Abstract:

Natural killer cells (NK cells) are lymphocytes that possess cytotoxic activity against tumour or virally infected cells independent of preceding antigen sensitisation. To kill such cells, they utilise their activating and inhibitory surface receptors that interact with target cell surface molecules. The immune response carried by NK cells depends on the balance of both activating and inhibitory signals.

Human NK cell surface receptor NKR-P1A belongs to the structural family of C-type lectin-like receptors. This receptor interacts with its ligand LLT1, which belongs to the same protein family, with low affinity and high specificity. The NKR-P1A:LLT1 complex formed between NK cell and its target cell inhibits NK cell cytotoxicity, and hence is a part of the regulation of immune response.

This thesis studied the effect of S159A mutation on the stoichiometric state of soluble human NKR-P1A ectodomain in solution. Therefore, a mutant form of NKR-P1A G90-S225 S159A ectodomain was successfully produced in stably transfected human embryonic kidney cells 293 (HEK293S GnTI^{-}). This construct was purified by affinity and size-exclusion chromatography, and analysed by SDS-PAGE and analytical ultracentrifugation. Our results show that the preclusion of *N*-linked glycosylation in the position 157 promotes the dimerization of the NKR-P1A ectodomain in solution.

(The thesis is written in Czech.)

Keywords:

immune system, recombinant expression, HEK293, NKR-P1A, CD161, NK cells, piggyBac