

Abstract

Casein kinase 1 (CK1) protein family is an important part of signalling apparatus in eukaryotic organisms. These serin-threonine kinases play roles in several important developmental and cell-maintaining signalling pathways including Wnt signalling. CK1 kinases influence this signalling pathway in various manners; some of them positively – leading to expression of Wnt regulated genes and some of them negatively – contributing to degradation of the transcription activator β -catenin.

In this thesis, we were focusing on casein kinase 1 gamma (CK1 γ), which in mammalian cells in the presence of Wnt signal phosphorylates Wnt co-receptor LRP5/6. This phosphorylation leads to binding and inhibition of a β -catenin degradation complex. In *C. elegans* LRP5/6 receptor is probably missing from the genome, yet we can still observe influence of CSNK-1/CK1 γ on Wnt signalling. Using reduction-of-function experiments we have found out that loss of *csnk-1* expression enhanced a canonical Wnt signalling defect in mutants with reduced Wnt production. Using overexpression approach, we were not able to uncover the tissue specificity of CSNK-1 action. In order to obtain a better tool to study the function of CSNK-1, we began to optimize conditions for the use of auxin-inducible degron system for conditional CSNK-1 depletion.