Abstract:

CRMP2 was first identified in 1995 as a mediator of Sema3A signalization pathway which leads to axon growth cone collapse. Since then CRMP2 was designated as an essential cue during neuronal polarity estabilishment and neuronal growth in embryonic life. CRMP2 was also found hyperphosphorylated in NFT´s and this finding led to further research of CRMP2 function in the pathogenesis of AD. The activity and proper function of CRMP2 is regulated by phosphorylation and a deeper look into the mechanism of this modification is necessary for understanding how CRMP2 influences the function of neural cells. In this thesis I focus on signaling pathways, kinases and interaction partners of CRMP2 and describe how aberrant regulation of these interactions leads to Alzheimer's disease development.