Therapeutics based on siRNA represent a promising hope for the treatment of many congenital and acquired disorders. This method is based on posttranscriptional silencing of pathological gene or set of genes (RNAi process), which are responsible for the actual cause of the disease. Access is therefore based on the assumption of treatment options for the disease at the point of origin of the defect intervention at the molecular level, which is different from the conventional, so-called symptomatic therapy, which focuses only on the treatment or suppression of symptoms.

Despite rapidly increasing understanding of gene function and cause a number of genetic diseases, the expansion of siRNA therapeutics limited the development of efficient and safe transport systems (vectors). In order to ensure efficient transport of siRNA *in vivo* conditions, the vectors must sufficiently reduce the size of the siRNA, protect it against degradation during transport, and release in the cytoplasm of the target cell. For this purpose they were developed sophisticated transport systems based on viral and non-viral origin.

This diploma thesis is focused on the preparation of new transport systems, siRNA-based synthetic hydrophilic polymers, such as non-viral vectors. For *in vitro* testing the effectiveness during transport of siRNA we were prepared two types of polymeric carriers and positively charged polymers (polycations), which electrostatically interact with the negatively charged siRNA to form a polyelectrolyte complex (PEC) and hydrophilic uncharged polymers which contain in their structure reactive groups for covalent binding of the siRNA.

The first part is devoted to the preparation and characterization of polycations and hydrophilic polymers with reactive groups and their ability to form a siRNA polyelectrolyte complexes or covalent conjugates. The second part is devoted to *in vitro* testing of biological activity of prepared PEC siRNA and polymer conjugates of siRNAs in terms of their ability to cause gene silencing GFP produced by GFP-modified HeLa cells via RNAi mechanism. The effectiveness of each siRNA vectors were evaluated as a decrease in fluorescence intensity of GFP.