

p-S939,T1462-TSC2 binding to 14-3-3 dimer is negatively regulated by DDIT4

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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

- Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. 7
- Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics*, 33, 3461-3467. A
- Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗
- Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology*, *14*, e1005968. *オ*

This document contains 1 reaction (see Table of Contents)

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Stable identifier: R-HSA-5632732

Type: binding

Compartments: cytosol



Phosphorylation of TSC2 by AKT enables association of TSC2 with 14-3-3 proteins YWHAB (14-3-3 protein beta/alpha), YWHAQ (14-3-3 protein theta), YWHAG (14-3-3 protein gamma), YWHAH (14-3-3 protein eta), YWHAE (14-3-3 protein epsilon), YWHAZ (14-3-3 protein zeta/delta) or SFN (14-3-3 protein sigma) (Liu et al. 2002). Binding to 14-3-3 proteins sequesters TSC2 to the cytosol and prevents its association with TSC1 (Cai et al. 2006).

Literature references

Liu, MY., Walker, CL., Bedford, MT., Cai, S., Espejo, A. (2002). 14-3-3 interacts with the tumor suppressor tuberin at Akt phosphorylation site(s). *Cancer Res.*, 62, 6475-80.

Kim, J., Guo, R., Walker, CL., Shen, J., Kiguchi, K., Cai, SL. et al. (2006). Activity of TSC2 is inhibited by AKT-mediated phosphorylation and membrane partitioning. J. Cell Biol., 173, 279-89. *¬*

Editions

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