AKT1 E17K mutant phosphorylates GSK3

Matthews, L., Orlic-Milacic, M., Thorpe, L., Yuzugullu, H., Zhao, JJ.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references

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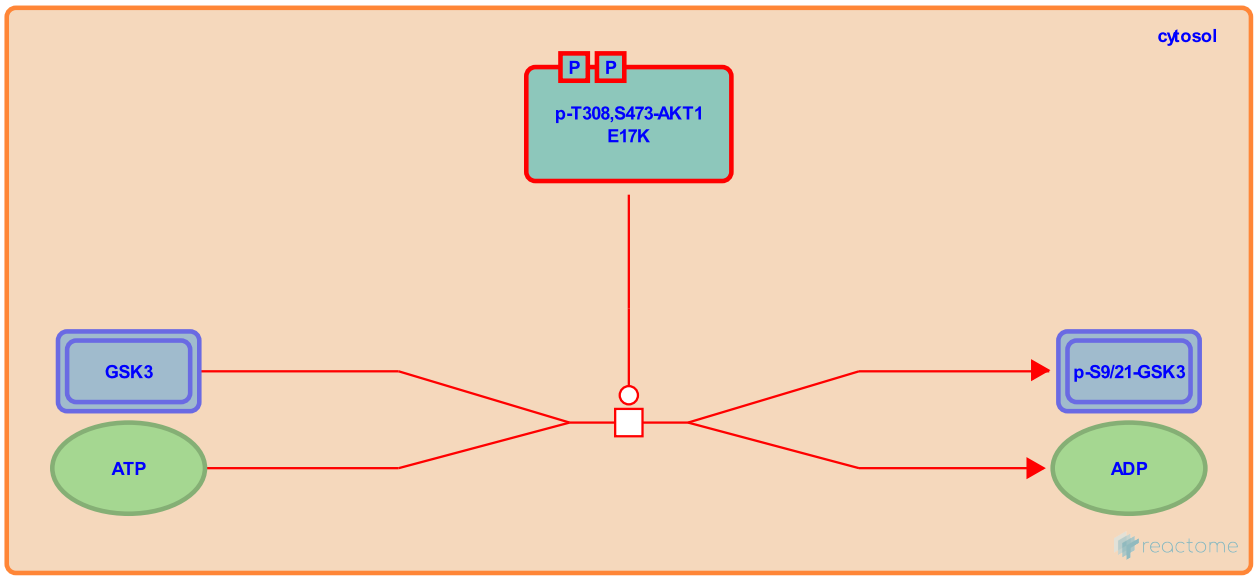
Stable identifier: R-HSA-2399966

Type: transition

Compartments: cytosol

Diseases: cancer



AKT1 E17K gain-of-function mutant preserves the ability to phosphorylate GSK3 (Malanga et al. 2008). AKT-mediated phosphorylation inactivates GSK3 and enables WNT-independent stabilization of beta-catenin (CTNNB1) (Haq et al. 2003). AKT-mediated inactivation of GSK3 also triggers changes in glucose metabolism (Ueki et al. 1997).

Literature references

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Editions

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