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RECEPTOR MEDIATED STIMULATION OF AKT PAN-KINASE IN NIH3T3 CELLS: REVIEW ARTICLE

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INTRODUCTION

AKT protein kinase, also known as protein kinase B (PKB), is a serine/threonine-specific protein kinase involved in multiple cellular processes such as cell growth, survival, metabolism, and motility. AKT is activated by a variety of extracellular stimuli, including growth factors and hormones that activate upstream receptor tyrosine kinases (RTKs). Upon binding of ligands to RTKs, various signaling pathways are activated, leading to the activation of AKT. One of the most studied RTK pathways is the phosphatidylinositol 3-kinase (PI3K) pathway, which stimulates AKT through its activation by the RTKs.

NIH3T3 cells are a widely used model system to study the signaling mechanisms of AKT activation in response to various stimuli. Here, we review the receptor-mediated stimulation of AKT in NIH3T3 cells, highlighting the different pathways and molecules involved in the process.

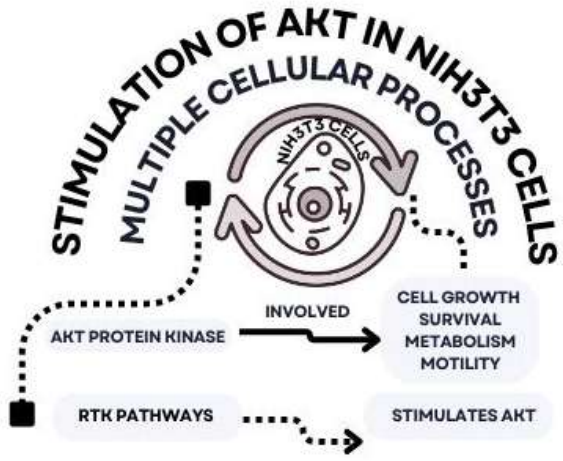

Fig. 1: Receptor-mediated stimulation of AKT in NIH3T3 cells, highlighting the different pathways and molecules.

PATHWAYS

AKT Pathways in NIH3T3 Cells

The activation of AKT in NIH3T3 cells can occur through two main pathways: the PI3K pathway and the mitogen-activated protein kinase (MAPK) pathway. The PI3K pathway consists of multiple steps, including the binding of growth factors to RTKs, the activation of PI3K, the production of phosphatidylinositol (3,4,5)-trisphosphate (PIP3), and the activation of AKT by phosphorylation at Thr308 and Ser473. The MAPK pathway, on the other hand, involves the activation of Ras, Raf, MEK, and ERK proteins, which can directly or indirectly phosphorylate AKT on Ser473.

PI3K PATHWAY

The PI3K pathway is the most well-characterized pathway for AKT activation in NIH3T3 cells. RTKs, such as the epidermal growth factor receptor (EGFR), platelet-derived growth factor receptor (PDGFR), and insulin-like growth factor 1 receptor (IGF-1R), are known to activate PI3K and its downstream targets, including AKT. For example, the binding of PDGF to PDGFR results in the recruitment of PI3K to the receptor complex, leading to the production of PIP3 and the activation of AKT. Additionally, AKT can also be activated by G protein-coupled receptors (GPCRs) that activate PI3K through the βγ subunits of G proteins. For instance, the activation of the GPCRs by lysophosphatidic acid (LPA) or sphingosine 1-phosphate (S1P) can cause the activation of PI3K and AKT in NIH3T3 cells.

MAPK PATHWAY

The MAPK pathway is also involved in the activation of AKT in NIH3T3 cells. The activation of Ras, a small GTPase, by upstream signaling events activates the Raf-MEK-ERK cascade, which is known to phosphorylate AKT on Ser473. The activation of Ras can be achieved