

B-WICH:histone acetyltransferase acetylates histone H3 at lysine-9

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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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Reactome database release: 88

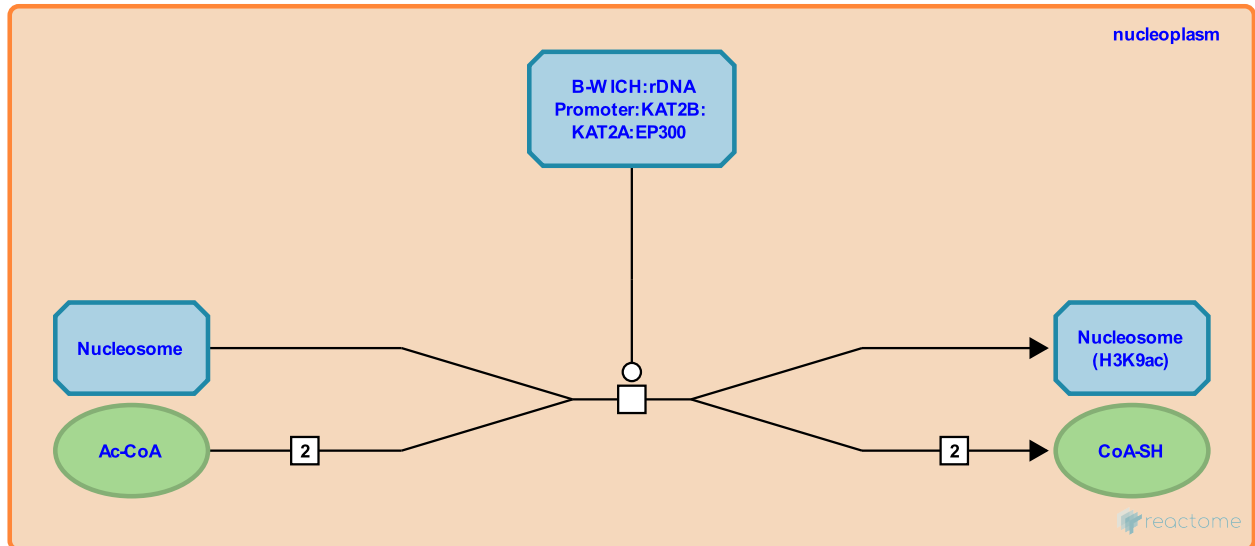
This document contains 1 reaction ([see Table of Contents](#))

B-WICH:histone acetyltransferase acetylates histone H3 at lysine-9 [↗](#)

Stable identifier: R-HSA-5250938

Type: transition

Compartments: nucleoplasm



Histone acetyltransferases recruited by the B-WICH complex acetylate histone H3 at lysine-9. Knockdown of the BAZ1B (WSTF) and MYOIC components of B-WICH cause a loss of histone acetyltransferases KAT2B (PCAF), KAT2A (GCN5), and EP300 (p300) and a reduction of acetylated histone H3. Knockdown of KAT2B (PCAF) causes a reduction in acetylation of histone H3 at lysine-9, leading to reduced rRNA synthesis levels (Sarshad et al. 2013, Shen et al. 2013).

Literature references

Lyu, G., Zhou, T., Zhu, Q., Shen, M., Zhang, F., Gao, Q. et al. (2013). The chromatin remodeling factor CSB recruits histone acetyltransferase PCAF to rRNA gene promoters in active state for transcription initiation. *PLoS ONE*, 8, e62668. [↗](#)

Böhm, S., Sarshad, A., Percipalle, P., Fomproix, N., Sadeghifar, F., Vintermist, A. et al. (2013). Nuclear myosin 1c facilitates the chromatin modifications required to activate rRNA gene transcription and cell cycle progression. *PLoS Genet.*, 9, e1003397. [↗](#)

Editions

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