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EDITED BY
Rafael Sanjuán,
University of Valencia, Spain

REVIEWED BY
Selma Gago-Zachert,
Martin Luther University of Halle-Wittenberg, Germany
Israel Pagan,
Polytechnic University of Madrid, Spain

*CORRESPONDENCE
Anamarija Butković
anamarija.butkovic@pasteur.fr
Rubén González
ruben.gonzalez@bio.ens.psl.eu

†These authors have contributed equally to this work

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A brief view of factors that affect plant virus evolution

Anamarija Butković[1*†] and Rubén González[2*†]

[1]Archaeal Virology Unit, Institut Pasteur, Paris, France, [2]Institut de Biologie de l'École Normale Supérieure, CNRS, INSERM, Paris, France

Viruses are highly evolvable biological entities capable of wreaking havoc on our society. Therefore, a better understanding of virus evolution is important for two main reasons: (i) it will lead to better management of current diseases and prevention of future ones, and (ii) it will contribute to a better understanding of evolutionary processes and their dynamics. In order to understand the evolution of viruses as a whole, it is necessary to consider different elements that shape virus evolution. In this review, we give a general overview of the most relevant factors that determine the evolution of plant viruses. We will focus on mutation rates, epistasis, robustness, recombination, genome organization, virus-host interactions, transmission, community interactions and abiotic factors. Since this review gives a summarized overview of the most important factors in virus evolution it can be a useful starting material for anyone interested in approaching (plant) virus evolution.

KEYWORDS

virus evolution, plant virus, virus interactions, viral community, virus transmission

Introduction

Viruses can rapidly tune the efficiencies of their replication, intra-host movement and between-host transmission to maximize their fitness (1). The consequent virus adaptation and propagation through host populations impacts the infected hosts' health as well as the ecosystem (2). The study of virus evolution provides useful knowledge about specific evolutionary parameters that can be targeted to disrupt virus populations and prevent or mitigate viral diseases (3). Furthermore, studying viruses allows us to address evolutionary questions in a relatively short period of time (4).

The dimensions of plant virus evolution

The evolution of viruses is shaped by numerous factors (5, 6). In this minireview we will use plant viruses as an exemplar to outline the most important factors described so far. In order to better address this, we have classified each component affecting virus

evolution into different levels (Figure 1). In the following sections, we will summarize each one.

Intrinsic viral factors

Plant viruses can have DNA or RNA genomes of different sizes, architectures, and with different mutation rates.

Sources of genetic variation

The main drivers of genetic variation in viruses are mutation, recombination and reassortment. Generally, single stranded RNA and reverse transcribing (RT) viruses have higher mutation rates (10-6 – 10-4 substitutions per nucleotide per cell infection) compared to double or single stranded DNA viruses (10-8 – 10-6) (7–11). The mutation rate is higher in RNA and RT viruses due to the high-error prone RNA-dependent RNA polymerase and RNA-dependent DNA polymerase (retrotranscriptases, RT) that lack the proof-reading ability or base excision repair (12). Viral mutation rates can be measured as per strand copying or per cell infection estimated rate. These two definitions stem from two different modes of replication: (i) linear replication (mutation rates defined as per strand copying), where multiple copies of the genome are made from the same template or (ii) geometric replication (mutation rates defined as per cell infection), where progeny strands can also be used as templates leading to a geometrical increase in the number of molecules and mutations (8). The actual replication mode of viruses can alternate between the two mechanisms (13). Some viruses can have lower mutation rates and generate less diversity due to their persistent or acute lifestyle (14). Other factors affecting mutation rates in plant viruses are mutagens, host antiviral enzymes and spontaneous chemical reactions (15). RNA viruses tend to have smaller genomes since the probability to accumulate detrimental mutations is smaller (16–19). Although there are exceptions to this rule, for example, closteroviruses that infect plants with genome sizes of up to 20kb (the usual size is 4-12 kb) (20). These large closterovirus genomes can be explained by the acquisition of new genes, such as heat shock protein 70, and the presence of duplicated genes (21). It appears 20kb is the size limit for an RNA virus without a proofreading activity (20). Compared to animal viruses, plant viruses have lower mutation rates and are genetically more stable since they are under weaker immune selection, strong stabilizing selection, strong bottlenecks or different replication modes (21–33). Mutations generate diversity and novelty upon which natural selection can act, thus giving rise to new variants in the virus population (15). A higher mutation rate does not confer greater adaptability, instead high mutation rates of viruses (especially RNA) are in line with the hypothesis in which faster replication is favored at the cost of fidelity (11, 34, 35). This type of replication generates a large proportion of detrimental mutations, and viruses live in an error-threshold or mutation-selection balance. Still, with so many new variants generated, the chances that a beneficial mutation will appear in the population will also increase. This is important in adaptation to new environments and evasion of host defenses (36–39).

A way that viruses can mitigate the detrimental effects of large mutation rates and possibly adapt better to new environments is through epistasis, the interaction between mutations resulting in a lower or higher fitness of the virus. Epistasis seems to be the main driving force behind across-host fitness trade-offs and adaptive processes since it determines the effect of mutations (40). Most studies on epistasis in plant viruses focused on RNA viruses and showed a prevalence of antagonistic epistasis, where two mutations combined have higher fitness than each of the single mutations. This antagonistic epistasis is

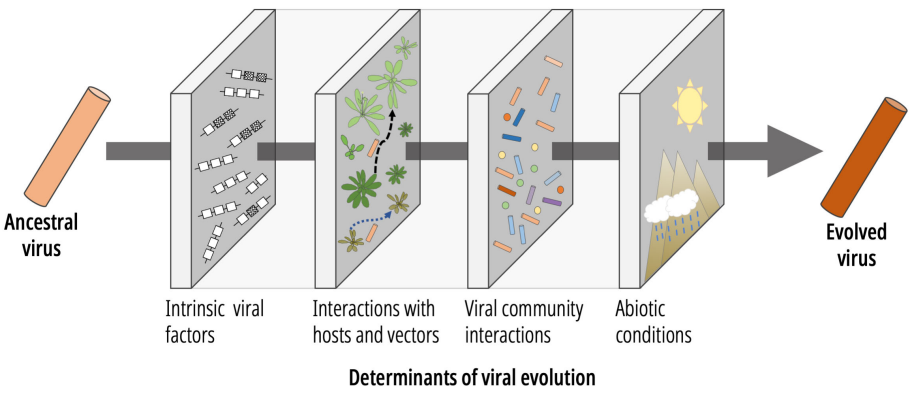

FIGURE 1
Representation of some of the most relevant factors affecting plant virus evolution, from an ancestral to the evolved virus.

highly dependent on the host background (41–45). In RNA viruses the effect of epistasis is large due to their small genome size, presence of secondary structures, overlapping genes and multifunctional proteins where disruption of one function affects many others (38). Another important phenomenon that affects the fitness of viruses in their hosts is antagonistic pleiotropy, where mutations that are beneficial in one host can be detrimental in another (46–48). Because of that, antagonistic pleiotropy can lead to viruses capable of crossing host or species barriers that provoke novel diseases.

Viruses can also buffer the effects of large mutation rates through robustness, or the constancy of the phenotype in the face of heritable perturbations (genetic or epigenetic) or non-heritable perturbations such as external stressors (for example heat, light changes or developmental noise) (49). Robustness acts on the population level and helps viruses deal with genome instability by increasing their tolerance to mutations (50, 51). Due to their nature (high mutational pressures, small genomes that maximize the impact of mutations, and constantly changing environmental conditions) and the fact that they constantly face changing and unpredictable environments (host switches, environmental perturbations, and changes in the physiological state and the immune responses of the host), robustness plays an important role in RNA viruses (52–57). Genetic robustness and environmental robustness have been described in an RNA plant virus (51), confirming the theoretical postulates (50). Generally, plant viruses that are more robust will have more chances of survival and further propagation in unpredictable environments.

Recombination is an event in which two RNA or DNA viruses coinfecting a single host cell exchange parts of their genome (15, 58–63). Recombination is frequent in (+) RNA, ssDNA and reverse transcribing viruses and rare in (-) RNA viruses (37, 62, 64–69). While reassortment is exclusive to segmented RNA and ssDNA viruses, where different viruses exchange genome segments and pack heterologous molecules into a single virion (70–74). These differences in recombination rates can be explained by the evident differences in the biology of (-) RNA viruses compared to (+) RNA, ssDNA and reverse transcribing viruses. In (-) RNA viruses the genomes are quickly bound to the nucleocapsid subunit which limits the recombination events (70). Many theories have emerged about the evolutionary advantages of recombination, explaining it as: (i) a sort of sexual reproduction, (ii) a derivate of the processivity of the RNA polymerase, or (iii) a 'recombination dependent replication' mechanism in geminiviruses, where fragments of the genome are recovered to create recombinant viruses (70, 75, 76). While the most plausible theory regarding the existence of reassortment in segmented RNA viruses and ssDNA (begomoviruses and nanoviruses) describes this process as a by-product of the segmented genome structure (77). Various studies have shown that recombination plays an important role in RNA and ssDNA virus evolution and epidemiology by affecting virulence, pathogenesis, host range, vector transmission, and evasion of host immunity (62, 78). Changes in virus genomes caused by recombination or reassortment can lead to the emergence of new virus diseases and cause significant changes in symptomatology (79–81).

Genome architecture

A considerable proportion of known plant viral species are multipartite and their genome architecture is composed of several segments which are packaged in different viral particles that are independently transmitted (82). A multipartite genome allows viruses to rapidly tune their gene expression by adjusting their relative copy number (83). The adjustments in the proportion of virus segments can regulate the expression of viral genes, thus possibly having a consequential impact on viral fitness and infectivity. This rapid adjustment is particularly useful under environmental changes, as it allows the multipartite virus to have an immediate adaptive response (84). The genome formula of a virus is adaptive and it changes in different hosts (85). Furthermore, even if infection starts with different relative abundances of each segment, their final abundance will evolve to a host-specific segment ratio equilibrium (86). This frequency-dependent selection may happen at the replicative level or during encapsidation. However, it has been shown that not all segments of a multipartite virus need to infect and replicate within the same host cell since viral proteins can be exchanged between host cells, ensuring the availability of all viral proteins needed to assemble new virus particles (87). This phenomenon implies that each fragment may be exposed to slightly different conditions depending on the status of the particular cell they are infecting.

Virus interactions with hosts and vectors

Interactions with hosts

In order to successfully infect a cell of a new host, viruses need to first overcome the host defense response. Plants have evolved mechanisms to restrict virus replication and movement in order to resist infection or tolerate it (88–94), while viruses evolved mechanisms to fight the host defense response (95, 96). This ongoing arms race between plant defense mechanisms and viruses drives co-evolutionary events in both of them. So far, multiple studies described how individuals with different degrees of susceptibility and tolerance to virus infection impact the evolution of the virus, where more permissive hosts select for less virulent and specialized viruses, while more restrictive hosts select for more virulent and generalist pathogens (97). Laboratory experiments show that deficiencies in different host immunity mechanisms determine the rate of evolution of a plant RNA virus, its genetic adaptations, and the degree of

specialization of the evolved virus (98). Virus adaptation to certain host defenses can be highly complex, since virus evolution is influenced by the host's genetic background (48). At the host population level, the defenses vary between individuals. This heterogeneity of plant defense responses (in tolerance or susceptibility to a virus) plays an important role in shaping the patterns of virus evolution (99, 100). For some viruses, the age of the host may influence and lead to an increased virulence of a virus (101). The age of the host can result in viruses with altered infectivity rates, that evolve at different rates and acquire different mutations (102). Notably, even when a virus adapts to a specific new host, the newly acquired adaptive mutations may be hampering the virus' fitness on their original host (103–106).

The evolution of a virus in a host is in part also shaped by the host factors that are necessary for virus replication and transmission. Virus not only needs to adapt to the host to overcome its defenses but also to efficiently hijack the cell, manipulating it for its benefit (107, 108). The host factors required by a virus for successful infection are specific to each host and virus and vary depending on the step of the infection (109).

Transmission

In order to spread to a new host and ensure its survival in a host population, a plant virus needs to be transmitted from the infected plant to a naïve one. This transmission can occur in two different manners: (i) vertically – a way of transmission where plant viruses can reach the often virus-free reproductive tissues of the same plant, accumulate in the seeds and infect the offspring (110, 111), and (ii) horizontally – where a virus is transmitted from an infected plant to a healthy one by physical contact. This mechanical transmission can be caused by the use of agricultural machinery, grazing animals, plants touching due to winds, exposure to infected plant-derived products, etc. (112–114). A key element in virus horizontal transmissions is vectors. Vectors are living organisms that can acquire and disseminate plant viruses by causing mechanical damage to the plant host, disrupting its mechanical barriers and allowing the virus to passively penetrate the plant cuticle and cell wall. In order to be horizontally transmitted by a vector, a virus needs to be efficiently acquired and transmitted by its vector. If a virus is acquired but inefficiently transmitted, the spread of the virus through a host population will be hampered (115). In addition, viruses may evolve different ways to manipulate their plant hosts to generate compounds or morphological phenotypes that attract vectors (116, 117), which will increase the chances of the virus being transmitted to a new host. The nature of the manipulation of the vector's choice and the success of the viral spread is complex (118, 119) and has deep evolutionary consequences (120), especially since the feeding preference of the vector affects the types of hosts a virus encounters, and therefore shapes the evolution of a virus toward a specialist or a generalist pathogen (121).

The transmission mode can also shape virus evolution. Experimental evolution has shown that adaptation to vertical transmission can result in less virulent viruses with a lower viral load (110, 122). Apart from epidemiological traits, the mode of transmission can also affect the nucleotide diversity of virus populations, where an RNA virus had higher nucleotide diversity when vertically transmitted (*via* tubers) in comparation with horizontal transmission (*via* vector or mechanic) (123). The horizontal transmission inflicts a stronger pressure on viruses as this mode of transmission constrains virus population in each of the different steps of the transmission (124) and the interactions with vectors seem to impose a stronger selective pressure on certain viral proteins than the interactions with hosts or other viruses (125). During the horizontal transmission of the virus and its posterior systemic movement through the new host, the virus population goes through bottleneck events. In these bottleneck events only a reduced fraction of the virus population finds a new host, thus limiting the genetic variation of the population and potentially leading to genetic drift (25, 27, 29). Narrow bottlenecks that impose a strong burden on the genetic variation of a virus population are frequent, yet still some viruses may be under wider bottlenecks (126). Soft bottlenecks may even be beneficial for the virus population as they allow the exploration of rugged genotypic spaces with multiple fitness peaks (127).

Viral community interactions

When a virus infects a new host, it might have to interact with other pathogens that are infecting the same host. In this section we will focus on viral interactions with other viruses and the evolutionary consequences of these interactions (128), still, there are complex interactions with other non-viral pathogens that will not be discussed here (129).

Hosts can be co-infected with more than one virus at the same time (130). It has been shown that a considerable proportion of both symptomatic and asymptomatic wild plants are coinfected by multiple virus species or strains (131). In mixed infections viruses interact with each other which can result in neutral interactions, a range of competitive interactions that are detrimental for at least one of the competitors (132), or beneficial interactions. There is an increased interest (and therefore more reported cases) in beneficial interactions, where at least one virus aids another virus' replication and propagation (133).

The structure of a viral community can be host-dependent (134). Therefore, a virus can reach different viral loads and be transmitted with different efficiencies depending on the host that is being infected with multiple viruses (135). This host effect on

co-infection and the way viruses interact with their hosts and their vectors will impact the virus transmission (136, 137). Furthermore, it will influence the fitness of the virus and affect the evolution of all viral populations interacting in the co-infected host. The competition between two viruses can occur within the host but also within the vector. Examples have been found where a virus that was a weaker competitor in the host became the dominant competitor in the vector (138). This suggests that evolutive pressures a virus has to face during competition are context-dependent. Within the conditions that influence viral communities, abiotic environmental factors can also play a key role by modifying the diversity of a virus population and the already complex virus-virus interactions (139, 140).

Abiotic environmental conditions

The environment plays a key role in virus evolution and it affects all other factors mentioned previously. As described in section 1, high mutation rate, recombination, robustness and segmented genomes would be favored in variable environments, since they promote a rapid response of the virus to changing conditions. At the host level, abiotic conditions modify the physiological, hormonal and transcriptional state of the plant (141–144). This change in the plant status affects the plant - virus interaction (145), especially considering that abiotic stressors and viral infection activate the same signaling pathways in the plant and often interfere with one another (146). Therefore, many abiotic conditions (such as drought, high temperatures, elevated salinity, CO2 levels, etc.) have been described as having an effect on the course of plant virus infection. These environmental stressors change the plant - virus interaction by inhibiting plant host defense responses and promoting plant susceptibility to viruses, which in turn aids viral infection and symptom development although reverse effects have also been observed (147–160). Effects of certain abiotic stressors on plant virus infection are becoming increasingly important in the wake of climate change, in particular, temperature increase (161). For example, seasonality (that is directly correlated with temperature) negatively affected an RNA virus accumulation during winter (162). Environmental conditions can also modify virus transmission. Higher light intensity, temperature, CO2 and water levels, changed the level of aphid propagation and seed transmission of plant viruses by affecting plant resistance or aphid behavior (155, 163–173).

In summary, the optimal fitness of a virus is highly dependent on the environment (174). However, environmental impact on virus epidemiology may also vary depending on the host's genetics (175). Notably, under certain stressful environments virus-infected plant hosts can even have higher fitness and survival compared to non-infected plants (176–179). This environmental-induced change in the host-virus interaction can have consequences on the nature of their relationship; for example, switching it from a parasitic relationship to a mutualistic one (180).

Conclusions

The study of the factors that shape the evolution of viruses is extremely important from both the evolutionary and epidemic point of view. In order to unveil these factors and their impacts, plant viruses have proven to be a great practical complex adaptive system used in experimental studies because of (i) their rapid evolutionary rate, (ii) lesser ethical concerns regarding plant hosts, (iii) the lower costs and requirements of plant maintenance compared to animal maintenance, making it feasible to perform experiments on a large number of hosts, and (iv) the safer side of plant virus research compared to animal research. However, there are many aspects characteristic to plant-virus pathosystems one has to keep in mind when doing plant virus evolutionary experiments: the existence of multipartite genomes, reassortment, specific modes of transport through the host, vector transmission, and a host that lacks an adaptive immune system. All in all, the particularities of plant viruses are highly interesting from the evolutionary point of view. The interesting traits of plant viruses as a system to study evolution should further heighten the interest of scientists (and funders).

Author contributions

AB and RG collected and reviewed the published literature, wrote, and revised the manuscript. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

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