

Autocatalytic phosphorylation of FGFR3

fusions

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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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Reactome database release: 88

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

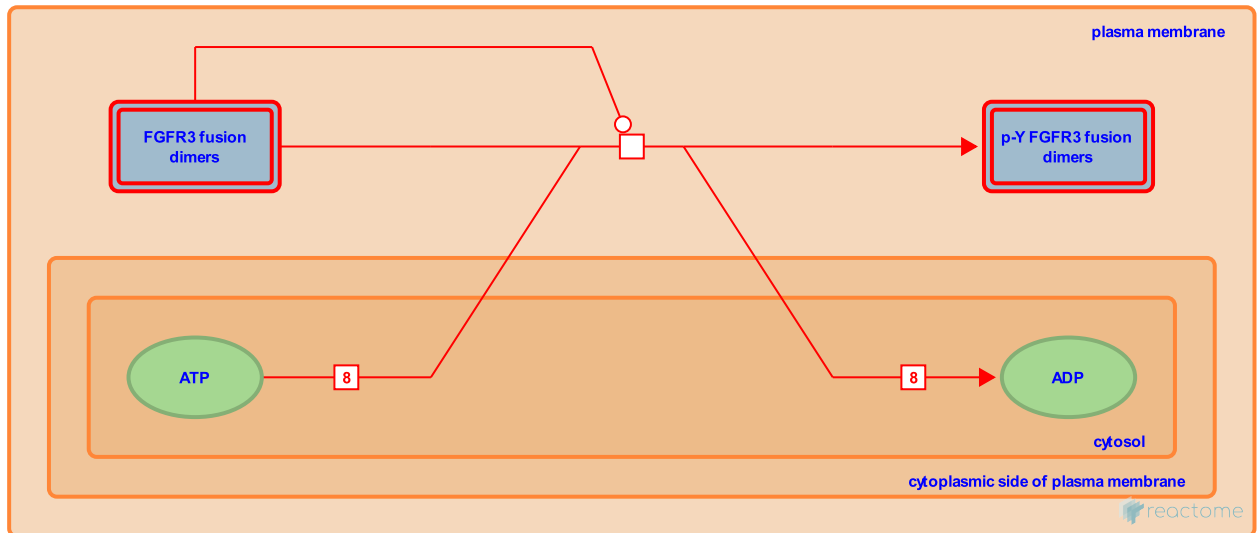
Autocatalytic phosphorylation of FGFR3 fusions [↗](#)

Stable identifier: R-HSA-8853309

Type: transition

Compartments: plasma membrane

Diseases: cancer



FGFR3 fusions promote cellular proliferation and tumorigenesis that can be inhibited by tyrosine kinase inhibitors, suggesting that signaling is dependent on autophosphorylation of tyrosine residues in the intracellular region as is the case for WT FGFR3 (Singh et al, 2012; Parker et al, 2013; Williams et al, 2013; Wu et al, 2013; Yuan et al, 2014). FGFR3 fusions are reported to activate the ERK, STAT and AKT pathways, but not the PLC gamma pathway as the fusions generally lack the tyrosine residue required for PLC gamma recruitment (Parker et al, 2013; Williams et al, 2013; Wu et al, 2013; reviewed in Parker et al, 2014; Carter et al, 2015).

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Editions

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