AcM-UBE2F transfers NEDD8 to CRL5 E3 ubiquitin ligase complex

Pick, E., Rothfels, K.

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

The contents of this document may be freely copied and distributed in any media, provided the authors, plus the institutions, are credited, as stated under the terms of Creative Commons Attribution 4.0 International (CC BY 4.0) License. For more information see our license.

16/01/2021
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


Reactome database release: 75

This document contains 1 reaction (see Table of Contents)
AcM-UBE2F transfers NEDD8 to CRL5 E3 ubiquitin ligase complex

Stable identifier: R-HSA-8952044

Type: transition

Compartments: cytosol

UBE2F transfers NEDD8 to lysine 724 of CUL5 in the E3 ligase complex (Duda et al, 2008). Neddylation increases the ubiquitination activity of the E3 complex towards its target, and prevents binding of the CUL5 complex with the CAND1 inhibitor (Hori et al, 1999; Duda et al, 2008; Kelsall et al, 2013). Targets of CUL5 RING complexes include a variety of cellular proteins including receptor and non-receptor tyrosine kinases, signaling molecules transcriptional regulators (reviewed in Okamura et al, 2016). CRL5 complexes are also hijacked by viruses such as HIV, HPV and adenovirus among others. Interaction with viral proteins redirects the ubiquitin ligase complex to target host proteins to promote conditions that favor viral propagation (Harada et al, 2002; Mehle et al, 2004; reviewed in Mahon et al, 2014).

Literature references


Editions

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016-12-13</td>
<td>Authored, Edited</td>
<td>Rothfels, K.</td>
</tr>
<tr>
<td>2017-02-22</td>
<td>Reviewed</td>
<td>Pick, E.</td>
</tr>
</tbody>
</table>