Living organisms are a phenomenal example of a highly complex and organized system of molecular processes engaging multiple factors of diverse function and activity. A great diversity in proteome despite of a relatively low, 20 000 of genes in human organisms is achieved through a process of alternative splicing. This process is responsible for inclusion or exclusion of different RNA sequences within one transcript, called alternative exons, resulting in production of proteins composed of or deprived of these alternative regions. In consequence, distinct isoforms of the same protein are produced, which differ in properties and function.

As every complex process so as alternative splicing is governed by multiple protein factors which themselves are subjected to alternative splicing. One of these factors which we are focused on is a family of Musceblind-like (MBNL) proteins. MBNL is represented by three paralogs (MBNL1, MBNL2 and MBNL3) which govern fetal-to-adult alternative splicing transition and consequently the production of adult isoforms of proteins. *MBNL1*, which is the object of our attention, is predominantly expressed in the skeletal muscles and heart. Their functional depletion, as one may assume, has severe consequences for body development causing a return to fetal-like pattern of alternative splicing. It occurs in a fairly frequent genetic neuromuscular disorder called myotonic dystrophy (*dystrophia myotonica*, DM).

MBNL1 isoforms are encoded by a few alternative exons which carry crucial information including MBNL1 cellular localization, capability of forming dimers, splicing activity and their stability in cells. Thus far, the splicing mechanism of *MBNL1* transcript remains elusive, even though the protein has an impact on hundreds of targets implicated mostly in skeletal muscle development, muscle regeneration and brain function. Our studies and others show that MBNL1 protein autoregulates its own alternative exons but information on co-regulators engaged in this process is sparse. Thus, in my research I desire to define the splicing mechanism of *MBNL1* alternative exons through identification of these co-regulators and *cis*-acting elements they interact with. Considering the fact of reduced functional pool of MBNLs in DM, I will undertake an approach to elevate the expression level of a stable and more splicing-potent isoform of MBNL1 in cells by splicing modulation of *MBNL1* alternative exons.

I anticipate that new findings will contribute to our deeper understanding of the regulatory mechanisms of splicing factors and alternative splicing process. Apart from a scientific value, anticipated findings will provide new prospects into the area of potential therapeutic strategies against DM.