

YAP1 binds TP73

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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.

The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

Literature references

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Reactome database release: 70

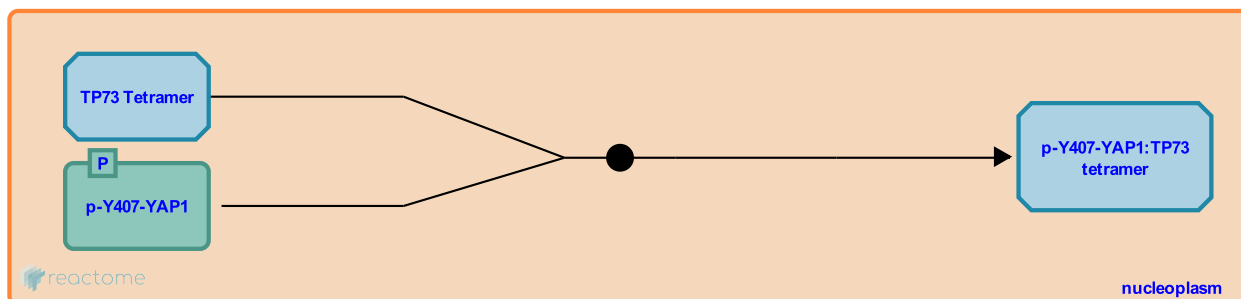
This document contains 1 reaction ([see Table of Contents](#))

YAP1 binds TP73 [↗](#)

Stable identifier: R-HSA-8956676

Type: binding

Compartments: nucleoplasm



YAP1, phosphorylated on tyrosine residue Y407 (Y357 in the splicing isoform 3, known as YAP1-1beta) by the protein tyrosine kinase ABL1, activated in response to DNA damage, forms a complex with TP73. ABL1-phosphorylated YAP1 can no longer bind RUNX1 (Levy, Adamovich et al. 2008; Levy, Reuven and Shaul 2008). Binding of phosphorylated YAP1 to TP73 may target TP73 to promoters of pro-apoptotic target genes instead of cell cycle arrest genes (Levy, Adamovich et al. 2008).

Literature references

Levy, D., Reuven, N., Shaul, Y. (2008). A regulatory circuit controlling Itch-mediated p73 degradation by Runx. *J. Biol. Chem.*, 283, 27462-8. [↗](#)

Levy, D., Adamovich, Y., Reuven, N., Shaul, Y. (2008). Yap1 phosphorylation by c-Abl is a critical step in selective activation of proapoptotic genes in response to DNA damage. *Mol. Cell*, 29, 350-61. [↗](#)

Editions

2016-09-14	Authored	Orlic-Milacic, M.
2016-12-20	Reviewed	Ito, Y., Chuang, LS.
2017-05-09	Edited	Orlic-Milacic, M.