scripts of the transgene, rather than the protein itself, were effective. Because high protein levels are usually associated with high transcript levels, correlation of CP levels with the degree of resistance suggests, but does not prove, that CP, rather than RNA, is effective. In some cases, the CP levels decline after subjecting the transgenic plants to elevated culture temperatures; the transcript levels change only little or not at all. If CPMR breaks down at high temperatures and is restored after shifting back to normal temperature, a protein effect is likely. The only sure way to exclude a protein effect is to transform plants with a gene construct that encodes a nontranslatable CP gene, usually a frame-shift mutation, and test the resulting plants for resistance. The characteristics of CPMR have not been studied in detail in all the host–virus combinations to which the approach has been successfully applied. In this chapter, some examples will be used to describe possible ways of interference of CP that accumulates in transgenic plants with virus infection. Mechanisms of resistance in cases in which protection is probably caused by RNA will be described in the following chapter.

2. The Viral Infection Cycle

Although there is a great variety in genome structure, replication strategies, and manners of transmission between plant viruses, most plant virus infections proceed in similar phases. **Figure 1** shows a model of the infection process and the steps that might be affected in transgenic CP-accumulating plants. The initial event is the introduction of one or more virus particles into single cells, either mechanically through wounding or by a vector organism. Inside these cells, the virions disassemble to release the viral genome, which, in most cases, is composed of one or more single-stranded RNA molecules. The viral genes are then transcribed either directly from the genomic RNA or from subgenomic RNAs that are synthesized using complementary replication intermediates as templates. Viral gene products are involved in genome replication, virion formation, and passage of infectious units to adjacent cells, where a new round of replication occurs. Plants that carry hypersensitivity genes respond to local infection with the formation of necrotic lesions, resulting in the arrest of virus spread, and induction of systemic defense mechanisms that prevent or delay subsequent infections. In plants that are susceptible, the infection spreads through the plasmodesmata from cell to cell, until it reaches the vascular tissue and infectious units can enter the phloem. Rapid systemic spread seems to occur mainly through the sieve elements with the assimilate flow. Little is known so far about the mechanisms of entry into, and exit from, the sieve elements.

Plant viruses are transmitted between plants in different ways, most commonly by mechanical means or through insect vectors. Insects usually acquire virus from systemically infected tissue through chewing or directly from the