DCL-1 (CD302) is a type I transmembrane C-type lectin receptor, which is expressed on monocytes, macrophages, granulocytes and dendritic cells. However, its extracellular domain lacks the amino acids motives essential for carbohydrate binding in the presence of calcium ions, suggesting that it does not have the classic binding capacity found in other C-type lectin receptors such as the mannose receptor. No exogenous or endogenous ligands have been identified yet, though. Due to internal colocalization with F-actin we can assume, that this unconventional lectin receptor plays a role not only in endocytosis and phagocytosis but also in the cell adhesion and migration. The receptor DCL-1 was first identified as a genetic fusion partner of human DEC-205 multilectin receptor in Hodgkin's lymphoma cell lines.

The experimental part of this thesis deals with the characterization of disulfide bonds and data acquisition for validation of DCL-1 crystal structure. First the production and refolding conditions were optimized to obtain the highest amount of DCL-1 protein, precisely its extracellular domain.

These optimal conditions were used to prepare the protein for in-gel digestion using specific endopeptidases in the presence of cystamine followed by LC-MS analysis. DCL-1 disulfide bonds were determined by comparing of LC-MS analysis data with software generated theoretical peptide masses.

Chemical cross-linking experiments in combination with LC-MS analysis were carried out to map the tertiary structure of the DCL-1 receptor. For this purpose, two homobifunctional (DSG and DSS) and one heterobifunctional (EDC) reagents were used. The model of DCL-1 crystal structure was validated by the obtained protein cross-links.