PIP2 binds inhibiting VAV

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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

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

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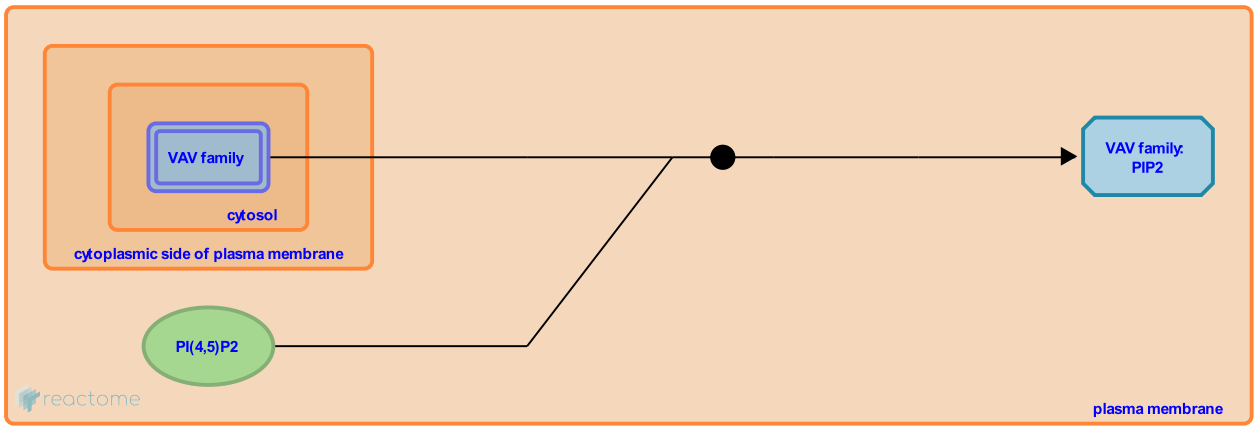
PIP2 binds inhibiting VAV ↗

Stable identifier: R-HSA-434633

Type: binding

Compartments: cytosol, plasma membrane

Inferred from: PIP2 inhibits Vav1 (Mus musculus)



Vav interacts directly with PIP2 and PIP3, with a fivefold selectivity for PIP3 over PIP2. PIP3 gives a twofold stimulation of Vav1 GEF activity while PIP2 leads to 90% inhibition. Binding probably occurs through the PH domain, known to bind phosphoinositides.

Literature references

White, MA., Falck, JR., Mosteller, RD., Xia, Y., Han, J., Shu, X. et al. (1998). Role of substrates and products of PI 3-kinase in regulating activation of Rac-related guanosine triphosphatases by Vav. *Science, 279*, 558-60. ↗

Editions

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