RAS intrinsic GTPase activity hydrolyzes GTP to GDP

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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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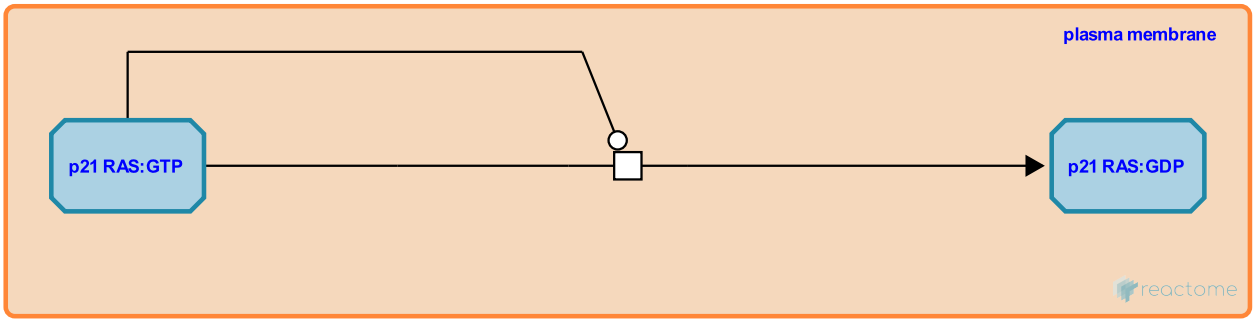
Stable identifier: R-HSA-9649736

Type: transition

Compartments: plasma membrane



RAS proteins have weak intrinsic GTPase activity in the absence of other effectors (Gibbs et al, 1984; reviewed in Pylayeva-Gupta et al, 2011). Nucleotide attack is mediated by residue Q61 and facilitated by van der Waals bonds contributed by glycine residues at position 12 and 13; these three residues account for the majority of oncogenic and pathogenic mutations found in RAS proteins (reviewed in Prior et al, 2012). GAP proteins stimulate the intrinsic GTPase activity of RAS proteins by inserting an arginine residue into the active site, which contributes to proper positioning of the critical Q61 RAS residue (reviewed in King et al, 2013).

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Editions

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