Constitutive phosphorylation by pERK1/2

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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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Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

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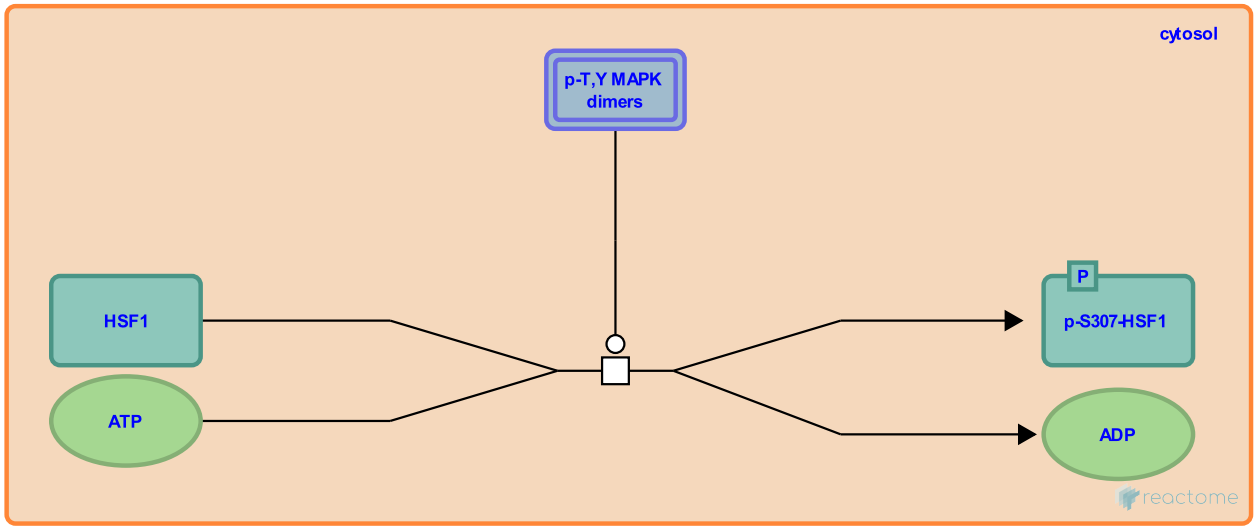
Stable identifier: R-HSA-3371531

Type: transition

Compartments: cytosol



Constitutive phosphorylation at Ser307 was shown to inhibit HSF1 transcriptional activity under normal temperatures. Substitution of Ser307 with alanine derepresses the transactivation domain such that the S307A mutant showed increased transcriptional activity in human and mouse cells (Knauf U et al. 1996; Kline MP & Morimoto RI 1997).
HSF1-ERK association was shown to promote ERK activity in human HeLa, acute monocytic leukemia THP1 and metastatic cutaneous SCC7 cells resulting in phosphorylation of HSF1 on Ser307 (Chu B et al. 1996; Wang X et al. 2004). This phosphorylation in turn promoted HSF1 association with YWHAE (14-3-3 epsilon), which may be involved in the attenuation of HSF1 activity during recovery and leads to accelerated cytoplasmic localization of HSF1 (Wang X et al. 2003, 2004).

Literature references

Kyriakis, J., Kingston, RE., Knauf, U., Newton, EM. (1996). Repression of human heat shock factor 1 activity at control temperature by phosphorylation. *Genes Dev., 10,* 2782-93.

Editions

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