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Vector-transmission of plant viruses and constraints imposed by virus–vector interactions

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Because plants are sessile and their cells protected by a cell wall, the contact transmission of plant viruses is very rare. Almost all plant viruses are transmitted by vectors, which can be insects, nematodes, mites or fungi. Although very efficient, this mode of transmission is not trivial and imposes numerous constraints on viruses. In this review we show that these constraints apply at all stages of the transmission process and at all scales, from the molecular to ecological interactions. We discuss several viral adaptations that likely reflect sophisticated means to alleviate these constraints and to maximize transmission, and we point at gaps and future directions in this field of research.

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Introduction

Once they have exploited their host and depleted its resources, viruses need to move on. In some cases, viruses are transmitted vertically to host offspring, but most of the time they spread in the host population(s) by horizontal transmission. In animal viruses, horizontal transmission occurs either 'mechanically', via the contact of the host cells (e.g. mucosa) with an airborne virus, with contaminated objects or parts of other living organisms, or through vectors that are most often blood-feeding arthropods. Because plants are sessile and their cells protected by a cell wall, plant viruses are rarely transmitted by contact in nature [1] and nearly always use vectors. These vectors are mostly insects (aphids, leafhoppers, thrips, beetles . . .), but can also be mites, nematodes or fungi [2]. Particularly prone to viral transmission are aphids and related hemipteran insects, for which a wealth of information is currently available. The mouthparts of these insects are needle-like organs adapted to pierce plant tissues and to suck the content of plant cells or the sap. Viruses taken up by hemipteran vectors are later directly injected into the plant tissue during salivation, allowing them to defeat the first plant defense: its outer cell wall. These insects can also travel long distances (from few meters to hundreds of kilometers in air currents), and often feed on numerous plant species, providing many migration options for the virus they carry [3].

One could think that there is nothing easier for a virus than being carried away by an insect vector. We will show in the following sections that on the contrary, the interaction with the vector is so critical and imposes so many constraints that sophisticated adaptations are selected wherever there is room for improving the transmission efficiency at all possible scales, from molecular interactions to host/vector community networks.

Constraints at the scale of the individual plant–vector interaction

Phytoviruses transmission can be divided in three main categories depending on how they interact with their respective vectors: first, non-circulative transmission where the virus does not penetrate the inner body of its vector and is retained in and released from the anterior alimentary tract, second, circulative non-propagative transmission where the virus circulates inside the vector passing from the gut to the salivary glands without replicating, third, circulative propagative transmission where the virus similarly circulates but also replicates in its vector. For all three categories, the transmission from one plant to the next requires the efficient acquisition of the virus, its retention as infectious units for a sufficient amount of time in the vector's body, and release in the new host. Each of these steps involves specific constraints that are summarized below.

Constraint 1: being spatiotemporally available to the vector

The first constraint that all phytoviruses have to face is to be available for ingestion by their vectors. Perhaps counter intuitively, this step is far from trivial. The infected hosts or cells are not mere bags where the virus would be homogeneously 'swimming', awaiting the streaming up into vectors when they feed into such bags. Instead, viruses accumulate in specific compartments of the host cells, they have to interact with countless host

factors and the viral components have to fulfill multiple functions ensuring primarily replication, cell-to-cell movement and systemic host colonization. The viral proteins interacting with vectors (e.g. capsid proteins and/or and helper components) most often have additional functions, and their activity must be coordinated in a way to ensure a successful encounter with the vectors without jeopardizing the intra-host cycle. Hence, it would not be surprising if viruses could generate specific transmission morphs and/or transiently and reversibly switch to a 'transmission mode'. This possibility has been extensively described for other parasites but has been too long ignored for viruses [4,5]. To date, there is only one study showing that, like other parasites, plant viruses can exhibit specific developmental processes yielding what can be considered as specific transmission morphs.

Cauliflower mosaic virus (CaMV) has evolved a complex but efficient mechanism to ensure its ingestion by the vector. This virus accumulates in each individual plant cell in two types of cytoplasmic inclusions: numerous viral factories where all viral proteins are produced and where the virus replicates, and one single transmission body where the helper component (HC) protein P2, that serves as a molecular bridge between virions and receptors in aphids 'mouthparts, is sequestered. When the plant senses a puncture by an aphid's stylets, the virus hijacks a very early step of the signal transduction pathway and responds within seconds by spreading both P2 from the transmission body [6••] and virions from the viral factories [7] all over the microtubule network of the cell, a phenomenon called 'transmission activation' [8]. The spread of the P2-virion transmissible complexes on the cell cytoskeleton ensures that the aphid will efficiently ingest viral particles wherever it punches the cell (Figure 1a-1). This stunningly fast redistribution of viral products upon aphid puncture is totally reversed within a few minutes, so that the viral within host cycle is barely affected [6••,7].

The fact that different viruses of plants and animals may have developed analogous processes has been previously discussed [8,9]. Surprisingly, however, the fascinating viral capacity to reversibly produce transmission-specific morphs precisely when the vector is present remains totally understudied, and no other cases of transmission activation have been characterized thus far.

Constraint 2: being retained by the vector

After ingestion, viruses have two possible fates: they can go through the gut of the vector and be excreted/lost, or they can be retained as infectious units within the vector. To achieve the latter, plant viruses have developed either capsid or HC proteins with vector-receptor specific binding domains (Figure 1a-2). Correlated with the necessity to evolve such binding systems, the main constraint imposed by the retention process lies in the specificity of the protein–protein interactions implying that each virus can efficiently interact and be retained solely in suitable species belonging to a specific vector family. This constraint of virus-vector specificity has consequences at a higher scale and is further discussed below. In the different transmission modes, plant viruses recognize different types of vector proteins. Non-circulative viruses bind to cuticular proteins of the vector's mouthparts or foregut [10• and Uzest *et al.* on this subject in the same issue of *Current Opinion in Virology*)]. Circulative and circulative-propagative viruses primarily interact with membrane receptors at the surface of the vector's midgut or hindgut epithelial cells, which leads to endocytosis of the viral particles [11–13,14•,15••]. Whether these various protein types (cuticular proteins and membrane receptors) impose different levels or patterns of virus-vector specificity remains to be explored.

Constraint 3: bottlenecks

Population bottlenecks are demographic events temporarily reducing population sizes. They occur at different scales during viral dissemination and, although viral populations are sometimes subjected to very narrow within plant-bottlenecks [16], we here focus on those imposed upon vector transmission (Figure 1a-3). Interestingly, a very small number of infectious units are most often transmitted for both circulative and non-circulative viruses.

Regarding non-circulative viruses transmitted by aphids, small bottleneck size could be somewhat expected because it has been estimated from the size of a CaMV particle relative to that of the area where it is retained in the stylets (the acrostyle) that there is only room for a few hundreds of virions at most (Uzest personal communication, but can also be estimated from Refs. [17,18]). In addition, only a fraction thereof is likely released in susceptible tissues and infects new host cells. Consistently, the bottlenecks for non-circulative viruses have been estimated to range between 0.5 and 3.2 genomes with potato virus Y (PVY — [19]), between 1 and 2 with cucumber mosaic virus (CMV — [20]), and between 1 and 5 with CaMV (our own unpublished data).

The transmission of circulative viruses can take various paths but they all have to cross at least the intestinal barrier through endocytosis/transcytosis, diffuse into the hemocoel and cross the salivary gland cells to finally reach the saliva. It has been shown that populations of arboviruses suffer several successive bottlenecks when progressing in the vector from gut lumen to hemolymph, from hemolymph to salivary gland cells and from salivary gland cells to saliva [21,22]. To our knowledge, no such detailed within-vector study has been done for circulative plant viruses. However, the 'overall vector bottleneck' (i.e. the number of genomes efficiently transmitted from one plant to the next by one insect vector) has been estimated to range between one and two for the