Abstract

Natural killer cells are large granular lymphocytes of innate immunity that are characterized by the ability to kill cancer and virus-damaged cells without prior activation. Cytotoxic functions of NK cells are regulated on the one hand through surface receptors recognizing MHC-I molecules, on the other hand by the presence of a set of activating and inhibitory receptors that are under normal conditions in balance with each other. Therefore, the fate of the target cell depends not only on the expression of MHC-I, but also on the expression of ligands that activate NK cell receptors. One of the activating receptors of NK cells is NKp30. Three specific cellular ligands have been discovered for NKp30: human BCL-2-associated athanogen 6 (BAG-6, also known as BAT3), tumour antigen B7-H6, and the newly discovered ligand galectin-3. All these ligands are often expressed by cancer cells, where BAG-6 and Gal-3 inhibit NK cell functions, which may be a mechanism for tumour escape from the immune system. Therefore, Gal-3 is a new potential drug target that, by inhibiting Gal-3, can help the immune system defend itself against malignantly transformed cells.

This bachelor's thesis includes the verification of the effect of the Cys^{173} – Ser^{173} mutation in the carbohydrate recognition domain of galectin-3 on the binding of the NKp30 receptor using gel permeation chromatography. It also deals with the preparation of galectin-3 with a C-terminal polyhistidine tag, which will be further used in the study of the interaction of galectin-3 with newly synthesized organic Gal-3 inhibitors using microscale thermophoresis. The exploitation of the results of this work may contribute to the unravelling of the structure of Gal-3 binding to the NKp30 receptor and the design of new effective drugs with therapeutic potential.

Key words

Galectin-3, NKp30, NK cells, recombinant protein expression, *E. coli*, liquid chromatography