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**Title:** "Going against the tide: a novel RNA element protects against viral-induced RNA decay"

**Abstract:** Chemical modifications are critical to guiding mRNA processing as well as the fate of mRNA. N6-methyladenosine (m6A) is one of the most abundant internal RNA modifications of cellular mRNAs. This modification recruits reader proteins that can influence RNA fate by directing their localization and/or stability. Our lab focuses on viruses that co-opt cell pathways to control RNA stability. We work with Kaposi sarcoma-associated herpesvirus (KSHV), a gamma-herpesvirus associated with the development of several cancers. KSHV triggers a massive RNA decay event where 70% of mRNA is degraded by SOX, a virally encoded endonuclease. This process is believed to allow the virus to free up cellular machinery to promote viral gene expression while dampening immune sensing at the same time. Of the 30% of mRNA that escape viral-induced decay, we found a class of mRNA that are protected from degradation by a novel type of RNA element named SRE for “SOX Resistant Element”. To better understand how this SRE mediates protection from SOX, we bioinformatically looked for possible motif in this RNA element and found potential m6A sites. Using MeRIP and mRNA sequencing, we confirmed that SRE-containing genes are m6A-modified in cells, and that mutations within this putative m6A site restores SOX-susceptibility. Recently, we demonstrated that m6A reader YTHDC2 protects the multiple SRE containing transcripts from SOX *in vivo* and we are in the process of characterizing the YTHDC2-mediated method of protection. Taken together, our results reveal a novel mechanism of resistance from virally induced degradation. Characterization of this protection phenotype could provide insights into RNA stability regulation during stress such as during viral infection but also in non-pathogenic settings.