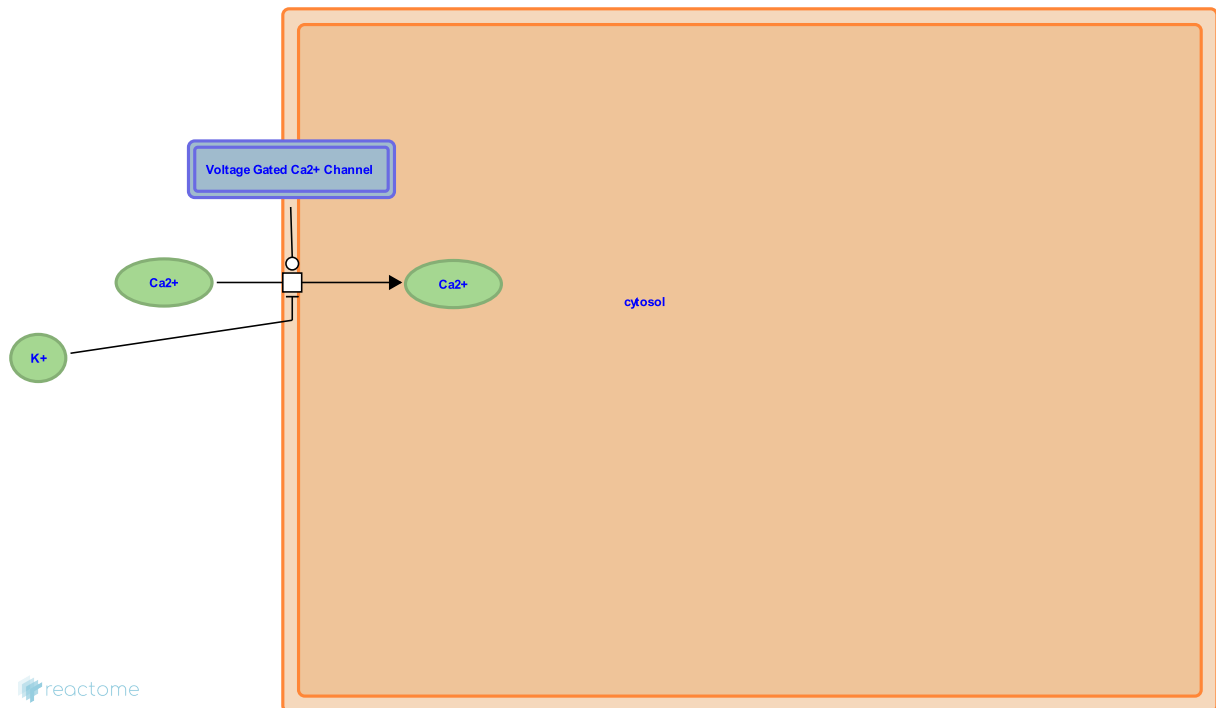


# Presynaptic depolarization and calcium channel opening



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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 [Reactome Textbook](#).

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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.

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

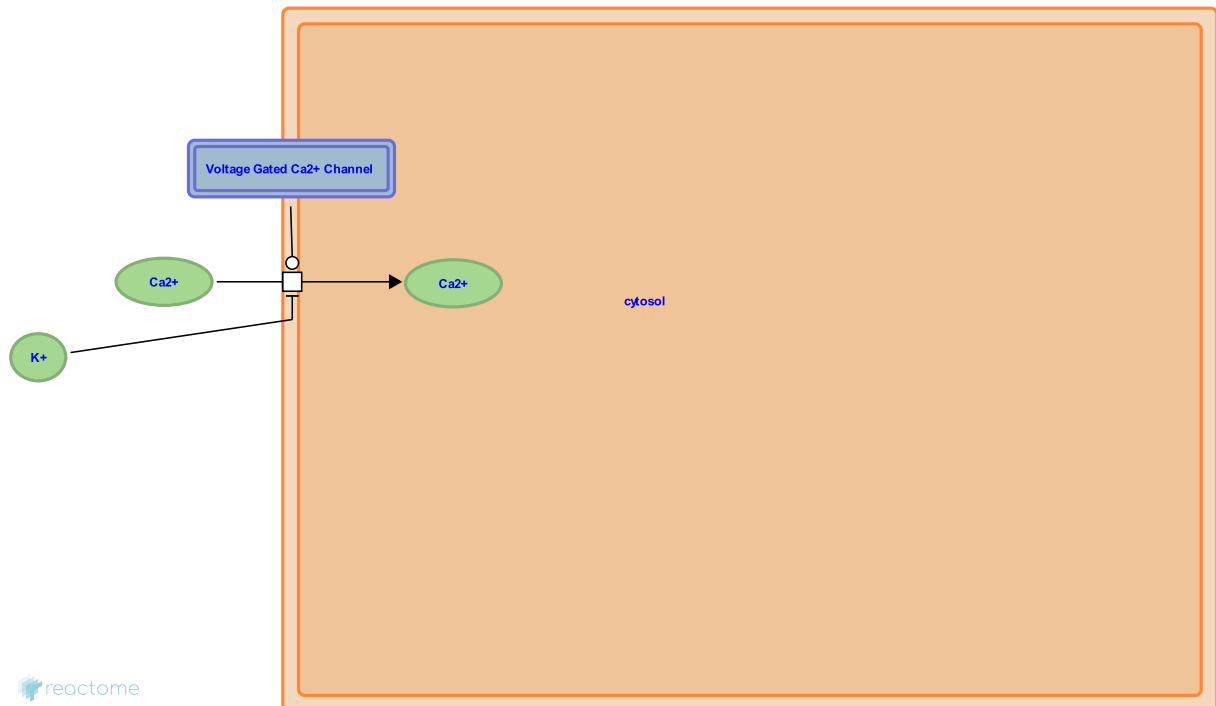
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Reactome database release: 88

This document contains 1 pathway and 1 reaction ([see Table of Contents](#))

## 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 Ca<sup>2+</sup> channels to open allowing the influx of Ca<sup>2+</sup> 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.

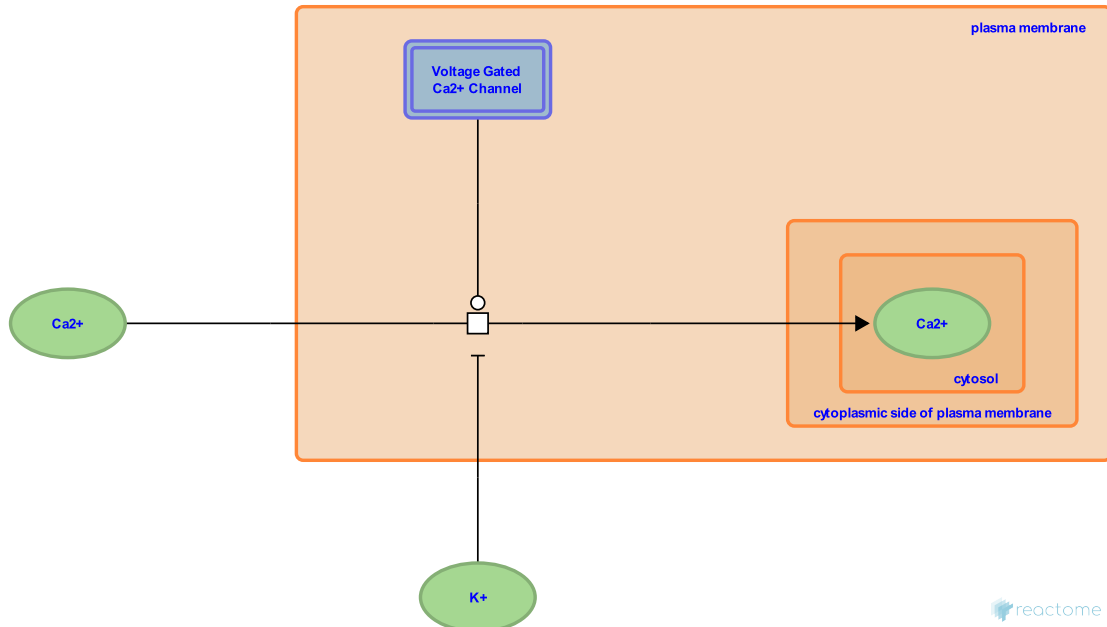
## Ca<sup>2+</sup> influx through voltage gated Ca<sup>2+</sup> channels ↗

**Location:** Presynaptic depolarization and calcium channel opening

**Stable identifier:** R-HSA-210420

**Type:** transition

**Compartments:** plasma membrane, extracellular region, cytosol



Ca<sup>2+</sup> influx from the extracellular space into the presynaptic neuron through the Voltage Gated Ca<sup>2+</sup> 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 Ca<sup>2+</sup>. 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 Ca<sup>2+</sup>.

### 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 Ca<sup>2+</sup> channel subunits. *Genomics*, 30, 605-9. ↗

### Editions

2008-01-14	Authored	Mahajan, SS.
2008-04-24	Reviewed	Kavalali, E.
2009-11-19	Edited	Gillespie, ME.

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