ONLINE JOURNAL OF BIOLOGICAL SCIENCES

REVIEW ARTICLE

Criniviruses at the Crossroads of Virology and Agroecology: Molecular Dialogues, Vector Synchrony, and Translational Control Strategies in Crop Systems

Md. Abdullah Al Sabbir, Nabela Akter, Ankita Saha, Md. Motaher Hossain*

Department of Plant Pathology, Gazipur Agricultural University, Gazipur, Bangladesh

*Corresponding Author: hossainmm@gau.edu.bd

Abstract: Criniviruses, members of the Closteroviridae, illustrate how molecular evolution, vector ecology, and climatic variability converge to determine plant virus emergence and persistence. Although individual genomic and transmission traits are well studied, their cross-scale integration has remained limited. Recent advances in comparative genomics and high-throughput sequencing have revealed that bipartite genome reassortment, recombination hotspots, and quasi-species microdiversity provide the genetic flexibility that sustains crinivirus adaptation across hosts. Parallel discoveries in vector biology show that endosymbiont-mediated modulation within *Bemisia tabaci* and *Trialeurodes vaporariorum* strongly influences viral retention and transmissibility. Coupled with climate-driven shifts in vector distribution and the microclimatic stability of protected cultivation systems, these processes collectively explain current expansion patterns of crinivirus diseases. Integrating molecular, ecological, and spatial perspectives offers a basis for predictive risk modeling and climate-responsive management, supported by advances in field-deployable diagnostics, precision breeding, and endosymbiont-targeted control strategies. Together, these developments frame a systems-level understanding of crinivirus epidemiology that aligns plant virology with adaptive, sustainable crop protection.

Keywords: Crinivirus, Vector Dynamics, Segment Reassortment, Endosymbiont Modulation, Climate Driven Epidemiology, Agroecological Modeling

Received: 13-11-2025 | **Revised:** 14-06-2026 | **Accepted:** 22-08-2026 | **DOI:** 10.3844/ojbsci.2026.26.03.076

Introduction

Criniviruses are a genetically distinct genus within the family Closteroviridae, characterized by a bipartite, positive-sense single-stranded RNA genome and a strict tropism for phloem tissues [1, 2]. Unlike members of the genera Closterovirus and Ampelovirus, criniviruses rely primarily on whitefly vectors for transmission, most notably *Bemisia tabaci* (MED, MEAM1) and *Trialeurodes vaporariorum*. They are transmitted in a semi-persistent manner, a vector interaction strategy that critically influences their field spread, host range, and overall epidemiological dynamics [3, 4]. Phylogenetic analyses based on conserved genomic regions including the RNA-dependent RNA polymerase (RdRp), heat shock protein 70 homolog

(HSP70h), and Coat Protein (CP) support the monophyly of crinivirus species and clearly separate them from other closteroviral lineages [5-7]. Importantly, the bipartite genome architecture of criniviruses enables functional partitioning, with RNA1 dedicated to replication and RNA2 governing movement and encapsidation. This modular organization provides adaptive flexibility during host colonization and intercellular trafficking [8]. This genomic configuration likely emerged under co-evolutionary pressure from vector specialization. Comparative analyses suggest that divergence within the genus was shaped by transmission efficiency optimizations toward specific whitefly species, paralleled by expansion into a broad range of dicotyledonous hosts [9]. In response to expanding molecular and ecological knowledge, the International Committee on Taxonomy of Viruses [2] has updated genus-level classification criteria. These now include genome organization, amino acid identity thresholds, and distinct whitefly-mediated transmission phenotypes to better demarcate species boundaries within Crinivirus.

Criniviruses represent a growing threat to vegetable production worldwide due to their bipartite RNA genomes and phloem-specific tropism [10, 11]. While all species share these fundamental traits, variations in host range, accessory movement proteins, and viral suppressors of RNA silencing create species-specific epidemiological outcomes [12]. Intensive agricultural systems have inadvertently created ecological conditions favorable for the emergence and persistence of criniviruses. Greenhouse environments extend crop duration, increase relative humidity, and reduce the abundance and diversity of natural enemies that collectively support elevated whitefly populations and enhance virus transmission rates [3, 12]. Concomitantly, intensive monoculture in high-input vegetable production systems reduces host genetic diversity and limits the activation of innate defense mechanisms. This reduction in genetic and physiological resilience renders crops highly susceptible to phloem-limited viruses, including criniviruses [13, 14]. Both of these threats are compounded by the spread of *Bemisia tabaci* around the world, supported by the rising temperatures and expanding agricultural frontiers (Figure 1). Climate warming enhances thermal suitability for whitefly reproduction, survival, and distribution, especially in tropical and subtropical agroecosystems where overlapping vector phenology and continuous cropping windows maximize infection pressure [15]. Compounding the problem is the widespread development of insecticide resistance in *B. tabaci* populations, which undermines conventional control strategies and enables long-term vector persistence [16]. Additionally, the international trade of vegetatively propagated materials such as seedlings that appear asymptomatic yet harbor latent infections serves as an efficient pathway for cross-border Crinivirus dissemination [17, 18]. The synchrony between whitefly population peaks and stages of crop susceptibility in controlled or year-round cultivation systems results in high viral transmission efficacy [7]. Emerging outbreaks in sub-Saharan Africa and South Asian regions further underscore the global relevance of these viruses [10].

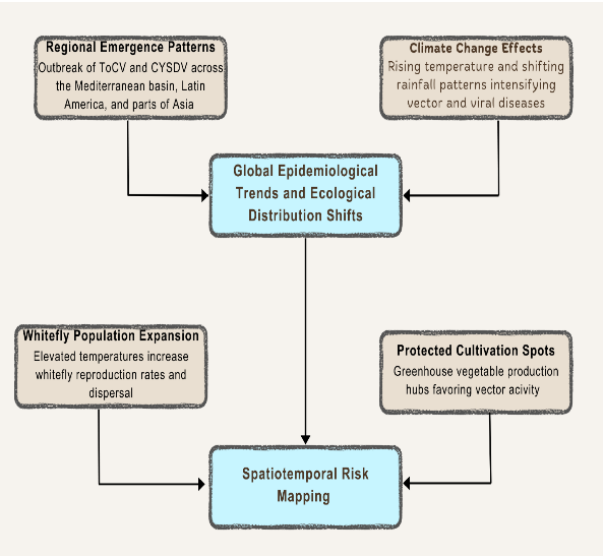

Fig. 1: Conceptual model of the global epidemiological trends and ecological distribution shifts in Crinivirus infections. (a) Regional Emergence Patterns highlight increasing outbreaks of Tomato chlorosis virus (ToCV) and Cucurbit yellow stunting disorder virus (CYSDV) across the Mediterranean basin, Latin America, and parts of Asia; (b) Climate Change Effects, including rising temperatures and altered precipitation regimes intensifying vector activity; (c) Whitefly Population Expansion driven by elevated thermal conditions; (d) Protected Cultivation Spots, particularly greenhouse-based vegetable production systems, serve as ecological refuges for whiteflies and hotspots for virus transmission; Spatiotemporal Risk Mapping frameworks essential for forecasting and managing Crinivirus epidemiology under global change scenarios

Amidst the escalating threat of emerging and re-emerging plant viruses, Criniviruses demand urgent attention due to their expanding geographic distribution, broadening host range, and persistent challenges in intensive horticultural systems. Despite substantial progress in understanding their bipartite genome organization and whitefly-mediated transmission, critical gaps remain in linking molecular evolution, intra-host microdiversity, and vector ecology to field-level epidemiology. This review addresses these gaps by synthesizing recent advances in Crinivirus molecular biology, vector virus host interactions, and climate-driven epidemiological shifts, while highlighting unresolved questions concerning transmission efficiency, host adaptation, and outbreak predictability. Furthermore, we critically evaluate persistent diagnostic bottlenecks and emerging surveillance technologies, including AI-driven risk forecasting and spatial epidemiology tools, to enhance early detection and proactive management. The review also emphasizes the underexplored role of vector endosymbionts and behavioral ecology in shaping virus persistence and epidemic dynamics. Structured around four integrative domains:

(i) Molecular and Evolutionary Foundations

(ii) Host Vector Virus Dynamics

(iii) Epidemiological Transitions under Climate Stress

(iv) Sustainable and Predictive Management; this review positions itself as a timely resource for advancing strategic research and translational applications in the Crinivirus pathosystem

Methodology

This review was conducted using a structured narrative approach to evaluate published research on crinivirus biology, virus–vector interactions, epidemiology, and disease management. Literature searches were performed in Web of Science, Scopus, PubMed, CAB Abstracts, and Google Scholar. Search terms included Crinivirus, *B. tabaci*, virus transmission, molecular evolution, endosymbionts, epidemiology, climate change, and disease management. Publications from 2000 to 2026 were considered. Earlier studies were included when relevant to taxonomy, genome organization, or transmission biology. Studies were selected based on their relevance to crinivirus genomics, host virus interactions, vector ecology, epidemiology, diagnostics, and management. Peer-reviewed articles, review papers, and authoritative reports were included. Duplicate records, studies lacking sufficient methodological information, and publications unrelated to crinivirus pathosystems were excluded. Titles, abstracts, and full texts were screened for relevance before inclusion. Due to substantial variation in study objectives, experimental systems, host species, vector populations, and reported outcomes, quantitative meta-analysis was not appropriate. Evidence was therefore evaluated through comparative narrative synthesis, with emphasis on recurrent patterns, major advances, unresolved questions, and research gaps across molecular, ecological, and epidemiological aspects of crinivirus research.

Architectural and Functional Dissection of the Crinivirus Genome

Functional Architecture and Partitioning of the Bipartite Genome

The bipartite organization of Crinivirus genomes (RNA1 and RNA2), each encapsidated separately, yet both essential for successful infection and systemic spread. RNA1 encodes the replication-associated proteins, including a methyltransferase (MET), a helicase (HEL), and an RNA-dependent RNA polymerase (RdRp), forming the core viral replication complex [19-21] In contrast, RNA2 carries genes essential for virion assembly and intercellular movement, including the major and minor coat proteins (CP, CPm), a heat shock protein 70 homolog (HSP70h), a ~60 kDa protein, and several movement-associated factors (Figure 2) [18]. These proteins are typically translated from subgenomic RNAs (sgRNAs) generated during infection, driven by internal promoters upstream of respective open reading frames, enabling temporal and tissue-specific expression during systemic colonization [19]. The bipartite genome organization of Criniviruses enables modular separation of replication (RNA1) and movement/encapsidation (RNA2) functions, enhancing adaptability to hosts and vectors [22]. It provides a modular genomic strategy that not only permits efficient control of gene expression but also supports genomic plasticity during host adaptation and vector transmission [23]. Evidence from recombinant genome studies and reassortment events suggests that this bipartition may facilitate evolutionary flexibility through inter-strain segment exchange, conferring novel transmission efficiencies or host range expansion [24]. Intergenic and intersegmental junctions particularly the conserved 3′ untranslated regions (UTRs) are implicated in replication regulation, subgenomic RNA synthesis initiation, and possible recombination hotspots, thus serving as key regulatory nodes [19, 25]. Importantly, the replication-movement coordination between RNA1

and RNA2 is tightly regulated; disruption of this balance often results in attenuated infection, highlighting the interdependence of these two genomic segments in viral fitness [26, 27]. The bipartite genome organization of criniviruses enables the independent evolution of RNA1 and RNA2, thereby enhancing genetic diversification [24]. Combined effects of mutation, recombination, and reassortment generate diverse viral populations capable of responding rapidly to host, vector, and environmental selection pressures [28, 29]. Such genetic microdiversity contributes to viral persistence, adaptation, and the emergence of novel epidemiological variants across agroecosystems [30].

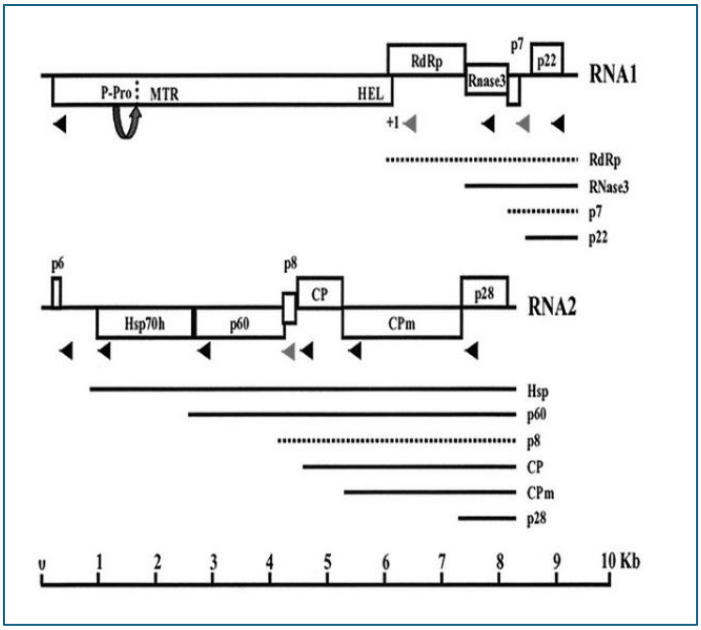

Fig. 2: Genomic architecture of SPCSV and the expression of sgRNAs. The genomic RNA1 and RNA2 are depicted as a line, with the Open Reading Frames (ORFs) represented by boxes. Open Reading Frames (ORFs) are depicted above, below, or within the line, signifying their presence in various reading frames. On RNA1, +1 denotes a potential +1 ribosomal frameshift location [26]

Accessory Proteins Mediated Systemic Movement and Regulatory Complexity

Beyond their core replication and encapsidation proteins, Crinivirus genomes encode a set of accessory proteins that play pivotal roles in facilitating systemic movement, modulating host responses, and enhancing vector transmissibility [31]. Several of these proteins arise from non-canonical Open Reading Frames (ORFs), often embedded within overlapping coding regions and translated via leaky scanning or non-AUG initiation [32]. In Tomato Chlorosis Virus (ToCV), small ORFs such as p4 and p8 have been implicated in modulating the stoichiometry of downstream structural proteins by acting as translational gatekeepers, ensuring optimal expression ratios during different infection stages [10]. Among the most critical viral tools deployed during infection, Viral Suppressors of RNA silencing (VSRs) play a central role in antagonizing host antiviral defense mechanisms. The P22 protein of Sweet Potato Chlorotic Stunt Virus (SPCSV) exemplifies a potent VSR, capable of sequestering small interfering RNAs (siRNAs) and impeding the systemic spread of RNA silencing signals, thereby facilitating long-distance viral movement [26, 33-34]. Other VSRs, such as p20, have been shown to interfere with Argonaute-mediated slicing and siRNA stabilization [35]. These suppressors often display multifunctionality, engaging in both immune evasion and interactions with host translation machinery.

Complementing the function of these suppressors, a hallmark feature of criniviruses is the expression of a HSP70h encoded on RNA2. HSP70h localizes to the Endoplasmic Reticulum (ER) and plasmodesmata, where it mediates virion intracellular trafficking, assists in virion tail formation, and facilitates systemic phloem loading [36]. This protein not only supports virion stabilization for whitefly acquisition but also interacts with plant cytoskeletal elements to assist in directional movement [37]. Together, these regulatory elements non-canonical ORFs, VSRs, and HSP70h form a coordinated network that governs infection dynamics. They allow criniviruses to fine-tune replication, suppress host defense, and ensure successful colonization and transmission. Their modular evolution and multifunctionality provide insights into how these viruses adapt to new hosts and vectors under shifting ecological pressures.

Translational Regulation and Inter Virus Synergy

Crinivirus gene expression is also fine-tuned by several levels of translational control. Ribosomal leaky scanning enables the downstream ORFs to avoid upstream ORFs and leads to balanced translation of replication-related and structural proteins [38]. Programmed −1 ribosomal frameshifting increases coding capacity, allowing for precise production of replication-related polyproteins in the amounts needed for efficient infection [39]. Complementing this, many criniviruses employ programmed ribosomal frameshifting, particularly the −1 frameshift, to expand coding capacity without increasing genome size. For instance, RNA1-encoded polyproteins involved in replication are often synthesized through frameshift events at conserved slippery sequences and downstream pseudoknot motifs [40]. Further regulatory sophistication is achieved via subgenomic RNAs (sgRNAs), which allow temporal and tissue-specific expression of structural proteins such as the major Coat Protein (CP), minor CP, and HSP70 homolog (HSP70h). These sgRNAs act as transcriptional hubs that respond dynamically to intracellular cues and vector-transmission cycles [29, 41].

During mixed infections, inter-virus synergy can amplify crinivirus RNA accumulation through mutual exploitation of host translation factors, including ribosomal subunits and *eIF4E/eIF(iso)4G* complexes [42]. For example, in co-infections of ToCV with potyviruses, viral suppressors of RNA silencing (VSRs) may jointly suppress host antiviral responses, while translational enhancers facilitate cross-viral access to the translational apparatus [40, 43]. These co-infections not only enhance crinivirus replication and systemic spread but also alter symptomatology, often intensifying host damage and complicating diagnosis and control.

Molecular Evolution and Vector Ecology Integration

The evolutionary dynamics of criniviruses are governed by the interplay between genomic diversification and vector-mediated selection. Continuous mutation, recombination, and reassortment between the bipartite genome segments generate genetically heterogeneous viral populations. It provides a reservoir of variants with distinct adaptive potential [24, 44]. Such genetic flexibility facilitates adaptation to diverse hosts, vectors, and agroecological conditions. However, only a subset of this diversity contributes to successful transmission. During acquisition, retention, and inoculation, whitefly vectors impose selective bottlenecks that preferentially favor variants with enhanced transmission competence and ecological fitness. Consequently, vector transmission functions not only as a dispersal mechanism but also as an evolutionary filter that shapes viral population structure and persistence [28, 4].

Vector ecology further modulates these evolutionary processes. Differences in transmission efficiency among *B. tabaci* cryptic species and *T. vaporariorum* together with variation in vector abundance, host utilization, and dispersal capacity, influence the frequency and outcome of virus vector encounters [9]. These ecological pressures can alter genotype frequencies within viral populations and favor the persistence of transmission-adapted variants. Collectively, molecular diversification generates adaptive potential, whereas vector-mediated selection determines which variants successfully establish and spread. The integration of these evolutionary and ecological processes provides a mechanistic framework for understanding crinivirus adaptation, emergence, and long-term epidemiological success across agricultural ecosystems.

Precision Vectoring and Viral Modulation of *Bemisia tabaci* and *Trialeurodes vaporariorum*

Proteome Level Virus Vector Interactions

The vector specificity of Crinivirus transmission by *Bemisia tabaci* and *Trialeurodes vaporariorum* is governed specific and selective interactions between the virion structural proteins and receptors in the midgut of virus vectors [9]. The virions are present in the anterior midgut as shown in Figure 3 and the filter chamber and the CP and minor coat protein (CPm) bind to membrane-associated proteins to form stable complexes. Studies using immunolocalization and proteomic binding assays have confirmed that crinivirus virions accumulate in these regions, where CPm interacts with midgut membrane-associated glycoproteins to mediate virus retention and protect against proteolytic degradation during passage [9, 45]. Proteomic analyses have identified sugar transporters and lectin-domain-containing proteins as candidate CPm-interacting proteins in Cucurbit chlorotic yellows virus CCYV-transmitting whiteflies. These host molecules likely function as receptors, mediating viral sequestration and circulative movement across the insect gut barrier [46].

The semi-persistent, non-propagative nature of Crinivirus transmission requires that virions traverse the midgut epithelium into the hemolymph and eventually reach the salivary glands without undergoing replication within the insect host [47-48]. Proteomic remodeling of whitefly gut epithelial tissues following virus acquisition reveals upregulation of receptor and effector

proteins, including cuticle-associated proteins and mucin-like molecules that contribute to virion stability and trafficking [49]. Notably, the transmission efficiency of criniviruses can vary between cryptic species of *B. tabaci* due to differential expression of gut-expressed viral receptors, as demonstrated in transcriptomic comparisons [50]. Functional antibody-blocking experiments targeting CPm–midgut protein complexes significantly reduced viral acquisition and transmission, functionally validating the central role of these interactions in crinivirus epidemiology [11]. These data underscore the importance of proteome-level specificity and post-acquisition molecular remodeling in vector competence, revealing potential targets for disruption in integrated vector management strategies.

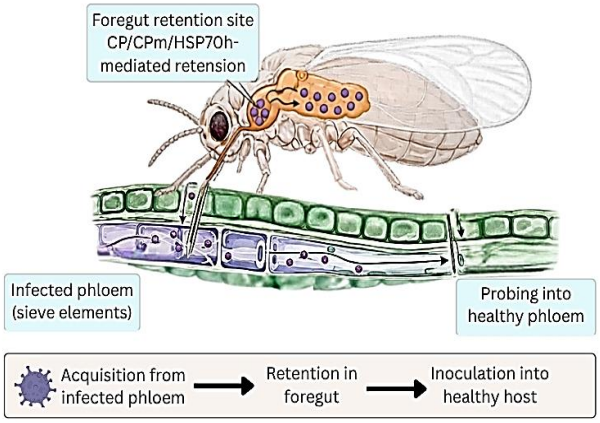

Fig. 3: Circulative pathway of criniviruses in whitefly vectors. Entry of criniviruses in the whitefly foregut during phloem feeding and translocation through the midgut into the hemocoel; Further reach the salivary glands via the hemolymph for subsequent transmission to new host plants; a circulative, non-propagative pathway, typical of semi-persistent virus transmission

Endosymbiont Mediation of Vector Competence

In addition to virus-induced proteomic changes within whitefly tissues, endosymbiotic bacteria constitute a distinct regulatory layer affecting vector competence and transmission efficiency. Endosymbiotic microorganisms in *B. tabaci* exert profound influence on the vector's capacity to acquire, retain, and transmit criniviruses. While the primary obligate symbiont *Portiera aleyrodidarum* fulfills essential nutritional functions, several facultative symbionts like *Hamiltonella defensa*, *Rickettsia, Arsenophonus, Wolbachia*, and *Cardinium* play direct and indirect roles in modulating vector competence through proteomic, immunological, and metabolic pathways [51-53].

Hamiltonella defensa has been most notably implicated in viral transmission. It produces GroEL-like chaperonins that bind to virions in the whitefly hemolymph, stabilizing them and preventing degradation prior to salivary gland passage, thereby enhancing systemic transmission efficiency [54, 55]. Similarly, *Rickettsia* spp. modulate host gene expression in midgut and salivary glands, with transcriptome-wide changes linked to increased viral acquisition and fecundity in infected *B. tabaci* populations [56]. *Arsenophonus* influences lipid metabolism and alters membrane fluidity in the gut epithelium, facilitating CPm-receptor binding by enhancing virion docking and passage across the midgut barrier [57]. These symbionts not only support viral circulative persistence but also suppress host immunity. RNA-seq studies reveal symbiont-induced downregulation of genes associated with antimicrobial peptide production and proteolysis, creating an immunosuppressed microenvironment that benefits crinivirus retention [51]. Importantly, ecological factors like temperature, insecticide exposure, and host plant species can shift the composition of the symbiont community, leading to dynamic alterations in transmission efficiency [58, 59].

Synergistic and antagonistic interactions among co-infecting symbionts further shape vector-virus dynamics. For instance, *Wolbachia* may suppress certain virus infections via ROS modulation and autophagy interference, though effects are context-dependent and vary by host background [60, 61]. Functional experiments using antibiotics to selectively eliminate symbionts have confirmed their essential role: removal of *Hamiltonella* or *Rickettsia* significantly impairs crinivirus acquisition and transmission, highlighting their utility as molecular targets for next-generation vector control strategies [62, 63]. Collectively, these findings highlight the contribution of endosymbionts (Table 1) to vector competence and identify them as potential targets for disrupting crinivirus transmission.

Table 1: Potential endosymbiont-mediated modulation of crinivirus vector competence

Symbiont	Mechanism	Ecological Outcome	Reference
Hamiltonella defensa	GroEL chaperonin stabilizes viral particles	↑ Crinivirus transmission	[54]
Rickettsia sp.	Alters lipid metabolism, boosts vector fitness	↑ Whitefly fecundity & virus spread	[56]
Arsenophonus sp.	Immune modulation via suppression of antimicrobial peptides	↑ Virus persistence	[57]
Portiera aleyrodidarum	Provides essential amino acids, enhancing host survival	Sustains long-term vector populations	[64]
Cardinium sp.	Manipulates host reproduction (cytoplasmic incompatibility)	↑ Symbiont prevalence, indirect virus stability	[65]
Wolbachia sp.	Immune reprogramming (ROS modulation, autophagy interference)	↓ Some virus infections, context-dependent	[60]
Fritschea bemisiae	Endocellular localization, affects gut physiology	Potential barrier or enhancer of transmission	[51]

Influence of Host Plant Volatiles and Virus Induced Phenotypic Shifts in Insect Behavior

In addition to vector physiology, criniviruses manipulate host plant biochemistry to maximize transmission. The infected plants produce a modified Volatile Organic Compound (VOC) profile including increased terpenes and aldehydes and specific monoterpenes, which serve as attractants of *B. tabaci*. Repellent Green Leaf Volatiles (GLVs) are also suppressed simultaneously [66, 67].

Quantitative VOC profiling has demonstrated significant upregulation of sesquiterpenes in Tomato Chlorosis Virus (ToCV)-infected tomato plants, which corresponds with increased whitefly landing and extended phloem ingestion time. This, in turn, facilitates both acquisition and inoculation events [68]. For Cucurbit Chlorotic Yellows Virus (CCYV), altered aldehyde emissions modulate vector foraging patterns by enhancing host attractiveness during early symptom expression phases [67]. Notably, virus-induced phenotypic plasticity in whiteflies such as altered locomotion, increased probing frequency, and modified oviposition preferences further aligns with the temporal window of maximal virus transmissibility [69]. These vector-favoring changes are not universal across crinivirus species. Rather, divergent VOC signatures suggest species-specific manipulation strategies. For example, Lettuce Infectious Yellows Virus (LIYV) triggers only subtle GLV suppression, promoting long-term virus circulation without triggering vector overloading or immune response. These findings reflect co-evolved specificity in virus-vector-host interaction networks [70].

Behavioral assays under controlled light and olfactory conditions indicate that visual cues complement VOC-mediated attraction. Infected plants are more strongly preferred under illuminated conditions, further enhancing viral acquisition and subsequent spread. This dual-cue mechanism (olfactory and visual) offers redundancy to ensure vector targeting [71, 72]. Comparison studies showed that the effects of different criniviruses on the VOC emission profiles as shown in Table 2, are not universal. From an applied perspective, interfering with virus-mediated VOC manipulation holds promise for vector control. Chemical masking agents, RNAi-based suppression of key biosynthetic enzymes in host VOC pathways, or transgenic approaches targeting terpene synthases represent viable strategies to disrupt vector orientation behavior and reduce transmission rates [67].

Crinivirus Host Vector Triangulation: Molecular Dialogues and Immunomodulation

Hormonal Crosstalk Modulation During Infection

Crinivirus infection triggers complex reprogramming of host hormonal milieu, with Salicylic Acid (SA), Jasmonic Acid (JA) and auxin signaling. SA accumulation promotes the expression of Pathogenesis Related (PR) genes [73]. On the other hand, it antagonizes JA responses, weakens anti-herbivore defenses and builds an environment conducive to vector colonization [74]. Such an unbalance is further supported by viral coat proteins that repress JA-responsive genes, eliminating resistance to *Bemisia tabaci* feeding and enabling vector-mediated viral transmission [75]. The coat protein of Tomato Chlorosis Virus (ToCV) has been shown to downregulate JA-responsive genes by interfering with transcription factors like MYC2 and ORA59, thus amplifying vector feeding efficiency and virus acquisition [76].

Auxin biosynthesis and transport are also disrupted; consequently, tissue architecture change to favor viral accumulation and systemic spread [82]. At the transcriptional level, WRKY and NAC-type transcription factors are nodes of convergence where SA–JA–auxin crosstalk is integrated as shown in Table 3. WRKY70 acts as a positive regulator of SA signaling and a

suppressor of JA responses. It is differentially regulated in virus-infected tissues, skewing the immune response balance toward antiviral immunity at the expense of anti-vector resistance [73, 83]. Similarly, NAC transcription factors such as NAC042 are downregulated during infection, impairing stress-responsive gene expression and facilitating viral persistence [84]. This results in establishing virus-induced hormonal bias. Vector feeding additionally synergizes with these viral manipulations, exaggerated hormonal shifts and tilted defense trade-offs toward infection [45]. Finally, this triangulation signifies a tactical virus hijacking of host defense cross-talk in which antiviral SA responses subvert anti-vector JA pathways to ensure effective crinivirus transmission [85].

Table 2: Volatile Organic Compound (VOC) Modifications in Crinivirus-Infected Plants and Their Vector-Behavioral Consequences

VOC Alteration	Behavioral Effect on Whitefly Vectors	Transmission Implication	References
↑ sesquiterpene emissions	Increased settling on ToCV-infected tomato leaves by *B. tabaci*	Enhances early acquisition opportunities	[68]
↓ Total volatiles and terpenes	Whiteflies displayed olfactory preference for healthy plants	Olfactory-mediated reduction may slow spread, though visual cues compensate	[77]
Visual cue dominance in infection	Infected plants were strongly preferred under light, but not in darkness indicating reliance on visual cues	Transmission facilitation through enhanced vector landing in illuminated environments	[78]
↑ Aldehydes (infection-specific)	Increased aldehydes detected in CCYV-infected cucurbits altered whitefly orientation behavior	May enhance vector probing efficiency, influencing virus acquisition	[79]
↑ Green Leaf Volatiles (GLVs)	Subtle GLV induction during Lettuce infectious yellows virus infection modulated vector settling patterns	Fine-tuned modulation of attraction aids persistent virus circulation	[80]
VOC suppression in systemic tissues	Infected cucurbit leaves emitted fewer attractant VOCs	long-term epidemiological balance by discouraging superinfection	[81]

Table 3: Crinivirus strategies for immune evasion via hormone-transcriptional cross-talk

Mechanism	Plant Response	Vector/Transmission Impact	References
SA suppression	Reduced expression of pathogenesis-related (PR) genes	Enhanced viral accumulation, improved persistence in tissues	[86]
JA pathway antagonism	Weakened defenses against herbivory	Increased feeding by *B. tabaci*, improved virus acquisition	[76]
Auxin modulation	Altered leaf morphology and reduced resistance	Enhanced suitability of leaf tissues for vector colonization	[87]
WRKY transcriptional reprogramming	Downregulation of WRKY70, involved in defense signaling	Facilitates systemic infection, improves viral spread	[88]
NAC transcription factor manipulation	Suppressed NAC042, stress-related transcription factors	Promotes long-term vector colonization and persistent infection	[84]
Hormonal synergy (SA–JA–Auxin)	Crosstalk disruption, delayed immune response	Facilitates virus–vector stable cycle, enhancing transmission	[73]

RNA Silencing Suppression Via P22 and CP-Mediated Evasion Strategies

RNA silencing is a major antiviral defense mechanism in plants, but some criniviruses have commandeered specific proteins to quash this machinery. The Lettuce Infectious Yellows Virus (LIYV) encoded P22 protein is a strong VSR, capable of binding to dsRNAs and siRNA duplexes to block their cleavage by cell Dicer-like enzymes [89]. This process interferes with the formation of the RNA-Induced Silencing Complex (RISC), resulting in attenuation of Argonaute-dependent antiviral responses [90]. Apart from local suppression of silencing, P22 inhibits systemic transmission of mobile silencing signals and thus facilitates viral long-distance spread within the host [89]. Co protein (CP and CPm) also participate by blocking RISC action, thus decreasing the host antiviral response [91]. This leads to the accumulation of viral titers in the phloem tissues, and subsequently results in increased efficiency of vector acquisition. Under such suppression, plants will endeavor to induce compensatory defense mechanisms by secondary siRNA amplification and modulation of AGO expression, however, plants

are largely incapable in opposing P22 potency [89]. Comparison studies suggest that this particular RNA silencing suppression is shared among closterovirids, which highlights recurring evolutionary VSR strategies.

Organelle and Redox Modulation in Host Defense Manipulation

Criniviruses have evolved sophisticated mechanisms to subvert host immune responses by targeting chloroplast functions and modulating the plant's redox environment. Chloroplasts are central in antiviral defense because they generate salicylic acid precursors and Reactive Oxygen Species (ROS) to inhibit viral spread [92]. As chloroplasts become swollen and stromules (membrane extensions) form, viral replication complexes localize at these sites, co-opting chloroplast-derived energy and resources for virion assembly [93-95]. This ROS-mediated redox imbalance has far-reaching consequences for the plant's ability to mount an immune response. NPR1 (Non-expressor of Pathogenesis-Related Genes 1), a key regulator of SA signaling and SAR, is repressed under these oxidative conditions, impairing the plant's capacity to mount a defense response [96-97]. Similarly, thioredoxin, a major component of redox signaling pathways involved in stress response, is modulated in such a way that it dampens immune signaling [98-100]. Moreover, criniviruses disrupt the isochorismate pathway, a crucial step in SA biosynthesis, by interfering with enzymes that normally synthesize SA from chorismic acid. This reduction in SA biosynthesis lowers the plant's systemic resistance to the virus and enables more efficient viral movement through the vascular system [83, 101]. This manipulation of the chloroplast–redox axis provides the virus with the means to both escape immune surveillance and create an environment conducive to long-term viral colonization.

Mosaicism Mutation and Evolutionary Plasticity in Reassortment Mechanisms

Segment Reassortment and Recombination Hotspots

Segment reassortment and recombination are pivotal mechanisms that drive the genomic fluidity and evolutionary adaptability of criniviruses. These can easily exchange segments from their bipartite RNA genome during mixed infection to produce reassorted variants with distinct phenotypic features [102, 103]. Recombination primarily occurs at specific hotspots in the intergenic regions of RNA1 and RNA2, where polymerase pausing facilitates template switching [104]. Here, malfunctioning RNA molecules generated through recombination events act as evolutionary intermediaries, enabling the virus to adapt rapidly to selective pressures such as host resistance mechanisms or environmental changes [105, 106]. From a functional perspective, segment reassortment can confer significant adaptive advantages. For example, reassorted viral genomes may allow criniviruses to expand their host range, enabling them to infect previously resistant plant species such as solanaceous or leguminous crops, in addition to their usual cucurbitaceous hosts [3, 107]. Population level studies show that there is extensive circulation of reassorted strains which enhance ingroup diversity and facilitate maintenance of adaptive potential in mixed cropping systems [108]. Recombination not only increases the evolutionary plasticity of criniviruses but also facilitates the maintenance of adaptive strategies in field conditions, where varying host susceptibilities and environmental stressors may favor certain genetic lineages [14, 109]. In the broader context of plant RNA virus evolution, these recombination hotspots in criniviruses share similarities with those found in other multipartite viruses, such as geminiviruses, where recombination has been identified as a major force driving viral diversity and viral fitness [110]. This suggests that recombination is a convergent evolutionary strategy among multipartite RNA viruses, providing a robust framework for understanding the evolutionary success of Closteroviridae and related families.

HTS-Driven Discovery of Cryptic Strains and Microvariants

The advent of High-Throughput Sequencing (HTS) has revolutionized the understanding of criniviruses by identifying cryptic lineages and within-host microvariants not detected by traditional molecular diagnostic assays [111]. Deep-sequencing methodologies have continually confirmed that crinivirus populations are dynamic quasi-species, in that a cloud of minor variants accompany individual hosts, which contribute to overall adaptive potential and evolutionary pleiotropy [112-113]. The application of HTS has led to the identification of divergent strains and cryptic species across diverse agroecosystems, prompting a re-evaluation of closterovirid taxonomy and challenging long-held species demarcation criteria [114]. Mixed infections uncovered through metagenomic surveys further highlight that reassortment and recombination are not isolated events but pervasive processes occurring under field conditions, often accelerating viral diversification [112]. Importantly, intra-host microvariants can serve as adaptive reservoirs, where minority haplotypes may rise to dominance under selective pressures imposed by host resistance or vector transmission dynamics, thereby fueling the emergence of novel pathogenic strains [102]. From an epidemiological perspective, the detection of cryptic strains and microvariants has profound implications for viral surveillance and disease management. By revealing hidden viral diversity, HTS enables early detection

of emergent strains that may be more pathogenic or capable of overcoming resistance barriers in crops [115]. This capability is particularly important in monitoring tomato and cucurbit production systems, where cryptic reservoirs of viral variants have been implicated in disease outbreaks and cross-crop transmission, complicating control strategies [107].

Phylogeographic Mapping in Tomato, Cucurbit, and Bean Systems

Crinivirus populations exhibit strong host-specific phylogeographic structuring, where viral clades are associated with distinct host plants, including tomato, cucurbit, and bean species. These phylogenetic clusters reflect the evolutionary pressures imposed by the host plant environment, shaping viral diversity and transmission dynamics across geographical regions [4, 116]. In tomato and cucurbit production systems, for instance, crinivirus strains have evolved distinct genetic markers that facilitate adaptation to specific plant defenses and vascular structures and influences the virus's systemic movement and persistence [107, 117].

Geographical distribution also plays a role in divergence, as Mediterranean, South American and Asian strains always form phylogenetic clads independently, suggesting regional evolutionary paths [14]. Dispersal of the vector *B. tabaci* cryptic species helps in cross-continental movement, and in turn the phylogeographic connectivity of virus populations [118]. Crucially, inter- or multi-cropping systems facilitate cross-crop transmission events, providing recombinant lineages the opportunity to expand their host ranges and complicating disease control options [43, 119]. The use of HTS has enabled the fine-scale reconstructions of phylogeographies by revealing low-frequency microvariants and cryptic lineages not identified by conventional methods [120, 121]. Yet, co‐detection of recombination between isolates carrying the type 1 and type 2 RNA segments on the genome, would also cause phylogenetic incongruence, complicating evolutionary tracing [22]. Comparative studies suggest that criniviruses may modify their movement and structural proteins for better compatibility with distinct hosts, such as members of the Solanaceae, Cucurbitaceae or Leguminosae [4, 22].

Crinivirus Synergy Complexes and Co-Infections: A Challenge to Diagnostics

Multipartite Disease Complexes With Begomovirus, Potyvirus, and Orthotospovirus

New disease complexes consisting of criniviruses, begomoviruses, potyviruses, and orthotospoviruses in multipartite arrangements are a rising menace to important horticulture systems [122, 123]. In potato and chili (pepper) crops, simultaneous infection by multiple viruses frequently results in synergistic interactions. Mixed infections expressing markedly more severe symptoms than single infections and, in some cases, even than the classic dual-virus complexes already described [124, 125]. Experimental evidence shows that mixed infections involving a crinivirus and a begomovirus (e.g., Tomato chlorosis virus + Tomato yellow leaf curl virus) can cause severe chlorosis, necrosis, and stunting symptomatically resembling abiotic or nutrient-stress disorders [122]. Tomato chlorosis virus (Crinivirus) and Okra enation leaf curl virus (Begomovirus) co-infection also results in chlorotic and necrotic lesions, mimicking nutrient deficiency [126]. Such family-to-family inter-familial viral associations improve replication efficacy and wider systemic migration that are required for epidemic expansions. Vector interactions also contribute to this synergy, where whiteflies and thrips may transmit these viruses together, increasing the establishment of both complex infections in a mutual host plant [127]. The epidemiological implications are serious, with multipartite complexes promoting fast epidemic development and major yield losses on different cropping systems.

Molecular Basis of Synergism and Exacerbated Symptomology

On a molecular mechanism level, viral synergism arises from the combinatory activities of RNA-silencing suppressors and the overlapping or redundant targeting of host defense pathways by co-infecting viruses [128]. The HC Pro suppressor in potyviruses can bind and sequester siRNAs and impair small-RNA-mediated antiviral silencing [129]. In criniviruses, the P22 suppressor has been shown to impair systemic silencing responses, thus compromising both local and systemic RNA-interference (RNAi) immunity [130]. These repressions act ultimately to stabilize and elevate the abundance of viral RNA and to increase viral titters. In addition to RNAi-suppression, crinivirus and potyvirus have chloroplast-targeting proteins that perturb photosynthetic electron transport and, following ROS overproduction, disrupt the equilibrium of previously delicate redox state. Disturbance of redox-regulated pathways leads to the disruption of NPR1-dependent signaling and SA-regulated defense cascades, which promote replication and systemic movement [131]. Symptom exacerbation is particularly evident in co-infection, including systemic chlorosis, dwarfing and premature aging, all of which are more severe than in the case of single infection [132-134]. The increased viral titers under synergism increase vector acquisition capability and thus close a positive feedback loop that can drive epidemiological spread.

Implications for Misdiagnosis and Management Failures

The intricacy of crinivirus-related co-infections is difficult to diagnose and manage. Such overlapping symptom profiles often resemble abiotc stress or single-virus infections, which led to frequent misdia gnosis in the fields [135]. With classical serological assays such as ELISA, distinguishing between co-infecting viruses can be challenging due to antigenic cross-reactivity or low viral titers, which may result in false negatives and underestimate the true prevalence of infection [136, 137]. These diagnostic drawbacks result in long lag phases and suboptimal quarantine strategies that allow unchecked transmission in cropping systems [138]. Breeding programs that target resistance to a single virus may inadvertently compromise durability, as such resistance can be bypassed by co-infecting or emerging viruses, especially under synergistic interactions or shared suppression mechanisms [139, 140]. Misidentification of the actual viral agents involved in disease outbreaks can result in ineffective vector control strategies. This is especially when management focuses on a single vector species while others, involved in co-infection cycles, remain active and unaddressed [141]. Diagnosis has dramatically changed following the emergence of high-throughput sequencing, which has uncovered cryptic strains, microvariants and new co-infections that had escaped notice of conventional diagnostic procedures [142]. Therefore, implementing multiplex PCR and HTS-based diagnostic tools is becoming increasingly critical for effective surveillance and control of complex multi-virus syndromes. Failure to promptly detect and manage these viruses can escalate epidemics, cause significant crop losses, and compromise vector management and resistance breeding strategies [143, 144].

Climate Change and Crinivirus Epidemiological Shifts

Climate change has emerged as a major determinant shaping the epidemiology and persistence of criniviruses. It acts primarily through its cascading effects on vector dynamics, host distribution, and agroecosystem suitability [145]. Ecological Niche Modeling (ENM) under RCP 4.5 and RCP 8.5 scenarios has consistently projected a northward and altitudinal expansion of *B. tabaci* populations. This expansion extends their ecological range into temperate and subtropical regions and thereby enhancing the risk of Crinivirus establishment in previously marginal areas [146]. Climate change may intensify crinivirus epidemics by accelerating whitefly population growth within favorable thermal limits. *B. tabaci* development can shorten from about 36 days at 20°C to about 15 days at 32°C, although development may fail under extreme heat such as 35°C, indicating that warming effects are nonlinear [147]. Protected cultivation systems like greenhouses, plastic tunnels, and net-houses serve as microclimatic refuges that promote year-round survival of *B. tabaci* populations. These conditions sustain elevated virus transmission rates even when outdoor environments are unfavorable for vector activity [148]. Simultaneously, climate-driven alterations in host phenology, cropping cycles, and regional crop distribution are expected to disrupt the synchrony between virus, vector, and host availability. These disruptions increase the risk of temporal and spatial overlap, favoring epidemic emergence especially in tomato and cucurbit systems [127, 149, 150]. The spread of Crinivirus is also strongly influenced by the duration of virus retention within vector populations. Tomato Chlorosis Virus (ToCV) can be retained by B. tabaci MED for at least 6 days following acquisition [151], whereas Cucurbit Yellow Stunting Disorder Virus (CYSDV) remains transmissible for at least 9 days [152]. Similarly, Cucurbit Chlorotic Yellows Virus (CCYV) may be retained in whiteflies for up to 12 days, although retention efficiency gradually declines over time [153]. These prolonged retention periods facilitate secondary spread through vector movement among infected crops, weeds, and newly established plantings. Transmission efficiency is also dependent on vector feeding duration. For CYSDV, successful transmission exceeding 80% has been observed when whiteflies were allowed acquisition access periods of at least 18 h and inoculation access periods of at least 24 h [154]. Such findings indicate that environmental conditions favoring prolonged vector feeding may substantially increase epidemic development.

Recent advances in spatial epidemiology have substantially enhanced the capacity to predict and monitor emerging Crinivirus outbreaks. ENM like Maximum Entropy (MaxEnt)-based approaches integrates vector occurrence records with climatic variables to identify regions suitable for the establishment and persistence of *B. tabaci* cryptic species, the principal vectors of criniviruses [4, 146, 155]. Geographic Information Systems (GIS) further refine these predictions by incorporating host-crop distribution, land-use patterns, and environmental variables to delineate transmission hotspots and support targeted surveillance programs [156, 157]. Complementary remote-sensing technologies, including multispectral and hyperspectral imaging platforms, enable large-scale assessment of crop health through the detection of changes in canopy reflectance, chlorophyll content, and photosynthetic activity associated with disease-induced stress. More recently, machine learning algorithms such as Random Forest, Support Vector Machine (SVM), and Extreme Gradient Boosting (EGB) have demonstrated considerable utility for integrating climatic, ecological, and epidemiological datasets to forecast outbreak probability and identify periods of elevated transmission risk [158, 159]. Collectively, these approaches provide a robust

framework for dynamic risk mapping, early-warning systems, and evidence-based disease management. More broadly, crinivirus emergence reflects the interaction of viral evolution, vector biology, endosymbiont-mediated processes, environmental change, and cropping-system dynamics operating across multiple biological scales (Figure 4).

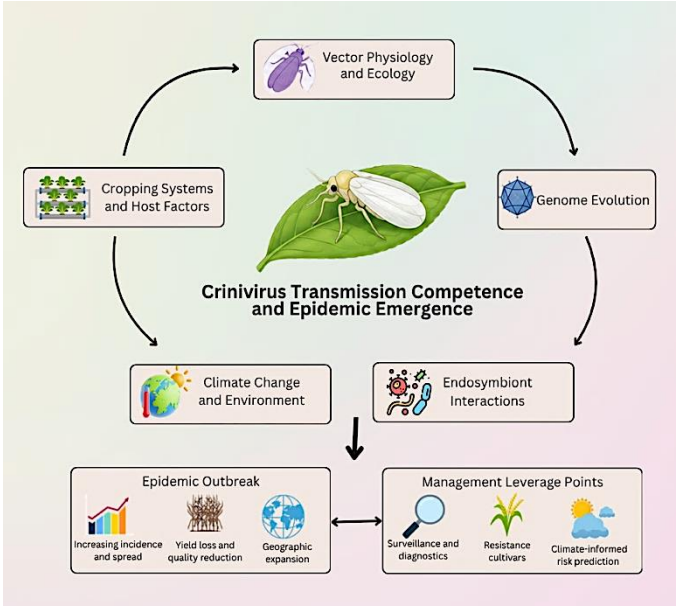

Fig. 4: Conceptual framework illustrating the multiscale drivers of crinivirus transmission competence and epidemic emergence. Viral genome evolution, vector physiology and ecology, endosymbiont-mediated interactions, climate and environmental factors, and cropping-system characteristics collectively influence transmission dynamics and disease emergence

Converging Virology and Agroecology Toward Sustainable Virus Management

Sustainable management of crinivirus and associated virus complexes necessitates a convergence between molecular virology, agroecology, and participatory innovation frameworks. The integration of portable high-throughput diagnostic technologies such as real-time nanopore sequencing, CRISPR-based assays, and multiplex PCR has revolutionized virus detection in the field. These tools enable rapid, sensitive identification of plant virus co-infections, significantly reducing diagnostic delays and strengthening real-time epidemic surveillance [60-63]. Yet, effective virus suppression extends beyond molecular diagnostics and technological innovation. It requires a recognition that epidemiological outcomes are shaped by landscape-scale metapopulation dynamics of vectors, whose spatial dispersal and ecological persistence drive viral establishment across heterogeneous agroecosystems [12, 163, 164]. Embedding ecological insights within farmer-responsive extension programs facilitates adaptive innovation. This approach ensures that novel diagnostic and Integrated Pest Management (IPM) tools are locally relevant and more readily adopted at the community level [165, 166]. Critically, policy and institutional frameworks play a catalytic role in ensuring sustainable plant health governance. They strengthen biosecurity infrastructure, promote cross-sectoral coordination, and maintain quarantine networks that are essential to preventing the incursion and spread of exotic or recombinant plant viral strains [167, 168]. Future-ready IPM strategies must evolve into adaptive, systems-based frameworks that integrate multiple layers of resilience. These include the deployment of host-plant resistance genes, utilization of vector biological control agents, and incorporation of climate-resilient predictive models that capture the dynamic interactions among viruses, vectors, and host plants under changing environmental conditions [169, 171]. Equally essential is bridging the research practice divide through interdisciplinary collaboration, wherein virologists, ecologists, and extension professionals co-develop locally relevant, feedback-driven management innovations [172]. Within an agroecological framework, integrative strategies enhance biodiversity, functional resilience, and overall agroecosystem adaptability. This paradigm shifts plant virus management from reactive control toward a more preventive, participatory, and ecologically sustainable approach [166, 173, 174].

Conclusion and Future Perspectives

Criniviruses represent one of the most sophisticated plant virus vector systems in modern agriculture, shaped by the continuous interaction of viral evolution, vector biology, host adaptation, and environmental change. Advances in viromics,

molecular genetics, and vector ecology have substantially improved our understanding of the mechanisms governing virus emergence, transmission, and persistence. Future research should prioritize the functional characterization of transmission-associated determinants, the evolutionary consequences of recombination and reassortment, and the molecular basis of vector competence across diverse whitefly species. Equally important is the integration of high-throughput diagnostics, coordinated surveillance networks, and spatially explicit epidemiological approaches to improve the detection of emerging variants, alternative hosts, and transmission hotspots. Climate-informed forecasting frameworks capable of linking vector dynamics, host distribution, and environmental suitability will be essential for anticipating future disease risks. From a management perspective, durable host resistance, strengthened surveillance, and evidence-based vector control remain the most immediate priorities. A central challenge for the field is no longer the discovery of new criniviruses, but the development of predictive frameworks that explain how viral evolution, vector adaptation, and ecological change interact to drive disease emergence across agricultural landscapes. Addressing this challenge will be critical for advancing predictive epidemiology and establishing resilient, sustainable strategies for crinivirus management in an era of increasing agricultural and climatic uncertainty.

Acknowledgment

The authors extend their sincere appreciation to colleagues and peers for their insightful discussions and constructive suggestions that enriched the conceptual depth of this review.

Funding Information

The author(s) disclosed receipt of no funding for conducting this article.

Author's Contributions

Md. Abdullah Al Sabbir: Conceptualization, investigation, data collection, and original draft preparation.
Nabela Akter: Drafting, data collection and contribution to manuscript drafting.
Ankita Saha: Visualization, figure preparation, and technical editing of the manuscript.
Md. Motaher Hossain: Conceptual supervision, critical review, and final approval of the submitted version.

Ethics

This paper is original and not submitted elsewhere. The corresponding author agrees that all authors have seen and approved the final version of the manuscript and there is no ethical problem to disclose.

References

1. ViralZone (ExPASy). (2024). Two Crinivirus-specific proteins of Lettuce infectious yellows virus (LIYV), P26 and P9, are self-interacting. *ViralZone*. https://viralzone.expasy.org/287
2. International Committee on Taxonomy of Viruses (ICTV). (2024). Family: Closteroviridae. *ICTV 9th Report on Virus Taxonomy*. https://ictv.global/report_9th/RNApos/Closteroviridae
3. Garcia, L. R., & Janssen, D. (2020). Epidemiology and control of emerging criniviruses in bean. Virus Research, 280, 197902. https://doi.org/10.1016/j.virusres.2020.197902
4. Tzanetakis, I. E., Martin, R. R., & Wintermantel, W. M. (2013). Epidemiology of criniviruses: an emerging problem in world agriculture. *Frontiers in Microbiology*, *4*, 00119. https://doi.org/10.3389/fmicb.2013.00119
5. Liu, Q., Zhang, S., Mei, S., Zhou, Y., Zhou, C., & Cao, M. (2021). Viromics Unveils Extraordinary Genetic Diversity of the Family Closteroviridae in Wild Citrus. *Research Square*.
6. Abrahamian, P. E., & Abou-Jawdah, Y. (2013). Whitefly-transmitted criniviruses of cucurbits: current status and future prospects. *VirusDisease*, *25*(1), 26–38. https://doi.org/10.1007/s13337-013-0173-9
7. Navas-Castillo, J., Fiallo-Olivé, E., & Sánchez-Campos, S. (2011). Emerging Virus Diseases Transmitted by Whiteflies. *Annual Review of Phytopathology*, *49*(1), 219–248. https://doi.org/10.1146/annurev-phyto-072910-095235
8. Coêlho, L. M., Lima, A. T. M., Inoue-Nagata, A. K., Rizo, A. A. I., Pontes, N. de C., Golynski, A., & Albuquerque, L. C. de. (2019). Genetic structure analyses of the tomato-infecting crinivirus Tomato chlorosis virus indicates low genetic variability in the p22 gene and evidence of segregation based on sampling location in Brazil. *Tropical Plant Pathology*, *44*(3), 292–296. https://doi.org/10.1007/s40858-019-00280-5

9. Ghosh, S., & Ghanim, M. (2021). Factors Determining Transmission of Persistent Viruses by Bemisia tabaci and Emergence of New Virus–Vector Relationships. *Viruses*, *13*(9), 1808. https://doi.org/10.3390/v13091808
10. Ontiveros, I. (2023). *Dynamics of virus-host interactions in mixed viral infections in tomato plants and effects on whitefly-mediated dissemination*.
11. Czosnek, H., Hariton-Shalev, A., Sobol, I., Gorovits, R., & Ghanim, M. (2017). The Incredible Journey of Begomoviruses in Their Whitefly Vector. *Viruses*, *9*(10), 273. https://doi.org/10.3390/v9100273
12. Krause-Sakate, R., Watanabe, L. F. M., Gorayeb, E. S., Silva, F. B. da, Alvarez, D. de L., Bello, V. H., Nogueira, A. M., Marchi, B. R. de, Vicentin, E., Ribeiro-Junior, M. R., Marubayashi, J. M., Rojas-Bertini, C. A., Muller, C., Bueno, R. C. O. de F., Rosales, M., Ghanim, M., & Pavan, M. A. (2020). Population Dynamics of Whiteflies and Associated Viruses in South America: Research Progress and Perspectives. *Insects*, *11*(12), 847. https://doi.org/10.3390/insects11120847
13. Srinivasan, R., Martini, X., Pandey, S., Netla, V. R., & Dutta, B. (2024). Management of insect-transmitted viruses in organic vegetable production. *Entomologia Generalis*, *44*(6), 1393–1407. https://doi.org/10.1127/entomologia/2024/2781
14. Rubio, L., Guerri, J., & Moreno, P. (2013). Genetic variability and evolutionary dynamics of viruses of the family Closteroviridae. *Frontiers in Microbiology*, *4*, 00151. https://doi.org/10.3389/fmicb.2013.00151
15. Lapidot, M., Legg, J. P., Wintermantel, W. M., & Polston, J. E. (2014). Management of Whitefly-Transmitted Viruses in Open-Field Production Systems. *Advances in Virus Research / Plant Virus-Vector Interactions*, 147–206. https://doi.org/10.1016/b978-0-12-801246-8.00003-2
16. Patra, B., & Kumar Hath, T. (2022). Insecticide Resistance in Whiteflies*Bemisia tabaci*(Gennadius): Current Global Status. *Insecticides - Impact and Benefits of Its Use for Humanity*. https://doi.org/10.5772/intechopen.101954
17. Ertunc, F. (2020). Emerging Plant Viruses. *Emerging and Reemerging Viral Pathogens*, *1*, 1041–1062. https://doi.org/10.1016/b978-0-12-819400-3.00046-6
18. Wintermantel, W. M., & Wisler, G. C. (2006). Vector Specificity, Host Range, and Genetic Diversity of Tomato chlorosis virus. *Plant Disease*, *90*(6), 814–819. https://doi.org/10.1094/pd-90-0814
19. Dolja, V. V., Kreuze, J. F., & Valkonen, J. P. T. (2006). Comparative and functional genomics of closteroviruses. *Virus Research*, *117*(1), 38–51. https://doi.org/10.1016/j.virusres.2006.02.002
20. IsHak, J. A., Kreuze, J. F., Johansson, A., Mukasa, S. B., Tairo, F., Abo El-Abbas, F. M., & Valkonen, J. P. T. (2003). Some molecular characteristics of three viruses from SPVD-affected sweet potato plants in Egypt. *Archives of Virology*, *148*(12), 2449–2460. https://doi.org/10.1007/s00705-003-0187-5
21. Mongkolsiriwattana, C., Chen, A. Y. S., & Ng, J. C. K. (2011). Replication of Lettuce chlorosis virus (LCV), a crinivirus in the family Closteroviridae, is accompanied by the production of LCV RNA 1-derived novel RNAs. *Virology*, *420*(2), 89–97. https://doi.org/10.1016/j.virol.2011.08.017
22. Kiss, Z. A., Medina, V., & Falk, B. W. (2013). Crinivirus replication and host interactions. *Frontiers in Microbiology*, *4*, 00099. https://doi.org/10.3389/fmicb.2013.00099
23. Martelli, G. P., Agranovsky, A. A., Bar-Joseph, M., Boscia, D., Candresse, T., Coutts, R. H. A., Dolja, V. V., Falk, B. W., Gonsalves, D., Jelkmann, W., Karasev, A. V., Minafra, A., Namba, S., Vetten, H. J., Wisler, G. C., & Yoshikawa, N. (2002). The family Closteroviridae revised. *Archives of Virology*, *147*(10), 2039–2044. https://doi.org/10.1007/s007050200048
24. Rubio, L., Galipienso, L., & Ferriol, I. (2020). Detection of Plant Viruses and Disease Management: Relevance of Genetic Diversity and Evolution. *Frontiers in Plant Science*, *11*, 01092. https://doi.org/10.3389/fpls.2020.01092
25. Fuchs, M. (2021). *Closteroviruses (Closteroviridae)*. *3*, 336–347. https://doi.org/10.1016/b978-0-12-809633-8.21285-5
26. Kreuze, J. F., Savenkov, E. I., & Valkonen, J. P. T. (2002). Complete genome sequence and analyses of the subgenomic RNAs of Sweet potato chlorotic stunt virus reveal several new features for the genus Crinivirus. *Journal of Virology*, *76*(18), 9260–9270. https://doi.org/10.1128/jvi.76.18.9260-9270.2002
27. Untiveros, M., Olspert, A., Artola, K., Firth, A. E., Kreuze, J. F., & Valkonen, J. P. T. (2016). A novel sweet potato potyvirus open reading frame (ORF) is expressed via polymerase slippage and suppresses RNA silencing. *Molecular Plant Pathology*, *17*(7), 1111–1123. https://doi.org/10.1111/mpp.12366
28. Domingo, E., Sheldon, J., & Perales, C. (2012). Viral Quasispecies Evolution. *Microbiology and Molecular Biology Reviews*, *76*(2), 159–216. https://doi.org/10.1128/mmbr.05023-11
29. Agranovsky, A. A. (2021). Structure and Expression of Large (+) RNA Genomes of Viruses of Higher Eukaryotes. *Biochemistry (Moscow)*, *86*(3), 248–256. https://doi.org/10.1134/s0006297921030020
30. Sanjuán, R., & Domingo-Calap, P. (2016). Mechanisms of viral mutation. *Cellular and Molecular Life Sciences*, *73*(23), 4433–4448. https://doi.org/10.1007/s00018-016-2299-6
31. Maliogka, V. I., Wintermantel, W. M., Orfanidou, C. G., & Katis, N. I. (2020). Criniviruses infecting vegetable crops. *Applied Plant Biotechnology for Improving Resistance to Biotic Stress*, 251–289. https://doi.org/10.1016/b978-0-12-816030-5.00012-4
32. Solovyev, A. G., & Morozov, S. Y. (2017). Non-replicative Integral Membrane Proteins Encoded by Plant Alpha-Like Viruses: Emergence of Diverse Orphan ORFs and Movement Protein Genes. *Frontiers in Plant Science*, *8*, 01820. https://doi.org/10.3389/fpls.2017.01820
33. Cuellar, W. J., Tairo, F., Kreuze, J. F., & Valkonen, J. P. T. (2008). Analysis of gene content in sweet potato chlorotic stunt virus RNA1 reveals the presence of the p22 RNA silencing suppressor in only a few isolates: implications for viral evolution and synergism. *Journal of General Virology*, *89*(2), 573–582. https://doi.org/10.1099/vir.0.83471-0
34. Hussain, M. D., Farooq, T., Chen, X., Tariqjaveed, M., Jiang, T., Liu, S., & Zhou, T. (2021). Viral suppressors from members of the family Closteroviridae combating antiviral RNA silencing: a tale of a sophisticated arms race in host-pathogen interactions. *Phytopathology Research*, *3*(1), 27. https://doi.org/10.1186/s42483-021-00104-y
35. Ambrós, S., Gómez-Muñoz, N., Giménez-Santamarina, S., Sánchez-Vicente, J., Navarro-López, J., Martínez, F., Daròs, J.-A., & Rodrigo, G. (2021). Molecular signatures of silencing suppression degeneracy from a complex RNA virus. *PLOS Computational Biology*, *17*(6), e1009166. https://doi.org/10.1371/journal.pcbi.1009166
36. Peremyslov, V. V., Hagiwara, Y., & Dolja, V. V. (1999). HSP70 homolog functions in cell-to-cell movement of a plant virus. *Proceedings of the National Academy of Sciences*, *96*(26), 14771–14776. https://doi.org/10.1073/pnas.96.26.14771

37. Qiao, W., Medina, V., & Falk, B. W. (2017). Inspirations on Virus Replication and Cell-to-Cell Movement from Studies Examining the Cytopathology Induced by Lettuce infectious yellows virus in Plant Cells. *Frontiers in Plant Science*, *8*, 01672. https://doi.org/10.3389/fpls.2017.01672
38. Kubota, K., & Ng, J. C. K. (2016). Lettuce chlorosis virus P23 Suppresses RNA Silencing and Induces Local Necrosis with Increased Severity at Raised Temperatures. *Phytopathology®*, *106*(6), 653–662. https://doi.org/10.1094/phyto-09-15-0219-r
39. Firth, A. E., & Brierley, I. (2012). Non-canonical translation in RNA viruses. *Journal of General Virology*, *93*(7), 1385–1409. https://doi.org/10.1099/vir.0.042499-0
40. Jiao, Z., Tian, Y., Wang, J., Ismail, R. G., Bondok, A., & Fan, Z. (2022). Advances in research on maize lethal necrosis, a devastating viral disease. *Phytopathology Research*, *4*(1), 14. https://doi.org/10.1186/s42483-022-00117-1
41. Agranovsky, A., & Morozov, S. (1999). Gene Expression in Positive Strand RNA Viruses: Conventional and Aberrant Strategies. *Molecular Biology of Plant Viruses*, 99–119. https://doi.org/10.1007/978-1-4615-5063-1_4
42. Miras, M., Miller, W. A., Truniger, V., & Aranda, M. A. (2017). Non-canonical Translation in Plant RNA Viruses. *Frontiers in Plant Science*, *8*, 00494. https://doi.org/10.3389/fpls.2017.00494
43. Sánchez-Tovar, M. R., Rivera-Bustamante, R. F., Saavedra-Trejo, D. L., Guevara-González, R. G., & Torres-Pacheco, I. (2025). Mixed Plant Viral Infections: Complementation, Interference and Their Effects, a Review. *Agronomy*, *15*(3), 620. https://doi.org/10.3390/agronomy15030620
44. Agranovsky, A. A. (2016). Closteroviruses: Molecular Biology, Evolution and Interactions with Cells. *Plant Viruses: Evolution and Management*, 231–252. https://doi.org/10.1007/978-981-10-1406-2_14
45. He, H.-F., Zhao, C.-C., Zhu, C.-Q., Yan, W.-L., Yan, M.-H., Zhang, Z.-L., Liu, J.-L., Shi, B.-Z., Bai, R.-E., Li, J.-J., & Yan, F.-M. (2023). Discovery of novel whitefly vector proteins that interact with a virus capsid component mediating virion retention and transmission. *International Journal of Biological Macromolecules*, *226*, 1154–1165. https://doi.org/10.1016/j.ijbiomac.2022.11.229
46. Jiménez, J., Mishra, R., Wang, X., Magee, C. M., & Bonning, B. C. (2024). Composition and abundance of midgut plasma membrane proteins in two major hemipteran vectors of plant viruses, Bemisia tabaci and Myzus persicae. *Archives of Insect Biochemistry and Physiology*, *116*(3), e22133. https://doi.org/10.1002/arch.22133
47. Rajarapu, S. P., Ullman, D. E., Uzest, M., Rotenberg, D., Ordaz, N. A., & Whitfield, A. E. (2021). *Plant–virus–vector interactions*.
48. Rao, Y. D., Kumar, D., & Sharma, S. (2024). A comprehensive review on transmission mechanism of plant viruses by insects. *International Journal of Research in Agronomy*, *7*(7S), 337–344. https://doi.org/10.33545/2618060x.2024.v7.i7se.1049
49. Mittapelly, P., & Rajarapu, S. P. (2020). Applications of Proteomic Tools to Study Insect Vector–Plant Virus Interactions. *Life*, *10*(8), 143. https://doi.org/10.3390/life10080143
50. Farooq, T., Lin, Q., She, X., Chen, T., Li, Z., Yu, L., Lan, G., Tang, Y., & He, Z. (2022). Cotton leaf curl Multan virus differentially regulates innate antiviral immunity of whitefly (Bemisia tabaci) vector to promote cryptic species-dependent virus acquisition. *Frontiers in Plant Science*, *13*, 1040547. https://doi.org/10.3389/fpls.2022.1040547
51. Andreason, S. A., Shelby, E. A., Moss, J. B., Moore, P. J., Moore, A. J., & Simmons, A. M. (2020). Whitefly Endosymbionts: Biology, Evolution, and Plant Virus Interactions. *Insects*, *11*(11), 775. https://doi.org/10.3390/insects11110775
52. Hussain, M. D., Farooq, T., Kamran, A., Basit, A., Wang, Y., Smagghe, G., & Chen, X. (2025). Endosymbionts as hidden players in tripartite pathosystem of interactions and potential candidates for sustainable viral disease management. *Critical Reviews in Biotechnology*, *45*(6), 1348–1370. https://doi.org/10.1080/07388551.2024.2449403
53. Rao, Q., Rollat-Farnier, P.-A., Zhu, D.-T., Santos-Garcia, D., Silva, F. J., Moya, A., Latorre, A., Klein, C. C., Vavre, F., Sagot, M.-F., Liu, S.-S., Mouton, L., & Wang, X.-W. (2015). Genome reduction and potential metabolic complementation of the dual endosymbionts in the whitefly Bemisia tabaci. *BMC Genomics*, *16*(1), 226. https://doi.org/10.1186/s12864-015-1379-6
54. Gottlieb, Y., Ghanim, M., Chiel, E., Gerling, D., Portnoy, V., Steinberg, S., Tzuri, G., Horowitz, A. R., Belausov, E., Mozes-Daube, N., Kontsedalov, S., Gershon, M., Gal, S., Katzir, N., & Zchori-Fein, E. (2006). Identification and Localization of a Rickettsia sp. in Bemisia tabaci (Homoptera: Aleyrodidae). *Applied and Environmental Microbiology*, *72*(5), 3646–3652. https://doi.org/10.1128/aem.72.5.3646-3652.2006
55. Kliot, A., & Ghanim, M. (2013). The Role of Bacterial Chaperones in the Circulative Transmission of Plant Viruses by Insect Vectors. *Viruses*, *5*(6), 1516–1535. https://doi.org/10.3390/v5061516
56. Li, Y.-H., Peng, J., Wu, Q.-J., Sun, J.-C., Zhang, P.-J., & Qiu, B.-L. (2024). Transcriptome data reveal beneficial effects of Rickettsia (Rickettsiales: Rickettsiaceae) on Bemisia tabaci(Hemiptera: Aleyrodidae) through nutritional factors and defense mechanisms. *Journal of Economic Entomology*, *117*(3), 817–824. https://doi.org/10.1093/jee/toae066
57. Singh, I., Kaur, R., Kumar, A., Singh, S., & Sharma, A. (2021). Differential expression of gut protein genes and population density of Arsenophonus contributes to sex-biased transmission of Bemisia tabaci vectored Cotton leaf curl virus. *PLOS ONE*, *16*(11), e0259374. https://doi.org/10.1371/journal.pone.0259374
58. Li, H., Jiang, Z., Zhou, J., Liu, X., Zhang, Y., & Chu, D. (2023). Ecological Factors Associated with the Distribution of Bemisia tabaci Cryptic Species and Their Facultative Endosymbionts. *Insects*, *14*(3), 252. https://doi.org/10.3390/insects14030252
59. Pan, H., Li, X., Ge, D., Wang, S., Wu, Q., Xie, W., Jiao, X., Chu, D., Liu, B., Xu, B., & Zhang, Y. (2012). Factors Affecting Population Dynamics of Maternally Transmitted Endosymbionts in Bemisia tabaci. *PLoS ONE*, *7*(2), e30760. https://doi.org/10.1371/journal.pone.0030760
60. Mushtaq, I., Sarwar, M. S., & Munzoor, I. (2024). A comprehensive review of Wolbachia-mediated mechanisms to control dengue virus transmission in Aedes aegypti through innate immune pathways. *Frontiers in Immunology*, *15*, 1434003. https://doi.org/10.3389/fimmu.2024.1434003
61. Palmer, W., Varghese, F., & Rij, R. V. (2018). Natural Variation in Resistance to Virus Infection in Dipteran Insects. *Viruses*, *10*(3), 118. https://doi.org/10.3390/v10030118
62. Hart, C. E., & Thangamani, S. (2021). Tick-virus interactions: Current understanding and future perspectives. *Parasite Immunology*, *43*(5), e12815. https://doi.org/10.1111/pim.12815
63. Laukaitis, H. J., & Macaluso, K. R. (2021). Unpacking the intricacies of Rickettsia–vector interactions. *Trends in Parasitology*, *37*(8), 734–746. https://doi.org/10.1016/j.pt.2021.05.008

64. Sun, X., Liu, B.-Q., Chen, Z.-B., Li, C.-Q., Li, X.-Y., Hong, J.-S., & Luan, J.-B. (2023). Vitellogenin Facilitates Associations between the Whitefly and a Bacteriocyte Symbiont. *MBio*, *14*(1), e02990-22. https://doi.org/10.1128/mbio.02990-22
65. Caballero, R. de J. (2007). *Systematics of the Bemisia tabaci complex and the role of endosymbionts in reproductive compatibility*.
66. Nebapure, S. M., Shankarganesh, K., Rajna, S., Naga, K. C., Pandey, D., Gambhir, S., Praveen, K. V., & Subramanian, S. (2022). Dynamic changes in virus-induced volatiles in cotton modulate the orientation and oviposition behavior of the whitefly Bemisia tabaci. *Frontiers in Physiology*, *13*, 1017948. https://doi.org/10.3389/fphys.2022.1017948
67. Shi, X., Zhang, Z., Zhang, C., Zhou, X., Zhang, D., & Liu, Y. (2021). The molecular mechanism of efficient transmission of plant viruses in variable virus–vector–plant interactions. *Horticultural Plant Journal*, *7*(6), 501–508. https://doi.org/10.1016/j.hpj.2021.04.006
68. Fereres, A., Peñaflor, M., Favaro, C., Azevedo, K., Landi, C., Maluta, N., Bento, J., & Lopes, J. (2016). Tomato Infection by Whitefly-Transmitted Circulative and Non-Circulative Viruses Induce Contrasting Changes in Plant Volatiles and Vector Behaviour. *Viruses*, *8*(8), 225. https://doi.org/10.3390/v8080225
69. Moreno-Delafuente, A., Garzo, E., Moreno, A., & Fereres, A. (2013). A Plant Virus Manipulates the Behavior of Its Whitefly Vector to Enhance Its Transmission Efficiency and Spread. *PLoS ONE*, *8*(4), e61543. https://doi.org/10.1371/journal.pone.0061543
70. Wang, J. (2009). *Reverse genetics systems and replication studies of a Crinivirus, Lettuce infectious yellows virus (LIYV*.
71. Sun, Z., Liu, Z., Zhou, W., Jin, H., Liu, H., Zhou, A., Zhang, A., & Wang, M.-Q. (2016). Temporal interactions of plant-insect-predator after infection of bacterial pathogen on rice plants. *Scientific Reports*, *6*(1), 26043. https://doi.org/10.1038/srep26043
72. Meijer, D. (2023). *The effects of far-red light on plant-arthropod interactions and the implications for greenhouse tomato cultivation*. https://doi.org/10.18174/631182
73. Zhao, S., & Li, Y. (2021). Current understanding of the interplays between host hormones and plant viral infections. *PLOS Pathogens*, *17*(2), e1009242. https://doi.org/10.1371/journal.ppat.1009242
74. Zenda, M. F. (2024). *Investigation into the exogenous priming effects of salicylic acid and methyl-jasmonate on tomato curly stunt virus infection*.
75. Ziegler-Graff, V. (2020). Molecular Insights into Host and Vector Manipulation by Plant Viruses. *Viruses*, *12*(3), 263. https://doi.org/10.3390/v12030263
76. Wu, X., & Ye, J. (2020). Manipulation of Jasmonate Signaling by Plant Viruses and Their Insect Vectors. *Viruses*, *12*(2), 148. https://doi.org/10.3390/v12020148
77. Schlaeger, S., Pickett, J. A., & Birkett, M. A. (2018). Prospects for management of whitefly using plant semiochemicals, compared with related pests. *Pest Management Science*, *74*(11), 2405–2411. https://doi.org/10.1002/ps.5058
78. Zhang, Z., Zhang, B., He, H., Yan, M., Li, J., & Yan, F. (2022). Changes in Visual and Olfactory Cues in Virus-Infected Host Plants Alter the Behavior of Bemisia tabaci. *Frontiers in Ecology and Evolution*, *10*. https://doi.org/10.3389/fevo.2022.766570
79. Zhao, K. (2020). *Two Orthotospoviruses Pathosystem: Interaction of Plantviruses-Vector*.
80. Dryburgh, J. L. (2019). *Effects of Virus Infection and Volatiles on Aphid Virus Vector Behavior on Sweetpotato*. https://doi.org/10.31390/gradschool_dissertations.5008
81. Yu, X., Zhu, Y., Yin, G., Wang, Y., Shi, X., & Cheng, G. (2024). Exploiting hosts and vectors: viral strategies for facilitating transmission. *EMBO Reports*, *25*(8), 3187–3201. https://doi.org/10.1038/s44319-024-00214-6
82. Padmanabhan, M. S., Kramer, S. R., Wang, X., & Culver, J. N. (2008). Tobacco Mosaic Virus Replicase-Auxin/Indole Acetic Acid Protein Interactions: Reprogramming the Auxin Response Pathway To Enhance Virus Infection. *Journal of Virology*, *82*(5), 2477–2485. https://doi.org/10.1128/jvi.01865-07
83. Sharma, S., & Prasad, M. (2025). Diverse roles of phytohormonal signaling in modulating plant–virus interactions. *Journal of Experimental Botany*, *76*(7), 1921–1940. https://doi.org/10.1093/jxb/erae468
84. Bhattacharjee, B., & Hallan, V. (2022). Role of Plant Transcription Factors in Virus Stress. *Transcription Factors for Biotic Stress Tolerance in Plants*, 79–102. https://doi.org/10.1007/978-3-031-12990-2_5
85. Zarate, S. I., Kempema, L. A., & Walling, L. L. (2006). Silverleaf Whitefly Induces Salicylic Acid Defenses and Suppresses Effectual Jasmonic Acid Defenses. *Plant Physiology*, *143*(2), 866–875. https://doi.org/10.1104/pp.106.090035
86. Murphy, A. M., Zhou, T., & Carr, J. P. (2020). An update on salicylic acid biosynthesis, its induction and potential exploitation by plant viruses. *Current Opinion in Virology*, *42*, 8–17. https://doi.org/10.1016/j.coviro.2020.02.008
87. Pan, L., Miao, H., Wang, Q., Walling, L. L., & Liu, S. (2021). Virus-induced phytohormone dynamics and their effects on plant–insect interactions. *New Phytologist*, *230*(4), 1305–1320. https://doi.org/10.1111/nph.17261
88. Qin, Y., Zhao, J., Wang, J., Ye, X., Zhou, C., & Zhou, Y. (2023). Regulation of Nicotiana benthamiana cell death induced by citrus chlorotic dwarf-associated virus-RepA protein by WRKY 1. *Frontiers in Plant Science*, *14*, 1164416. https://doi.org/10.3389/fpls.2023.1164416
89. Landeo-Ríos, Y., Navas-Castillo, J., Moriones, E., & Cañizares, M. C. (2016). The p22 RNA silencing suppressor of the crinivirus Tomato chlorosis virus preferentially binds long dsRNAs preventing them from cleavage. *Virology*, *488*, 129–136. https://doi.org/10.1016/j.virol.2015.11.008
90. Silva, E. G., de Faria, I. J. S., Ferreira, Á. G. A., Jiran, T. H. L., Estevez-Castro, C. F., Armache, J. N., Amadou, S. C. G., Verdier, Y., Vinh, J., Majzoub, K., Meignin, C., Haas, G., Martin, F., Imler, J.-L., & Marques, J. T. (2025). Argonaute 2 targets viral transcripts but not genomes of RNA viruses during antiviral RNA interference in Drosophila. *PLOS Pathogens*, *21*(2), e1012184. https://doi.org/10.1371/journal.ppat.1012184
91. Cañizares, M. C., Navas-Castillo, J., & Moriones, E. (2008). Multiple suppressors of RNA silencing encoded by both genomic RNAs of the crinivirus, Tomato chlorosis virus. *Virology*, *379*(1), 168–174. https://doi.org/10.1016/j.virol.2008.06.020
92. Xu, Y., Zhang, S., Zhang, M., Jiao, S., Guo, Y., & Jiang, T. (2024). The role of reactive oxygen species in plant-virus interactions. *Plant Cell Reports*, *43*(8), 197. https://doi.org/10.1007/s00299-024-03280-1
93. Jin, X., Jiang, Z., Zhang, K., Wang, P., Cao, X., Yue, N., Wang, X., Zhang, X., Li, Y., Li, D., Kang, B.-H., & Zhang, Y. (2018). Three-Dimensional Analysis of Chloroplast Structures Associated with Virus Infection. *Plant Physiology*, *176*(1), 282–294. https://doi.org/10.1104/pp.17.00871

94. Shahriari, A. G., Tahmasebi, A., Ghodoum Parizipour, M. H., Soltani, Z., Tahmasebi, A., & Shahid, M. S. (2025). The crucial role of mitochondrial/chloroplast-related genes in viral genome replication and host defense: integrative systems biology analysis in plant-virus interaction. *Frontiers in Microbiology*, *16*, 1551123. https://doi.org/10.3389/fmicb.2025.1551123
95. Zhao, J., Zhang, X., Hong, Y., & Liu, Y. (2016). Chloroplast in plant-virus interaction. *Frontiers in Microbiology*, *7*, 1565. https://doi.org/10.3389/fmicb.2016.01565
96. Liu, C., Liu, Q., & Mou, Z. (2024). Redox signaling and oxidative stress in systemic acquired resistance. *Journal of Experimental Botany*, *75*(15), 4535–4548. https://doi.org/10.1093/jxb/erae193
97. Zhang, J., Ren, Z., Zhou, Y., Ma, Z., Ma, Y., Hou, D., Xu, Z., & Huang, X. (2019). NPR1 and Redox Rhythm: Connections, between Circadian Clock and Plant Immunity. *International Journal of Molecular Sciences*, *20*(5), 1211. https://doi.org/10.3390/ijms20051211
98. Dagah, O. M. A., Silaa, B. B., Zhu, M., Pan, Q., Qi, L., Liu, X., Liu, Y., Peng, W., Ullah, Z., Yudas, A. F., Muhammad, A., Zhang, X., & Lu, J. (2024). Exploring Immune Redox Modulation in Bacterial Infections: Insights into Thioredoxin-Mediated Interactions and Implications for Understanding Host–Pathogen Dynamics. *Antioxidants*, *13*(5), 545. https://doi.org/10.3390/antiox13050545
99. Gostner, J. M., Becker, K., Fuchs, D., & Sucher, R. (2013). Redox regulation of the immune response. *Redox Report*, *18*(3), 88–94. https://doi.org/10.1179/1351000213y.0000000044
100. Morris, G., Gevezova, M., Sarafian, V., & Maes, M. (2022). Redox regulation of the immune response. *Cellular & Molecular Immunology*, *19*(10), 1079–1101. https://doi.org/10.1038/s41423-022-00902-0
101. Jiang, T., Hao, T., Chen, W., Li, C., Pang, S., Fu, C., Cheng, J., Zhang, C., Ghorbanpour, M., & Miao, S. (2025). Reprogrammed Plant Metabolism During Viral Infections: Mechanisms, Pathways and Implications. *Molecular Plant Pathology*, *26*(2), e70066. https://doi.org/10.1111/mpp.70066
102. Lowen, A. C. (2018). It's in the mix: Reassortment of segmented viral genomes. *PLOS Pathogens*, *14*(9), e1007200. https://doi.org/10.1371/journal.ppat.1007200
103. McDonald, S. M., Nelson, M. I., Turner, P. E., & Patton, J. T. (2016). Reassortment in segmented RNA viruses: mechanisms and outcomes. *Nature Reviews Microbiology*, *14*(7), 448–460. https://doi.org/10.1038/nrmicro.2016.46
104. Ruiz, L., Simón, A., García, C., Velasco, L., & Janssen, D. (2018). First natural crossover recombination between two distinct species of the family Closteroviridae leads to the emergence of a new disease. *PLOS ONE*, *13*(9), e0198228. https://doi.org/10.1371/journal.pone.0198228
105. Bentley, K., & Evans, D. J. (2018). Mechanisms and consequences of positive-strand RNA virus recombination. *Journal of General Virology*, *99*(10), 1345–1356. https://doi.org/10.1099/jgv.0.001142
106. Simon-Loriere, E., & Holmes, E. C. (2011). Why do RNA viruses recombine? *Nature Reviews Microbiology*, *9*(8), 617–626. https://doi.org/10.1038/nrmicro2614
107. Ansar, M., Agnihotri, A. K., & Srinivasaraghavan, A. (2019). Virus Diseases: An Inimitable Pathosystem of Vegetable Crop. *The Vegetable Pathosystem: Ecology, Disease Mechanism, and Management*, 313–341. https://doi.org/10.1201/9780429022999-12
108. Wuest, S. E., Peter, R., & Niklaus, P. A. (2021). Ecological and evolutionary approaches to improving crop variety mixtures. *Nature Ecology & Evolution*, *5*(8), 1068–1077. https://doi.org/10.1038/s41559-021-01497-x
109. LaTourrette, K., & Garcia-Ruiz, H. (2022). Determinants of Virus Variation, Evolution, and Host Adaptation. *Pathogens*, *11*(9), 1039. https://doi.org/10.3390/pathogens11091039
110. Fiallo-Olivé, E., & Navas-Castillo, J. (2023). The Role of Extensive Recombination in the Evolution of Geminiviruses. *Viral Fitness and Evolution*, *439*, 139–166. https://doi.org/10.1007/978-3-031-15640-3_4
111. Saldarelli, P., Giampetruzzi, A., Maree, H. J., & Rwahnih, M. A. (2017). High-Throughput Sequencing: Advantages Beyond Virus Identification. *Grapevine Viruses: Molecular Biology, Diagnostics and Management*, 625–642. https://doi.org/10.1007/978-3-319-57706-7_30
112. Domingo, E., & Perales, C. (2019). Viral quasispecies. *PLOS Genetics*, *15*(10), e1008271. https://doi.org/10.1371/journal.pgen.1008271
113. Tangkanchanapas, P., Haegeman, A., Ruttink, T., Höfte, M., & De Jonghe, K. (2020). Whole-Genome Deep Sequencing Reveals Host-Driven in-planta Evolution of Columnea Latent Viroid (CLVd) Quasi-Species Populations. *International Journal of Molecular Sciences*, *21*(9), 3262. https://doi.org/10.3390/ijms21093262
114. Williamson, C. H. D., Stone, N. E., Nunnally, A. E., Roe, C. C., Vazquez, A. J., Lucero, S. A., Hornstra, H., Wagner, D. M., Keim, P., Rupnik, M., Janezic, S., & Sahl, J. W. (2022). Identification of novel, cryptic Clostridioides species isolates from environmental samples collected from diverse geographical locations. *Microbial Genomics*, *8*(2), 000742. https://doi.org/10.1099/mgen.0.000742
115. Torralba, B., Blanc, S., & Michalakis, Y. (2024). Reassortments in single-stranded DNA multipartite viruses: Confronting expectations based on molecular constraints with field observations. *Virus Evolution*, *10*(1), veae010. https://doi.org/10.1093/ve/veae010
116. Orfanidou, C. G., Baltzi, A., Dimou, N. A., Katis, N. I., & Maliogka, V. I. (2017). Cucurbit chlorotic yellows virus: Insights Into Its Natural Host Range, Genetic Variability, and Transmission Parameters. *Plant Disease*, *101*(12), 2053–2058. https://doi.org/10.1094/pdis-02-17-0164-re
117. Grumet, R., McCreight, J. D., McGregor, C., Weng, Y., Mazourek, M., Reitsma, K., Labate, J., Davis, A., & Fei, Z. (2021). Genetic Resources and Vulnerabilities of Major Cucurbit Crops. *Genes*, *12*(8), 1222. https://doi.org/10.3390/genes12081222
118. Paredes-Montero, J. R., Haq, Q. M. I., Mohamed, A. A., & Brown, J. K. (2021). Phylogeographic and SNPs Analyses of Bemisia tabaci B Mitotype Populations Reveal Only Two of Eight Haplotypes Are Invasive. *Biology*, *10*(10), 1048. https://doi.org/10.3390/biology10101048
119. Meynard, J.-M., Doré, T., & Lucas, P. (2003). Agronomic approach: cropping systems and plant diseases. *Comptes Rendus. Biologies*, *326*(1), 37–46. https://doi.org/10.1016/s1631-0691(03)00006-4

120. Bester, R., Cook, G., Breytenbach, J. H. J., Steyn, C., De Bruyn, R., & Maree, H. J. (2021). Towards the validation of high-throughput sequencing (HTS) for routine plant virus diagnostics: measurement of variation linked to HTS detection of citrus viruses and viroids. *Virology Journal*, *18*(1), 61. https://doi.org/10.1186/s12985-021-01523-1
121. Kutnjak, D., Tamisier, L., Adams, I., Boonham, N., Candresse, T., Chiumenti, M., Jonghe, K. D., Kreuze, J. F., Lefebvre, M., Silva, G., Malapi-Wight, M., Margaria, P., Mavrič Pleško, I., McGreig, S., Miozzi, L., Remenant, B., Reynard, J.-S., Rollin, J., Rott, M., … Haegeman, A. (2021). A Primer on the Analysis of High-Throughput Sequencing Data for Detection of Plant Viruses. *Microorganisms*, *9*(4), 841. https://doi.org/10.3390/microorganisms9040841
122. Sánchez-Sánchez, M., Vargas-Arispuro, I., Tovar-Pedraza, J. M., González-Pérez, C. J., Aispuro-Hernández, E., Quintana-Obregón, E. A., & Martínez-Téllez, M. Á. (2024). Mixed viral infections in vegetable crops: biochemical and molecular aspects. *Revista Mexicana de Fitopatología, Mexican Journal of Phytopathology*, *42*(3), 2404–3. https://doi.org/10.18781/r.mex.fit.2404-3
123. Tatineni, S., & Hein, G. L. (2023). Plant Viruses of Agricultural Importance: Current and Future Perspectives of Virus Disease Management Strategies. *Phytopathology*, *113*(2), 117–141. https://doi.org/10.1094/phyto-05-22-0167-rvw
124. Murphy, J. F., & Bowen, K. L. (2006). Synergistic Disease in Pepper Caused by the Mixed Infection of Cucumber mosaic virus and Pepper mottle virus. *Phytopathology®*, *96*(3), 240–247. https://doi.org/10.1094/phyto-96-0240
125. Srinivasan, R., & Alvarez, J. M. (2007). Effect of Mixed Viral Infections (Potato Virus Y–Potato Leafroll Virus) on Biology and Preference of Vectors Myzus persicae and Macrosiphum euphorbiae (Hemiptera: Aphididae). *Journal of Economic Entomology*, *100*(3), 646–655. https://doi.org/10.1093/jee/100.3.646
126. Angira, A., Sharma, T., Choudhary, N., Rishi, N., Verma, H. N., & Awasthi, L. P. (2024). Viral diseases of field and horticultural crops. *Plant Pathology and Plant Diseases*, 15–21. https://doi.org/10.1016/b978-0-323-90899-3.00015-x
127. Gautam, S., Gadhave, K. R., Buck, J. W., Dutta, B., Coolong, T., Adkins, S., & Srinivasan, R. (2020). Virus-virus interactions in a plant host and in a hemipteran vector: Implications for vector fitness and virus epidemics. *Virus Research*, *286*, 198069. https://doi.org/10.1016/j.virusres.2020.198069
128. Pruss, G., Ge, X., Shi, X. M., Carrington, J. C., & Bowman Vance, V. (1997). Plant viral synergism: the potyviral genome encodes a broad-range pathogenicity enhancer that transactivates replication of heterologous viruses. *The Plant Cell*, *9*(6), 859–868. https://doi.org/10.1105/tpc.9.6.859
129. Del, T. F. J., Donaire, L., Aguilar, E., Chung, B.-N., Tenllado, F., & Canto, T. (2017). Potato Virus Y HCPro Suppression of Antiviral Silencing in Nicotiana benthamiana Plants Correlates with Its Ability To Bind In Vivo to 21- and 22-Nucleotide Small RNAs of Viral Sequence. *Journal of Virology*, *91*(12), e00367. https://doi.org/10.1128/jvi.00367-17
130. Landeo-Ríos, Y., Navas-Castillo, J., Moriones, E., & Cañizares, M. (2017). The Heterologous Expression of the p22 RNA Silencing Suppressor of the Crinivirus Tomato Chlorosis Virus from Tobacco Rattle Virus and Potato Virus X Enhances Disease Severity but Does Not Complement Suppressor-Defective Mutant Viruses. *Viruses*, *9*(12), 358. https://doi.org/10.3390/v9120358
131. Sangeetha, E., & Jebasingh, T. (2021). Virus-host interactome of Potyviridae. *Plant Virus-Host Interaction: Molecular Approaches and Viral Evolution*, 169–189. https://doi.org/10.1016/b978-0-12-821629-3.00015-4
132. Abrahamian, P., Sobh, H., Seblani, R., & Abou-Jawdah, Y. (2015). Co-infection of two criniviruses and a begomovirus enhances the disease severity in cucumber. *European Journal of Plant Pathology*, *142*(3), 521–530. https://doi.org/10.1007/s10658-015-0630-y
133. Jiang, T., & Zhou, T. (2023). Unraveling the Mechanisms of Virus-Induced Symptom Development in Plants. *Plants*, *12*(15), 2830. https://doi.org/10.3390/plants12152830
134. Morales, K., Zambrano, J. L., & Stewart, L. R. (2014). Co-infection and Disease Severity of Ohio Maize dwarf mosaic virus and Maize chlorotic dwarf virus Strains. *Plant Disease*, *98*(12), 1661–1665. https://doi.org/10.1094/pdis-12-13-1230-re
135. Mehetre, G. T., Leo, V. V., Singh, G., Sorokan, A., Maksimov, I., Yadav, M. K., Upadhyaya, K., Hashem, A., Alsaleh, A. N., Dawoud, T. M., Almaary, K. S., & Singh, B. P. (2021). Current Developments and Challenges in Plant Viral Diagnostics: A Systematic Review. *Viruses*, *13*(3), 412. https://doi.org/10.3390/v13030412
136. Mrkvová, M., Hančinský, R., Grešíková, S., Kaňuková, Š., Barilla, J., Glasa, M., Hauptvogel, P., Kraic, J., & Mihálik, D. (2022). Evaluation of New Polyclonal Antibody Developed for Serological Diagnostics of Tomato Mosaic Virus. *Viruses*, *14*(6), 1331. https://doi.org/10.3390/v14061331
137. Roy, S. D., Ramasamy, S., & Obbineni, J. M. (2024). An evaluation of nucleic acid-based molecular methods for the detection of plant viruses: a systematic review. *VirusDisease*, *35*(2), 357–376. https://doi.org/10.1007/s13337-024-00863-0
138. Massart, S., Olmos, A., Jijakli, H., & Candresse, T. (2014). Current impact and future directions of high throughput sequencing in plant virus diagnostics. *Virus Research*, *188*, 90–96. https://doi.org/10.1016/j.virusres.2014.03.029
139. Alazem, M., & Lin, N. (2015). Roles of plant hormones in the regulation of host–virus interactions. *Molecular Plant Pathology*, *16*(5), 529–540. https://doi.org/10.1111/mpp.12204
140. Syller, J., & Grupa, A. (2016). Antagonistic within-host interactions between plant viruses: molecular basis and impact on viral and host fitness. *Molecular Plant Pathology*, *17*(5), 769–782. https://doi.org/10.1111/mpp.12322
141. Gilbertson, R. L., Batuman, O., Webster, C. G., & Adkins, S. (2015). Role of the Insect Supervectors Bemisia tabaci and Frankliniella occidentalis in the Emergence and Global Spread of Plant Viruses. *Annual Review of Virology*, *2*(1), 67–93. https://doi.org/10.1146/annurev-virology-031413-085410
142. Minicka, J., Zarzyńska-Nowak, A., Budzyńska, D., Borodynko-Filas, N., & Hasiów-Jaroszewska, B. (2020). High-Throughput Sequencing Facilitates Discovery of New Plant Viruses in Poland. *Plants*, *9*(7), 820. https://doi.org/10.3390/plants9070820
143. Kutnjak, D., Rupar, M., Gutierrez-Aguirre, I., Curk, T., Kreuze, J. F., & Ravnikar, M. (2015). Deep Sequencing of Virus-Derived Small Interfering RNAs and RNA from Viral Particles Shows Highly Similar Mutational Landscapes of a Plant Virus Population. *Journal of Virology*, *89*(9), 4760–4769. https://doi.org/10.1128/jvi.03685-14
144. Ghosh, D. K. (2025). Innovations in molecular diagnostics, characterization and novel. *Journal of Mycopathological Research*, *63*(63), 477–490. https://doi.org/10.57023/jmycr.63.3.2025.477
145. Jones, R. A. C. (2021). Global Plant Virus Disease Pandemics and Epidemics. *Plants*, *10*(2), 233. https://doi.org/10.3390/plants10020233

146. Xue, Y., Lin, C., Wang, Y., Liu, W., Wan, F., Zhang, Y., & Ji, L. (2022). Predicting Climate Change Effects on the Potential Distribution of Two Invasive Cryptic Species of the Bemisia tabaci Species Complex in China. *Insects*, *13*(12), 1081. https://doi.org/10.3390/insects13121081
147. Nava-Camberos, U., Riley, D. G., & Harris, M. K. (2001). Temperature and Host Plant Effects on Development, Survival, and Fecundity of Bemisia argentifolii (Homoptera: Aleyrodidae). *Environmental Entomology*, *30*(1), 55–63. https://doi.org/10.1603/0046-225x-30.1.55
148. Liu, B., Preisser, E. L., Chu, D., Pan, H., Xie, W., Wang, S., Wu, Q., Zhou, X., & Zhang, Y. (2013). Multiple Forms of Vector Manipulation by a Plant-Infecting Virus: Bemisia tabaci and Tomato Yellow Leaf Curl Virus. *Journal of Virology*, *87*(9), 4929–4937. https://doi.org/10.1128/jvi.03571-12
149. Moya-Ruiz, C. de, Ferriol, I., & Gómez, P. (2024). The Temporal Order of Mixed Viral Infections Matters: Common Events That Are Neglected in Plant Viral Diseases. *Viruses*, *16*(12), 1954. https://doi.org/10.3390/v16121954
150. Trebicki, P. (2020). Climate change and plant virus epidemiology. *Virus Research*, *286*, 198059. https://doi.org/10.1016/j.virusres.2020.198059
151. Orfanidou, C. G., Pappi, P. G., Efthimiou, K. E., Katis, N. I., & Maliogka, V. I. (2016). Transmission of Tomato chlorosis virus (ToCV) by Bemisia tabaci Biotype Q and Evaluation of Four Weed Species as Viral Sources. *Plant Disease*, *100*(10), 2043–2049. https://doi.org/10.1094/pdis-01-16-0054-re
152. Wisler, G. C., Duffus, J. E., Liu, H.-Y., & Li, R. H. (1998). Ecology and Epidemiology of Whitefly-Transmitted Closteroviruses. *Plant Disease*, *82*(3), 270–280. https://doi.org/10.1094/pdis.1998.82.3.270
153. Li, J., Liang, X., Wang, X., Shi, Y., Gu, Q., Kuo, Y.-W., Falk, B. W., & Yan, F. (2016). Direct evidence for the semipersistent transmission of Cucurbit chlorotic yellows virus by a whitefly vector. *Scientific Reports*, *6*(1), 36604. https://doi.org/10.1038/srep36604
154. Celix, A., Almarza, N., Rodríguez-Cerezo, E., López-Sesé, A., & Gómez-Guillamón, M. L. (1996). Characterization of Cucurbit yellow stunting disorder virus, a Bemisia tabaci-transmitted closterovirus. *Phytopathology*, *86*(12), 1370–1376. https://doi.org/10.1094/Phyto-86-1370.
155. Elith, J., & Leathwick, J. R. (2009). Species Distribution Models: Ecological Explanation and Prediction Across Space and Time. *Annual Review of Ecology, Evolution, and Systematics*, *40*(1), 677–697. https://doi.org/10.1146/annurev.ecolsys.110308.120159
156. Madden, L. V., Hughes, G., & Bosch, F. van den. (2007). *The study of plant disease epidemics*. *100*.
157. Palaniyandi, M., Sharmila, T., Manivel, P., Thirumalai, P., & Anand, P. (2021). Multispectral Satellite Data and GIS for Mapping Vector Ecology, Monitoring, Risk Assessment, and Forecast of Vector Borne Disease Epidemics: A Systematic Review. *Applied Ecology and Environmental Sciences*, *9*(8), 751–760. https://doi.org/10.12691/aees-9-8-6
158. Kamilaris, A., & Prenafeta-Boldú, F. X. (2018). Deep learning in agriculture: A survey. In *Computers and Electronics in Agriculture* (Vol. 147, pp. 70–90). https://doi.org/10.1016/j.compag.2018.02.016
159. Liakos, K., Busato, P., Moshou, D., Pearson, S., & Bochtis, D. (2018). Machine Learning in Agriculture: A Review. In *Sensors* (Vol. 18, Issue 8, p. 2674). https://doi.org/10.3390/s18082674
160. Boykin, L., Ghalab, A., Rossitto De Marchi, B., Savill, A., Wainaina, J. M., Kinene, T., Lamb, S., Rodrigues, M., Kehoe, M., Ndunguru, J., Tairo, F., Sseruwagi, P., Kayuki, C., Mark, D., Erasto, J., Bachwenkizi, H., Alicai, T., Okao-Okuja, G., Abridrabo, P., … Kiarie, S. (2018). Real time portable genome sequencing for global food security. *F1000Research*, *7*, 1101. https://doi.org/10.12688/f1000research.15507.1
161. Pandey, S., & Mishra, S. (2023). CRISPR/Cas-Based Strategies for Resistance Against Plant Viruses. *CRISPR/Cas Systems: Plant Genome Editing and Agriculture*, 391–414. https://doi.org/10.1201/9781003331759-19
162. Liu, H., Wu, K., Wu, W., Mi, W., Hao, X., & Wu, Y. (2019). A multiplex reverse transcription PCR assay for simultaneous detection of six main RNA viruses in tomato plants. *Journal of Virological Methods*, *265*, 53–58. https://doi.org/10.1016/j.jviromet.2018.12.011
163. Naranjo, S. E., Castle, S. J., De Barro, P. J., & Liu, S.-S. (2009). Population Dynamics, Demography, Dispersal and Spread of Bemisia tabaci. *Bemisia: Bionomics and Management of a Global Pest*, 185–226. https://doi.org/10.1007/978-90-481-2460-2_6
164. Togni, P. H. B., Harterreiten-Souza, É. S., Novaes, D. R., & Sujii, E. R. (2021). Spatial dynamic and spillover of the polyphagous pest Bemisia tabaci is influenced by differences in farmland habitats on tropical organic farms. *Agriculture, Ecosystems & Environment*, *320*, 107610. https://doi.org/10.1016/j.agee.2021.107610
165. Braun, A., & Duveskog, D. (2011). *The Farmer Field School approach–History, global assessment and success stories*.
166. Kiptot, E., Karuhanga, M., Franzel, S., & Nzigamasabo, P. B. (2016). Volunteer farmer-trainer motivations in East Africa: practical implications for enhancing farmer-to-farmer extension. *International Journal of Agricultural Sustainability*, *14*(3), 339–356. https://doi.org/10.1080/14735903.2015.1137685
167. Garrett, K. A., Nita, M., Wolf, E. D., Esker, P. D., Gomez-Montano, L., & Sparks, A. H. (2021). Plant pathogens as indicators of climate change. *Climate Change and Crop Production*, 499–513. https://doi.org/10.1016/b978-0-12-821575-3.00024-4
168. Ristaino, J. B., Anderson, P. K., Bebber, D. P., Brauman, K. A., Cunniffe, N. J., Fedoroff, N. V., Finegold, C., Garrett, K. A., Gilligan, C. A., Jones, C. M., Martin, M. D., MacDonald, G. K., Neenan, P., Records, A., Schmale, D. G., Tateosian, L., & Wei, Q. (2021). The persistent threat of emerging plant disease pandemics to global food security. *Proceedings of the National Academy of Sciences*, *118*(23), 2022239118. https://doi.org/10.1073/pnas.2022239118
169. Donatelli, M., Magarey, R. D., Bregaglio, S., Willocquet, L., Whish, J. P. M., & Savary, S. (2017). Modelling the impacts of pests and diseases on agricultural systems. *Agricultural Systems*, *155*, 213–224. https://doi.org/10.1016/j.agsy.2017.01.019
170. Jeger, M. J., Pautasso, M., Holdenrieder, O., & Shaw, M. W. (2007). Modelling disease spread and control in networks: implications for plant sciences. *New Phytologist*, *174*(2), 279–297. https://doi.org/10.1111/j.1469-8137.2007.02028.x
171. Savary, S., Willocquet, L., Pethybridge, S. J., Esker, P., McRoberts, N., & Nelson, A. (2019). The Global Burden of Pathogens and Pests on Major Food Crops. *Nature Ecology and Evolution*, *3*(3), 430–439. https://doi.org/10.1038/s41559-018-0793-y
172. Nelson, R., Coe, R., & Haussmann, B. I. G. (2019). Farmer research networks as a strategy for matching diverse options and contexts in smallholder agriculture. *Experimental Agriculture*, *55*(S1), 125–144. https://doi.org/10.1017/s0014479716000454

173. Altieri, M. A., & Nicholls, C. I. (2020). Agroecology and the emergence of a post COVID-19 agriculture. *Agriculture and Human Values*, *37*(3), 525–526. https://doi.org/10.1007/s10460-020-10043-7
174. Ratnadass, A., Fernandes, P., Avelino, J., & Habib, R. (2012). Plant species diversity for sustainable management of crop pests and diseases in agroecosystems: a review. *Agronomy for Sustainable Development*, *32*(1), 273–303. https://doi.org/10.1007/s13593-011-0022-4