Plant viruses can therefore only be controlled by taking a number of phytosanitary measures, including using pesticides, crop rotation, applying crossprotection, or, ultimately, breeding for resistance ***(1,2)***.

Infection of plants with a mild strain of a given virus, which prevents infection by aggressive strains of the virus, has been referred to as classical crossprotection, or premunition. Commercial application of this method, however, has a number of disadvantages. Although the plant tolerates the mild protecting virus, infection still leads to reduced yields. Moreover, the protecting virus can spread to neighboring crops, in which it may cause significant yield losses. In addition, coinfection with another virus might increase symptom development in crop plants. Finally, since viral RNA molecules easily undergo mutations during replication, aggressive strains may arise from the mild protecting strains. It has long been debated whether accumulation of viral CP or replication of viral RNA is responsible for the observed interference with virus replication. The fact that virus mutants, which do not encode CP, are still capable of crossprotecting plants, supports the hypothesis that the replicating viral RNA is involved in crossprotection (*see* **Subheading 5.**) ***(3)***.

Plants find themselves protected from viral infections using passive or active defense mechanisms. Passive defense implies that the plant has factors that suppress, or lack factors that support, virus replication. These passive mechanisms may act on the level of transmission (often linked to insect resistance), multiplication (the plant is immune), transport (a virus causes a subliminal infection), and symptom development (the plant is tolerant). Passive virus resistance can be monogenic, but usually is multigenic and is then referred to as partial or horizontal resistance. Each separate gene contributes to the total level of protection.

In addition to passive defense mechanisms, plants may protect themselves from viral infections using active defense responses. Upon infection, resistant plants produce factors that suppress virus multiplication. The most important example of such modes of resistance is the hypersensitive response (HR). The HR can be explained by the classical gene-for-gene model, as outlined by Flor ***(4)***, in which a product of an avirulence gene of the pathogen (a virus) is recognized by a receptor, e.g., the product of a plant resistance gene. Recognition of the pathogen leads to programmed cell death (apoptosis) at the site of infection, giving rise to the formation of local lesions and to induction of systemic acquired resistance (SAR) ***(4)***. Active resistance is usually monogenic and is often referred to as cultivar or vertical resistance.

In breeding, introgression of horizontal sources of resistance is inconvenient. Since these sources are multigenic, breeding becomes time-consuming and labor-intensive. Moreover, genes contributing to the resistance can be closely linked to undesired agronomic traits. For these reasons, the use of ver-