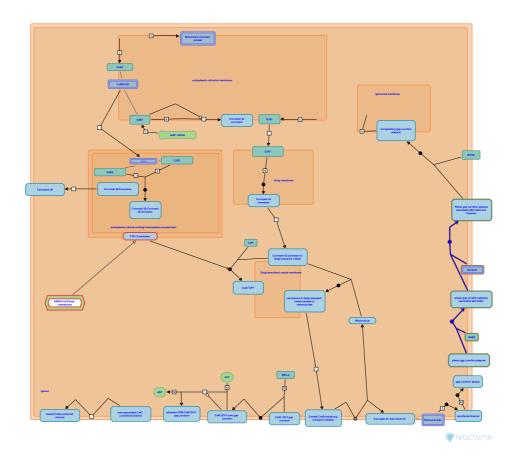


Formation of annular gap junctions



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This is just an excerpt of a full-length report for this pathway. To access the complete report, please download it at the <u>Reactome Textbook</u>.

11/10/2024

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).

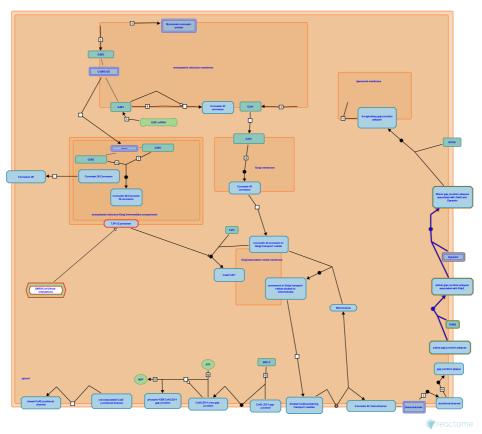
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- Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics*, 33, 3461-3467. A
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This document contains 1 pathway and 2 reactions (see Table of Contents)

Formation of annular gap junctions 7

Stable identifier: R-HSA-196025



Gap junction plaque internalization and the disruption cell communication requires a reorganization of Cx molecular interactions. Proteins including Dab-2, AP-2, Dynamin and Myosin VI associate with gap junction plaques permitting the internalisation of plaques after clathrin association (Piehl et al., 2007). Until now, two kinds of annular gap junctions have been described. The first is a small vesicle like structure which permits gap junction plaque renewal without arrest of functionality (Jordan et al., 2001). The second is a large annular structure, composed primarily of the junctional plaques of two adjacent cells (Jordan et al., 2001; Segretain et al., 2004).

Literature references

Gumpert, A., Segretain, D., Falk, MM., Piehl, M., Denizot, JP., Lehmann, C. (2007). Internalization of Large Double-Membrane Intercellular Vesicles by a Clathrin-dependent Endocytic Process. *Mol Biol Cell*. 7

Editions

2007-01-03

Authored

Gilleron, J., Segretain, D., Falk, MM.

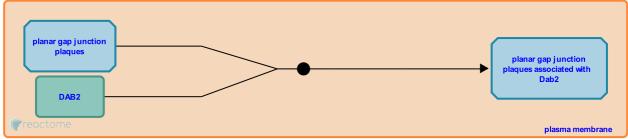
Dab2 is recruited to the junctional plaques 7

Location: Formation of annular gap junctions

Stable identifier: R-HSA-196026

Type: binding

Compartments: plasma membrane



Dab2 is recruited to Cx43-based GJs possibly through a direct interaction between its N-terminal phosphotyrosine binding (PTB) domain and a putative XPXY internalization motif found in the C-terminal tail of Cx43 as well as a number of other connexin family members (Piehl et al., 2007). The distal portion of Dab2 on its opposite end binds the globular N-terminal domain of clathrin heavy chains (Piehl et al., 2007).

Followed by: Dynamin is recruited to the gap junction plaque

Literature references

Gumpert, A., Segretain, D., Falk, MM., Piehl, M., Denizot, JP., Lehmann, C. (2007). Internalization of Large Double-Membrane Intercellular Vesicles by a Clathrin-dependent Endocytic Process. *Mol Biol Cell*. 7

Editions

2007-01-03	Authored	Gilleron, J., Segretain, D., Falk, MM.
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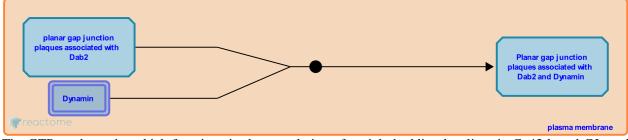
Dynamin is recruited to the gap junction plaque 7

Location: Formation of annular gap junctions

Stable identifier: R-HSA-196017

Type: binding

Compartments: plasma membrane



The GTPase dynamin, which functions in the completion of vesicle budding localizes in Cx43-based GJs and especially invaginating plaques and AGJ vesicles (Piehl et al., 2007).

Preceded by: Dab2 is recruited to the junctional plaques

Literature references

Gumpert, A., Segretain, D., Falk, MM., Piehl, M., Denizot, JP., Lehmann, C. (2007). Internalization of Large Double-Membrane Intercellular Vesicles by a Clathrin-dependent Endocytic Process. *Mol Biol Cell*.

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Table of Contents

Introduction	1
Formation of annular gap junctions	2
Dab2 is recruited to the junctional plaques	3
Dynamin is recruited to the gap junction plaque	4
Table of Contents	5