# Antagonistic interactions between plant viruses in mixed infections.

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## Introduction

Plant viruses that share a host typically interact either synergistically or antagonistically. An increase in virus (es) replication in the host plant is the result of a synergistic interaction, which has a facilitative effect on both, or at least one, of the viral partners. When one virus helps another virus spread via vectors, a distinct situation arises. This process, which naturally happens in specific virus complexes, is frequently referred to as "helper reliance." In contrast, only one of the viruses is likely to benefit from an antagonistic form of contact, and its existence and activity reduce the fitness of the second virus. Furthermore, it is anticipated that plants will experience a variety of antagonistic and synergistic virus-virus interactions, leading to more or less predictable biological and epidemiological outcomes. Because of their intricacy and the absence of appropriate laboratory tools, interference interactions have so far been inferred from population-level events rather than laboratory trials, such as changes in plant fitness or the presence of shared vectors [1].

Co-infection and super-infection are two distinct routes of numerous infections. Co-infection occurs when two or more viruses attack the host at the same time or quickly after each other. Different viruses (strains) infect the host at various points during a superinfection. Different circumstances can lead to host infection during spontaneous viral outbreaks. A host typically contracts a single virus at a time during the early stages of an epidemic when prospective hosts are abundant but viral density is low. But as the pandemic spreads, more and more hosts get the virus, increasing the amount of virus in the population. As the epidemic advances, the likelihood that a freshly released viral variation would come into contact with and infect an unoccupied host reduces, and the likelihood that mixed viral infections will develop rises over time. However, the primary virus has a numerical advantage for utilising the finite resources, regardless of any changes in fitness between viral types at the time of invasion. When two homologous viruses infect a susceptible host cell, things change. Since neither variety gets a numerical advantage in this situation, the environmental niche is open to both, and their future fate will primarily be determined by their respective fitnesses [2].

### *Cross-protection*

A past infection with one (protecting) virus inhibits or interferes with a subsequent infection by a homologous virus. This sort of competitive virus-virus interaction is also known as "super-infection exclusion" or "homologous interference." Due to the fact that only related viruses would exhibit the response, this phenomenon was previously used to establish virus connections. Currently, this procedure is much less appealing and practical due to the availability of serological and nucleic acid-based techniques [3].

The two viruses can independently multiply; travel large distances, and migrates from cell to cell. However, the host plant becomes resistant to superinfection with a related challenging virus when infected with the protective virus, or disease signs brought on by the latter are repressed. Cross-protection is similar to the idea of a "vaccine" in both human and veterinary medicine in this regard. Numerous explanations for the occurrence have been put forth. These include, among other things, preventing the challenging virus from disassembling by expressing its coat protein and inducing RNA silence by the protective virus, likely through sequence-specific destruction of the challenging viral RNA. The former idea has the best supporting data. However, there is evidence that the coat protein may prevent the difficult virus from replicating, suggesting that the difficulty of virus uncoating may not be the only method of cross-protection [4].

### *Mutual exclusion*

It is still unclear how mutual exclusion works. It has recently been suggested that a plant may be thought of as a spatially structured environment for plant viruses based on the existing understanding of interactions between viruses and host plants. There is growing proof that closely related viruses are excluded from one another in space. The two viral populations competed with one another during the colonisation of epidermal cells when plants were double-inoculated with cDNA clones of the potyviruses plum pox virus, tobacco vein mottling virus, and clover yellow vein virus expressing green and red fluorescent proteins. Only a small portion of the cells on the border of two adjacent, dissimilarly coloured cell clusters could see both fluorescence signals. Even though they belonged to separate CMV subgroups, two cytomegalovirus strains did not co-infect the same cells in cowpea plants. The location of identical, but expressing yellow vs. cyan fluorescent proteins, viral populations in co-infected Chenopodium quinoa plants was investigated, and similar findings were made utilising Apple latent spherical virus [5].

## References

1. Chiu Mh, Chen Ih, Baulcombe DC, et al. The silencing suppressor P25 of Potato virus X interacts with Argonaute1

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***Received:** 27-Jan-2023, Manuscript No. AAJBP-23-88992; **Editor assigned:** 30-Jan-2023, PreQC No. AAJBP-23-88992(PQ); **Reviewed:** 13-Feb-2023, QC No. AAJBP-23-88992; **Revised:** 20-Feb-2023, Manuscript No. AAJBP-23-88992(R); **Published:** 27-Feb-2023, DOI: 10.35841/aajbp-7.1.133*

***Citation:** Virdich B. Antagonistic interactions between plant viruses in mixed infections. 2023;7(1):133*

and mediates its degradation through the proteasome pathway. Mol Plant Pathol. 2010;11(5):641-9.

2. DaPalma T, Doonan BP, Trager NM, et al. A systematic approach to virus–virus interactions. Virus Res. 2010;149(1):1-9.
3. Folimonova SY, Robertson CJ, Shilts T, et al. Infection with strains of Citrus tristeza virus does not exclude superinfection by other strains of the virus. J Virol. 2010;84(3):1314-25.
4. Froissart R, Doumayrou J, Vuillaume F, et al. The virulence–transmission trade-off in vector-borne plant viruses: a review of (non-) existing studies. Philos Trans R Soc Lond B Biol Sci. 2010;365(1548):1907-18.
5. González-Jara P, Fraile A, Canto T, et al. The multiplicity of infection of a plant virus varies during colonization of its eukaryotic host. J Virol. 2009;83(15):7487-94.

***Citation:*** *Virdich B. Antagonistic interactions between plant viruses in mixed infections. 2023;7(1):133*