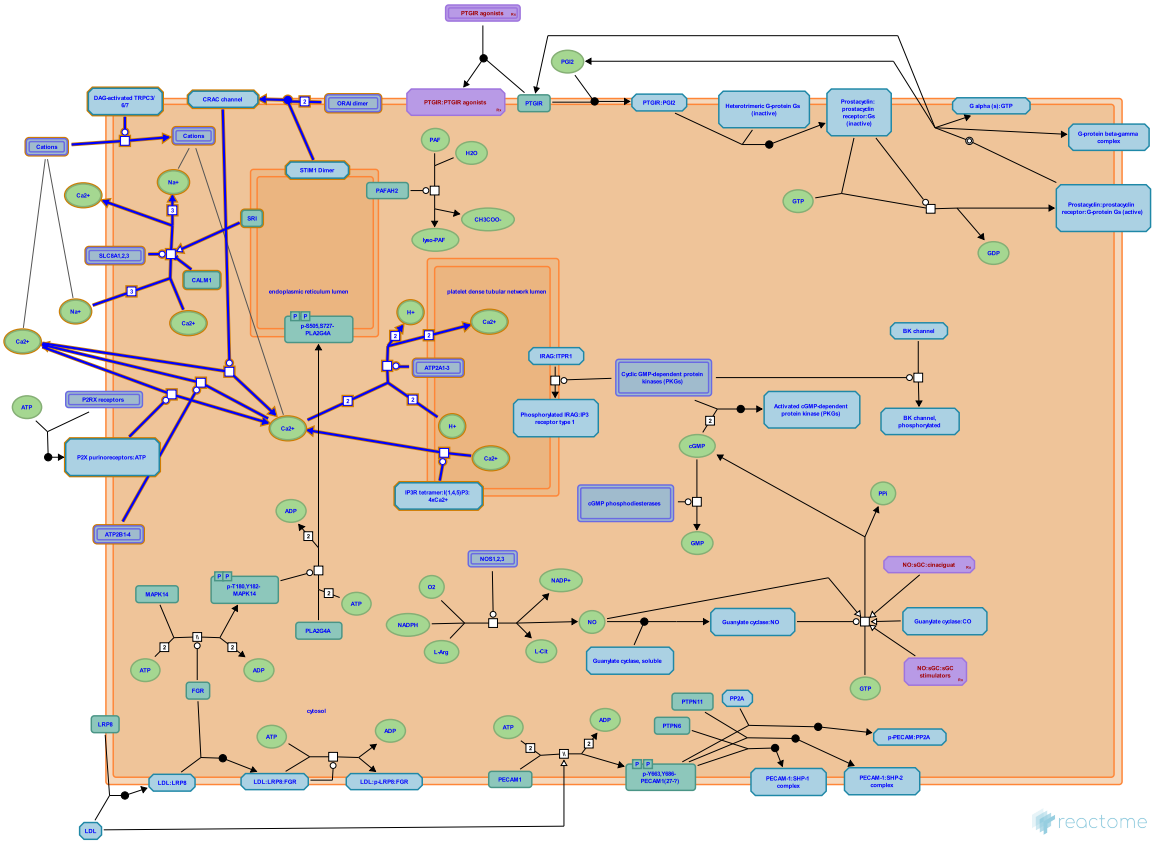
Platelet calcium homeostasis



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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.

16/05/2024

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).

Literature references

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

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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. ↗

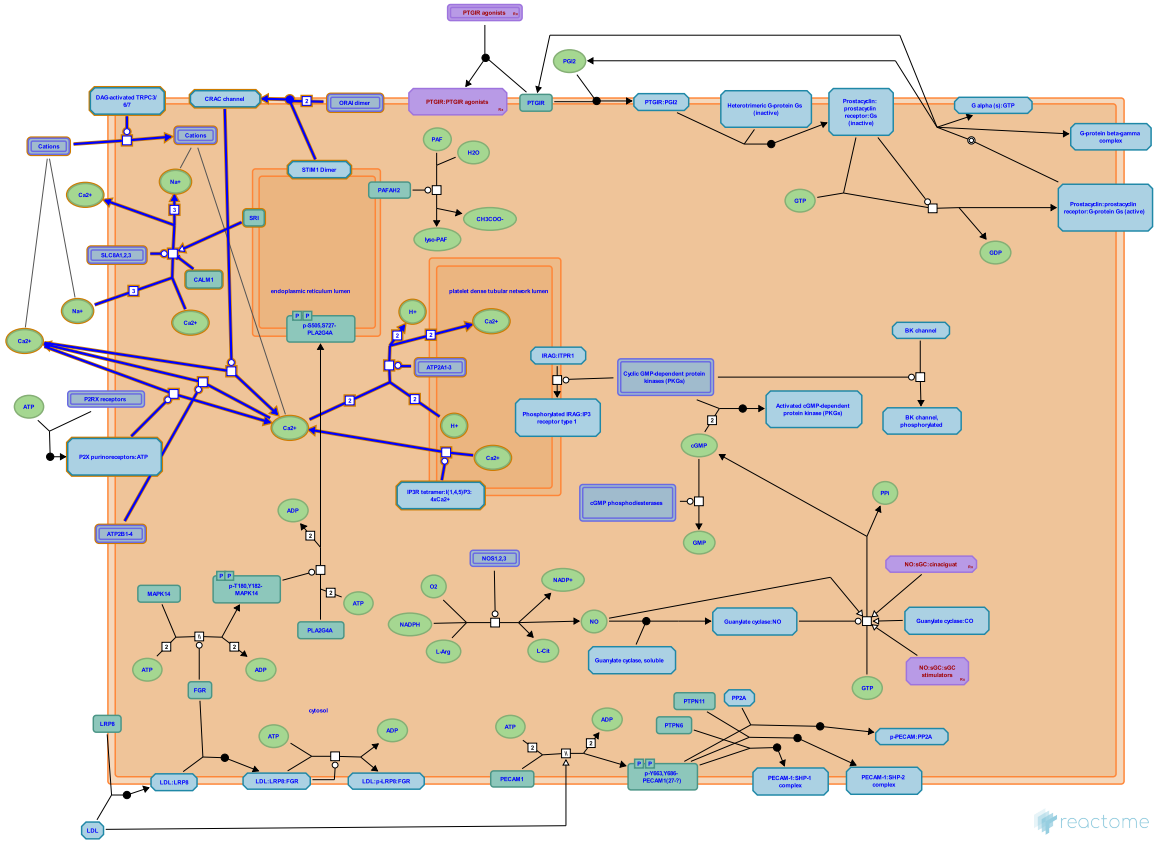
Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

Reactome database release: 88

This document contains 3 pathways (see Table of Contents)

Platelet calcium homeostasis ↗

Stable identifier: R-HSA-418360



Ca2+ homeostasis is controlled by processes that elevate or counter the elevation of cytosolic Ca2+. During steady state conditions, cytoplasmic Ca2+ is reduced by the accumulation of Ca2+ in intracellular stores and by Ca2+ extrusion. The primary intracellular calcium store in platelets is the dense tubular system, the equivalent of the ER system in other cell types. Ca2+ is extruded by Ca2+-ATPases including plasma membrane Ca2+ ATPases (PMCAs) and sarco/endoplasmic reticulum Ca2+ -ATPase isoforms (SERCAs).

Activation of non- excitable cells involves the agonist-induced elevation of cytosolic Ca2+, an essential process for platelet activation. It occurs through Ca2+ release from intracellular stores and Ca2+ entry through the plasma membrane. Ca2+ store release involves phospholipase C (PLC)-mediated production of inositol-1,4,5-trisphosphate (IP3), which in turn stimulates IP3 receptor channels to release Ca2+ from intracellular stores. This is followed by Ca2+ entry into the cell through plasma membrane calcium channels, a process referred to as store-operated calcium entry (SOCE). Stromal interaction molecule 1 (STIM1), a Ca2+ sensor molecule in intracellular stores, and the four transmembrane channel protein Orai1 are the key players in platelet SOCE. Other major Ca2+ entry mechanisms are mediated by the direct receptor-operated calcium (ROC) channel, P2X1 and transient receptor potential channels (TRPCs).

Literature references

Varga-Szabo, D., Braun, A., Nieswandt, B. (2009). Calcium signaling in platelets. *J Thromb Haemost, 7*, 1057-66. ↗

Jardin, I., López, JJ., Rosado, JA., Salido, GM., Pariente, JA. (2008). Intracellular calcium release from human platelets: different messengers for multiple stores. *Trends Cardiovasc Med, 18*, 57-61. ↗

Editions

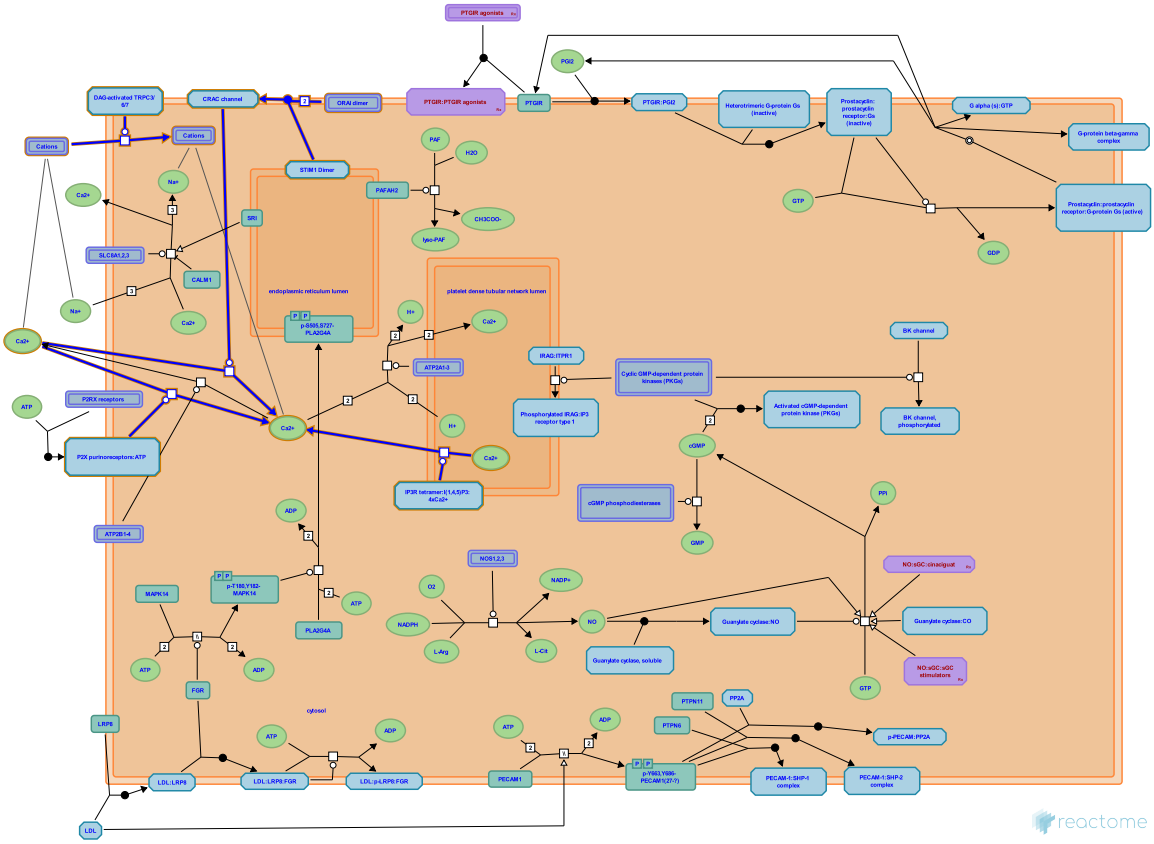
2009-06-03	Authored	Akkerman, JW.
2010-06-07	Edited	Jupe, S.
2010-06-07	Reviewed	Kunapuli, SP.

Elevation of cytosolic Ca2+ levels ↗

Location: Platelet calcium homeostasis

Stable identifier: R-HSA-139853

Compartments: cytosol



Activation of non- excitable cells involves the agonist-induced elevation of cytosolic Ca2+, an essential process for platelet activation. It occurs through Ca2+ release from intracellular stores and Ca2+ entry through the plasma membrane. Ca2+ store release involves phospholipase C (PLC)-mediated production of inositol-1,4,5-trisphosphate (IP3), which in turn stimulates IP3 receptor channels to release Ca2+ from intracellular stores. This is followed by Ca2+ entry into the cell through plasma membrane calcium channels, a process referred to as store-operated calcium entry (SOCE). Stromal interaction molecule 1 (STIM1), a Ca2+ sensor molecule in intracellular stores, and the four transmembrane channel protein Orai1 are the key players in platelet SOCE. Other major Ca2+ entry mechanisms are mediated by the direct receptor-operated calcium (ROC) channel, P2X1 and transient receptor potential channels (TRPCs).

Literature references

Feske, S. (2007). Calcium signalling in lymphocyte activation and disease. *Nat Rev Immunol, 7*, 690-702. ↗

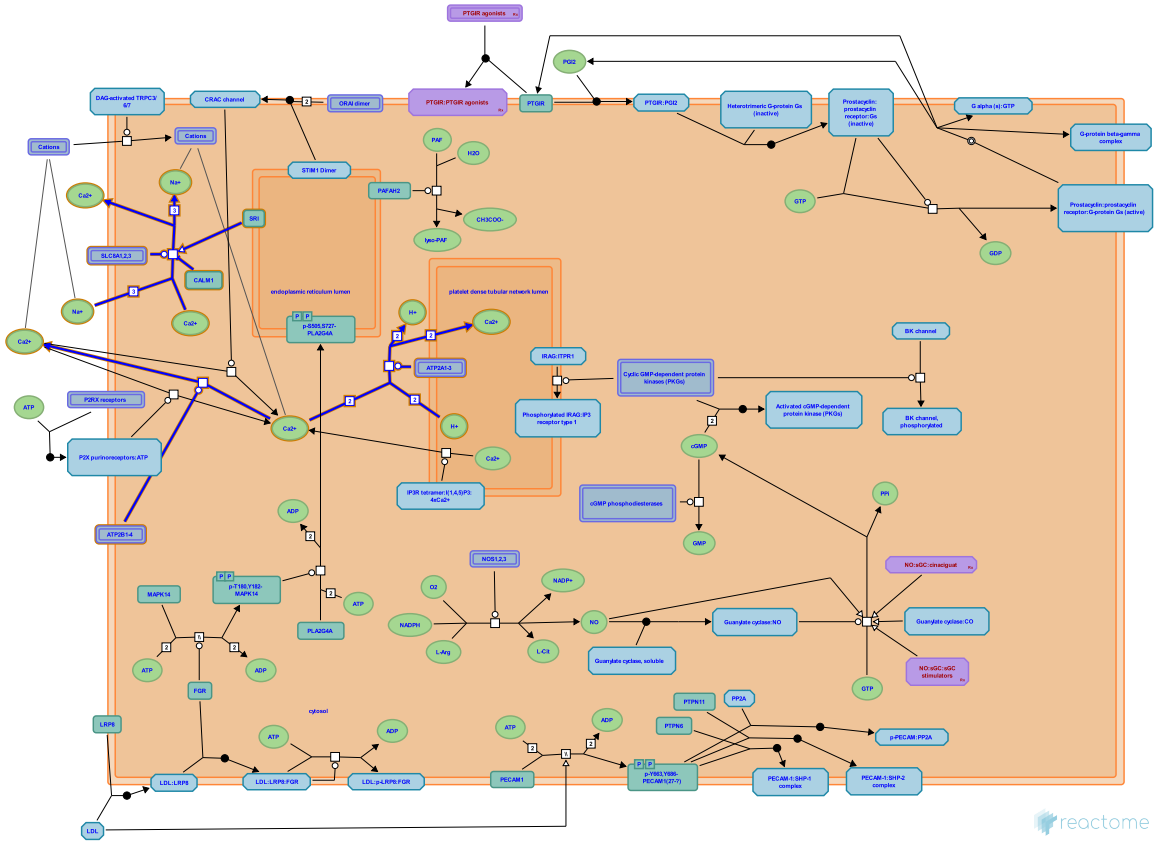
Varga-Szabo, D., Braun, A., Nieswandt, B. (2009). Calcium signaling in platelets. *J Thromb Haemost, 7*, 1057-66. ↗

Jardin, I., López, JJ., Rosado, JA., Salido, GM., Pariente, JA. (2008). Intracellular calcium release from human platelets: different messengers for multiple stores. *Trends Cardiovasc Med, 18*, 57-61. ↗

Reduction of cytosolic Ca++ levels ↗

Location: Platelet calcium homeostasis

Stable identifier: R-HSA-418359



During steady state conditions, cytoplasmic [Ca2+] is reduced by the accumulation of Ca2+ in intracellular stores and Ca2+ extrusion.

Literature references

Jardin, I., López, JJ., Rosado, JA., Salido, GM., Pariente, JA. (2008). Intracellular calcium release from human platelets: different messengers for multiple stores. *Trends Cardiovasc Med, 18*, 57-61. ↗

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2009-06-03	Authored	Akkerman, JW.
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