Available online at www.sciencedirect.com

ScienceDirect

Current Opinion in Insect Science

Disruption of insect transmission of plant viruses

Anna E Whitfield and Dorith Rotenberg

Plant-infecting viruses are transmitted by a diverse array of organisms including insects, mites, nematodes, fungi, and plasmodiophorids. Virus interactions with these vectors are diverse, but there are some commonalities. Generally the infection cycle begins with the vector encountering the virus in the plant and the virus is acquired by the vector. The virus must then persist in or on the vector long enough for the virus to be transported to a new host and delivered into the plant cell. Plant viruses rely on their vectors for breaching the plant cell wall to be delivered directly into the cytosol. In most cases, viral capsid or membrane glycoproteins are the specific viral proteins that are required for transmission and determinants of vector specificity. Specific molecules in vectors also interact with the virus and while there are few-identified to no-identified receptors, candidate recognition molecules are being further explored in these systems. Due to the specificity of virus transmission by vectors, there are defined steps that represent good targets for interdiction strategies to disrupt the disease cycle. This review focuses on new technologies that aim to disrupt the virus–vector interaction and focuses on a few of the well-characterized virus–vector interactions in the field. In closing, we discuss the importance of integration of these technologies with current methods for plant virus disease control.

Addresses
Department of Plant Pathology, Kansas State University, Manhattan, KS 66502, USA

Corresponding author: Whitfield, Anna E (aewtospo@ksu.edu)

Current Opinion in Insect Science 2015, **8**:79–87

This review comes from a themed issue on **Parasites/Parasitoids/Biological Control**

Edited by **Bryony C Bonning**

For a complete overview see the Issue and the Editorial

Available online 22nd January 2015

http://dx.doi.org/10.1016/j.cois.2015.01.009

2214-5745/© 2015 Elsevier Inc. All rights reserved.

Introduction

The virus transmission cycle involves host-finding, feeding and acquisition of virus, transport and delivery of virus to a new host plant (Figure 1). Each step in the transmission process provides an opportunity for interdiction. Strategies for disrupting transmission are the focus of this review and we highlight recent biotech-based approaches to reduce vectorial capacity and population reduction approaches that utilize the specificity of the virus–vector interaction to target insects.

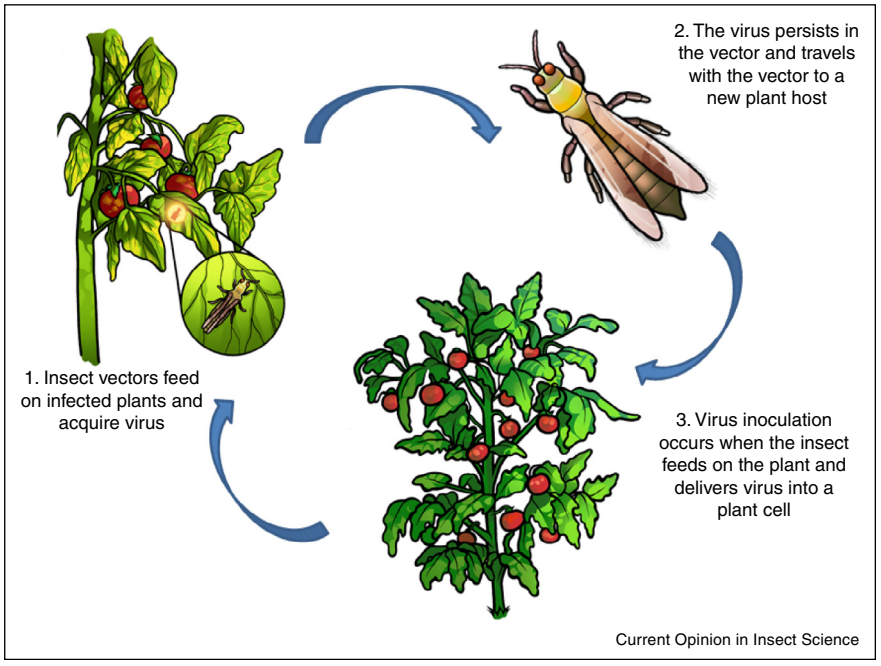
Overview of the mechanisms and methods of plant virus transmission

Plant virus transmission by insects is classified into two major categories: non-circulative and circulative transmission. The non-circulative-externally borne viruses associate with specific cuticular structures of the insect stylet or foregut (Figure 2) and the attached virus particles are lost during the insect molt (reviewed in [1,2]). Non-circulative viruses are transmitted in a non-persistent or semi-persistent manner which means that they are acquired within seconds to minutes of feeding and transmitted rapidly as well. Semi-persistent viruses require longer periods to be acquired and transmitted (minutes to hours). By contrast, the circulative or internally-borne viruses require a greater time for acquisition and transmission (hours to days) and must traverse the gut and reach the salivary glands for transmission to occur. These viruses are not lost during insect molts and have a latent period between initial acquisition and transmission. The latent period is synonymous with extrinsic incubation period in animal vector biology. For all types of insect transmission, viral determinants of transmissibility have been defined. For the non-circulative viruses, some viruses bind directly to insect stylets or foreguts and other viruses need the assistance of another viral protein(s) that serves as a bridge between the insect structures and the virion [3•,4–6]. For the circulative viruses, the viral capsid proteins and glycoproteins have been identified as viral determinants of insect transmission (reviewed in [7]). Similarly, for the viruses transmitted by soil-dwelling plant–virus vectors (nematodes, fungi, and plasmodiophorids) the viral coat protein(s) is responsible for binding and retention in the vector [8–10]. Despite being transmitted by different mechanisms, the requirement of a viral protein–insect molecular interaction is a consistent theme in transmission by insects and provides a common target for interrupting the transmission process.

Blocking virus transmission with viral capsid proteins and glycoproteins

Viral proteins are required for attachment and/or entry into the insect vector. Therefore, exploiting these proteins for their specific binding affinities to vector tissues is an obvious approach for blocking virus acquisition and transmission. For all the vector-borne plant viruses, a specific viral protein(s) is required for virus transmission. Genomes of plant viruses are quite small, and defining the viral attachment protein(s) (VAP) has been completed for diverse and seemingly intractable virus-vector systems.

Figure 1



The transmission cycle for insect-borne plant viruses. Each step in the transmission process represents a unique opportunity for disruption.

Using this knowledge, recombinant VAP can be used to (1) reduce transmission of viruses by blocking virus binding and subsequent dissemination in the vector and (2) reducing the vector population using the viral protein to deliver toxic cargo to the insect (Table 1).

Exploiting viral proteins to control vectors of circulative viruses

For circulative viruses, the structural proteins of the viral capsid are the determinants of insect vector specificity (reviewed in [11]). The route of virus dissemination has been well-characterized for members of the family *Luteoviridae* and the coat protein (CP) and the readthrough extension of the coat protein are required for transmission. Luteovirids are small icosahedral virions (25–30 nm) that are composed of a major coat protein and a minor protein that has a carboxy-terminal extension termed the readthrough domain (RTD). Initial virus entry occurs in the insect gut and the specific region for entry varies with virus species, occurring in the midgut or hindgut. Several studies have documented that the coat protein is sufficient for delivery of virus into the hemocoel and the RTD is crucial for transmission. It is thought that the salivary glands are the barrier to transmission of particles with mutations in the RTD [12–14]. Knowledge of Pea enation mosaic virus (PEMV) CP binding and movement through the insect gut was used to target a hemocoel-active toxin to aphids [15••]. The authors found that a recombinant CP fused to non-viral toxin peptides could be delivered via transcytosis from the aphid gut to the hemocoel to be aphicidal. The benefit of using this system is that luteovirids are transmitted specifically by aphids. Additionally, the insect gut is not the major barrier to luteovirids entry into the insect and the salivary gland appears to be a more significant barrier to aphid transmission of these viruses. Additionally, the CP-toxin fusion killed non-vector aphids but had no apparent effect on an off-target lepidopteran species, *Heliothis virescens*. Begomoviruses are transmitted in a similar circulative manner by whitefly vectors and the viral CP was shown to bind to whitefly midguts and reduce the amount of virus in whiteflies in feeding experiments [16]. The ability of viral CPs to bind to insect guts and block virus entry indicates that preventing virus entry and delivering toxic peptides may prove to be transmission inhibition-based approaches for other viruses that circulate through the insect body.

An alternative strategy to CP-mediated transport of toxins to aphid vectors has been documented with the use of aphid gut-binding peptides. A bio-panning approach identified a 12 amino acid peptide that bound to pea aphid guts [17]. Interestingly, this peptide, GBP3.1, reduced PEMV abundance in the vector for up to 70 min after acquisition of the peptide. Although the primary amino acid sequence of GBP3.1 was dissimilar to the PEMV CP sequence, structural similarity was

Download English Version:

https://daneshyari.com/en/article/4508240

Download Persian Version:

https://daneshyari.com/article/4508240

Daneshyari.com