HuR Spatial Localization Is Affected by p38 MAPK Phosphorylation upon T-Cell Activation

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The post-transcriptional regulation controls RNA gene expression. This occurs between the process of transcription and translation of the gene. For this, it’s important to know the structure of the mRNA and the proteins that bind to stabilize, suppress or degrade. These proteins are affected by transductional modifications. One of these, is the phosphorylation caused by protein kinases. An example of this is p38: a Mitogen-Activated Protein Kinase of pharmaceutical interest, because of its important role in inflammatory diseases such as: Psoriasis, Arthritis and Chronic Obstructive Pulmonary Disease. On the other hand, it’s known that the protein HuR binds to the 3'-UTR of mRNAs and regulates its stability and translation. But, what effects does p38 phosphorylation have on HuR? This led us to pose the research question: Does p38 MAPK pathway affect the sub-cellular localization of HuR on activated T-Cells? For this, we propose the following hypothesis: p38 MAPK signaling pathway promotes HuR shuttling to the cytoplasm in Jurkat T-Cells. Our aim is to: Elucidate the role of p38 MAPK in the sub-cellular localization of HuR in activated T-Cells. As the independent variable: Cells treated with the p38 inhibitor and as dependent variable: HuR levels in the nucleus and cytoplasm. The methodologies performed were: Cell Culture, Determination of Proteins with the Bradford Method, SDS-PAGE, Western Blot and Immunohistochemistry. Finally, the hypothesis was accepted. HuR translocates from the nucleus to the cytoplasm after stimulation of T-Cells and it’s shown how this translocation is affected by the inhibition of p38.

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