**Description for the general public**

Molecules of ribonucleic acid (RNAs), which are produced by RNA polymerase II possess a characteristic structure at the 5′ end, called a cap. This structure protects the RNA molecules against degradation by exonucleases, which contributes to a greater stability of the transcripts. In humans, the RNA cap consists of an inverted methylguanosine linked to the RNA by a unique triphosphate bond followed by methylation of the first and often the second transcribed nucleotides. These modifications are introduced by enzymes called methyltransferases (MTases). Methylation of the first nucleotide plays a crucial role in synthesis of encoded protein, but importantly, it blocks recognition of the RNA as a non-self molecule. Many viruses evolved their own MTases, which can introduce this modification to viral RNAs. In this manner, viruses can avoid recognition by components of the antiviral immune response. This fact makes viral MTases a potential antiviral drug target. Consequently, to identify inhibitors of viral MTases it is crucial to investigate the role and regulation of human MTases.

In this project we focus on the regulation of a human MTase, which is responsible for cap methylation. Recently, the crystal structure of the catalytic domain of this MTase was solved. It was confirmed that human MTase can methylate capped RNAs *in vitro*, but still, there is no information about its regulation *in vivo*. In preliminary studies we revealed that RNA helicase interacts with this cap MTase. Interestingly, this RNA helicase is involved in splicing and ribosome biogenesis. The question arises, why are these two enzymes, which play a crucial role in completely different processes, interacting with each other? Thus, the aim of postulated project is to carry out a comprehensive characterization of this interaction. In the literature we find instances of regulation of MTase by other proteins. We assume that this helicase could regulate the activity of MTase *in* vivo. We will determine (i) the interacting regions of both proteins, (ii) the impact of RNA helicase on transcript methylation by MTase (iii) and the role of this interaction in the context of RNA secondary structure. Our findings will certainly expand the knowledge of gene expression regulation at this very early stage of mRNA processing which is cap methylation. Moreover, the results may contribute to the development of antiviral drugs that inhibit viral MTases, but do not influence the activity of human MTase that is essential for cell survival.