B-WICH complex positively regulates rRNA expression

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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.

14/11/2022
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


Reactome database release: 82

This document contains 1 pathway and 3 reactions (see Table of Contents)
B-WICH complex positively regulates rRNA expression

Stable identifier: R-HSA-5250924

Compartments: nucleoplasm

The B-WICH complex is a large 3 Mdalton complex containing SMARCA5 (SNF2H), BAZ1B (WSTF), ERCC6 (CSB), MYO1C (Nuclear myosin 1c), SF3B1, DEK, MYBBP1A, and DDX21 (Cavellán et al. 2006, Percipalle et al. 2006, Vintermist et al. 2001, Sarshad et al. 2013, Shen et al. 2013, reviewed in Percipalle and Farrants 2006). B-WICH is found at active rRNA genes as well as at 5S rRNA and 7SL RNA genes. B-WICH appears to remodel chromatin and recruit histone acetyltransferases that modify histones to transcriptionally active states.

Literature references


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B-WICH complex binds rDNA promoter

Location: B-WICH complex positively regulates rRNA expression

Stable identifier: R-HSA-5250947

Type: binding

Compartments: nucleoplasm

Nuclear myosin 1 is positively regulated by the glycogen synthase kinase (GSK3beta) by direct phosphorylation (Sarshad et al. 2014). NM1 phosphorylation is a requirement for NM1 to bind to the chromatin. In the absence of GSK3beta dependent phosphorylation NM1 is polyubiquitinated by the E3 ligase UBR5 and degraded by the proteasome and rRNA synthesis is not activated (Sarshad et al. 2014).

Active rRNA genes are bound by the B-WICH multiprotein complex (Cavellan et al. 2006, Percipalle et al. 2006). B-WICH binds the promoter region of the gene (Percipalle et al. 2006, Sarshad et al. 2013). The MYO1C component of the B-WICH complex binds chromatin and interacts with SMARCA5. Binding causes 200 bp of chromatin at the promoter to adopt a more open configuration and contributes to epigenetic modifications compatible with transcription activation (Vintermist et al. 2011, Sarshad et al. 2013). At the rRNA gene promoter the SMARCA5-MYOIC interaction is excluded when MYOIC interacts with actin in complex with RNA polymerase I (Sarshad et al. 2013). Binding of MYOIC to chromatin is regulated by GSK3beta-dependent phosphorylation that targets the MYOIC chromatin binding domain (Sarshad et al. 2014). Binding of MYOIC to the RNA polymerase I is partly mediated via phosphorylated TIF1A (Philimonenko et al. 2004). Binding of B-WICH to rRNA genes requires MYOIC to be recruited to active rRNA genes and this mechanism appears to be a requirement to activate and maintain transcription by RNA polymerase I (Percipalle et al. 2006, Sarshad et al. 2013, Sarshad et al. 2014, reviewed in Sarshad and Percipalle 2014).

Followed by: B-WICH recruits histone acetyltransferases

Literature references


**Editions**

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B-WICH recruits histone acetyltransferases

**Location:** B-WICH complex positively regulates rRNA expression

**Stable identifier:** R-HSA-5250930

**Type:** binding

**Compartments:** nucleoplasm

Direct interactions between BAZ1B (WSTF) and histone acetyltransferases KAT2B, KAT2A, and EP300 are weak (Vintermist et al. 2011) so the acetyltransferases may interact with other subunits of B-WICH or with proteins not in the B-WICH complex. The ERCC6 (CSB) component of B-WICH and MYOIC interact with KAT2B (PCAF) (Sarshad et al. 2013, Shen et al. 2013). The histone acetyltransferases are believed to acetylinate histone H3 at lysine9 in rDNA since this modification is reduced in WSTF and MYOIC knockdown cells (Vintermist et al. 2011, Sarshad et al. 2013). Knockdown of KAT2B causes loss of acetylation on histone H4 and on histone H3 at lysine9 (Shen et al. 2013).

**Preceded by:** B-WICH complex binds rDNA promoter

**Followed by:** B-WICH:histone acetyltransferase acetylates histone H3 at lysine-9

**Literature references**


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

Location: B-WICH complex positively regulates rRNA expression

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 MYO1C 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).

Preceded by: B-WICH recruits histone acetyltransferases

Literature references


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