Abstract:
The eIF4E is an important eukaryotic translation initiation factor, because of its ability to bind cap at 5'end of mRNA. There are three members of this protein family found in humans: eIF4E1, eIF4E2 and eIF4E3. eIF4E1 also plays role in in export of some mRNA from nucleus to cytoplasm. This protein is mostly regulated by mTOR signaling pathway and malfunctions in regulation leads to increased cell proliferation and thus tumorogenesis. eIF4E2 plays a role in regulating of translation during embryogenesis and it is known to mediate translation in terms of hypoxia. Role of eIF4E3 is so far shrouded in mystery. Some studies suggest it might be able to suppress tumor growth, but no studies have been done on human eIF4E3. Big potential of our work is, that all proteins we work with, are human. Based on our results, the endogenous amount of eIF4E3 protein is higher than it was thought. This is one of the reasons, why this protein should not escape our attention.
In my diploma thesis, I have studied physiological characteristics of cell cultures overexpressing eIF4E proteins after mTOR inhibition treatment. I have realized that the most efficient inhibitor in all tested cell cultures is PP-242, which binds directly into active site of mTOR kinase.
I have cloned 3xC FLAG tagged eIF4Es constructs and used them to describe subcellular localization of eIF4E3 in respect to P-bodies and stress granules. I confirmed that eIF4E3 does not localize to P-bodies which is in agreement with our previous observations using eIF4E proteins fused with GFP

Keywords: eIF4E1, eIF4E2, eIF4E3, mTOR, stress granules, P-bodies, polysomes, Rapamycine, PP-242, Resazurin, translation