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PLANT-MICROBE-INSECT INTERACTIONS

Ecological and phytohormonal aspects of plant volatile emission in response to single and dual infestations with herbivores and phytopathogens

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Summary

1. In their natural environment, plants are faced with a multitude of attackers, of which insect herbivores and plant pathogens are an important component. In response to these attacks, plants release volatile organic compounds (VOCs), which play an important role in the communication between plants and the associated community members, such as other herbivores, phytopathogens and the natural enemies of herbivores.

2. While numerous studies have focused on either plant–pathogen or plant–insect interactions, less is known when these two sets of interactions co-occur. Depending on the mode of attack of the pathogen (necrotroph vs. biotroph) or herbivore (chewing vs. piercing-sucking) they will activate different defence pathways in the plant in which the phytohormones salicylic acid (SA), jasmonic acid (JA) and ethylene (ET) play key roles. As these pathways can crosstalk, a pathogen infection can interfere in a plant's defence response to herbivory, and vice versa.

3. Infestation of a plant with organisms inducing SA signalling prior to – or simultaneously with – attack by organisms that induce the JA pathway often suppresses JA signalling. However, the impact of this signalling pathway crosstalk on VOC induction is not clear cut, as there is high variability in the effects on volatile emissions, ranging from suppression to enhanced emission. The effects of the modified volatile blends on the foraging success of carnivorous natural enemies of herbivorous insects have started to be investigated. Foraging success of natural enemies generally withstands this modification of the host-induced VOC blend, but the presence or absence of key compounds is an important determinant of the response of certain carnivores.

4. Further studies incorporating plant–insect and plant–pathogen interactions at different levels of biological integration will provide valuable insight in how plants integrate signals from different suites of attacking organisms into an adaptive defence response.

Key-words: natural enemies, phytohormones, plant volatiles, signal transduction, species interactions, tripartite interactions, tritrophic interactions

Introduction

In nature, plants are members of complex communities, and they interact with a wide range of organisms, both beneficial and deleterious. Among these are a plethora of attackers, including herbivorous arthropods and plant pathogens. Half of the estimated 6 million insect species are herbivorous (Schoonhoven, Van Loon & Dicke 2005) and while the diversity of plant pathogenic microbes has not been quantified, they are an equally major threat to plants (Strange & Scott 2005). Faced with this multitude of enemies, it should come as no surprise that plants have evolved sophisticated defence strategies that allow them to recognize herbivores or pathogens (Mithöfer & Boland 2008) and then implement an often tailor-made defence

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response. When a plant is under attack, a wide range of responses are initiated, including physical and chemical defences (Walters 2011). The latter of these include the production of secondary metabolites (Iason, Dicke & Hartley 2012) of which the emission of volatile organic compounds (VOCs) are an important component (Heil 2008; Dicke & Baldwin 2010; Hare 2011).

Volatile organic compounds are highly diverse and are of strong ecological importance as they contribute to shaping the assemblage of, and interactions between, the organisms within a plant's community (Poelman, van Loon & Dicke 2008b). VOCs induced by herbivory attract natural enemies of herbivores, which may confer protection to the plant (Kessler & Baldwin 2001; Dicke & Baldwin 2010). Induced volatiles can have repellent effects on herbivores (Delphia, Mescher & De Moraes 2007; Bleeker *et al.* 2009) and inhibit pathogen colonization (Brown *et al.* 1995). In addition, they are involved in information transfer between and within plants, through the airborne transport of signals that lead to the priming or expression of defences in neighbouring plants or in distal parts of the emitting plant (Engelberth *et al.* 2004; Kessler *et al.* 2006; Frost *et al.* 2008). These VOCs, however, can be a double-edged sword as they can also be used by herbivores as host-plant location cues (Bolter *et al.* 1997). For instance, in *Nicotiana attenuata*, the same volatile chemical signals are exploited by both herbivores and carnivores for host location (Halitschke *et al.* 2008) underlining the complexity in the interactions mediated by VOCs (Dicke & Baldwin 2010).

While induced VOCs are receiving increasing attention, especially their role in mediating tritrophic interactions, the study of VOCs in a multiple attack situation, notably with pathogens and herbivores, has been largely unexplored. In nature, plants are often confronted with simultaneous or sequential attack, yet even until recently research was largely conducted on study systems comprising of single plant-attacker combinations (Dicke, van Loon & Soler 2009). Rapid advances in our knowledge on the underlying mechanisms of plant defences have shown that the interactions under a multiple attack scenario are complex (Pieterse *et al.* 2012). At least three phytohormones, that is, salicylic acid (SA), jasmonic acid (JA) and ethylene (ET), play key regulatory roles in the interconnecting signal-transduction pathways that mediate induced defences in response to herbivore and pathogen infestation. Crosstalk between these pathways can mould the final defence response, including VOC production, when challenged by different attacker species. Induced responses of plants to pathogens and herbivores were long treated as two separate fields, and the two areas of study have evolved largely independently of one another until quite recently (Pieterse & Dicke 2007). In this review, we will give an overview of (i) the existing literature on crosstalk between signalling pathways affected by insect herbivore and pathogen attack, (ii) the effect of pathway crosstalk on plant VOC emission, and (iii) the effects of these emissions on community members at higher trophic levels. Finally, we will provide an outlook to the future by focussing on the integration of studies on plant–pathogen and plant–insect interactions.

Signal-transduction pathways regulating induced plant defences

At the crux of a plant's interactions with attacking pathogens and insect herbivores lays a complex interconnecting signalling network regulated by phytohormones. These are crucial not only for regulating plant growth, development and reproduction, but also for induced defences, including the production of VOCs. It has been well established that SA, JA and ET play pivotal roles in the regulation of the signal-transduction pathways that lead to the activation of different sets of defence-related genes (von Dahl & Baldwin 2007; Howe & Jander 2008; Robert-Seilaniantz, Grant & Jones 2011; Pieterse *et al.* 2012). While the common division is that pathogens induce SA and herbivores activate JA-mediated defences, the reality is much more complex. For example, SA is generally thought to mediate defences against piercing-sucking insects and pathogens that are biotrophic for all or part of their lifecycle, while JA/ET induction is usually associated with chewing insect herbivores and necrotrophic pathogens (Glazebrook 2005; Pieterse & Dicke 2007; Spoel, Johnson & Dong 2007). Moreover, defences against herbivores and necrotrophs are seemingly regulated by two distinct and antagonistic branches of the JA signalling pathway (Kazan & Manners 2008; Verhage *et al.* 2011) (Fig. 1). In addition, plant-attacker combinations of organisms inducing the same general pathways generate different dynamics of SA, JA and ET production and, consequently, different transcriptomic responses (De Vos *et al.* 2005). This unique 'signal signature' induced by an attack results in major differences in the expression levels and timing of the different gene sets, contributing to the specificity of a plant's induced response which is likely to have important consequences for the volatile blends induced by the signal-transduction pathways.

In nature, plants often face multiple attackers and they need to be able to adapt to their ever-changing environment. There is increasing evidence that signalling pathways cross-communicate with each other, and it is clear that this crosstalk is a powerful regulatory mechanism that allows plants to fine-tune their final defence response in reaction to pathogen or herbivore attack (Koornneef & Pieterse 2008; Grant & Jones 2009; Verhage, van Wees & Pieterse 2010). The outcome of crosstalk between different signal-transduction pathways can be antagonistic or synergistic and the interaction between the SA and JA signal-transduction pathways is one of the best studied examples, with much evidence that they are often mutually antagonistic (Stout, Thaler & Thomma 2006; Koornneef & Pieterse 2008; Koornneef *et al.* 2008). SA has an especially strong effect, in that its accumulation inhibits JA biosynthesis and signalling when it is induced prior to or concomitantly

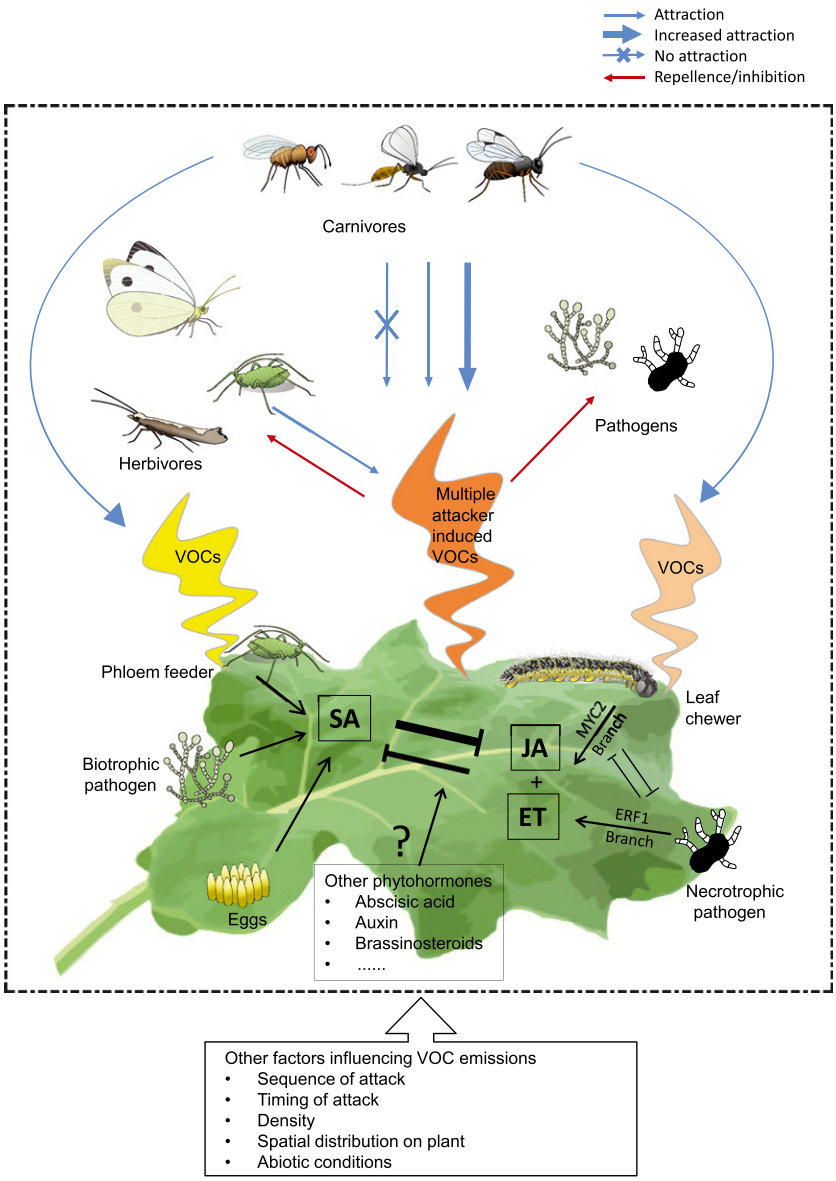

Fig. 1. Overview of the effects of single attack and multiple attacks of plants on volatile organic compound (VOC) emission, including subcellular mechanisms such as phytohormone-mediated signal transduction and effects on interactions with microbial and insect species.

with JA. In Arabidopsis, infection by the biotroph *Pseudomonas syringae* leads to greater susceptibility to the nectrotroph *Alternaria brassicicola*, through impairment of JA-mediated defences (Spoel, Johnson & Dong 2007). ET is involved in crosstalk with both of these pathways, as it acts synergistically with JA in activating defence-related genes (Penninckx *et al.* 1998) and can heighten a plant's sensitivity to SA, leading to enhanced SA-mediated defences (De Vos *et al.* 2006). Other phytohormones, such as abscisic acid, auxins, cytokinins, brassinosteroids and gibberellins appear to play a much larger role in shaping defence-related signalling than previously thought, although for some the exact mechanisms of their involvement in the backbone SA-JA-ET network still needs to be further explored (Erb *et al.* 2011a; Robert-Seilaniantz, Grant & Jones 2011; Giron *et al.* 2013; Pieterse *et al.* 2012). In the context of multiple attackers, prioritization of one pathway over another can have strong consequences in terms of defence expression. Order of attack becomes important as the initial attacker may compromise a plant's induced responses to secondary challengers, driving community-wide effects (Bruinsma & Dicke 2008; Poelman, van Loon & Dicke 2008b). At the molecular level, it has also been demonstrated that timing of induction is a major factor determining JA and SA signalling (Thaler, Fidantsef & Bostock 2002; Koornneef *et al.* 2008).

Interestingly, dose-dependent effects have been suggested to influence JA-SA crosstalk. Low concentrations of both exogenous SA and JA enhance the expression of defence-related genes, leading to a synergistic effect. However, at higher concentrations or prolonged treatment duration this effect disappears, indicating that there may be threshold levels for defence trade-offs (Mur *et al.* 2006; Kazan & Manners 2008). Recent studies show an important modulatory role of ET in crosstalk in that when JA/ET is induced first, the ET burst renders JA-dependent defences insensitive to SA-mediated suppression (Diezel *et al.* 2009; Leon-Reyes *et al.* 2010). Herbivores and pathogens have

evolved ways of manipulating hormonal crosstalk to their own advantage, and so can change the outcome of defence. A well-known example is *P. syringae* that produces coronatine, a JA analogue that suppresses SA-dependent defences, rendering plants more susceptible to the pathogen (Nomura, Melotto & He 2005). More recently, similar hijacking mechanisms were found for a necrotrophic fungus, *Botrytis cinerea* (El Oirdi *et al.* 2011). Similar decoy tactics exist among insects, such as in the case of *Bemisia tabaci* nymphs that promote their own development as well as spider mite reproduction by down-regulating JA defences via SA induction (Zarate, Kempema & Walling 2007; Zhang *et al.* 2009). The spider mite *Tetranychus evansi* takes this one step further by not only preventing defence induction, but also supressing the constitutive plant defences, which enhances the performance of conspecifics (Sarmento *et al.* 2011).

Single-species herbivory

When a plant is attacked by an herbivore, this induces the emission of a specific blend of volatile compounds, known as herbivore-induced plant volatiles (HIPV) (Fig. 1). HIPV are complex blends that can be composed of up to several hundred individual compounds (Pichersky & Gershenzon 2002; Dudareva *et al.* 2006; Pichersky, Noel & Dudareva 2006). These volatile blends are generally dominated by two major classes of compounds: terpenoids and fatty acid –derived green leaf volatiles (GLV) (Arimura, Matsui & Takabayashi 2009; Mumm & Dicke 2010). Some compounds, primarily GLV, are mainly released immediately upon wounding while others, including terpenoids, are synthesized 'de novo' and released from several hours up to several days after attack (Paré & Tumlinson 1997; Turlings *et al.* 1998). Many HIPV are not induced by mechanically damaging tissues alone, and in many cases they will be emitted both locally from damaged leaves and systemically from undamaged plant tissues. Some compounds may already be produced by undamaged plants and are simply emitted in greater quantities or different ratios after herbivory (Holopainen 2004). HIPV blends can vary quantitatively and qualitatively, depending on the plant species (Takabayashi, Dicke & Posthumus 1991; Van Poecke & Dicke 2004), plant genotype (Degen *et al.* 2004; Kappers *et al.* 2011), herbivore species (De Moraes *et al.* 1998) and even the developmental stage of herbivores (Takabayashi *et al.* 1995).

Disparities in volatile responses to different herbivore species can be in part explained by differences in the damage inflicted by insects of contrasting feeding guilds, due to the distinctive signalling pathways that they induce (Leitner, Boland & Mithöfer 2005). However, as even insects of the same guild can induce different HIPV blends, variation in volatile profiles can also be attributed to the composition of elicitors present in oral secretions of herbivores (Halitschke *et al.* 2003; Tumlinson & Engelberth 2008). Application of oral secretions, or of elicitors present in herbivore regurgitant, to wounded leaf tissue can mimic the effects of herbivory and activate the signal-transduction pathways, leading to the biosynthesis and emission of HIPV (Turlings, Tumlinson & Lewis 1990; Mattiacci, Dicke & Posthumus 1995; Alborn *et al.* 1997; Bonaventure, VanDoorn & Baldwin 2011).

Further complicating matters is that larval feeding is usually preceded by oviposition, which is a fact that has been long neglected in studies on HIPV. Insect egg deposition can also lead to the induction of volatile emission, which then mediates the attraction of egg parasitoids (Hilker *et al.* 2002; Mumm *et al.* 2003; Fatouros *et al.* 2008; Tamiru *et al.* 2011). In the pine and elm systems, elicitation of volatiles attractive to the parasitoids was shown to be a response to compounds in the oviduct secretions coating the eggs, which then come in contact with leaf tissue during oviposition (Meiners & Hilker 1997; Hilker *et al.* 2002). In other cases, egg parasitoids respond to volatiles that are only induced by the combination of both feeding damage and oviposition, as oviposition or feeding alone were not attractive to parasitoids (Colazza, McElfresh & Millar 2004b; Colazza *et al.* 2004a). The presence of both feeding damage and eggs can also have a synergistic effect on the emissions of certain compounds (Conti *et al.* 2008). However, Bruessow *et al.* (2010) found, unexpectedly, that egg deposition by *Pieris brassicae* butterflies leads to the local accumulation of SA, which then suppresses expression of caterpillar-induced JA-dependent defence genes. This crosstalk can have important consequences for plant defence, and there is evidence that HIPV emissions normally induced by subsequent caterpillar herbivory can be suppressed (Peñaflor *et al.* 2011). From a mechanistic perspective, looking at different stages of the plant-herbivore interaction independently is a suitable approach. However, these studies demonstrate that it is imperative to investigate the sequence of events as they occur in nature, with eggs first, to have an accurate overview from an ecological point of view.

Multiple herbivory

There are an increasing number of studies that investigate the dynamics of plant–insect interactions under multiple attacks. However, many of these focus primarily on how direct defences mediate interactions between herbivores (Rodriguez-Saona *et al.* 2005; Kaplan & Denno 2007). Knowing that there is potential for crosstalk between the SA and JA signalling pathways in particular, it is likely that interactions between attackers of different feeding guilds can also affect induction of indirect plant defences. Attack by an SA-pathway-inducing herbivore can be expected to modify or attenuate the volatile response towards a subsequent JA-pathway-inducing herbivore, and vice versa.

What is clear from the literature to date is that the effect of dual herbivory on HIPV emissions is difficult to predict and highly variable. Considering feeding guilds alone is

not enough to make predictions, as the same combination of herbivore species may have drastically diverging effects on ensuing VOC emissions in different plant species (de Boer *et al*. 2008). Multiple herbivory by insects of different feeding guilds can result in a few specific compounds being emitted in significantly higher or lower amounts than by attack by one herbivore only, with an overall volatile profile similar to what is induced in singly damaged plants (Rodriguez-Saona, Crafts-Brandner & Cañas 2003; Delphia, Mescher & De Moraes 2007; Zhang *et al*. 2009). In other instances, total emission rates are affected. Dual herbivory involving different feeding guilds can lead to the majority of compounds being emitted in greater amounts than during single-species herbivory (Moayeri *et al*. 2007; Hare & Sun 2011) and HIPV emissions may also increase to levels higher than the expected additive effect of the two herbivores, indicating a synergistic effect on induction (de Boer *et al*. 2008). Total emissions can also be suppressed, as was demonstrated in cotton plants infested by piercing silverleaf whiteflies (*B. tabaci*) and chewing *Spodoptera exigua* caterpillars, where HIPV emissions in response to dual herbivory were reduced by 60% in comparison with caterpillar feeding alone (Rodriguez-Saona, Crafts-Brandner & Cañas 2003). Finally, in still other cases, damage by a phloem feeder did not lead to any measurable effects on HIPV emissions induced by caterpillars (Erb, Foresti & Turlings 2010). Moreover, changes in HIPV emissions are not limited to those brought about by two folivorous herbivores, but also by combinations of shoot and root herbivory, which can also lead to altered patterns of emission in one or both spheres (Rasmann & Turlings 2007; Soler *et al*. 2007; Pierre *et al*. 2011).

One trend that emerges from the literature is that when an herbivore, usually a phloem feeder, induces low HIPV emissions, volatile emission elicited by a co-attacking chewing herbivore can be negatively affected (Rodriguez-Saona *et al*. 2005; Zhang *et al*. 2009; Schwartzberg, Böröczky & Tumlinson 2011). Although not a phloem feeder, maggots of the gall-inducing tephritid fly, *Eurosta solidaginis* attacking *Solidago altissima*, had similar effects, in that they dampened total HIPV production normally induced by *Heliothis virescens* caterpillars. Interestingly, two other herbivores that did not induce HIPV, that is, *Gnorimoschema gallaesolidaginis* (galling moth) and *Philaenus spumarius* (piercing-sucking insect), failed to have the same suppressive effect on *H. virescens*-induced volatiles. However, researchers were unable to clearly establish the role of the primary defence-related phytohormones, in part due to high variability within the data set (Tooker *et al*. 2008).

Of all the multiple herbivory studies specifically addressing HIPV, few have simultaneously investigated the phytohormonal basis for the observed changes in HIPV production. Zhang *et al*. (2009) observed that whitefly (*B. tabaci*) infestation negatively affected the emission of a spider mite (*Tetranychus urticae*)-induced monoterpene and this interference was positively damage dependent. JA levels in dually infested leaves were significantly lower compared to levels in leaves infested with *T. urticae* only, and two JA-regulated genes involved in induced defence signalling and the terpenoid's biosynthesis showed reduced expression levels. Application of exogenous SA mimicked these results, indicating that the interference of *B. tabaci* in volatile emission may be mediated by the SA signalling pathway. In another study, attack by the phloem feeder *Euscelidius variegatus* did not alter the volatile blend induced by *Spodoptera littoralis* caterpillars, suggesting an absence of negative crosstalk in this system (Erb, Foresti & Turlings 2010). Although no phytohormonal analyses have been made in the latter study, transcriptional data supports this suggestion, as it showed that contrary to many phloem feeders, *E. variegatus* induced the same JA biosynthetic genes as *S. littoralis*. However, it remains to be investigated whether the effect is dependent on the amount of damage.

VOC induction by pathogens

Plant pathogens are also capable of inducing plant volatiles, although this has been far less studied than induction by herbivorous insects (Fig. 1). As with herbivores, the induced VOC blend can exhibit attacker specificity, with different strains of a same pathogen inducing quantitatively and qualitatively differing VOC blends (Huang *et al*. 2003). In comparison with HIPV, the ecological function of pathogen-induced plant volatiles is not very clear yet, though it is thought that they may function as an additional defence mechanism against pathogen attack. Volatile emission from infected plants has been found to correlate with a hypersensitive response (local cell death in the region surrounding the infection site) in the plant (Croft, Juttner & Slusarenko 1993; Huang *et al*. 2003). Furthermore, several pathogen-induced volatile compounds have been shown to severely inhibit pathogen growth. Such antimicrobial activity was shown for compounds such as (*Z*)-3-hexenol and (*E*)-2-hexenal (Croft, Juttner & Slusarenko 1993) as well as for methyl salicylate (MeSA) and linalool (Shulaev, Silverman & Raskin 1997; Cardoza, Alborn & Tumlinson 2002), which are compounds that are also often emitted in response to herbivory.

The mechanisms underlying volatile production by plants in response to pathogen infection are not well studied, particularly in relation to the phytohormonal signalling pathways. Several microbial elicitors of VOC induction have been identified, and interestingly it appears that some pathogens induce similar patterns of VOCs as those induced by herbivory. One example of this is cellulysin, derived from the fungus *Trichoderma viridae,* which was found to induce volatiles via the JA signalling pathway (Piel *et al*. 1997). Interestingly, alamethicin, isolated from the same fungus, induced only a few volatile compounds; two homoterpenes, whose biosynthesis is dependent on the JA signalling pathway, and MeSA (Engelberth

et al. 2001). The implication of phytohormones in volatile induction is not so straightforward, as was recently shown. High accumulation of JA does not systematically lead to VOC induction. The application of two microbial elicitors, β-(1,3)-β-(1,6)-glucans and *N,N′,N″,N‴*-tetra acetylchitotetraose, both give rise to an accumulation of JA 8-fold higher than levels observed during herbivory. Despite these high JA levels, only the elicitor β-(1,3)-β-(1,6)-glucans led to substantial VOC induction (Leitner *et al.* 2008). This indicates that induction of the JA signalling cascade is not solely involved in triggering volatile induction in this plant species and that other mechanisms, yet to be determined, are likely to be implicated as well. As phytopathogen infection may result in specific plant responses in terms of VOC emissions, the influence of phytopathogens, alone or in combination with an insect herbivore, on VOC induction in plants, as well as the unravelling of the underlying signalling pathways, deserve more attention.

Induced plant VOCs in interactions between phytopathogens and insect herbivores

A number of studies have examined indirect plant-mediated interactions between pathogens and herbivores, primarily in terms of attacker performance. The outcome of such interactions is dependent on the plant, herbivore and pathogen species involved. Pathogen infection may have either beneficial, neutral or detrimental effects on herbivore performance, and likewise herbivory may affect pathogen growth (for a comprehensive overview see Rostás, Simon & Hilker 2003). However, crosstalk effects between phytohormonal signalling pathways and the impact of pathogens on plant volatile emissions remain largely unexplored.

Plant volatiles induced by pathogen infection alone can influence interactions between a plant and its herbivores. These volatile emissions can make the plant more attractive to herbivores (Piesik *et al.* 2011) and can also be used by female insects to discriminate against infected plants for oviposition (Dötterl *et al.* 2009; Tasin, Knudsen & Pertot 2012) and even to differentiate between VOC profiles induced by two different pathogenic fungi (Johne, Weissbecker & Schütz 2008).

The very limited number of studies that have examined plant responses to dual stresses showed that co-occurring attack by a pathogen and an herbivore affects the plant's VOC emissions in response to herbivory, and effects are consistent with expectations in the light of knowledge of underlying phytohormonal signal transduction. Concomitant attack by a hemibiotrophic fungus, *Setosphaeria turcica,* and a leaf chewing herbivore, *S. exigua,* resulted in strongly suppressed volatile emissions in comparison with herbivory alone (Rostás *et al.* 2006). Classically categorized as a necrotroph, recent work suggests that *S. turica* is in fact a hemibiotroph (Chung *et al.* 2010), and the plant defence response to the fungus in the early infection stages appears to be SA and ET pathway regulated (Erb *et al.* 2009). In light of this, the reduced volatile emissions may be an effect of crosstalk with herbivore-induced JA-dependent defences. A similarly attenuated volatile emission (60% reduction) was reported for dual attack by a leaf chewer and a phloem feeder (Rodriguez-Saona, Crafts-Brandner & Cañas 2003), and it is generally accepted that phloem feeders and (hemi-)biotrophic pathogens elicit similar responses in the plant (Walling 2000). In contrast, infection of peanut plants by the necrotrophic pathogen *Sclerotium rolfsii*, thought to primarily induce JA, had no negative effect on VOC emissions in response to herbivory by *S. exigua* caterpillars. Along with pathogen-induced volatiles, the plants emitted all the compounds that are produced in response to *S. exigua* herbivory alone, often in greater amounts (Cardoza, Alborn & Tumlinson 2002). Dual induction of the JA signal-transduction pathway may explain the enhanced VOC emissions. Cardoza *et al.* (2003a) later showed for this system that dually challenged plants had significantly higher levels of JA than the expected additive effect of the individual attackers. The nature of the interaction between a plant and pathogen strain can add a further layer of complexity to the outcome of the plant-pathogen-insect interaction. A plant's volatile response to herbivory can substantially differ if the co-occurring strain of *Xanthomonas campestris* has either a compatible (infection) or incompatible (plant resistance) interaction with the plant. Compared to herbivory alone, co-attack by a compatible strain enhanced VOC emissions, while infection with an incompatible strain led to the suppression of herbivore-induced compounds (Cardoza & Tumlinson 2006).

Volatile emissions can also be manipulated by pathogens to their own benefit, namely the attraction of insect vectors to ensure dispersal to other host plants. This is commonly the case with viruses, which can alter plant volatile emissions resulting in attraction of aphid vectors to plants that may even be of suboptimal quality for them (Fereres & Moreno 2009; Bosque-Pérez & Eigenbrode 2011), or decrease the attraction of non-vectoring herbivores (van Molken *et al.* 2012). Virus infection can lead to elevated levels of the same volatile blend produced by healthy plants (Eigenbrode *et al.* 2002; Mauck, De Moraes & Mescher 2010) which can make infected plants more attractive to herbivores. Such manipulation of plant volatiles is not restricted to viruses. Both bacterial (Mayer, Vilcinskas & Gross 2008) and fungal pathogens (McLeod *et al.* 2005) can induce changes in their host plant's volatile emissions that result in the attraction of insect vectors, which will then carry the pathogen to new host plants.

Effects of VOCs on the natural enemies of herbivores

Many of the studies on HIPV in the context of tritrophic interactions were conducted with one plant, herbivore and carnivorous species. However, even in these simple systems, it is apparent that there exists a large amount of variation in HIPV induction between organisms and study

systems. Moreover, our knowledge on the mechanisms of defence induction indicates that simultaneous or sequential attack by different herbivore species, particularly when they belong to different feeding guilds, can have important consequences for the ecological dynamics of a tritrophic system (for a review see Poelman, van Loon & Dicke 2008b; Dicke, van Loon & Soler 2009). It is widely recognized that carnivorous insects exploit plant volatiles that are produced in response to feeding damage as a navigational system for prey/host location, and much attention has been given to the roles of induced VOCs in mediating tritrophic interactions (Sabelis & Van De Baan 1983; Turlings *et al*. 1995; Heil 2008; Dicke & Baldwin 2010; Reddy 2012).

The effects of multiple attack on natural enemies foraging success can be positive, negative or neutral. For instance, when two organisms inducing the JA signalling pathway co-occur on a plant, natural enemies often become more attracted to such plants (Shiojiri *et al*. 2001; de Boer *et al*. 2008), also when the second attacker is a pathogen (Cardoza, Teal & Tumlinson 2003b). However, this may depend on the natural enemy species. Shiojiri *et al*. (2001) showed that herbivory by *Plutella xylostella* and *Pieris rapae* caterpillars enhanced attraction of the parasitoid *Cotesia glomerata* even though it can only parasitize one of the lepidopterans, while *C. plutellae* preferred VOCs of plants infested with only its host. On dually infested plants this then translates into increased *C. glomerata* parasitism rates, and a decrease for *C. plutellae*, compared to parasitism rates on plants with only their respective hosts (Shiojiri *et al*. 2002). Some studies have also examined effects on the third trophic level when both the JA and SA signal-transduction pathways are assumed to be induced, by combinations of herbivores and/or pathogens. While in one case attack by two herbivore species, respectively affecting JA and SA signalling, led to a decrease in carnivore attraction compared to single-species herbivory (Zhang *et al*. 2009), in all other cases, carnivores were either equally attracted to, or preferred dually damaged plants over plants damaged by the host herbivore alone, for both dual attack by two herbivores (Moayeri *et al*. 2007; Erb, Foresti & Turlings 2010) and a combination of herbivore and pathogen (Rostás *et al*. 2006). Interactions between the two primary signal-transduction pathways may play a role in the changes in VOC emissions in response to dual attacker events. This in turn may affect the response of natural enemies depending on the blend characteristics a species uses in VOC-mediated foraging behaviour.

While higher levels of VOC emissions can increase the attraction of natural enemies in both single and multiple attack scenarios, changes in levels of specific compounds after dual attack can have serious implications for foraging decisions. Absence of a key compound can lead to a greatly diminished response of a predator. Predatory mites showed a strongly diminished response to plants infested by spider mites and nonprey whiteflies (Zhang *et al*. 2009). Dually damaged plants were shown to have reduced levels of (*E*)-β-ocimene emission, which is a compound known to be attractive to the predator (Dicke *et al*. 1990). Supplementing the blend from spider mite plus whitefly infested plants with this compound restored predator attraction, demonstrating its importance for locating prey by the predators (Zhang *et al*. 2009). The reduction in (*E*)-β-ocimene emission in response to whitefly infestation correlated with reduced JA titre and reduced transcription of the JA-regulated gene encoding for the enzyme ocimene synthase that is crucial for in (*E*)-β-ocimene production (Zhang *et al*. 2009). Likewise, a sharp decrease in overall volatile emissions may not have any consequences for parasitoid attraction if the compounds involved in attraction remain relatively unaffected in their emission rates (Rostás *et al*. 2006). Thus, to understand the consequences of dual infestation on the behavioural responses of carnivorous insects, knowledge of the underlying mechanisms is crucial.

Conclusions and future perspectives

In natural systems, co-occurring attack by herbivorous insects and phytopathogens is frequent, and there is a tendency to increase the complexity of the studies investigating plant defence to more realistically reflect natural conditions. Yet, while plant-mediated effects of plant pathogens on insect herbivores, and vice versa, have been examined in some detail, for example, in terms of attacker performance, their combined effects on underlying signal-transduction networks and induced volatile responses have only rarely been considered. Recent studies show that including another insect attacker greatly increases the complexity of interactions between the plant and its attackers, both in terms of direct and indirect plant-mediated defences (Dicke, van Loon & Soler 2009), so the inclusion of phytopathogens into the system can be expected to have similar effects. The very limited research on the subject so far indicates that not only are phytopathogens capable of inducing plant volatiles, sometimes similarly to the response to herbivory, they can also have an influence on host-plant searching behaviour of herbivores, and also on insects of the third trophic level which often rely on volatile cues for locating their herbivorous victims. This topic warrants further research, as the combination of plant–insect and plant–pathogen interactions into one system will provide important insight into how plants prioritize and integrate signals coming from different suites of attacking organisms.

Our knowledge of VOC induction under multiple attack is still too limited to be able to draw solid conclusions about how plants determine VOC induction patterns, Even in the more intensively studied area of HIPV it is apparent that the knowledge of what happens during single herbivory is not sufficient to predict a plant's responses when facing multiple herbivores (Dicke, van Loon & Soler 2009). Dual attack appears to lead to high variability in the effects on VOC emission; several factors influencing

signal-transduction networks and subsequent VOC induction (below) must be taken into consideration to reveal emerging patterns.

ATTACKER IDENTITY

Different combinations of attacking organisms may induce different signalling pathways, so the final plant defences will be highly dependent on the combination used. Induction with two organisms that induce the same defence pathways will have different effects than two organisms inducing different pathways.

SEVERITY OF ATTACK

Plant responses to herbivory, both in single and in multiple herbivore attack are dependent on the amount of damage inflicted by attacking herbivores (Geervliet *et al*. 1998; Zhang *et al*. 2009). It is likely that the severity of pathogen infection also exhibits dose-dependent effects on volatile emissions.

SEQUENCE OF ATTACK

The order in which organisms attack can be an important determinant of the plant's response (Viswanathan, Lifchits & Thaler 2007; Erb *et al*. 2011b), and SA-mediated induction prior to – or simultaneously with – JA-mediated induction is most often researched. Though the inhibitive effects of SA on JA are stronger than the contrary (Koornneef *et al*. 2008), the effect of reverse induction on volatile induction still warrants investigation.

TIMING OF ATTACK

Induction of the signal-transduction pathways occurs with a specific temporal pattern, and so a second attacker will have a certain timeframe during which it can have the greatest impact on the defence signalling network and subsequent volatile induction. Of particular interest is the case of hemi-biotrophic pathogens, which are biotrophs in the initial stages of infection, and then adopt a necrotrophic lifestyle, each theoretically inducing different signal-transduction pathways in the plant. Knowing this, how is crosstalk and volatile induction affected if an herbivore subsequently arrives in either the biotrophic or nectrotrophic phase?

ABIOTIC CONDITIONS

Changes in environmental conditions hold great potential to alter induced volatile emissions, both in terms of composition and quantity. Factors such as light, temperature, humidity, soil moisture, nutrient availability and ozone can all affect HIPV emission (Gouinguené & Turlings 2002; Holopainen & Gershenzon 2010; Loreto & Schnitzler 2010). Such factors should be considered as they can have significant effects on volatile blend characteristics.

OTHER PHYTOHORMONES

Recent research in plant–pathogen interactions indicates that several other phytohormones play a larger role in modulating plant defence than previously thought. Among many examples, auxin appears to disrupt biosynthesis of SA and to modify JA signalling, abscisic acid can antagonize JA-ET signalling (Pineda *et al*. 2013), and brassinosteriods can lead to increased plant resistance towards biotrophic pathogens (Robert-Seilaniantz, Grant & Jones 2011). It is clear that research on subcellular mechanisms of plant defence should look further than the classic trio of SA, JA and ET, and the impact other phytohormones may have on volatile induction is not yet known.

A more holistic approach is needed in the study of the ecology of induced plant volatiles during multiple attack, by integrating research approaches from the molecular to the ecological level, to gain a more comprehensive view of the mechanisms involved throughout the different levels of biological organization. More specifically, issues to address are how multiple attack affects the signal-transduction network, and how these changes then affect biosynthesis of volatiles compounds, as well as their emission patterns. And finally, how does variation in emission patterns affect the members of the third trophic level that depend on VOCs for, for example, host or prey location. However, most of the knowledge on the mechanisms was gained from the model plant *Arabidopsis thaliana*. There is a need to use current knowledge gained from this system as a starting point for investigating ecologically relevant model systems. Moreover, research needs to be conducted out in the field as well as in the laboratory, as VOC emission patterns obtained under laboratory conditions may be different from patterns observed when a study system is in a natural environment, where various biotic and abiotic factors can have a strong influence on the emission of compounds (Kigathi *et al*. 2009). Only by combining both approaches can we have a better understanding of the ecological functions of plant volatiles.

Plants are members of complex communities. Initial attack by one of the community members may have long-term effects on community dynamics (Van Zandt & Agrawal 2004; Poelman *et al*. 2008a, 2010). To understand such community dynamics and the underlying processes that shape them, it is important to understand how community members modify the expressed plant phenotype. An important component of the plant phenotype consists of its emission of VOCs as the VOC blend provides phenotypic information at a distance of the plant. This VOC-aspect of the plant phenotype mediates interactions with various community members (Dicke & Baldwin 2010). So far, induced plant volatiles have been especially investigated in the context of plant–arthropod interactions. How-

ever, first evidence shows that plant pathogens can have important impacts on induced plant volatiles as well and, therefore, integrating plant-arthropod and plant–pathogen interactions into studies on the effects of HIPV on community dynamics of plants and their attackers and members of higher trophic levels will be an important next step. This integrative approach is likely to unravel new insights into the community ecology of plant-based communities.

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