

Phosphorylation of HSF1 at Ser326 induces

transactivation

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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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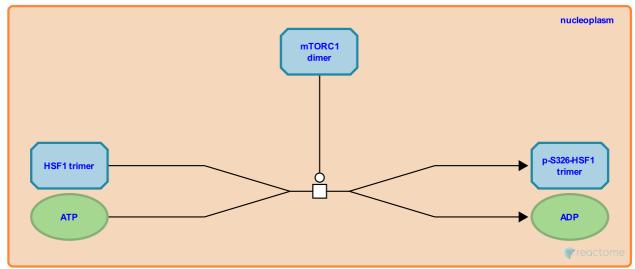
This document contains 1 reaction (see Table of Contents)

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Stable identifier: R-HSA-5082405

Type: transition

Compartments: nucleoplasm



Mutagenesis experiments and functional studies suggest that phosphorylation of HSF1 residue Ser326 promotes induction of the HSF1 transcriptional competence in response to heat and other cell stressors including proteasome inhibitors and sodium arsenite (Guettouche T et al. 2005; Chou SD et al. 2012).

The mammalian target of rapamycin complex 1 (mTORC1) has been implicated in sensing intracellular protein misfolding (Qian SB et al. 2010; Chou SD et al. 2012). RNA interference?mediated repression of mTOR kinase activity in human HeLa cells was found to increase sensitivity to heat shock. Moreover, inhibition of HSF1 phosphorylation on Ser326 by rapamycin suggests that this site in HSF1 is a target for the mTORC1complex (Chou SD et al. 2012).

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Editions

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