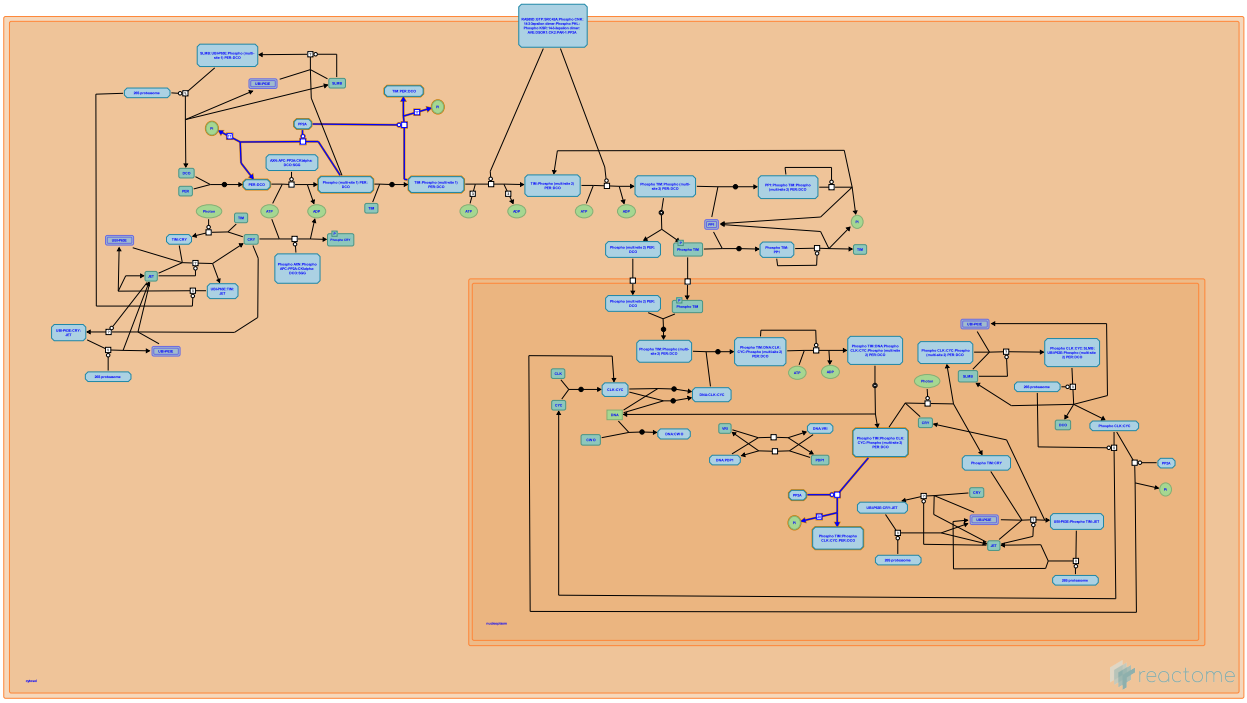
Dephosphorylation of PER



Edery, I., Williams, MG.

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

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.

01/05/2024

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. ↗

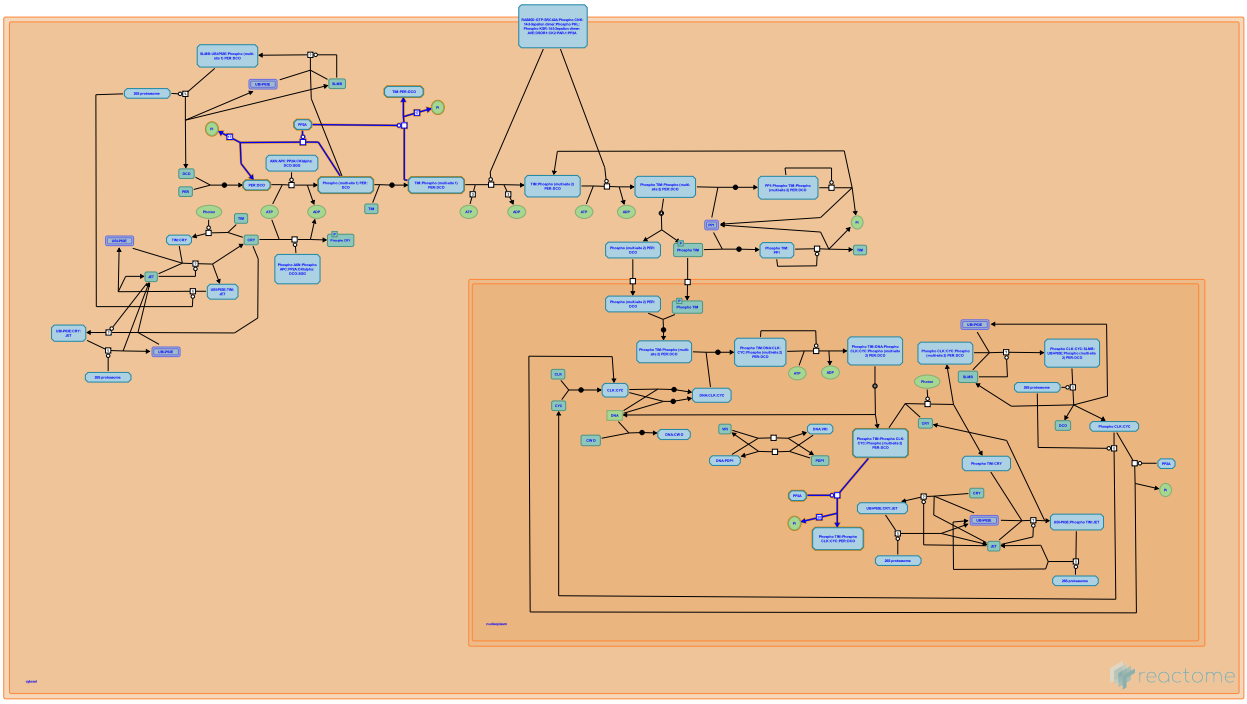
Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

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. ↗

Dephosphorylation of PER ↗

Stable identifier: R-DME-432620



Spatzle (SPZ) dimer binding leads to Toll (TL) receptor homodimerisation and activation.

Literature references

Hardin, PE. (2005). The circadian timekeeping system of Drosophila. *Curr Biol, 15*, R714-22. ↗

Editions

2009-08-13	Authored	Williams, MG.
2009-08-14	Edited	Williams, MG.
2010-07-06	Reviewed	Edery, I.

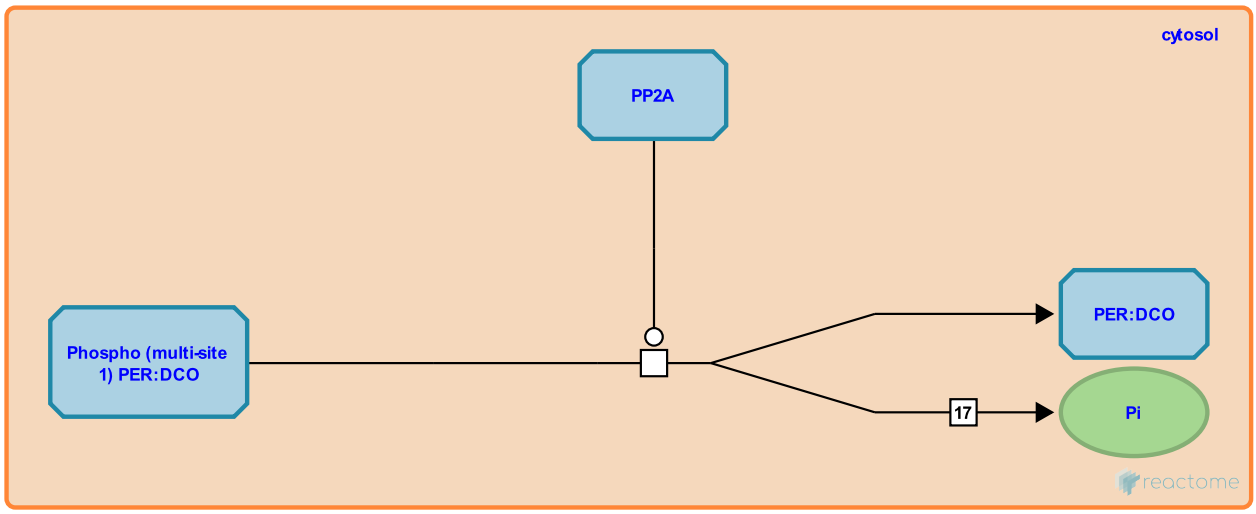
Cytosolic PP2A dephosphorylates phosphorylated PER complexed with DCO ↗

Location: Dephosphorylation of PER

Stable identifier: R-DME-432556

Type: transition

Compartments: cytosol



The protein phosphatase PP2A, formed of PP2A-29B:Microtubule star (MTS):Twins (TWS), helps stabilise hyperphosphorylated Period (PER), within the PER:DCO complex, by dephosphorylating it and so prevent it from degradation. PP2A likely dephosphorylates the site phosphorylated by Casein kinase 1epsilon orthologue, Discs Overgrown (DCO) as it stabilises PER.

Literature references

Fang, Y., Sehgal, A., Sathyanarayanan, S. (2007). Post-translational regulation of the Drosophila circadian clock requires protein phosphatase 1 (PP1). *Genes Dev, 21*, 1506-18. ↗

Vanselow, JT., Edery, I., Kramer, A., Chiu, JC. (2008). The phospho-occupancy of an atypical SLIMB-binding site on PERIOD that is phosphorylated by DOUBLETIME controls the pace of the clock. *Genes Dev, 22*, 1758-72. ↗

Xiao, R., Sehgal, A., Sathyanarayanan, S., Zheng, X. (2004). Posttranslational regulation of Drosophila PERIOD protein by protein phosphatase 2A. *Cell, 116*, 603-15. ↗

Editions

2009-08-13	Authored	Williams, MG.
2010-07-06	Reviewed	Edery, I.
2014-05-20	Edited	Williams, MG.

Cytosolic PP2A dephosphorylates phosphorylated PER complexed with TIM and DCO

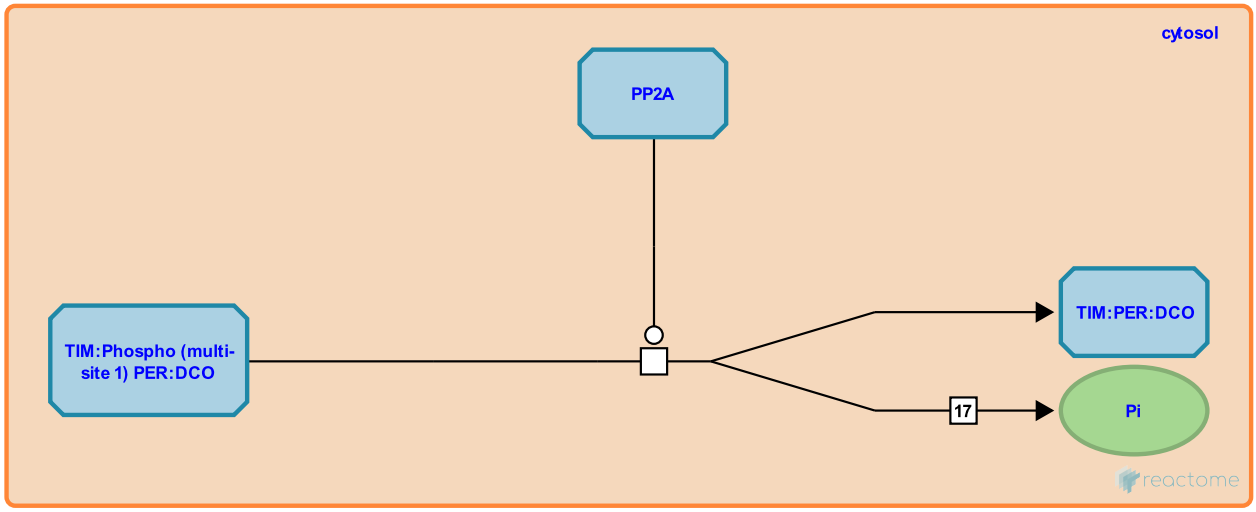
↗

Location: Dephosphorylation of PER

Stable identifier: R-DME-538822

Type: transition

Compartments: cytosol



The protein phosphatase PP2A, formed of PP2A-29B:Microtubule star (MTS):Twins (TWS), helps stabilise hyperphosphorylated Period (PER), within the PER:DCO complex, by dephosphorylating it and so prevent it from degradation. PP2A likely dephosphorylates the site phosphorylated by Casein kinase 1epsilon orthologue, Discs Overgrown (DCO) as it stabilises PER.

Literature references

Fang, Y., Sehgal, A., Sathyanarayanan, S. (2007). Post-translational regulation of the Drosophila circadian clock requires protein phosphatase 1 (PP1). *Genes Dev, 21*, 1506-18. ↗

Vanselow, JT., Edery, I., Kramer, A., Chiu, JC. (2008). The phospho-occupancy of an atypical SLIMB-binding site on PERIOD that is phosphorylated by DOUBLETIME controls the pace of the clock. *Genes Dev, 22*, 1758-72. ↗

Xiao, R., Sehgal, A., Sathyanarayanan, S., Zheng, X. (2004). Posttranslational regulation of Drosophila PERIOD protein by protein phosphatase 2A. *Cell, 116*, 603-15. ↗

Editions

2010-03-08	Authored	Williams, MG.
2010-07-06	Reviewed	Edery, I.
2014-05-20	Edited	Williams, MG.

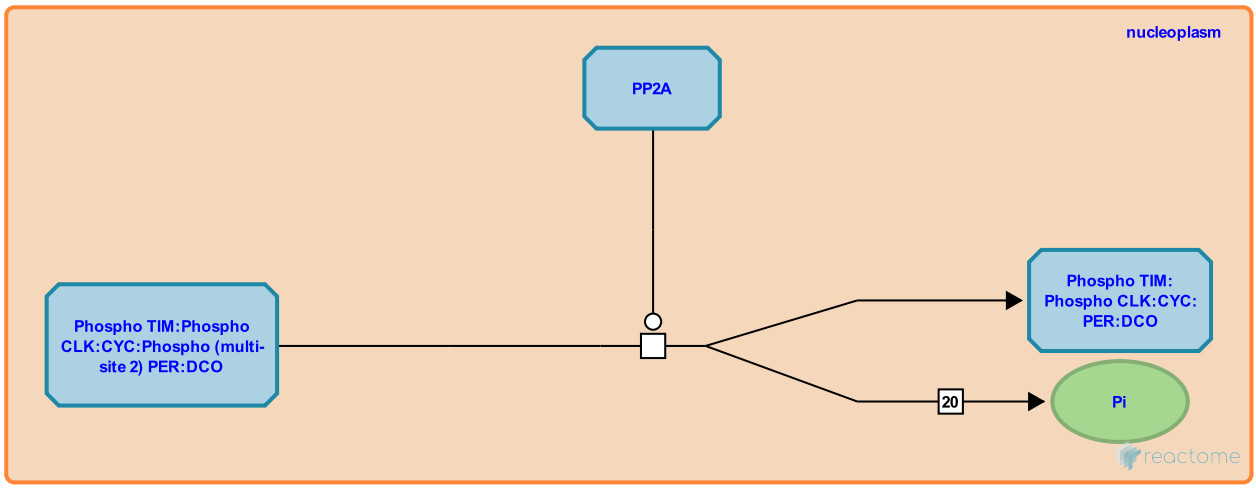
Nuclear PP2A dephosphorylates phosphorylated PER ↗

Location: Dephosphorylation of PER

Stable identifier: R-DME-432527

Type: transition

Compartments: nucleoplasm



The protein phosphatase PP2A, formed of PP2A-29B:Microtubule star (MTS):Widerborst (WDB), helps stabilise hyperphosphorylated Period (PER) by dephosphorylating it and so prevent it from degradation. PP2A also seems to speed up nuclear import of PER. This is probably a secondary effect of stabilising PER.

Literature references

Vanselow, JT., Edery, I., Kramer, A., Chiu, JC. (2008). The phospho-occupancy of an atypical SLIMB-binding site on PERIOD that is phosphorylated by DOUBLETIME controls the pace of the clock. *Genes Dev, 22*, 1758-72. ↗

Xiao, R., Sehgal, A., Sathyanarayanan, S., Zheng, X. (2004). Posttranslational regulation of Drosophila PERIOD protein by protein phosphatase 2A. *Cell, 116*, 603-15. ↗

Editions

2009-08-13	Authored	Williams, MG.
2010-07-06	Reviewed	Edery, I.
2014-05-20	Edited	Williams, MG.

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