infect cucumber cells and spread locally in inoculated leaves, but is unable to cause systemic infection. Addition of CMV RNA-3, but not the other CMV RNAs, to the inoculum complemented for the defect and led to systemic infection by TAV. Complementation was also observed when the RNA-3 carried a mutation that left only the CP gene intact. Different properties of the plasmodesmata that connect mesophyll cells and mesophyll to phloem-parenchyma cells have been shown by dye-injection studies using transgenic tobacco plants that express the TMV movement protein gene ***(30)***. It is possible that the CMV CP is involved in the transport of infectious units from mesophyll cells into phloem cells and that accumulation of CP in transgenic plants interferes with that process.

7. Common Features of CPMR

Accumulation of viral CP in transgenic plants can interfere with different steps of virus infection, depending on the host–virus combination. However, there are several features that are shared in cases in which CP accumulation leads to virus resistance. One common observation of CPMR is limitation of resistance to the virus from which the CP gene is derived and to closely related viruses. This specificity suggests that host defense responses, like those that are involved in the development of nonspecific systemic acquired resistance, do not play a major role in CPMR.

In cases in which virion disassembly is affected in transgenic plants, the range of protection might depend on the ability of the accumulated CP to aggregate with CP subunits of the infecting virus. Such aggregation would lead to stabilization of the virion and prevent replication. The correlation of the degree of resistance of TMV CP-accumulating plants to other tobamoviruses with the degree of amino acid homology between their CPs might be an indication that this is the case.

Classical crossprotection seems sometimes to be caused by inhibition of viral uncoating. For example, infection of tobacco, tomato, or squash plants with the mild strain S of cucumber mosaic virus leads to resistance to CMV-P ***(31)***. Crossprotection can be overcome by infection with CMV-P RNA. The same applies to CPMR to CMV in tobacco protoplasts ***(28)***, and to TMV-infected *Nicotiana sylvestris* plants that can be superinfected with RNA or partially uncoated virions, but not virions of a necrotizing strain of TMV ***(32)***. The presence of high concentrations of protecting virus has been shown to be more efficient in crossprotection between TMV strains than lower concentrations ***(33)***. High concentrations of crossprotecting virus might result in higher concentrations of free CP that can function in stabilizing the virions of the superinfecting virus. The correlation of CP levels in transgenic tobacco with resistance to TMV suggests that both CPMP and crossprotection might in this case work by the same mechanism.