ally involved inoculation of 10–20 plants from self-fertilized progeny with different concentrations of viral inoculum. Plants were monitored for symptom development and analyzed for presence of viral CP production by Western, ELISA, or probing of dot-blots (*see* Chapters 46 and 47). Sometimes, they were also analyzed for presence of infectious virus by inoculation of extracts derived from protected transgenic plants onto new plants (*see* Chapter 49). Typically, the protection phenotype was delay in symptom development, reduction in symptoms on inoculated leaves, decrease or absence of systemic movement, and reduced virus accumulation. The extent of protection was related to the levels of CP expressed in transgenic plants and the inoculum concentration used in protection experiments.

van Dun et al. ***(7)*** and Powell et al. ***(8)*** showed that transgenic plants expressing translationally defective transcripts of AlMV or TMV CP genes, respectively, were not protected from infection, indicating that protection was caused by the protein rather than the transcript. Thus, the general concensus among researchers at the time was that CP levels were associated with the extent of protection. However, initial experiments on potyviral CP systems indicated that plants with very low or nondetectable levels of CP were protected, as were plants expressing only transcripts ***(9–12)***. This issue of correlations between transgene expression and protection, which is discussed below, indicates that there are multiple mechanisms involved in the protection phenotype that may reflect entry, replication, and movement mechanisms for each virus.

3. Range of Protection

Protection has been demonstrated in 10 different plant hosts transformed with CP or nucleocapsid protein (NCP) genes derived from 14 groups of plant viruses. As indicated in **Table 1**, most examples of protection are conferred against closely related viruses. Generally, the highest level of protection is against the same virus or closely related strains from which the transgene was derived. Barker et al. ***(13)*** determined that combining potato leaf-roll virus (PLRV) CP and host resistance genes in potato gave additive effects on protection against PLRV infection. Stark et al. ***(9)*** first described a broader resistance in plants expressing soybean mosaic virus (SMV) CP that were protected against another potyvirus, tobacco etch virus (TEV). Ling et al. ***(14)***, Namba et al. ***(15)***, and Murry et al. ***(16)*** also reported that transgenic plants expressing potyviral CP genes were protected against heterologous potyviruses. For tobamoviruses, Nejidat and Beachy ***(17)*** reported that protection was effective against different viruses in this group when the CP of the challenge virus exhibited at least 60% homology to the TMV-U1 CP expressed in transgenic tobacco.