

# ICAM1-5 bind Integrin alphaLbeta2 (LFA-1)

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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

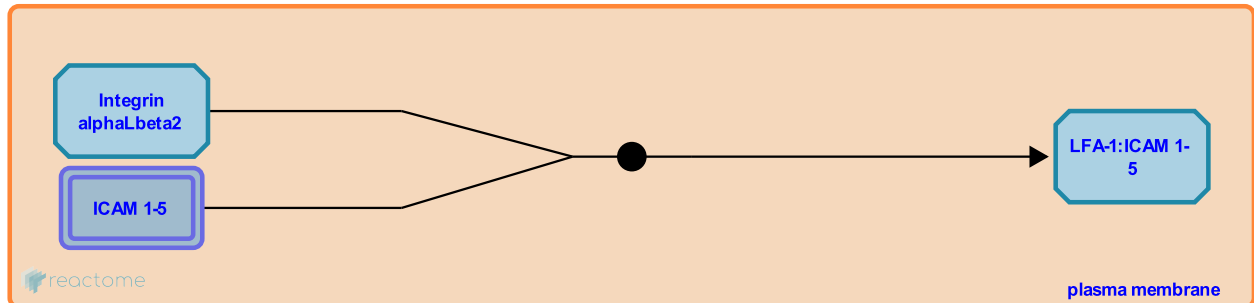
This document contains 1 reaction ([see Table of Contents](#))

## ICAM1-5 bind Integrin alphaLbeta2 (LFA-1) [↗](#)

**Stable identifier:** R-HSA-199050

**Type:** binding

**Compartments:** plasma membrane



Integrins play a central role in mediating lymphocyte adhesion to a number of surfaces. Integrin alphaLbeta2 (LFA-1) interacts with Intercellular adhesion molecule (ICAM)1-5, which are typically expressed on other immune system cells. ICAM4 and 5 are known to be expressed on telencephalic neurons. VCAM-1 regulates lymphocyte adhesion to activated endothelial cells via Very Late Antigen-4 (VLA-4). To function in a circulating mode, leukocytes express LFA-1 and VLA-4 in a low ligand binding capacity. When leukocytes reach sites of inflammation, these integrins are switched to a higher binding state to guide the complex process of transmigration, tethering, rolling, arrest, adhesion and shape change. Signal cascades between LFA-1 and VLA-4 may cross-talk affecting binding affinities in a reciprocal fashion.

### Literature references

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### Editions

2007-07-08	Authored	de Bono, B.
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