The activation of bone marrow-derived mast cells (BMMCs) induces a number of cell processes such as degranulation, proliferation and cytoskeleton rearrangements. Although microtubules are important in these processes, molecular mechanisms that control changes in microtubule organisation during cell activation are unknown. Activation of BMMCs can be achieved in several ways. Under physiological conditions, the aggregation of IgE receptors (FcRI) on the surface of BMMCs leads to the initiation of specific signaling pathways. Cells can be also activated nonspecifically by a tyrosine phosphatase inhibitor pervanadate, or by thapsigargin that inhibits Ca2+ ATPase pumps located on the endoplasmic reticulum. In this diploma thesis it was found out that rapid morphological changes can be monitored when BMMC are immobilised on the fibronectin before their activation. It was proved that specific and nonspecific activation events lead to microtubule reorganization, as well as to generation of a large number of microtubule-dependent protrusions. In the course of FcRI aggregation, generation of microtubule protrusions depends on the activity of Src family protein tyrosine kinases and on the intracellular Ca2+ concentration. STIM1, an endoplasmic reticulum Ca2+ sensor, which participates in the activation of store-operated Ca2+ channels in the plasma membrane, plays a key role in this process. Recently obtained data suggest that Ca2+ might affect microtubule nucleation from MTOC, as well as their polymerization.

Obtained results demonstrate that microtubule reorganization during BMMC activation depends on changes in the intracellular Ca2+ concentration, modulated by STIM1. Knowledge of molecular mechanisms reorganising microtubules could be used in new approaches to the treatment of inflammatory and allergic diseases.