Crebbp acetylates replicative histone H2B, H3, H4

Jupe, S., Karagiannis, T.

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

27/01/2021

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. ↗

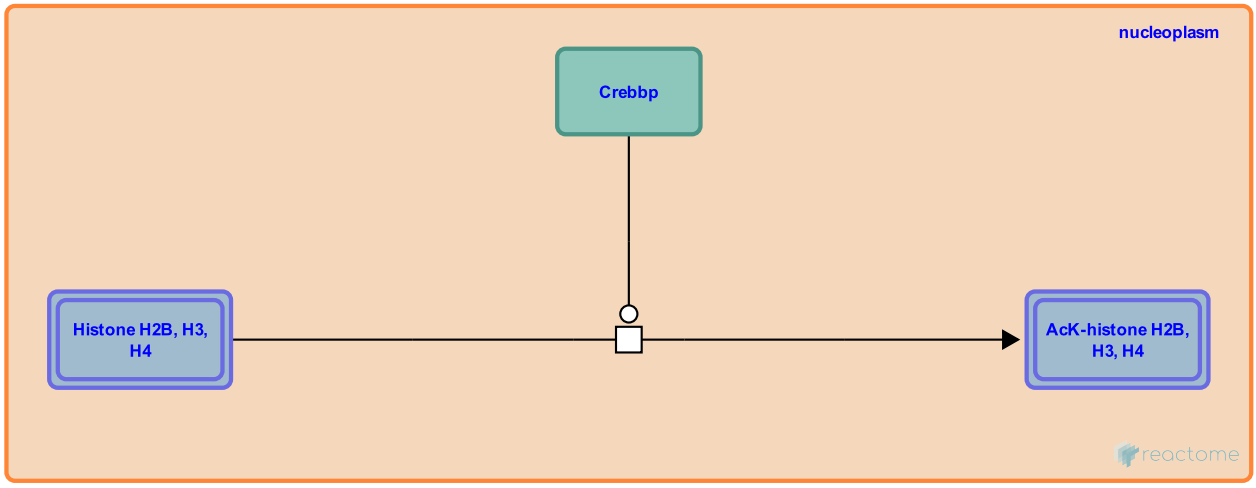
Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

Crebbp acetylates replicative histone H2B, H3, H4 ↗

Stable identifier: R-MMU-4568768

Type: transition

Compartments: nucleoplasm



CREBBP (CBP) is named after its interaction with the CRE-binding protein CREB, though it interacts with many other proteins. It is thought to act as an integrator of signals from various pathways (Goodman & Smolik 2000), which compete for a limited amount of nuclear CREBBP. CREBBP and EP300 (p300) are closely related and have overlapping functions but also unique properties, particularly in vivo (Kalkhoven 2004). Both proteins form a physical bridge between DNA-binding transcription factors and the RNA polymerase II complex. Histones are believed to be the main acetylation targets of CREBBP and EP300, but their ability to acetylate and thereby regulate transcription factors such as p53 (Gu & Roeder 1997) is considered significant additional function (Kasper et al. 2006).

CREBBP has intrinsic histone acetyltransferase (HAT) activity on lysine-12 of H2B, lysine-14 of H3 and lysine-8 of H4 (Bannister & Kouzarides 1996, Rekowski & Giannis 2010, Barrett et al. 2011).

Homozygous knockout of Crebbp results in embryonic lethality (Tanaka et al. 1997). Focal deletion of Crebbp demonstrates that it is critical for the in vivo acetylation of lysines on histones H2B, H3 and H4, and cannot be compensated for by the p300 (Barrett et al. 2011).

Genomic aberrations in CREBBP are associated with Rubinstein-Taybi syndrome (Torress et al. 2013).

Literature references

Barrett, RM., Malvaez, M., Kramar, E., Matheos, DP., Arrizon, A., Cabrera, SM. et al. (2011). Hippocampal focal knockout of CBP affects specific histone modifications, long-term potentiation, and long-term memory. *Neuropsychopharmacology, 36*, 1545-56. ↗

Editions

2013-03-12	Authored	Jupe, S.
2013-11-18	Edited	Jupe, S.
2013-11-18	Reviewed	Karagiannis, T.