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❋ **Viruses are obligate molecular pathogens. They depend on living host cells for their multiplication, including synthesis of the viral nucleic acids and proteins. The infection cycle of viruses in plants includes three main phases: i) replication, ii) cell to cell movement via plasmodesmata, and iii) long distance movement to different parts of the plant. During all these steps of the infection cycle viruses are challenged by the genetic variability of their hosts, which requires the virus to be adjusted to minor or major differences in virus-host interactions. These adjustments require mutations in the viral genome. Most plant viruses are also dependent on vector organisms for their spread to new host plants. The changes in virus genomes for better adaptability to the host should not compromise vector-transmissibility of progeny viruses. Host adaptation and vector adaptation can therefore be seen as the main forces influencing plant virus evolution.**

EVOLUTION OF PLANT VIRUSES: ADAPTATION TO HOSTS AND VECTORS

THE MAIN STEPS OF THE VIRAL INFECTION CYCLE IN PLANTS

For replication, the virus particle disassembles in the initially infected cell and the viral genome becomes 'activated' (Atabekov et al., 2007). RNA viruses replicate in the cytoplasm. The viral RNA encodes an RNA-dependent RNA polymerase enzyme (replicase) for synthesis of new copies of the viral RNA genome. The first step of replication is hence translation of the replicase directly from the exposed viral genome. However, viruses that have a non-coding RNA strand ("negative strand") or double-stranded RNA genome need to carry the replicase protein incorporated in their virions in order to initiate replication following disassembly (Ueda et al., 1997; Jackson et al., 2005). DNA viruses replicate in the nucleus where they utilize the DNA polymerase of the host for synthesis of new genomes (Rojas et al., 2005).

Cell-to-cell movement of plant viruses takes places via plasmodesmata connecting the cytoplasma of adjacent cells. Movement from cell to cell through cellular membranes is possible in animal tissues but not in plants that have cell walls. Cell-to-cell movement of viruses requires active participation of the plant cells. Viral nucleic acids or virus particles need to be recognized by putative receptors at the openings of plasmodesmata and the plasmodesmata widened for the virus to pass through (Lucas and Lee, 2004). Viruses encode dedicated movement proteins to augment this step of the infection cycle (Lucas, 2006). Successful cell-to-cell movement is also a prerequisite for the viral long distance movement. Viruses must pass through plasmodesmata connecting several types of cells in leaf veins before reaching the sieve elements (SE) in which transport occurs to other parts of the plant (Lough and Lucas, 2006). The plasmodesmata connecting companion cells (CC) and phloem parenchyma cells (PPC) exhibit strict control on movement of macromolecules. This cell boundary may often be to step where systemic infection fails (Rajamäki and Valkonen, 2002). On the other hand, viruses that are injected directly into SE by their vector insects are not able to move out from SE and CC through the CC-PPC boundary and remain phloem-limited (Savenkov and Valkonen, 2001).

Systemic movement of viruses occurs according to the source-sink transition of tissues, during which import of photoassimilates ceases and export is initiated at the transition boundary (Lough and Lucas, 2004). It is commonly thought that long distance movement occurs passively in SE. However, there are data that do not fully fit in this scenario but suggest a more complicated mechanism in which long distance movement takes places via repeated cycles of movement through SE, unloading, replication, and re-entry into the SE (Germundsson and Valkonen, 2006). After systemic movement to distant parts of the plant the virus is unloaded from in veins and invades the cells and tissues by cell-to-cell movement.

SOURCES OF VARIABILITY IN PLANT VIRUSES

The main sources of variability providing the basis for virus evolution are point mutations and recombination (Worobey and Holmes, 1999). Point mutations occur frequently during the replication of RNA viruses whose RNA-dependent RNA polymerase lacks the proof-reading properties characteristic of DNA polymerases (Domingo and Holland, 1997). However, mutations occur also during DNA virus replication (Delatte et al., 2007; Lefeuvre et al., 2007). Thus, plant virus populations possess intrinsic heterogeneity. They consist of a major geno-

type that usually becomes characterized as 'the isolate' and a set of minor variants that are generated by mutation and kept at lower frequency by selection (García-Arenal et al., 2001). A population of nearly-identical sequences, a quasi-species (Domingo and Holland, 1997), is therefore always found in virus-infected plants.

Recombination occurs frequently between virus isolates and sometime even virus species that co-infect plants, as shown in potyviruses (family *Potyviridae*) that have an (+)ssRNA genome (Tomitaka and Ohshima, 2006) and be-gomoviruses (family *Geminiviridae*) whose genome consists of two circular ssDNA molecules (Rojas et al., 2005). These viral taxa contain the largest numbers of plant-infecting viruses (Fauquet et al., 2005).

Clostero- and criniviruses (family *Closteroviridae*) have some of the largest genomes among plant-infecting viruses. They possess a unique ability to integrate foreign genes into their genome (Dolja et al., 2006). A plant heat shock protein homolog encoded by these viruses is involved in viral movement. Recently, a crinivirus was found to encode a Class I dsRNA-specific endonuclease that is homologous to the well-characterized Class I RNase in *Escherichia coli*. The viral RNase is used for suppression of RNA silencing, the fundamental antiviral system in eukaryotic organisms (Kreuze et al., 2005). No other RNA viruses are known to carry an RNase gene. It seems to be recently incorporated into the viral genome (Cuellar et al. 2008). Tenui- and to-spoviruses (family *Bunyaviridae*) snatch ca. 10−20 nucleotides from the 7mG-capped 5′-end of host mRNAs and use them to prime transcription of the viral genome. However, the snatched nucleotides do not become an integral part of progeny viruses (Duijsings et al., 2001).

MEANS AND MECHANISMS FOR ADAPTATION

A member of the quasispecies may become the major genotype if it shows higher compatibility in virus-vector and/or virus-host interactions than other virus variants. However, genetic bottlenecks are common during vector-mediated transmissions of viruses (Moury et al. 2007). They also occur during systemic infection that results in somewhat diverse viral populations in different leaves or parts of the plant (De la Iglesia and Elena 2007; Moury et al. 2007; and refs. cited). When repeated, the genetic bottleneck events may reduce viral fitness because purifying selection does not work efficiently in small populations. Hence, reversions and second-site compensatory mutations that could restore fitness remain rare. The role of genetic bottlenecks in plant virus evolution has gained little attention in experimental research, until recently (De la Iglesia and Elena 2007).

Intramolecular interactions of the viral genome and interactions between the virus-encoded proteins drive a genome-wide coordinated evolution in which mutation at one site may cause complementary mutations in other parts of the genome. Therefore, a mutation that could provide bet-

ter compatibility for interaction with the host may impair intramolecular interactions of the virus and hence be of no benefit. In potyviruses that do not contain genes but express all proteins initially as a single large polyprotein the intramolecular interactions at protein and RNA levels seem particularly important (Rajamaki et al., 2004).

Viruses utilize the cellular transcription, translation and macromolecular transport systems for their infection cycle. Identification of viral, host and vector proteins which interact during the infection cycle should offer means to make function-based molecular predictions about factors that provide selection pressures and drive evolution of plant viruses (Singh et al., 2008). However, the knowledge in this area is still limited. Currently, members of the eukaryotic translation initiation complex, such as eIF4E and eIF(iso)4E, belong to the most widely studied host proteins regarding infection with potyviruses. The viral genome-linked protein (VPg) is studied as the interacting counterpart (Robaglia and Caranta, 2006).

Viruses need to suppress, circumvent or bypass antiviral defence mechanisms of the host plant, which constitutes an important challenge in host adaptation. RNA silencing is one of the most crucial obstacles that the virus needs to cope with. In the past 10 years it has become evident that all viruses encode at least one protein or possess RNA structures whose main purpose is to suppress or help to circumvent RNA silencing (Ding and Voinnet, 2007). The aforementioned incorporation of an RNase gene into the genome of a crinivirus is one example of the evolution for better counter-defence against RNA silencing (Kreuze et al., 2005). Plant cells can detect viral RNA also by a mechanism that resembles the dsRNA-activated protein kinase pathway of animals. However, viruses can suppress this defence mechanism by recruiting a cellular protein to block the pathway (Bilgin et al., 2003). Plants have also evolved to specifically recognize pathogens, including viruses, with the products of resistance (R) genes. Recognition by an R gene elicits a large number and variety of defence responses of which some are detrimental to the virus. Viruses can circumvent recognition via mutation in the corresponding viral aviru-lence gene, unless this turns out to cause a serious loss of fitness for other reasons (Taraporewala and Culver, 1996; Weber and Pfitzner, 1998).

Advanced adaptation of viruses to hosts and vectors can take intriguing and complicated forms. Chlorosis and yellowing of leaves and stunted growth make the infected plants more easily detectable for the vector aphids, but *Potato leaf roll virus* has evolved also additional means to attract the aphids, which it is fully dependent on for transmission. This virus affects the volatiles of potato plants in a manner that makes the plant more attractive to aphids and increases the numbers of aphids landing on the infected plant (Eigen-brode et al., 2002).

Coat protein (CP) is the key player in transmission of viruses by vectors and determines vector-specificity, but still

few vector proteins interacting with the CP are known (Ng and Falk, 2006). However, there are recent break-through findings in mapping genetic loci controlling virus transmission in aphids (Gray et al., 2007) and identification of aphid receptors that interact with other viral proteins required for transmission (Uzest et al., 2007). Analysis of the CP sequences from a broad range of viruses suggests that the virus-vector interaction dominates over the virus-host interactions as a selective force in virus evolution (Chare and Holmes, 2004).

FUTURE PERSPECTIVES

Recent advances in DNA sequencing technology have made it feasible to characterize not only specific genes but partial or full viral genomes of many virus isolates (Ala-Poikela et al. 2006; Dijkeng et al. 2008). The comprehensive amount of sequence data analyzed by phylogenetic and other methods will provide a strong basis for further development of theories on genetic drift and adaptation in plants viruses. The molecular features are also becoming the predominant criteria in viral taxonomy.

However, most of the plant viruses studied so far have been described isolated from cultivated plants in the agricultural or horticultural environments. There is little knowledge of the natural hosts and especially the variability of plant viruses in wild species (Tugume et al. 2008). Therefore, the current picture of plant virus evolution is probably incomplete and will gain from studies on viruses in the wild vegetation.

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**Эволюция вирусов растений:
адаптация к хозяевам и к векторам**

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❈ РЕЗЮМЕ: Вирусы являются облигатными молекулярными патогенами. Для размножения, в том числе синтеза нуклеиновых кислот и белков, вирусу необходимы живые клетки-хозяева. Цикл инфекции вирусов в растениях включает три основных фазы: i) репликация, ii) перемещение из клетки в клетку через плазмодесмы, и iii) дальний транспорт в различные части растений. В ходе этих стадий на инфекционный цикл вирусов оказывает влияние генетическая изменчивость их хозяев, в результате чего вирус должен «приспосабливаться» к незначительным или крупным различиям во взаимодействиях между вирусом и хозяином. Для таких «приспособлений» необходимы мутации в вирусном геноме. Многие вирусы растений также зависят от организмов-переносчиков для заражения новых растений-хозяев. Изменения в вирусном геноме для лучшей «адаптации» к хозяину не должны нарушать способность организма-переносчика передавать вирусное потомство. Адаптацию хозяина и адаптацию организма-переносчика, таким образом, можно рассматривать как основные факторы, оказывающие влияние на эволюцию вирусов растений.

❈ КЛЮЧЕВЫЕ СЛОВА: вирусы растений, эволюция, организмы-переносчики, плазмодесмы, рекомбинация.