JAK2 Autophosphorylates in Response to Leptin

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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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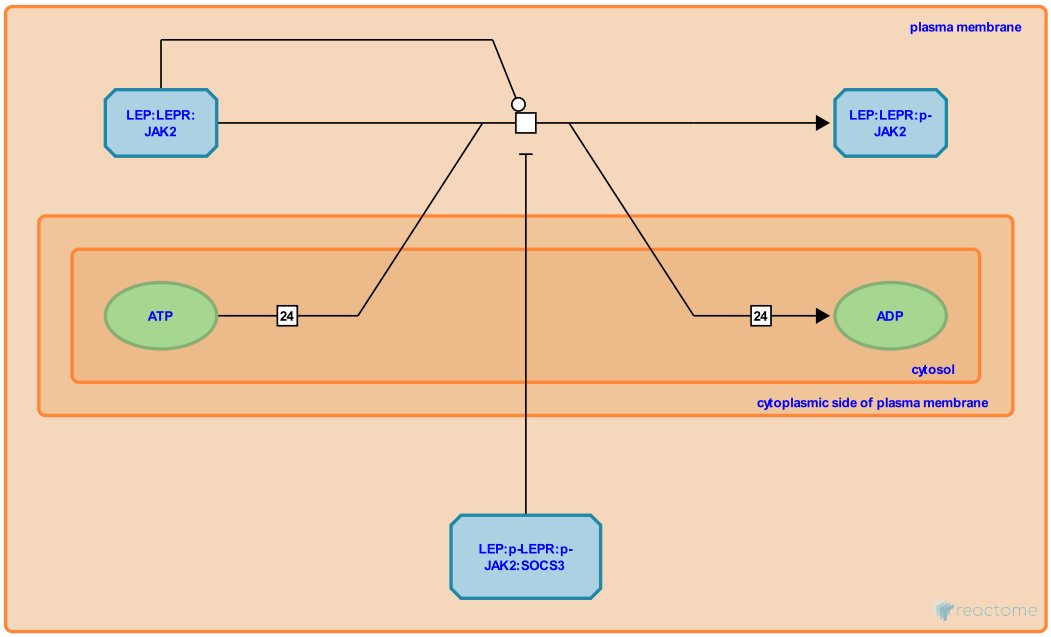
Stable identifier: R-HSA-2586555

Type: transition

Compartments: plasma membrane, cytosol

Inferred from: Jak2 Autophosphorylates in Response to Leptin (Mus musculus)



As inferred from mouse, binding of Leptin (LEP) to the Leptin receptor (LEPR) causes a conformational change in LEPR that activates autophosphorylation of JAK2 at multiple tyrosine residues. Phosphorylated JAK2 has much higher kinase activity than unphosphorylated JAK2.
As inferred from mouse, the kinase inhibitory domain of SOCS3 interacts with the activation loop of jAK2 and inhibits the phosphorylation of JAK2.

Literature references

Frantz, JD., Flier, JS., El-Haschimi, K., Bjørbaek, C. (1999). The role of SOCS-3 in leptin signaling and leptin resistance. *J. Biol. Chem., 274*, 30059-65. ↗

Editions

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