutive emission of linalool (2) from maize plants can prime neighboring plants by activating root jasmonate signaling and promoting the exudation of 2-(2-hydroxy-4,7-dimethoxy-1,4-benzoxazin-3-one)-β-D glucopyranose (HDMBOA-Glc), a benzoxazinoid defense metabolite, from the roots. This response subsequently alters the composition of the rhizosphere microbiome and reciprocally enhances plant defense.[44] Together, these observations illustrate a direct and indirect metabolic link between pVOC emissions and microbial colonization of plants.

Similarly, constitutive floral pVOC blends rich in benzenoids and terpenoids have been shown to influence microbial assemblages on floral surfaces. For example, genotypic variation in floral traits can alter the emission of terpenes such as α- and β-pinene (**6** and **7**) and ocimene isomers, as well as benzenoids including benzaldehyde (**18**) and *p*-anisaldehyde (**19**), which in turn influence the composition of floral bacterial and fungal communities.[45] Recent work also suggests that floral scent composition can shape microbial diversity in the anthosphere, with higher chemical diversity often associated with reduced microbial diversity, consistent with floral pVOCs acting as selective filters that regulate microbial colonization of the flower.[5]

Constitutive emissions are also common belowground, where plant roots release pVOCs into the surrounding soil. For instance, *Arabidopsis thaliana* roots synthesize monoterpenes such as 1,8-cineole (**5**), while roots of *Daucus carota* release myrcene (**3**), γ-terpinene (**9**), and *p*-cymene (**11**), with notable variation across genotypes.[46,47] Similarly, several root-emitted monoterpenes, including α-pinene (**6**), 2-carene (**8**), and α-terpinene (**10**), have been reported from tomato plants, with emission profiles varying between domesticated and wild genotypes.[48,49] Experimental evidence further supports a role for terpenes in shaping rhizosphere microbiomes. For example, exogenous application of monoterpenes (*e.g.* **2** and **5**) and a sesquiterpene (nerolidol (**4**)) delivered through an artificial root system altered bacterial and fungal community compositions in the sorghum rhizobiome.[50] Notably, these effects varied depending on terpene identity and concentration as well as strain specificity, highlighting spatially specific impacts of terpene exposure on root-associated microbiota. Recently, the emission of the volatile plant hormone methyl jasmonate (**14**) by roots was also found to change soil microbial community structure and induce biofilm formation.[6] Collectively, these findings indicate that steady-state pVOC emissions may function as persistent ecological filters that help define baseline microbial niches and microbiome assembly (Fig. 5).

4. Stress-induced pVOCs and their role in plant–microbe interactions

Plants produce a subset of pVOCs when exposed to biotic stresses, including herbivore attack and pathogen invasion.[28] These stress-induced pVOC emissions are typically rapid and transient, reflecting dynamic reprogramming of core metabolic pathways including the isoprenoid, lipoxygenase, and benzenoid/phenylpropanoid routes.[51] Importantly, stress-induced pVOC emissions are often spatially and temporally heterogeneous, generating fluctuating chemical landscapes that contrast with the relatively stable profiles of constitutive pVOC emissions.[52] Such transient pVOC bursts may act as perturbations that reshape plant-associated microbiomes by selectively inhibiting growth or motility of sensitive taxa while favoring stress-tolerant microbes. In this way, inducible pVOCs may function as dynamic ecological filters that promote microbiome plasticity under stress conditions.

A hallmark of stress-responsive pVOC emissions is their rapid induction upon stress perception. For example, herbivore attack triggers the release of herbivore-induced pVOCs (also referred to as HIPVs), including green leaf volatiles (GLVs), terpenoids, and aromatic compounds that are largely regulated by jasmonate-dependent signaling pathways.[34] While these compounds are well known for mediating multitrophic interactions such as predatory insect recruitment, emerging evidence suggests that these HIPVs can also recruit beneficial microbial genera. HIPVs blends have been associated with shifts in phyllosphere microbiota, potentially through

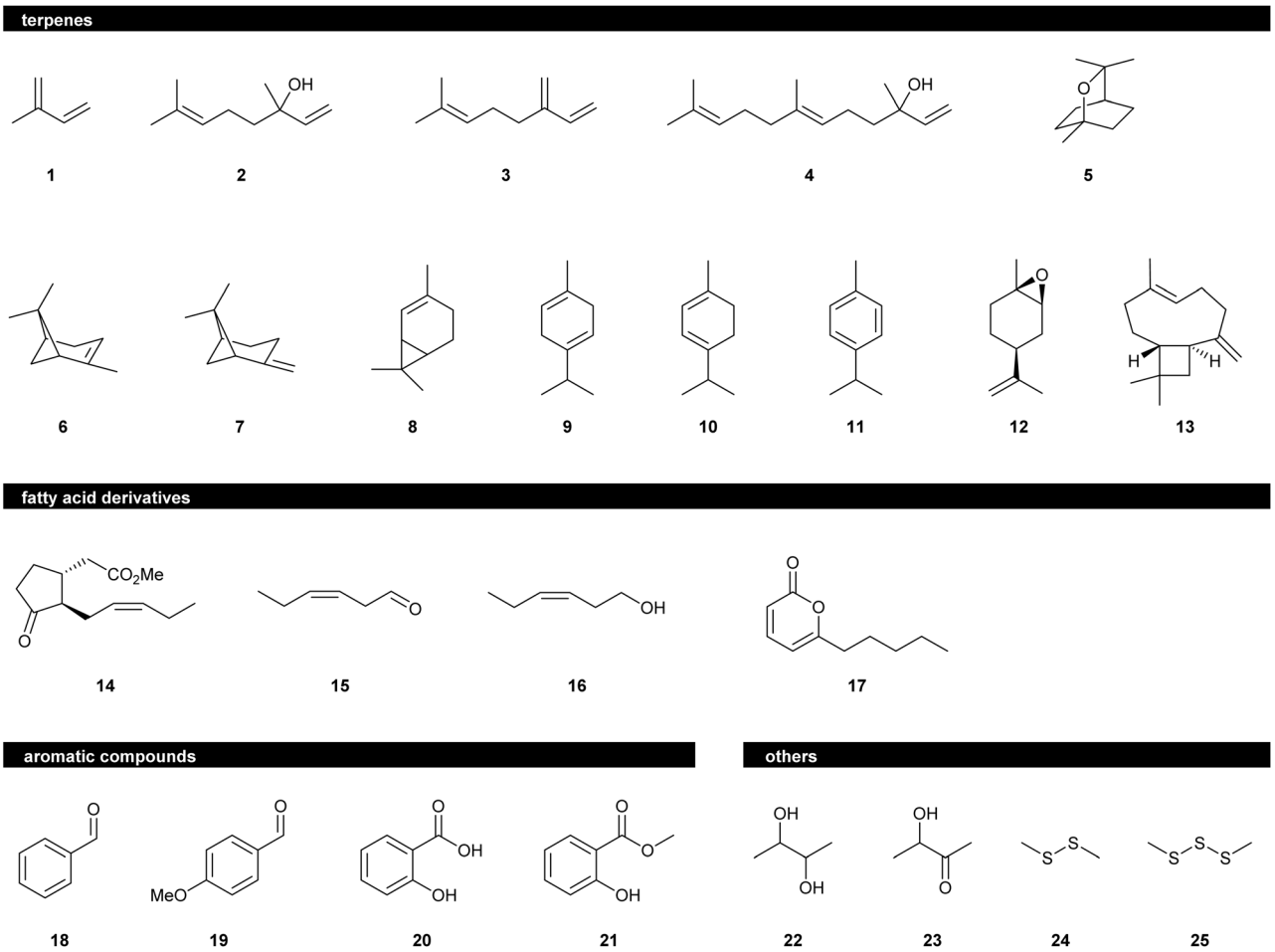

Fig. 4 Structures of the plant and microbial-emitted VOCs involving in plant–microbe interactions with compound numbers corresponding to those discussed in the text (indicated by the bold numbers in text).

antimicrobial activity or changes in surface chemistry and nutrient availability.[53] GLVs derived from the lipoxygenase pathway, such as (*Z*)-3-hexenal (**15**) and (*Z*)-3-hexen-1-ol (**16**), possess antimicrobial properties and may transiently suppress susceptible microbial taxa, acting as short-lived ecological filters on leaf surfaces.[54]

Stress-induced pVOCs can also systemically influence the physiology of neighboring plants or induce changes in their metabolic profiles, which may ultimately affect rhizosphere microbiome assembly. For instance, herbivory-induced GLVs can activate jasmonate-dependent systemic defense signaling in neighboring receiver plants, resulting in shifts in their rhizosphere microbiome and a concomitant positive effect on plant growth and defense.[55] Similarly, indole, a nitrogen-containing volatile released by herbivore-damaged maize plants, functions as an airborne priming signal that enhances jasmonate-dependent defense responses and resistance to subsequent herbivore attack in neighboring plants.[56] Although its effects on microbiome assembly remain less well characterized, indole-induced changes in plant defense metabolism may indirectly influence root exudation patterns and the recruitment of beneficial rhizosphere microorganisms.[55,57]

Additionally, exposure of tomato plants to microbially induced pVOCs, such as β-caryophyllene (**13**) emitted by neighboring tomato plants, can trigger salicylic acid (**20**) accumulation in the roots, which in turn reshapes the root-associated microbiome to resemble that of the volatile-producing plants.[58] Furthermore, foliar infection by *Botrytis cinerea* systemically altered root-emitted VOC composition including benzyl alcohol and benzofuran (Fig. 5), a shift that was accompanied by changes in the fungal and bacterial communities.[49] Together, these studies reveal that stress-induced pVOCs can act as dynamic regulators that shape plant microbiome structure and function. Notably, this interaction is bidirectional. While aboveground volatile signaling can modify root-associated microbial communities, root-associated microorganisms can also alter plant defense signaling and induce systemic resistance, resulting in changes in shoot volatile emissions and enhanced resistance against herbivores and pathogens.[58–60] Consequently, pVOC-mediated plant communication and microbiome assembly operate as a dynamic feedback loop linking aboveground stress responses with belowground microbial community structure and function. By promoting the establishment of microbiomes associated with enhanced defense capacity, these volatile-

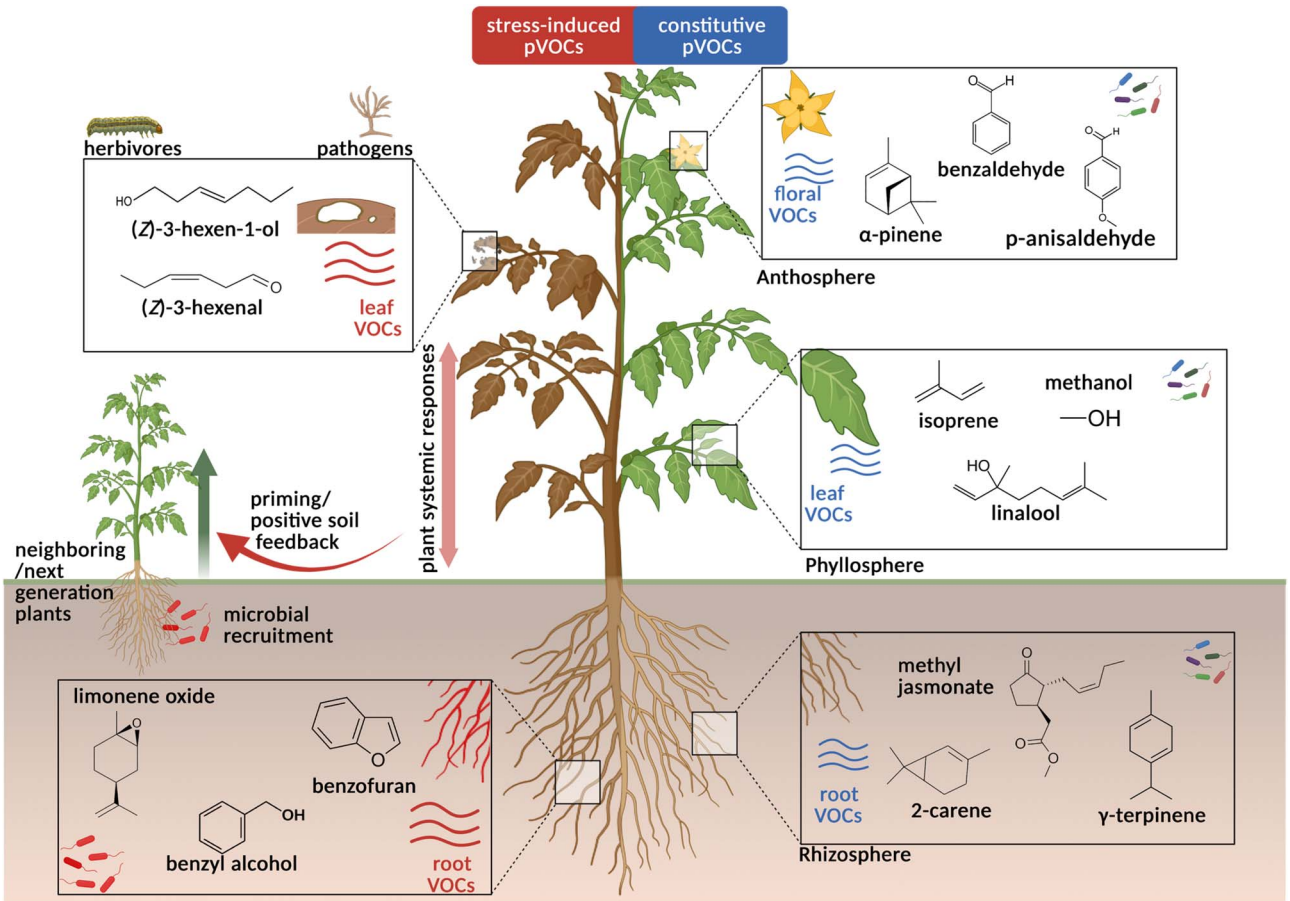

Fig. 5 Representative volatile organic compounds (VOCs) emitted by plants under constitutive and stress-induced conditions which affect microbiome assembly across plant compartments. Plants emit pVOCs from flowers, leaves, and roots under constitutive conditions (*e.g.*, benzaldehyde, isoprene, methyl jasmonate), contributing to microbial community assembly in different plant compartments. Under stress, plants release pVOCs in response to biotic stresses such as herbivores (*e.g.* (*Z*)-3-hexenal and ((*Z*)-3-hexen-1-ol), which function in defense signalling. Plants also locally or systemically respond to aboveground and belowground biotic stresses (*e.g.*, herbivores or pathogens), by changing their emission profile belowground (*e.g.*, limonene oxide, benzofuran and benzyl alcohol) which can recruit beneficial microbial communities. Additionally, pVOCs can indirectly modify root exudate composition in neighboring plants, thereby influencing the microbial establishment in the rhizosphere which promote positive feedback in plants.

mediated interactions may generate a positive microbial legacy that benefits neighboring plants and, potentially, subsequent plant generations through the persistence and transmission of beneficial microbiome configurations.[61]

Recent findings further suggest that plants can recruit beneficial microbes *via* stress-induced rVOCs. For instance, roots of *Carex arenaria* infected with the fungal pathogen *Fusarium culmorum* emitted a distinctive volatile blend containing (*Z*)-limonene oxide (**12**). This compound, which was not detectable in healthy plants and selectively attracted specific bacterial taxa over distances of more than 12 cm.[11] The recruited bacterial taxa in return suppressed the fungal pathogen, illustrating the plant's ability to fine-tune rVOC profiles as part of the "cry-for-help" strategy. Similarly, the volatile hormone ethylene, produced by peanut roots in response to cyanide released by neighboring cassava plants, altered the microbial composition of the peanut rhizosphere by shifting the abundance of Actinobacterial species, ultimately resulting in enhanced peanut seed production.[62] Collectively, these studies demonstrate that stress-responsive pVOCs represent a key ecological interface between plant defense and microbiome assembly, orchestrating a "cry-for-help" response in plants belowground.

5. Impact of microbial VOCs on plant growth and defense

Microorganisms inhabiting the rhizosphere, phyllosphere and anthosphere emit a diverse array of mVOCs that can influence plant physiology. Over the past two decades, a large body of work ranging from single compound assays to community-level volatilomics has shown that mVOCs can (i) promote plant growth, (ii) prime immunity and enhance systemic resistance, and (iii) rewire hormonal networks that connect plant development and defense (Fig. 6).[2,7] At the same time, the outcomes of mVOCs exposure are highly concentration and context-dependent, reflecting variation in microbial taxonomy, environmental conditions, mVOC composition, and plant species.[63]

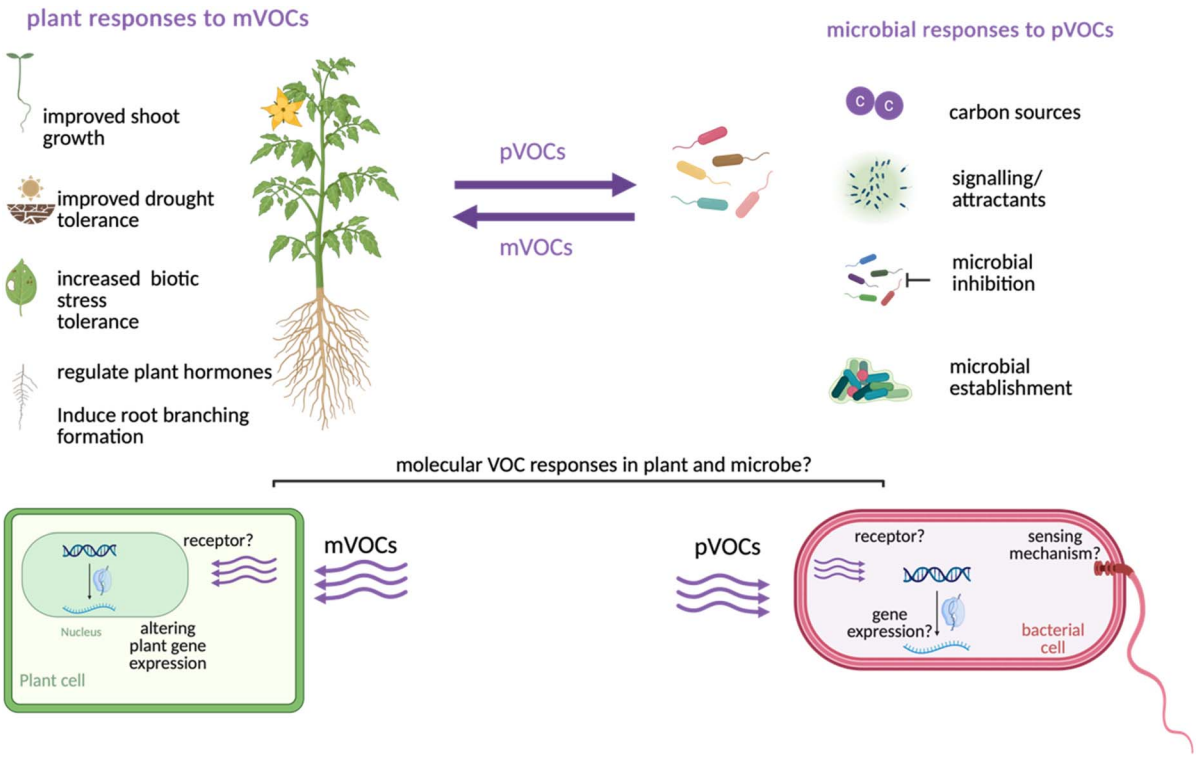

Fig. 6 Bidirectional communication between plants and microbes mediated by volatile organic compounds (VOCs). Plants and associated microorganisms exchange volatile organic compounds (plant-derived VOCs-pVOCs; microbial VOCs-mVOCs) that function as signalling molecules influencing both partners. mVOCs can enhance plant performance by promoting shoot growth, improving drought tolerance, increasing resistance to biotic stress, regulating plant hormone pathways, and inducing root branching. In turn, pVOCs shape microbial communities by serving as carbon sources, acting as signalling or attractant molecules, inhibiting competing microbes, and facilitating microbial establishment. At the molecular level, VOC perception remains incompletely understood but is proposed to involve receptor-mediated sensing and downstream changes in gene expression in both plant and microbial cells.

Among the best-characterized mVOCs are 2,3-butanediol (**22**) and its precursor acetoin (**23**), commonly emitted by plant growth-promoting rhizosphere bacteria such as *Bacillus* and *Enterobacter* spp. These alcohols have been repeatedly linked to plant growth promotion and induced systemic resistance.[64] More specifically, plant exposure to the bacterial VOCs 2,3-butanediol (**22**) and acetoin (**23**) has been associated with transcriptional changes in auxin-responsive genes, modulation of auxin transport pathways, and increased lateral root formation and density.[64,65] In addition, these VOCs can affect cytokinin-associated signaling and growth responses, resulting in enhanced shoot growth and altered patterns of cell proliferation and development. Whether mVOCs directly mimic plant hormones or instead trigger endogenous hormone biosynthesis remains an active area of research, but transcriptome analyses of plants exposed to these mVOCs consistently reveal enrichment of auxin- and cytokinin-responsive pathways. Also other studies have shown that mVOC blends from growth-promoting bacteria and endophytic fungi can reconfigure signalling in the auxin, cytokinin, ethylene, abscisic acid (ABA), jasmonic acid (JA) and salicylic acid (SA) pathways, with consequences for both growth and defense.[58,63,66,67]

A recent study revealed that 2,3-butanediol (**22**) and acetoin (**23**) can also suppress storage root formation in the medicinal plant *Pseudostellaria heterophylla* while reshaping its rhizosphere microbiome especially the fungal communities.[68] 2,3-butanediol (**22**) and acetoin (**23**) also triggered reprogramming of root gene expression, including suppression of photosynthesis and secondary metabolic pathway[69]s. Both compounds down-regulated photosynthesis, cutin, suberin and wax biosynthesis, pathways that are crucial for plant carbon allocation, root barrier integrity and storage organ development. The diol 2,3-Butanediol **(22)** specifically suppressed brassinosteroid biosynthesis, carotenoid biosynthesis, linoleic acid metabolism and alkaloid biosynthesis, while acetoine specifically suppressed flavonoid biosynthesis and isoquinoline alkaloid biosynthesis.[68]

mVOC such as 2*R*,3*R*-butanediol can adjust stomatal behaviour and ABA-associated signaling, contributing to changes in transpiration and potentially in drought tolerance.[69] A recent study demonstrated that in *Arabidopsis* mVOCs emitted by *Pseudomonas* strains can reprogram root gene expression, suppressing ABA and sugar transport pathways while activating ABA-independent drought regulators, iron uptake genes, and glucosinolate-related pathways.[70] Metabolite changes (*e.g.*, altered glucosinolates, increased coumarins, modified sugar levels) and mutant analyses indicated that ABA and aliphatic glucosinolates negatively regulated the mVOC response. The mVOC blend which include, among others, dimethyl disulfide and hexanoic acid also

improved growth and water content of soil-grown *Brassica oleracea* under drought and reshaped the root microbiome toward a composition resembling well-watered plants. Hence, mVOCs can enhance drought resilience through coordinated changes in plant transcription, physiology, root architecture, and microbiome assembly.[70] A widely discussed group of mVOCs is the sulfur-containing mVOCs, in particular dimethyl disulfide (DMDS, **24**) and dimethyl trisulfide (DMTS, **25**). DMDS can influence *Arabidopsis* root system architecture *via* modulation of canonical auxin signaling, illustrating how a relatively simple molecule may intersect with core developmental pathways.[2] Recent community-scale volatilomics further suggests that sulfur-containing compounds (including DMDS) can emerge as dominant features of community mVOCs blend and correlate with plant growth responses, highlighting that the biologically relevant unit is often a volatile bouquet rather than a single mVOC.[71–73]

Nitrogen-containing mVOCs also contribute to plant–microbe and microbe–microbe interactions, although they have received less attention than alcohols and sulfur-containing volatiles. Among these compounds, pyrazines are emitted by diverse soil- and plant-associated bacteria, including species of *Bacillus*, *Pseudomonas*, and *Streptomyces*.[74–76] Several pyrazines exhibit antimicrobial activity against competing microorganisms and plant pathogens, suggesting a role in structuring rhizosphere microbial communities and contributing to disease-suppressive soils.[8,77,78] In addition to their direct inhibitory effects, pyrazines have been proposed to function as infochemicals that mediate interactions among microorganisms and influence the composition of community-level volatile blends.[8,78] These observations indicate that nitrogen-containing mVOCs may contribute to plant health not only through direct effects on plant physiology but also indirectly through modulation of microbial community dynamics.

How plants perceive mVOCs remains one of the most exciting open questions (Fig. 6). Some mVOCs likely act through passive diffusion across membranes followed by intracellular impacts on redox state, mitochondrial function, or membrane properties. For example, small microbial volatiles such as acetoin (**23**) and 2,3-butanediol (**22**) can readily enter plant tissues and trigger extensive transcriptional and physiological responses without the involvement of a dedicated receptor having yet been identified.[64,79] Recent work indicates that mVOC perception rapidly activates Ca^{2+} and K^{+} fluxes, reactive oxygen species (ROS) and nitric oxide (NO) signaling, and plasmodesmata-associated signaling pathways. These responses converge on hormone signaling networks, particularly auxin transport, enabling plants to adjust development and defense in response to microbial volatile cues.[80] In addition, VOC partitioning into lipid bilayers may alter membrane fluidity and ion transport processes, triggering downstream signaling events such as Ca^{2+} fluxes and MAPK activation.[81,82] However, increasing evidence suggests that at least some volatile responses may involve dedicated perception and signaling mechanisms rather than solely physicochemical effects. A striking example is the discovery that volatile communication in plants can rely on a α/β-hydrolase KARRIKIN INSENSITIVE 2 (KAI2)-mediated signaling pathway, a signaling protein best known for perceiving smoke-derived karrikins and endogenous karrikin-like molecules, which was recently shown to be required for plant responses to certain airborne volatiles. This demonstrates that plants possess genetically encoded modules for volatile perception and downstream signaling.[83] Although comparable receptors or signaling proteins for mVOCs have not yet been identified, these discoveries raise the possibility that microbial volatiles may also be perceived through dedicated sensing machinery. Such mechanisms would differ from physicochemical effects, in which volatile molecules influence cellular processes directly through their chemical properties, for example by partitioning into membranes or altering redox homeostasis, without requiring specific receptor-mediated recognition. Future studies will be needed to determine whether mVOC perception relies predominantly on physicochemical interactions, dedicated sensing components, or a combination of both mechanisms.

6. Experimental challenges and future perspectives

Both plants and microorganisms produce complex blends of pVOCs and mVOCs, including terpenoids, alcohols, ketones, sulfur-containing compounds, and nitrogenous compounds.[84] These compounds can act over a distance without requiring physical contact, thereby expanding the spatial scale of plant–microbe interactions. Despite rapid advances in analytical chemistry and molecular biology, major conceptual and technical challenges remain exist, particularly in understanding the ecological relevance, mechanistic basis, and practical application of volatile-mediated signalling. One of the primary challenges lies in the chemical complexity and context dependency of pVOC and mVOC blends. In natural ecosystems, organisms emit dynamic mixtures rather than single compounds, and the biological effects often depend on concentration, exposure duration, and combinatorial interactions. Low concentrations of mVOCs may prime plant immunity or promote growth, whereas higher doses can be inhibitory or phytotoxic. In natural soils, mVOCs rarely act in isolation; they interact with other metabolites and signalling molecules, creating complex feedback loops. mVOCs often act as modulators of plant signaling networks, influencing hormonal pathways such as auxin, cytokinin, ethylene, and jasmonic acid.[85] Hence, depending on the plant's physiological state and the presence of biotic stress (*e.g.*, pathogens or herbivores), the same volatile signal can shift the balance between growth and defense.

Different microbial species and even different strains of the same species may produce distinct mVOC blends depending on their genetic background and metabolic state. Nutrient availability, oxygen levels, pH, and interactions with other microbes can all shift the quantity and composition of emitted volatiles.[78] In microbial communities, interactions among community members can substantially alter mVOC emission profiles and thereby modify plant responses. Through processes such as competition, cooperation, or metabolic cross-feeding, microorganisms may enhance, suppress, or induce the production of

specific volatile compounds, resulting in volatile blends that differ from those emitted by individual strains grown in isolation. Consequently, plant responses are often shaped by the collective volatile profile of the microbial community rather than by the activity of a single microorganism.[86] Plant species and genotypes differ in their sensitivity to specific VOCs. Developmental stage is also crucial, seedlings often show stronger growth responses, while mature plants may respond with altered defence signalling. Therefore, the environmental context strongly shapes outcomes. Furthermore, abiotic factors such as temperature, moisture, and nutrient availability modify both microbial metabolism and plant responsiveness. Additionally, soil structure can influence VOC diffusion and concentration belowground and therefore their perception among receiver organisms.[87] Moreover, the mechanisms of VOC perception and signal transduction in plant and microbes are largely unknown. In plants, only a limited number of receptors or molecular targets for mVOCs have been identified. For example, the α/β-hydrolase KARRIKIN INSENSITIVE 2 (KAI2) has recently been implicated in the perception of the volatile sesquiterpene germacrene-D in petunia, providing evidence that plants can employ genetically encoded signaling components for volatile-mediated communication.[83] In addition, some volatile compounds are perceived after metabolic conversion into bioactive molecules, as illustrated by methyl salicylate, which is enzymatically converted into salicylic acid by SALICYLIC ACID-BINDING PROTEIN 2 (SABP2), thereby activating downstream defense signaling pathways.[88]

It is often unclear whether VOCs are perceived through dedicated receptors, passive diffusion and through metabolic incorporation, or *via* perturbations of redox or membrane states. Similarly, microbial responses to pVOCs are poorly characterized at the molecular level (Fig. 6). Elucidating the genetic and biochemical pathways underlying volatile perception and downstream signalling will be essential for predictive modelling and rational microbial applications in agriculture.

Another challenge concerns the experimental systems to study pVOCs and mVOCs. Much of the current knowledge derives from *in vitro* plate-based assays in which plants and microbes are physically separated but share headspace. While these systems have been instrumental in demonstrating proof-of-principle effects, they do not fully recapitulate the physical and chemical heterogeneity of soil and plant environments. Soil structure, moisture, pH, and organic matter content influence volatile diffusion, adsorption, and degradation. Additionally, microbial communities in natural soils are highly diverse, and interspecific interactions can modulate volatile production. Translating findings from simplified *in vitro* systems to complex rhizosphere, phyllosphere or anthosphere environments remains a significant hurdle. Standardization and reproducibility represent additional technical challenges. Variability in growth media, headspace volume, airflow, sampling methods, trapping materials and analytical platforms (*e.g.*, SPME-GC-MS vs. PTR-MS) can yield divergent volatile profiles. Community-wide adoption of standardized experimental and reporting guidelines will be essential to facilitate cross-study comparisons and meta-analyses. In this context, emerging resources such as the Global Natural Products Social Molecular Networking (GNPS) platform provide a valuable framework for the annotation and sharing of metabolomic and volatile datasets, enabling more comprehensive comparisons across studies.[89] However, important limitations remain, including incomplete spectral libraries, challenges in annotating unknown compounds, and constraints in metabolomic data analysis that hinder accurate compound identification and quantification.

Importantly, several VOCs including sulfur containing compounds, certain terpenes, and short-chain alcohols can be produced by both plants and microbes.[84] This complicates both the identification of VOCs and the attribution of their precise biological origin. Integration of high-resolution volatilomics with transcriptomics, proteomics, and metabolomics will therefore be needed to further strengthen causal inference.

In conclusion, volatile-mediated interactions are embedded within multi-trophic networks in natural environments. Plants and microbes emit and respond to VOCs, creating a chemically mediated communication web. The effects of pVOCs on microbes or mVOCs on plants emerge from dynamic interactions among VOC producers/receivers, and their surrounding environments. Such context dependency highlights the importance of studying pVOCs and mVOCs under ecologically realistic conditions, especially when considering their potential application in sustainable agriculture. Future research should therefore move beyond pairwise systems toward synthetic and natural communities and application of pVOCs and mVOCs for improving plant health and sustainable crop protection.

7. Author contributions

All authors conceived the idea for this review. MSR prepared the schematic figures, and JD designed the chemical drawings. MSR and PG wrote the initial draft of the manuscript, which was then critically revised by JSD and JMR. All authors made substantial contributions to the final version of the manuscript.

8. Conflicts of interest

The authors have no conflicts to declare.

9. Data availability

Data availability statement is not applicable.

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11. References

1 E. Pichersky, J. P. Noel and N. Dudareva, *Science*, 2006, **311**, 808–811.
2 L. Weisskopf, S. Schulz and P. Garbeva, *Nat. Rev. Microbiol.*, 2021, **19**, 391–404.

3 T. C. J. Turlings, J. H. Tumlinson and W. J. Lewis, *Science*, 1990, **250**, 1251–1253.
4 R. Sohrabi, B. C. Paasch, J. A. Liber and S. Y. He, *Annu. Rev. Plant Biol.*, 2023, **74**, 539–568.
5 M. Hanusch, S. Dötterl, A.-A. C. Larue-Kontić, A. Keller and R. R. Junker, *New Phytol.*, 2025, **248**, 3270–3279.
6 O. S. Kulkarni, M. Mazumder, S. Kini, E. D. Hill, J. S. B. Aow, S. M. L. Phua, U. Elejalde, S. Kjelleberg and S. Swarup, *Nat. Chem. Biol.*, 2024, **20**, 473–483.
7 R. R. Junker and D. Tholl, *J. Chem. Ecol.*, 2013, **39**, 810–825.
8 R. Schmidt, V. Cordovez, W. de Boer, J. Raaijmakers and P. Garbeva, *ISME J.*, 2015, **9**, 2329–2335.
9 S. A. Rolfe, J. Griffiths and J. Ton, *Curr. Opin. Microbiol.*, 2019, **49**, 73–82.
10 M. S. Rizaludin, N. Stopnisek, J. M. Raaijmakers and P. Garbeva, *Metabolites*, 2021, **11**(6), DOI: 10.3390/metabo11060357.
11 K. Schulz-Bohm, S. Gerards, M. Hundscheid, J. Melenhorst, W. de Boer and P. Garbeva, *ISME J.*, 2018, **12**, 1252–1262.
12 N. M. van Dam, A. Weinhold and P. Garbeva, in *Deciphering Chemical Language of Plant Communication*, ed. J. D. Blande and R. Glinwood, Springer International Publishing, Cham, 2016, pp. 175–210.
13 R. Escobar-Bravo, P.-A. Lin, J. M. Waterman and M. Erb, *Nat. Prod. Rep.*, 2023, **40**, 840–865.
14 N. Dudareva, A. Klempien, J. K. Muhlemann and I. Kaplan, *New Phytol.*, 2013, **198**, 16–32.
15 S. Schulz and J. S. Dickschat, *Nat. Prod. Rep.*, 2007, **24**, 814–842.
16 F. Zhou and E. Pichersky, *Curr. Opin. Plant Biol.*, 2020, **55**, 1–10.
17 J. S. Dickschat, *Nat. Prod. Rep.*, 2011, **28**, 1917–1936.
18 M. Rohmer, M. Seemann, S. Horbach, S. Bringer-Meyer and H. Sahm, *J. Am. Chem. Soc.*, 1996, **118**, 2564–2566.
19 F. M. Hahn, A. P. Hurlburt and C. D. Poulter, *J. Bacteriol.*, 1999, **181**, 4499–4504.
20 F. Lynen, H. Eggerer, U. Henning and I. Kessel, *Angew. Chem.*, 1958, **70**, 738–742.
21 J. S. Dickschat, *Nat. Prod. Rep.*, 2015, **33**, 87–110.
22 T. D. Sharkey, M. Bellucci, F. Loreto, M. G. Mostofa, A. Sahu, B. M. L. Serda, S. M. Weraduwage and Y. Xu, *J. Plant Physiol.*, 2026, **318**, 154712.
23 J. Byron, J. Kreuzwieser, G. Purser, J. van Haren, S. N. Ladd, L. K. Meredith, C. Werner and J. Williams, *Nature*, 2022, **609**, 307–312.
24 M. Lv, L. Zhang, Y. Wang, L. Ma, Y. Yang, X. Zhou, L. Wang, X. Yu and S. Li, *Hortic. Res.*, 2024, **11**, uhae220.
25 T. Vogt, *Mol. Plant*, 2010, **3**, 2–20.
26 J. R. Widhalm and N. Dudareva, *Mol. Plant*, 2015, **8**, 83–97.
27 S. D. Cook, *Plant Cell Physiol.*, 2019, **60**, 243–254.
28 K. Matsui, *J. Exp. Bot.*, 2026, **77**, 3267–3280.
29 J. Čivić, I. Tuñón and J. N. Harvey, *ACS Catal.*, 2025, **15**, 1684–1692.
30 I. L. Pirtle, W. Kongcharoensuntorn, M. Nampaisansuk, J. E. Knesek, K. D. Chapman and R. M. Pirtle, *Biochim. Biophys. Acta Gene Struct. Expr.*, 2001, **1522**, 122–129.
31 Y. L. Peng, Y. Shirano, H. Ohta, T. Hibino, K. Tanaka and D. Shibata, *J. Biol. Chem.*, 1994, **269**, 3755–3761.
32 J. Sekiya, T. Kajiwara and A. Hatanaka, *Agric. Biol. Chem.*, 1979, **43**, 969–980.
33 Cambrex, *Partial purification and properties of a hydroperoxide lyase from fruits of pear*, https://pubs.acs.org/doi/pdf/10.1021/jf00108a030, accessed April 1, 2026.
34 T. Hirao, A. Okazawa, K. Harada, A. Kobayashi, T. Muranaka and K. Hirata, *J. Biosci. Bioeng.*, 2012, **114**, 540–545.
35 A. Clavijo McCormick, S. B. Unsicker and J. Gershenzon, *Trends Plant Sci.*, 2012, **17**, 303–310.
36 A. Maruyama-Nakashita, *Curr. Opin. Plant Biol.*, 2017, **39**, 144–151.
37 J. S. Dickschat, S. Wickel, C. J. Bolten, T. Nawrath, S. Schulz and C. Wittmann, *Eur. J. Org Chem.*, 2010, **2010**, 2687–2695.
38 T. Nawrath, J. S. Dickschat, B. Kunze and S. Schulz, *Chem. Biodiversity*, 2010, **7**, 2129–2144.
39 C. Sá, D. Matos, A. Pires, P. Cardoso and E. Figueira, *Sci. Total Environ.*, 2021, **800**, 149478.
40 Y. J. Wong, Y. Luo, X. J. Loh and X. Chen, *Anal. Sens.*, 2025, **5**, e202500037.
41 D. Abanda-Nkpwatt, M. Müsch, J. Tschiersch, M. Boettner and W. Schwab, *J. Exp. Bot.*, 2006, **57**, 4025–4032.
42 X. Mo, D. Cai, H. Yang, Q. Chen, C. Xu, J. Wang, Z. Tong and B. Xu, *Food Chem.*, 2025, **27**, 102377.
43 O. Carrión, L. Gibson, D. M. O. Elias, N. P. McNamara, T. A. van Alen, H. J. M. Op den Camp, C. V. Supramaniam, T. J. McGenity and J. C. Murrell, *Microbiome*, 2020, **8**, 81.
44 D. Guo, Z. Liu, J. M. Raaijmakers, Y. Xu, J. Yang, M. Erb, J. Zhang, Y.-G. Zhu, J. Xu and L. Hu, *Science*, 2025, **389**, eadv6675.
45 N. Wei, R. L. Whyle, T.-L. Ashman and M. A. Jamieson, *Hortic. Res.*, 2022, **9**, uhab005.
46 F. Chen, D.-K. Ro, J. Petri, J. Gershenzon, J. Bohlmann, E. Pichersky and D. Tholl, *Plant Physiol.*, 2004, **135**, 1956–1966.
47 M. Yahyaa, D. Tholl, G. Cormier, R. Jensen, P. W. Simon and M. Ibdah, *J. Agric. Food Chem.*, 2015, **63**, 4870–4878.
48 A. S. L. Díaz, M. S. Rizaludin, H. Zweers, J. M. Raaijmakers and P. Garbeva, *Molecules*, 2022, **27**, DOI: 10.3390/molecules27051612.
49 M. S. Rizaludin, A. S. L. Díaz, H. Zweers, J. M. Raaijmakers and P. Garbeva, *FEMS Microbiol. Ecol.*, 2025, **101**, fiaf042.
50 M.-Y. Chou, T. B. Andersen, M. E. Mechan Llontop, N. Beculheimer, A. Sow, N. Moreno, A. Shade, B. Hamberger and G. Bonito, *Microbiol. Spectr.*, 2023, **11**, e01332–23.
51 L. Hu, *New Phytol.*, 2022, **233**, 618–623.
52 K. G. S. Dani and F. Loreto, *New Phytol.*, 2022, **234**, 804–812.
53 G. Farré-Armengol, I. Filella, J. Llusia and J. Peñuelas, *Trends Plant Sci.*, 2016, **21**, 854–860.
54 J. Jin, M. Zhao, T. Jing, M. Zhang, M. Lu, G. Yu, J. Wang, D. Guo, Y. Pan, T. D. Hoffmann, W. Schwab and C. Song, *Hortic. Res.*, 2023, **10**, uhad143.
55 L. Hu, K. Zhang, Y. Xu, X. Zheng, J. M. Waterman, X. Ouyang, Z. Wu, Z. Shen, Y. He, B. Ma, C. A. M. Robert,

J. M. Raaijmakers, M. Ye, M. Erb and J. Xu, *Nat. Plants*, 2025, **11**, 1001–1017.
56 M. Erb, N. Veyrat, C. A. M. Robert, H. Xu, M. Frey, J. Ton and T. C. J. Turlings, *Nat. Commun.*, 2015, **6**, 6273.
57 H. Liu and L. E. Brettell, *Trends Plant Sci.*, 2019, **24**, 187–189.
58 H. G. Kong, G. C. Song, H.-J. Sim and C.-M. Ryu, *ISME J.*, 2021, **15**, 397–408.
59 R. Sharifi, S.-M. Lee and C.-M. Ryu, *New Phytol.*, 2018, **220**, 684–691.
60 C. M. J. Pieterse, C. Zamioudis, R. L. Berendsen, D. M. Weller, S. C. M. V. Wees and P. A. H. M. Bakker, *Annu. Rev. Phytopathol.*, 2014, **52**, 347–375.
61 Y. Chen, R. A. Wilschut and P. Garbeva, *Trends Microbiol.*, 2026, **34**(7), 723–734.
62 Y. Chen, M. Bonkowski, Y. Shen, B. S. Griffiths, Y. Jiang, X. Wang and B. Sun, *Microbiome*, 2020, **8**, 4.
63 R. Sharifi, J.-S. Jeon and C.-M. Ryu, *J. Exp. Bot.*, 2022, **73**, 463–486.
64 C.-M. Ryu, M. A. Farag, C.-H. Hu, M. S. Reddy, H.-X. Wei, P. W. Paré and J. W. Kloepper, *Proc. Natl. Acad. Sci. U. S. A.*, 2003, **100**, 4927–4932.
65 A. Bailly, U. Groenhagen, S. Schulz, M. Geisler, L. Eberl and L. Weisskopf, *Plant J.*, 2014, **80**, 758–771.
66 S. Tyagi, K. Kim, M. Cho and K. J. Lee, *Environ. Sustain.*, 2019, **2**, 211–216.
67 A. Garnica-Vergara, S. Barrera-Ortiz, E. Muñoz-Parra, J. Raya-González, A. Méndez-Bravo, L. Macías-Rodríguez, L. F. Ruiz-Herrera and J. López-Bucio, *New Phytol.*, 2016, **209**, 1496–1512.
68 Y. Yang, C. Xu, D. Lin, C. Zheng, X. Dai, Z. Zheng, N. Wang, B. Hu, L. Xia, X. Qian and L. Zhang, *Biology*, 2026, **15**, DOI: 10.3390/biology15050403.
69 S. M. Cho, B. R. Kang, S. H. Han, A. J. Anderson, J.-Y. Park, Y.-H. Lee, B. H. Cho, K.-Y. Yang, C.-M. Ryu and Y. C. Kim, *Mol. Plant-Microbe Interact.*, 2008, **21**, 1067–1075.
70 Z. Carracedo Lorenzo, M. S. Rizaludin, J. Wang, R. Berdaguer, C. Brito-López, C. Sánchez-Arcos, P. Garbeva, C. M. J. Pieterse, M. Dicke, C. Testerink, K. J. Kloth and R. Karlova, *New Phytol.*, 2026, **251**, 405–423.
71 B. Piechulla, M. C. Lemfack and M. Kai, *Plant Cell Environ.*, 2017, **40**, 2042–2067.
72 G. M. Türksoy, M. Berka, K. Wippel, A. Koprivova, R. A. Carron, L. Rüger, M. Černý, T. G. Andersen and S. Kopriva, *Plant Commun.*, 2025, **6**, 101351.
73 J. F. Jiménez-Bremont, E. González-Pérez, M. A. Ortega-Amaro, S. Madrigal-Ortiz, A. Duque-Ortiz and A. Mendoza-Mendoza, *Sci. Hortic.*, 2024, **325**, 112656.
74 T. K. S. Janssens, O. Tyc, H. Besselink, W. de Boer and P. Garbeva, *FEMS Microbiol. Lett.*, 2019, **366**, fnz023.
75 K. Lee, S.-H. Kwon, S. Song, D.-Y. Lee, M. K. Park and Y.-S. Kim, *Metabolites*, 2023, **13**, DOI: 10.3390/metabo13020219.
76 Y. Gong, J.-Q. Liu, M.-J. Xu, C.-M. Zhang, J. Gao, C.-G. Li, K. Xing and S. Qin, *Appl. Environ. Microbiol.*, 2022, **88**, e02317–e02321.
77 P. Garbeva and L. Weisskopf, *New Phytol.*, 2020, **226**, 32–43.
78 K. Schulz-Bohm, L. Martín-Sánchez and P. Garbeva, *Front. Microbiol.*, 2017, **8**, DOI: 10.3389/fmicb.2017.02484.
79 P. Fincheira and A. Quiroz, *Microbiol. Res.*, 2018, **208**, 63–75.
80 C. N. Kanchiswamy, I. A. Paponov, M. Malnoy, F. Caldo, S. Roychoudhry and M. E. Maffei, *Plant Stress*, 2026, 101438.
81 G. Arimura and T. Uemura, *Trends Plant Sci.*, 2025, **30**, 105–115.
82 F. Loreto and S. D'Auria, *Trends Plant Sci.*, 2022, **27**, 29–38.
83 S. A. Stirling, A. M. Guercio, R. M. Patrick, X.-Q. Huang, M. E. Bergman, V. Dwivedi, R. W. J. Kortbeek, Y.-K. Liu, F. Sun, W. A. Tao, Y. Li, B. Boachon, N. Shabek and N. Dudareva, *Science*, 2024, **383**, 1318–1325.
84 D. Schenkel, M. C. Lemfack, B. Piechulla and R. Splivallo, *Front. Plant Sci.*, 2015, **6**, DOI: 10.3389/fpls.2015.00707.
85 N. Kanjana, P. Guo, Z. Deng, M. A. Ahmed, I. Shah and L. Zhang, *Curr. Plant Biol.*, 2026, **45**, 100584.
86 G. M. Türksoy, M. Berka, K. Wippel, A. Koprivova, R. A. Carron, L. Rüger, M. Černý, T. G. Andersen and S. Kopriva, *Plant Commun.*, 2025, DOI: 10.1016/j.xplc.2025.101351.
87 S. Som, D. S. Willett and H. T. Alborn, *Rhizosphere*, 2017, **4**, 70–74.
88 M. E. Bergman, X.-Q. Huang, S. Baudino, J.-C. Caissard and N. Dudareva, *Curr. Opin. Plant Biol.*, 2025, **85**, 102706.
89 M. Wang, J. J. Carver, V. V. Phelan, L. M. Sanchez, N. Garg, Y. Peng, D. D. Nguyen, J. Watrous, C. A. Kapono, T. Luzzatto-Knaan, C. Porto, A. Bouslimani, A. V. Melnik, M. J. Meehan, W.-T. Liu, M. Crüsemann, P. D. Boudreau, E. Esquenazi, M. Sandoval-Calderón, R. D. Kersten, L. A. Pace, R. A. Quinn, K. R. Duncan, C.-C. Hsu, D. J. Floros, R. G. Gavilan, K. Kleigrewe, T. Northen, R. J. Dutton, D. Parrot, E. E. Carlson, B. Aigle, C. F. Michelsen, L. Jelsbak, C. Sohlenkamp, P. Pevzner, A. Edlund, J. McLean, J. Piel, B. T. Murphy, L. Gerwick, C.-C. Liaw, Y.-L. Yang, H.-U. Humpf, M. Maansson, R. A. Keyzers, A. C. Sims, A. R. Johnson, A. M. Sidebottom, B. E. Sedio, A. Klitgaard, C. B. Larson, C. A. Boya P, D. Torres-Mendoza, D. J. Gonzalez, D. B. Silva, L. M. Marques, D. P. Demarque, E. Pociute, E. C. O'Neill, E. Briand, E. J. N. Helfrich, E. A. Granatosky, E. Glukhov, F. Ryffel, H. Houson, H. Mohimani, J. J. Kharbush, Y. Zeng, J. A. Vorholt, K. L. Kurita, P. Charusanti, K. L. McPhail, K. F. Nielsen, L. Vuong, M. Elfeki, M. F. Traxler, N. Engene, N. Koyama, O. B. Vining, R. Baric, R. R. Silva, S. J. Mascuch, S. Tomasi, S. Jenkins, V. Macherla, T. Hoffman, V. Agarwal, P. G. Williams, J. Dai, R. Neupane, J. Gurr, A. M. C. Rodríguez, A. Lamsa, C. Zhang, K. Dorrestein, B. M. Duggan, J. Almaliti, P.-M. Allard, P. Phapale, L.-F. Nothias, T. Alexandrov, M. Litaudon, J.-L. Wolfender, J. E. Kyle, T. O. Metz, T. Peryea, D.-T. Nguyen, D. VanLeer, P. Shinn, A. Jadhav, R. Müller, K. M. Waters, W. Shi, X. Liu, L. Zhang, R. Knight, P. R. Jensen, B. Ø. Palsson, K. Pogliano, R. G. Linington, M. Gutiérrez, N. P. Lopes, W. H. Gerwick, B. S. Moore, P. C. Dorrestein and N. Bandeira, *Nat. Biotechnol.*, 2016, **34**, 828–837.

