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Commentary

Interaction between RNA Structure and Non-Canonical Translation

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DESCRIPTION

Infected cells' translational machinery is used by viruses to create their proteins. As a result, cellular mRNAs and viral mRNAs must compete for ribosomes and translational factors. Eukaryotic viruses use a variety of tactics to prosper. One is to use alternate tools for ribosome recruitment to get around the requirement for m7G-cap. These include Cap-Independent Translational Enhancers (CITEs), which encourage initiation at the uncapped 5′ end even when they are found in 3′ Untranslated Regions (3′ UTRs), and Internal Ribosome Entry Sites (IRESs), which enable translation to be independent of the free 5′ end. A virus can nevertheless interfere with standard ribosomal scanning and start codon selection even if it uses the typical cap-dependent ribosome recruitment. Internal and overlapping open reading frames are frequently created as a result of genome compression pressure.

Specific methods, such as linked termination-reinitiation, 43S sliding, leaky scanning, or shunting, are used to start their translation. By reducing the reliance of viral mRNAs on translation initiation components, deviations from the canonical initiation offer resistance to antiviral systems and cellular stress responses. Viruses can also gain an advantage in a fight for the translational machinery by deactivating specific translational factors or substituting viral analogues for them. Even more advanced intracellular "translation factories" are built by some viruses to spatially separate their protein synthesis sites from cellular antiviral defences and improve the availability of translational components. These virus-specific mechanisms, however, can end up being the virus's fatal flaw. Therefore, a deeper comprehension of the unusual mechanisms behind viral mRNA translation initiation offers crucial knowledge for creating fresh methods of antiviral therapy.

Base pairing between a Shine-Dalgarno (SD) sequence upstream of the start codon and the complementary anti-SD sequence at the 3′ end of 16S rRNA in a 30S ribosomal subunit is the typical scenario for how translation starts in bacteria. It has long been assumed that this manner of initiation, in which the SD plays a significant role at the Ribosome Binding Site (RBS), is the main initiation pathway in bacteria and *archaea*. According to the model, the SD-aSD interaction raises the local concentration of 30S ribosomal subunits close to the start codon, which facilitates the subsequent steps in the development of the initiation complex. The importance of SD length and its distance from the start codon has been shown using systematic approaches in *Escherichia coli*. Although the link is nonlinear and binding affinity by itself does not entirely explain the observed protein expression levels, longer SD elements often support higher expression. While lengthy (8 to 10-nucleotide [nt]) SD sequences limit translation, it is likely because they trap the ribosome on the RBS, weaker SD sequences are more vulnerable to the effects of mRNA degradation and transcription termination. The average SD length and beginning codon spacing for *E. coli* genes are 6.3 nt and 4.4 nt, respectively. The SD sequences from Bacillus subtilis are often longer than those from *E. coli*, a difference that may be explained by the fact that many Gram-positive organisms lack the big ribosomal protein bS1.

The mRNA structure surrounding the RBS has a significant impact on 30S subunit binding and successful initiation. Numerous initiation-targeting gene regulation systems change the local RNA structure surrounding SD sequences in order to function. One of these mechanisms is translational coupling, in which the translation of an upstream open reading frame (uORF) causes the RNA structure enclosing the downstream RBS to be disrupted. The local RNA structure can be influenced by the binding of tiny regulatory RNAs or RNA binding proteins, metabolites (in riboswitches), or temperature rises (in thermosensors), with corresponding effects on initiation. How some mRNAs with RBSs sequestered in stable structures can be translated efficiently needs to be explained by an extra model. According to the "standby binding" theory, 30S subunits initially attach to the single-stranded RNA that surrounds the structured, RBS-containing region. When the nearby RNA structure briefly opens, the bound standby 30S subunit can then successfully compete for RBS capture. More instances of standby binding imply that this initiation style might be common.

During times of cellular stress, noncanonical translation is crucial for controlling gene expression. The lack of correlation

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between transcriptomic study of mRNA levels and proteomic analysis of their expression points to extensive posttranscriptional modulation of mRNA levels. Undoubtedly, the importance of noncanonical translation in cancer, early development, and cellular stress has gone unappreciated. Understanding noncanonical translation's contributions and methods is essential for comprehending how genes are expressed. Overall, translation researchers have labelled the various initiation methods as distinct or special. It is tempting to hypothesise that there may be some shared mechanisms between IRESs and ribosomal shunts, however, given some of the similarities between ribosomal shunting and IRES-mediated translation, such as ribosome landing occurring at a pyrimidine-rich site, base-pairing with the 18S rRNA, and the requirement for eS25. Additionally, certain noncanonical processes recruit or assemble ribosomes using a structure resembling tRNA. The processes by which ribosomes are manipulated by CITES, ribosomal shunting, TISU, IRESs, and m6A to load the mRNA and start translation are mainly unclear. Both similarities and distinctions will emerge as the mechanisms underpinning noncanonical initiation are clarified.