GLI:SUFU dissociates

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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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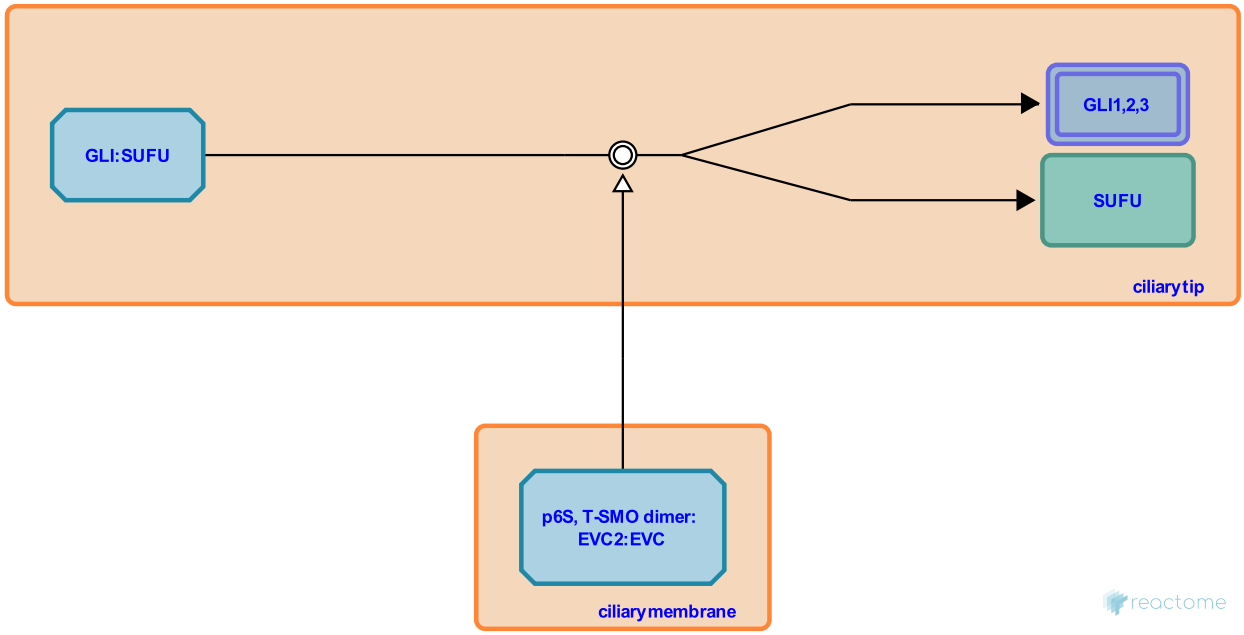
GLI:SUFU dissociates ↗

Stable identifier: R-HSA-5635859

Type: dissociation

Compartments: ciliary tip



Hh signaling promotes the dissociation of the GLI:SUFU complex in the cilium downstream of SMO activation (Humke et al, 2010; Tukachinsky et al, 2010). This appears to divert the transcription factors away from the partial processing/degradation pathway and allow the full-length forms to translocate to the nucleus where they are converted to labile transcriptional activators (Humke et al, 2010; Tukachinsky et al, 2010; Pan et al, 2006; Kim et al, 2009). How the Hh signal is transmited from SMO to promote the dissociation of the GLI:SUFU complex is not clear, however it may involve changes in PKA activity as a result of lowered cAMP levels upon pathway stimulation. (Tukachinsky et al, 2010; Wen et al, 2010; Tuson et al, 2011; Barzi et al, 2010; reviewed in Briscoe and Therond, 2013). GPR161, which localizes to the cilium in a TULP3-dependent manner and which increases cAMP levels in the absence of ligand, is cleared from the cilium upon pathway activation, and deletion of GPR161 increases Hh-dependent signaling (Mukhopadhyay et al, 2010; Mukhopadhyay et al, 2013). These data suggest that removal of ciliary GPR161 upon Hh stimulation may contribute to pathway activity by downregulating PKA activity through cAMP levels (reviewed in Mukhopadhyay and Rohatgi, 2014).

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Editions

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