

# CDC42:GTP binds PAK2

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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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Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics*, 33, 3461-3467. [↗](#)

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Reactome database release: 88

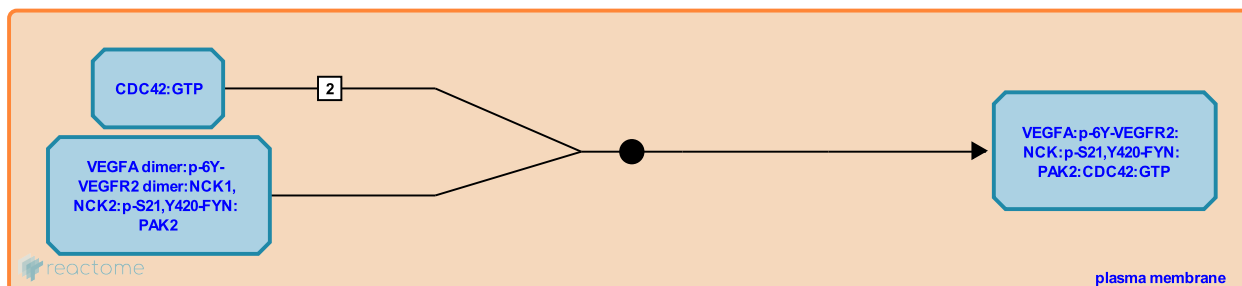
This document contains 1 reaction ([see Table of Contents](#))

## CDC42:GTP binds PAK2 ↗

**Stable identifier:** R-HSA-5218832

**Type:** binding

**Compartments:** plasma membrane



The PAK family of serine/threonine kinases are known to be activated by binding to the GTP-bound form of CDC42 or RAC1, small GTPases of the Rho family that are involved in regulating the organization of the actin cytoskeleton. PAK exists as homodimer in a trans-inhibited conformation. The kinase inhibitory (KI) domain of one PAK molecule binds to the C-terminal catalytic domain of the other and inhibits catalytic activity. Association of GTP-bound forms of CDC42 or RAC1 with the PAK PBD/CRIB domain induces conformational changes in the N-terminal domain that no longer support its autoinhibitory function. CDC42-mediated activation primes PAK2 for superactivation by tyrosine phosphorylation (Renkema et al. 2002).

### Literature references

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### Editions

2013-08-30	Authored, Edited	Garapati, P V.
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