SUMMARY

Prostate cancer is the second most common malignancy and is the fifth most common cause of cancer death in the male population worldwide. Recent statistics show that prostate cancer is diagnosed at the late stage in 5.1% of cases, and the 5-year survival rate for these patients is only 30%. It is therefore crucial to understand the mechanisms of migration and invasion of prostate cancer cells and the impact of deregulated factors responsible for modulating the Epithelial-Mesenchymal Transition (EMT) process as the first step in metastasis formation. The ability to rapidly assess the expression levels of factors involved in the EMT process could help improve the diagnosis of prostate cancer, provide a basis for the development of effective targeted therapies, and influence the therapeutic outcome of the late stage of this disease.

The EMT phenomenon is a multi-track program with numerous regulatory factors responsible for specific cellular signaling pathways. These factors interact to form a communication network through which cancer progression occurs. A modern model of cancer cells spread to lymph nodes or distant organs shows that metastatic pathologies are determined by a unique set of altered tissue-specific factors leading to activation of the EMT process and initiation of the invasive-metastatic cascade in prostate cancer patients. In published scientific papers, the nuclear fraction of the MIF protein has been shown to play a role in the progression of prostate cancer. It has been proven that nuclear MIF negatively correlates with the overall expression of β-catenin, so it is likely that the MIF factor affects the abnormal activation of the Wnt/β-catenin signaling pathway, stimulating the translocation of the active form of β-catenin into the cell nucleus to act as an activator of transcription. Moreover, the nuclear fraction of the MIF protein is responsible for participating in the process of lymph node metastasis formation in prostate cancer. It has also been proven that both the SDF-1 factor and its binding receptors CXCR4 and CXCR7 are involved in the formation of lymph node metastasis. There was a decrease in the expression of a stromal fraction of the SDF-1 protein in patients with lymph node metastases, compared to patients without metastases. A similar phenomenon was noted for the

chemokine receptors: the nuclear-cytoplasmic fraction of CXCR4 protein and the cytoplasmic fraction of CXCR7 protein - a decrease in the expression of these proteins was seen in patients with lymph node metastases present. In addition, miR-210-3p overexpression and miR-141-3p deregulation have been shown to be associated with the presence of metastases in prostate cancer patients, in particular distant metastases and bone metastases.

Further scientific progress in epithelial-mesenchymal transdifferentiation in prostate cancer patients can allow the implementation of advanced oncological diagnostics, early disease prognosis, and treatment aimed at stabilizing miRNA and mRNA/protein levels. Using miRNAs or mRNAs/proteins as biomarkers or direct pharmacological targets may change the approach to treating patients with metastatic prostate cancer and bring measurable therapeutic benefits.