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REVIEW

Plant-pathogen interactions and transmissions: Unraveling the complex role of pathogen vectors in disease ecology

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Highlights

- Multifaceted roles of vectors in pathogen transmissions.
- Mechanistic insights into transmission pathways.
- Physiological interactions in vector-pathogen-host systems.
- Strategic significance for sustainable plant disease management.

Abstract

Plant–pathogen interactions are complex, multifaceted processes involving various participants, including insect vectors and parasitic plants, that play a crucial role in the spread of plant diseases. This review explores the intricate relationships between plants, pathogens, and insect vectors, emphasizing these interactions' ecological and epidemiological significance. Insect vectors, such as aphids, leafhoppers, whiteflies, and beetles, transmit various plant pathogens, including viruses, bacteria, fungi, and phytoplasmas, through different mechanisms. The transmission mode can be direct or indirect, continuous or discontinuous, depending on the biology of the pathogen and the insect vector. We differentiate between noncirculative and circulative pathogen transmission pathways and describe how pathogen movement within insect bodies influences their ability to spread diseases to new plant hosts. The impacts of these interactions extend beyond agricultural productivity to encompass significant economic losses, environmental challenges, and potential human health risks due to excessive use of chemical controls. Understanding these complex dynamics is essential for designing effective disease management strategies and developing environmentally sustainable control measures. This review synthesizes current knowledge on the transmission mechanisms, types of plant pathogens, and the consequences of insect-mediated disease spread, providing insights crucial for advancing plant protection and integrated pest management practices.

Keywords: phytopathogen–insect interactions, transmission mechanism, insect vector borne diseases, host vulnerability, environmental influences

1. Introduction

Plant–pathogen interactions represent a dynamic and complex network of biological, ecological, and environmental factors. Insect vectors are central to this web of interactions, which play a critical role in the transmission of plant pathogens. These pathogens, viruses, bacteria, fungi, and nematodes, depend on various insect species, such as aphids, whiteflies, thrips, and planthoppers to move between plants, facilitating the onset of diseases that can devastate crops. The relationship between insect vectors and plant pathogens is highly specialized, with many pathogens relying on specific insect species for successful transmission. This coevolved relationship has a significant impact on agricultural productivity and broader ecological processes, as the distribution and abundance of these diseases can reshape ecosystems (Bosque-Pérez and Eigenbrode 2011).

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One of the key drivers of plant disease outbreaks is the transmission of pathogens by insect vectors (Fig. 1), which have both direct and indirect economic impacts. Insect-borne diseases can decimate crop yields, increase agricultural production costs, and disrupt global food supply chains. For instance, the rice tungro spherical virus (RTSV), transmitted by leafhoppers, and the wheat streak mosaic virus (WSMV), vectored by mites, are responsible for major economic losses in Asia and North America, respectively (Eigenbrode *et al.* 2018). The ability of insect vectors to mediate these plant–pathogen interactions is critical to understanding how disease outbreaks can be managed, particularly as agricultural ecosystems face the dual challenges of evolving pathogen resistance and changing environmental conditions (He *et al.* 2016; Lahlali *et al.* 2024). Recent studies have revealed that the relationship between insect vectors and plant pathogens is not passive. In many cases, plant pathogens actively manipulate their insect vectors (Fig. 2) to enhance their own transmission efficiency (Islam *et al.* 2020). For instance, aphids infected with plant viruses often exhibit altered feeding behavior, facilitating faster and more effective transmission of the virus to new host plants (Wamonje *et al.* 2020). Research as demonstrated that aphid-transmitted viruses such as barley yellow dwarf virus (BYDV) modify the feeding behavior of their aphid vectors, causing them to probe more frequently and for longer durations on healthy plants. Similarly, the bacterial pathogen *Candidatus* Liberibacter asiaticus (CLas), which causes citrus greening disease, has been shown to alter the physiology of its insect vector, the Asian citrus psyllid (*Diaphorina citri*), enhancing the fitness and lifespan of the vector while promoting the disease spread (Armijo *et al.* 2016; Pelz-Stelinski and Killiny 2016).

This manipulation extends beyond viral and bacterial pathogens. Fungal as well as bacterial pathogens such as *Xylella fastidiosa*, which causes Pierce's disease in grapevines, are transmitted by sharpshooters and spittlebugs (Armijo *et al.* 2016). The bacteria's spread has devastated vineyards and olive orchards, particularly in southern Europe, where there is currently no known cure. In these cases, the insect vectors not only carry the pathogen but also

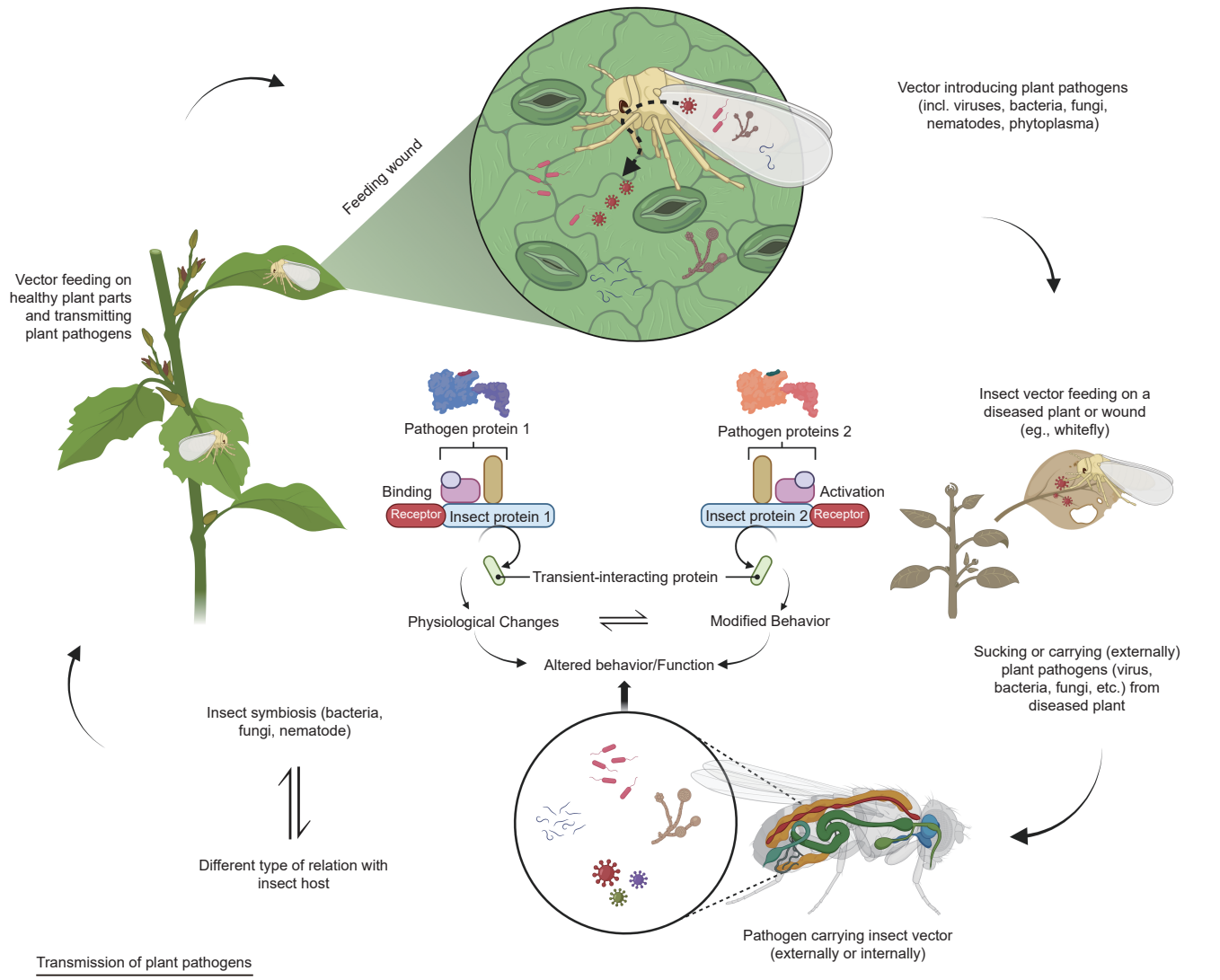


Fig. 1 Flow diagram of transmission of plant pathogens carried by various insect vectors that may acquire it from affected plant part (cut or wound) and transfer to a healthy host plant.

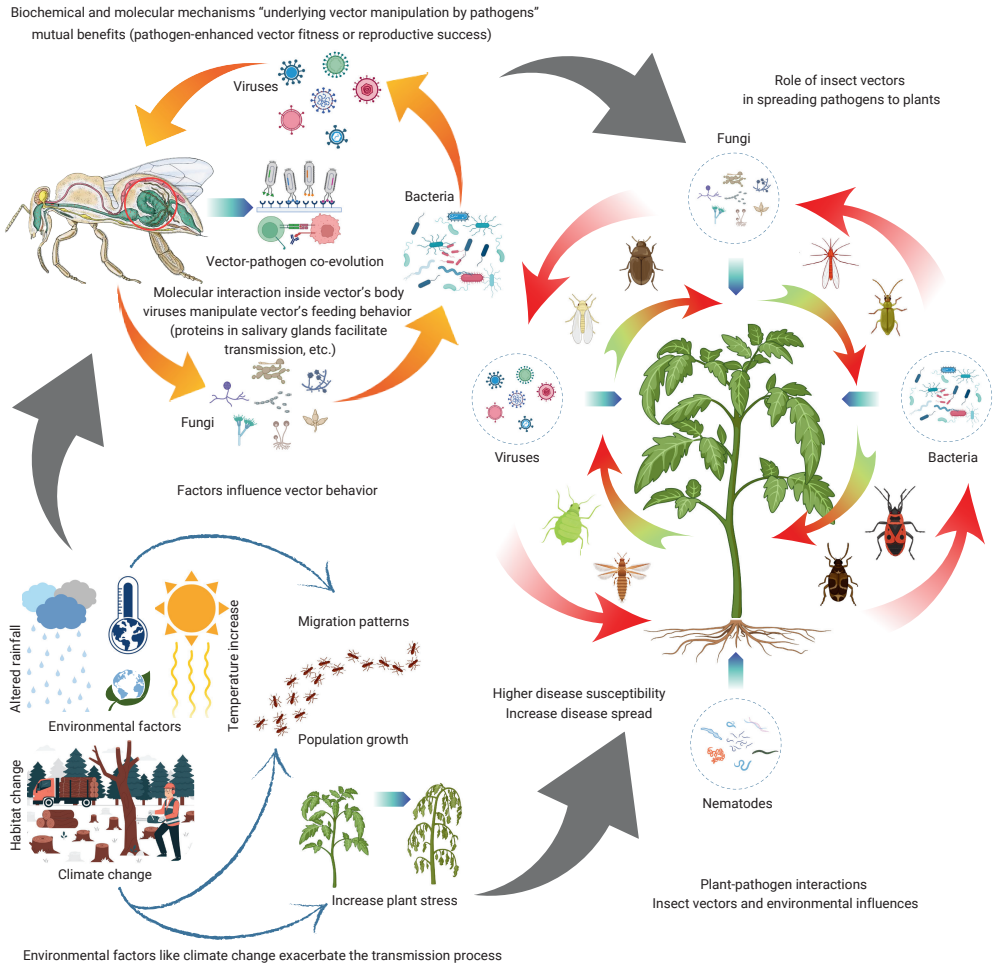


Fig. 2 Solid red arrows indicate direct transmission pathways (e.g., pathogens moving from vectors to plants or between hosts), demonstrating that pathogens influence vector survival and reproduction through pathogen-enhanced vector fitness. Solid orange arrows highlight vector–pathogen co-evolution in reciprocal adaptations (e.g., pathogens manipulating vector behavior and vectors enhancing pathogen fitness), emphasizing that vectors increase pathogen transmission efficiency through vector-driven pathogen spread. Solid blue arrows represent the environmental influence of abiotic and biotic factors affecting vectors or plants (e.g., climate change altering vector migration) and link climate change to vector behavior or plant stress. Solid grey arrows denote bidirectional or cyclic interaction loops (e.g., increased pathogen spread leading to higher vector populations). Dashed arrows illustrate indirect effects and influences (e.g., climate stress indirectly increasing plant susceptibility). The directional flow of arrows points from the influencing factor to the affected element (e.g., pathogens-vectors-plants). Key points of complex interactions among insect vectors, pathogens, and environmental factors of this figure: (1) This loop leads to a self-reinforcing cycle of disease susceptibility, spread, and ecological impact, emphasizing the need to consider these factors in plant disease management and ecological research. Pathogens, including viruses, bacteria, and fungi, enhance vector fitness or reproductive success, creating a symbiotic relationship that benefits both the pathogen and the vector. (2) Pathogens manipulate the vector's biochemistry, often modifying the vector's proteins or influencing feeding behaviors. For instance, viruses and bacteria can alter the insect's behavior, enabling more efficient transmission to the host plant. This co-evolution highlights how vectors are not merely passive carriers but are manipulated at a molecular level to enhance pathogen transmission. (3) A dynamic co-evolutionary relationship between pathogens and their insect vectors, wherein both adapt over time. Pathogens evolve mechanisms to better infect or exploit vectors, while vectors may develop physiological or behavioral adaptations. (4) Climate change and environmental factors play a significant role in modifying vector behavior. Changes in temperature, precipitation, and habitat degradation lead to altered migration patterns, shifts in vector population dynamics, and expansion into new geographical areas. (5) Environmental stressors such as drought, heat, and nutrient limitations can weaken plants, making them more susceptible to infections. Stressed plants often emit different volatile compounds that can attract insect vectors, thus increasing the likelihood of pathogen transmission. (6) Insect vectors carry a variety of pathogens (e.g., viruses, bacteria, fungi) and are capable of infecting multiple plant hosts. This facilitates widespread transmission as vectors move from plant to plant, introducing pathogens to each new host they encounter. (7) The vectors acquire pathogens from infected plants, then transfer these pathogens to healthy plants through their feeding activities, perpetuating a continuous cycle of infection that leads to widespread plant diseases. (8) Environmental stress factors, vector manipulation by pathogens, and vector population dynamics all contribute to the higher susceptibility of plants to diseases. This amplification effect results in greater disease spread and severity across plant populations. (9) As these interactions intensify, there is a broader ecological impact. Plant diseases affect not only individual plant health but also agricultural productivity and biodiversity within ecosystems. (10) Climate change influences vector migration patterns, survival rates, and reproductive cycles. Warmer temperatures often accelerate vector life cycles, increase population densities, and expand geographical ranges, intensifying disease transmission risks but extreme temperatures may also negatively impact vector populations in some cases. (11) Habitat changes resulting from deforestation, urbanization, and extreme weather events disrupt ecological balances, leading to increased interactions between vectors, pathogens, and host plants. This results in an increased risk of plant diseases in newly introduced areas.

assist in creating favorable conditions for its transmission. The economic impact of these plant diseases is immense, with outbreaks of pierce's disease and olive quick decline syndrome (OQDS) causing losses in the millions of euros annually in Europe (Shaw and Catteruccia 2019; Morelli *et al.* 2021). At the molecular and biochemical level, plant–pathogen–insect interactions are highly specialized. Many plant pathogens have evolved mechanisms to exploit the biology of their insect vectors to enhance their transmission (Fig. 1) (Moyano *et al.* 2023). This can include altering the vector's gut and salivary gland proteins, making it easier for the pathogen to pass from the insect to the plant during feeding. For example, the aphid *Myzus persicae* has been shown to harbor potato leafroll virus (PLRV), which manipulates the aphid's feeding behavior to increase transmission rates (Raccah and Fereres 2009). Similar interactions have been observed in other vector–pathogen systems, including the cucumber mosaic virus (CMV), which alters the behavior of its aphid vectors to enhance its spread (Carr *et al.* 2020). These studies underscore the need for a deeper understanding of the biochemical and molecular pathways involved in these interactions to develop effective strategies for disrupting disease transmission (Chiquito-Contreras *et al.* 2024). One of the most significant challenges in managing plant diseases transmitted by insect vectors is the role of climate change in altering vector distribution and abundance (Fig. 2). Changes in global temperatures, precipitation patterns, and the frequency of extreme weather events influence the geographical range and population dynamics of many insect species, affecting plant disease epidemiology (Skendžić *et al.* 2021). For example, warming temperatures have allowed aphid species such as *Rhopalosiphum padi* to expand into previously colder regions of northern Europe, increasing the incidence of BYDV. Similarly, the spread of tomato spotted wilt virus (TSWV) has been exacerbated by the expansion of thrips populations into warmer regions of southern Europe and North America (Batuman *et al.* 2020).

Besides insects, plant pathogens can be vectored by parasitic plants (Fig. 3). The impacts of climate change on plant–pathogen interactions are not limited to shifts in vector populations. While climate change has generally expanded the range of many insect vectors, extreme temperatures may also negatively impact vector populations in some cases, leading to larger populations and more frequent disease outbreaks (Skendžić *et al.* 2021). Moreover, stressed plants, more common in warmer or drier climates, may be more susceptible to infection, creating a feedback loop that exacerbates disease pressure in agricultural systems. Research has shown that the interaction between environmental stressors and plant–pathogen–vector dynamics can intensify the spread of diseases in both

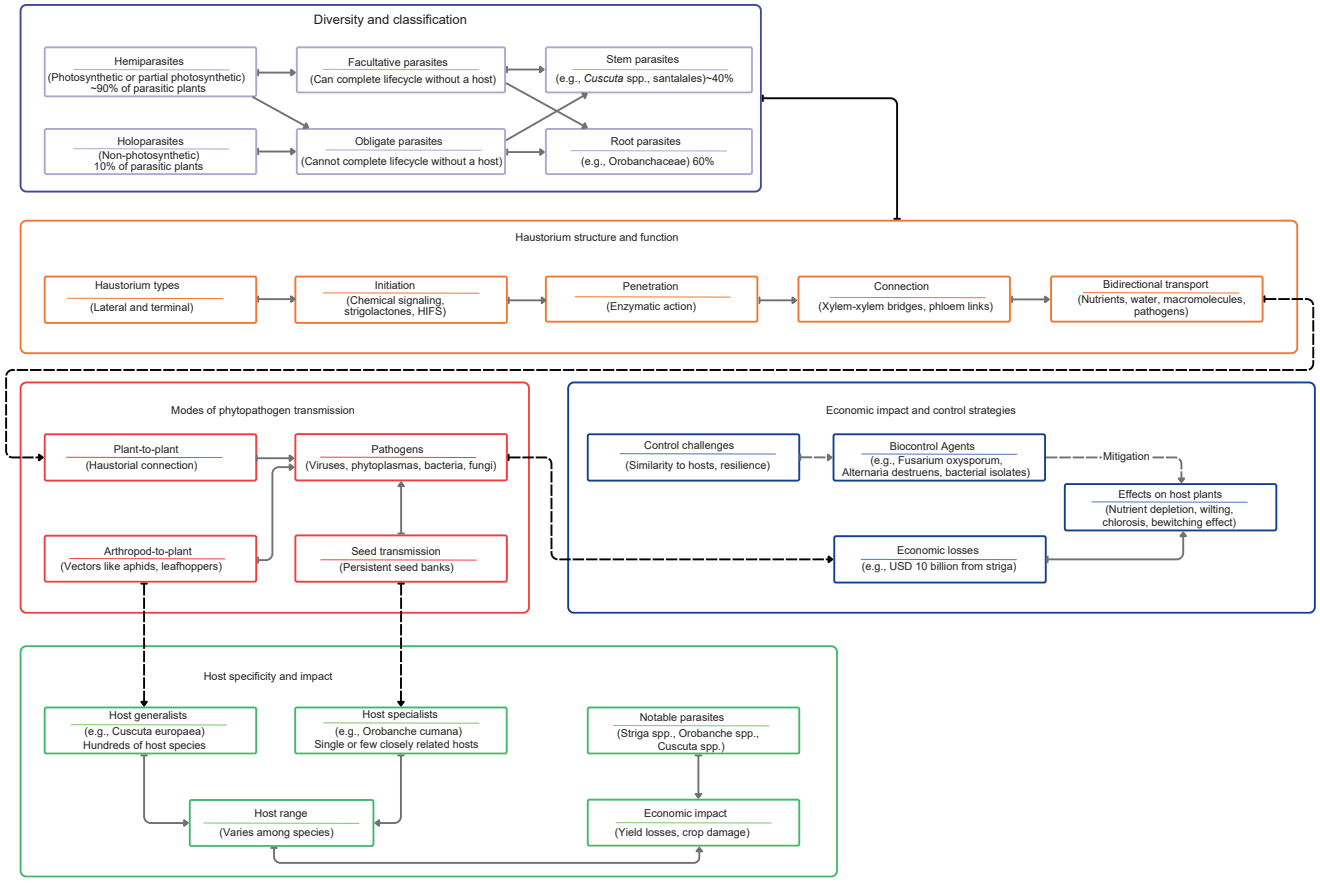


Fig. 3 Multi-layered flowchart that integrates the key components of parasitic plants, emphasizing with key aspects of diversity and classification of parasitic plants, host specificity and impact, haustorium structure and function, modes of phytopathogen transmission and economic impact and control.

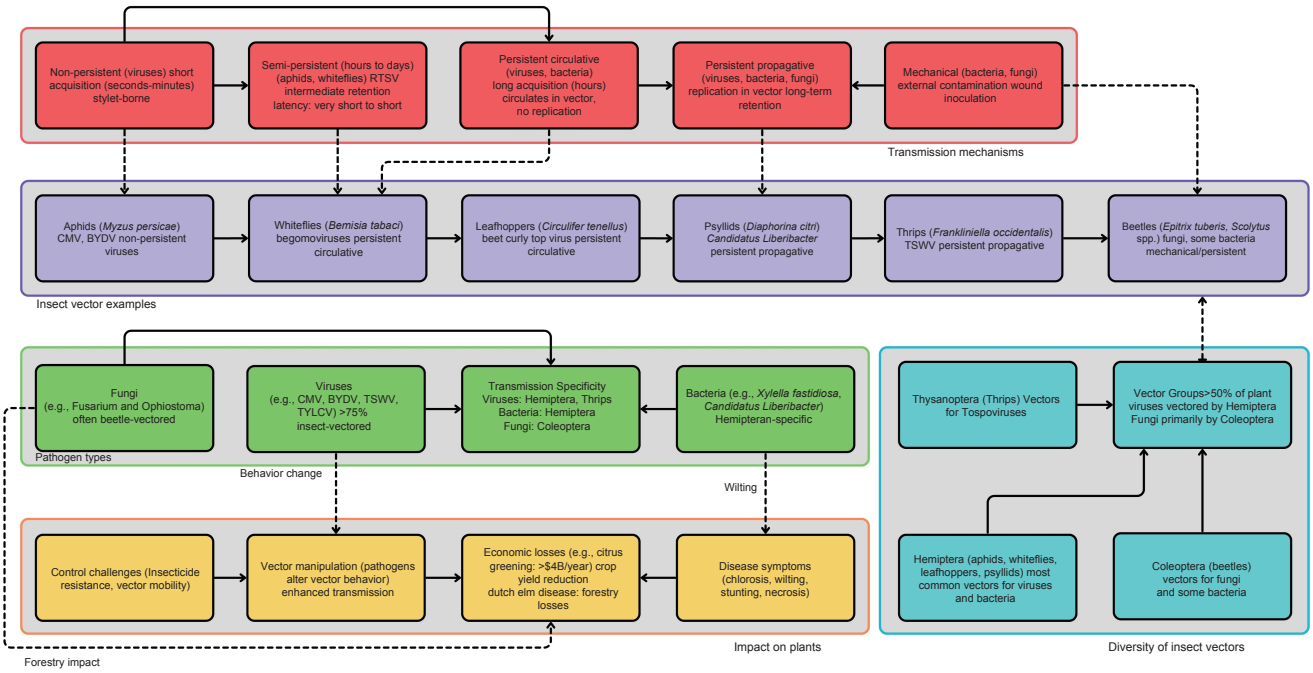


Fig. 4 Relationships between insect vectors and plant pathogens (viruses, bacteria, fungi, etc.), elaborating transmission mechanisms, vector–pathogen types and interactions, economic impacts, and diversity. It highlights specific examples (e.g., aphids transmitting viruses, bark beetles spreading fungi) and emphasizes quantitative data on crop losses.

cultivated and wild plants (Singh *et al.* 2023). Understanding how climate change will influence the future dynamics of plant–pathogen–insect interactions is critical for developing sustainable disease management strategies. Predictive models that integrate climate variables, vector biology, and pathogen dynamics are essential for forecasting disease risks in a changing environment (Peters *et al.* 2020). Moreover, advances in genetic and biochemical tools, such as gene editing and ribonucleic acid interference (RNAi), offer new possibilities for controlling vector populations or interrupting pathogen transmission at the molecular level (Ratcliffe *et al.* 2022). These technologies, combined with traditional integrated pest management approaches, hold promise for reducing the burden of vector-borne plant diseases in the face of global climate change (Holt *et al.* 2020).

Insect vectors are not merely passive carriers of plant pathogens but active participants in the complex interactions that govern plant disease transmission (Fig. 4). The coevolutionary relationships between plants, pathogens, and their insect vectors have profound implications for disease ecology, particularly in a changing climate (Gougherty and Davies 2021). As research continues to uncover the molecular and ecological mechanisms underlying these interactions, it is clear that integrated approaches to pest and disease management will be necessary to safeguard agricultural productivity and biodiversity. The future of plant disease management lies in developing strategies that consider the dynamic interplay among plants, pathogens, and insect vectors, while also addressing the environmental challenges of climate change. Addressing these challenges will require interdisciplinary collaboration and innovative solutions that integrate molecular, ecological, and environmental perspectives (Schmidt *et al.* 2021).

2. Overview of plant pathogens and insects vectors

Plants cover many of the earth's surface, including trees, grasses, and flowers. They are vital in maintaining ecosystem balance and providing food for animals and humans. Additionally, plants, whether in the wild or cultivated, act as potent biofertilizers for the soil. When they decompose, they enrich the soil with essential organic matter (Usman *et al.* 2014). Consequently, taking care of plants is a crucial and ongoing responsibility. The study of plant pathogens falls under the interdisciplinary fields of biology. Plant pathology is dedicated to understanding and mitigating plant diseases from biotic and abiotic origins. Biotic diseases result from phytopathogenic microbes that cause infections and later spread diseases in hosts. Abiotic diseases are caused by various environmental factors (Abdulkhair and Alghuthaymi 2016). An insect vector is an insect that spreads disease causing pathogens in other living organisms or individuals. Plant pathology, the scientific discipline focused on studying plant diseases and their causes, is essential for humans' and animals' economic and hygienic well-being. Phytopathogens can be categorized into two types: biotic factors, which include all microbes and parasitic plants, and abiotic factors, encompassing environmental elements. Plant pathology is closely linked to other sciences, including entomology, bacteriology, mycology, virology and weed science (Agrios 2008). These interdisciplinary connections are essential due to the detrimental impacts of insects, bacteria, fungi, viruses/viroids, and weeds on plants (Oliver 2024). Insect vectors

are responsible for the majority of plant virus transmissions (Appendix A). Insects that feed by sucking and those that chew or bite may both spread viruses. Both "mechanical and biological" modes of transmission are possible. The latter scenario describes an association between a particular insect and a viral infection. Insects known to spread a particular virus are known as vectors for that virus. Mechanical transmission occurs when an insect carries a pathogen on its body or digestive tract (Agrios 2008). An insect vector is an organism with the ability to transfer pathogens from one host to another. In the context of plant diseases, insects that can acquire plant pathogens, while feeding on diseased plants or through contact and then transmit it to healthy plants are referred to as insect vectors. Examples include *Aphis craccivora*, *Koch*, *Cestius phycitis*, bees, wasps, flies, ants and some other species of aphids (Ziegler-Graff and Brault 2008).

Vectors are responsible for introducing pathogens like bacteria or viruses into plants (Fig. 4), causing infections. Insects, mites, and nematodes serve as vectors, facilitating the movement of plant pathogens among immobile plants. It is important to note that many insects and arthropods may contain plant pathogens but cannot transmit them to plants, so they are not considered vectors. Mobile vectors are essential for certain significant plant diseases (Wilson *et al.* 2017). Certain insects are carriers (passive) that only pick up diseases externally on their bodies, usually on their legs or mouthparts, through contact with infected or contaminated plant parts (Fig. 1). Even though some insects can carry the disease, they cannot actively spread it to a new host in a way that causes infection. For the pathogen to establish itself in the new host, such as flies (Weintraub 2012) or beetles (fungi), it typically requires an additional factor, such as entering a wound or being consumed. A real insect vector possesses the capacity to actively spread the infection, frequently through internal harboring and growth.

2.1. Pathogen carrying vectors and their role in transmitting plant diseases

Plant pathogens are transmitted through a variety of pathways, including contamination *via* soil, direct contact between infected and healthy plants, and other biological agents such as insects and mites. While mechanical transmission and soil-borne pollution contribute to the spread of plant diseases, these pathways account for only a small percentage of infections (Agrios 2008). In contrast, insects and other arthropods, including mites, dominate in transmitting the most economically significant plant diseases. The importance of insect-mediated transmission lies in the frequency of occurrence and the efficiency with which these insects spread pathogens to susceptible hosts (Van Bogaert 2016). Insect vectors are classified into two main functional groups based on their feeding mechanisms: chewing insects and piercing-sucking insects. These two feeding modes determine the pathogens that vectors can transmit and influence the nature of their interactions with the plant (Krenn 2019). Chewing insects, such as beetles (Coleoptera), grasshoppers (Orthoptera), and earwigs (Dermaptera), cause physical damage to plant tissues and may transmit fungal spores or bacteria through open wounds. However, their role in disease transmission is generally secondary to piercing-sucking insects. Piercing-sucking insects such as aphids, leafhoppers, whiteflies, and mealybugs have specialized mouthparts that penetrate plant tissues to feed on phloem or xylem sap, making them highly effective vectors of plant pathogens (Chandi *et al.* 2018).

Hemiptera plays a particularly significant role among the many insect orders involved in pathogen transmission. This group includes vectors such as aphids, leafhoppers, and whiteflies, responsible for spreading numerous viral, bacterial, and phytoplasmic diseases. Within Hemiptera, species in the suborder Homoptera are considered especially important in plant disease transmission. Approximately 90% of all known insect-borne plant diseases are transmitted by Homoptera insects, underscoring their central role in agricultural disease outbreaks. Aphids, for example, are the primary vectors for plant viruses like BYDV and potato virus Y (PVY), while leafhoppers spread phytoplasma diseases such as aster yellows and rice tungro (Wilson *et al.* 2019). Whiteflies, including *Bemisia tabaci*, are responsible for transmitting begomoviruses like tomato yellow leaf curl virus (TYLCV), a significant threat to tomato production worldwide (Cathrin and Ghanim 2014). The ability of these vectors to transmit pathogens efficiently depends on both their biology and feeding behavior. Piercing-sucking insects introduce pathogens directly into the vascular system of plants, bypassing natural defenses and enabling rapid systemic infection (Nadarasah and Stavrinides 2011). For example, aphids transmit non-persistent viruses, which attach to the stylets of their mouthparts and are released into the phloem as the aphid probes different plants. Leafhoppers and planthoppers, on the other hand, transmit persistent viruses and phytoplasmas, which can circulate within the vector's body before being delivered to the plant during feeding (Mitchell *et al.* 2018). This circulative mode of transmission allows pathogens to persist within the insect vector for extended periods, increasing the likelihood of transmission to multiple plants over time (Mauck *et al.* 2012).

In addition to viruses, bacterial pathogens such as CLas and fungal pathogens like *X. fastidiosa* are also transmitted by Hemipteran vectors. The transmission of these pathogens involves complex biochemical interactions between the pathogen and the insect vector (Fig. 2) (Kruse *et al.* 2019). For example, CLas alters the behavior and physiology of the *D. citri*, enhancing vector fitness and promoting further disease spread (Pelz-Stelinski and Killiny 2016). These interactions underscore the mutualistic nature of some vector–pathogen relationships, where the pathogen benefits from the vector's mobility while the vector gains improved survival or reproductive success (Kruse 2019). Insects from other orders, such as Thysanoptera (thrips) and Coleoptera (beetles), also contribute to plant disease transmission, although to a lesser extent than Hemiptera. Thrips are significant vectors for Tospoviruses, including TSWV, a globally destructive pathogen affecting over 1,000 plant species (Rotenberg *et al.* 2015). TSWV is transmitted by at least 10 thrips species, with *Frankliniella occidentalis* (western

flower thrips) being the most efficient vector (Kumar *et al.* 2016). In contrast, beetles, particularly those in the family Curculionidae (e.g., weevils), are primarily associated with the transmission of fungal spores and bacterial pathogens. Their feeding or boring activities facilitate fungal infections (e.g., Dutch elm disease) and bacterial colonization leaving plants vulnerable to secondary infections (Balla *et al.* 2021). The significance of insect vectors in plant disease ecology extends beyond pathogen transmission. In many cases, vectors also influence the epidemiology and evolution of plant diseases (Eigenbrode *et al.* 2018). For example, viral infections can alter vector behavior, making the insect more likely to spread the pathogen. Research has shown that some viruses manipulate aphid feeding behavior, causing them to move between plants more frequently, increasing transmission efficiency (Mauck *et al.* 2012). Similarly, specific bacterial and fungal pathogens modify vector physiology to enhance reproductive success, resulting in larger vector populations and a greater risk of disease outbreaks (Luan *et al.* 2014).

2.2. Vectors interaction and transmission of plant pathogens

Insect vector's role in the transmission of plant pathogens is significantly impacting agricultural productivity and global food security. These complex relationships involve various feeding mechanisms, biotic interactions, and environmental factors (West 2014). A plant disease begins with a pathogen's invasion and multiplicates inside the plant's tissue. The most common ways for pathogens to infiltrate a plant are *via* artificial or natural holes like stomata, lenticels, hydathodes, nectar-producing glands, and leaf scars; or by wounds caused by things like insects, nematodes, tools, machinery, and extreme weather conditions (Fig. 1) (Aditya *et al.* 2021). Aphids and mealybugs have needle-like mouthparts that directly penetrate plant phloem (nutrient-rich sap). This permits infections to enter the phloem, where they can be acquired and disseminated. Chewing mouthparts of beetles, grasshoppers, and caterpillars cause superficial bruises on plant tissue (Douglas 2022). Although these wounds mainly harm the plant, they can allow fungal spores or bacterial inoculums to enter. When bees and butterflies pollinate flowers, they collect nectar. Although they are not immediately dangerous, they can mistakenly transport pollen containing fungal spores or bacterial cells, allowing the infection to spread farther (Nagaraja and Rajagopal 2019)

Plant pathogens like viruses bind to the insect's mouth cavity's outer surface. The virus is mechanically transferred without acquisition after feeding on complete plants. Lettuce mosaic virus (LMV), for example, is spread by aphids (Dietzgen *et al.* 2016). Plant wounds are required for pathogens such as bacteria and fungi to enter inside the plant. Through feeding activity, insects can directly transfer these pathogens. For example, the codling moth transmits *Erwinia amylovora*, which is the causal agent of fire blight of apples (Steele and Gibbs 2018). Viruses enter the insect's gut during feeding, grow within its internal organs, and then secreted with the saliva, where they might infect new plants. Citrus tristeza virus (CTV), for instance, is spread by citrus psyllids. The infectivity of pathogens can be chemically influenced by vector saliva altering host immune responses, or mechanically increased by insects, such as cucumber beetle larval feeding damage creating wounds that facilitate the infection and spread of *Fusarium oxysporum* in host roots (Steele and Gibbs 2018). Plant cell walls are broken down by salivary enzymes, which make it easier for pathogens to enter plant tissues. Insects inject toxins into plants while they feed, weakening their defensive systems and enhancing the growth of pathogens. Insect saliva contains sugars and amino acids that plant pathogens like and use to increase their growth (Noman *et al.* 2021). The interaction between the pathogen and the vector insect determines the pathogen's capability to survive and replicate within the vector. Insects' host-seeking and feeding preferences impact their interactions with specific plant species and associated pathogens (Wilson *et al.* 2017).

Humans transfer pathogens through cultivation, grafting, pruning, and transporting diseased plant material. Animals are other common agents that transfer pathogens to plants (West 2014). However, insect vectors play a crucial role in insect transmission from infected to healthy plants. They can cause fatal diseases, badly affect plants, and cause significant yield losses (Parker and Grünwald 2012). This transmission mode has often resulted in some of the worst plant disease epidemics in new areas, with the local host plant stock not adapted to the pathogen and consequently remains highly susceptible (Jones 2021).

Insect vectors are responsible for the majority of plant virus transmissions. Insects that feed by sucking and those that chew or bite may both spread viruses. Both "mechanical and biological" modes of transmission are possible (Sarwar 2020a). The latter scenario describes an association between a particular insect and a viral infection. Insects known to spread a particular virus are known as vectors for that virus. Mechanical transmission occurs externally without pathogen retention or biological processing when an insect carries a pathogen on its body (e.g., aphid stylets) (Boucias and Pendland 2012). When insects feed on plants, they often cause damage, allowing pathogens to enter the plant or root system (Fig. 1). By providing a host for the disease to overwinter in, insects may contribute to its continued existence.

3. Mechanism of transmission (types)

The transmission of plant pathogens by insect vectors is a highly specialized biological process involving molecular interactions between the pathogen, insect vector, and plant host. These interactions ensure successful pathogen acquisition, movement through the vector's body, and eventual delivery into a new plant host (Agrios 2008). At the molecular level, specific proteins (Appendix B), enzymes, and genes facilitate the transmission cycle, while biochemical processes within the vector modulate physiology to enhance pathogen survival and spread. Understanding these molecular mechanisms is critical for designing targeted strategies to disrupt vector-borne disease cycles and protect crops from devastating losses. Certain types of plant

pathogen transmission by vectors are given below.

3.1. Direct and indirect transmission

The transmission of plant diseases can also be classified as direct or indirect, depending on whether the pathogen is transferred directly through planting material or indirectly *via* vectors or environmental factors. Direct transmission occurs when pathogens are transferred to a new host along with the planting material itself. This involves pathogens residing within or on infected propagules such as seeds, cuttings, tubers, bulbs, or sets. When these materials are planted, the pathogen is directly introduced to the new location. For instance, pathogens like *Ralstonia solanacearum*, causing bacterial wilt, are directly transmitted through infected potato tubers used for planting (Dey and Sen 2023). Similarly, many seed-borne pathogens, including certain fungal and bacterial species, are directly transferred *via* contaminated seeds.

Indirect transmission, in contrast, involves pathogen movement to new plants via an external agent or vector, separate from the planting material. These agents can be biotic, such as insects (Fig. 2), animals, or other plants, or abiotic, like wind, water, or contaminated tools and equipment. Insects are particularly significant indirect vectors for numerous plant pathogens, including viruses, bacteria, and fungi (West 2014; Kumar *et al.* 2022). They can carry pathogens externally on their bodies or internally and transmit them while feeding or moving between plants. For example, wind dispersal is a crucial indirect transmission method for many fungal pathogens, such as rust fungi, enabling spore spread over considerable distances and leading to epidemics in cereal crops (Goyal and Prasad 2010).

3.2. Continuous and discontinuous transmission

The spread of plant pathogens can also be described as continuous or discontinuous, focusing on the temporal and geographic pattern of disease occurrence rather than the transfer agent. Continuous transmission refers to the ongoing, localized spread of pathogens within a region where the pathogen is already established or endemic (Whitfield *et al.* 2015). This type of transmission is characterized by the pathogen's sustained presence and propagation within a defined geographic area. Pathogens spread from infected to healthy plants within the same area, often facilitated by local vectors or environmental conditions. For example, TYLCV exhibits continuous transmission in regions where its whitefly vector *B. tabaci* is consistently present, allowing the virus to persist and spread within the crop cycle and between seasons (Czosnek and Ghanim 2012). Continuous transmission is typical in areas with favorable environments that support both the pathogen and its vectors, leading to persistent disease pressure.

Discontinuous transmission, conversely, describes the introduction of pathogens into new, previously unaffected areas, often across geographical barriers. This mode of transmission is usually associated with long-distance movement, frequently facilitated by human activities such as trade in infected plant material or accidental introduction (Stukenbrock and Mcdonald 2008). For example, the emergence of OQDS in Europe, caused by *X. fastidiosa*, is a prime example of discontinuous transmission, resulting from the importation of infected plant material from the America (Raparelli *et al.* 2024). Discontinuous transmission events can lead to sudden disease outbreaks in new regions, often causing severe impacts due to the lack of co-evolved resistance in local plant populations and potentially introducing novel pathogens to new ecosystems. Unlike continuous transmission which is localized and ongoing, discontinuous transmission represents a jump dispersal event leading to disease emergence in a new location (Jones 2009).

3.3. Noncirculative insect-borne plant pathogens

In noncirculative transmission, plant pathogens are acquired and transmitted by insect vectors without crossing cellular barriers and circulating within the insect's body. These pathogens are either retained on the external surfaces (e.g., mouthparts, legs, or body) or localized within specific parts of the insect's foregut or digestive system (Fig. 1). A key characteristic of noncirculative transmission is that these pathogens do not enter the insect's hemolymph and are typically transmitted during brief feeding events (Ratcliffe *et al.* 2022).

Noncirculative pathogens are further classified into two subtypes: non-persistent and semi-persistent.

(1) Non-persistent transmission. This mode is defined by its rapidity; the pathogen is acquired and transmitted rapidly, often within seconds to minutes. Viruses and pathogens attach to stylet receptors located on the insect's mouthparts. The interaction is temporary, and pathogens are released quickly during probing or test bites. The viral particles interact with proteins like peritrophins, ensuring that they remain attached to the foregut and are not lost during feeding. The virus particles attach to the stylets of the insect's mouthparts and are released during probing or biting on a new plant (Rajarapu *et al.* 2021). An example of a non-persistent pathogen is the CMV, transmitted by aphids like *M. persicae*. The virus is lost after a few feedings since no internal replication occurs within the vector. Coat proteins (CPs) on CMV interact with cuticular proteins (CuPs) on the insect's stylet, facilitating rapid transmission. No replication occurs inside the vector. Helper Component-Protease (HC-Pro) bridges interactions between viral particles and insect receptors in Potyviruses, stabilizing virus binding. Viral CPs interact with stylet receptors in non-persistent transmission. Cauliflower mosaic virus (CaMV) uses its P2 protein to bind aphid receptors, ensuring rapid transmission during brief probing events (Mubashir 2022).

(2) Semi-persistent transmission. In contrast to non-persistent transmission, semi-persistent pathogens require a more extended feeding period to be acquired and transmitted. Pathogens are retained in the foregut lining for several hours or days, allowing multiple transmissions from the same vector. Similar to non-persistent transmission, they are retained in the foregut but do not circulate within the vector's body. The defining feature is their retention in the foregut for a longer duration than non-persistent pathogens (Weintraub 2012).

An example is PVY, which is transmitted by aphids in a semi-persistent manner. Though semi-persistent pathogens last longer within the vector than non-persistent ones, they are still eventually lost (Bahrami Kamangar 2021).

3.4. Circulative insect-borne plant pathogens

Circulative transmission is fundamentally different from noncirculative transmission because it involves pathogens crossing cellular barriers and circulating within the insect vector. Circulative pathogens enter the insect vector's digestive tract, cross the intestinal barrier, and circulate within the vector's hemolymph (Reyes Caldas *et al.* 2022). This internal route necessitates longer feeding periods for acquisition and transmission, as they must move from the digestive system to the salivary glands before being introduced to a new host during feeding. Receptor-mediated endocytosis is when pathogens bind to specific receptors on midgut epithelial cells, triggering endocytosis (Fig. 2). GroEL chaperone proteins (from endosymbiotic bacteria) assist in folding viral particles, allowing successful transport across the gut lining (Gray *et al.* 2014).

Circulative pathogens can be divided into two types; Propagative and non-propagative transmission of circulative pathogens.

(1) Propagative transmission. The defining characteristic of propagative transmission is pathogen replication within the insect vector. In propagative mode, the pathogen not only circulates within the vector but also replicates within its tissues. Many bacterial pathogens, such as CLas, responsible for citrus greening, multiply within the insect vector (*D. citri*). Propagative transmission enables pathogens to remain infectious within the vector for extended periods, increasing the likelihood of spreading to new plants. The bacterium produces effector proteins that manipulate vector physiology and immune responses to enhance survival and reproduction (Pelz-Stelinski and Killiny 2016).

(2) Non-propagative transmission. In contrast to propagative transmission, in the non-propagative type, the pathogen circulates within the vector but does not replicate inside it. Many plant viruses, such as BYDV, follow this transmission mode. After being acquired, the virus circulates within the insect's hemolymph and eventually accumulates in the salivary glands, from where it is transmitted to a new host plant (Bosque-Pérez and Eigenbrode 2011). HSP70 (heat shock proteins) facilitate viral survival under stress and assist in the pathogen's movement through insect tissues (Gray *et al.* 2014). Thus, while both propagative and non-propagative circulative pathogens circulate within the insect, replication is the key differentiator.

4. Molecular interactions in non-circulative pathogen transmissions

4.1. Protein–protein binding mechanisms

Non-circulative transmission, characterized by its rapid acquisition and inoculation phases, fundamentally relies on specific protein-protein interactions at the interface of the insect vector and the plant pathogen. At the molecular level, these interactions dictate the efficiency and specificity of pathogen attachment to and detachment from the insect vector's stylet or foregut (Zhou *et al.* 2018). The binding of the pathogen to the insect's stylet or foregut proteins ensures that the pathogen remains attached during initial probing and feeding, although for a transient period (Han 2022). This transient nature is a characteristic of non-circulative transmission, distinguishing it from the persistent interactions observed in circulative transmission modes. This interaction is particular, meaning only certain combinations of viral CPs and insect receptors will result in effective transmission. Understanding these molecular interactions is critical for identifying weak points in the transmission cycle that could be targeted for innovative disease control strategies (Deshoux *et al.* 2018).

4.2. Viral CPs and insect receptors

Viral CPs play a pivotal role in determining the transmission efficiency of non-circulative pathogens. These proteins, exposed on the surface of viral particles, are the primary determinants mediating attachment to insect receptors during the acquisition phase (Whitfield and Rotenberg 2015). The precise molecular architecture of these CPs, including their amino acid sequence and glycosylation patterns, dictates their ability to interact with specific insect receptors. The specificity of these interactions is determined by the compatibility between the viral CPs and the glycoproteins on the insect's stylet or foregut. These glycoproteins proteins act as molecular docking sites, allowing the virus to bind securely to the vector during feeding. These glycoprotein receptors on the insect cuticle are often complex carbohydrates conjugated to proteins, providing a diverse array of potential binding motifs (Heck 2018). The viral CPs recognize and bind to glycoprotein receptors on the insect's stylet or gut surfaces. This interaction ensures that the virus remains attached to the vector for a short period required to infect another plant (Idrees *et al.* 2024). The strength of the interaction between viral CPs and insect receptors (binding affinity) determines the transmission efficiency. Weak binding may result in the pathogen dislodging before transmission, while strong interactions enhance the likelihood of successful infection (Figure 1) (Andret-Link and Fuchs 2005).

4.3. Helper protein mechanisms

In Potyviruses, such as turnip mosaic virus (TuMV), the HC-Pro acts as a molecular bridge, stabilizing the interaction between the viral CPs and the receptor on the aphid stylet (Fernandez-Calvino *et al.* 2010). HC-Pro binds the viral particle and glycoproteins on the stylet, ensuring the virus stays attached during feeding. This interaction ensures the virus is transferred efficiently to plant tissues during the aphid's initial probing behavior. Without HC-Pro, the virus would not bind efficiently to the vector's stylet, reducing the probability of transmission (Jangra *et al.* 2024a). The HC-Pro protein, through its RNA binding and protein interaction domains, is thought to prevent virus aggregation and facilitate virion attachment to the aphid stylet, thereby acting as a true helper component (Pantaleo and Masuta 2022). Glycoproteins serve as attachment points for viruses. Glycoproteins on the cuticle lining the stylet or foregut serve

as virus attachment points. These glycoproteins have specific binding domains that recognize viral CPs. The binding interaction is temporary, as non-persistent viruses are typically transmitted during the first few seconds of feeding or probing (Fig. 2) (Marwal *et al.* 2018).

4.4. Viral protein P2 and stylet receptors

The CaMV is another example of a virus transmitted non-circulatively by aphids. CaMV employs a unique molecular strategy involving the P2 viral protein, which interacts directly with receptors on the aphid's stylet. This mechanism highlights the diversity of molecular strategies used by non-circulative pathogens to exploit insect vectors for transmission (Berthelot *et al.* 2019). The P2 protein is unique to caulimoviruses and is critical for vector transmission, acting as a virion-associated protein that mediates direct binding to aphid stylets. The P2 protein of CaMV also acts as the primary attachment factor, binding to specific receptors on aphid stylets. Unlike the HC-Pro protein in Potyviruses, which serves as a bridge, the P2 protein interacts directly with stylet receptors without the need for a helper component (Webster *et al.* 2018). When an aphid probes the plant's tissues, the virus adheres to the stylet through the P2-stylet interaction and is released into the plant's vascular tissues, leading to cause infection in the plant. This transmission is rapid, occurring within the initial feeding events, often in seconds (Cornara *et al.* 2020).

4.5. Specificity of the P2-stylet interaction

The P2 protein binds selectively to stylet receptors unique to certain aphid species, which limits the transmission of CaMV to aphids that express the compatible receptor proteins. This specificity plays a role in determining the virus's host range and disease distribution. This vector specificity underscores the intricate co-evolutionary relationships between viruses and their insect vectors (Fig. 4), where subtle variations in receptor-ligand compatibility can have significant epidemiological consequences (Verdier *et al.* 2023). Disrupting the P2-stylet interaction, either through genetic modification of aphid vectors or chemical interventions that block the binding sites, could offer a potential strategy for preventing CaMV transmission (Thango 2019). The molecular interactions involved in non-circulative transmission are governed by highly specific protein–protein interactions between viral CPs and insect receptors. These interactions ensure that pathogens remain attached to the vector during feeding and are efficiently transmitted to new host plants (Wilson *et al.* 2019). In Potyviruses, the HC-Pro protein acts as a molecular bridge between the virus and the vector, enhancing transmission efficiency. In contrast, the P2 protein of CaMV directly interacts with aphid stylet receptors, enabling rapid transmission during the initial feeding events (Rajarapu *et al.* 2021). These molecular mechanisms highlight the importance of understanding the precise protein–receptor interactions involved in transmission, as they offer potential targets for disease control strategies. Disrupting these interactions could prevent the spread of non-circulative pathogens, protecting crops from devastating disease outbreaks (Vallad *et al.* 2018). Therefore, targeting these specific protein interactions represents a promising avenue for developing environmentally sound and highly effective disease management tools.

4.6. Molecular variation in non-circulative transmissions

Non-circulative pathogens exhibit a diverse range of molecular strategies to attach to insect vectors, depending on the type of virus and vector species involved. Beyond CPs, a range of viral proteins and non-proteinaceous mechanisms facilitate transient surface binding (Then *et al.* 2021). Some pathogens, such as geminiviruses, rely on interactions with insect CuPs in the foregut, while others may use transient electrostatic interactions with the stylet for temporary adhesion (Navarro *et al.* 2019). This molecular variation reflects the diverse evolutionary pressures and ecological contexts shaping virus–vector interactions. Interactions in which some non-circulative viruses bind weakly to the stylet, allowing rapid detachment and transmission during feeding (Verdier *et al.* 2023). This mechanism enables viruses to spread efficiently between multiple plants during a single feeding session. In some insect vectors, endosymbiotic bacteria play a role in stabilizing the viral particles within the stylet or gut lining. For example, *Buchnera aphidicola*, an aphid endosymbiont, produces chaperone proteins that assist in the retention of viral particles (Chesnais *et al.* 2022). While the precise role of endosymbionts in non-circulative transmission is still being elucidated, they may provide a stabilizing effect enhancing viral persistence on the stylet during acquisition and inoculation.

4.7. Environmental and genetic factors influencing protein–protein binding efficiency

Temperature and environmental conditions, genetic variation in vectors, and co-evolution of pathogens and vectors are the major factors that influence the efficiency of these transient, surface-level protein-protein. These factors can modulate the strength and specificity of viral protein–insect receptor interactions, thereby impacting transmission dynamics. Such as temperature fluctuations may alter the binding affinity between viral CPs and insect receptors (Fig. 2) (Samuel *et al.* 2016; Gale 2020). Elevated temperatures can reduce the stability of glycoprotein interactions, leading to decreased transmission efficiency in warmer climates (Viglietta *et al.* 2021). Different populations of the same insect vector species may express genetically distinct receptor proteins, resulting in variable transmission efficiency across different regions. This is particularly evident in aphids, where genetic polymorphisms influence their ability to transmit specific viral strains (Sanches *et al.* 2023). Pathogens and their insect vectors often undergo co-evolution, with both the pathogen's CPs and the vector's receptors evolving to maintain compatibility. This evolutionary pressure ensures that the virus remains transmissible, even in the presence of environmental changes or genetic mutations (Gray *et al.* 2007). These factors emphasize the delicate and environmentally sensitive nature of transient protein interactions in non-circulative

transmission. Understanding these multifaceted factors is crucial for predicting disease outbreaks and developing robust disease management strategies that account for environmental variability and evolutionary dynamics (Zhou *et al.* 2024).

5. Molecular interactions in circulative pathogen transmission

Circulative transmission, conversely, requires pathogen internalization and systemic vector interactions, contrasting with non-circulative surface binding. And defined by the pathogen's requirement to traverse multiple internal barriers within the insect vector, necessitating a distinct and more intricate array of molecular interactions (Kumaraswamy and Huang 2024). Circulative transmission is a highly complex and specialized mechanism by which pathogens enter the insect vector's digestive system, cross biological barriers, circulate within the hemolymph, and ultimately invade the salivary glands for transmission to a plant host during feeding (Sarwar 2020a). Protein interactions here are not merely for surface attachment but for navigating multiple internal barriers and systems (Appendix B). Unlike non-circulative transmission, circulative pathogens interact with multiple tissues in the insect's body, requiring precise molecular mechanisms to overcome immune responses and navigate internal structures (Ng *et al.* 2015). This type of transmission is essential for certain persistent viruses, bacterial pathogens, and phytoplasmas, making it a critical component in plant–pathogen interactions. Understanding the molecular and biochemical processes involved in circulative transmission offers valuable insights into disease management strategies (Mahanta *et al.* 2023). The molecular composition of circulative transmission involves a cascade of sequential protein-protein interactions at each critical step, distinctly different from the single-step surface interactions in non-circulative transmission, highlighting the greater complexity and specificity required for systemic pathogen spread.

5.1. Gut barrier interaction

The gut barrier is a significant obstacle for pathogens entering the insect vector. Unlike external non-circulative pathogens, circulative pathogens must actively penetrate this barrier. To successfully move from the digestive tract into the hemolymph (insect blood), pathogens must bind to specific receptors on the midgut epithelial cells (Hillyer 2016). The pathogen's ability to cross this barrier involves highly specific receptor-mediated endocytosis and protein–protein interactions that facilitate transport across the midgut lining (Linser *et al.* 2014). This interaction triggers endocytosis, a cellular process by which the cell membrane invaginates and engulfs the pathogen, allowing it to pass into the cytoplasm and cross the midgut barrier. The role of endocytic vesicles starts after internalization; the pathogen is enclosed in endocytic, which transport it across the epithelial cells to release it into the basal lamina for entry into the hemolymph (Eleftherianos *et al.* 2021a). A key molecular interaction in gut barrier crossing is thus receptor-mediated endocytosis. The specificity of these receptor–ligand interactions at the midgut membrane determines the host range of the pathogen within the insect vector, representing the first critical molecular checkpoint in circulative transmission (He *et al.* 2021).

5.2. Gut barrier crossing with chaperone protein assistance

Beyond receptor-mediated endocytosis, chaperone proteins play a crucial role in facilitating pathogen transit across the gut barrier, highlighting a cooperative molecular strategy that is not typically observed in non-circulative transmission (Navarro *et al.* 2019). Among these, GroEL chaperone proteins, produced by endosymbiotic bacteria (e.g., *B. aphidicola* in aphids), assist in the folding, stabilization, and transport of viral particles across the gut lining. GroEL binds to viral CPs, protecting the virus from degradation in the harsh environment of the insect's digestive system and facilitating its successful transit to the hemolymph (Rao *et al.* 2024). These chaperone proteins act as molecular shields, preserving the structural integrity and infectious potential of the pathogen during its transit across the gut. HSP70 plays a vital role in maintaining viral particle stability under stressful conditions within the insect vector (Henderson *et al.* 2006). It binds to viral proteins and prevents them from unfolding or degrading as they cross the gut barrier. Additionally, HSP70 helps the virus evade cellular defenses by modulating protein-folding pathways, ensuring that the pathogen retains its infectious potential as it moves into the hemolymph (Xu *et al.* 2021). The involvement of chaperone proteins underscores the complexity and energy-dependent nature of gut barrier crossing in circulative transmission, contrasting with the passive surface attachment of non-circulative pathogens (Singh *et al.* 2024).

5.3. Carrier protein mediated movement through the hemolymph

Once the pathogen crosses the gut barrier, it enters the hemolymph, the insect's circulatory fluid, where it must navigate through the body to reach the salivary glands. Hemolymph transport, a systemic step absent in non-circulative modes, involves carrier protein binding and immune evasion (Sigle and Hillyer 2018). This movement involves binding to carrier proteins that facilitate transport through the hemolymph while evading the insect's immune responses (Terra *et al.* 2019). Carrier proteins in hemolymph transport are lipophorins and vitellogenins. Lipophorins are lipid-transporting proteins in the insect hemolymph. Pathogens exploit lipophorins by binding them, hitching a ride through the circulatory system to the salivary glands. Vitellogenins are precursor proteins for egg yolk formation, can also bind pathogens, transporting them through the hemolymph. This strategy allows pathogens to avoid free circulation, reducing the likelihood of detection by the insect's immune system (Sheehan *et al.* 2018). Thus, carrier protein interactions are essential for directed hemolymph transport in circulative pathogens.

5.4. Immune evasion mechanisms in hemolymph

During hemolymph transport, active immune evasion becomes a critical necessity for circulative pathogens during

hemolymph transport, contrasting sharply with the rapid, superficial interactions characteristic of non-circulative pathogens. The prolonged presence of circulative pathogens within the hemolymph necessitates robust strategies to counteract the insect's immune defenses (Milgroom 2023). Pathogens employ various sophisticated strategies to evade or actively suppress insect immune responses. Pathogens may engage in molecular mimicry, mimicking insect host molecules to evade immune recognition. For example, some viruses display glycoproteins on their surface that resemble insect cell-surface molecules, effectively camouflaging themselves and preventing recognition as foreign entities by the insect's immune system (Mondino *et al*. 2020). Furthermore, some pathogens directly target and interfere with insect immune signaling pathways, particularly the Toll and Imd (immune deficiency) pathways, which are crucial for producing antimicrobial peptides (AMPs) and initiating other immune responses in insects. Some circulative pathogens have evolved mechanisms to suppress these key immune pathways, ensuring they are not effectively detected and attacked by the insect's antimicrobial defenses (Alejandro *et al*. 2022). Begomoviruses, transmitted by whiteflies, provide a clear example of immune suppression, as they have been shown to actively suppress immune signaling in the insect vector, allowing the virus to circulate relatively freely within the hemolymph without triggering a robust immune response (Ronald and Joe 2018; Eleftherianos *et al.* 2021b). These intricate immune evasion mechanisms are a defining feature of circulative transmission, highlighting the prolonged and intimate interaction between pathogen and vector in this mode of transmission.

5.5. Salivary gland invasion (receptor specificity and systemic spread)

Salivary gland invasion represents the final critical step in circulative transmission. This invasion of salivary glands is a crucial step entirely absent in non-circulative transmission (Rao *et al*. 2024). Once the pathogen reaches the hemolymph, it must invade the salivary glands to ensure transmission during feeding. Salivary glands are highly selective barriers, and pathogens need specific molecular mechanisms to recognize and bind to receptors on gland cells. This invasion process is particularly critical for propagative pathogens that replicating within the vector (Schorderet-Weber *et al*. 2018; Schneider *et al*. 2021). Similar to gut entry, receptor recognition and binding are essential molecular events for salivary gland invasion. Pathogens recognize and bind to specific receptors on the salivary gland epithelial cells, which allows them to selectively invade the gland tissues (Chibly *et al.* 2022). The precise and specific interaction between viral or bacterial surface proteins and salivary gland receptors ultimately determine the efficiency of pathogen accumulation in the salivary glands and, consequently, exhibit the overall efficiency of transmission (Baker *et al*. 2014). Specific receptor interactions are therefore crucial for salivary gland entry, completing the circulative pathway.

5.6 Vector gene regulation and replication in propagative transmission

A key aspect further differentiating circulative transmission, particularly the propagative subtype, is vector gene regulation and pathogen replication within vector cells, a phenomenon absent in other transmission modes. Some propagative pathogens modify the gene expression within the salivary glands to create a more favorable environment for replication and survival (Lin *et al.* 2024). For example, bacterial pathogens may upregulate genes related to nutrient uptake and secretion, ensuring optimal growth within the vector. CLas, a bacterium causing citrus greening, modifies the expression of metabolic and reproductive genes in the *D*. *citri*, enhancing the vector's ability to spread the pathogen effectively (Yan *et al.* 2013). This intricate series of interactions ensures that the pathogen is transmitted efficiently to new plant hosts, highlighting the co-evolutionary relationship between pathogens and their insect vectors (Mondal 2017). In addition to this, vector replication and gene regulation are defining molecular features of propagative circulative transmission. This intricate and bidirectional series of molecular interactions, involving pathogen-mediated manipulation of vector gene expression and cellular processes, ensures that the pathogen is transmitted efficiently to new plant hosts, highlighting the sophisticated and co-evolved relationship between propagative circulative pathogens and their insect vectors (Fig. 2) (Lin *et al.* 2024).

These molecular and biochemical manipulations underscore the complexity of plant–vector–pathogen interactions, where both the pathogen and the vector benefit from the relationship. The transmission of plant pathogens by insect vectors involves multiple pathways, each governed by distinct biological and biochemical processes (Heck 2018). Continuous and discontinuous transmission highlights the natural and human-mediated spread of pathogens, while direct and indirect transmission emphasizes the roles of planting materials and biological agents. The distinction between noncirculative and circulative pathogens provides insight into the molecular mechanisms that enable pathogens to persist within vectors and spread effectively. Understanding these processes at the molecular level is essential for developing targeted disease management strategies, particularly in an era of global trade and climate change (Rao *et al.* 2024).

6. Mechanisms of pathogen-vector interactions and transmissions

6.1. Manipulation of insect vector behavior by pathogens

The ability of plant pathogens to manipulate the behavior and physiology of their insect vectors is a striking example of co-evolution (Fig. 2). These manipulations ensure the pathogen's efficient transmission by altering the vector's feeding behavior, reproduction, and immune responses (Chesnais *et al.* 2020). Such behavioral and physiological modifications allow pathogens to enhance their spread while also benefiting the vector, creating a complex relationship with far-reaching implications for disease epidemiology and control strategies (Ahmad *et al.* 2024).

Pathogen induced changes in vector feeding behavior and physiology In addition to altering feeding behavior, many plant pathogens manipulate their insect vector's physiology and reproductive capacity (Fig. 2). These changes ensure the sustained availability of vectors for pathogen transmission over time and across large areas (Eigenbrode *et al.* 2018). Plant pathogens alter neurotransmitters, hormones, and sensory signals in their insect vectors. This leads to increased feeding, movement, and probing, enhancing pathogen transmission (Moyano *et al.* 2023). At the molecular level, the neurotransmitter dopamine and signaling molecules like serotonin and acetylcholine may be involved in modulating vector-feeding patterns. The pathogens influence these pathways to alter the vector's ability to detect and respond to host plant cues, encouraging behaviors that promote the pathogen's spread (Yang and Chiu 2017). By modulating the dopamine signaling pathway, viruses like the CMV alter the sensory and locomotory behavior of aphids, causing them to increase probing and movement between plants. These behavioral changes ensure the virus is transmitted efficiently from infected to healthy plants (Mauck *et al.* 2012). Short probing behavior is encouraged in non-persistent virus transmission, where the insect briefly taps multiple plants. Longer probes, required for circulative or persistent pathogens, are achieved by suppressing host defenses and altering feeding signals to extend insect feeding on infected plants.

CMV provides an illustrative example of how pathogens manipulate insect vector behavior to their advantage. Infected plants emit altered volatile organic compounds (VOCs), which attract aphids. These VOCs attract more aphids and stimulate exploratory probing behavior, increasing the frequency of brief feedings on multiple plants (Milonas *et al.* 2023). The infected plant, thus, becomes a hub for virus dissemination, enabling rapid spread across a field or ecosystem. Pathogen-altered VOC emissions serve as olfactory cues for insects. Such VOC changes have been linked to increased vector movement and can shift aphid preferences from healthy plants to infected ones, further enhancing pathogen dissemination (Grunseich *et al.* 2019).

Physiological changes in vectors (reproduction, lifespan, immune suppression) Some pathogens boost the reproductive rates of their insect vectors, either by enhancing fecundity or extending the vector's lifespan. These changes are often driven by the modulation of hormonal pathways in the vector (Nijhout and Laub 2018; Force *et al.* 2025). For example, pathogens may increase juvenile hormone production in vectors, stimulating reproduction and delaying senescence. This ensures that the insect population remains high, enhancing the chances of pathogen spread. By suppressing stress responses and reducing metabolic costs, pathogens can prolong the lifespan of vectors, allowing them to feed and transmit pathogens over more extended periods (Lucas 2011). The bacterium, CLas enhances the reproductive success of the *D. citri*). CLas infection modulates the expression of genes involved in reproduction and energy metabolism, leading to increased fecundity and prolonged lifespan of the psyllid vector (Nian *et al.* 2024). This adaptation ensures that the psyllid population remains stable or increases, promoting long-term pathogen transmission. Pathogens such as CLas manipulate gene expression to increase the vector's reproductive output. For example, genes involved in lipid metabolism and reproduction are upregulated, providing the vector with the energy and resources needed for sustained reproduction (Killiny *et al.* 2017).

Pathogens must suppress the insect vector's immune system to survive and reproduce effectively within the vector's body. Immune suppression allows the pathogen to persist without being cleared by the vector's defenses. This is achieved by interfering with the insect immune pathways, particularly the Toll and Imd pathways, which regulate the production of AMPs (Manniello *et al.* 2021; de Angeli Dutra *et al.* 2022). These receptors detect invading pathogens and trigger immune responses. However, some pathogens manipulate pattern recognition receptors (PRRs) to block immune signaling, preventing the insect from recognizing and eliminating the pathogen. Many pathogens reduce the production of AMPs, weakening the vector's immune system and allowing pathogens to survive in the hemolymph or gut tissues (Zhao *et al.* 2023). Begomoviruses suppress the *B. tabaci* immune response by mimicking insect host molecules and disrupting the expression of immune pathway genes. The viruses inhibit the Toll and Imd pathways, reducing the production of AMPs and enabling the virus to persist and spread efficiently through the vector population (Czosnek *et al.* 2017). Together, these behavioral and physiological manipulations illustrate the coevolutionary dynamics (Fig. 2) between plant pathogens and their insect vectors, highlighting the intricate molecular and biochemical processes involved in the spreading of plant diseases. Understanding these interactions offers valuable insights for the development of targeted vector control strategies and disease management programs to mitigate crop losses and enhance agricultural productivity (Eigenbrode *et al.* 2018).

6.2. Genes and proteins responsible for transmission

The transmission of plant pathogens by insect vectors depends heavily on specific genes and proteins (Appendix B) involved in various stages of the transmission cycle. These factors play a critical role in the molecular interactions between the pathogen, insect vector, and plant host (Eigenbrode *et al.* 2018). Each component, whether viral CP, bacterial effector, or insect receptor, serves a specialized function, contributing to the efficient acquisition, retention, and transmission of the pathogen. Understanding these genes and proteins provides insights into the molecular basis of transmission and offers targets for disease management strategies (Perilla-Henao and Casteel 2016). Pathogens, including viruses, bacteria, and fungi, rely on several specific genes and their protein products to ensure successful interactions with insect vectors. These proteins mediate attachment to receptors on the vector's body, facilitate pathogen movement within the host plant, and manipulate the insect vector's physiology to promote transmission (Jangra *et al.* 2024a).

Coat proteins (CPs) genes and movement protein (MP)

genes The CPs is a structural protein encoded by the virus that forms the protective outer layer of viral particles. It plays a key role in binding to receptors on the insect's stylet or gut, facilitating acquisition and transmission during feeding. Viral CPs interact with glycoproteins or lectins on the stylet or foregut of the vector, ensuring the virus attaches securely during brief probing events (Zhang *et al.* 2022). In non-circulative viruses, like as Potyviruses, CPs form transient interactions with receptors on the stylet, enabling efficient transmission during short feeding probes. As TuMV utilizes its CPsto bind to the aphid's stylet, ensuring rapid virus acquisition and transmission during initial feeding bouts (Stevens and Lacomme 2017). In CaMV, the CPs interacts with specific aphid stylet receptors, with the assistance of P2 helper proteins, ensuring successful non-circulative transmission (Hogenhout *et al.* 2008). Movement proteins are essential for intra-floral and intercellular movement of viruses within plant tissues. They facilitate the movement of viral particles between plant cells, allowing the virus to infect new tissues and become available for acquisition by insect vectors. The role in insect transmission of MPs is not only to assist viral movement within the host plant but may also modulate the interactions between the virus and the insect vector (Kumar and Dasgupta 2021). Some viruses rely on MPs to enhance vector retention or target specific tissues within the plant to optimize acquisition by insects, as the tobacco mosaic virus (TMV) encodes movement proteins that facilitate its spread within the plant. Similarly, begomoviruses use movement proteins to reach the phloem, which whiteflies can acquire during feeding (Atabekova *et al.* 2023).

Bacterial effector proteins In bacterial pathogens, effector proteins are secreted molecules that manipulate host and vector physiology to suppress immune responses and enhance pathogen survival (Milgroom 2023). These proteins are often injected into the host or vector cells through specialized secretion systems, such as Type III secretion systems. Bacterial effectors interfere with the insect's immune pathways, such as the Toll and Imd pathways, reducing the production of AMPs (Brivio and Mastore 2018). They also manipulate vector physiology by altering gene expression, promoting reproductive success, or modulating metabolism. For example, CLas secretes effector proteins that enhance the reproductive capacity and mobility of the *D. citri* vector. These effectors interfere with the psyllid's immune response, allowing the bacterium to persist and replicate within the vector (Pelz-Stelinski *et al.* 2010).

Insect and vector interaction proteins (gut and stylet receptors) Several proteins play essential roles in the acquisition, retention, and transmission of pathogens. These proteins include receptors on the gut and stylet, transport proteins for pathogen movement through the hemolymph, and immune proteins that regulate the vector's immune response (Perilla-Henao and Casteel 2016). The stylet and gut receptors are surface proteins on the vector that recognize and bind to pathogen molecules, facilitating acquisition during feeding (Gale 2020). There are different types of receptors, including Aminopeptidases, Cadherins, and Lectins: Aminopeptidases enzymes, located on the surface of midgut cells, serve as binding sites for certain viruses, such as begomoviruses (Singh *et al.* 2021). They play a role in both pathogen acquisition and retention. Cadherins are adhesion molecules that facilitate viral attachment to the gut lining, allowing the pathogen to cross the epithelial barrier and enter the hemolymph (Badillo-Vargas *et al.* 2019; Gale 2020). Meanwhile, lectins bind to specific carbohydrate molecules on viral CPs, stabilizing the interaction between the pathogen and the insect vector. For example, whiteflies express cadherins that bind to the CPs of begomoviruses, enabling the virus to cross the gut barrier and reach the hemolymph, where it can be transported to the salivary glands (Dietzgen *et al.* 2016).

Transport proteins and immune proteins Pathogens exploit transport proteins within the insect's hemolymph to move efficiently through the circulatory system and reach the salivary glands for transmission. There are a few types of transport proteins, such as lipophorins that transport lipids through the hemolymph and also bind to viral particles, facilitating their movement through the vectors (Bonnet *et al.* 2018) as well as vitellogenins are precursors to proteins. However, they can also bind to bacterial and viral particles, aiding pathogen transport through the hemolymph. Lipophorins bind to the CPs of viruses or bacterial outer membrane proteins, preventing the pathogen from being degraded and assisting in transport to the salivary glands (Müller and Müller 2023). In whiteflies, begomoviruses exploit lipophorins to move through the hemolymph, ensuring the virus reaches the salivary glands for transmission to the next plant host (He *et al.* 2021).

The insect immune system relies PRRs to detect pathogens and initiate immune responses. However, many pathogens evade or suppress these immune responses, ensuring their survival within the vector (Wang Y *et al.* 2022). A critical pathway involved is the Toll pathway, which activates the production of AMPs in response to fungal and bacterial pathogens. And Imd pathway, which regulates the immune response to Gram-negative bacteria by producing AMPs (Jo *et al.* 2019). Some pathogens suppress PRR activity, preventing the insect from recognizing and eliminating the pathogen. Others interfere with the signaling pathways downstream of PRRs, blocking the production of AMPs and other immune responses (Zhao *et al.* 2023). Some pathogens can evade recognition by peptidoglycan recognition protein (PGRPs), which are key PRRs in insects. For instance, certain Gram-negative bacteria such as *Pseudomonas aeruginosa*, produce molecules that mimic or mask their peptidoglycan (PGN) structures, preventing PGRP recognition and to evade recognition by host proteins like NGAL, which typically scavenge bacterial siderophores. This stealth mechanism allows the bacteria to thrive in hostile environments (Dziarski and Gupta 2006; Kordaczuk *et al.* 2020). This prevents the activation of the Imd pathway, which is crucial for the production of AMPs. Fungi can evade recognition by β-1,3-glucan recognition protein (βGRP) by modifying their cell wall components (Peek *et al.* 2012; Pel and Pieterse 2013; Sohail Sajid *et al.* 2022). For example, some fungi such as *Aspergillus fumigatus* produce enzymes that degrade β-glucans or alter their structure, making it difficult for βGRPs to bind and

initiate an immune response. Baculoviruses, *Autographa californica* multiple nucleopolyhedrovirus (AcMNPV) which infect insects, produce proteins that can inhibit the activation of the prophenoloxidase (proPO) system, a key component of the insect immune response. This prevents melanization and encapsulation of the virus, allowing it to evade the immune system (Clem and Passarelli 2013; Martínez-Solís *et al.* 2020). Begomoviruses such as TYLCV disrupt the Toll and Imd pathways in *B. tabaci*, suppressing AMP production and enabling the virus to persist and replicate within the vector without being eliminated (Zhao *et al.* 2023). Pathogens can interfere with the Toll and Imd signaling pathways, which are central to the insect immune response. For example, *Metarhizium rileyi* can inhibit the activation of these pathways by secreting molecules that interfere with the signaling cascades. This prevents the production of AMPs and other immune effectors (Xu *et al.* 2025).

6.3. Biochemical processes involved in transmission

The transmission of plant pathogens through insect vectors involves multiple biochemical processes that influence the efficiency of pathogen acquisition, retention, and transmission to plant hosts. These processes include signal transduction pathways, protein–protein interactions, and metabolic pathways that govern how pathogens evade immune responses, interact with vector proteins, and manipulate vector physiology for their benefit. A deeper understanding of these biochemical mechanisms reveals how pathogens survive within insect vectors and spread between plants (Mahanta *et al.* 2023).

Signal transduction pathways Insect vectors rely on innate immune systems, particularly the Toll and Imd pathways, to detect and respond to invading pathogens. These pathways are crucial for activating the production of AMPs, which inhibit or eliminate bacterial, fungal, and viral invaders (Zhao *et al.* 2023). Toll pathway primarily responds to fungal and Gram-positive bacterial infections. Upon pathogen detection, PRRs bind to pathogen-associated molecular patterns (PAMPs), triggering a cascade that activates the NF-κB transcription factor. NF-κB then promotes the expression of genes encoding AMPs (e.g., defensins) (Valanne *et al.* 2011). The Toll pathway is triggered in insects like mosquitoes in response to fungal infections, producing defensins that restrict pathogen proliferation. While the Imd pathway is activated by Gram-negative bacteria. Upon detecting bacterial lipopolysaccharides (LPS) through receptors like PGRP-LC, this pathway triggers the production of AMPs such as cecropins and attacins. When vectors such as whiteflies encounter pathogens, the Imd pathway is expected to limit bacterial infections through the production of these AMPs (Manniello *et al.* 2021).

Pathogen suppression of immunity Many pathogens have evolved mechanisms to suppress or evade immune responses in insect vectors, allowing them to survive, replicate, and transmit without being eliminated. Suppression is a mechanism in which some viruses mimic host molecules, reducing immune detection by the Toll or Imd pathways. Bacterial pathogens, such as CLas, inhibit the expression of AMP genes, weakening the vector's immune response and allowing the bacterium to persist within the insect gut and hemolymph (Stevens and Lacomme 2017). Immune evasion in whiteflies such as Begomovirus suppresses Toll pathway signaling to avoid AMP-mediated clearance. This immune suppression allows the virus to remain viable within the vector, increasing transmission success (Dombrovsky *et al.* 2014).

Protein interactions, metabolic pathways and nutrient acquisition The efficiency of pathogen transmission depends heavily on the binding affinity and specificity of interactions between pathogen proteins and insect vector receptors. Structural compatibility between these molecules ensures that the pathogen binds effectively to receptors on the stylet, gut lining, or salivary glands of the insect vector (Rao *et al.* 2024). According to the mechanism of binding, the viral CPs bind to complementary glycoprotein receptors on the stylet or gut epithelial cells. The stronger the binding affinity between these molecules, the more efficiently the virus attaches and transmits. In begomoviruses, the CPs interacts with cadherin-like receptors in whitefly vectors, facilitating the viruses entry across the gut barrier and ensuring transport to the salivary glands for further transmission (Santos Díaz *et al.* 2018). There are factors affecting affinity and strength of binding between pathogen proteins and insect receptors were influenced by environmental factors such as temperature and pH (Fig. 2), which can alter receptor structure or protein folding. Post-translational modifications (PTMs) such as phosphorylation and glycosylation play essential roles in regulating protein interactions, including those between pathogens and insect vectors (Duan and Walther 2015). Phosphorylation is the addition of phosphate groups to proteins, often catalyzed by kinases, alters protein conformation and affects binding interactions. Some insect receptors involved in pathogen transmission are regulated by phosphorylation, influencing their ability to bind viral particles (Luan *et al.* 2014). While glycosylation is the addition of carbohydrate groups to proteins that can affect binding affinity by modifying protein charge or structure. Viral CPs with glycosylation sites that may bind more effectively to glycoprotein receptors on insect stylets, enhancing transmission efficiency (Müller and Müller 2023). Pathogens manipulate the insect vector's metabolic pathways to acquire nutrients and generate the energy needed for replication and transmission. These metabolic changes ensure the vector provides a favorable environment for pathogen survival and propagation (Mahanta *et al.* 2023). Pathogens place significant energy demands on the insect vector by diverting metabolic resources to support their replication and transport. The metabolic burden can alter the vector's behavior, physiology, and reproductive output. For example, viruses often increase energy consumption by promoting cellular processes such as glycolysis and ATP production, providing the energy needed for viral replication (Abraham *et al.* 2017; Javed *et al.* 2021). Bacterial pathogens, like CLas, modify the metabolic pathways of psyllid vectors to reduce energy expenditure and ensure the survival of the infected vector. Although pathogen transmission provides evolutionary advantages to the pathogen, the metabolic demands can reduce the vector's overall fitness, leading to trade-offs in

behavior, reproduction, or longevity (Killiny *et al.* 2017).

Some pathogens alter the nutrient absorption pathways of insect vectors to promote their growth and replication. This manipulation can involve changes in gene expression related to nutrient uptake or storage. Pathogens such as CLas reprogram the psyllid's metabolism to increase the absorption of lipids and carbohydrates, providing the resources needed for bacterial growth within the vector (Vyas *et al.* 2015; Zhang *et al.* 2025). Pathogens often exploit lipophorins and vitellogenins, essential nutrient transport proteins for hemolymph transport and nutrient acquisition. Lipophorins bind to viral or bacterial particles, facilitating their movement through the vector's body and ensuring they reach the salivary glands for transmission. While pathogens benefit from enhanced nutrient absorption, the vector may suffer from reduced fitness due to nutrient depletion, compromising reproduction or lifespan (Matsuno *et al.* 2018).

Understanding these biochemical processes provides critical insights into the molecular basis of pathogen transmission and offers potential targets for disrupting transmission cycles. Future research focusing on post-translational modifications, nutrient manipulation, and immune suppression mechanisms will help develop innovative strategies for vector control and disease management in agricultural systems (de Angeli Dutra *et al.* 2022; Idrees *et al.* 2024). The molecular and biochemical mechanisms governing the transmission of plant pathogens by insect vectors involve a series of highly specialized interactions. Non-circulative transmission relies on protein-protein interactions at the stylet or foregut, while circulative transmission requires the pathogen to navigate complex biological barriers within the vector. Pathogens also manipulate vector behavior and physiology to enhance their transmission potential, highlighting the co-evolution between vectors and pathogens (Rau *et al.* 2015; Mondino *et al.* 2020). So, understanding these mechanisms at a molecular level is essential for developing innovative strategies to disrupt transmission cycles and mitigate the impact of vector-borne plant diseases.

7. Insect vectors and specific pathogens

7.1. Insect vectors responsible for the transmission of viruses

Most plant viruses need the assistance of a vector in order to spread from plant to plant (Fig. 1). The study of virus–vector interactions has shown standard modes of transmission. At least one of the structural virion proteins is required for transmission in all plant viruses investigated so far. While some viruses can replicate and spread using just their capsid proteins, others need a "helper" protein to aid retention by connecting the virus to the vector's surface (Verdier *et al.* 2023). All viruses, whether they can replicate or not, have a similar life cycle and are acquired by vectors, starting in the insect's digestive system. Even within same virus family, differences exist in how viruses spread from infected gut epithelial cells to other organs (Mubashir 2022). Similarities between viral infection cycles in insect and plant hosts have been highlighted by the discovery of virus movement in vectors *via* tubule structures made of non-structural virus proteins and the participation of the actin cytoskeleton in this process. These tubules provide a novel approach for insects to overcome midgut obstructions in certain cases. The use of the vitellogenin absorption route for viral invasion of insect eggs is a relatively new finding in the vector-borne transmission of plant viruses (Wei *et al.* 2017; He *et al.* 2021). Transovarial transmission of viruses is similar to that of other insect-associated microorganisms. Identifying vector molecules that interact with and react to viruses is a new subject, even though the viral components of vector interactions are well-defined in most commercially relevant systems. Adopting cutting-edge research tools should help us figure out how these vector parts work to spread viruses. Many different vectors may be disrupted in their ability to spread disease by focusing on the similarities between the roles played by viral structural proteins and the first locations of virus entry or retention (Whitfield *et al.* 2015).

Most plant-infecting viruses rely on various insects for transmission to their host plants (Appendix A). These viruses and responsible insects (that play their role as vectors to transmit it to the host plant) interactions can vary in duration and specificity (Rao *et al.* 2024). However, some common patterns have emerged as plant viruses typically contain structural proteins on the surface of their virions that play a crucial role in transmission. In some cases, additional non-structural helper proteins serve as bridges, facilitating the attachment of the virion to the vector's binding site (Mubashir 2022). Our current knowledge of the fundamental mechanics of viral transmission has been dramatically expanded by recent scientific findings, allowing for the creation of novel approaches to interrupting the infection cycle (Lin *et al.* 2024). For example, most plant-infecting viruses rely on insect vectors to transmit them to their host plants. These virus–vector interactions can vary in duration and specificity, but some common patterns have emerged; likewise, plant viruses typically contain structural proteins on the surface of their virions that play a crucial role in transmission (Van Bogaert 2016; Fermin 2018). In some cases, additional non-structural helper proteins serve as bridges, facilitating the attachment of the virion to the vector's binding site. Viruses have a specific affinity for particular sites within or on vectors, where they remain until they are eventually transmitted to their plant hosts (Verdier *et al.* 2023). The factors within viruses that determine vector transmission represent promising targets for translational research aimed at disrupting transmission or reducing vector populations (Whitfield and Rotenberg 2015). Several terminologies characterizing transmission connections based on acquisition and inoculation thresholds and retention by the vectors emerged from the first studies of the biology of insect transmission of plant viruses. There are four insect vector plant virus transmission types: non-persistent, semi-persistent, circulating, and propagating (Ng *et al.* 2015). Some penetrate the insect stomach and circulate or proliferate within the insect vector body (circulative), whereas others attach to particular cuticular regions without accessing cells (noncirculative). Recently, the relationship between plant viruses and the insects that transmit them has been given more attention (Blanc *et al.* 2014). Viruses

that are not actively being circulated are kept in the insect's stylet or foregut. The phloem limits the infectivity of non-propagative circulative viruses, which often enter the insect *via* the midgut or hindgut. Certain circulatory plant viruses get access to the salivary glands through the hemolymph (Zhao *et al.* 2020; He *et al.* 2021). Conversely, viruses (circulative propagative) enter the insect *via* its filtering organs in its anterior midgut. Some viruses (propagative) are capable of replication utilize the hemolymph system to spread. Several terminologies characterizing transmission connections based on acquisition and inoculation thresholds and retention by the vectors emerged from the first studies of the biology of insect transmission of plant viruses (Andret-Link and Fuchs 2005; Blanc *et al.* 2014). Some viruses that replicate rapidly are restricted to the phloem of plants, whereas others may be found throughout the plant. In order to complete their journey, viruses must first enter the hemolymph before moving on to the salivary glands through the nerve tissue (neurotropic pathway) or the connective tissues. One kind of reovirus travels from cell to cell in the midgut by way of tubules, while another utilizes tubules to cross the basal lamina (Purcell and Almeida 2005; Huo *et al.* 2014; Jia *et al.* 2018).

In case of persistent transmission, an infected vector may continue to spread a pathogen for many days or weeks. For example, beet leafhopper (*Circulifer tenellus*) is the only insect species known to transmit the beet curly top virus (BCTV) in a non-propagative circulative manner. The incubation time for the virus in beet leafhoppers is just a few hours (Ammar and Nault 2002; Weintraub *et al.* 2019). Their ability to spread this geminivirus is proportional to how much time they spend feeding on a plant infected by it. BCTV is a non-replicating virus that may be spread from one vector to another (Carter 1973). To complete the vector transmission process, pathogens must proliferate inside the vector until they infect enough of the vector's salivary glands, which might take a long time. Some plant diseases, such as viruses and bacteria, may cause damage to the vector because they replicate in a variety of vector organs and tissues that are probably not necessary for transmission (Purcell and Almeida 2005). Reoviruses, like the rice dwarf virus (RDV), may replicate in the insects that spread them and the plants they infect. Pathogens may be guaranteed to survive the vector's developmental molts by multiplying and circulating inside the vector's bodily cavity (hemocoel). Once the vector has shed its exoskeleton, transmission of viruses that are not persistent or just intermittent is terminated (Marwal *et al.* 2018).

Aphids and viruses Aphids are phloem-feeding insects known as significant pests in agriculture due to their ability to weaken plants by feeding and to transmit numerous plant viruses. Various strategies have been employed to identify viral receptors within aphid vectors, leading to the discovery of several key proteins involved in virus transmission (Brault *et al.* 2010). Using virus overlay and immunoblot assays, two accessory salivary gland (ASG)-associated proteins, SaM35 and SaM50, were identified in the wheat aphid (*Sitobion avenae*) as receptors for the BYDV-MAV isolate (Li *et al.* 2001). Additionally, a 50-kD protein (P50) extracted from wheat aphid (*Schizaphis graminum*) and *S. avenae* exhibited specific binding to purified virus particles of the BYDV-GAV isolate. Immunogold labeling localized P50 to the plasma membrane surrounding the ASG in *S. graminum*, suggesting its role in virus transmission (Wang and Zhou 2003). Seddas *et al.* (2004) identified three proteins, Rack-1, glyceraldehyde-3-phosphate dehydrogenase (GAPDH3), and actin, in the *M. persicae* that may be involved in the transcytosis of BWYV particles (Seddas *et al.* 2004). For CaMV, the viral P2 protein acts as a receptor for a non-glycosylated protein embedded in the chitin matrix of aphid stylets (Uzest *et al.* 2007). Furthermore, CuPs in *M. persicae* have been identified as receptors interacting with the helper-component protease (HCPro) of zucchini yellow mosaic virus (ZYMV) (Dombrovsky *et al.* 2014). Similarly, ribosomal protein S2 (RPS2) was found to interact with the HC-Pro of tobacco etch virus (TEV), suggesting its involvement in virus transmission (Fernandez-Calvino *et al.* 2010). Cyclophilin A and B proteins in *S. graminum* interact with the RPV strain of cereal yellow dwarf virus (CYDV-RPV) and may mediate virus transport from the hindgut lumen into the hemocoel (Tamborindeguy *et al.* 2013). Moreover, membrane alanyl aminopeptidase N (APN) has been identified as a receptor for the pea enation mosaic virus (PEMV)-encoded CPs in the gut of the pea aphid (*Acyrthosiphon pisum*) (Linz *et al.* 2015).

Other identified receptors such as MPCP4 which is a cuticular protein in *M. persicae* that binds with the CP of CMV. RNAi-mediated silencing of MPCP4 suppressed the aphid's ability to acquire CMV, indicating its role as a viral receptor (Liang *et al.* 2021). And from Stylin-01 and Stylin-02, silencing of stylin-01 reduced CaMV transmission by aphids, suggesting its function as a receptor in the stylet (Webster *et al.* 2018). Ephrin receptor (Eph) is a novel membrane-bound protein involved in the transmission of turnip yellows virus (TuYV) by *M. persicae*. Feeding aphids with dsRNA targeting Eph-mRNA reduced TuYV accumulation and transmission (Shingote *et al.* 2022). In addition, MPCP2 interacts with PVY, and its silencing resulted in a 47% reduction in virus transmission efficiency, highlighting its potential role in virus transmission (Bahrami Kamangar *et al.* 2019). Complement component 1Q subcomponent-binding protein (C1QBP) which interacts with structural proteins of PLRV. Inhibiting C1QBP increased PLRV acquisition and transmission by aphids, indicating its regulatory role (DeBlasio *et al.* 2021).

Planthoppers, leafhoppers and viruses Planthoppers and leafhoppers are significant vectors for viruses affecting cereal crops. Identifying viral receptors in these insects is crucial for understanding virus transmission mechanisms. A 32-kD membrane-associated protein in the rice brown planthopper (*Nilaparvata lugens*) acts as a potential receptor for rice ragged stunt virus (RRSV) (Zhou *et al.* 1999). In the small brown planthopper (SBPH, *Laodelphax striatellus*), proteins such as receptor for activated protein C kinase (RACK) and GAPDH3 were found to be involved in epithelial transcytosis of RSV particles (Li *et al.* 2011). The vitellogenin receptor in SBPH is required for the transovarial transmission of RSV, facilitating virus movement into the ovaries and subsequent transmission to offspring (Huo *et al.* 2014). Other interacting proteins, such as G-protein pathway suppressor 2 (GPS2) which interacts with RSV capsid protein, activating the c-Jun

N-terminal kinase (JNK) signaling pathway, promoting RSV replication in the vector (Wang *et al.* 2017). CuPs (CPR1) which interacts with the nucleocapsid protein of RSV, aiding viral survival in the hemolymph (Liu *et al.* 2015).

The Pns10 protein of RDV interacts with cytoplasmic actin in the rice green leafhopper (*Nephotettix cincticeps*), emphasizing actin's role in virus transmission and specificity (Chen *et al.* 2015). Oligomycin-sensitivity conferral protein (OSCP) interacts with RRSV Pns10 in *N*. *lugens*, with silencing of OSCP reducing viral load, indicating its role in viral proliferation (Huang *et al.* 2017). Furthermore, proteins such as RPL18, sugar transporter 6 (STP), α-tubulin 2, and flotillin 2 in SBPH have been identified as interacting with RSV components, facilitating viral translation, entry, and transmission (Li *et al.* 2020; Wang W *et al.* 2022). Moreover, in corn planthopper (*Peregrinus maidis*), cyclophilin A and apolipophorin III interact with maize mosaic virus (MMV) glycoproteins (Alviar *et al.* 2022). In addition, a voltage-dependent anion channel 2 (VDAC2) interacts with RSV-encoded RNA-dependent RNA polymerase (RdRP), facilitating virus accumulation (Zhang *et al.* 2023).

Thrips and viruses Thrips are vectors for several viruses including TSWV, which causes significant crop losses (Jangra *et al.* 2024a). Young thrips larvae acquire the virus, which is then transmitted by adult thrips throughout their lifespan (Gabryś and Kordan 2022). Identified receptors in thrips includes 50-kD midgut protein in *F*. *occidentalis*, which is a potential receptor for TSWV glycoproteins (Bandla *et al.* 1995). Additionally, a 94-kD protein was found to interact with the envelope glycoprotein (G2) of TSWV in *F*. *occidentalis* and cotton thrips (*Thrips tabaci*) (Bandla *et al.* 1998; Jangra *et al.* 2024a;). C-Type lectin is proposed as the primary cellular receptor for groundnut bud necrosis virus (GBNV) glycoprotein (GN) in melon thrips (*T*. *palmi*) (Jagdale and Ghosh 2019). Moreover, six TSWV-interacting proteins (TIPS) were identified from *F*. *occidentalis*, including endocuticle structural glycoprotein and cyclophilin, which interact with the viral GN protein and may facilitate virus entry or infection processes (Badillo-Vargas *et al.* 2019). Ubiquitin-related proteins, such as UBR7, also interact with TSWV GN, with silencing of UBR7 leading to decreased viral infection in thrips (Khan *et al.* 2023; Shi *et al.* 2023). Other proteins like apolipoprotein-D (ApoD), orai-2-like (Orai), and obstructor-E-like isoform X2 (Obst) have been identified as interacting with TSWV in *F*. *occidentalis*. Overexpression of Obst in viruliferous thrips and reduced virus acquisition and transmission upon Obst silencing indicate its role in TSWV transmission (Zheng *et al.* 2023).

Whiteflies and viruses Whiteflies (*B*. *tabaci*) are vectors for many severe plant viruses, particularly begomoviruses, which cause significant crop losses (Czosnek *et al.* 2017). Heat shock proteins BtHSP16 and BtHSP70 in *B*. *tabaci* interact with the CPs of tomato yellow leaf curl sardinia virus (TYLCSV), protecting other begomoviruses during translocation within the whitefly (Ohnesorge and Bejarano 2009). *Bemisia tabaci* PGRP acts as a binding site for TYLCV, with interactions confirmed *via* immunocapture PCR (Wang *et al.* 2016). Cyclophilin B protein also interacts with TYLCV, potentially facilitating virus movement across membrane barriers in the vector (Kanakala and Ghanim 2016). Additionally, a midgut protein (MGP) has been identified as a putative receptor for both tomato leaf curl New Delhi virus (ToLCVNDV) and cotton leaf curl Rajasthan virus (CLCuV-Ra) in *B*. *tabaci* (Rana *et al.* 2016). Vitellogenin, an egg yolk precursor protein, is essential for whiteflies' transovarial transmission of TYLCV in *B*. *tabaci* (Wei *et al.* 2017).

Other interacting proteins and factors such as vesicle-associated membrane protein-associated protein B (VAPB) is abundant in midgut tissues of *B*. *tabaci*, VAPB interacts with TYLCV and is involved in virus transmission (Zhao *et al.* 2019). The proliferating cell nuclear antigen (PCNA) interacts with TYLCV-encoded replication-associated protein (Rep), aiding in virus replication within the vector (He *et al.* 2020). BtCubam receptor complex comprising of cubilin (BtCUBN) and aminoless (BtAMN), facilitates the entry of begomoviruses into the whitefly midgut *via* clathrin-dependent endocytosis. Additionally, transcription factors of whitefly C2H2 zinc finger protein (VEZF) interacts with the CP of begomoviruses. Silencing of VEZF increased virus retention, suggesting its inhibitory role in transmission (Zhao *et al.* 2020; Ghosh *et al.* 2023). Finally, the phosphatidylethanolamine-binding protein (PEBP) interacts with TYLCV-CP, leading to downregulation of the MAPK signaling cascade, activation of apoptosis in whiteflies, and an increase in viral titer (Wang S *et al.* 2022).

7.2. Role of viruses in "plant–virus–vector" interactions

The uptake and transmission of viruses by insect vectors require specific associations between viral proteins and vector proteins, often referred to as receptors. Identifying these receptors is key to unravel the mechanisms of virus transmission, which can help develop novel approaches for managing virus–vector relationships (Fig. 4) (Sastry *et al.* 2019). In *B*. *tabaci*, proteins such as tubulin beta chain (Stubbs), keratin type I cytoskeletal 9-like (KRT), and cytochrome-c-oxidase subunit 5A (COX) interact with the minor coat protein (CPm) of cucurbit chlorotic yellows virus (CCYV), affecting virus retention and transmission (He H F *et al.* 2023). The complex interactions between viral proteins and vector proteins determine the efficiency of virus acquisition, retention, and transmission. By unraveling these interactions, we can pave the way toward safer alternatives to insecticides and novel methods for managing insect vectors and the viruses they transmit (Ziegler-Graff and Brault 2008; Isman 2015).

7.3. Insect vectors responsible for bacterial pathogens

Insect vectors are central to the transmission of bacterial pathogens that cause severe plant diseases, significantly impacting global agriculture (Appendix C). These bacteria including *Candidatus* Liberibacter spp., *Phytoplasma* spp., *Spiroplasma* spp., and have evolved intricate strategies to interact with their insect carriers and plant hosts (Fig. 2) (Purcell and Almeida 2005; Reyes caldas *et al.* 2022). The pathogens rely heavily on the plant's vascular systems,

primarily the phloem and xylem, to establish infections and ensure systemic spread. Understanding these complex interactions between bacteria, insect vectors, and plant hosts is essential for developing strategies to mitigate these vector-borne diseases (Huang *et al.* 2020). The transmission of bacterial pathogens involves multiple steps, beginning with the acquisition by the vector during feeding and followed by intracellular migration, colonization, and transmission to new plant hosts. These bacteria utilize different transmission mechanisms depending on their biology and host preferences (Perilla-Henao and Casteel 2016). In some cases, such as *Candidatus* Liberibacter and *Phytoplasma* species, the pathogens are injected into the phloem, which remains confined within sieve elements, moving slowly against the phloem's natural flow (Vyas *et al.* 2015). Others, like *X. fastidiosa*, colonize the xylem and spread throughout the plant by moving across xylem vessels and through pit membranes. These vascular pathogens often accumulate in plant tissues such as roots and sink organs, ensuring persistence within the host (Killiny *et al.* 2017).

A key feature of phloem-limited pathogens is their structural flexibility, which allows them to move through the narrow spaces between sieve plates, even when the plates are reinforced by callose. *Candidatus* Liberibacter and *Phytoplasma* aggregate near sieve pores, facilitating their movement and spread within the phloem. These bacteria also adhere to the inner surfaces of sieve tube membranes, which enhances their colonization potential (Weintraub *et al.* 2019). In contrast, *X. fastidiosa* cells distribute unevenly within the xylem, forming biofilms that block water flow and damage plant tissues, resulting in severe symptoms such as leaf scorch and dieback (Perilla-Henao and Casteel 2016). The unique biology of these bacterial pathogens complicates their study, mainly due to their reliance on insect vectors and their inability to grow in culture. With the exception of *Liberibacter crescens BT-1*, most phloem-limited bacteria have resisted cultivation, limiting research efforts to identify the genetic determinants responsible for host specificity and vector compatibility (Sena-Vélez *et al.* 2019). However, *Spiroplasma* spp. offer an advantageous model for studying phloem-limited bacteria, as they are cultivable, have a broad host range, and can be genetically manipulated. These attributes provide a foundation for exploring the molecular mechanisms underlying bacterial movement, adhesion, and pathogenicity (Pelz-Stelinski *et al.* 2010). Vector-mediated bacterial transmission relies heavily on molecular communication between the pathogen, insect vector, and plant host. During feeding, bacteria like *Candidatus* Liberibacter and *Phytoplasma* exploit specialized tip structures and exposed surface proteins to attach to insect membranes, facilitating their movement into the insect's body (Pandey *et al.* 2021). Interestingly, while CLas carries flagellar genes, these are only expressed within the insect vector, underscoring how the vector environment shapes bacterial behavior. In *Spiroplasma citri*, twitching motility suffices for pathogenicity, suggesting that surface proteins and molecular interactions with insect tissues play a more critical role than traditional motility mechanisms (Alma *et al.* 2019; Nian *et al.* 2024).

Once ingested by the insect vector, bacterial pathogens face multiple barriers that must be overcome to complete the transmission cycle. In persistent propagative transmission, bacteria like CLas circulate within the vector's hemolymph and may replicate in the salivary glands, where they are transmitted to new plant hosts. The interaction between bacterial adhesins and gut receptors plays a vital role in allowing pathogens to traverse gut membranes (Perilla-Henao and Casteel 2016). For example, CLas interacts with microvilli proteins in the psyllid midgut, facilitating its movement across the gut lining. Similarly, *X. fastidiosa* employs hemagglutinin-like proteins to adhere to the foregut cuticle of sharpshooter leafhoppers, promoting colonization and biofilm formation (Kruse *et al.* 2019). Biofilm formation is a crucial element in the pathogenicity of xylem-colonizing bacteria. In *X. fastidiosa*, cell-wall-degrading enzymes like CbsA aid bacterial proliferation and movement within the xylem. Deletion of the CbsA gene in *X. fastidiosa* severely impairs vascular colonization, demonstrating the importance of these enzymes in bacterial pathogenicity (Batarseh Tiffany *et al.* 2022). Similarly, non-vector-borne *Xanthomonas oryzae* pv. *oryzae* mutants lacking CbsA exhibited increased symptom development and apoplastic colonization, further highlighting how these enzymes determine niche specificity and bacterial fitness (Morelli *et al.* 2021).

The interactions between bacterial pathogens and insect vectors are further influenced by immune modulation within the vector. CLas manipulates the psyllid immune system by downregulating immune genes, such as those involved in the Toll and Imd pathways, evading immune responses and enhancing bacterial transmission (Pelz-Stelinski *et al.* 2010). Similarly, *S. citri* secretes proteins that interfere with the vector's immune signaling, allowing the bacteria to persist and multiply within the insect. Such strategies underscore the complexity of vector–pathogen interactions and demonstrate how pathogens evolve mechanisms to circumvent immune defenses (Jones 2018; Mahanta *et al.* 2023). The transmission of bacterial pathogens also has profound economic implications. Diseases such as Huanglongbing (HLB), caused by CLas, have devastated the citrus industry, resulting in billions of dollars in losses. Similarly, phytoplasma diseases including lime witches' broom and lethal yellowing of coconut palms have wreaked havoc on the agricultural economies of the Middle East and Southeast Asia (Pandey *et al.* 2021). In the grape industry, pierce's disease caused by *X. fastidiosa* has resulted in annual losses exceeding US$100 million in California alone (Morelli *et al.* 2021; Raparelli *et al.* 2024). Moreover, olive rapid decline syndrome, also caused by *X. fastidiosa*, has threatened olive production in Europe, with potential losses estimated to reach billions of euros over the next few decades if effective disease control strategies are not implemented (Huang *et al.* 2020).

Recent advances in molecular and genomic tools pave the way for a deeper understanding of the interactions between bacterial pathogens, insect vectors, and plant hosts. Techniques such as transposon sequencing (TNseq) allow researchers to identify genes that are critical for bacterial movement, adhesion, and persistence. Additionally, genomic studies of insect vectors have revealed genes

involved in pathogen acquisition and transmission, providing new insights into how these insects interact with bacterial pathogens (Jangra *et al.* 2024b). For example, studies of psyllid feeding behavior have demonstrated how the structure of the phloem influences the acquisition and transmission of CLas, highlighting the role of plant vascular architecture in disease dynamics (Huang *et al.* 2020).

Overall, bacterial plant pathogens, particularly those transmitted by insect vectors, present a complex and significant challenge to global agriculture. These pathogens such as phytoplasmas, *Spiroplasma*, and *X. fastidiosa* exhibit diverse modes of interaction with both their plant hosts and the insect vectors that carry them (Reyes Caldas *et al.* 2022). Hemipteran insects, including psyllids, leafhoppers, sharpshooters, and spittlebugs, serve as the primary vectors, facilitating the transfer of bacterial pathogens directly into plant vascular tissues (Perilla-Henao and Casteel 2016). The intricate biological processes involved in vector-borne transmission highlight the importance of understanding the molecular mechanisms that govern these relationships to develop effective control strategies (Alma *et al.* 2019).

Each bacterial pathogen demonstrates unique adaptations to colonize both plant and insect hosts. *Xylella fastidiosa* colonizes the xylem vessels of plants and forms biofilms on the cuticular surface of its insect vectors' foreguts (Cornara *et al.* 2020). In contrast, phloem-limited bacteria, including *Candidatus* Liberibacter, *Phytoplasmas*, and *Spiroplasma*, are transmitted by sap-sucking insects that feed on phloem sap, such as psyllids and leafhoppers. These bacteria invade both the plant's phloem and the insect vector's tissues, affecting not only the plant host but also the vector itself (Alma *et al.* 2019; Weintraub *et al.* 2019).

One of the key differences between these pathogens lies in their virulence mechanisms. For instance, while *X. fastidiosa* does not possess the type III secretion systems (TTSSs) typically found in many Gram-negative bacteria for injecting effectors into plant cells, it has evolved alternative virulence factors to facilitate its colonization and spread within the xylem (Lee and Rogers 2015). Conversely, phytoplasmas contain pathogenicity islands known as potential mobile units (PMUs) that encode effectors capable of manipulating plant development. These PMUs are prone to recombination and mutation, providing phytoplasmas with a highly flexible and adaptive genome that enhances their virulence and capacity to infect new hosts (Weintraub 2012; Alma *et al.* 2019; Tokuda *et al.* 2023).

Effector biology and plant manipulation A central feature of bacterial vector-borne pathogens is their ability to produce effectors-secreted proteins that manipulate host cell processes to benefit the pathogen. Phytoplasmas, for instance, secrete effectors like SAP11 and SAP54, which profoundly impact plant development. SAP11 destabilizes plant transcription factors that regulate growth and architecture, leading to abnormal tissue formation, while SAP54 induces the transformation of flowers into leaf-like structures, a phenomenon known as phyllody (Pecher *et al.* 2019). These alterations not only facilitate nutrient acquisition by the pathogen but also make the infected plant more attractive to insect vectors, thereby promoting the transmission of the bacteria to new hosts (Mondal 2017). The *Candidatus* Liberibacter species, including CLas, have also been found to produce effectors that suppress plant immune responses. For example, the CLas Sec-delivered effector 1 (SDE1) can inhibit immune-related protease activity, suppress reactive oxygen species (ROS) production, and even induce cell death in host plants. These multifunctional effectors, reminiscent of viral proteins, allow the pathogen to manipulate multiple host processes simultaneously, enhancing its survival and transmission (Mahanta *et al.* 2023; Zhao *et al.* 2023). Effectors from *Spiroplasma* remain largely unexplored, although it is known that this mollicute bacterium can produce antigenic variation at its surface through post-translational modifications, which may play an important role in insect colonization. The precise functions of *Spiroplasma* effectors in either plant or insect hosts remain an essential area of future research (Sheehan *et al.* 2018).

Vector and host interactions Vector-borne bacterial pathogens exhibit highly specialized interactions with plant and insect hosts, often deploying different effectors to manipulate each organism (Fig. 2). For instance, some phytoplasma and Liberibacter effectors are preferentially expressed in plant hosts, while others are primarily expressed in the insect vector. This differential expression pattern ensures that the pathogen can successfully switch between plant and vector environments, allowing it to thrive in both (Atabekova *et al.* 2023). The specific interactions between bacterial adhesins and insect gut receptors influence the transmission of these pathogens. Bacterial pathogens such as CLas use their surface proteins to bind to the gut epithelium of their insect vectors, enabling them to traverse the gut barrier and colonize other tissues, such as the salivary glands, before being transmitted to new plant hosts during feeding (Bento *et al.* 2021). Evading or suppressing insect immune responses is also crucial for bacterial survival within the vector. For example, CLas has been shown to downregulate genes involved in the Toll and Imd immune pathways in the *D. citri*, which enhances bacterial persistence and transmission (Jangra *et al.* 2024a).

Phytoplasmas and vector compatibility Phytoplasmas, a group of plant pathogens, belongs to the phylum Mycoplasmatota, class Mollicutes, only found in the internal organs of phloem-feeding hemipteran insects and the phloem sieve components of vascular plants. Phytoplasmas are primarily transmitted by insect vectors such as leafhoppers, planthoppers, and psyllids. The spread of phytoplasma-associated diseases is heavily influenced by various ecological factors, including the vectors' ability to acquire, inoculate, and disperse the pathogen (Hogenhout *et al.* 2008; Gurr *et al.* 2015). The efficiency of phytoplasma transmission can depend on factors like the phytoplasma load in the source plants and the life stage of the vector (nymphs versus adults). In some cases, transovarial transmission, where the pathogen is passed from parent to offspring may occur, affecting the timing and seasonality of inoculation (Alma *et al.* 2019; Weintraub *et al.* 2019). Vectors with narrow host ranges, such as monophagous and oligophagous species, are generally more efficient at spreading phytoplasmas than polyphagous species that feed on a wider variety of

plants. Additionally, factors like plant architecture, habitat structure, and ecotones (the transitional areas between different ecosystems) impact the movement and survival of these vectors (Harris and Maramorosch 2013). The flight activity and spatial distribution of vectors, which can vary between male and female insects, also play significant roles in the spread of phytoplasmas. Depending on the movement patterns of the vectors, phytoplasma epidemics may follow either an open or closed transmission cycle (Gurr *et al.* 2015).

A wide range of commercial and native plant species are susceptible to phytoplasma-caused diseases, ranging from minor to severe in their impact. However, insect vectors have been identified for only a fraction of known phytoplasma diseases, limiting the ability to make well-informed decisions about crop protection and the conservation of endangered plant species (Weintraub *et al.* 2006). Over the past two decades, a deeper understanding of insect vector-phytoplasma interactions has led to improved forecasting of disease distribution and prevalence. Examples of key diseases include grapevine flavescence dorée and palatinate grapevine yellows in Europe, aster yellows in the USA, sugarcane white leaf yellows in Southeast Asia, and coconut lethal yellowing in North and Central America. This knowledge is essential for devising effective disease management strategies and safeguarding agricultural productivity and biodiversity (West 2014; Alma *et al.* 2019).

Phytoplasmas, along with three vector-borne Mollicutes species in the genus *Spiroplasma* (Kwon *et al.* 1999), and the Alphaproteo-bacteria "*Candidatus* Liberibacter" taxa, have a unique life cycle among known plant pathogenic bacteria and are obligate residents of hosts representing two distantly related Kingdoms, plants (Plantae) and insects (Animalia). A hemipteran insect picks up the bacteria from an infected plant, carries them around in their bodies, allows them to replicate over several days, and then inoculates other plants (i.e., a persistent-propagative transmission mode) so that they can spread throughout the environment (Huang *et al.* 2020). Phytoplasmas are acquired by phloem-feeding insects during their feeding on phytoplasma-infected plants, however, a significant amount of time is required for the insect to acquire a sufficient titer of phytoplasma to establish an infection within its own body. This period is termed the "acquisition access period" (AAP) and can vary from minutes to hours (Weintraub *et al.* 2019).

In addition, the latent period (LP), also known as the incubation period, is the time it takes for a feeding insect to get infected with phytoplasma and transfer it to a plant. When an insect vector is exposed to phytoplasma during the LP, the insect's digestive system becomes infected with the pathogen (Tokuda *et al.* 2023). Once within the insect, these cells enter the hemolymph and begin circulating, ultimately infecting and multiplying in different insect organs. Phytoplasmas infect particular cells in the salivary glands, multiply, and are then transferred to plants by injections of saliva into the phloem tissue of the plant during feeding (Legg *et al.* 2014).

Meanwhile, researchers are also exploring a promising approach for controlling phytoplasma transmission by insect vectors. This approach involves manipulating the symbiotic bacteria present in these insects. Many arthropods, including insect vectors, rely on symbiotic microorganisms for essential life functions (Hogenhout *et al.* 2008; Weintraub *et al.* 2019). It is hypothesized that the symbiotic bacteria on the insect's body may be manipulated genetically to stop the spread of infections like phytoplasmas. Another hypothesis is that the carbohydrate-binding capabilities of substances like plant lectins may be utilized to disrupt the insect's diet and limit the vector's efficacy since phytoplasma vectors receive nutrition by feeding on phloem tissue (Hogenhout *et al.* 2008).

Immunity factor in the transmission of bacterial plant pathogens Vectors facilitate the spread of bacteria to plant hosts through a series of intricate biological processes. The interaction between vectors and pathogens is complex, with some bacteria providing fitness benefits to their insect vectors while others may have neutral or even detrimental effects (Fig. 2). Understanding these interactions' mechanisms is critical for controlling vector-borne plant diseases (Hogenhout *et al.* 2008). The process of bacterial transmission through vectors follows distinct phases: acquisition of the pathogen from infected plants, movement of the bacteria through the insect's tissues (including propagation in some cases), and the eventual inoculation of a new host plant. The tissue tropism of the bacteria within the vector, where the pathogen localizes and propagates, determines the efficiency and mode of transmission (Gurr *et al.* 2015). For example, in the case of the onion yellows phytoplasma, the anterior midgut and salivary glands of the leafhopper vector are the primary infection sites, while for CLas, which causes HLB in citrus, the pathogen must be acquired by psyllid nymphs for efficient transmission during the adult stage (Kruse 2019).

Research using advanced molecular techniques such as in situ hybridization, electron microscopy, and qPCR has provided valuable insights into the localization of bacterial pathogens within vector tissues. For instance, CLas induces apoptosis and nuclear disruption in the midgut and malpighian tubules of adult psyllids, which poses a barrier to bacterial acquisition (Huang *et al.* 2020). However, this phenomenon is not observed in nymphs, which can acquire and transmit the pathogen more efficiently. Studies on other vectors, such as the potato psyllid, reveal that certain bacterial species, like *Candidatus* Liberibacter solanacearum, do not cause midgut cell apoptosis, allowing adult psyllids to successfully acquire and transmit the bacteria (Heck 2018). These differences in cellular responses between nymphal and adult stages underscore the complexity of vector–pathogen interactions and highlight the potential for novel control strategies that manipulate vector immune responses to limit pathogen acquisition (Alma *et al.* 2019).

The relationship between vectors and the pathogens they transmit often reflects a co-evolutionary process (Fig. 2). In some cases, bacterial colonization confers benefits to the vector, while in others, the interaction may be neutral or even harmful (Eigenbrode *et al.* 2018). Studies on maize stunting pathogens, such as maize bushy stunt phytoplasma (MBSP) and *Spiroplasma kunkelii*, illustrate how insect vectors that have co-evolved with these pathogens, such as *Dalbulus maidis* and *D. elimatus*, are more tolerant to infection, whereas other leafhoppers, such as *D. gelbus* and *D. quinquenotatus*, often succumb to the pathogens

before transmission can occur. This suggests that long-term exposure to bacterial pathogens may drive the evolution of vector tolerance and fitness benefits (de Angeli Dutra *et al.* 2022). Bacterial pathogens exhibit varying degrees of host specificity depending on their vector associations. For instance, *Liberibacter* species are highly specific to psyllid vectors, and this specialization is reflected in their limited host range (Lin *et al.* 2024). By contrast, phytoplasmas can infect a wide variety of plant species and are transmitted by a large number of polyphagous insect species, including leafhoppers, planthoppers, and psyllids. On the other hand, *Spiroplasmas* exhibit a narrower host range and vector specificity, as they are transmitted by a limited group of leafhopper species. *X. fastidiosa* represents a unique case, as it can colonize a wide range of host plants, often without causing disease, and is transmitted by a broad array of xylem-sap-feeding insects, most of which are generalists. The broad host range of *X. fastidiosa* is likely facilitated by the low host-specificity of its vectors, which allows these xylem-feeding insects to transmit the pathogen across hundreds of different plant species (Ahmad *et al.* 2024).

The immune responses of insect vectors play a significant role in determining the success of pathogen transmission. Bacterial pathogens often must overcome vector immune defenses to establish infection. For example, CLas has been shown to modulate the immune response in its psyllid vector, *D. citri*, downregulating genes involved in the ubiquitin-proteasome and immune pathways (Huang *et al.* 2020). Comparative transcriptomic studies of infected and uninfected psyllids have revealed that CLas induces extensive transcriptional changes, particularly in the midgut, which is the first line of defense against bacterial invasion. By suppressing apoptosis and other immune responses, CLas enhances its ability to colonize the vector and ensures its transmission to new host plants (Pelz-Stelinski and Killiny 2016; Nian *et al.* 2024).

Vector-borne pathogens have evolved various strategies to manipulate the immune responses of their insect hosts. For instance, *Candidatus* Liberibacter species and phytoplasmas deploy specific suites of effector proteins to manipulate both plant and insect hosts (Shaw and Catteruccia 2019). These effectors are differentially expressed depending on the host, allowing the pathogens to adapt to the varying environments of plants and insects (Fig. 2). In addition, studies have shown that bacterial effector expression can vary depending on the genotype of the plant host, suggesting that the interaction between bacterial effectors and plant immune systems plays a critical role in determining the success of infection (Killiny *et al.* 2017).

7.4. Fungal pathogens and insect vector interaction

Fungal transmission by insects often involves more intricate biochemical and molecular interactions (Appendix C). For example, the adhesion of fungal spores to the insect cuticle is facilitated by specific proteins and polysaccharides, allowing the fungus to be transported to the insect's body (de Angeli Dutra *et al.* 2022). In the case of *Raffaelea* spp., spores adhere to the cuticle of ambrosia beetles through specialized proteins, enabling the beetles to transport fungal spores as they move between trees. Insects, such as bark beetles, that bore into plant tissues, create wounds, which serve as entry points for fungal pathogens. For instance, the boring activities of bark beetles in elms allow *Ophiostoma ulmi* to enter the vascular system of the tree, where it can establish infection (McLeod *et al.* 2005). Furthermore, some insects have developed specialized structures, like mycangia, for carrying fungal spores. Ambrosia beetles, for example, use mycangia to transport and cultivate fungal spores, creating a mutualistic relationship where the fungus serves as a food source for the developing beetle larvae. This symbiotic relationship enhances the efficiency of fungal transmission and infection, as seen with ambrosia beetles and their associated fungi. These mutualistic interactions highlight how insect vectors can facilitate the spread of fungal pathogens while also benefiting from the relationship (Fraedrich *et al.* 2008).

Furthermore, fungal pathogens transmitted by insects often produce secondary metabolites and virulence factors that aid in infection and colonization. For example, *Gibberella moniliformis* produces mycotoxins, such as fumonisins, which suppress the plant's immune system and facilitate colonization (Pusztahelyi *et al.* 2015). Similarly, *F. oxysporum* produces enzymes that degrade plant cell walls, allowing the fungus to penetrate plant tissues and evade host defenses. The combination of physical damage caused by insect vectors and the biochemical activities of fungal pathogens can lead to severe disease outbreaks in plants (Ahmad *et al.* 2024). Insects' immune responses also play a critical role in the transmission process. Insect vectors may mount immune defenses to limit fungal colonization, such as producing AMPs or activating the melanin pathway to encapsulate and kill fungal spores. However, specific fungal pathogens have evolved mechanisms to overcome these defenses. For instance, some fungi can evade or suppress insect immune responses, enabling them to survive within the insect and be transmitted to new plant hosts. In some cases, pathogens like *Pythium aphanidermatum* and *Thielaviopsis basicola* have been shown to benefit from passing through the insect gut, with their spores becoming more infectious after excreted (Stanghellini *et al.* 1999).

In greenhouses and controlled agricultural environments, insects can facilitate the spread of fungal and oomycete pathogens in closed ecosystems. For example, fungus gnats (*Bradysia impatiens*), shore flies (*Scatella stagnalis*), and moth flies (*Psychoda* spp.) have been implicated in the transmission of fungal and oomycete pathogens in greenhouses (El-Hamalawi 2008). The larvae of these insects may carry oospores, fungal spores, or other propagules, which can survive digestion and be transmitted to healthy plants (Hyder *et al.* 2009). This transmission can occur in seedling flats or hydroponic systems, where the limited mobility of larvae and restricted water movement can amplify the spread of the pathogens (de Angeli Dutra *et al.* 2022).

Insects spread fungal pathogens unintentionally, may pick up the fungus or its spores from infected plants they visit, either *via* direct contact or indirect contact (Willsey

et al. 2017). However, it is challenging to establish a strong relationship between root-feeding insects and the dispersion of fungus and oomycetes due to the restricted mobility of subterranean insects and the difficulty of recognizing interactions in the rhizosphere (Agrios 2008). As a result, scientists know very little about the vast majority of the insects that potentially serve as vectors for soil-dwelling fungi and oomycetes. When insects go from plant to plant in search of food, they also physically transmit the fungus and oomycetes that inhabit their feeding wounds (Fig. 2). *Fusarium* spp., (including *F. avenaceum*) identified from head capsules of *S. hispidulus* larvae have been connected to the spread of clover root borers (Leath and Hower 1993). There is some evidence that root borers transfer fungi like *Kabatiella caulivora* (Kirch.) Karak and *Colletotrichum trifolii* Bain, which may have ingested and then excreted. Given that injured roots are a source of attraction for root borers (Leath and Hower 1993), this correlation is especially fascinating since it shows that as the root borer population rises, so does their exposure to phytopathogenic fungi and, therefore, their effectiveness as carriers would also rise (Amman 1983).

7.5. Parasitic plants as vectors of plant pathogens

Parasitic plants are unique, highly specialized vascular plants that partially or fully rely on other plants for survival. These plants establish a vascular connection with their hosts through a structure called the haustorium, which allows them to extract water, minerals, and organic compounds from their host plants (Teixeira-Costa and Davis 2021). This parasitic behavior leads to significant agricultural challenges, as parasitic plants can cause severe yield losses by draining the resources of economically important crops.

Parasitic plants have evolved in various forms, classified as hemiparasites (partially photosynthetic) and holoparasites (non-photosynthetic) based on their photosynthetic ability. They are further divided into root and stem parasites, depending on their attachment site on the host plants (Těšitel 2016). Of the approximately 5,000 known species of parasitic plants, holoparasites make up around 10% of the total, while the remaining species are almost equally divided between roots and stem parasites.

Significant groups of parasitic plants include the Orobanchaceae family, which contains root parasites, and the Santalales order, which consists of stem parasites. Another well-known group is the genus *Cuscuta* (dodder), which contains over 200 species and is notorious for its broad host range and ability to parasitize multiple plants simultaneously (Teixeira-Costa and Davis 2021; Folimonova and Sun 2022; Chanchala 2025).

Parasitic plants exhibit a range of host specificity. Some species are highly specialized and infect only a few closely related host species, while others are generalists that can infect hundreds of plant species (Fig. 3). For instance, *Orobanche cumana* is specific to sunflowers, but may expand its host range based on environmental factors (Dor *et al.* 2020). On the other hand, generalists like *Cuscuta europaea* can parasitize over 200 host species, making them more effective vectors for pathogen transmission due to their broad interactions with multiple plant hosts. Parasitic plants like *Striga* (witchweeds) are among the most devastating, particularly in sub-Saharan Africa, where they cause billions of dollars in crop losses annually by infecting cereals such as rice, sorghum, maize, and millet (Těšitel 2016). Broomrapes (*Orobanche* and *Phelypanche*) are similarly destructive, affecting crops such as legumes, carrots, and tomatoes. The parasitic plant *C. campestris* is also considered a major agricultural pest, causing significant losses worldwide (Parker 2012).

The primary mechanism by which parasitic plants extract nutrients is through the haustorium, a specialized invasive organ that connects to the host plant's vascular system. The haustorium allows the parasite to draw water, minerals, and photosynthates from the host, and is often compared to grafting, as it creates a continuous connection between the host and the parasite (Okayasu *et al.* 2021). In root parasites like *Orobanche*, the haustorium can take the form of lateral or terminal structures, which can penetrate and establish connections with the host's xylem and, in some cases, phloem. Stem parasites like *Cuscuta* rely on similar haustorial structures to attach to their hosts, using hydrolytic enzymes to penetrate the host tissue and establish vascular connections. These connections are essential for nutrient transfer and serve as highways for the exchange of various macromolecules, including RNA, DNA, and proteins, between the host and parasite (Kokla and Melnyk 2018).

This vascular connection also opens the possibility for parasitic plants to act as vectors for phytopathogens, transferring viruses, phytoplasma, bacteria, and fungi from one host plant to another. The haustorial connection facilitates the movement of these pathogens, enabling parasitic plants to play a significant role in the transmission of diseases in agricultural systems (Kokla and Melnyk 2018). Studies have shown that *Cuscuta* species can act as reservoirs for multiple viruses and are capable of transmitting viruses between hosts. For example, *Cuscuta* has been reported to harbor over 50 different viruses, including cucumber mosaic virus (CMV), which has a wide host range and is transmitted by aphids. While some parasitic plants exhibit symptoms of viral infections, many do not, making them asymptomatic carriers of viruses (Scholthof *et al.* 2011).

Phytoplasma, which are phloem-restricted bacteria that cause devastating diseases in plants, can also be transmitted by parasitic plants. *Cuscuta* spp. and root parasites like *Orobanche* have been shown to acquire and transmit phytoplasma, contributing to the spread of diseases such as tomato stolbur and big bud (Kundu *et al.* 2024). The ability of parasitic plants to transmit phytoplasma is particularly concerning due to the wide host range of these pathogens and their capacity to cause significant crop losses. Despite their role as vectors, parasitic plants often exhibit few or no symptoms of phytoplasma infection, further complicating efforts to detect and manage these diseases (Shokri *et al.* 2023). Phytopathogenic bacteria, such as *R. solanacearum* and *X. campestris*, are responsible for some of the most destructive plant diseases and have also been associated with parasitic plants. While the role of parasitic plants in bacterial transmission is less well-documented, there is evidence that parasitic plants can acquire bacterial pathogens

from their hosts and act as reservoirs for future infections. Interestingly, extracts from parasitic plants have been shown to exhibit antibacterial properties, suggesting a complex interaction between parasitic plants and bacterial pathogens (Scholthof *et al.* 2011).

Fungi are the most significant group of pathogens in terms of agricultural yield losses, and parasitic plants can both acquire and transmit fungal infections. *Cuscuta gronovii* has been reported to exhibit symptoms of fungal infection, such as necrotic lesions, while fungal pathogens like *F. oxysporum* have been explored as biological control agents for parasitic plants. Fungal pathogens from the *Fusarium* and *Alternaria* genera have shown promise in controlling the growth and germination of parasitic plants, offering a potential strategy for managing these destructive weeds (Gibot-Leclerc *et al.* 2022).

The transmission of phytopathogens through parasitic plants occurs *via* multiple routes (Fig. 2). One major pathway is plant-to-plant transmission, where parasitic plants acquire pathogens from an infected host and transmit them to other plants through the haustorial connection. This process is particularly effective in generalist parasitic plants like *Cuscuta*, which can simultaneously infect multiple host species, facilitating the spread of pathogens across diverse plant communities (Kundu *et al.* 2024). Another route of transmission involves arthropods, such as aphids and leafhoppers, which feed on both parasitic and host plants. These insects are known vectors for viruses and phytoplasma, and their interactions with parasitic plants may enhance the spread of pathogens between plant species (Sarwar 2020b). Seed transmission is another potential pathway for the dissemination of phytopathogens by parasitic plants. Many parasitic plants, particularly root parasites like *Orobanche*, produce large quantities of seeds that can remain viable in the soil for decades, providing a long-term reservoir for phytopathogens. While seed transmission of viruses, bacteria, and fungi is less well-studied, the presence of pathogens in parasitic plant seeds raises concerns about the role of parasitic plants in the long-term spread of diseases (Rubiales *et al.* 2009).

To manage the spread of parasitic plants and the pathogens they carry, researchers have explored the use of phytopathogens as biocontrol agents. Fungal pathogens, particularly species from the *Fusarium* genus, have shown great potential in controlling parasitic plants like *Striga* and *Orobanche* (Singh 2018). These biocontrol agents work by infecting the parasitic plants and inhibiting their growth or germination. For instance, strains of *F*. *oxysporum* have been highly effective in reducing *Striga* infestations, which are a major threat to cereal crops in Africa. Additionally, bacterial isolates from the *Bacillus* and *Pseudomonas* genera have shown inhibitory effects on the seed germination of parasitic plants, offering another avenue for biological control (Baker *et al.* 2024). Parasitic plants are not only direct threats to agricultural productivity but also act as vectors for a range of phytopathogens. Their haustorial connections enable the transmission between plants, contributing to the spread of diseases in agricultural systems. Understanding the complex interactions between parasitic plants, their hosts, and phytopathogens (Fig. 3) is critical for developing effective management strategies. Biocontrol agents, such as phytopathogenic fungi and bacteria, offer promising solutions for controlling parasitic plant infestations and mitigating their impact on crop yields (Parker 2012; Kokla and Melnyk 2018).

8. Vector-borne diseases and plants interactions

Plants possess sophisticated immune systems that recognize insect vectors and the bacterial pathogens they transmit. These immune responses involve both surface-localized PRRs and intracellular nucleotide-binding leucine-rich repeat (NLR) receptors, which detect microbial-associated molecular patterns (MAMPs) and herbivore-associated molecular patterns (HAMPs) released during insect feeding and pathogen invasion (Pel and Pieterse 2013). When hemipteran insects, such as psyllids and leafhoppers, penetrate the plant's apoplast with their stylets, they release watery saliva containing HAMPs, MAMPs, and bacterial effectors. These molecules activate the plant's immune system, triggering a cascade of defense responses aimed at restricting pathogen spread (Zhao *et al.* 2023; Jangra *et al.* 2024a). Watery saliva, secreted during vector probing, contains effectors that suppress plant immunity, while honeydew, excreted by the insect, carries additional MAMPs and HAMPs that further influence plant defenses (Zhao *et al.* 2023). When PRRs on the plant surface recognize these molecules, they initiate a range of immune responses, including the production of ROS and calcium (Ca^{2+}) signaling. Calcium bursts induced by vector feeding are critical in activating downstream defense pathways, resulting in the production of callose at sieve plates and the activation of phloem-specific proteins such as forisomes and sieve element occluding relatives (SEOR) proteins. SEOR proteins form aggregates at feeding sites to block phloem flow, limiting pathogen transmission (Chiquito-Contreras *et al.* 2024).

Callose deposition plays a pivotal role in restricting the movement of phloem-limited pathogens like *Candidatus* Liberibacter spp. and *Phytoplasmas*. For example, CLas infection triggers the expression of multiple callose synthases in citrus, including CalS3, CalS7, and CalS9, resulting in the formation of hydrogels that restrict bacterial movement without impairing mass flow (Zhang *et al.* 2025). However, the role of SEOR proteins in plant defense is complex, as recent research has shown that mutations in the SEOR1 gene reduce the growth of certain phytoplasma strains, suggesting that these proteins may also act as susceptibility factors (Huang *et al.* 2020). The phloem serves as a highway for systemic immune signaling, allowing plants to coordinate defense responses across distant tissues. Rapid waves of calcium signaling travel through the phloem in response to vector feeding and pathogen invasion, facilitating the deployment of immune responses in non-infected tissues (Reyes Caldas *et al.* 2022). Advances in single-cell RNA sequencing and vascular proteomics are helping to identify genes and proteins involved in these responses, providing new insights into how plants regulate immune responses at the cellular level (Ziegler-Graff and Brault 2008).

In the xylem, plants respond to bacterial invasion by forming tyloses, which are outgrowths of living parenchyma cells that block xylem vessels. Although tyloses are common defense structures, they are not always effective (Zhang *et al.* 2025). In interactions with *X. fastidiosa*, for example, tyloses can exacerbate disease symptoms by obstructing water flow, leading to wilting and dieback. The delayed immune recognition of *Xylella* may be due to the pathogen's surface lipopolysaccharides, which mask the bacterial cells and delay immune activation (Schneider *et al.* 2020; Morelli *et al.* 2021).

Plant genome sequencing projects have enabled quantitative studies of host–pathogen interactions, revealing genotype-specific responses to vector-borne bacterial pathogens. For instance, research on citrus responses to CLas has identified differential expression of defense-related proteins and carbohydrate metabolism pathways, with some responses varying across citrus cultivars (Reyes Caldas *et al.* 2022; Sanches *et al.* 2023). However, capturing vascular-specific immune responses remains challenging, as bulk tissue analysis may obscure the unique responses occurring within phloem and xylem cells. Technologies such as RNA aptamers and single-cell RNA sequencing are beginning to address these limitations, enabling researchers to explore immune responses at the level of individual vascular cells (Huang *et al.* 2020). Plants deploy a complex immune strategy to defend against vector-borne bacterial pathogens, involving localized and systemic responses. Understanding how plants detect and respond to insect vectors and the pathogens they transmit is essential for developing sustainable disease management strategies. These insights offer new avenues for improving plant resistance through genetic engineering and breeding programs, providing alternatives to chemical insecticides and helping to safeguard agricultural ecosystems from the threat of vector-borne diseases (Javed *et al.* 2021; de Angeli Dutra *et al.* 2022).

9. Conclusion

The intricate dynamics of plant–pathogen interactions and the pivotal role of insect vectors are paramount in managing and controlling plant diseases that pose significant threats to global agriculture and ecosystems. Plant diseases can lead to substantial economic losses and compromise food security worldwide, making it essential to delve deeply into the mechanisms that facilitate pathogen transmission (Fig. 4). This comprehensive review has dissected the complex processes by which pathogens are transmitted by insect vectors, highlighting the distinctions between noncirculative and circulative transmissions and underscoring the molecular and biochemical interactions that govern these processes. In noncirculative transmission, the specificity of protein–protein interactions is crucial for successfully transferring pathogens. Viral pathogens, for instance, often rely on directly binding their CPs to specific receptors located on the insect vector's stylet, the piercing-sucking mouthpart used during feeding. The viral protein P2 has been identified as a critical player in this process, interacting specifically with stylet receptors in a manner that determines both the specificity and efficiency of transmission. Molecular variations in these proteins and environmental conditions can influence the binding efficiency, affecting transmission rates. Understanding these nuances is critical, as it can aid in developing targeted strategies to interrupt the transmission cycle at this critical point, such as designing molecules that block these protein–protein interactions.

Conversely, circulative transmission involves a more elaborate journey of the pathogen through the vector's body. After ingestion, the pathogen must cross the gut barrier, a process mediated by specific proteins that facilitate passage through the gut epithelium while evading digestive enzymes and immune responses. Proteins such as the GroEL chaperone in *B. aphidicola* (an endosymbiont of aphids) protect the virus during its transit. Once in the hemolymph, the pathogen navigates through the vector's circulatory system, utilizing transport proteins to reach the salivary glands. Salivary gland invasion is another critical step, where the pathogen must again cross cellular barriers to be introduced into plant hosts during subsequent feeding. Molecular and biochemical mechanisms, including the regulation of specific genes and replication in propagative pathogens, are essential for successful transmission. These processes often involve intricate signal transduction pathways and manipulation of the vector's metabolic pathways, allowing the pathogen to exploit the vector's biology to its advantage. The ability of pathogens to manipulate insect vector behavior and physiology adds another layer of complexity to these interactions. Pathogens can induce physiological changes in vectors, such as altering feeding behavior to increase the frequency or duration of feeding events, thus enhancing transmission opportunities. For example, infected aphids may exhibit increased probing behavior, leading to more frequent virus transmission. Pathogens may also affect reproduction rates and lifespan enhancements, often by suppressing the vector's immune responses or regulating hormonal pathways. Essential genes and proteins, such as CPs, movement proteins (MP), bacterial effector proteins, and transport and immune proteins, have been identified as instrumental in these manipulations. Bacterial effectors, for instance, can modulate the vector's gene expression to suppress immune responses or alter metabolic functions. These molecular interactions offer potential targets for intervention; by disrupting these processes, it may be possible to reduce pathogen spread without adversely affecting the vector populations necessary for ecological balance.

Diverse insect vectors, including aphids, planthoppers, leafhoppers, thrips, and whiteflies, play significant roles in transmitting various pathogens. For example, *B. tabaci* are known vectors for numerous plant viruses, such as begomoviruses, which cause devastating diseases in crops like tomatoes and cotton. Specific whitefly proteins interact with viral components to facilitate transmission, with factors like the *B. tabaci*'s GroEL homolog playing a protective role for the virus. The "plant–virus–vector" interactions are multifaceted, involving the physical transfer of pathogens and intricate biochemical and molecular dialogues that determine transmission efficiency and specificity. Understanding the compatibility factors in phytoplasma transmission is crucial, as phytoplasmas require specific vector species for

transmission, governed by interactions between phytoplasma effector proteins and vector proteins. Similarly, immunity factors affecting bacterial pathogen spread highlight the importance of vector immune responses in determining transmission success. Insect vectors are also responsible for the transmission of bacterial and fungal pathogens. The effector biology of bacterial pathogens, such as *Candidatus* Liberibacter spp., involves manipulating plant host cells to facilitate infection and transmission. These bacteria produce effector proteins that alter plant physiology, making the plant more conducive to bacterial survival and spread. Vector and host interactions can influence the spread of these pathogens, with factors such as vector feeding habits, host plant resistance, and environmental conditions playing critical roles. In the case of fungal pathogens, insect vectors can facilitate the spread of spores; for example, beetles can carry fungal spores of *Ophiostoma* species, contributing to Dutch elm disease. Interactions between fungal structures and insect tissues influence transmission dynamics, with factors like adhesion mechanisms and spore viability affecting the spread efficiency.

Given the complexity and adaptability of these systems, sustainable strategies for managing insect vectors are essential. Traditional reliance on chemical pesticides is becoming less effective due to the development of resistance among insect populations and growing environmental and health concerns. Therefore, approaches that integrate an understanding of molecular interactions hold significant promise. Disrupting protein binding's essential for transmission, such as blocking viral CPs and insect receptor interactions using competitive inhibitors or RNA interference (RNAi) techniques, could prevent pathogens from establishing in vectors. Enhancing plant immune responses through genetic engineering or breeding for resistance can reduce susceptibility to pathogens, for example, by introducing genes that strengthen plant defenses or interfere with pathogen recognition. Additionally, manipulating signal transduction pathways and metabolic processes within vectors could reduce pathogen spread by impairing the vector's ability to harbor or transmit the pathogen. For instance, silencing genes essential for pathogen passage through the gut or salivary glands could be a viable strategy. Biological control methods, such as introducing natural predators, parasitoids, or entomopathogenic fungi that target insect vectors, offer environmentally friendly alternatives that can be integrated into pest management programs.

In conclusion, a comprehensive grasp of the mechanisms governing plant–pathogen–vector interactions is critical for developing innovative and sustainable disease management strategies. The intricate molecular and biochemical interactions between pathogens, vectors, and plants present challenges and opportunities for researchers and agricultural practitioners. Continued research is necessary to unravel the mysteries of these complex relationships, particularly in the face of emerging pathogens and changing environmental conditions due to climate change.

A multidisciplinary approach that combines molecular biology, ecology, entomology, plant sciences, genomics, and bioinformatics is essential to advance our understanding. Collaborative efforts can lead to developing novel control strategies, such as using biotechnological tools to create resistant plant varieties or deploying RNAi-based approaches to disrupt pathogen transmission. Such efforts will be instrumental in safeguarding crop health and ensuring food security in the face of evolving plant diseases. By advancing our understanding and developing targeted interventions, we can mitigate the impact of plant diseases on global agriculture and promote sustainable ecosystem management. Addressing these challenges has economic benefits and contributes to environmental conservation and the well-being of societies dependent on agricultural productivity. Integrating scientific knowledge into practical applications will be vital in overcoming the threats posed by plant pathogens and their insect vectors, ultimately supporting the resilience and sustainability of global food systems.

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Declaration of competing interest

The authors declare that they have no conflict of interest.

Declaration of generative AI and AI-assisted technologies in the writing process

The authors declare that they did not use AI in the preparation and writing of this manuscript.

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