p23 (PTGES3) binds

HSP90:ATP:FKBP4:nascent protein

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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

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

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. ↗

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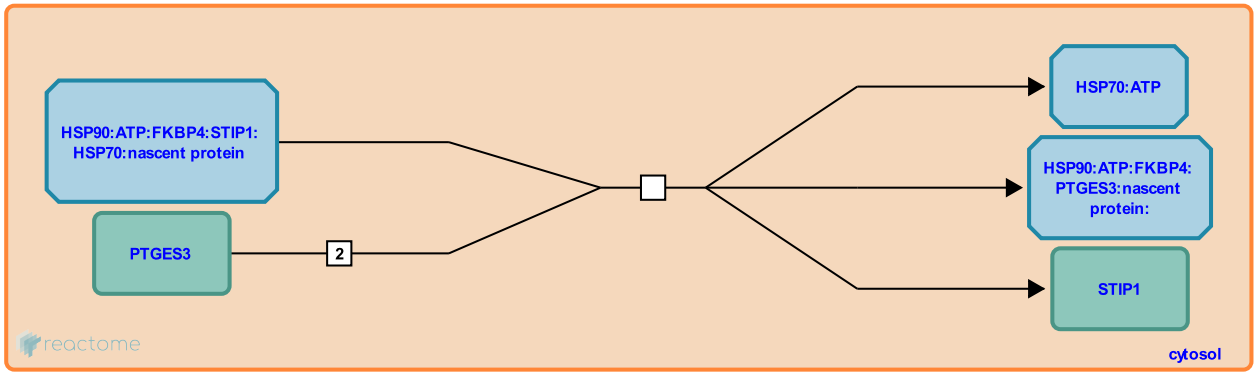
Stable identifier: R-HSA-5618110

Type: transition

Compartments: cytosol



Immunophilin p23 (also known as PTGES3) binds selectively to the ATP-bound state of HSP90. p23 stabilizes the closed state of HSP90, which weakens the binding of STIP1(HOP) and promotes its exit from the complex (McLaughlin H et al. 2006; Karagöz GE et al. 2011). When p23 is added to the client-transfer complex in the absence of the immunophilin or with FKBP51 (FKBP5), two copies of p23 are incorporated with concomitant loss of HSP70 and HOP (Ebong I et al. 2016). By contrast no stable complex with two p23 subunits is observed in the presence of FKBP52 (FKBP4); expulsion of HSP70, HOP and p23 occur with a low population of a complex incorporating only one p23 subunit (Ebong I et al. 2016).

Literature references

Yao, ZP., Nielsen, PR., Sobott, F., McLaughlin, SH., Robinson, CV., Laue, ED. et al. (2006). The co-chaperone p23 arrests the Hsp90 ATPase cycle to trap client proteins. *J. Mol. Biol., 356*, 746-58. ↗

Boelens, R., van Rosmalen, M., Uetrecht, C., Sinnige, T., Heck, AJ., Duarte, AM. et al. (2011). N-terminal domain of human Hsp90 triggers binding to the cochaperone p23. *Proc. Natl. Acad. Sci. U.S.A., 108*, 580-5. ↗

Editions

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