Recruitment of Acetylated SL1 to phosUBF-1:rDNA Promoter

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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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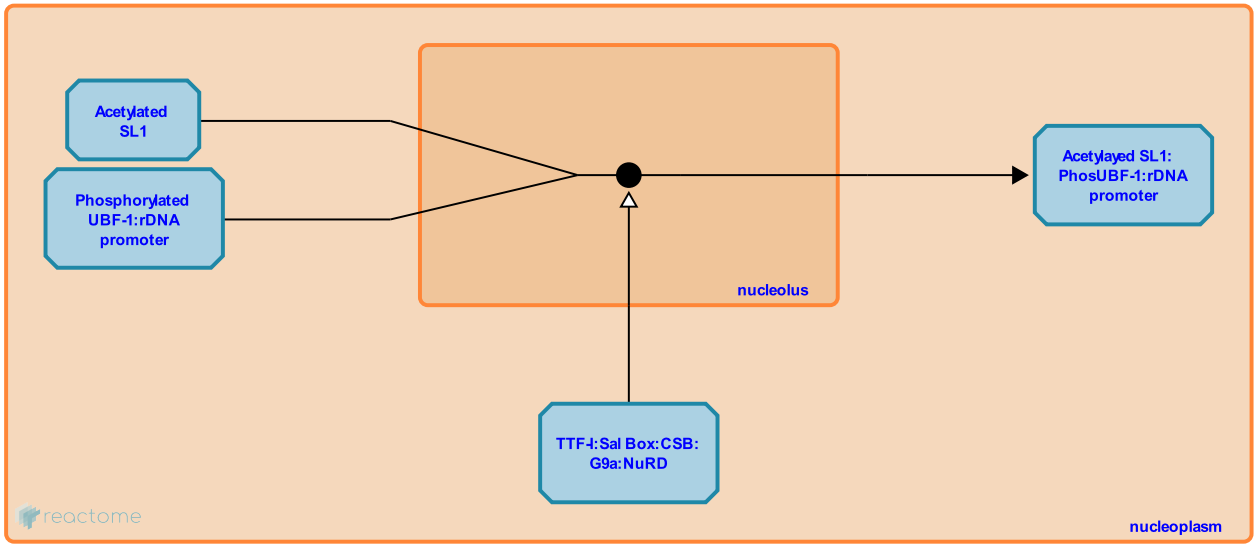
Stable identifier: R-HSA-73739

Type: binding

Compartments: nucleolus



Knockdown of CSB reduces recruitment of SL1 and RNA Polymerase I to active rRNA genes.
Human SL1 does not bind to DNA itself, rather it is recruited to the rDNA promoter through a physical interaction with UBF-1. Phosphorylation of UBF-1 within the carboxy-terminal region is required for SL1 binding. SL1 consists of TATA-binding protein (TBP) and three associated factors (TAFIs). SL1 has no sequence-specific DNA binding activity its recruitment to the promoter being mediated by specific interactions with UBF. Once bound the SL1 complex makes direct contact with the DNA promoter and guides promoter-specific initiation.

Studies to identify the mechanistic relationship between SL1 and UBF-1 have indicated that the interaction between UBF-1 and SL1 is regulated by tumor suppressor proteins such as Rb and P53, although it has also been proposed that Rb prevents UBF-1 from binding to DNA itself.

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Editions

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