

TP53 binds the GLS2 promoter

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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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Reactome database release: 88

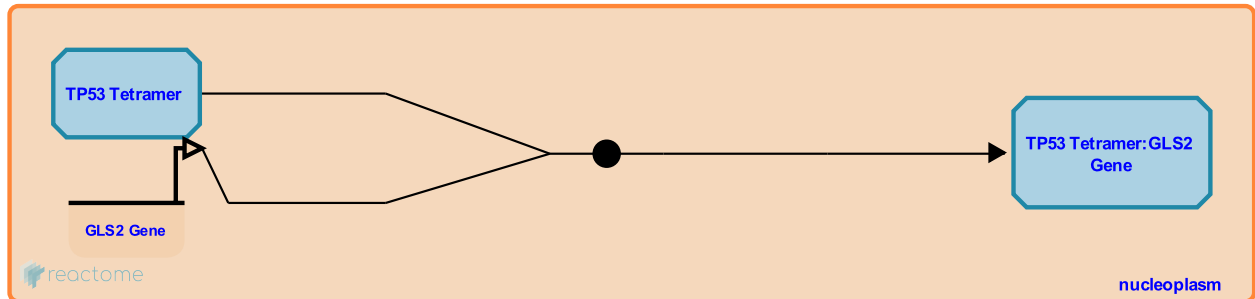
This document contains 1 reaction ([see Table of Contents](#))

TP53 binds the GLS2 promoter [↗](#)

Stable identifier: R-HSA-5632914

Type: binding

Compartments: nucleoplasm



The mitochondrial glutaminase GLS2 gene possesses two putative p53-binding sites in its promoter and one putative p53 binding site in the first intron. TP53 was demonstrated to bind to p53-response elements in the promoter but not intron 1 of GLS2 (Hu et al. 2010, Suzuki et al. 2010).

Literature references

Zhang, C., Hu, W., Sun, Y., Wu, R., Levine, A., Feng, Z. (2010). Glutaminase 2, a novel p53 target gene regulating energy metabolism and antioxidant function. *Proc. Natl. Acad. Sci. U.S.A.*, 107, 7455-60. [↗](#)

Hosokawa, H., Poyurovsky, MV., Nagano, H., Mayama, T., Tanaka, T., Prives, C. et al. (2010). Phosphate-activated glutaminase (GLS2), a p53-inducible regulator of glutamine metabolism and reactive oxygen species. *Proc. Natl. Acad. Sci. U.S.A.*, 107, 7461-6. [↗](#)

Editions

2014-12-23	Authored, Edited	Orlic-Milacic, M.
2014-12-30	Reviewed	Hwang, PM., Kang, JG., Wang, PY.
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