

## ABSTRACT

NK cells play a key role in the defence against cells that have been infected by a virus, a protozoan or have undergone malignant transformation. In addition, they also regulate the activity and quantity of other cells of the immune system. Target cells are recognized using their activating and inhibitory receptors, from which they receive activating and inhibitory signals, on which the cytotoxic response of NK cells depends. There is a dynamic balance between the signals that determines the life and death of the target cell. If activation signals prevail, the target cell will be eliminated. If inhibitory signals prevail, then a cytotoxic response will not be triggered.

The NKp30 receptor, which belongs to the immunoglobulin-like receptor superfamily, is an important activating receptor that recognizes a number of ligands, including hemagglutinin of vaccinia and ectromelia virus, human cytomegalovirus pp65 protein, B7-H6, BAG-6, and galectin-3. The extracellular domain of the NKp30 receptor is capable of homooligomerization in solution under certain conditions. The first requirement is the presence of N-glycosylation, the second requirement is the presence of a 15 amino acid long "stalk" domain that connects the ligand binding domain with the transmembrane  $\alpha$ -helix. The aim of this thesis was to assess which of the three available N-glycosylation sites of the NKp30 receptor is responsible for its homooligomerization. Another aim was the biophysical characterization of these homooligomers, including solving their structure.