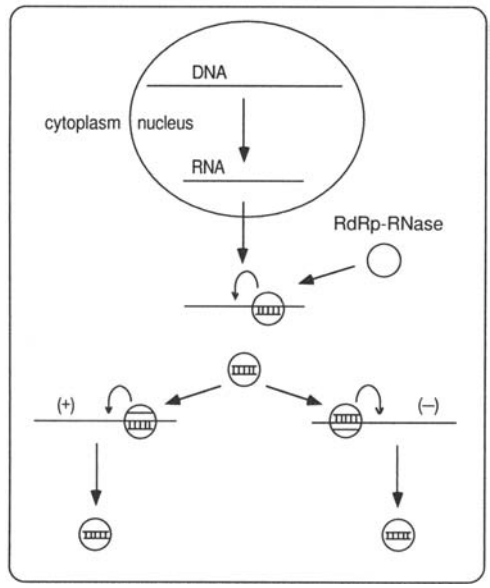

Fig. 1. Model to explain RMR. The transgene is transcribed in the nucleus and the mRNAs are transported to the cytoplasm. Some mRNAs are recognized by an RNA-dependent RNA polymerase (RdRp)–RNase complex, which synthesizes short complementary RNA molecules. The transgenic mRNAs are rapidly degraded and the RdRp–RNase complex uses the short duplex RNA to tag homologous (+) or possibly complementary (–) RNA sequences, and subsequently degrades them.

transgenically produced antisense RNAs were too short in these experiments, or the target viral RNAs, e.g., the minus-sense RNAs, somehow are protected and are not capable of interacting with the transgenic RNA *(38)*.

Taking all data on RMR together, it seems that, at least for most viruses, the polarity of the transgenic transcripts is not important for their capability to confer resistance. This leads to the hypothesis that RMR and antisense inhibition of gene expression might operate via similar or even identical mechanisms.

Figure 1 is a schematic representation of a possible model to explain RMR. In the transgenic virus-resistant plant cells, the transgenic mRNAs are recognized by a cytoplasmic host factor. This factor can be conceived as a RdRp-RNase complex, since it first synthesizes a short complementary RNA, and subsequently degrades the single-stranded parts of the mRNAs. With the help of the remaining short nucleic acid duplex, the complex is able to tag and degrade sequences homologous, or possibly complementary, to the transgenic mRNAs.