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Mechanisms of Resistance

Expression of Coat Protein

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1. Introduction

The expression of viral coat protein (CP) genes in transgenic plants can lead to different phenotypes of resistance ***(1)***. Occasionally, transgenic plants escape infection completely and do not accumulate virus or develop symptoms. In other cases, local and systemic virus accumulation and development of systemic infection proceed at a rate slower than in nontransgenic plants. In transgenic plant lines, the proportion of plants that develop symptoms after inoculation is frequently lower than in control lines. It has also been shown that transgenic plants can become locally infected and accumulate virus in the inoculated leaf, but do not support systemic infection. The different phenotypes of resistance suggest that there is not one common mechanism by which virus infection is affected in transgenic plants, but different steps of virus infection are inhibited in different host–virus combinations.

When coat protein-mediated resistance (CPMR) or coat protein-mediated protection (CPMP) of tobacco to tobacco mosaic virus (TMV) was first demonstrated, the degree of resistance correlated with the level of CP accumulation in the transgenic plants ***(2)***. Those plant lines that accumulated higher levels of CP were more resistant to TMV than those with low CP accumulation. Since then, it has been attempted to apply the approach to a variety of other host–virus combinations, in many cases successfully. In some examples, the degree of resistance was also correlated with CP accumulation; in others, that was not the case. Plant lines with low CP levels, or even with no detectable CP accumulation, were shown to be highly resistant. This indicated that CP gene products can lead to virus resistance in different ways, and that in some cases the tran-

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