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INVITED REVIEW

The evolutionary strategies of plant defenses have a dynamic impact on the adaptations and interactions of vectors and pathogens

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Abstract Plants have evolved and diversified to reduce the damages imposed by infectious pathogens and herbivorous insects. Living in a sedentary lifestyle, plants are constantly adapting to their environment. They employ various strategies to increase performance and fitness. Thus, plants developed cost-effective strategies to defend against specific insects and pathogens. Plant defense, however, imposes selective pressure on insects and pathogens. This selective pressure provides incentives for pathogens and insects to diversify and develop strategies to counter plant defense. This results in an evolutionary arms race among plants, pathogens and insects. The ever-changing adaptations and physiological alterations among these organisms make studying plant–vector–pathogen interactions a challenging and fascinating field. Studying plant defense and plant protection requires knowledge of the relationship among organisms and the adaptive strategies each organism utilize. Therefore, this review focuses on the integral parts of plant–vector–pathogen interactions in order to understand the factors that affect plant defense and disease development. The review addresses plant–vector–pathogen co-evolution, plant defense strategies, specificity of plant defenses and plant–vector–pathogen interactions. Improving the comprehension of these factors will provide a multi-dimensional perspective for the future research in pest and disease management.

Key words coevolution, induced defense, phytohormones, plant–vector–pathogen interactions, specificity of defense, transmission mechanism

Introduction

Plants have evolved sophisticated strategies to defend against insect pests and pathogens, their two main enemies. Those defenses include constitutive defenses, present continuously such as wax or thorns, but also induced defenses only produced while under attack. When an attack is detected, plants can allocate energy to produce toxic compounds for defense or to increase growth to compensate for loss of tissue due to herbivory. According to the resource availability and plant optimal defense theories, allocation of more energy for defense is done at the detriment of growth and development since plants have finite resources (Coley *et al.*, 1985; Karban & Baldwin, 1997). Since defense is very costly, some defenses are only produced when plants are under attack and the production of defensive chemicals can affect plant growth (Zhang & Turner, 2008). Consequentially, plants induce defense under stress (i.e., herbivory or infection) when the benefits of defense outweigh the cost (Karban & Baldwin, 1997). Here, we highlight some of the features in plant defense but focus on (i) the plant defense against

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pathogens and vectors and (ii) the roles of plant defenses in plant–vector–pathogen interaction. The study of plant–vector–pathogen interactions is complex. To make sense of this intricate relationship, knowledge on the general evolutionary relationship among them is essential.

Plant–vector–pathogen coevolution

The interactions among plant, vector and pathogen often result in a selective adaptation that favors traits for better fitness. The genetic traits of victim and exploiter can potentiate for generations with limitless escalation resulting in a coevolutionary arms race among species (Bergelson *et al.*, 2001).

The evolutionary arms race between plants and herbivorous insects has resulted in the development of a battery of plant defenses and of several insect strategies used to counteract plant defenses (Karban & Baldwin, 1997). As plants evolved to be more toxic to fence off herbivory, insects adapted by developing strategies to manipulate host plant defenses. For instance, plants produce phenolic compounds (compounds with aromatic 6-carbon ring bonded to a hydroxyl group) that when ingested by insects autoxidize and combine with proteins in the insect guts that can be very toxic to insects (Miles, 1999). Phloem-feeding insects in the family of Aphididae have evolved a strategy to counter this plant defense: during feeding they secrete saliva containing polyphenol oxidases that detoxify phenolic compounds (Urbanska *et al.*, 1998; Fig. 1). Certain phloem-sucking insects can also manipulate plant defenses (Will *et al.*, 2007) and increase plant susceptibility to subsequent attacks. For example, *Myzus persicae* and *Aphis fabae* were found to improve sugar beet (*Beta vulgaris*) host quality for subsequent infestation by the same aphid species (Williams *et al.*, 1998).

Many environmental factors have affected the evolution of plant–enemies interaction. For instance, plants and insects did not evolve alone. They also have interacted and evolved with pathogens. Aside from defending from insects, plants also constantly defend against exploiting pathogens. Although it is assumed that most pathogens have negative effects on host plant fitness, the result is variable. Pathogen infection may have positive effects on plant host (Goss & Bergelson, 2007). Some pathogens benefit from maintaining their host alive to proliferate and spread whereas others kill their host. Plants surviving pathogen attack might develop resistance as plants with favorable traits survive, similarly advantageous pathogen strains that can overcome plant defenses can proliferate and increase the infection efficiency. Thus, as with insects, the interactions between plants and their pathogens result in a coevolutionary arms race. The coevolution between plants and pathogens is mediated by the gene-specific arms race that led for example to the diversification of pathogen avirulence (Avr) genes and plant resistance (R) genes (Fig. 1). As pathogen Avr genes diversified, plant R genes also evolved and adapted to enhance plant resistance against infection (Dodds *et al.*, 2006).

Plant defense strategies have a huge impact on the adaptation of corresponding insects and pathogens. Depending on the plant species, plant modifications following pathogen infection can also affect insects (Stout *et al.*, 2006). A particular case in this category is the study of vector-borne plant pathogen transmission in which infestation is accompanied by plant infection. Although some studies have addressed plant–vector–pathogen interactions, knowledge on this topic remains limited. Interactions between plant, vector, and pathogen are complex. Understanding of the molecular dialog established among the three partners involved in the interaction requires the adoption of a systemic approach to account for all possible interactions.

Plant defenses

Specificity of plant defense

Over time, plants have evolved to utilize phytohormones in a conservative manner to regulate the production of various toxic compounds for defense. Since overproduction of toxic compounds can be costly, plants do not activate all defenses upon attack but they utilize a strategy called specificity of induction. Instead of producing multiple kinds of toxic compounds for defense, they produce specific compounds that target particular attackers. For instance, tomato (*Solanum lycopersicum*) responds to leaf-chewers like *Helicoverpa zea* by producing polyphenol oxidases and proteinase inhibitor while it responds to phloem feeders like aphids by producing peroxidases (Stout *et al.*, 1998). By producing certain antinutritive enzymes, plants can effectively reduce subsequent damage imposed by specific attackers that continue to feed on the plant (Fig. 1). When damaged by the specialist leaf-chewer *Plutella xylostella,* wild radish (*Raphanus sativus*) can induce defenses resulting in reduced feeding and decreased insect biomass in subsequent attacks by the same and other leaf-chewers (Agrawal, 2000).

Similarly, plant responses against pathogens are highly specific. Plants resistance (R) genes encode R proteins. The R proteins recognize the corresponding avirulence (Avr) proteins encoded by specific pathogens. When the

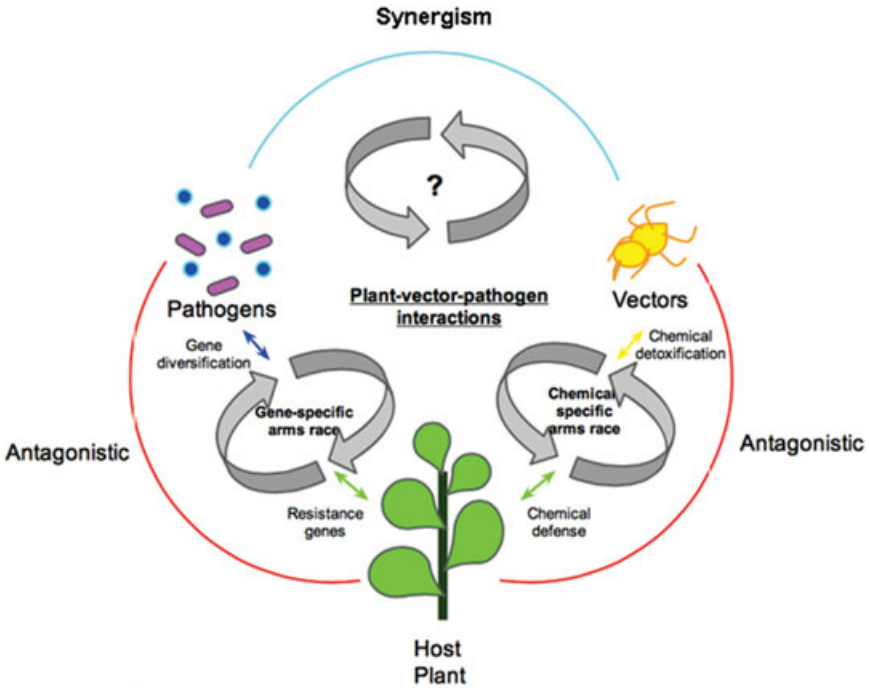

Fig. 1 Plant–vector–pathogen interactions. The relationship among host plant, insect vector and infectious pathogen is influenced by the adaptation to each organism. Their evolutionary interactions have resulted in arms race (curved gray arrows) improving enemy attack strategies (yellow and blue arrows) and host defense strategies (green arrows). Often, insect vectors and infectious pathogens have antagonistic relationships with host plants (red line). Moreover, their negative effects can synergize when attacking the same plant (blue line). Although there are exceptions, vector and pathogen often impose multiple stresses on the plant. Any stress that reduces plant performance and fitness can increase plant susceptibility to vector infestation and pathogen infection.

plant R proteins directly interact with the corresponding pathogen Avr proteins, plant defenses are triggered resulting in hypersensitive response (HR). During HR, cell death is induced surrounding the attack site resulting in necrosis at the region of infection and effectively limiting the spread of pathogens (Morel & Dangl, 1997). During evolution time, pathogen Avr proteins and plant R proteins diversified, but each individual only encodes a subset of them. For instance, flax *(Linum usitatissimum)* R genes only recognize seven variations of the Avr proteins of the flax rust fungus *(Melampsora lini)*. Only seven of twelve variants of *AvrL567* alleles derived from the flax rust fungus induced necrosis in flax plants as plant induced defense against the pathogen (Dodds *et al.*, 2006). Therefore, plant response to pathogen infection is certainly specific.

Induced plant defenses

When plants detect an attack by herbivorous insects or pathogens, they activate plant induced defenses, which are defense cascades producing antinutritive factors (e.g., proteinase inhibitor) or with antimicrobial properties (e.g., phytoalexins). The induction of plant defenses is regulated by phytohormones such as jasmonic acid (JA), salicylic acid (SA), and ethylene (ET) that signal the production of defensive compounds. With a few exceptions, JA has generally been associated with plant defense against leaf-chewing herbivores and wounding whereas SA has generally been associated with plant defense against phloem feeding herbivores and biotrophic pathogen (Karban & Baldwin, 1997; Walling, 2000; Thaler *et al.*, 2001; Kessler & Baldwin, 2002; Glazebrook, 2005; Howe & Jander, 2008). The activation of these pathways leads to a myriad of responses including the production of defensive compounds aimed at protecting the plant and fighting off attackers. Several studies have shown that these pathways can interact or crosstalk to fine-tune the response to different attackers (Reymond & Farmer, 1998; Rojo *et al.*, 2003). For instance, ET and JA are involved in the resistance against many pathogens. Similarly, the negative crosstalk between SA and JA has been reported in different systems (Doherty *et al.*, 1988; Penacortes *et al.*, 1993; Baldwin *et al.*, 1996; Harms *et al.*, 1998) whereas positive interactions have been found in the case of aphid infestations (Moran & Thompson, 2001; Ellis *et al.*,

2002; Zhu-Salzman *et al.*, 2004; De Vos *et al.*, 2005) or pathogen infection (van Wees *et al.*, 2000). It appears that the type of interaction (antagonist or positive) depends on phytohormone concentration (Devadas *et al.*, 2002).

Plant defense against pathogens

The mechanism of plant defense against pathogens has been well documented in the study of SA signaling pathway. In this pathway, one quick strategy of defense against pathogen infection is HR. As previously stated, during HR plants activate programmed cell death (PCD) at local leaves isolating infected cells from the main system thus preventing systemic proliferation of the pathogen (Mur *et al.*, 2008). Plants exposed to avirulent pathogen strains, rapidly produce an oxidative burst resulting in reactive oxygen species (ROS) production such as hydrogen peroxide (H_2O_2), which triggers PCD, and activates HR (Levine *et al.*, 1994). ROS and HR have been associated with SA signaling transduction pathway (Chen *et al.*, 1993). In plants, SA functions as a phytohormone and is involved with not only the signaling of ROS for HR but also with the activation of pathogenesis related (PR) genes. PR genes encode proteins that elicits plant systemic acquire resistance (SAR). SAR is a plant innate immune response that enhances the resistance against infection to subsequent attacks by the same or related pathogens (Chester, 1933; Enkerli *et al.*, 1993). While the activation of HR is quick, the induction of SAR is relatively slow. Hence, HR serves as the primary wall of defense whereas SAR serves as the secondary wall of defense. In plants, SAR and HR are also associated with protection against phloem-feeding vectors (Karban & Baldwin, 1997).

Other phytohormone pathways also play a role in plant defense against vectors and pathogens. JA defense pathway has been known to elicit compounds that have antimicrobial properties helping plant defense against pathogen (Creelman & Mullet, 1997). Furthermore, mutant *Arabidopsis* plants that cannot synthesize JA are more susceptible to infection by fungus *Pythium mastophorum* (Vijayan *et al.*, 1998). This indicates that JA can also play a role in plant defense against pathogen infection. JA also interacts with other phytohormones in the defense against pathogen. For instance, treatment with abscisic acid (ABA) can elicit lipoxygenase gene expression that control the biosynthesis of JA (Melan *et al.*, 1993), and ABA was shown to cooperate or precede JA in the activation of *Arabidopsis thaliana* defense genes against *Pythium irregulare* and other necrotrophic pathogens (Adie *et al.*, 2007). ABA is a phytohormone that regulates plant response to abiotic and biotic stresses. ABA is usually associated with cold and drought stress, but also commonly associated with leaf abscission. Upon pathogen infection, ABA production might be induced to regulate leaf abscission, which reduces the spread of the pathogen or delays the development of the disease since by abscising the infected leaf, plants can stop the infection progression. ABA might also be involved in priming of callose biosynthesis after pathogen recognition (Ton & Mauch-Mani, 2004), a plant response to fungal pathogens. Therefore, ABA might not only play a direct role but also an indirect role in plant defense against pathogen. As shown, various phytohormones have been documented to play a vital role in plant defense against pathogens and insects. Thus, phytohormones affect plant and may mediate pathogen transmission by vectors.

Plant and vector interactions

Insects with piercing-sucking mouthparts are major vectors of plant pathogens; they are responsible of transmitting more than 50% of vector-borne viruses (Hogenhout *et al.*, 2008) and a great number of vector-borne bacterial pathogens.

In spite of common feeding mechanisms, piercing-sucking insects have been shown to activate different defense pathways (Thompson & Goggin, 2006). Phloem feeding insects can activate only SA-defenses or SA- and JA/ET-defenses. Several studies showed that aphids (*M. persicae*, *Schizaphis graminum*, and *Acyrthosiphon kondoi*) activate the SA pathway and the JA pathway albeit at a lower level in *A. thaliana* (Moran & Thompson, 2001; Ellis *et al.*, 2002; De Vos *et al.*, 2005), *Sorghum bicolor* (Zhu-Salzman *et al.*, 2004; Park *et al.*, 2006), and *Medicago truncatula* (Gao *et al.*, 2007), respectively. However, studies performed with whiteflies showed that plant responses differ according to the whitefly species and insect developmental stage (van de Ven *et al.*, 2000). In *A. thaliana*, *Bemisia tabaci* Biotype B nymphs were shown to induce genes involved in SA-biosynthesis or SA-regulated whereas genes involved in JA biosynthesis and JA-regulated were repressed or showed modest to no changes in RNA levels using a microarray analysis (Kempema *et al.*, 2007) and RT-PCR (Zarate *et al.*, 2007). Nevertheless, studies carried out in *S. lycopersicon* demonstrated that *B. tabaci* and *Trialeurodes vaporariorum* induced the expression of JA-regulated genes and a low-level induction of some SA-regulated genes (Puthoff *et al.*, 2010). Similarly, studies conducted in *Nicotiana attenuata* plants deficient in JA biosynthesis or perception showed that JA signaling mediates plant choice by *Empoasca* leafhoppers (Kessler *et al.*, 2004; Kallenbach

et al., 2012). Therefore, even within the same feeding guild or insect species, plant responses against infestation can differ.

Several reasons might explain the differences in plant responses induced upon vector feeding. Herbivore insects with piercing-sucking mouthparts cause little plant damage during feeding. For instance, phloem-feeding insects use their piercing sucking mouthparts to reach the phloem. The path used to access the phloem has been shown to go in-between cells. During probing, or plant penetration, aphids "taste" the host plant by puncturing epidermal and mesophyll cells (López-Abella & Bradley, 1969; Powell, 1991), while whiteflies have been shown to rarely puncture cells (Janssen *et al.*, 1989; Johnson & Walker, 1999). Activation of JA/ET defenses is generally weak when occurring, and it is probably in response to light damage caused by the stylet during probing. Difference in feeding behavior (frequency of cell punctures during probing) is therefore one of the hypotheses to explain differences in induction of plant defense pathways by phloem feeding insects.

Sucking insects secrete saliva during feeding. Therefore, it is possible that variations in saliva composition of sucking insects (Miles, 1999) could explain the observed differences in the induction of plant defense mechanisms upon herbivory. Two types of secretion have been identified for phloem feeding insects, the sheath saliva and the watery saliva. Sheath saliva is secreted during stylet penetration limiting plant cell damage as well as protecting the stylet from plant defenses. Sheath saliva plugs the punctured sieve elements preventing phloem sap leakage into the apoplast, which could trigger plant wound responses resulting in plugging of the sieve pore (Knoblauch & van Bel, 1998; Will & van Bel, 2006). Watery saliva is continuously secreted during feeding on the phloem (Tjallingii, 2006) and might participate in prevention of sieve element plugging and suppression of plant defenses (Tjallingii, 2006). Analysis of *Acyrthosiphon pisum* salivary glands using proteomic and transcriptomic approaches identified more than 300 potentially secreted proteins (Carolan *et al.*, 2011) whereas analysis of whitefly salivary gland transcriptome identified 295 genes encoding putative secreted proteins (Su *et al.*, 2012). These findings highlight the complexity of phloem feeder–plant interaction.

Finally, the observed differences in the induction of plant defense mechanisms upon herbivory might be a consequence of the ability of some insects to actively suppress plant defense mechanisms. In several instances, phloem feeders were shown to actively suppress JA-based defenses (Thompson & Goggin, 2006; Kempema *et al.*, 2007; Zarate *et al.*, 2007).

Unraveling the intricacy of plant–vector–pathogen interaction

Mechanisms of pathogen transmission

Arthropods are major vectors of plant pathogens, some of which are the causative agent of several important emerging diseases. Four transmission mechanisms have been described based on where on the vector the pathogen is transported: attached to the cuticle lining of the mouthparts or the foregut (nonpersistent and semipersistent transmission, respectively) or internalized by vector cells (persistent transmission) which can be persistent circulative transmission (if the pathogen does not propagate within the vector) or persistent propagative (if the pathogen propagates within the vector) (Gray & Banerjee, 1999). The transmission mechanism determines several parameters of the vector–pathogen interaction. For instance, viruses that are transmitted in a nonpersistent manner remain in the vector for a short period of time (minutes to hours), while vectors remain viruliferous for longer periods of time in the case of persistently transmitted viruses (from days up to the vector lifespan). Therefore, the time the vector spends in one plant could play an important role in pathogen transmission. In this context, the ability of pathogens to manipulate plants, including plant defenses, could affect vector and pathogen transmission. Indeed, many examples of plant modification by pathogen affecting vectors have been shown (discussed later).

The roles of plant defenses in plant–vector–pathogen interaction

Plants are subject to attack by insects and pathogens either individually or simultaneously. Numerous studies have demonstrated the importance of phytohormones SA, JA, and ET in plant defenses against either insect herbivore attack (reviewed in Walling, 2000; Kessler & Baldwin, 2002; Kaloshian & Walling, 2005; Howe & Jander, 2008) or pathogen attack (reviewed in Felton & Korth, 2000; Glazebrook, 2005). Only a handful of studies have examined the role of defense signaling pathways when insects and pathogens coinvade a plant as in the case of vector-borne plant diseases. The main mechanism that is thought to underlie plant–insect vector–pathogen interaction is the antagonistic crosstalk between SA and JA signaling pathways (Stout *et al.*, 2006). As a result of a SA–JA antagonism, a pathogen-infected plant is thought to become more susceptible to attack by the insect vector that was previously resisted via JA-related resistance

(Belliure *et al.*, 2005). This would lead to increased viruliferous vectors on infected plants causing disease epidemics. Furthermore, nonvectors which induce similar antiherbivore defenses as the vector can benefit from antagonistic crosstalk by feeding on infected plants (Belliure *et al.*, 2010).

Plant viral infection affects vectors

To date, most plant–pathogen–vector interactions have focused on plant viruses and their insect vectors. Recently, it was demonstrated that *Tomato spotted wilt virus* (TSWV) increased SA levels and SA-related marker gene expression but reduced JA-related gene expression in TSWV-infected *Arabidopsis* plants (Abe *et al.*, 2012). The insect vector that transmit TSWV, western flower thrips (*Frankliniella occidentalis*) has been shown to induce JA-regulated defenses in plants (Abe *et al.*, 2008). On TSWV-infected plants, thrips showed increased feeding and fecundity presumably because JA-related defenses were reduced. Twospotted spider mites (*Tetranychus urticae*), another herbivorous arthropod also had greater population growth on TSWV-infected plants compared to healthy plants (Belliure *et al.*, 2010). The Begomovirus, *Tomato yellow leaf curl China virus* (TYLCCNV) and betasatellite coinfection repressed JA-related defenses, which resulted in increased whitefly *B. tabaci* Biotype B vector populations on virus-infected tobacco plants (Zhang *et al.*, 2012). Evidence suggest that the pathogenicity factors, βC1 of TYLCCN (Zhang *et al.*, 2012) and 2b of cucumber mosaic virus (CMV) (Lewsey *et al.*, 2010) inhibit JA-responsive genes, which can influence virus transmission by vectors (Fig. 1). Geminivirus infection is also known to reduce JA responses by the action of virus transcriptional activator protein C2. Reduction in JA responses due to virus infection may serve as a crucial viral defense mechanism and may also have an impact on the whitefly vector (Lozano-Durán *et al.*, 2011). Interestingly, whitefly nymphs induced SA-related gene expression and repressed JA-related genes (Kempema *et al.*, 2007). However, JA-related defenses are critical for whitefly nymph development (Zarate *et al.*, 2007). Volatile Organic Compounds (VOCs), which are released as a result of induction of defense signaling pathways due to pathogen infection is also known to mediate host plant preference by the insect vector. For example, squash plants infected with CMV released volatiles that attracted the aphid vector at first, but were poor hosts, which resulted in subsequent dispersal of the vector to uninfected plants (Mauck *et al.*, 2010). This is particularly interesting since CMV is transmitted in a nonpersistent manner; aphids remain viruliferous for a short period of time. Therefore, in this case virus transmission improves if the insect vector visits a new plant shortly after virus acquisition. The transmission mechanism of a virus has been shown to affect plant preference and time the vector spent on infected plants (Sisterson, 2008). With short persistence periods, efficient transmission only occurred if aphids were first attracted to infected plants but only remained a short period on these plants before moving to an uninfected host. Several factors might affect the attractiveness of the plant and the amount of time the vector spends in an infected plant. Those factors might include visual and olfactory cues, immune responses as previously discussed or plant composition (Khan & Saxena, 1985). Similarly, vectors can manipulate host plant and affect viral infection and the epidemiology of plant diseases. Prior infestation of *M. persicae* has been shown to increase plant susceptibility and significantly increase infection by *Beet mild yellowing virus* (BMYV; Williams *et al.*, 1998).

Plant bacterial infection affects vectors

Unlike viruses that are predominantly vector-transmitted, most bacterial plant pathogens are not vector-borne, which may explain the limited literature on the latter topic. Nevertheless, there appears to be commonalities in the published literature about plant virus and plant bacteria–vector interactions. Bacterial plant pathogens also modify host plant defense responses to benefit population growth of its insect vector and pathogen spread. For example, transcriptomic analyses of tomato plants following infestation with potato/tomato psyllid, *Bactericera cockerelli*, nymphs harboring "*Candidatus* Liberibacter solanaceraum" (CLs), the bacterial plant pathogen causative agent of potato zebra chip disease, induced distinct SA and JA gene expression compared to control plants (Casteel *et al.*, 2012). However, these responses were suppressed when plants were inoculated with the pathogen, CLs alone. These results were obtained using a potato microarray and relaxing statistical criteria to $P < 0.1$. No analyses of the effects of the vector alone were performed. A bacterial plant pathogen related to CLs, "*Candidatus* Liberibacter asiaticus" (Las) the causal agent of huanglongbing (citrus greening) caused the release of large quantities of methyl salicylate, a product of the SA signaling pathway. This compound was attractive to the vector, the Asian citrus psyllid, *Diaphorina citri*, and resulted in greater psyllid populations on infected citrus plants compared to uninfected plants (Mann *et al.*, 2012). In another study, lipoxygenase (LOX) was

found to be upregulated in Las-infected sweet orange leaves compared to uninfected leaves using iTRAQ (Fan *et al.*, 2011). Phytoplasmas are unculturable bacterial pathogens that are exclusively transmitted by phloem-feeding insects in the order Hemiptera (Weintraub & Beanland, 2006). It was demonstrated that an effector from Aster yellows phytoplasma, SAP11 destabilized cincinnata (CIN)-related teosinte branched1, cycloidea, proliferating cell factors 1 and 2 (TCP) transcription factors resulting in the reduced expression of JA-related marker gene, LOX2 in Aster yellow-infected *Arabidopsis* plants (Sugio *et al.*, 2011). Furthermore, the insect vector, showed increased fecundity on Aster yellows-infected plants, transgenic plants expressing SAP11 protein and plants compromised in CIN-TCP and JA synthesis. Similarly, transcriptomic analysis of mandarin infected with *Xylella fastidiosa*, a xylem restricted bacterium infecting a large range of hosts, revealed induction of LOX and S-adenosyl-L-methionine:salicylic acid methyltransferase genes, encoding precursor enzymes of JA and SA, respectively as well as induction of several genes involved in defense and oxidative burst (de Souza *et al.*, 2007). These studies show the complexity of plant responses to vector-borne bacterial pathogens and the potential role of phytohormone crosstalk. As with viruses, vector-borne bacterial transmission increases if vectors are attracted to infected plants. "*Candidatus* Phytoplasma mali," the causal agent of apple proliferation, was shown to induce release of β-caryophyllene by apple trees, which attracts apple psyllid vector, *Cacopsylla picta*, and facilitates pathogen spread (Mayer *et al.*, 2008).

Conclusion

Insect vectors and plant pathogens have largely contributed to the economic loss in agriculture production. Each year much money has been invested in biological and chemical control to reduce the crop loss caused by insects and pathogens. Yet, the world is still facing a challenging battle. With climate change, emerging plant diseases and pesticide resistance, insects are on the rise. Developing a feasible and sustainable strategy to combat plant diseases and vector pests is more challenging than ever. To protect crops and feed the ever-increasing population, innovative strategies are required. The strategy for pest and disease management should be based on a concrete knowledge of the biology and ecology of the vector pests and the pathogens they transmit.

Unfortunately, the current knowledge on plant–vector–pathogen interactions is still limited but data are starting to accumulate. Most studies focus on the interaction between plant and pathogen, plant and vector, or vector and pathogen, but very few studies actually focus on all aspects of the interactions among plant, vector and pathogen. Without a doubt this type of multidisciplinary study faces many challenges. For instance, any cost and benefit resulting from the coevolutionary arms race between any two organisms can influence their interactions with other organisms. As shown in this review, plant defenses against pathogen and vector are very specific. An attack from a pathogen can elicit plant regulatory genes that trigger the production of toxic compounds for defense. The triggered response might compromise the ability of the plant to defend against the vector and vice versa. Moreover, the biology of the vector can affect the transmission of the pathogen and subsequently affect plant defense. Not to mention, the synergistic and antagonistic effects of the vector and pathogen on plant defense can complicate the picture. This intricate relationship among plant, vector, and pathogen is fascinating indeed. To understand the interactions among these groups of organisms, a multidisciplinary approach is necessary which will provide a multidimensional perspective. Knowledge on the interactions among plant, vector and pathogen is imperative to develop better agricultural practices.

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Disclosure

The authors have declared that no competing interests exist.

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