Abstract

Protein kinases ERK1 and ERK2 are one of the most studied proteins in cell signalling. Both proteins are involved in a plethora of processes, such as phosphorylation and activation of kinases as part of signalling pathways. Enzymes ERK1 and ERK2 are part of MAPK/ERK signalling cascade, connected to many cellular including cell proliferation, cell growth or differentiation. The MAPK/ERK signalling cascade is often activated in different types of tumors, making it a candidate for developing new chemical inhibitors.

One of the important questions in fundamental research of ERK1 and ERK2 protein kinases is the search for difference between these proteins. Current knowledge points to redundancy of both proteins, howver several examples suggest otherwise. Recently, the work presented in Casanova *et al*. 2012 indirectly suggests divergent effect of ERK1 and ERK2 on cap-independent translation initiation.

In the Laboratory of RNA biochemistry we focus on HCV IRES (Hepatitis C Virus Internal Ribosome Entry Site) dependent translation initiation. This diploma thesis lead to establish RNA interference method in our laboratory and to establish reporter system to study ERK1 and ERK2 effect on HCV IRES dependent translation initiation. Based on our data acquired during our research, we present in this work results describing effect of ERK1 and ERK2 on HCV IRES dependent translation initiation. We conclude that ERK1 and ERK2 are both positive regulators of HCV IRES dependent translation initiation, thus ERK1 and ERK2 act redundantly in the regulation of this process. Protein ERK2 is stronger positive regulator of observed phenomenon than ERK1. This corresponds with intracellular ratio of ERK1 and ERK2.

Key words: ERK1, ERK2, MAPK/ERK, HCV IRES, translation, RNA interference, siRNA.