UNC79:UNC80:NALCN transports Na+ extracellular region to cytosol

He, L., Jassal, B.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.

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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**


Reactome database release: 78

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https://reactome.org
The sodium leak channel non-selective protein NALCN, a nonselective cation channel, forms the background Na+ leak conductance and controls neuronal excitability (Lu et al. 2007, Ren 2011). Mice with mutant NALCN have a severely disrupted respiratory rhythm and die within 24 hours of birth. Calcium (Ca2+) influences neuronal excitability via the NALCN:UNC79:UNC80 complex, with high Ca2+ concentrations inhibiting transport of Na+ (Lu et al. 2010). Mutations in human NALCN lead to complex neurodevelopmental syndromes, including infantile hypotonia with psychomotor retardation and characteristic facies (IHPRF) and congenital contractures of limbs and face, hypotonia and developmental delay (CLIFAHDD) (Bouasse et al. 2019).

**Literature references**


**Editions**

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