PARK2 K63-Ubiquitinates SNCAIP

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15/05/2024

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

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Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

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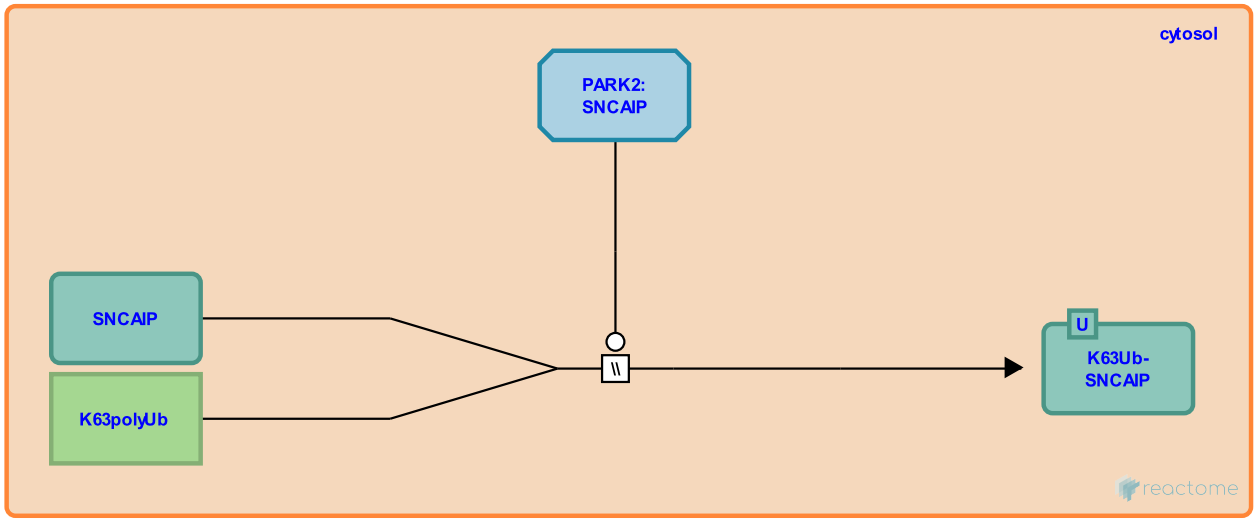
Stable identifier: R-HSA-5667111

Type: omitted

Compartments: cytosol



SNCAIP is ubiquitinated by several different E3 ubiquitin-ligases, including Parkin (PARK2). PARK2 overexpression with SNCAIP in cell culture leads to the formation of protein aggregates (Chung et al. 2001). PARK2 preferentially mediates the addition of lysine-63 (K63)-linked polyubiquitination of SNCAIP (Lim et al. 2005). This leads to SNCAIP degradation only at an unusually high PARK2 to SNCAIP ratio (Lim et al. 2005). K63-linked ubiquitination may be a signal that leads to the degradation of inclusions by autophagy when the ubiquitin-proteasome system is dysfunctional (Lim et al. 2005, Tan et al. 2008).

Literature references

Zhang, Y., Dawson, TM., Ross, CA., Dawson, VL., Lim, KL., Gao, J. et al. (2001). Parkin ubiquitinates the alpha-synuclein-interacting protein, synphilin-1: implications for Lewy-body formation in Parkinson disease. *Nat. Med., 7*, 1144-50. ↗

Editions

2015-01-23	Authored	Jupe, S.
2015-11-04	Edited	Jupe, S.
2015-11-09	Reviewed	Perry, G.