S-Farn-Me KRAS4B binds calmodulin

Gavathiotis, E., Rothfels, K.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.

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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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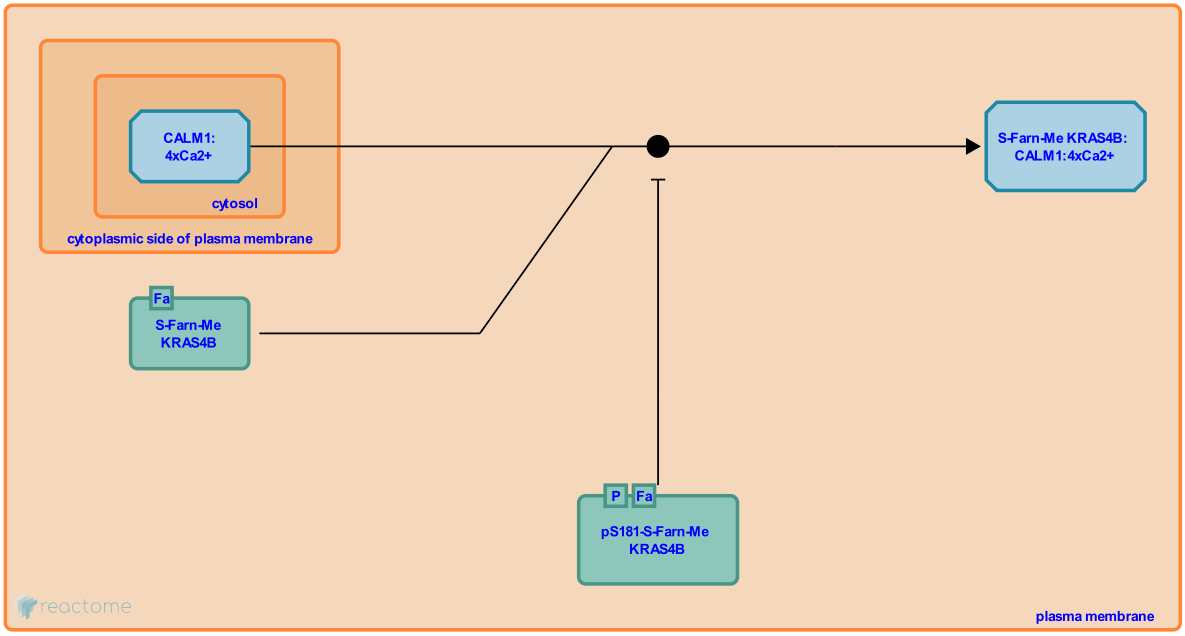
Stable identifier: R-HSA-9653585

Type: binding

Compartments: plasma membrane, cytosol



KRAS4B, unique among RAS isoforms, has been shown to bind to calmodulin (Villalonga et al, 2001; Lopez-Alcala et al, 2008). This interaction is thought to decrease the affinity of KRAS4B for the plasma membrane (Fivaz and Meyer, 2005; Sidhu et al, 2003; reviewed in Ahearn et al, 2018; Nussinov et al, 2015). Interaction between oncogenic KRAS4B and calmodulin has been shown to promote tumorigenesis by interfering with the activation of CAMK2. This in turn relieves the suppression of beta-catenin dependent signaling mediated by the non-canonical WNT signaling pathway (Wang et al, 2015).
The interaction between KRAS4B and calmodulin is inhibited by PKC- or PRKG2-dependent KRAS4B phosphorylation at serine 181 (Wang et al, 2015; Alvarez-Moya et al, 2010; reviewed in Ahearn et al, 2018).

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Editions

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