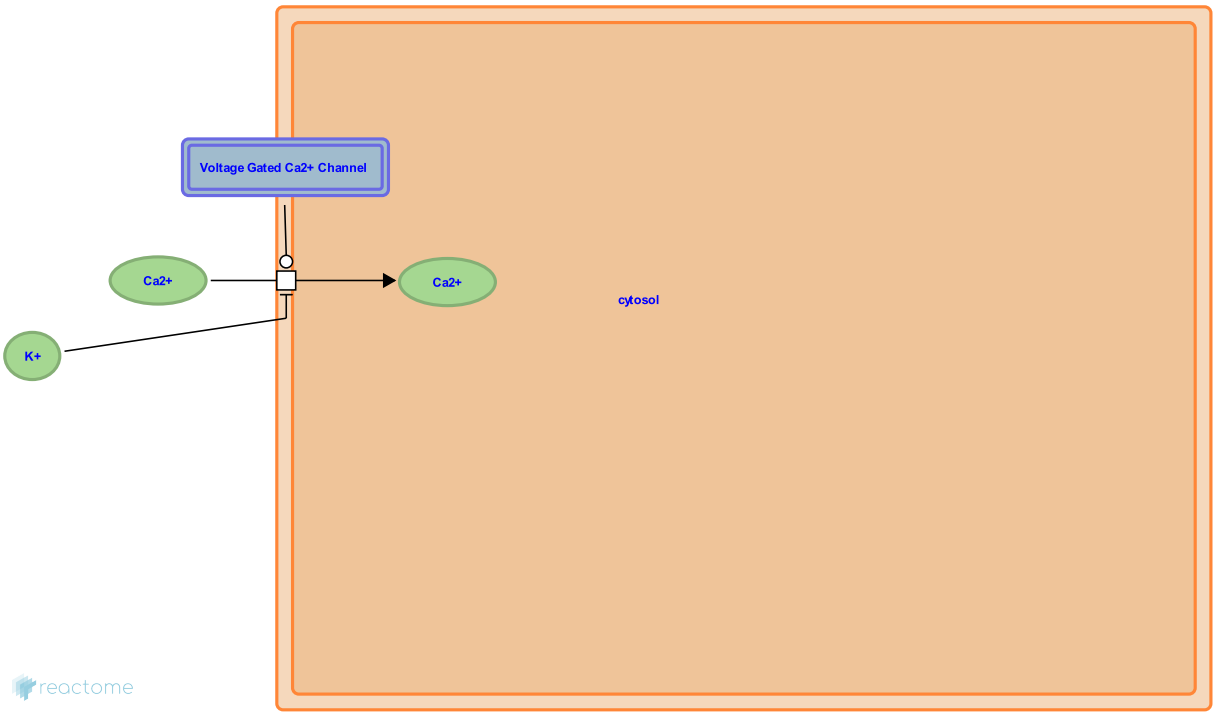
Presynaptic depolarization and calcium channel opening



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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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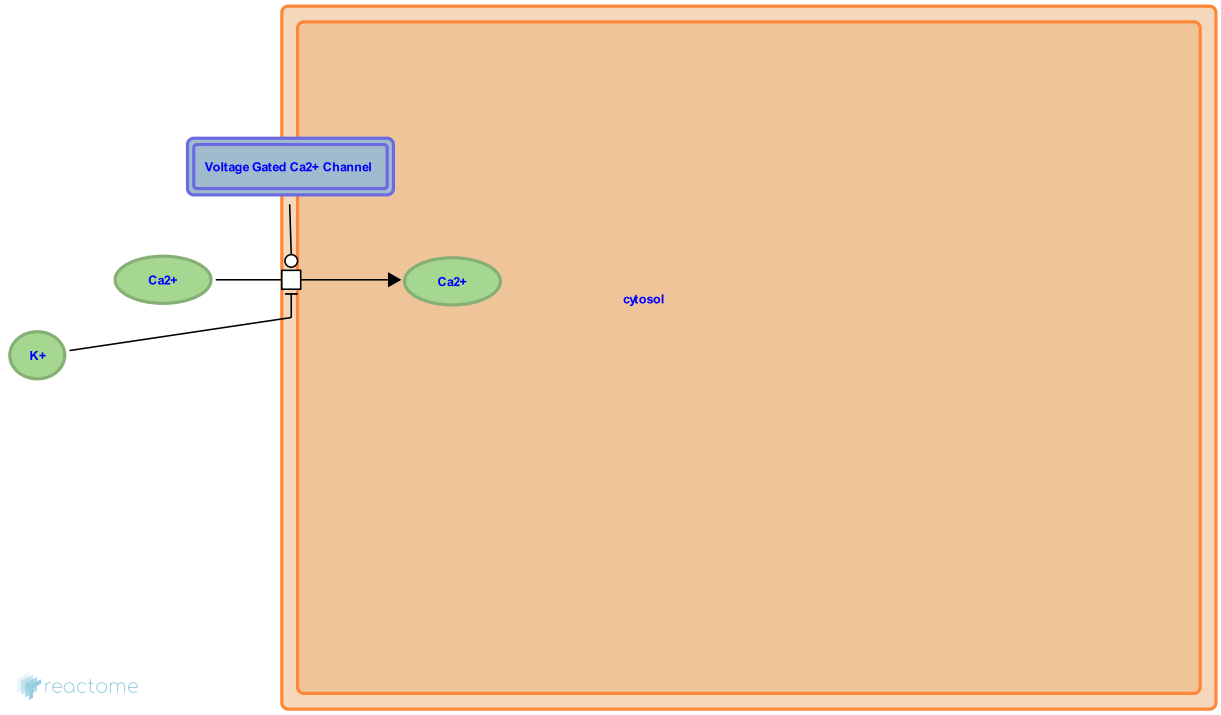
Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

Presynaptic depolarization and calcium channel opening ↗

Stable identifier: R-HSA-112308



Action potentials occur in electrically excitable cells such as neurons, muscles, and endocrine cells. They are initiated by transient opening of voltage dependent sodium channels, causing a rapid, large depolarization of membrane potentials that spread along the axon membrane.
The action potential travels down the axon and reaches the presynaptic terminal depolarizing the membrane in the pre synaptic terminal. The depolarization causes the voltage gated Ca2+ channels to open allowing the influx of Ca2+ that signals the release of neurotransmitter into the synaptic cleft.

Editions

2008-01-14	Authored, Edited	Mahajan, SS.
2008-04-24	Reviewed	Kavalali, E.
2020-01-24	Reviewed	Wen, H.

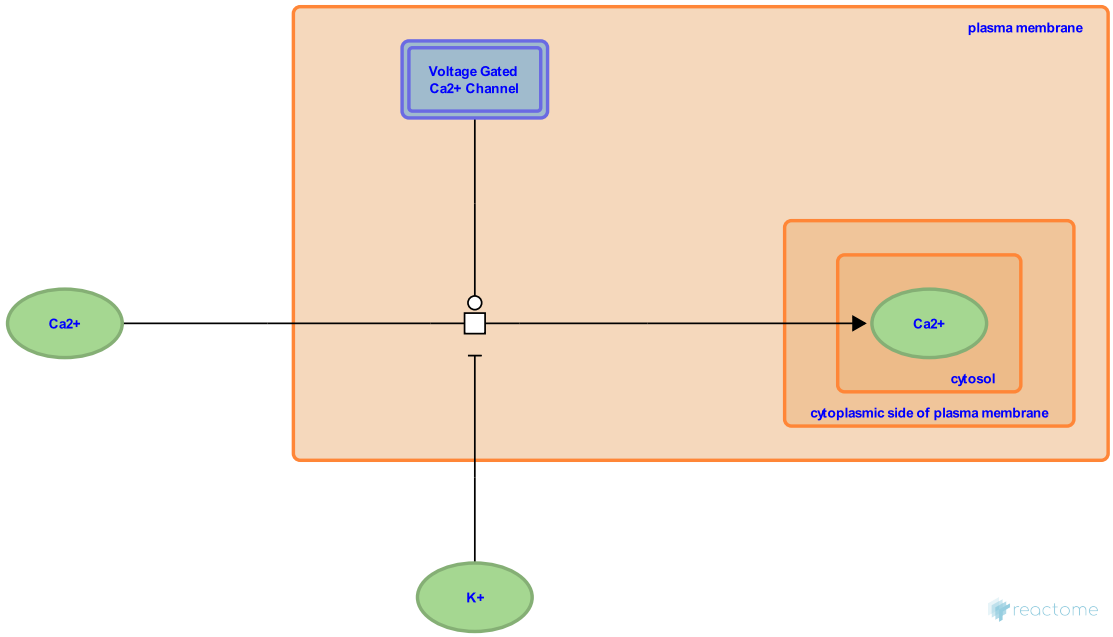
Ca2+ influx through voltage gated Ca2+ channels ↗

Location: Presynaptic depolarization and calcium channel opening

Stable identifier: R-HSA-210420

Type: transition

Compartments: plasma membrane, extracellular region, cytosol



Ca2+ influx from the extracellular space into the presynaptic neuron through the Voltage Gated Ca2+ Channels (VGCC), is dependant on the arrival of an action potential at the synaptic bulb. The vesicle fusion and subsequent release of glutamate into the synapse is triggered by this influx of Ca2+. The VGCCs involved here could be of the N, P/Q or R , and L type.
Hyperpolarization of the cell membrane due to KIR3 potassium channel activity inhibits this influx of Ca2+.

Literature references

Harpold, MM., Lory, P., Williams, ME., Taviaux, S., Diriong, S., Ellis, SB. (1995). Chromosomal localization of the human genes for alpha 1A, alpha 1B, and alpha 1E voltage-dependent Ca2+ channel subunits. *Genomics, 30*, 605-9. ↗

Editions

2008-01-14	Authored	Mahajan, SS.
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