ADRB2:Catecholamine binds ARRB1, ARRB2

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21/10/2019

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.

The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

Literature references

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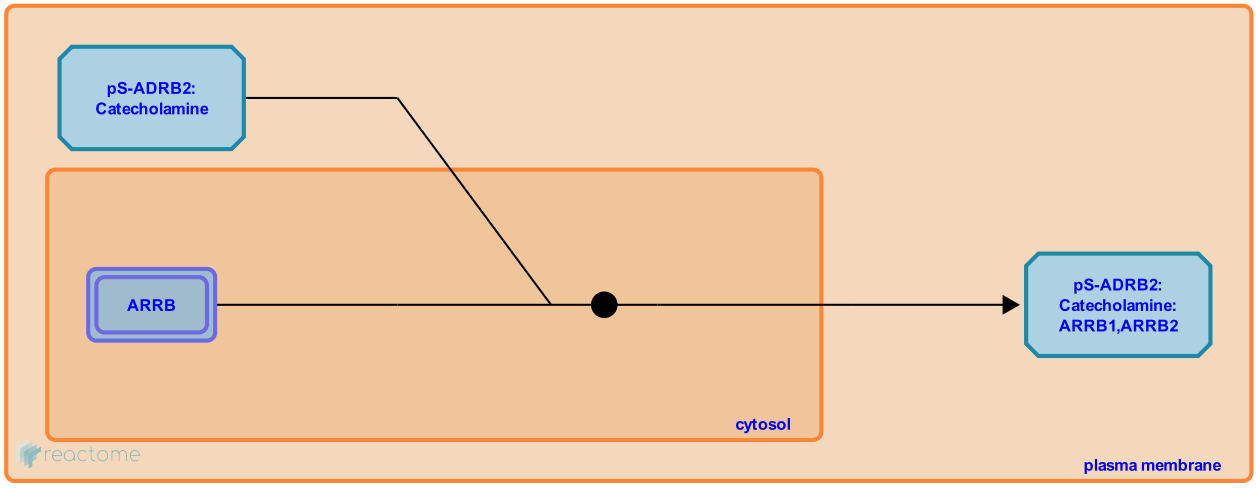
Stable identifier: R-HSA-8852167

Type: binding

Compartments: cytosol, extracellular region, plasma membrane



Two ubiquitously expressed forms of arrestin, arrestin-2 (ARRB1) and arrestin-3 (ARRB2), can bind and desensitize B2AR (Lohse et al. 1990, Attramadal et al. 1992, Sterne-Marr et al. 1993). Unlike visual arrestin, which has 10-fold greater affinity for phosphorylated rather than unphosphorylated rhodopsin, ARRB1 and ARRB2 show only a 2-fold difference in binding levels and are much less selective in their receptor preferences (Gurevich & Gurevich 2006). GRK-mediated receptor phosphorylation followed by arrestin binding and internalization is the classical model for GPCR desensitization. Many GPCRs have been demonstrated to require phosphorylation before they can bind arrestin, but other receptors do not appear to require phosphorylation in order to bind arrestin (see refs. included in Gurevich & Gurevich 2006). In these receptors, spatially close acidic amino acids are thought to provide sites that can bind the arrestin phosphate sensing region.

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Editions

2016-01-11	Authored	Jupe, S.
2016-01-15	Reviewed	Jassal, B.
2016-02-24	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.