Satellite tobacco necrosis virus (STNV) RNA is naturally uncapped at its 5′ end and lacks polyadenylation at its 3′ end. Despite lacking these two hallmarks of eukaryotic mRNAs, STNV-1 RNA is translated very efficiently. A ~130-nucleotide translational enhancer (TED), located 3′ to the termination codon, is necessary for efficient cap-independent translation of STNV-1 RNA. The STNV-1 TED RNA fragment binds to the eukaryotic cap-binding complexes, initiation factor (eIF) 4F and eIF(iso)4F, as measured by nitrocellular bind- ing and fluorescence titration. STNV-1 TED is a potent inhibitor of in vitro translation when added in trans. This inhibition is reversed by the addition of eIF4F or eIF(iso)4F, and the subunits of eIF4F and eIF(iso)4F cross-link to STNV-1 TED, providing additional evidence that these factors interact directly with STNV-1 TED. Deletion mutagenesis of the STNV-1 TED indicates that a minimal region of ~100 nucleotides is necessary to promote cap-independent translation primarily through interaction with the cap binding subunits (eIF4E or eIF(iso)4E) of eIF4F or eIF(iso)4F.

Initiation of protein synthesis in eukaryotic cells is a complicated process requiring 8–10 initiation factors for proper alignment of the initiation codon of messenger RNA on the 40 S ribosome and subsequent joining of the 60 S ribosome (for review, see Refs. 1–6). For eukaryotes the first step in this process is the recognition of the m7GpppX cap structure at the 5′ end of mRNA by eIF4E, the cap-binding protein component of the eIF4F complex. The eIF4G component of eIF4F then acts as a scaffold for the assembly of other initiation factors (for review, see Refs. 7–11). Initiation factors eIF4A, eIF3, and poly(A)-binding protein (PABP) are known to interact with eIF4F during this process (for review, see Refs. 7, 9, and 10). PABP binds the poly(A) tail present at the 3′ end of most cellular mRNAs, and the interaction of PABP with eIF4G implies that the 5′ and 3′ ends of the cellular mRNA are brought into close proximity during the initiation process (for review, see Refs. 9 and 12). This assembly of factors on the mRNA presumably functions in an ATP-dependent unwinding of secondary structure in the 5′-UTR of the mRNA before binding and scanning of the 40 S ribosome to find the correct initiation codon (5).

Plant eIF4F consists of two subunits, a small cap-binding protein, eIF4E (26 kDa), and a large subunit, eIF4G (180 kDa). Higher plants possess an isozyme form of eIF4F, termed eIF(iso)4F (13, 14). eIF(iso)4E, like eIF4F, consists of two subunits, eIF(iso)4E (28 kDa), and a large subunit, eIF(iso)4G (86 kDa). eIF(iso)4F has functions similar to eIF4F in the initiation of translation of plant mRNAs (14). The amino acid sequence of plant eIF(iso)4E is ~50% similar to plant eIF4E and retains all the conserved tryptophan residues found in cap-binding proteins (15, 16). The large subunit, eIF(iso)4G, is significantly smaller than plant eIF4G (86 versus 180 kDa, (15)), sharing ~30–40% similarity with central and C-terminal regions of eIF4G. Rodriguez et al. (17) show that the mRNA for eIF4E is present in all tissues except the specialization zone of roots, and the mRNA for eIF(iso)4E is abundant in developing tissues such as floral organs, meristems, and leaf primordia. Distribution of the eIF4E or eIF(iso)4E proteins within plant cells is not known, although eIF(iso)4G co-localizes with microtubules in situ as measured by immunolocalization (18).

There are alternative forms of initiation of translation that do not have an absolute requirement for the cap structure and initiate translation in a cap-independent manner (19, 20). The most studied of these alternate forms of initiation, internal initiation, is exemplified by the mammalian picornaviruses. Typically, viral RNAs that initiate by internal initiation have long 5′-UTRs and a region of secondary structure that functions as an internal ribosome entry site, and they do not require cap-binding protein for their initiation. Internal ribosome entry sites are found on some cellular mRNAs and are thought to promote initiation of these mRNAs when cap-binding protein is unavailable (for review, see Ref. 21).

Viruses have a number of mechanisms for internal initiation and a variety of initiation factor requirements (22–25), including no initiation factors at all for the cricket paralysis virus internal ribosome entry site (26). Rotoviruses and influenza virus use a viral protein to structurally mimic PABP to recruit eIF4G (27, 28). The picornavirus-like viruses in plants, the potyviruses, lack a cap at their 5′ end but possess a virus-encoded covalently linked protein (VPg) at the 5′ end of the viral RNA (29). It has been shown that the potyvirus VPg or its precursor interacts specifically with eIF(iso)4E or eIF4E (30–33). The genes for naturally occurring potyvirus-resistant plant
varieties have been shown to encode defective forms of eIF(iso)4E or eIF4E (34–36).

Barley yellow dwarf virus (BYDV) RNA, a luteovirus, also lacks a 5' cap structure (37). Wang and Miller (38) previously identified a cap-independent translational enhancer (TE) domain located within the 3'-UTR of BYDV. The TE stimulates translation of viral and heterologous genes 30–100-fold (38), and inactivation of this enhancer region renders BYDV RNA cap-dependent (37, 39). The addition of the BYDV TE domain in trans specifically inhibits in vitro translation and is reversed by the addition of eIF4F (39). The BYDV TE has been shown to interact through base pairing with a stem-loop located in the 5'-UTR (40, 41), serving as a way for the viral RNA to bring the 5'- and 3'-UTRs into contact mimicking the eIF4G-PABP interaction.

Satellite tobacco necrosis virus (STNV), a positive strand RNA necrovirus, requires co-infection of tobacco necrosis virus (TNV) for its replication (42–44). STNV RNA contains a 29-nucleotide 5'-UTR, a 588-nucleotide coding region for its own viral coat protein, and a 622-nucleotide 3'-UTR (45). STNV RNA lacks a 5' cap structure and a poly(A) tail (46). The sequences of three strains of STNV were determined, STNV-1 (45), STNV-2 (47), and STNV-C (48). Sequence comparison shows that STNV-1 and STNV-2 are more closely related to each other than STNV-C. Previous studies from this laboratory for STNV-1 (49) and others for STNV-2 (50) identified a translational enhancer domain (TED) in the 3'-UTR just after the termination codon for the STNV coat protein. Deletion of the TED domain of STNV-1 RNA lowers the translational efficiency and increases the amount of eIF4F necessary for half-maximal translation by about 10-fold (49). Translational efficiency is restored, and the eIF4F requirement is lowered if the TED-deleted STNV RNA is transcribed with a 5' cap group (49). These results suggest that the 3' STNV-1 TED functions in place of a 5' cap group. The STNV-2 TED functions independently in a heterologous mRNA at various positions to stimulate cap-independent translation (51). Maximal cap independence of STNV-1 RNA (49) or STNV-2 RNA (50–52) occurs when both the 5'- and 3'-UTRs are present with the coding region. These results suggest that the tertiary structure of the viral RNA may be important for maximal cap independence. The STNV-2 TED was recently shown by van Lipzig et al. (53) in UV-cross-linking assays to bind 28- and 30-kDa proteins in wheat germ extracts (53). These proteins were not identified but were suggested to correspond to the plant cap-binding proteins eIF4E and eIF(iso)4E (53). Further deletion studies more finely mapped the 5' and 3' boundaries of the STNV-2 TED (54).

Mutation or deletion of the STNV-1 5'-UTR reduces translational efficiency up to 50%; however, unlike the alterations of the 3'-UTR, deletions or mutations in the 5'-UTR do not significantly impact the amount of eIF4F required for initiation of translation (49). These findings suggest that although the 3'-UTR of STNV may be sufficient for recruitment of translation initiation factors and ribosomes, the 5'-UTR is necessary for efficient 5'-3' communication and proper initiation at the initiator AUG.

Several models for ribosomal recruitment by the STNV TED have been proposed, including base pairing to 18 S mRNA, base pairing of the 5' and 3'-UTRs and direct recruitment of a component(s) of the translational apparatus (49, 51–54). Given the profound effects of the STNV-1 TED on eIF4F requirement (49), it seems likely that eIF4F is directly involved in the cap-independent translation of this viral RNA. We provide biochemical evidence for specific interaction of eIF4E and eIF(iso)4E, the cap binding subunits of eIF4F and eIF(iso)4F, respectively, with the 3' TED of STNV-1 RNA.

### Table 1

| Oligonucleotides used for DNA amplification and cloning of STNV-1 TED deletion mutants |
|-----------------------------------------------|
| Oligonucleotides used for cloning or direct amplification of templates for transcription |

| Oligonucleotides | Source |
|------------------|--------|
| 5'-UTR            |        |
| 3'-UTR            |        |
| 5'-3'UTR          |        |
| 5'-UTR and coding region |        |
| 3'-UTR and coding region |        |

**Experimental Procedures**

**Plasmid Construction**—All STNV clones used in this study were derived from a full-length STNV-1 cDNA (L06057 (49)). Sets of appropriate oligonucleotides containing a HindIII site and the T7 promoter in the forward primer and a BamHI site in the reverse primer were used to prepare STNV-1 TED or the truncation mutants by DNA amplification (see Table I). The resulting purified amplification products were then either used directly as transcription templates or cloned into pUC18 digested with HindIII and BamHI. All plasmid constructs were verified by DNA sequencing at the DNA Sequencing Facility (Institute of Cellular and Molecular Biology, University of Texas at Austin). Plasmids were linearized with BamHI before transcription.

The TED truncation mutants were placed back onto the STNV-1 5'-UTR and coding region using NsiI and BamHI sites (see Fig. 1) to replace the wild type TED. The plasmid for these constructs contained nucleotides 1–750 (STNV-1 750) of STNV RNA (49). Briefly, truncation mutants were amplified using the corresponding primers listed in Table I. DNA amplification products were precipitated with ethanol, cleaved with NsiI and BamHI, and gel-purified. Purified mutant DNA was ligated to gel-purified STNV-1 750 that had been previously digested with NsiI and BamHI. The resulting constructs were confirmed by DNA sequencing. Plasmids were linearized with BamHI and utilized as the template for in vitro transcription.

**In Vitro Transcription of Plasmids**—RNA was transcribed from amplified DNA templates or linearized plasmid using the Megascript, MegaScript, or Message Machine T7 Kits (Ambion) according to the manufacturer's instructions. The template for the 125-nucleotide fragment of 18 S was the control template for the Megascript kit (Ambion). The multiple cloning region RNA (~150 nucleotides) was transcribed from amplified DNA made using M13 forward and M13 reverse primers and Bluescript plasmid as the template. Transcription reactions (0.5–1.0-ml reactions) were purified on sterile 20 cc Sephadeq G100 columns pre-equilibrated with sterile buffer (10 mM Tris-HCl, pH 8.0, 1 mM EDTA, 150 mM KC1). Peak fractions were pooled, and the RNA was precipitated with ethanol and resuspended in sterile water. For radiolabeled RNAs, each 20-μl reaction was supplemented with 0.02 μCi of 3000 Ci/mmol [α-32P]UTP or [α-32P]ATP (PerkinElmer Life Sciences). Radiolabeled RNAs averaged ~2 × 105 cpm/μmol. Fluorescent TED was transcribed using coumarin-labeled 5-UTP (PerkinElmer Life Sciences).
Interaction of eIF4F and eIF(iso)4F with STNV 3' UTR

**A**

![Interaction of eIF4F and eIF(iso)4F with STNV 3' UTR](image)

**B**

![binding of STNV-1 TED to initiation factors](image)

### Table II

| Initiation factor | Total TED RNA bound<sup>a</sup> |
|-------------------|---------------------------------|
| n, native protein | 69 ± 6 |
| r, recombinant protein | 79 ± 2 |
| neIF4F | 12 ± 1 |
| reIF4F | 11 ± 1 |
| neIF(iso)4F | 55 ± 4 |
| reIF(iso)4F | 51 ± 5 |
| neIF4E | 3 ± 0.5 |
| reIF(iso)4E | 7 ± 0.5 |
| neIF4A | 5 ± 1 |

<sup>a</sup>The amount of RNA bound was calculated as a percent of the total TED RNA (0.02 pmol, ~2 × 10<sup>10</sup> cpm/pmol) bound by 5 pmol of added protein.

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1. M. L. Allen, K. Ruud, L. Campbell, P. Murphy, and K. S. Browning, manuscript in preparation.
2. E. Gottlieb, personal communication.
3. Dose responses to native or recombinant eIF4F or eIF(iso)4F and their recombinant subunits are shown in Fig. 2. The native and recombinant complexes of eIF4F and eIF(iso)4F bind to STNV-1 TED very strongly, whereas the individual subunits bind the STNV-1 TED poorly even at high levels of protein.

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**FIG. 1.** Schematic of STNV-1 RNA. The TED is shown in gray. Nucleotide sequence of the TED. The NsiI and BamHI sites used for cloning of 5' and 3' mutants are overlined. The termination codon for the STNV-1 coat protein-coding region is underlined; every 10th nucleotide is highlighted. The STNV-1 RNA sequence is from GenBank<sup>33</sup>, accession number L06057 (49).
binding subunits, eIF4E and eIF(iso)4E, to bind to coumarin-labeled STNV-1 TED was measured by fluorescence titration. The fluorescence binding curves shown in Fig. 3 confirm the nitrocellulose binding assay results. The binding affinities of eIF4F or eIF(iso)4F complexes to STNV-1 TED are much higher than for the individual eIF4E or eIF(iso)4E subunits. The estimated $K_d$ values for the complexes in both the nitrocellulose binding and fluorescence assays are compared in Table III. The $K_d$ values for the eIF4F and eIF(iso)4F complexes are in good agreement between the two assays. The binding affinities for eIF4E and eIF(iso)4E estimated from the fluorescence titration are $\sim$10-fold lower than for the complexes. The binding affinity in the nitrocellulose assay was too low to make an estimate. It should also be noted that the binding of eIF4F to the STNV-1 TED appears to be slightly better than for eIF(iso)4F ($\sim$1.5-fold) in both assays.

The specificity of the binding of the complexes and individual subunits to STNV-1 TED was shown by competition with a fragment of unlabeled 18 S rRNA (125 nucleotides) or unlabeled STNV-1 TED in the nitrocellulose binding assay. As shown in Table IV, binding to the native and recombinant complexes of eIF4F was very specific with $\sim$95% inhibition of binding by unlabeled STNV-1 TED. eIF4E binding to STNV-1 TED, but not eIF4G binding, is specifically inhibited by unlabeled STNV-1 TED. Similar results were obtained for eIF(iso)4F and its subunits. These results show that the binding of the STNV-1 TED to eIF4F and eIF(iso)4F is specific and likely mediated through interaction of the eIF4E and eIF(iso)4E subunits with STNV-1 TED.

Translation Inhibition by STNV-1 TED in Trans Is Reversed by eIF4F or eIF(iso)4F—The 125-nucleotide TED of STNV-1 RNA was added in trans to a S30 wheat germ translation assay programmed with AMV RNA 4. AMV RNA 4, a naturally capped mRNA, uses both eIF(iso)4F and eIF4F equally well for initiation of translation in vitro (data not shown). As shown in Fig. 4A, a 120-nucleotide fragment of 18 S rRNA does not inhibit translation, whereas the addition of the STNV-1 TED strongly inhibits translation.

The inhibition of translation by STNV-1 TED was reversed by the addition of eIF4F or eIF(iso)4F as shown in Fig. 4B.
These results strongly suggest that STNV-1 TED sequesters eIF4F and eIF(iso)4F. Interestingly, the reversal curve for eIF(iso)4F was sigmoidal. This sigmoidal shape may reflect the lower affinity of eIF(iso)4F for the STNV-1 TED at low protein concentrations, or it may indicate that an additional component(s) in the wheat germ extract may interact with eIF(iso)4F.

The reversal above 100% is likely due to less than optimal concentrations, or it may indicate that an additional component(s) in the wheat germ extract may interact with eIF(iso)4F.

UV Cross-linking of STNV-1 TED and Immunoprecipitation of Protein-RNA Complexes—Further evidence of direct interaction of STNV-1 TED with eIF4F and eIF(iso)4F was obtained by UV cross-linking of STNV-1 TED in wheat germ extracts followed by immunoprecipitation of the reaction mixtures with rabbit pre-immune, anti-eIF4F, or anti-eIF(iso)4F serum. The results in Fig. 5 show STNV-1 TED specifically cross-links to the subunits of eIF4F and eIF(iso)4F (lanes 1–3). The 120-nucleotide fragment of 18 S RNA does not cross-link to subunits of either complex (lanes 4–6). These results confirm that the proteins in wheat germ extract that UV cross-link to STNV-1 TED reported by van Lipzig et al. (53) are subunits of eIF4F and eIF(iso)4F.

Deletion Mutagenesis Defines the Core Region of the STNV-1 TED—Deletion mutants from the 5′ and 3′ ends of the TED were prepared to delineate the minimum functional length of STNV-1 TED.

Each STNV-1 TED deletion mutant was transcribed and added in trans to a S30 translation assay to measure the ability of the mutant RNA to inhibit translation of capped AMV RNA 4. RNA transcribed from the multiple cloning region (~120 nucleotides) was used as a negative control. As shown in Table V, STNV-1 TED inhibited translation to 60% compared with no inhibition by multiple cloning region RNA. Deletion of nucleotides 619–648 from the 5′ end had very little effect on the ability of the STNV-1 TED to inhibit translation. Deletion up to nucleotide 656 from the 5′ end showed some loss of inhibitory function, and deletion up to nucleotide 659 abrogated the ability of the RNA to inhibit translation. Similarly, deletion from the 3′ end up to nucleotide 741 had very little effect on the ability to inhibit translation. Deletion to nucleotide 731 abrogated the ability of the RNA to inhibit translation. These results suggest that the minimal portion of the STNV-1 TED that functions in the in trans inhibition assay lies between nucleotides 648 and 741.

Cap-independent Translation of Deletion Mutants—The deletion mutants were placed back into the context of STNV-1 RNA (in cis) to determine the effect of the TED deletions on translational activity of the viral RNA. STNV-1 RNA deleted to nucleotide 750 (STNV-1 750) was previously shown to translate in vitro as well as full-length STNV-1 RNA, whereas a deletion to nucleotide 700 (STNV-1 700) abrogated the ability of the RNA to translate (49). Translational activity could be restored to STNV-1 700 if the RNA was transcribed with a m7GpppG cap group (49). The TED of STNV-1 750 was replaced with each

FIG. 4. Inhibition of translation by STNV-1 TED in trans and reversal by cap binding complexes. A, STNV-1 TED (■) or a 125-nucleotide fragment of 18 S RNA (▲) were each added as indicated to a wheat germ S30 translation assay programmed with 2 pmol of capped AMV RNA 4. B, increasing amounts of eIF4F (○) or eIF(iso)4F (●) were added to a wheat germ S30 translation assay programmed with 2 pmol of capped AMV RNA 4 and inhibited with 16 pmol of STNV-1 TED. The values shown are an average of at least three experiments.
with other initiation factors were not observed. Lack of interaction with eIF4E may be due to any number of issues related to expression or folding of the fusion protein produced in this assay (63).

The 5’ and 3’ deletion mutants for STNV-1 TED were cloned into the RNA three-hybrid vector and co-transformed with the pGAD/eIF(iso)4E plasmid into yeast. The results are shown in Table VII. Deletions from the 5’ end up to nucleotide 641 and 3’ deletions to nucleotide 747 were able to interact in the three-hybrid assay. These results suggest that the region important for interaction of STNV-1 TED with the cap-binding protein in vivo lies between nucleotides 641–747. This is similar to the region defined by in vitro assay (nucleotides 649–741), although the in vivo conditions of the three-hybrid assay appear to be more stringent, requiring slightly longer 5’ and 3’ boundaries.

**DISCUSSION**

**STNV-1 TED Binds eIF4F and eIF(iso)4F**—Previous work from this laboratory (49) and others (50–52) has shown that the TED in the 3’-UTR of STNV RNA is required for efficient cap-independent translation. Furthermore, if this region is deleted, efficient translation is restored if the RNA is transcribed with a cap group (49, 51). These results suggest that the TED in the 3’-UTR mimics a 5’ cap group.

In this paper we have shown by nitrocellulose filter binding, fluorescence assays, and UV-cross-linking that eIF4F and eIF(iso)4F bind specifically to the STNV-1 TED. In addition, we show that the cap-binding proteins eIF4E and eIF(iso)4E bind to the STNV-1 TED in the absence of the eIF4G or eIF(iso)4G subunits; however, the binding affinity is more than 10-fold higher when eIF4G or eIF(iso)4G is present. These results suggest that the interaction of the two subunits causes a conformational change in one or both subunits to increase the
binding affinity of the cap binding subunit for the STNV-1 TED. The nitrocellulose and fluorescence binding data also indicate that eIF4F has an ~1.5-fold higher binding affinity than eIF(iso)4F for STNV-1 TED. This is consistent with an ~2-fold difference in the amount of eIF(iso)4F compared with eIF4F required for translation of STNV RNA in vitro (Ref. 14 and data not shown).

The STNV-1 TED specifically inhibits translation of a capped mRNA when added in trans, and the inhibition is reversed by the addition of either eIF4F or eIF(iso)4F complexes (Fig. 4). The apparent 2-fold difference in affinity for STNV-1 TED by eIF4F and eIF(iso)4F also is reflected in Fig. 4B. The eIF(iso)4F reversal curve has a more sigmoidal shape, suggesting that the affinity of the eIF(iso)4F complex may be reduced at low concentrations of protein. Interestingly, wheat germ extracts contain at least 5-fold more eIF(iso)4F than eIF4F (61) so that the difference in affinity of eIF(iso)4F for STNV-1 TED may be offset by its higher concentration in vivo. This difference in affinity may play a role in the virus life cycle. STNV must compete not only with cellular mRNAs but also with its helper virus TNV RNA during infection. TNV also possesses a TE similar to that of BYDV (40) and, thus, may compete with STNV for the cap binding complexes during co-infection.

Additional evidence of direct interaction of the subunits of eIF4F and eIF(iso)4F with STNV-1 TED was obtained by UV cross-linking of STNV-1 TED in wheat germ extracts followed by immunoprecipitation of eIF4F and eIF(iso)4F (Fig. 5). Comparison of the intensity of the RNA-protein complexes obtained in lanes 2 and 3 reflect the ~5-fold higher amount of eIF(iso)4F present in wheat germ extracts (61).

5' and 3' Truncation Analysis of STNV-1 TED—The borders for the 5' and 3' ends of the STNV-1 TED were determined by truncation analysis using three different assay methods, in trans inhibition of in vitro translation, in cis stimulation of in vitro translation, and the in vivo yeast three-hybrid system.

The two in vitro assays show that the 5' border region of the STNV-1 TED is between nucleotides 656 and 659, and the 3' border region is between nucleotides 731 and 741. The yeast three-hybrid in vivo data show that the 5' border region is between nucleotides 641 and 647, and the 3' border region is between nucleotides 740 and 747. The three-hybrid assay requires slightly longer 5' and 3' regions for the protein-RNA interaction, perhaps for stabilization of structure in the more rigorous in vivo environment.

The border regions of the STNV-2 TED were previously determined, and a secondary structure was proposed by van Lipzig et al. (54); a similar structure for STNV-1 TED was also proposed. Fig. 6 shows portions of these proposed structures with the border regions for STNV-1 TED identified in this paper. The border regions for both STNV-1 TED and STNV-2 TED fall in a predicted stem. Both the STNV-1 and STNV-2 TED require a longer 3' border region (Fig. 6, A and B, region III) to function in vivo environments compared with in vitro assays. The 5' border region on the STNV-2 TED was the same for both in vivo and in vitro systems (Fig. 6B, region I), whereas, the STNV-1 TED required longer 5' and 3' regions for in vivo function (Fig. 6A, region I) compared with in vitro (Fig. 6A, region II). These proposed secondary structures do not have any experimental basis, and many alternative structures are possible and should be considered. Attempts at structure mapping STNV-1 TED by either chemical or enzymatic methods were unsuccessful in the presence or absence of cap binding protein or complexes (data not shown). In addition, NMR analysis of the STNV-1 TED indicated that there were only a few G-C base pairs present in solution.5

These results, although not conclusive, suggest that the STNV-1 TED is very dynamic and is able to assume multiple conformations.

**STNV-1 TED Functions as a Cap Mimic in the 3'-UTR**—The ability of STNV RNA to initiate cap-independent translation is dependent upon the presence of the STNV-1 TED in the 3'-UTR. However, the ability of STNV RNA lacking a functional TED to translate can be restored by the addition of a m^7GpppG cap group at the 5' end of the RNA (49, 51). The STNV-1 TED truncation mutants showed similar behavior; those that had lost the ability to support translation in a cap-independent manner regained translational activity by the addition of a 5' cap group. These results and the ability of the cap binding complexes, eIF4F and eIF(iso)4F, to bind to the STNV-1 TED

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4 A. Miller, personal communication.

5 D. Hoffman, personal communication.
how STNV and TNV compete for the translational machinery during co-infection.

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A Novel Interaction of Cap-binding Protein Complexes Eukaryotic Initiation Factor (eIF) 4F and eIF(iso)4F with a Region in the 3′-Untranslated Region of Satellite Tobacco Necrosis Virus

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