A number of observations support this model, with a cytoplasmic RdRp-RNase complex as the major active component. First, many eukaryotic RNA polymerases and RNases contain small nucleic acid fragments. Second, RdRp activity is occasionally present in plant cells. Third, a host-encoded RdRp could be purified from virus-infected plant tissue ***(39,40)***.

Experiments on transgenic expression of pigmentation genes in *Petunia* ***(41)***, on softening enzymes in tomato ***(42)***, and on β-1,3-glucanase in tobacco ***(43)***, revealed that cosuppression of gene expression results from an increase in RNA turnover in the cytoplasm. In addition, the carotenoid biosynthesis in tobacco plants can be modified by inoculation with RNA viral vectors containing the tomato phytoene synthase gene, encoding the key enzyme in the carotenoid biosynthesis, under the transcriptional control of a tobamovirus subgenomic promoter ***(44)***. These experiments clearly demonstrate that cosuppression and RMR reflect identical or similar posttranscriptional and cytoplasmic events. Both virus-resistant and cosuppressing plant cells contain a genetically stable memory for specific RNAs, which are readily degraded upon recognition ***(45)***.

Besides cosuppression, the proposed RMR model can also be adapted to explain antisense inhibition of gene expression. Moreover, classical plant virological phenomena, such as virus crossprotection (**Subheading 2.**), or even mosaic symptoms with light–dark-green islands ***(36)***, might be explained by this model.

An intriguing observation that still needs to be explained is the fact that, in a given transformation experiment, not all different transgenic plants show the virus-resistant or cosuppression phenotype. In tobacco, in our hands, on average 1 out of 10–20 transformants shows the desired phenotype. This frequency, however, differs from crop to crop, and, moreover, it has become clear that it is dependent on many other factors. First, the position of transgene copies on the host chromosomes is of importance. Host sequences flanking the T-DNA inserts have an influence on the levels and patterns of expression of the transgenes on the T-DNA. Second, the number of inserted T-DNA copies and the orientation toward each other also appear to affect the frequency of cosuppression ***(41)***.

From the run-on transcription experiments, it can be deduced that there is a tendency that only highly active copies activate this sequence-specific RNase activity ***(20,24,36,37)***. However, it has been demonstrated that promoterless DNA constructs are also capable of cosuppressing expression of host homologs ***(41)***. These observations suggest that, not the levels of transcription of the transgenes, but the properties of the transgene loci may be responsible for the observed phenotype.

In summary, RMR or cosuppression is caused by overaccumulation of transgenic transcripts or by accumulation of transcripts with aberrant structures.