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Ecosystem simplification, biodiversity loss and plant virus emergence

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Plant viruses can emerge into crops from wild plant hosts, or conversely from domestic (crop) plants into wild hosts. Changes in ecosystems, including loss of biodiversity and increases in managed croplands, can impact the emergence of plant virus disease. Although data are limited, in general the loss of biodiversity is thought to contribute to disease emergence. More in-depth studies have been done for human viruses, but studies with plant viruses suggest similar patterns, and indicate that simplification of ecosystems through increased human management may increase the emergence of viral diseases in crops.

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Introduction

Plant virus emergence is a complex, incompletely understood, process involving multiple ecological and genetic factors that may be considered as acting sequentially: first, the encounter between the virus and the new host must occur; then the virus must adapt to the new host so that infection is productive enough to ensure between-host transmission; and, last, epidemiological dynamics must change to optimize between-host transmission in the new host population [1•]. Here we are concerned with the first and third steps of emergence. It is widely accepted, although empirical and experimental evidence is scarce, that ecosystem simplification including biodiversity loss will favor both new virus–host encounters, and new epidemiological dynamics resulting in emergence. Linked to this idea is the concept that emergent viruses generally are multi-host pathogens that spillover into a new host population from the original host population in which the pathogen is permanently maintained, that is, from a reservoir host [2], usually a wild host. The focus has been on virus emergence in crops; however, understanding plant virus emergence in crops is limited by a lack of knowledge about the occurrence and dynamics of virus species and strains in wild plant communities [3]. As this gap is being filled it is becoming increasingly clear that crops may also be reservoirs for emergence into wild plants, with consequences potentially as socioeconomically relevant as emergence in crops. Although plant virus-like sequences have also been found in the feces of a number of animals, including humans [4•], these are almost certainly acquired in the diet, and there is no evidence that any of these can replicate, disseminate or cause disease in humans.

Virus infections in crops and wild plants

In general viruses in crop plants have been discovered because they caused symptoms, and surveys for asymptomatic viruses are extremely limited. Viruses that cause disease in some crops can be asymptomatic in others. For example *Peanut stunt virus* causes severe disease in peanuts and beans, but establishes a completely asymptomatic infection in clover [5,6]. *Cucumber mosaic virus* (CMV) can asymptomatically infect some cultivars of rice (Xu P, Roossinck MJ, unpublished data), and even though both the crop and the virus are extremely well-studied, this has not been reported. The result is that the literature on plant viruses in crops is inherently biased, and the real situation in crops is still unknown. Similar scenarios exist for human and other animal hosts and their viruses.

Until recently very few studies looked at viruses in wild plants, with the exception of a few surveys conducted on weeds in areas surrounding crops, and these only tested for known viruses [3]. With the advent of next generation sequencing however, methods have been developed to look for virus-like sequence elements without the bias of only looking for known viruses, and plant viruses are being found in wild plants and a number of other environments [4•,7]. These studies indicate that what is found in wild plants is vastly different from what is known in crop plants [4•]. This does not mean that the viruses are truly vastly different however, because in depth surveys for all viruses in crop plants have not yet been done. Perhaps most interesting in the wild plants is the preponderance of persistent viruses [8] that make up more than half of the sequences that have relatives in GenBank.

Another interesting observation is that although some known acute plant viruses are found in wild plants they are most often asymptomatic, and there is no correlation between the presence of a virus and the presence of symptoms in wild plants [8] (but see [9•]). In addition, many wild plants appear to have multiple infections, including both acute and persistent viruses [10•].

At least 60% of the sequences obtained from virus-enriched samples in plants have no relatives in GenBank, and are currently classified as unknowns. This is common in virus surveys, and in marine virus surveys these numbers are close to 90%. New methods are being developed to try to understand these entities [11], but we are still along way from definitive answers to the question: what viruses are infecting crops and wild plants?

Another question relevant to understanding emergence is if virus infection patterns are the same in crops and in wild plants. Again, data are limited, and consider only acute viruses infecting both crops and wild plants. In one study, the incidence of two begomoviruses with narrow host ranges, *Pepper golden mosaic virus* and *Pepper huasteco yellow vein virus* in wild pepper (*Capsicum annuum* var. *glabrius-culum*) populations in Mexico increased with ecosystem simplification due to habitat anthropization [9•]. The main predictor of begomovirus incidence was the species diversity of the habitat (Figure 1). However, diversity was unrelated to the incidence of the generalist virus CMV [9•]. Patterns of begomovirus and CMV incidence may be related to the dilution effect and amplification effect hypotheses linking host diversity and emergence risk (see below). The yearly variation in begomovirus incidence was higher in cultivated than in wild populations of the host plant [9•,12••], suggesting that ecosystem simplification favors epidemic (vs. endemic) dynamics of virus infection. Large yearly incidence variation in crops, but not in wild hosts, was also reported for CMV, with the

Figure 1

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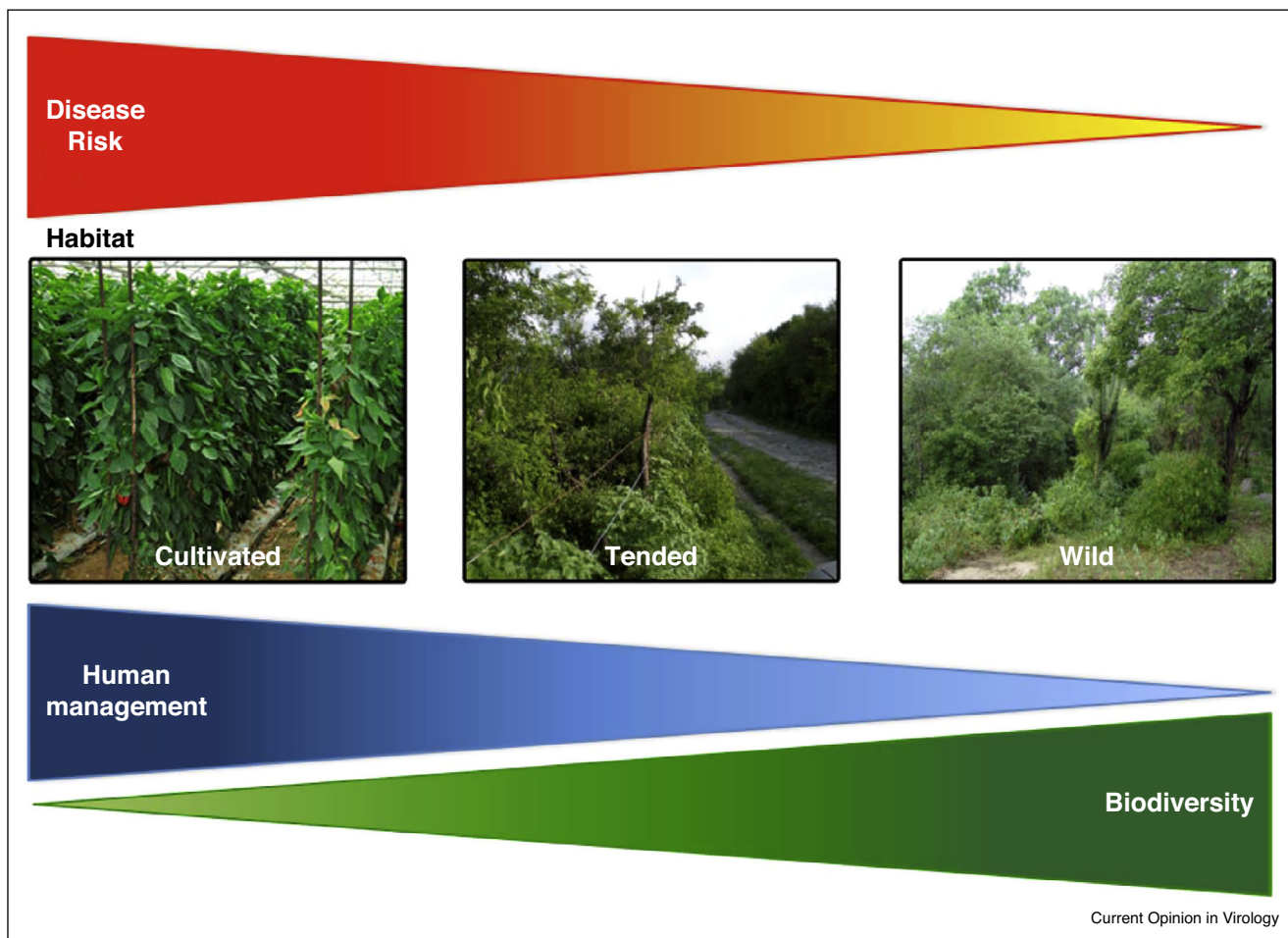
Schematic of the relationship between habitat heterogenenity and disease risk. Disease is greatest in fully cultivated plants, intermediate in wild plants that are tended by humans in anthropic habitats, and lowest in fully wild plants. On the other hand, biodiversity is highest in the habitats of wild plants, and lowest in those of cultivated plants.

extent of temporal variation in wild hosts being positively correlated with ecosystem simplification [13]. Further work is required to determine if these patterns can be generalized to other acute viruses, or to persistent viruses.

The relationships between biodiversity and infection risks

Loss of biodiversity can increase incidence of emerging diseases [14••], and change disease dynamics [15] although with very limited data on the incidence of infectious agents in wild hosts it is difficult to know if this assumption is general [16]. Diversity of available hosts could drive virus divergence by providing expanded niches for viruses when competition is strong [17]. Critically, we have a very limited inventory of the *global pathome*, the worldwide diversity and distribution of potentially pathogenic microbes in wild and domesticated plants and the environment. Some recent large-scale statistical studies have set the stage for identifying *hot spots* for potential emergence events for human pathogens [18–22]. These consider many potential drivers of pathogen biodiversity and emergence including climate factors, human demographics, population changes, land use changes, prior host species and pathogen control. While they disagree on some fundamental points (e.g. the proportion of emergence events due to viruses, the relationship between latitude and propensity for emergence), they highlight the importance of pathogen host range, local host species diversity, land use changes and agriculture. Although similar analyses have not been reported for plant viruses, plant pathologists have postulated for a long time that ecological simplification associated with agriculture favors the appearance of new diseases in crops, as well as their incidence and severity. Specifically, it is considered that reduced biodiversity in agroecosystems, in terms of both plant species richness and the genetic diversity of the focal host, will favor emergence [23,24]. Support for these hypotheses derives mostly from circumstantial or historical evidence. For plant viruses, empirical evidence has been reported from the analysis of *Cereal/Barley yellow dwarf* luteoviruses which have been the object of detailed experimental studies on their effects in wild grassland ecosystems in western North America [25•,26–28,29•], and for the begomovirus infecting wild pepper in Mexico, as described above [9•]. Current declines in biodiversity are at the root of a renewed interest on the relationship between biodiversity and disease emergence [14••]. For multi-host pathogens, heterogeneity in the ability of the pathogen to infect and multiply in different hosts has been shown to either reduce ('dilution effect' [30]) or increase ('amplification effect' [31]) the overall prevalence of infection in diverse host communities. The 'dilution effect' and 'amplification effect' represent extremes of a continuum based on the degree of host specialization by the pathogen. Again, available knowledge is scant here, as information on the host ranges and the degree of host preference of plant viruses derives mostly from assays on collections of experimental hosts, rather than from host range analyses in the field. Theoretical studies have speculated that host heterogeneity should drive selection for pathogen virulence [32] and may drive evolutionary branching in the parasite population [33]. These predictions have been tested for CMV though model analyses with biologically realistic, experimentally determined, model parameters [34].

The role of parasite biodiversity in disease emergence is equivocal, though many studies point to the critical role played by parasite intraspecific diversity in parasite adaptability [35]. Most studies of disease emergence do not directly address plant-virus systems but many of the questions and hypotheses they raise are relevant. For example, pathogens with broad host ranges (generalists) are hypothesized to be more likely to emerge in new species than those with narrow host ranges (specialists), and only a small fraction of parasites capable of moving from animals into humans are likely to spread from person to person [22]. Underlying this hypothesis is the concept of across-host fitness trade-offs (see Elena and Bedhomme in this volume), which should drive evolution toward specialists.

Phylogenetic approaches to study virus emergence, evolution and spread

Little is known about the selective forces that drive viral evolution in natural ecosystems, which contrasts with the more detailed population genetics studies in crop plants that have revealed the importance of mutation rates [36,37], recombination [38–40], genetic drift [1•], and, to a lesser extent, migration in virus evolution. In recent years, the methodology of phylogenetic inference, relying on maximum likelihood, Bayesian and other quantitative frameworks, has made great strides and started to unravel life histories of viruses with a sensitivity not attainable previously [36,41–46]. Approaches based on the coalescent theory and other statistical models have been developed [47–49], allowing investigators to quantify the effects of the individual evolutionary factors, including particularly revealing results on positive *vs.* purifying selection, interhost *vs.* intrahost evolution patterns, etc. (see Ref. [50] for a recent monographic treatment).

Phylogenetics can be used to model patterns of evolution and migration [51]. Various mathematical and statistical approaches to model emergence events have been proposed, providing retrospective understanding of parasites that have previously moved into novel host species [52–54] and tools for rapidly identifying hotspots of molecular evolution [37,55,56]. These methods recently have been used to assess whether environmental heterogeneity determines the evolutionary dynamics of plants viruses, and to reconstruct the spatio-temporal pattern of the colonization of new host populations [12••]. However,

we still lack the data and data-driven models that would allow us to anticipate future movement of parasites among different host species/populations [57].
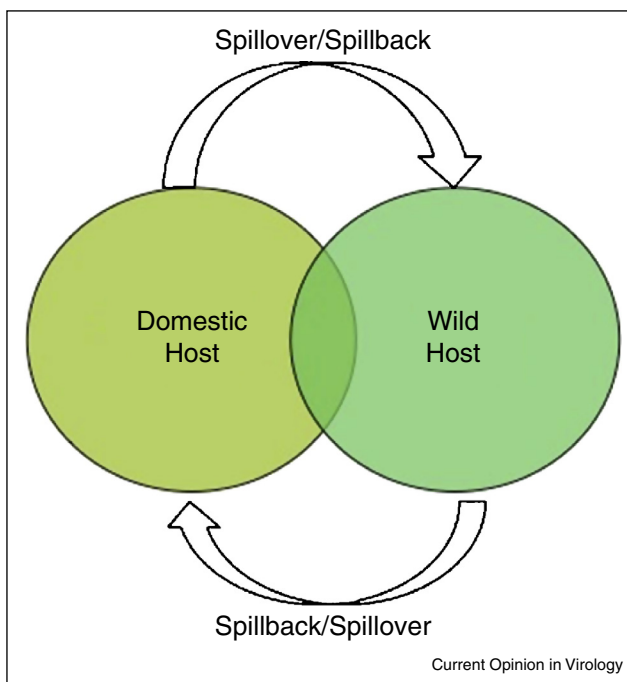
Spillover and spillback

Emergence requires that a virus infects a new host from a reservoir, that is, a natural host that may be either wild or domestic. This first encounter between virus and host is termed spillover. Virus adaptation to the new host results in infection dynamics becoming independent from further spillover [1•], and eventually the virus may spillback from the new host to the natural host (Figure 2).

There have been very few studies on the role of wildlands in plant virus diseases in crops, although considerably more work has been done in human virology. Human pathogenic viruses can emerge from unapparent infections in wildlife via a range of routes, including air-born, vector-born and body fluid-born transmission, as documented for *Human immunodeficiency virus* that apparently emerged numerous times from the closely related *Simian immunodeficiency virus*, endemic in chimpanzees [58], and only rarely pathogenic [59], and more recently for *Severe acute respiratory syndrome virus* that apparently emerged from bats [60,61]. *West Nile virus* has been a spillover

Figure 2

Spillover and spillback between wild and domestic hosts. Spillover occurs when a virus moves from its natural (either a domestic or a wild host) into a new host (either wild or domestic). Spillback occurs when a virus moves from the new host back into the natural host. Natural host here means the source of the virus in this ecosystem, but does not necessarily mean the host where the virus originally evolved (this is often unknown).

human pathogen for some time, but only recently appeared in North America. It is carried by wild birds, and transmitted by mosquitoes [62]. *Influenza virus* has been a human pathogen for a long time, but new strains emerge periodically from wild waterfowl populations, usually via a secondary domesticated host like swine [63–66], and are a frequent source of concern. Disease dynamics between domestic and wild hosts have been studied in *Dengue virus* (mosquito-born), mostly in the context of movement from sylvatic hosts to humans. The movement of Dengue from humans to simian hosts may have occurred but has not been studied beyond phylogenetics of extant viruses [67] and even extensive treatments of the sylvatic cycle of Dengue and other zoonotic viruses do not consider spillback [68], although understanding the sylvatic cycle is recognized as critical for understanding human Dengue dynamics [67]. Often assumptions are made that humans are dead-end hosts for zoonotic viruses [69], but this has not been well tested, and the role of wild host diversity in disease dynamics has been studied for only a few cases [14••,16]. Although the studies relating plant virus emergence to wild host reservoirs are fewer than for human or animal viruses, the classical work on the emergence of *Cacao swollen shoot virus* as an important pathogen of cacao (*Malvaceae* family, native to the Americas) in West Africa from lowland forest and native savanna trees belonging to the *Malvaceae*, and the more recent studies on the emergence of *Maize streak virus* on maize in Africa after recombination of strains infecting local grasses [70•,71] demonstrate the role of spillover in plant virus disease emergence (Table 1). In other instances, evidence strongly suggests emergence from a wild reservoir, as for *Sweet potato feathery mottle virus* [72] or *Pepino mosaic virus* [73] or a wild reservoir is suspected, but has not been identified, as for *African cassava mosaic virus* and *East African cassava mosaic virus* [70•]. As with humans, crops may be epidemiological dead ends. With *Mal de Rio Cuarto virus* that causes the major maize viral disease in Argentina, maize infection is strictly dependent on the migration of viruliferous delphacid vectors from wild grasses or winter grain crops to the young maize plants [74]. In western Australia *Hardenbergia mosaic virus* that naturally infects a native legume can spillover into introduced legumes and cause severe crop losses [75].

The role of domestic hosts as reservoirs for virus emergence in wild hosts has been documented for viruses affecting wildlife, due to the high impact of emerging viruses in conservation programs, particularly for endangered species. Thus, the introduction of Rinderpest virus into Africa with infected cattle from India had a catastrophic impact on native wild African ruminants in the late 19th century [76]. More recent examples from the late 20th century include the outbreaks of rabies that decimated the populations of African wild dogs and Ethiopian wolves, probably from domestic dogs [77] or

Table 1

Examples of plant viruses that have emerged in crops grown in non-native environments

Virus	Geographical emergence	Date first reported	Origin of crop plant	Source of virus	Reference
Cassava mosaic geminiviruses	East Africa	1894	South America	Unknown	[70•]
Cacao swollen shoot virus	West Africa	1900	Central America	Local trees from the Malvaceae	[80]
Maize streak virus	Africa	1928	Central America	Wild native grasses	[81]
Tomato yellow leaf curl virus	Israel	1930	South America	Infects many wild hosts, origin not clear	[82]
Sugarcane yellow leaf virus	Southern United States, Central and South America	1994	Southern Asia	Host unknown, but originated in Columbia	[83]
Pepino mosaic virus	Peru, but has emerged in tomato around the world	1980	South America	Wild native *Solanum* species	[73]
Tomato torroado virus	Spain	1996	South America	Unknown, infects many Solanaceae	[84]
Iris yellow spot virus	Brazil	1981	Worldwide	Unknown, but common in weeds	[85]
Plum pox virus	United States[a]	1999	China	Unknown, may have arrived from Europe on nursery stock	[86]
Wheat mosaic virus[b]	United States	1993	Turkey	Unknown but also found in maize (a native crop)	[87]

[a] Widespread in Europe, emerged in eastern US recently.
[b] Also called High plains virus.

the outbreaks of *Porcine distemper virus* that decimated North Sea and Caspian seal populations, the virus evolving from *Canine distemper virus* after jumping species [76,78]. No similarly documented cases have been reported for plants, but broad-range strains of *Bean yellow mosaic virus* have spilled over from ornamental plants to the native flora of Australia, possibly with negative effects [79]. In Costa Rica, surveys have found acute viruses from local crops in wild plants, but without any associated negative effects. There is also evidence that some of these viruses spillback into domestic crops with attenuation of their pathology (Roossinck, unpublished data). Other data on spillback and its potential affects on disease dynamics are lacking.

Conclusions

As human populations increase, the need for food continuously increases, with concomitant increases in conversion of wild lands for agricultural use. Increased use of wild lands for crops has resulted in significant losses in biodiversity, or ecosystem simplification. Virus disease emergence in crops is a complex process that involves interactions among wild and domestic hosts, insect and other virus vectors, and changes in ecosystems. Although the data is quite limited, evidence to date indicates that loss of biodiversity will lead to increased incidence of virus disease. The need for more studies on the presence of viruses in wild and domestic plants, particularly without regard to symptoms, is apparent, and a thorough understanding of these dynamic processes will not be possible until more data are available. In addition, the role of crop viruses in diseases of wild plants is largely unknown. Spillover from domestic plants to wild plants could contribute to increased simplification of wild ecosystems, further increasing the potential for newly emerging diseases in crops.

Acknowledgements

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- •• of outstanding interest

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