

JAM2 binds JAM3

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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

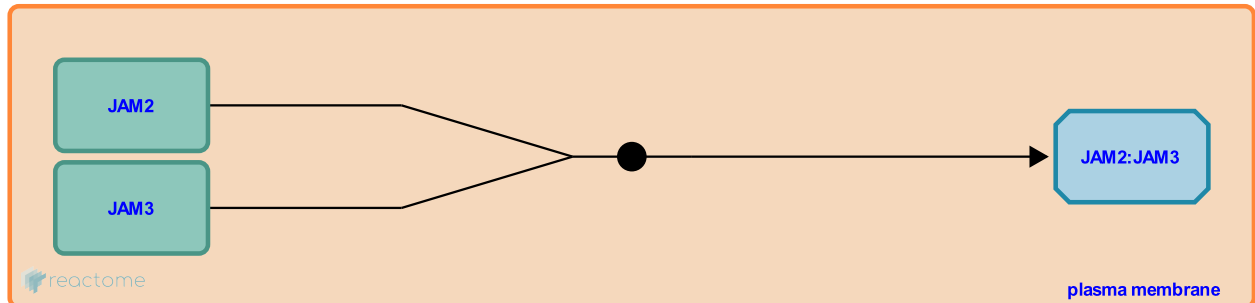
This document contains 1 reaction ([see Table of Contents](#))

JAM2 binds JAM3 [↗](#)

Stable identifier: R-HSA-202721

Type: binding

Compartments: plasma membrane



JAM2 and JAM3 bind each other and are strongly expressed by endothelial cells of high endothelial venules, the predominant site of leukocyte extravasation. JAM2 and JAM3 also bind to the leukocyte integrins VLA-4 and Mac-1 respectively.

Literature references

Ludwig, R.J., Baatz, H., Santoso, S., Podda, M., Hardt, K., Boehncke, W.H. et al. (2005). Junctional adhesion molecules (JAM)-B and -C contribute to leukocyte extravasation to the skin and mediate cutaneous inflammation. *J Invest Dermatol*, 125, 969-76. [↗](#)

Weber, C., Fraemohs, L., Dejana, E. (2007). The role of junctional adhesion molecules in vascular inflammation. *Nat Rev Immunol*, 7, 467-77. [↗](#)

Editions

2007-11-12	Authored	Ouwehand, W.H.
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