Brief Note

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The Eukaryotic Proteome is Expanded by IRES-Dependent mRNA Translation Initiation

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Until recently, it was thought that mRNA translation in eukaryotic cells began wholly with the attachment of the mRNA 5'-m7G cap-structure to the cap-binding complex E1F4F. Nonetheless, fresh data suggests that eukaryotic cells have evolved different translation initiation techniques from a single mRNA, such as IRES-dependent mRNA translation initiation, which can expand the size and function of a cell proteome.

In light of recent findings, we briefly explore the growing importance of protein isoforms derived from IRES-dependent translation start in mammalian cells. Popular projections only a few years before the Human Genome Project's completion claimed that humans have up to 100,000 genes. However, new data from extensive genome sequencing and analysis has reduced that estimate to a more modest range of 19,000 genes.

In any case, numerous processes affect the content of the cellular proteome, which can increase protein number, notably in eukaryotes, where alternative splicing allows more proteins to be synthesised from a single gene. The discovery of various translation initiation mechanisms from a single mRNA in eukaryotic cells recently generated fresh doubts about the true amount of a particular proteome. Internal Ribosome Entry Site (IRES) elements in their 5'UTRs can attract the ribosome independently of the 5'-cap, which is an alternate route to start viral mRNA translation. Secondary structures in IRES elements are complicated, and IRES-mediated translation is frequently reliant on specific IRES transacting components (ITAFs).

Given that eukaryotic viruses rely on host cell components to carry out their biochemical pathways, the recent identification of IRES elements in mRNAs transcribed in animal cells, plants, and yeasts is somewhat remarkable. Multiple molecular mechanisms regulate the expression of genes having IRES elements in their mRNAs, with IRES mediated translation favoured when 5'capdependent translation is hampered.

As a result, it's now thought that cellular IRES-mediated translation plays a key part in cell destiny decisions under a range of circumstances. *FGF-2* is a member of a vast family of proteins that bind heparin and heparin sulphate and influence the activity of a variety of cell types. Five *FGF2* isoforms with distinct localization and activities are produced by an alternate translational process: A Low Molecular Weight (LMW-18 KDa) and four High Molecular Weight (HMWs-22, 22.5, 24, 34 KDa). All isoforms are controlled by IRES except the 34 KDa HMW, which is controlled by the typical cap-dependent mechanism.

Furthermore, *FGF2* expression has been linked to a variety of human cancers, including melanoma, where it is thought to have a role in the formation and progression of the disease. While LMW *FGF2* expression confers stem-like traits and a proangiogenic profile to melanoma cells, we found that HMWs isoforms are involved in migratory processes and the maintenance of tumour perfusion when endothelial cell-driven angiogenesis is absent by promoting vasculogenic mimicry in a human metastatic melanoma cell line.

This study compares the roles of *FGF2* isoforms in melanoma progression, demonstrating that, although acting in opposite directions, they collaborate in distinct stages of the metastatic cascade, giving melanoma cells a higher malignancy and aggressiveness.

Because eukaryotic mRNA IRES are difficult to discover, study is focused on IRESs that have been experimentally validated to some extent. We can conclude that demonstrating IRES in the 5' UTR experimentally remains a difficult issue, obscuring the true contribution of this translation initiation mechanism to the size and functions of a eukaryotic cell proteome.