CDT1-mediated formation of MCM2-7 double hexamer at the replication origin

Kusic-Tisma, J., Orlic-Milacic, M.
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 reaction (see Table of Contents)
CDT1-mediated formation of MCM2-7 double hexamer at the replication origin

**Stable identifier:** R-HSA-9749351

**Type:** transition

**Compartments:** nucleoplasm

**Inferred from:** CDT1-mediated formation of MCM2-7 double hexamers at the replication origins in budding yeast (Saccharomyces cerevisiae)

Based on studies in budding yeast, after CDT1-mediated loading of the MCM2-7 complex to CDC6 bound to the ORC(1-6) complex at the replication origin and ATP-dependent release of CDT1, another complex of CDT1 and MCM2-7 is loaded, resulting in the formation of the salt-stable double hexamer of MCM2-7 (Fernández-Cid et al. 2013). MCM2-7 double hexamers are connected head-to-head via their N-terminal rings. DNA runs through a central channel in the double hexamer (Remus et al. 2009). In a study using human proteins, it was suggested that geminin inhibits the formation of salt-resistant pre-replicative complexes (Wu et al. 2014), possibly by interfering with CDT1-mediated loading of the second MCM2-7 hexamer.

**Editions**

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2021-10-27</td>
<td>-authored</td>
<td>Kusic-Tisma, J.</td>
</tr>
<tr>
<td>2021-11-03</td>
<td>edited</td>
<td>Orlic-Milacic, M.</td>
</tr>
</tbody>
</table>