clearly sidesteps some of the practical and ecological problems involved in using whole viruses for crossprotection.

Finally, the European Community Directive 91/414, aimed at securing harmonization of pesticide registration and availability in the member states, classifies microbiological biocontrol agents as pesticides. For these purposes, attenuated virus strains are classified as biological pesticides and therefore require registration as such. The procedures for registration are expensive and not simple. Until a degree of experience in registering biologicals as pesticides is built up, this regulatory requirement may impose a further block to the introduction of effective cross-protecting viral agents.

5. Possible Mechanisms of Crossprotection

Numerous theories have been advanced and there has been some spirited debate in the literature ***(58,59)***. The situation is probably complicated by the fact that viruses have a number of patterns of interaction within a doubly infected plant. Experiments demonstrating one particular type of interaction do not necessarily exclude the occurrence of another. For example, many systemic virus infections of plants induce the formation of dark green islands of tissue that contain few or no virus particles, but are resistant to challenge inoculation with the same virus ***(60)***. This mechanism of resistance is probably quite separate from crossprotection in virus-containing parts of the leaf, but has confused some of the literature on the subject.

An early theory was that the cross-protecting virus depleted certain metabolites required for virus multiplication or blocked host sites specifically involved in replication. The former explanation would seem unlikely to apply to those viruses, such as potyviruses, which only multiply to very low concentrations in the host. There is a lack of specific evidence for the latter explanation.

Palukaitis and Zaitlin ***(61)*** developed a model in which interference was at the level of the viral RNAs. This involved sequestration of the (–)-strand RNA produced by the challenging virus by the excess progeny positive-sense RNA of the protecting virus.

The strongest evidence is for a central role for the CP of the protected strain in crossprotection, possibly by sequestering the nucleic acid of the challenging strain, or, more likely, by preventing its uncoating ***(62,63)***. Overwhelming support for the involvement of CP in crossprotection is given from the numerous examples of transgenic plants expressing the CP gene for various viruses, which show a protective effect very similar to whole virus crossprotection ***(64)*** (*see* Chapter 3). Earlier reports that crossprotection could be induced by protein-free virus mutants ***(65)*** probably involve a different mechanism of interaction ***(60)***. Further evidence for the operation of parallel non–CP-based mechanisms comes from the demonstration of crossprotection between viroids that lack pro-