

Prolonged inflammation contributes to the development of cancer. Some of the currently used antitumor therapies are based on inhibition of this process. Key factors of inflammatory suppression in the healthy body are proteins that damage the molecules stimulating the immune response.

One of the mentioned proteins is MCPIP1. The role of this protein in the development of cancer has recently been increasingly explored. It turns out that the high level of MCPIP1 protein acts as a natural therapeutic agent for several types of cancer cells. One of them is neuroblastoma - a cancer affecting mainly infants and toddlers.

The MCPIP1 protein has a specific fragment - the PIN domain - which recognizes and destroys the pro-inflammatory molecules. Recent reports show that MCPIP1 may also degrade tumor-promoting molecules (oncogenes). In this way, MCPIP1 inhibits the growth of kidney and breast cancer cells.

Deregulated levels of small regulatory RNA molecules (miRNAs) contributes to cancer progression. It is thought that the MCPIP1 protein can degrade the precursor forms of these molecules. This phenomenon has not yet been well recognized in cancer cells.

In our proposed project, we will answer the question whether MCPIP1 destroys oncogenes in neuroblastoma cells. For molecules degraded by the protein, we will find fragments necessary for recognition by MCPIP1. We will also look at the effect of elevated MCPIP1 levels in neuroblastoma cells on small regulatory RNA molecules. In addition, we will investigate whether precursor forms of miRNA molecules are degraded by MCPIP1.

Determining the role of the PIN domain of MCPIP1 protein in inhibition of neuroblastoma cell growth will allow for a better understanding of the regressive mechanisms of this tumor. In the future, this knowledge may help to develop new therapies based on selective induction of MCPIP1 levels in cancer cells.