virions. There is only a slight delay in the development of systemic symptoms, compared to nontransgenic control plants. Protoplasts prepared from transgenic plant tissue do not support TMV replication after electroporation with TMV virions, but do so when electroporated with TMV RNA or partially uncoated TMV ***(15)***. Transgenic plants have been generated that express the TMV CP gene from different tissue-specific promoters. These plants accumulated CP either mainly in the upper leaf epidermis, the mesophyll, or the phloem. Only plants that accumulated CP in the upper epidermis, the site of initial infection under laboratory conditions, showed resistance ***(16)***.

Apparently a step prior to virion disassembly is blocked in transgenic tobacco cells. This could be the initial dissociation of some CP molecules from the 5' end of the viral RNA, a process called swelling. Virion disassembly is then completed by binding of ribosomes to the exposed RNA and translation of the TMV replicase proteins from the genomic RNA. It is not yet known whether swelling is caused by the chemical environment in the cytoplasm alone, or whether it occurs at specific sites inside the cell that function as receptors that recognize the 5' end of TMV rods. The latter was suggested by studies in which protoplasts were coelectroporated with isolated CP and TMV ***(17)***. TMV CP can aggregate to different states, depending on the salt and pH conditions. Large aggregates that resembled virions were more efficient in inhibiting TMV replication than small aggregates or monomers, suggesting that the larger CP aggregates blocked an intracellular swelling site. However, it was not possible to monitor the aggregation state of the CP in the protoplasts after electroporation, and the different efficiency of inhibition might have been caused by differences in uptake or stability of the CP. Furthermore, in vitro-generated TMV mutants with altered virion surface structure were unable to overcome resistance of plants accumulating wild-type TMV CP ***(18)***. It is possible that an equilibrium exists between release of CP molecules from the 5' end of the TMV RNA and binding of CP that accumulated in the transgenic cells, resulting in stabilization of the virion.

Inoculation with TMV RNA does not completely overcome CPMR. Local and systemic spread of TMV after inoculation with TMV RNA is delayed in transgenic plants ***(19)***. It was also observed that movement of TMV through transgenic stem sections that were grafted into nontransgenic tobacco plants was delayed, but only if the transgenic section contained a leaf that might have functioned as a sink for infectious units. TMV accumulation in protoplasts prepared from transgenic plants occurs slower than in protoplasts prepared from control plants after electroporation with TMV RNA, except when very high levels of TMV RNA were used. These results suggest that TMV replication in transgenic cells is not only affected at the step of virion disassembly, but also at a later stage. There is no convincing evidence yet for interference with cell-to-