

FGFR3 fusions dimerize

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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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This document contains 1 reaction (see Table of Contents)

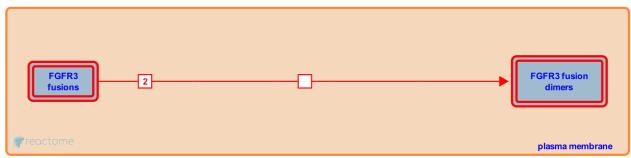
FGFR3 fusions dimerize 7

Stable identifier: R-HSA-8853317

Type: transition

Compartments: plasma membrane

Diseases: cancer



Constitutively active fusions of FGFR3 have been identified in glioblastoma, non-small cell lung cancer and bladder cancer, among others (Singh et al, 2012; Williams et al, 2013; Wu et al, 2013; Wang et al, 2014; Capelletti et al, 2014; Yuan et al, 2015; Carneiro et al, 2015; reviewed in Parker et al, 2014). The most prevalent fusion partner is TACC3 (transforming acidic coiled-coil-containing protein 3), a microtubule binding protein with roles in microtubule spindle assembly and chromosome segregation (Singh et al, 2014; Burgess et al, 2015; reviewed in Parker et al, 2014). There are conflicting reports about whether FGFR3 fusions form constitutive dimers, however ligand-independent autophosphorylation and downstream signaling has been demonstrated. FGFR3 fusions promote cellular proliferation and tumorigenesis and appear to escaped miRNA-mediated downregulation (Singh et al, 2012; Williams et al, 2013; Wu et al, 2013; Parker et al, 2013; reviewed in Parker et al, 2014).

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Editions

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