

# RAC1 and CDC42 activate PAK3

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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

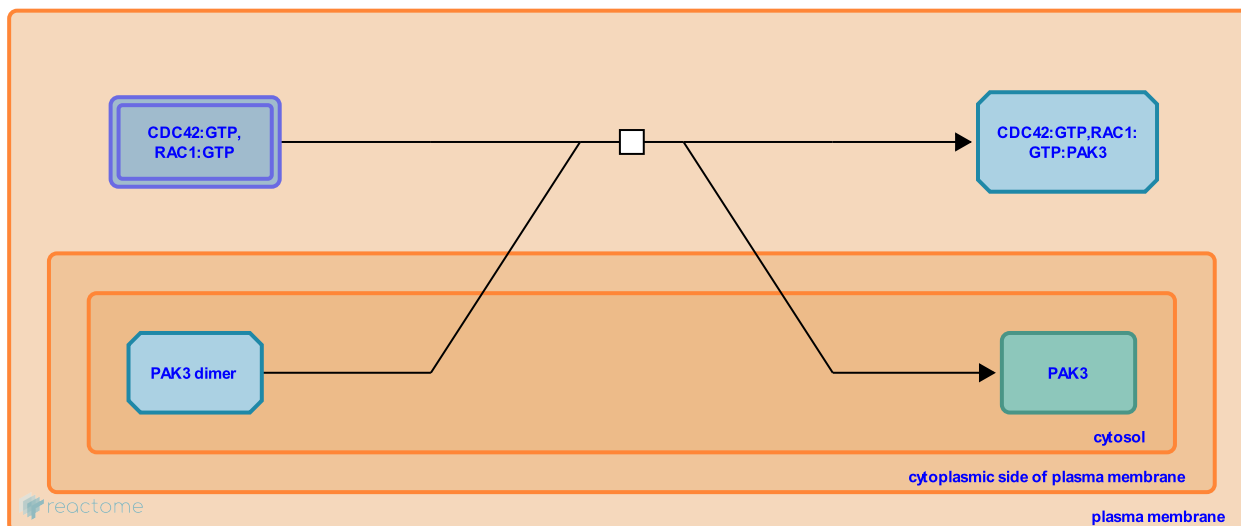
This document contains 1 reaction ([see Table of Contents](#))

## RAC1 and CDC42 activate PAK3 [↗](#)

**Stable identifier:** R-HSA-8981926

**Type:** transition

**Compartments:** cytosol, plasma membrane



Inactive p21-associated kinases (PAKs), PAK1, PAK2 and PAK3, form homodimers that are autoinhibited through in trans interaction between the inhibitory N-terminus of one PAK molecule and the catalytic domain of the other PAK molecule. PAK3, like other PAK isoforms, is a direct effector of RAC1 and CDC42 GTPases. RAC1 and CDC42 bind to a highly conserved motif in the amino terminus of PAK3 known as p21-binding domain (PBD) or Cdc42/Rac interactive binding (CRIB) domain. This binding induces a conformational change that disrupts PAK3 homodimers and relieves autoinhibition of the catalytic carboxyl terminal domain, thereby inducing autophosphorylation at several sites and enabling the phosphorylation of exogenous substrates (Manser et al. 1994, Manser et al. 1995, Zhang et al. 1998, Lei et al. 2000, Parrini et al. 2002; reviewed by Daniels and Bokoch 1999, Szczepanowska 2009).

### Literature references

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### Editions

2008-07-16	Authored	Garapati, P V.
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