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BE SMART TO SURVIVE: VIRUS-HOST RELATIONSHIPS IN NATURE

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ABSTRACT

In order to survive in nature, different pathogens follow different procedures to manipulate their host plants for the pathogen favor. Plant viruses are not an exception of this rule. They are often found to alter the host plant traits in the way that affects the community of organisms in the host plant as well as the vectoring insects. It has been indicated that virus-infected plants are more preferable than virus-free plants with respect to the growth rates, longevity and reproduction of the vector. Viruses use several strategies in order to reprogram their host's cell to make it more conducive to replication and spread. Consequently, phytohormone signaling pathway in virus-infected plants can be disrupted either directly or indirectly. In plants, there are hormone pathways contribute to all aspects of plant physiology. Sometimes, virus infection can be advantageous to the infected host by providing the plant with tolerance to biotic and abiotic stresses. This article summarizes some aspects where the virus found to reprogram the host's cell to make it more conducive to virus' cycle of life. It also provides an important basic knowledge about how biotic and abiotic stress affects the interaction among virus, vector and the host plant; this knowledge could open the gate to understand the effect of multi-stress effect on the host plant in future studies through recognizing the necessity for plants to have an integrated system of defense against different threats.

Keywords: Virus-vector relationship, Plant viruses, virus manipulation of host, virus reproduction

INTRODUCTION

Viral diseases cause severe economic losses by lowering yields and reduce quality of plant products around the world. For example, *Tomato yellow leaf curl virus* (TYLCV) causes quantitative and qualitative yield losses often reach 100% in tomato crop (**Polston *et al.*, 1994; Al-Ani *et al.*, 2011a**); other viruses such as Potato leaf roll virus (PLRV), Barley yellow dwarf virus (BYDV), Potato virus Y (PVY), Cucumber mosaic virus (CMV), Bean yellow mosaic virus (BYMV) and Zucchini yellow mosaic virus (ZYMV) are good examples of the most damaging viruses, which causes yield losses between 30% and 100% in different crops (**Al-Ani *et al.*, 2009; Adhab, 2010; Al-Ani *et al.*, 2010, 2011b, 2011c, 2013; Adhab and Al-Ani, 2011; Al-Ani and Adhab, 2013**). Plant viruses use the host plant resources to support their own survival and spread, so that, in agricultural systems, they are normally considered harmful to the host plant. But, viruses, plant and the environment have a very complex interaction. Some viruses are beneficial to their hosts. For instance, **Xu *et al.*, (2008)** have shown that infection with different RNA viruses provides water stress tolerance to multiple species of plants.

Historically, plant virologists focused on viruses that cause disease in crops, but more recently it has been shown that viruses are asymptomatically very abundant in wild plants (**Roossinck 2012, Roossinck *et al.*, 2015**). Further research on the role of viruses in wild plants has revealed details of virus-arthropod-plant relationships and indicated long-standing interactions among multiple partners (**Khalaf et al., 2019; Wen-Po *et al.*, 2017**). Virus ecologists look at viruses as symbionts (**Roossinck, 2015**). Symbiosis is the situation when two various entities living in or on one another in a mutually beneficial relationship (**deBary 1879**). Symbionts (e.g. viruses) can move from pathogenic to mutualistic depending on the environmental conditions of the host (**Roossinck 2015**). Some viruses show mutualistic relationships with their hosts. For example, *Cucumber mosaic virus* (CMV), *Tobacco mosaic virus* (TMV) and *Brome mosaic virus* (BMV) provide tolerance to water deficit stress in their host plants (**Xu *et al.*, 2008**).

Until recently, most virus-host studies focused on the two interactors, the virus and the plant. However, in the recent fifteen years, research shifted to the fact that the virus-host-environment relationships depend on multilayered interactions that include other microbes, invertebrates, neighboring plants and all abiotic stresses in the location of interaction. Indeed, plants, insects and viruses' relationships are ancient and very complex. Virus transmission through vectoring insect is very well evolved. Insects can transmit viruses using different mechanisms depending on a variety of factors (**Bragard *et al.*, 2013**); these different transmission modes can affect the plant-virus-insect relationships. Virus infection alters the host plant traits the way that serves virus spread and reproduction. In many cases, plants infected with viruses change volatile organic compound profiles that elicit better settling of their non-infective vectors (**Eigenbrode *et al.*, 2002, Jiménez-Martínez *et al.*, 2004**). **Adhab et al., (2019)** have found that turnip plant infected with W260 strain of *Cauliflower mosaic virus* (CaMV) attracted more turnip aphids, which is the natural vector of W260 strain (**Adhab & Schoelz 2015**), than uninfected plants at both detached leaf and whole plant levels. The turnip aphid choice is clearly affected by CaMV infection. These collective results have suggested that the CaMV infection of turnip plants affected their susceptibility to biotic and abiotic stresses.

This review is aimed to advance our understanding of how viruses behave in plants in order to spread and survive in nature, and what pathways in the host plants are targeted by the virus.

Virus infection causes changes in phytohormone signaling pathway

Viruses use several strategies in order to reprogram their host's cell to make it more conducive to replication and spread. Consequently, phytohormone signaling pathway in virus-infected plants can be disrupted either directly or indirectly. In plants, there are hormone pathways contribute to all aspects of plant physiology. Salicylic acid (SA), Jasmonic acid (JA), ethylene (Et) are involved in defense systems (**Derksen *et al.*, 2013**) while abscisic acid (ABA), auxin (Aux), gibberellins (GA) and cytokinins (CK) contribute to both defense and plant development and physiological processes (**Robert-Seilaniantz *et al.*, 2011; Durbak *et al.*, 2012**). Viral and host-component interactions involving phytohormone pathways have been identified recently providing some explanation for how viruses manipulate phytohormone regulatory systems to serve the virus development within the host cell. SA and ethylene responsive genes have been shown to be strongly activated in response to CaMV infection in Arabidopsis (**Love *et al.*, 2005**). In the latter study, authors showed that Arabidopsis responds to CaMV by elevating the levels of Salicylic acid, Jasmonic acid/ethylene and reactive oxygen species (ROS). One interpretation of that finding is that plants show systemic response to an elicitor encoded by CaMV. Authors called this phenomenon as the rapid systemic response

(RSR).The phytohormone ABA strongly regulates a subset of plant developmental stages, is the key hormone in the modulation of plant responses to many abiotic stresses including drought (**Atkinson and Urwin 2012; Sung and Luan 2012**), and can be antagonistic to defense hormone pathways, such as JA/Et and SA. Depending on the stage of infection, ABA has multiple roles in defending the plant against the pathogen attack (**Ton *et al.*, 2009**). These results suggest that the virus infection plays a role in changing the phytohormone signaling pathways.

Virus infection increases host plant's tolerance to stressors

Virus infection can be advantageous to the infected host by providing the plant with tolerance to biotic and abiotic stresses. For example, plants infected with viruses have shown more tolerance to water deficit stress (**Xu *et al.*, 2008**). Different plant species such as rice, beet, tobacco, cucumber, pepper, watermelon, squash, and *N. benthamiana*, that were infected with RNA viruses, including *Cucumber mosaic virus* (CMV), *Tobacco mosaic virus* (TMV) and *Brome mosaic virus* (BMV), showed drought symptoms 2-5 days later than non-infected plants. The infected leaves also maintained water content for a longer period of time than the control. The mechanism of this phenomenon is still unknown, but metabolite analysis in some virus-infected plants showed a higher level of osmoprotectants and antioxidants, such as anthcyanins, than uninfected plants (**Xu *et al.*, 2008**). Similarly, specific virus-infected fungal endophytes provided heat tolerance to the host plant *Dichanthelium lanuginosum* (**Márquez *et al.*, 2007**).

Another study found that the benthi plants showed higher drought tolerance when co-infected with potato virus X (PVX) and plum pox virus (PPV) synergistically (**Aguilar *et al.*, 2017**). Also, plants expressing PVX virulence protein P25 showed higher level of tolerance when infected with PPV. Similar results were gained from infected Arabidopsis plants, where virus infections resulted in higher water content in plants. However, the virus infection reduced the host production, which indicates that a significant tradeoff exists between drought tolerance and virulence in infected plants (**Aguilar *et al.*, 2017**).

These results indicate a mutualistic relationship between viruses and host plants. Previous study showed that plants infected with rhizobacterium *Paenibacillus polymyxa* expressed drought tolerance after bacterial attack; this was explained as an effect associated with expression of *ERD15* (early response to dehydration 15) gene (**Timmusk and Wagner, 1999**). Figure 1 shows the complicated events when the plant exposed to different types of stresses. Cellular receptors sense biotic and/or abiotic stresses and trigger gene regulation via signal transduction including Mitogen-activated protein kinases (MAP kinase) cascade, reactive oxygen species (ROS) accumulation and Hormone signaling; then, multiple and individual stress-induced transcription factors, such as WRKY and NAC, will be induced. That leads to post-translational regulation of transcription factors (TFs) and, then, to expression of functional downstream response genes that leads, eventually, to post-transcriptional regulation and stress tolerance (**Atkinson and Urwin, 2012**).

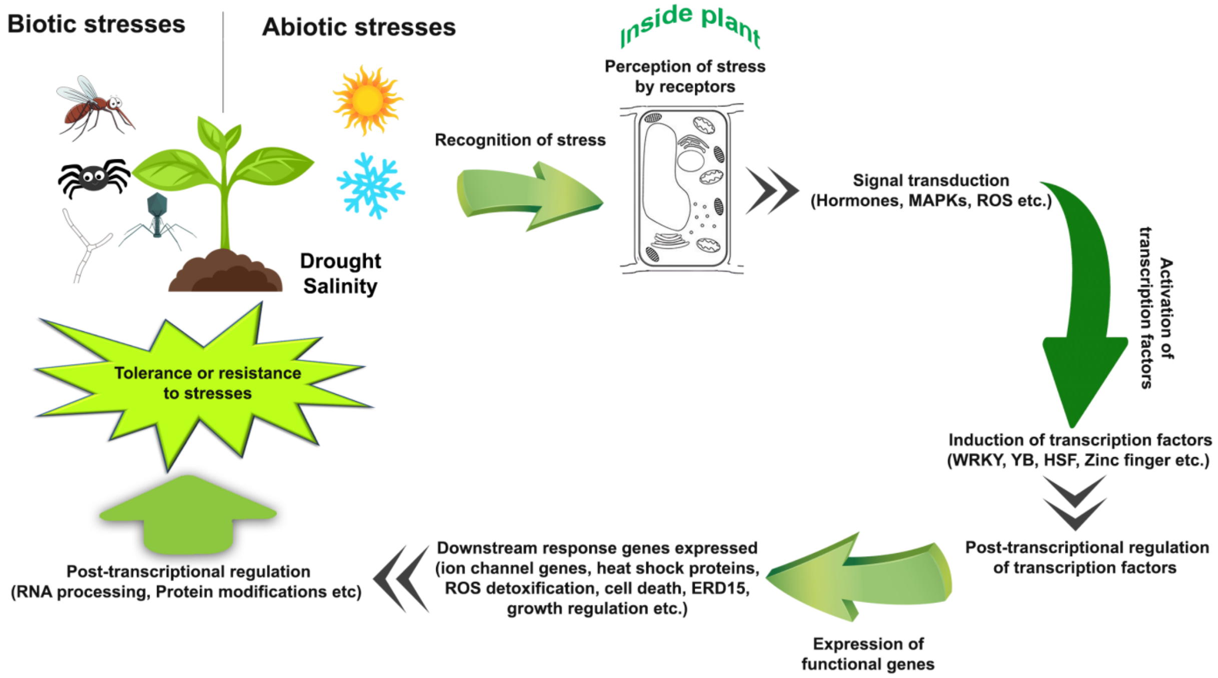

Figure 1 Main activated events in the signal transduction pathway responding to biotic and abiotic stresses. In the model, biotic and/or abiotic stress is recognized and perceived by different receptors. The second step is the activation of transcription factors; this step includes the induction of transcription factors and post-transcriptional regulation of these factors. The third step represents the expression of functional genes and proteins, which includes the downstream response genes expression and the post-transcriptional regulation. These steps eventually lead to the response to stress; this response may result in tolerance or resistance to the stress.

There are studies that have showed a connection between virus infection and heat resistance, however, the relationship between heat resistance and plant immunity remains mostly unknown. Some studies suggested that there are synergistic effects when abiotic and biotic stresses are combined. For example, it is thought that elevated temperature benefit plant viruses by increasing the vectors' availability and weakening the host's resistance to viruses. *Tobacco mosaic virus* (TMV) and *Tomato spotted wilt virus* (TSWV) suppress temperature-dependent resistance in their host; TMV overcame N-gene mediated resistance when temperature exceeded 28°C in tobacco (**Kiraly *et al.*, 2008**), but TSWV required higher temperature to suppress TSW-mediated resistance in pepper (**Moury *et al.*, 1998**). It has been reported that the R protein's, the plant resistance proteins, temperature-induced conformational change is responsible for temperature sensitivity of N-gene of tobacco (**Zhu *et al.*, 2010**).The *Arabidopsis thaliana* ecotype En-2 is known for its resistance to most CaMV strains. However, it has shown weaker level of resistance in the months of June and August when the temperature is high and the day is long (**Adhab et al., 2018**). CaMV inoculation to En-2 plants in the months of June and August accelerated the appearance of disease symptoms on infected plants when compared to symptoms occurrence in tests conducted in the months November – March. This suggests that the higher temperature induces the long-distance transport of CaMV in resistant Arabidopsis plants.

Plant hormones are involved in biotic stress responses such as herbivory and pathogen infection (Bostock 2005; Rostas and Turlings 2008). SA promotes systemic acquired resistance in plants further the infection with pathogen (**Vasyukova and Ozeretskovkaya, 2007**). Virus infection was shown to be inducing both SA and ABA in many studies (**Whenham *et al.*, 1986; Xu *et al.*, 2008**). The manifold roles of plant hormones may explain the effect of virus infection on stress tolerance. In other words, the plant phenotype could be affected by increase in plant hormone concentrations caused by virus infection, and the changes in phenotype may protect plants against environmental stress (**Márquez *et al.*, 2007**).

Viral infection alters vectoring insects' behavior

Host plant traits can be altered by an attack of vector-borne pathogens, and this plant response affects the community of organisms in the host plant as well as the vectoring insects (**Eigenbrode *et al.*, 2002; Stout *et al.*, 2006; Mauck *et al.*, 2010; Mauck *et al.*, 2012; Kersch-Becker and Thaler 2014**). For example, the suitability of host plants for aphid vectors can be altered by plant virus infection (**Kersch-Becker and Thaler, 2014**).

Transmission and dispersal of the majority of plant viruses depends on specific vector species (**Ng and Falk, 2006**). Aphids are the most common vectors of insect-transmissible plant viruses and responsible for transmitting about 50% of insect-transmissible plant viruses (**Nault, 1997; Ng and Perry, 2004**). Many studies indicate that virus-infected plants are more preferable than virus-free plants with respect to the growth rates, longevity and reproduction of the vector (**Blua *et al.*, 1994; Fereres *et al.*, 1999; Jiménez-Martínez *et al.*, 2004; Srinivasan *et al.*, 2008**). Vector behavior is shaped by natural selection in response to virus-induced changes in host plant traits (**Eigenbrode *et al.*, 2002**). The virus and the vector are potentially linked in a mutualistic interaction if vector performance on virus-infected plants also enhances the spread of the virus (**Mauck *et al.*, 2012**). Different viruses alter plant traits and affect their vector species differently (**Eigenbrode *et al.*, 2002; Belliure *et al.*, 2005; Belliure *et al.*, 2008; Hodge and Powell, 2008**). Some viruses produce symptoms on their infected host plants that can be considered as mechanisms by which the virus manipulates its vector through the host plant (**Musser *et al.*, 2003; Belliure *et al.*, 2005; Belliure *et al.*, 2008; Hodge and Powell, 2008**). In some cases, different strains of the same virus can manipulate host plant differently. For example, turnip aphid transmits both two CaMV strains NY8153 and W260 in a semi-persistent manner (**Adhab & Schoelz, 2015**), but it behaves differently towards plants infected with each strain. The aphid vector could recognize plants infected with the two strains and would choose turnip plants infected with W260 over other choice. The result suggests that infection of the host plant with different strains of CaMV has changed the plant traits and made one is more preferable to turnip aphid than the other one (**Adhab *et al.*, 2019**).

The mechanism of viral transmission in many cases determines the way that the virus manipulates its vector and alters the host. There are different kinds of mechanisms to transmit viruses through aphids; these types have been defined based on the inoculation and acquisition periods (**Hull, 2002**). Persistent viruses are acquired and transmitted by their aphid vector after long and continuing feeding periods (hours to days) (**Hull, 2002**). Non-persistent transmission occurs through acquisition access of seconds to a few minutes and usually retention by the vector for no more than a few minutes to hours. On the other hand, semi-persistent viruses, such as *Cauliflower mosaic virus* (CaMV)(*Caulimovirus*: *Caulimoviridae*), are transmitted by vectors following minutes to several hours of vector acquisition access and have a retention time of several hours to a few days (**Schoelz and Adhab, 2020**). It is predicted that persistent and semi-persistent viruses attract vectors and encourage their long-term feeding by promoting plant quality (**Eigenbrode *et al.*, 2002; Alvarez *et al.*, 2007; Jiu *et al.*, 2007; Mauck *et al.*, 2012**). On the other hand, non-persistent viruses will reduce host plant quality to encourage vector dispersal since this kind of virus is rapidly acquired by the vector and can be immediately transmitted to other plants (**Mauck *et al.*, 2010; Mauck *et al.*, 2012**). The pattern of vector behavior when exposed to healthy and infected plants has been described as following. The alate (winged) aphid (the vector) must locate on the infected plants in order to acquire the virus and transmit it to other plants; this attraction to the virus-infected plant is due to either chemical cues (volatiles detectable by vector) or visual cues (color or shape associated with infection) or both. After locating the infected plant, the vector must acquire the virus by feeding on the infected plant. Infection-induced changes in the host defenses, morphological and chemical traits, or host nutritional status may affect the duration and the nature of feeding of the vector. Once the virus is acquired, the vector should locate a susceptible host to transmit the virus. Dispersal in the short term can be affected by pathogen-induced changes in the cues that control host selection behavior; on the other hand, virus dispersal in the long term can be affected by vector performance, which may be affected by infected host. Some viruses can be transmitted multiple times after one acquisition, while some must be re-acquired to be transmitted to multiple hosts by the same individual vector; in the case of re-acquisition needed, the vector will get attracted to the virus-infected plant again to acquire the virus and then will disperse to find another susceptible non-infected host to feed on. In this case, the virus dispersal is successful (**Mauck *et al.*, 2012**).

It has been shown that *Barley yellow dwarf virus* (BYDV) on wheat and *Potato leafroll virus* (PLRV) infecting potato induce changes in the host selection behavior of their aphid vector indirectly (**Eigenbrode *et al.*, 2002; Jiménez-Martínez *et al.*, 2004**). It has been reported that plants infected with these viruses change volatile organic compound profiles that elicit better settling of their non-infective vectors (**Eigenbrode *et al.*, 2002; Jiménez-Martínez *et al.*, 2004**). Different strains of the same virus may cause real diversity in induced plant defenses that could affect vectors in different ways (**Herbers *et al.*, 2000; Kogovšek *et al.*, 2010; Verbeek *et al.*, 2010; Kersch-Becker and Thaler 2014**). Although many studies demonstrate that pathogen infection has the ability to change plant susceptibility to viral vectors, there is no clear known mechanism that explains how vectored viruses can alter plant quality.

Plant volatiles play a key role in virus-aphid-plant interaction

As one of the normal physiological activities, plants release volatile compounds; the quality and amount of those compounds could be affected by biotic and abiotic stressors (**Pare´ and Tumlinson, 1997; Farmer, 2001**). Many plant-arthropod interactions are indeed mediated by volatile cues (**De Moraes *et al.*, 1998, 2001; Verheggen *et al.*, 2008**). The volatile-mediated interactions are very complex. For instance, insect herbivores use information from volatile cues to choose among potential hosts (**De Moraes *et al.*, 2001; Webster *et al.*, 2008**). Aphids usually cause less tissue damage when feeding than chewing herbivores, but this type of feeding induces changes in plant volatiles as well (**de Vos and Jander, 2010**). Aphids feed by inserting stylet into the phloem inducing wound responses in the host plant, which most of the time induce SA signaling pathway but not JA pathway that typically mediates volatile induction to chewing herbivores (**Walling, 2000**).

Many plant viruses depend on insect vector in their spread to other distant hosts (**Ng and Perry, 2004**). It serves the virus spread the best if virus could make the plant more attractive for aphid probing and/or suitable for long-term feeding. Visual and chemical cues from infected plants generally attract aphids settling. Because persistent viruses require relatively long uptake periods, they either do not change or they make the plant more attractive for aphid feeding. Unlikely, non-persistent viruses, which need less time to be acquired by aphids, can make plant less attractive for aphids feeding. For example, the persistently transmitted PLRV-infected potato showed better growth of green peach aphids than non-infected potato (**Srinivasan *et al.*, 2008**), and the volatiles from PLRV-infected were more attractive to the same species of aphids (**Werner *et al.*, 2009**). The same plant infected with different viruses may show different volatile compounds. For instance, faba bean *Vicia fabae* infected with *Bean yellow mosaic virus* (BYMV), the non-persistent virus, reduced aphid growth and survival, but same host infected with *Pea enation mosaic virus* (PEMV), the persistent virus, did not affect the growth of the same species of aphid (**Hodge and Powell, 2008**), suggesting that the manner in which virus transmitted affects the way it manipulates the host and alters the volatiles.

Relationships between plant viruses and other microbes

Vectors are also colonized by other entities that play roles in virus transmission nature. For example, endosymbiotic bacteria have been found to produce compounds that affect transmission of plant viruses (**Morin *et al.*, 1999, vandenHeuvel *et al.*, 1994**).

A clear example for the relationships found between plant virus and other entities is the Tobacco mosaic virus (TMV) in nature. This virus is spread mechanically; it enters plants through wounds, so any herbivore can serve as a vector of TMV. In turn, volatile signals are released from wounds in plants in order to cue neighboring hosts to trigger a response that can be general resistance to any pathogen including bacteria. It has been shown that wound-response volatile signals increased the reproduction and cell-to-cell movement capacity of TMV, which enhances TMV transmission (**Dorokhov *et al*,. 2013, Gutiérrez *et al.*, 2013**).

Bacterial endosymbiont of the vector have been found to play a role in the interaction process. This interaction involve volatile signals and subviral satellites of geminiviruses. For example, tobacco plants are not the preferred host for whiteflies; however, infection of tobacco with the geminvirus *Tomato yellow leaf curl China virus* (TYLCCV) can improve the host quality to make it more attractive to whiteflies (**Zhang *et al.*, 2012, Luan *et al.*, 2013**).

Another example of the interaction between plant virus and other entities is the case of white clover and *White clover mosaic virus* (WClMV). When white clover is infected with WClMV, it produces volatiles (like β-carophyllene) and becomes less attractive to fungus gnats (**vanMolken *et al.*, 2012**). The infection of wild gourds with ZYMV reduces the attraction to beetles that transmit *Erwinia trachiephila* (a destructive pathogen on gourds); this may protect the plants from the infection with the Erwinia (**Shapiro et al., 2013**).

From the above examples, we conclude that interactions between plant viruses and the environment include many other players, including arthropods, fungi, bacteria and other plants. Virus infection may be beneficial to the host by protecting it from infection with other pathogens or from arthropods. More research in this direction is required to understand the relationship between plant viruses and the environment.

CONCLUSION REMARKS

Plant viruses use the host plant resources to support their own survival and spread, so that, in agricultural systems, they are normally considered harmful to the host plant. But, the virus-host relationship is much more complex.

Plant hormones get affected by the virus infection. Several viral-host components interactions involved phytohormone pathways have been identified recently providing some explanation for how viruses manipulate phytohormone regulatory systems to serve the virus development within the host cell. Also, virus infection can be an advantage to the infected host. In other words, virus infection can provide infected-host with tolerance against some biotic and abiotic stresses. Doing more research to understand the role of virus-infection in phytohormone changes in host plants is important to provide more information towards understanding the mechanism of virus-host relationships and interactions. Also, proving that virus-infection provides tolerance to stresses has its own impact on agriculture because it changes the view about plant virus infection in the field. It might be a valid strategy to fight drought and high temperature problems in the future. Understanding the way in which virus control phytohormones provide researchers with ideas that could be used or applied with other strategies to find a solution for drought problems around the world.

Not only virus infection changes the plant hormone pathways in infected plants, but also volatile compounds profile gets affected by the infection as well. Virus infection alters some plant organic compounds such us volatiles, some of which are involved in plant indirect defense. Research suggests that the virus uses this technique to support its survival and spread. For example, virus infected-plants release different volatiles than healthy plants do. Most of the time, there are some attractive volatiles coming out of the infected plants (**Eigenbrode *et al.*, 2002, Alvarez *et al.*, 2007; Jiu *et al.*, 2007; Mauck *et al.*, 2012**). Researchers have tried to determine which volatile attracts the vector the most. Some studies have shown certain volatiles serve as arrestants to virus vectors; for example, β-pinene, that are released from PLRV-infected potato, is a mild arrestant to green peach aphids, the PLRV vector (**Ngumbi *et al.*, 2007**). This is an important finding because by knowing the chemical cues that attract aphids, we can apply it in the field by using similar synthesized chemicals to mimic the same action to trick and trap aphids in the field and serve agriculture. In other words, we can make aphids traps using those chemicals and spread them around the field away from main crop to attract aphids and protect the main crop from the damage occurs by aphid feeding. Also, such results will help researchers understanding the mechanism by which the virus alters plant traits and attract vectors. The agriculture industry may benefit from those results as well because it provides a chance for determining a specific chemical that could be commercially used instead of pesticides to decrease the pest community and loses in crops. Also, by knowing the specific volatiles attracting pest, we can choose and recommend the non-crop hosts that could produce good amount of the same volatile to be planted around the crop to trap pests and protect the main crop. In addition, the findings we provide may be applied on other viruses that manipulate hosts the same way CaMV does. Consequently, we provide a database for future research by establishing the knowledge about how virus alters phytohormones and volatiles in the host. Taking together, this review provides an important basic knowledge about how biotic and abiotic stress affects the interaction among virus, vector and the host plant; this knowledge could open the gate to understand the effect of multi-stress effect on the host plant in future studies through recognizing the necessity for plants to have an integrated system of defense against different threats. Also, it increases the cooperation among plant pathologists, ecologists and entomologists.

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