Viruses rely heavily on RNA binding proteins for their success as pathogens. In this presentation, I will first talk about RNA tail modification which impacts viral and cellular gene expression. We found that TENT4 enzymes extend poly(A) tail of mRNAs with ‘mixed tails’ to delay deadenylation and stabilize the RNAs. Hepatitis B virus and human cytomegalovirus hijack this mechanism to efficiently stabilize their own RNAs. In the later part of my presentation, I will discuss our recent work on SARS-CoV-2. To delineate the viral transcriptomic architecture and provide a high-resolution map of SARS-CoV-2, we performed deep sequencing of infected cells. Our data define the canonical transcripts and noncanonical transcripts encoding unknown ORFs. More recently, we have also performed proteomic analyses of the SARS-CoV-2 ribonucleoprotein complex. We identify many proteins that directly interact with viral RNAs and modulate viral growth. Functional investigation of the viral transcripts and host proteins discovered in this study will open new directions to the research efforts to elucidate the life cycle and pathogenicity of SARS-CoV-2.