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Do plant viruses benefit their insect vectors?

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Abstract
The relationship between insect vectors, the pathogens, and the plant are more complex. The attempt to unravel the complexity elucidated much theory. In this study, we would like to analyze the various types of interactions between the viruses and their insects' vectors. The study reviewed the various work done on the relationship between the insect vectors and their plant viruses, to understand that do the viruses during the course of evolution offered any benefit for their vectors? The study clearly states that the evolved relationship between the plant viruses and its insect vectors can be classified into three categories *viz.*, Mutualistic, Non-mutualistic (Neutral) and Antagonistic. In mutualistic interaction, the viruses offer some benefits to their insect vectors for their own benefits. This was mostly observed in persistent transmission type where the virus should be in the insect system for a longer period. The non-mutualistic interaction was observed in semi- persistent transmission, here the relationship between virus and vectors are going to be lost for few hours. So the manipulation of the vector behaviour occurred, without any cost effective or deleterious effect to the vectors. The antagonistic relationships were normally observed in non-persistent transmission. Here, the virus has to be transferred immediately to the host plant, so the virus manipulates the vector, which is cost-effective to the vectors. We would like to conclude the answer to the question, "Do plant viruses benefit their insect vectors?" as, yes it is! Perhaps if the relationship is the long lasting (Persistent).

Keywords: Screening, germplasms/varieties, *H. armigera* and chickpea

Introduction
Arthropods are the most abundant species on the Earth and act as important vectors for many diseases of both animal and plants [1]. They are vectors for thousands of pathogens including bacteria, fungus, viruses, phytoplasma, trypanosomes, and plasmodia. The manipulation of vector biology and behaviour by the pathogen in the animal system were well studied and documented [2].

In contrast to animal pathosystems, plant pathosystems have been less well studied for evidence of host or vector manipulation by pathogens. While animal pathogens can alter the behaviour of both hosts and vectors in ways that increase the frequency of host-host or host-vector encounters, in plant pathosystems the host is sessile, so the potential for behavioural manipulation is restricted to the vector, the mobile component in these systems. The observed changes in vector behaviour include those related to pathogen transmission [3].

Plant viruses are viruses that affect plants. Like all other viruses, plant viruses are obligate intracellular parasites that do not have the molecular machinery to replicate without a host. Plant viruses are pathogenic to higher plants [4]. The first virus to be discovered was Tobacco mosaic virus (TMV). This and other viruses cause an estimated US$60 billion loss in crop yields worldwide each year. Plant viruses are grouped into 73 genera and 49 families [5].

The first plant viruses causing disease is "mosaic disease" by Martinus Beijerinck in 1898, he referred it as "contagium vivum fluidum", thus become "virus" in the modern terminology [6]. In 1939, Holmes published a classification list of 129 plant viruses. This was expanded and in 1999 there were 977 officially recognized, and some provisional, plant virus species [7].

More recently virus research has been focused on understanding the genetics and molecular biology of plant virus genomes, with a particular interest in determining how the virus can replicate, move and infect plants [8]. Understanding the virus genetics and protein functions have been used to explore the potential for commercial use by biotechnology companies. In particular, viral-derived sequences have been used to provide an understanding of novel forms of resistance. The recent boom in technology allowing humans to manipulate plant viruses may provide new strategies for production of value-added proteins in plants [9].

Plant Viruses Transmission

The transmission is the most important aspects of the successful diseases establishment. The plant viruses have to employ some of the strategies to tackle the challenges from the plants to move from one plant to another plant or from one plant cell to another cell inside the system [10]. Plants are stationary, so plant to plant transmission was carried out by vectors. Wherein to move from cell to cell, the plant viruses were evolved to use some of the plant's own mechanisms, for example, especially RNA viruses the plants have some specialized mechanisms of transporting their own mRNA from cell to cell *via* plasmodesmata, the virions are chosen to move through plasmodesmata in order to avoid the barrier from solid cell [11]. One of the other challenges faced by the plant viruses was the plant defense *i.e* production of siRNA in response to dsRNA of the viruses, most of the plant RNA viruses code for a suppressor protein for the siRNA response of the plants [12]. So the 50% of the challenges of the viruses transmission was met out by vectors, some of the feeding habits of the vectors and some coding proteins from the vectors actually protect and help for the effective transmission of the viruses [13]. Plant viruses are typically spread by two common mechanisms: horizontal transmission and vertical transmission [14]. The vertical transmission is *via* the seeding material (seed and pollen), where in the horizontal transmission includes transmission through sap, insect vectors, nematodes, and Plasmodiophorids [15, 16].

The plant viruses that are transmitted by insect vectors are classified as nonpersistent, semi- persistent, persistent. In non-persistent transmission, viruses become attached to the distal tip of the stylet of the insect and to the next plant it feeds on; it inoculates it with the virus. Semi-persistent viral transmission involves the virus entering the foregut of the insect. Those viruses that manage to pass through the gut into the hemolymph and then to the salivary glands are known as persistent There are two sub-classes of persistent viruses: propagative and circulative. Propagative viruses are able to replicate in both the plant and the insect (and may have originally been insect viruses), whereas circulative can't replicate [17].

The important challenge of the circulative viruses was to overcome the insect defense system; it should not elicit the defense signals. For this matter the helping hand was from the other partner living inside the insect system i.e, the endosymbionts, especially in aphids the chaperone protein symbionin was produced by bacterial symbionts which protect the circulative viruses inside the insect body [18]. Many plant viruses encode within their genome polypeptides with domains essential for transmission by insects. In non-persistent and semi-persistent viruses, these domains are in the coat protein and another protein is known as the helper component. The helper component will bind to the specific domain of the coat protein, and then the insect mouthparts creating a bridge. In persistent propagative viruses, such as tomato spotted wilt virus (TSWV), there is often a lipid coat surrounding the proteins that are not seen in other classes of plant viruses. In the case of TSWV, 2 viral proteins are expressed in this lipid envelope. It has been proposed that the viruses bind via these proteins and are then taken into the insect cell by receptor-mediated endocytosis [13].

The plant virus family that are transmitted by insect vectors and the type of their classification are given in table.1.

Table 1: Plant virus genera organised according to transmission mode by insects (Modified from Gray *et al.* [1].

Nonpersistent Noncirculative	Semipersistent Noncirculative	Persistent Circulative	Persistent Propagative
Alfamovirus	Badnavirus	Begmovirus	Tospovirus
Carlavirus	Caulimovirus	Curtovirus	Marafivirus
Cucumovirus	Closterovirus	Mastrevirus	Phytoreovirus
Fabavirus	Sequivirus	Enamovirus	Fijivirus
Machlomovirus	Trichovirus	Nanavirus	Oryzavirus
Macluravirus	Waikavirus	Umbravirus	Phytorhabdovirus
Potevirus		Bromovirus	Cytorhabdovirus
Potyvirus		Carmovirus	Nucleorhabdovirus
		Comovirus	Tenuivirus
		Sobemovirus Tymovirus	

Insect and plant virus relationship can be grouped into following 3 major categories.

1. Mutualistic

In this kind of relation, it is beneficial for both virus and vectors. This can be further divided into two categories *viz*., direct benefit and indirect benefit. In this mutual interaction, the plant viruses offer the number of benefit to the host.

1.1 Viruses Improved the Vectors Immune

Medeiros *et al*. [13], reported that the plant virus "Tospovirus" on tomato plant activate the immune system of its main insect vector, *Frankiniella occidentalis* Pergande. Tospoviruses have the ability to infect plants and their insect vectors. Tomato spotted wilt virus (TSWV), the type species in the Tospovirus genus, infects its most important insect vector, *F. occidentalis*, the western flower thrips (WFT) [19]. However, no detrimental effects on the life cycle or cytopathological changes have been reported in the WFT after TSWV infection, and relatively few viral particles can be observed even several days after infection. Medeiros [13] hypothesized that TSWV infection triggers an immune response in the WFT. Using subtractive cDNA libraries to probe WFT DNA microarrays, they found that the WFT's immune system is activated by TSWV infection.

The activated genes included (i) those encoding antimicrobial peptides, such as defensin and cecropin; (ii) genes involved in pathogen recognition, such as those encoding lectins; (iii) those encoding receptors that activate the innate immune response, such as Toll-3; and (iv) those encoding members of signal transduction pathways activated by Toll-like receptors, such as JNK kinase [20]. Transcriptional upregulation of these genes after TSWV infection was confirmed by Northern analysis, and the kinetics of the immune response was measured over time. Several of the detected genes were activated at the same time that viral replication was first detected by reverse transcription-PCR [21]. This is the first report of the activation of an insect vector immune response by a plant virus. The results may lead to a better understanding of insects' immune responses against viruses and may help in the future development of novel control strategies against plant viruses, as well as human and animal

viruses transmitted by insect vectors [13].

1.2 Vector reduces the induced defense anti-herbivore product

Belliure *et al.* [4] reported, the increased in developmental rate and survival of *F. occidentalis* on TSWV infected plants. This is mainly due to the negative cross- talk between the anti-pathogen (virus) signaling in the virus infected plants will reduce the anti-herbivore signaling. Therefore in virus infected plants the anti-herbivore components will not be produced and thus help in the vector to perform better in the virus infected host.

Mauck *et al.* [22], have recently investigated the effects of several plant viruses on the quality of host plants as a resource for aphid vectors and on the host-derived cues presented to foraging aphids [23, 28]. The results of these studies suggest an interesting pattern of variation among viruses that differ in their mode of transmission: Mauck *et al.* [22], have documented increased aphid performance on plants infected by persistently transmitted (PT) viruses, which require sustained aphid feeding for effective transmission.

Viruses can make plants more attractive to insects. Recent studies have shown that virus infection can affect the volatile compounds the plants produce, and this, in turn, can attract insects [28]. Belliure *et al.* [4] reported that that infection of plants with Tomato spotted wilt virus (TSWV) increased the developmental rate of and juvenile survival of its vector, Thrips *F. occidentalis* (Pergande), is the main vector of Tomato spotted wilt virus (TSWV). Thrips induce anti-herbivore defenses in pepper plants (*Capsicum annum* L.) that are detrimental to thrips.

However, thrips have a higher growth rate and juvenile survival on pepper plants infected with TSWV. Hence, virus infection seems to reduce anti-herbivore defenses, in agreement with the negative cross-talk between signal ling pathways triggering anti-herbivore defenses and those triggering anti-pathogen defenses. Moreover, the increased juvenile growth rate results in a shorter period of vulnerability of juvenile thrips to predation [5]. In line with this increased performance of juvenile thrips, adult thrips aggregate on virus-infected plants [29], and their offspring will vector the virus when became the adult. Such mechanisms that reduce induced plant defenses against vectors promote the spread of the virus by the vector and may, therefore, have evolved in vector-borne plant viruses and are a mutual relationship with the vectors. In some cases virus-infected plants are better hosts for insects, resulting in increased feeding (Mutual interaction) [29].

1.3 Reduces the competition by reducing the fitness of non-vector insects

Pan *et al.* [30], reported that the performance of the sweet potato whitefly, *Bemisia tabaci* (Gennadius) biotype Q, was altered when raised on pepper infected with Tomato spotted wilt virus (TSWV). TSWV infection reduced *B. tabaci* fecundity and longevity and increased *B. tabaci* developmental time but did not affect the insect's survival or female body lengths. Results demonstrate that TSWV infection can decrease the fitness of *B. tabaci* biotype Q on pepper plants which is a non-vector host for Tospovirus, which indirectly benefit the host vectors by reducing the competition from another herbivore [30].

2. Non- Mutualistic

Under this relation, the solely beneficial organism is the plant virus. At the same time, it did not interfere with the vector biology or fitness attributes. This kind of relation will neither offer benefit nor offer any harmfulness to the vectors. The plant viruses evolved to manipulate the host vector without any negative rewards or positive rewards [31, 32].

In the case of non-mutual interaction, the virus gets to benefit by modifying the host behaviour but which does not make any cost-effective to the vectors [33, 34]. For example, the recent report plant viruses were found to modify insect behaviour more directly. Aphids that had already acquired *Barley yellow dwarf virus* were attracted to uninfected plants, whereas aphids that had not acquired the virus were attracted to infected plants, another striking behaviour modification that can enhance the spread of the virus [23]. In this case, the vector aphid is not having any deleterious effects. So it is a neutral interaction or non-mutual interaction.

Laura *et al.* [35], reported that the aphid *Rhopalosiphum padi*, after acquiring Barley yellow dwarf virus (BYDV) during in vitro feeding, prefers no infected wheat plants, while non-infective aphids also fed in vitro prefer BYDV-infected plants. The plant viruses alter the volatile components of the plant in such a way that it will attract only the potential vectors which are yet to be acquired with the virus. At the same time, the vectors which are already acquired the viruses are not get attracted. This behavioral change should promote pathogen spread since noninfective vector preference for infected plants will promote acquisition, while infective vector preference for non -infected hosts will promote transmission [35].

3. Antagonistic

This kind of relationship is not beneficial to the insect's vectors. In antagonistic, the plant viruses are getting benefited at the cost of the insect vectors. These plant viruses interfere either directly to the physiology of the vectors or affect the fitness attributes of the vectors. Whereas in some cases, though virus infected plants are poor hosts, it attracted the most, and insects leave quickly after probing the plants. This type of interaction will benefit only the virus at the cost of vectors, so this relationship is an antagonistic interaction [36, 37].

Mauck *et al.* [22] reported the role of host volatiles to attract the insect vectors. Comparatively, only a few studies have documented the odour cues role in the ecology of insect-vectored plant diseases. Change in host-derived volatiles increased the attraction of sand flies, and also another report from Kenya revealed that the malaria parasite *Plasmodium falciparum* infected children attracted considerably more mosquito than the uninfected children are the example for the pathogen altering the host volatile to attract the vectors even though the pathogen was at the non-transmissible stage [38, 39].

Several studies also documented plant pathogens manipulating host odours. For example, the Dutch elm disease causing fungi attract its vectors- bark beetle to the infected trees, by producing pseudo- flowers that emit the volatile compounds imitating the true floral odours, in addition secrete sucrose as the rewards for the foraging insects [40, 41, 42]. There were only a few reports available on the pattern of vector behaviour for the transmission of nonpersistent viruses. The non-persistent viruses are expected to repel the aphids once they acquired the viruses rather encouraging the arrestment and colonization on the infected plants. Few available documents suggested that aphid population growth often was reduced on plants infected by non-persistently transmitted viruses but this study lack how virus infection influences plant chemistry (e.g., volatile and contact cues) or document vector behaviors relating directly to the transmission [43].

Mauck *et al.* [22] found that aphid performance was strongly

reduced in plants infected by the non-persistently transmitted (NPT) Cucumber Mosaic virus (CMV, Cucumovirus, Bromoviridae) which is quickly acquired during aphid feeding and benefits from rapid vector dispersal [23]. Interestingly, aphids exhibited preferential attraction to the olfactory cues of plants infected by both persistently and NPT viruses compared with healthy controls [23, 24, 26, 27, 28] in the case of CMV, this attraction to infected plants, despite their decreased quality as a resource for aphids, appeared to result from the increased emission of the volatile compounds.

Although few studies have been done, there seems to be an interesting correlation between the type of transmission and the quality of the host for insect feeding [35]. Viruses that are transmitted in a non-persistent manner (i.e., the insect acquires and transmits the virus rapidly through simple probing) seem to make the plants poorer hosts, whereas viruses that are transmitted in a persistent manner (i.e., the insect is viruliferous for a long period of time and the virus is usually processed through the gut of the insect) become better quality hosts for insects [22]. This makes sense from the virus-centric viewpoint: if the transmission is rapid it is better for the insect to quickly move off to a new plant, whereas if the transmission requires a stronger insect association increased feeding will increase the chances of proper acquisition [43, 44].

Table 2. Important Plant Viruses and their vectors

Virus taxon	No. of members	Principal vector
No circulative, no persistent		
Caulimovirus	17	Aphids
Alfamovirus	1	Aphids
Carlavirus	55	Aphids
Cucumovirus	3	Aphids
Fabavirus	2	Aphids
Machlomovirus	1	Thrips and Beetles
Macluravirus	2	Aphids
Potyvirus	55	Aphids, Mites, and Mechanical
Potyvirus		
No circulative, semi persistent		
Badnavirus	16	Mealy bug and Leafhoppers
Closterovirus	25	Aphids, Whiteflies and Mealy bug
Nepovirus	39	Nematodes
Sequivirus	2	Aphids
Tobravirus	4	Nematodes
Sequivirus	2	Aphids
Tobravirus	4	Nematodes
Trichovirus	6	Aphids, Mealybug, and mites
Waikavirus	3	Aphids and Leafhoppers

Conclusion

Persistent viruses benefit their vectors

There were many scientific reports documented the benefit offered by the persistent viruses to their vectors. The aphid vectors *R. padi* and *M. persicae* develop more rapidly, produce more offspring, or both, on their respective virus infected hosts [25, 26]. Apterae of each vector species settle preferentially on virus infected host plants, and this discrimination occurs in darkness in the absence of visual cues suggested the definite benefit of the viruses to their insect vectors [24, 25].This discrimination by apterate occurs even when aphids are prevented from contacting the leaf surface, demonstrating they are attracted or arrested by VOC (volatile compounds) in the vicinity of infected hosts [25, 26, 27].

The VOC profile in the headspace of virus-infected plants differs substantially in overall concentration and relative composition compared to virus-free plants [24, 25, 26, 28, 42, 43].

In some of the virus-induced volatiles (VIV) (those produced in higher concentrations by infected potato plants) were electrophysiologically and behaviorally activated *M. persicae*, and a blend of VIV elicited aphid responses [43, 44]. Individual VOC and blends that mimic the VIV in the headspace of BYDV-infected wheat plants elicited a response by *R. padi* [2].

As the disease progresses in each of these crop plants, aphid responses to virus-infected versus virus-free plants shift, with stronger arrestment or attraction occurring during intermediate [28] or later stages of infection suggested the benefit of the viruses to vectors in host finding process [27]. In some cases, for example, PLRV disease, during disease progression the head space VOC profiles varied and accordingly the dynamic phase of the response of the aphid was observed. The non-viruliferous *R. padi* responded preferentially to virus-infected wheat, wherein the viruliferous apterate do not attract towards the diseased plants not [27]. The same way in case of *M. persicae*, the viruliferous were less responsive to host VOC than the nonviruliferous [43, 44].

From the literature review, the evolved relationship between the plant viruses and its insect vectors can be classified into three categories *viz*., Mutualistic, Non-Mutualistic (Neutral) and Antagonistic. In mutualistic interaction, the viruses offer some benefits to their insect vectors for their own benefits. This was mostly observed in Persistent transmission type where the virus should be in the insect system for a longer period. The non- mutualistic interaction was observed in Semi- Persistent transmission, here the relationship between virus and vectors are going to be lost for few hours. So the manipulation of the vector behaviour occurred, without any cost effective or deleterious effect to the vectors. The antagonistic relationships were normally observed in Non-Persistent transmission. Here, the virus has to be transferred immediately to the host plant, so the virus manipulates the vector, which is cost-effective to the vectors. Therefore, we would like to conclude the answer to the question,"Do plant viruses benefit their insect vectors?" as, yes it is! Perhaps if the relationship is long lasting one (Persistent).

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