Syncytin-1 is an endogenous retroviral envelope glycoprotein specifically expressed in human placenta, where the protein was adopted for its physiological function. After interaction with specific receptors, transmembrane proteins ASCT1 and ASCT2, Syncytin-1 initiates cell-cell fusion leading to formation of multinucleated syncytiotrophoblast, which is essential for feto-maternal nutrients exchange. In this diploma thesis a new cell-cell fusion quantification assay was implemented for characterisation of Syncytin-1 fusion determinants. The assay uses Syncytin-1 and ASCT2 expressed separately with fragments of luciferase in heterologous cell-culture system. The assay enables to specifically quantify cell-cell fusions based on activity of reconstituted luciferase reporter. This study discovered new facts about the role of intracytoplasmic tail of Syncytin-1 in the process of the cell-cell fusion. This specific part of protein contains a tandem motif sensitive to changes in amino acid sequence that led to loss of fusogenic potential of Syncytin-1. It was further confirmed, that the protein Suppressyn works as an inhibitor of cell-cell fusions initiated by Syncytin-1. Suppressyn however does not bind to receptors of Syncytin-1 and the mechanism of its inhibition remains unsolved. Finally, it was demonstrated that the C-region of extracellular domain 2 of the ASCT2 protein is not the binding site of Syncytin-1. Introduction of several major deletions in the C-region did not abolish the ASCT2 and Syncytin-1 interaction.