Abstract

Rhabdomyosarcomas (RMS) are heterogeneous group of sarcomas with mesenchymal origin. These rhabdomyosarcomas in some detail keep their cell differentiation in structure, but they are not able to finish their differentiation cycle. At the moment RMS are the most frequently occurring malignant soft tissue tumors in paediatric pacients.

In recent years, the attention is focused on the research of small single strand molecules, so called microRNA (miRNA). As highly conserved, 18 to 24 nucleotides long, uncoding molecules of RNA, they plays important role as regulators of gene expression. They are controlling both, physiological and pathological processes in organism. Their important role in pathogenesis of different tumor diseases was described at multiple levels. Abnormal levels of miRNA leads to variable, although significant consequences, what indicates miRNA expression also as tumor specific. These miRNA sometimes represents the role of oncogenes, but mostly they occur in the form of tumor suppressors.

In my diploma thesis I am focusing on the family of so called myomiRNAs, muscle-specific microRNAs (miR-133a, miR-133b and miR-206), which regulates cell determination of myogenic precursors, whereas I focus on potentially changed level of their expression in the samples of alveolar, embryonal, or very rare form of pleomorphic rhabdomyosarcoma in the comparison with samples of different types of mesenchymal tumors or muscle tissue without presence of a tumor.

Analysis of the results pointed out statistically significant differences in the levels of expression in individual myomiRs (most significant are miR-206 and miR-133b) by RMS, in comparsion with control samples, or different levels of expression in terms of different subtypes of RMS. The results of my work could have an imapet on candidature of these muscle-specific microRNAs as a potential diagnostic or prognostic markers in pathogenesis of rhabdomyosarcoma.

Key words: rhabdomyosarcoma, microRNA, myomiRs, RQ-RT-PCR, gene expression