

FFAR1:FFAR1 ligands activate Gq

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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: 88

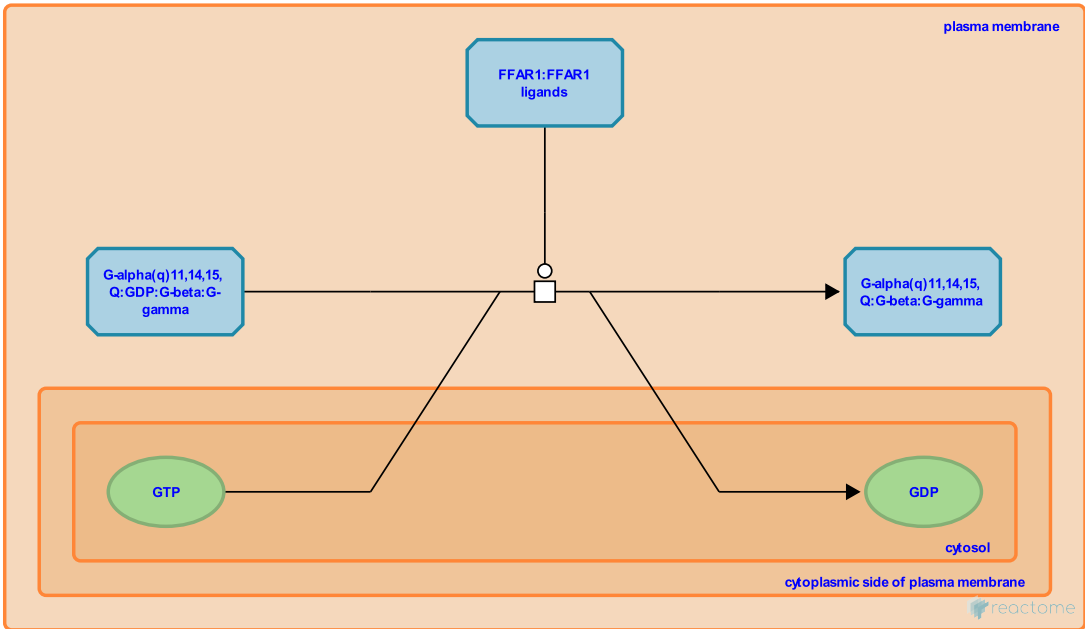
This document contains 1 reaction ([see Table of Contents](#))

FFAR1:FFAR1 ligands activate Gq ↗

Stable identifier: R-HSA-416530

Type: transition

Compartments: plasma membrane, cytosol



FFAR1 (GPR40) is a G-protein coupled receptor. Based on studies with inhibitors of G proteins such as pertussis toxin FFAR1 is believed to signal through Gq/11. Binding of free fatty acids by FFAR1 activates the heterotrimeric Gq complex, which then activates Phospholipase C. From experiments in knockout mice it is estimated that signaling through FFAR1 is responsible for about 50% of the augmentation of insulin secretion produced by free fatty acids. The rest of the augmentation is due to metabolism of the free fatty acids within the pancreatic beta cell.

Literature references

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Editions

2009-06-08	Authored, Edited	May, B.
2009-09-09	Reviewed	Poitout, V., Kebede, M.
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