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Current Opinion in Virology

Vector mediated transmission of persistently transmitted plant viruses

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Many vector-borne plant viruses of agricultural importance are persistently transmitted from plant to plant by sap-sucking insects. So far, the mechanisms for vector-mediated horizontal transmission of the viruses to plant hosts and for vertical transmission to insect offspring have been poorly understood. During horizontal transmission, intact virions or virus-induced inclusions are exploited by persistently transmitted viruses to overcome the midgut and salivary gland barriers. The existing oocyte entry paths used by vitellogenin or symbiont bacteria can mediate the vertical transmission of viruses by female insects. We hypothesize that the viruses may also be vertically transmitted by male insects via attachment to the surface of sperm. Inhibiting vertical transmission of the viruses by insect vectors in the overwintering season unfavorable for horizontal transmission may open new perspectives for viral control.

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Current Opinion in Virology 2018, **28**:127–132

This review comes from a themed issue on **Emerging viruses: intraspecies transmission**

Edited by **Sander Herfst** and **Martin Ludlow**

https://doi.org/10.1016/j.coviro.2017.12.004

Introduction

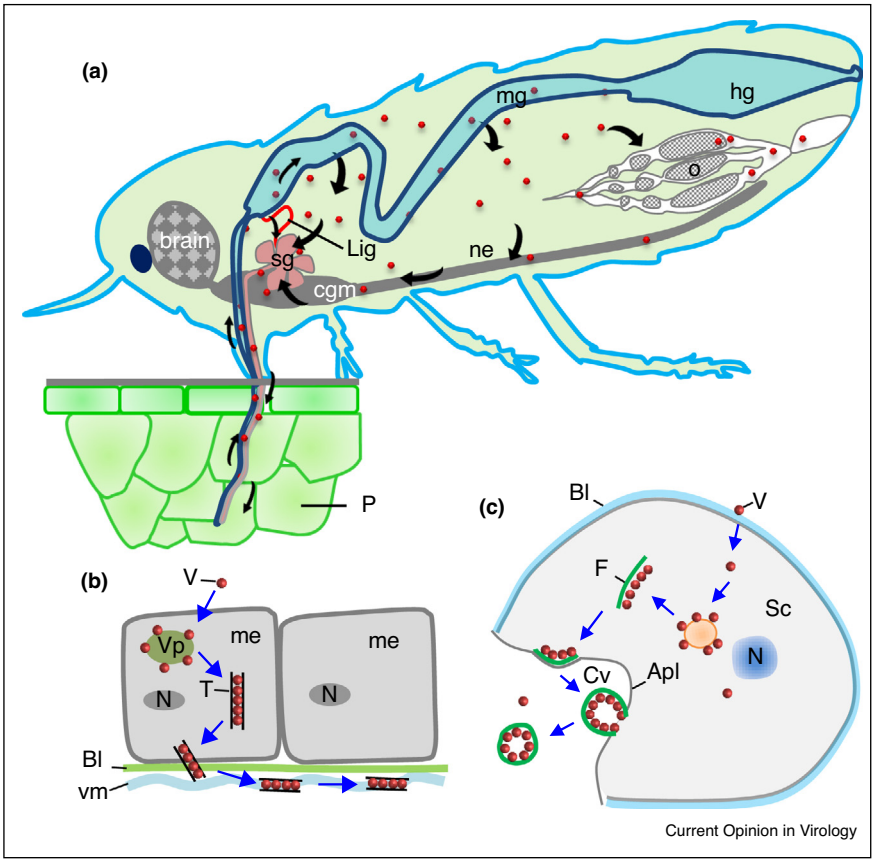
Many plant viral pathogens that cause significant agricultural problems are transmitted from plant to plant in a persistent manner by sap-sucking insects, including aphids, whiteflies, leafhoppers, planthoppers, and thrips [1]. Persistently transmitted viruses are classified into two groups: propagative and non-propagative viruses. In persistent-propagative transmission, viruses can replicate and induce viral inclusions in the insect vectors [2,3]. Persistently transmitted plant viruses, when ingested by their insect vectors, establish initial infection in the midgut epithelium, from where they disseminate to the midgut visceral muscles [4,5]. Viruses then spread into the hemolymph and eventually either into the salivary glands to be horizontally transmitted to healthy plants, or into the female ovary to be vertically transmitted to offspring [1,6]. In this review, we will describe how persistently transmitted plant viruses overcome multiple tissue or membrane barriers in the insect vectors during horizontal transmission to plant hosts or vertical transmission to insect offspring.

Horizontal transmission to plant hosts

Midgut barriers

The first bottleneck for viral infection, the midgut, has been long recognized as a major determinant of vector competence for a variety of viruses. However, the molecular mechanisms that specifically modulate the persistent infection of plant viruses within insect midguts are poorly understood. Electron microscopy observations reveal that persistently transmitted nonpropagative luteoviruses and begomoviruses overcome midgut barriers by intact virions [7,8]. Persistently transmitted propagative viruses, however, take advantage of viral inclusions to facilitate viral spread in the insect midgut [5,9••]. For example, rice reoviruses exploit the tubular structures, which are constituted by viral nonstructural proteins, to pass through the microvilli of the midgut epithelium into the lumen, or to pass through the basal lamina from the midgut epithelium into the visceral circular muscles of planthopper or leafhopper vectors [5,9••] (Figure 1). The midgut actin–myosin complex provides the necessary power to propel the tubules of rice reoviruses through the tightly regulated membrane or tissue barriers of the insect midgut epithelium [9••,10–12]. We name this mechanism as 'actin-based tubule motility'. Aside from the physical barriers in the insects, several antiviral pathways are used to modulate the persistent transmission of plant viruses by insect vectors. Generally, a conserved small interfering RNA (siRNA) antiviral pathway can be triggered in response to the propagation of plant viruses in insect vectors [13,14]. Such a virus-induced siRNA antiviral pathway has been shown to efficiently restrict the accumulation of southern rice black streaked dwarf virus, a plant reovirus, in the midgut epithelium of small brown planthopper, which finally affects the capacity for viral dissemination [14]. In addition, an autophagy pathway is induced by tomato yellow leaf curl virus (TYLCV), a

Figure 1



Infection route of plant viruses in an insect vector during horizontal transmission. **(a)** In the insect vector, plant viruses first infect and replicate in the midgut epithelium, then overcome the basal lamina barrier, move through the hemolymph, nervous system or ligament-like structure direct contact the midgut and salivary glands to infect the salivary glands. **(b)** In the midgut, rice reoviruses exploit virus-containing tubules to pass through the basal lamina from the midgut epithelium to the visceral muscle. **(c)** In salivary glands, RGDV crosses the primary salivary glands (PSG) via an exocytosis-like process mediated by virus-induced filaments (for details, see [29••]). Arrows indicate the process of viral spread. Apl, apical plasmalemma; Bl, basal lamina; cgm, compound ganglionic mass; Cv, cavity; F, filaments; hg, hindgut; Lig, ligament-like structure; me, midgut epithelium; mg, midgut; N, nucleus; ne, nerves; O, ovary; P, phloem; Sc, salivary cytoplasm; sg, salivary gland; T, tubule; V, virus particle; vm, visceral muscle; Vp, viroplasm.

begomovirus, in the midgut of the whitely *Bemisia tabaci* as an intrinsic antiviral defense mechanism [15••]. By contrast, plant viruses may also manipulate vector immune systems for efficient infection of insect cells. Wang *et al.* recently reported that the outer capsid protein of rice stripe virus (RSV), a tenuivirus, can activate the c-Jun N-terminal kinase pathway of its planthopper vector to promote its replication [16]. After viral dissemination from the visceral muscles into the hemolymph, a cuticular protein seems to interact with the outer capsid protein of RSV to prevent the degradation of virus particles by the insect immune system [17]. Thus, we deduce that a metastable balance may exist between the ability of a virus to overcome the midgut barrier and the ability of an insect vector to control viral dissemination, allowing for viral persistence and highly efficient spread in nature [18,19].

Salivary gland barriers

Insect salivary glands represent the last barrier for viral transmission. In general, virions are secreted along with saliva to the salivary cavities from the secretory region and then pass through the stylet canal, from which the virions are ejected during feeding. The mechanism by which persistently transmitted plant viruses overcome the salivary gland barriers is poorly understood. It has been well documented that the hemolymph, nervous system, or cellular structures providing the direct contact between the midgut and salivary gland have been used by persistently transmitted plant viruses as the routes to spread into the salivary glands of insect vectors [20,21•,22•] (Figure 1). In the hemolymph, the GroEL homolog proteins encoded by endosymbiotic bacteria of vector aphids or whitefly have been demonstrated to interact with the capsid proteins of luteoviruses or begomoviruses