Inactivation of Wee1 kinase

Gillespie, ME., Hunter, T., Watanabe, N.

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

The contents of this document may be freely copied and distributed in any media, provided the authors, plus the institutions, are credited, as stated under the terms of Creative Commons Attribution 4.0 International (CC BY 4.0) License. For more information see our license.

02/05/2024

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. ↗

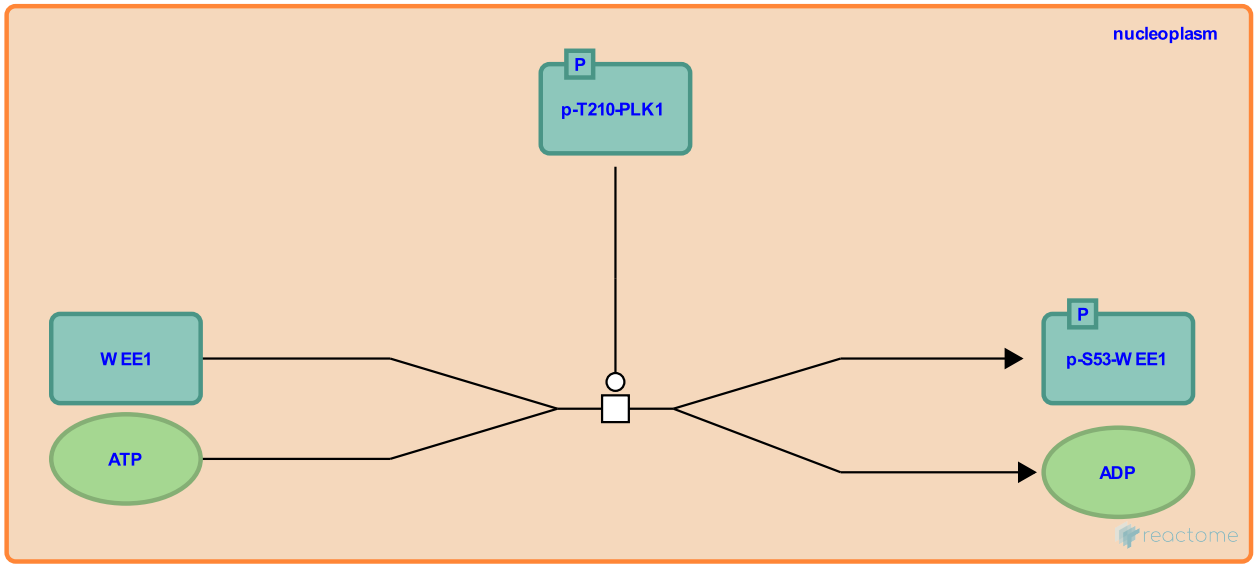
Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

Inactivation of Wee1 kinase ↗

Stable identifier: R-HSA-156699

Type: transition

Compartments: nucleoplasm



*Plk1 is shown to phosphorylate Wee1A, an event that is likely critical for recognition and ubiquitination of Wee1A by SCF and therefore for the subsequent degradation of Wee1A . **Plk1 phosphorylates Wee1A at S53, creating the second phosphodegron, PD53. ** Evidence also exists in budding yeast that the budding yeast polo homolog Cdc5 directly phosphorylates and down-regulate the budding yeast Wee1 ortholog Swe1. Thus, polo kinase-dependent phosphorylation and degradation of Wee1A (or Swe1) is likely conserved throughout evolution and is critical for normal mitotic entry.

Literature references

Nishihara, Y., Taniguchi, M., Arai, H., Watanabe, N., Osada, H., Hunter, T. et al. (2004). M-phase kinases induce phospho-dependent ubiquitination of somatic Wee1 by SCFbeta-TrCP. *Proc Natl Acad Sci U S A, 101,* 4419-24. ↗

Park, JE., Lee, KS., Veenstra, TD., Cho, YW., Sakchaisri, K., Yu, LR. et al. (2004). Coupling morphogenesis to mitotic entry. *Proc Natl Acad Sci U S A, 101,* 4124-9. ↗

Editions

2008-05-08	Authored	Hunter, T., Watanabe, N.
2024-03-06	Edited	Gillespie, ME.