

Formation of di-heterotetramers of GluN1 (GRIN1) and GluN3A (GRIN3A)

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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](#))

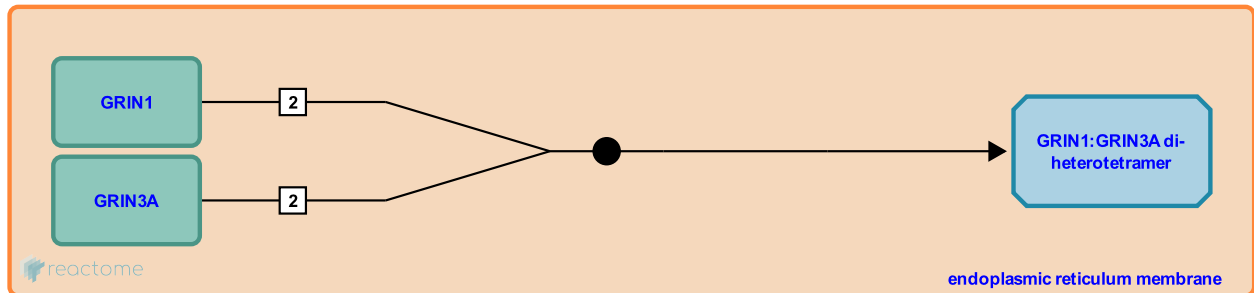
Formation of di-heterotetramers of GluN1 (GRIN1) and GluN3A (GRIN3A) ↗

Stable identifier: R-HSA-9609747

Type: binding

Compartments: endoplasmic reticulum membrane

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GluN3A (GRIN3A) interacts with GluN1 (GRIN1) to form a di-heteromeric NMDA receptor (Perez-Otano et al. 2001, Chatterton et al. 2002). The tetrameric structure of the GluN1:GluN3A (GRIN1:GRIN3A) di-heteromer is inferred from the tetrameric architecture of all known glutamate receptors (Traynelis et al. 2010). GluN3A expression reaches the highest level in early postnatal period and then declines (reviewed by Paoletti et al. 2013). GluN1:GluN3A di-heteromers can be activated by glycine in *Xenopus* oocytes (Smothers and Woodward 2007), and the action of glycine can be greatly enhanced by diminishing the ability of GluN1 subunit to interact with glycine (Awobuluyi et al. 2007, Kvist et al. 2013, Grand et al. 2018).

Editions

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