The SARS-CoV-2 pandemic had made it clear that viruses possess one of the biggest threat not only to the health of infected individuals but also cause serious problems for healthcare systems and economies worldwide. Intriguingly, highly pathogenic RNA viruses are the most important group responsible for zoonotic and epidemic diseases such as COVID-19 and flu, outbreaks of hemorrhagic fever such as yellow fever, Dengue fever, and Ebola disease, or encephalitis such as Japanese encephalitis and Zika fever. Anthropogenic changes of natural ecosystem and constant growth of word population are the factors that increase risk of pathogen transmission from an animal to a human what may develop into a new pandemic. Therefore, only combining molecular and epidemiological knowledge concerning RNA viruses gives promise to control these emergent pathogens. However, we believe that exact molecular characterization of interplay between RNA viruses and host cells is the first step towards preventing future pandemics.

As early as 24 hours after RNA virus infection, up to 25% of all RNA molecules present in host cells are pathogenic viral RNA (vRNA), i.e. viral messenger RNA (vmRNA), viral genomic RNA (vgRNA), and double stranded replication intermediates (dsRNA). To prevent such a takeover of the host metabolism, the innate immune system of the infected organism must detect threat as soon as possible. When considering RNA viruses recognition, activation of timely proper immune response capable of sensing and neutralizing viral genetic material is crucial for cell survival. Viral nucleic acids are one of the strongest pathogen-associated molecular patterns (PAMPs), molecules causing particular immune system reactions. Human cells are armed with a variety of pattern recognition receptors (PRRs) responsible for PAMPs recognition. The main factors responsible for detecting foreign nucleic acids in mammalian cells have already been identified. For activation of any RNA sensor, detecting an abnormal molecular RNA pattern, not present under normal conditions, is obligatory. These patterns may be some chemical modification of RNA, or the absence of such one, specific secondary or tertiary RNA structure, particular sequence, or dsRNA that can derive from viral genome as it is in the case of dsRNA viruses or from annealed complementary RNA strands, which are generated as RNA virus replication intermediates. However, yet there is still very limited understanding of how different epitranscriptomic marks modulate host immune response. Therefore, we will attempt to comprehensively understand how chemical modifications of viral RNA influence its immunogenic potential and stability in infected cells. Moreover, we will study how epitranscriptomic marks deposited on viral RNAs shield transcripts from being recognized by host antiviral factors.