Until recently, majority of studies were focused on proteins. RNA molecules were seen only as a passive element transferring the genetic information from DNA into protein. Now it is known that a major portion of the genomes is transcribed into RNA molecules that do not code proteins but are involved in almost all aspects of the cell physiology. Although it is single-stranded and composed of only four nucleotides RNA folds into complex secondary and tertiary structures. This "intrinsic" ability forms structural backbone important in intramolecular RNA-RNA contacts and intermolecular interactions with proteins and small ligands. Therefore, RNA structure plays vital functions on many levels of gene expression. As a structural biologists we are interested in the role of the messenger RNA (mRNA) structure during translation initiation process.

Protein translation is the final step of decoding the genetic information. In this process nucleotide sequence of the mRNA molecule is translated into a protein. Translation is divided into three consecutive stages – initiation, elongation and termination. It is believed that translation initiation is the rate-limiting step. Hence, it is highly regulated, allowing rapid adaptation of the cell to changes in physiological conditions without the need of *de novo* mRNA synthesis and transport from the nucleus to the cytoplasm.

Protein synthesis is a highly energy-consuming process. Therefore, under cellular stress translation is downregulated in order to save energy and nutrients. However, to adopt and survive the cells must maintain translation of key regulatory proteins allowing rapid and coordinated response to stress conditions. The most important is p53 protein.

p53 is a transcription factor that controls expression of hundreds of genes. The way the p53 protein acts depends on the type and extent of stress. While cell-cycle arrest allows induction of repair mechanisms and cell survival, apoptosis eliminate damaged and potentially malignant cells. Since p53 is a key regulator of important cellular functions, its level is highly regulated. Until recently it was thought that the main mechanism of stress-dependent p53 protein induction relies on posttranslational modifications increasing its stability. However, recent investigations has revealed, that the level of p53 protein can be effectively and rapidly modulated also during the translation initiation process.

Although p53 protein translation has been characterised functionally the role of p53 mRNA structure is poorly understood. This project focuses on learning RNA structure-dependent, fundamental mechanisms controlling translation initiation of p53 protein. Using various *in vitro* and *in vivo* biochemical approaches we will identify structural motifs in p53 mRNA which regulate p53 translation by formation of RNA-RNA contacts and protein binding sites. The obtained results will contribute to the basic knowledge of regulatory RNAs. Additionally, they can be used for development of effective anti-cancer therapies targeting structured regions of the p53 mRNA.