

Deacetylation of cleaved cohesin

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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

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- Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics*, 33, 3461-3467. [↗](#)
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Reactome database release: 70

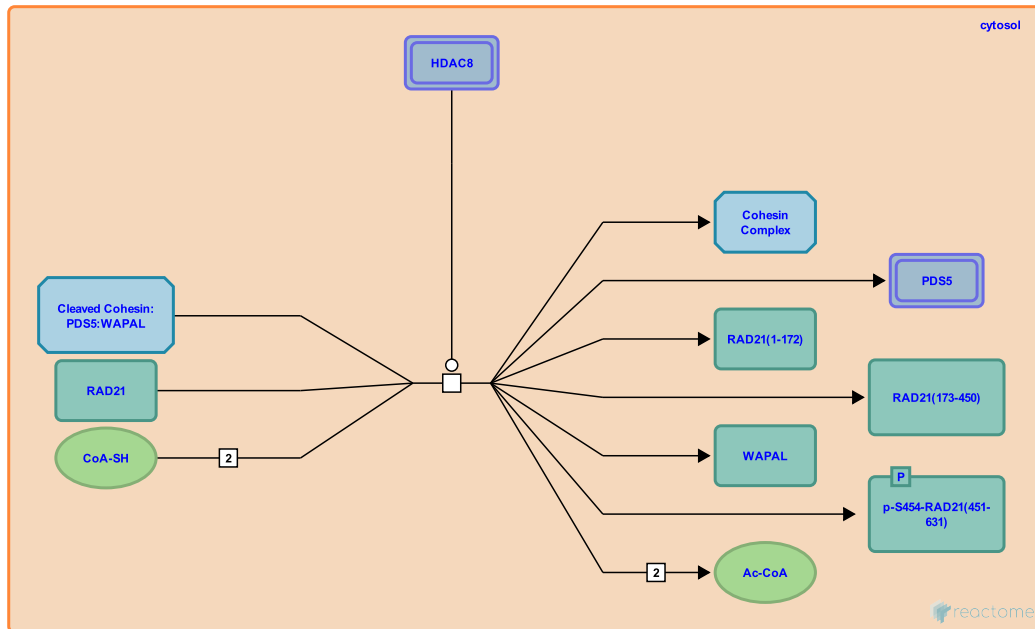
This document contains 1 reaction ([see Table of Contents](#))

Deacetylation of cleaved cohesin ↗

Stable identifier: R-HSA-2545203

Type: transition

Compartments: cytosol



Histone deacetylase HDAC8 deacetylates SMC3 cohesin subunit. SMC3 deacetylation promotes dissociation of cleaved RAD21 fragments from other cohesin proteins and their replacement with intact RAD21, thereby allowing restoration of the cohesin complex (Deardorff et al. 2012). HDAC8 mutations, as well as mutations in NIPBL, SMC1A and SMC3, can cause Cornelia de Lange syndrome (Deardorff et al. 2012).

Literature references

Deardorff, MA., Bando, M., Nakato, R., Watrin, E., Itoh, T., Minamino, M. et al. (2012). HDAC8 mutations in Cornelia de Lange syndrome affect the cohesin acetylation cycle. *Nature*, 489, 313-7. ↗

Editions

2012-10-02	Authored	Orlic-Milacic, M.
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