DESCRIPTION FOR THE GENERAL PUBLIC (IN ENGLISH)

Our genetic material is permanently exposed to factors causing DNA damage. External factors, such as ionizing radiation and chemical mutagens can lead to DNA double strand breaks. A well-functioning mechanism of repairing damage prevents permanent mutation occurrence. One path of the DNA repair is homologous recombination. This process leads to repair DNA double strand breaks without loss of sequence information. Moreover, this mechanism is necessary for genetic recombination during meiosis. In the process of homologous recombination are involved many proteins, with the central role of Rad51 recombinase forming nucleofilaments around site of the damage. However, it is also group of essential mediator proteins that stabilize the resulting structure.

The genetic material in the nucleus is highly condensed. Each 147 bp is wrapped around a histone core to form a nucleosome. The genome is not homogenous - some regions are stronger condensed than others. This leads to differences in the transcription activity of individual regions. The region of reduced availability is the heterochromatin, occurring in *Schizosaccharomyces pombe* in the centromeres, telomeres, rDNA and mating type switching region. Maintaining the stability of the chromatin is necessary to preserve the integrity of the genome. Disruption in the regulation of silencing may lead to illegitimate recombination in particularly sensitive areas such as the series of repetitive sequence within the centromere. For the maintenance of this structure are responsible regulatory proteins, most of them belonging to the family of Snf2. These are the DNA-dependent ATPases, which can remodel chromatin structure.

Genomic organization and DNA repair mechanisms are highly conserved in eukaryotic organisms. This enables the use of simple model organisms such as *Schizosaccharomyces pombe* in studies, allowing to carry out high-speed and low cost experiments, and the results can be applied to human cells.

Proteins involved in the processes described above are often present in the form of complexes. One of them are occurring in *S. pombe* protein Rrp1 and Rrp2. They are paralogs of protein Uls1, SUMO- dependent ubiquitin ligase which is present in Saccharomyces cerevisiae. In previous studies demonstrated the role they play in the process of homologous recombination. In addition, they were included to the family Snf2 DNA-dependent ATPases. Structural similarity and the presence of specific motifs suggests that they may function as regulators of chromatin structure. As a result of earlier work showing interaction with Rad52 protein, a major mediator of homologous recombination.

In the project, it is planned to test the activity of proteins Rrp1 and Rrp2 *in vitro*. They will be tested for their ability to ubiquitination of SUMOylated proteins. Proteins Rrp1 and Rrp2 may undergo post-translational modifications - phosphorylation and SUMOylation. Responsible for this are specific motifs present in their sequence. Analyze for presence of SUMO molecules and phospho residues will be performed. Using endonuclease MN, ability of Rrp1 and Rrp2 for remodeling chromatin structure will be tested.

The information generated by the project will allow for the enlargement of knowledge about the homologous recombination mediators, and provide new data on regulation of the organization of chromatin structure. This will enable a more accurate knowledge of the mechanisms to maintain genomic stability which is very important in the context of many human diseases.