is severe. Symptom response may, but does not necessarily, reflect the degree to which viral functions are restricted.

Essential viral functions in the virus–host interaction include initial establishment of infection, multiplication in the initially infected cell, movement of the virus or its message for replication to adjoining cells, and systemic transport throughout the host. Epidemiological processes necessary for a virus to be acquired from one plant and transmitted to another include compatible virus–vector and vector–host interactions. Processes involved in virus–vector interactions may be controlled by viral genes, but those associated with vector–host interactions, including feeding of the vector on the host and deposition of the virus into the plant, are not controlled by viral genes and should not be affected by any pathogen-derived transgenic resistance.

3.2.2. Elucidation of Viral Function Involved in Resistance

The kind of field resistance expressed may depend on the function repressed. Reduced incidence of disease, for example, may reflect repression of the initial establishment of infection or failure of virus to move from initially infected cells. Resistance to spread of virus from plant to plant could reflect a longer period of time after inoculation of a plant before that plant could itself serve as a source of virus for further spread, or it could reflect a reduced transmission efficiency from plants with resistance to virus replication.

Incidence of disease expression, severity of symptoms caused by disease, and timing of disease development can be determined visually in the field. Beyond these observations, the determination of the specific viral function repressed in disease resistance usually involves virus detection technology. It is difficult to distinguish between true immunity to initial infection and failure of virus to move from initially infected cells, since there may be very few initially infected cells, and the virus accumulated in those cells may be insufficient for detection. In either case, infection cannot be demonstrated after repeated inoculation, and the plants may be described as field-immune according to Cooper and Jones *(4)*. Graft inoculation techniques may be used to distinguish between failure of virus to move from the site of initial infection and its failure to infect new cells after it has moved *(5)*. Varying degrees of resistance to virus multiplication in plants may be demonstrated using quantitative local lesion assays *(6)*. Quantitative serological methods may also be used for this purpose, but only to the extent that the differences in virus antigen accumulation detected serologically reflect real differences in virus multiplication. Very low levels of virus accumulation in plants below those detectable by clinical methods can usually be demonstrated by graft transmission to susceptible plants *(5)*, if the virus moves systemically. Tolerance is easily demonstrated by showing that the plant contains virus, but expresses mild or no symptoms.