

TP53 stimulates expression of SESN1,2,3 genes

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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. [↗](#)

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. [↗](#)

Reactome database release: 90

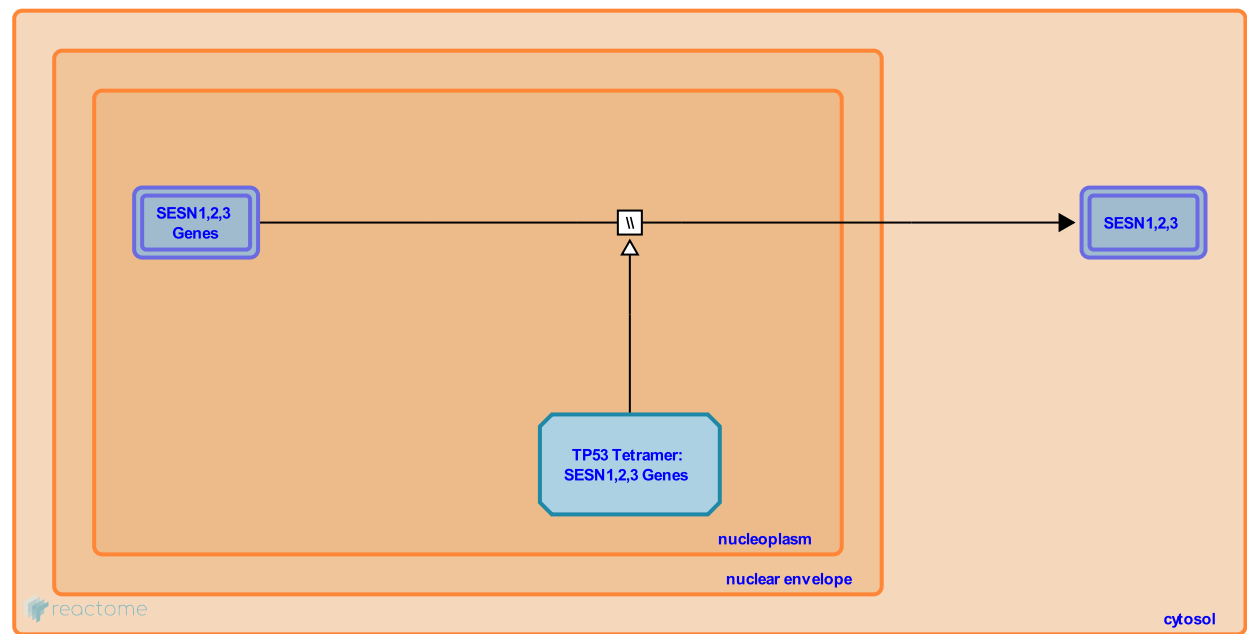
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TP53 stimulates expression of SESN1,2,3 genes ↗

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Sestrins (SESN) are a small family of stress-sensitive gene that are conserved across several species. Mammals express three different SESN family members characterized as SESN1-3. Sestrin genes, SESN1, SESN2 and SESN3, are upregulated in response to TP53-mediated transcriptional regulation. SESN1 and SESN2 were classified as members of the growth arrest and DNA damage (GADD) gene family that can regulate cell growth and viability under different cellular pressures. In particular, p53 negatively modulates the mTOR pathway via SESN1 and SESN2 upregulation (Feng 2010). SESN3 was identified shortly after SESN2 through in silico analysis and was found to be a target of the forkhead transcription factor (FOXO) family. A specific TP53 binding site on the human SESN3 promoter has not been identified yet, but was found in the rat ortholog (Velasco-Miguel et al. 1999, Budanov et al. 2002, Brynczka et al. 2007).

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Editions

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