Bradykinin receptors B1 and B2 bind to bradykinin

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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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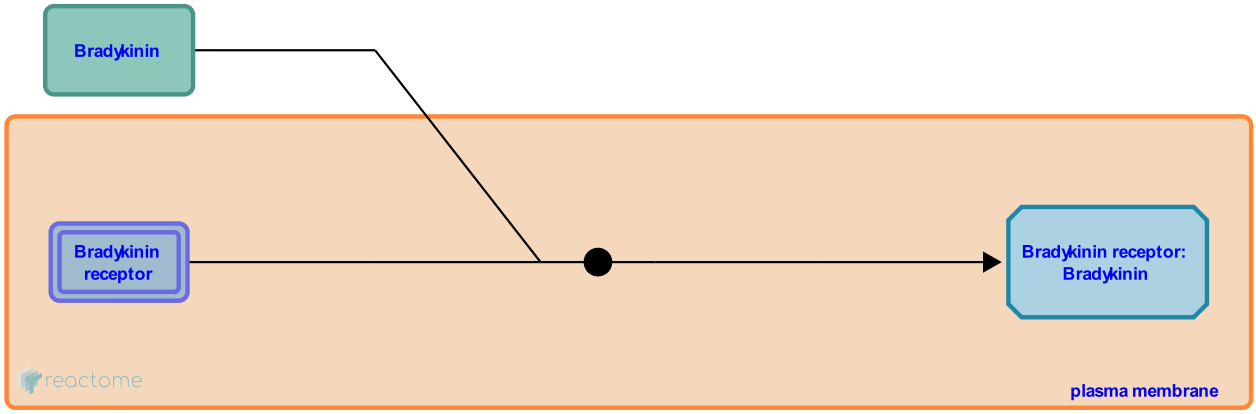
Stable identifier: R-HSA-374331

Type: binding

Compartments: extracellular region, plasma membrane



Bradykinin (Rocha e Silva M, et al, 1949) is a 9 amino-acid peptide belonging to the kinin group of proteins. It causes blood vessel dilation via the release of prostacyclin, nitric oxide and endothelial-derived hyperpolarizing factor, resulting in lower blood pressure. It is also involved in the pain mechanism. Bradykinin exerts its effects through two receptors, bradykinin receptor B1 and 2 (B1R and B2R respectively). B1R (Menke JG et al, 1994) is synthesized de novo following tissue injury and receptor binding leads to an increase in cytosolic calcium concentration, resulting in chronic and acute inflammatory responses. Unlike B1R, B2R (Hess JF et al, 1992) is ubiquitously and constitutively expressed in healthy tissues. It also increase cytosolic calcium concentration and stimulates the mitogen-activated protein kinase pathways.

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Editions

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