Symptom modulation by subviral RNAs associated with turnip crinkle virus

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Abstract

Many plant RNA viruses provide replication and encapsidation functions for one or more subviral RNAs that can modulate the symptoms of the associated helper virus. In this dissertation, I report my studies on symptom modulation in *Arabidopsis thaliana* by subviral RNAs associated with turnip crinkle virus (TCV), a single-stranded, positive-sense, plant RNA virus. ^ Satellite RNA C (satC) is a virulent satRNA that normally intensifies symptoms of wild-type (wt) TCV but can attenuate symptoms if the TCV coat protein (CP) is either replaced with that of cardamine chlorotic fleck carmovirus (TCV-CP_{CCFV}) (Kong *et al.*, 1995) or if TCV contains an alteration in the CP initiation codon (TCV-CPm) (Kong *et al.*, 1997b). I found that TCV-CPm produced reduced level of a CP (10∼20% of wt level) that contained two additional amino acids at its N terminus and did not form virions in infected protoplasts. SatC did not substantially affect the accumulation of TCV-CPm genomic RNA in protoplasts. These results, along with data reported previously (Kong, 1996), led to the conclusion that satC-mediated symptom attenuation of TCV-CPm involves a reduction in virus long-distance movement (Kong *et al.*, 1997b). By characterizing the promoter for the CP mRNA, i.e., the 1.45-kb sgRNA promoter and defining the sequence and structural elements required for promoter activity, I was able to construct TCV variants expressing a level of wt CP similar to TCV-CPm (10∼20% of wt). I found that these mutants also have their symptoms attenuated by satC, indicating that the level of viral CP, not the mutation in the N terminus, is the crucial factor in determining whether satC is going to attenuate or exacerbate symptoms. ^ Another normally virulent subviral RNA, namely defective interfering RNA G (diG) exhibited different symptom modulation of TCV-CPm compared with satC, and the determinants for this differential symptom modulation were previously localized to the T-terminal 100 bases of the subviral RNAs containing six positional differences (Kong *et al.*, 1997a). In this dissertation I report the further characterization of the determinants in these six positions and the possible mechanism underlying the differential symptom modulation by these two subviral RNAs. My results revealed that two positions located in the 3′-terminal stem-loop structures of satC and diG, which also serve as promoters for complementary strand synthesis, are critical for symptom modulation. Furthermore, the hairpin CP binding capacity correlates with the symptom modulation. Several models for symptom modulation by the subviral RNAs associated with TCV are proposed. ^

Subject Area

Molecular biology|Plant pathology

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