

ROS positively regulates misfolding of cilia proteins

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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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Reactome database release: 88

This document contains 1 reaction ([see Table of Contents](#))

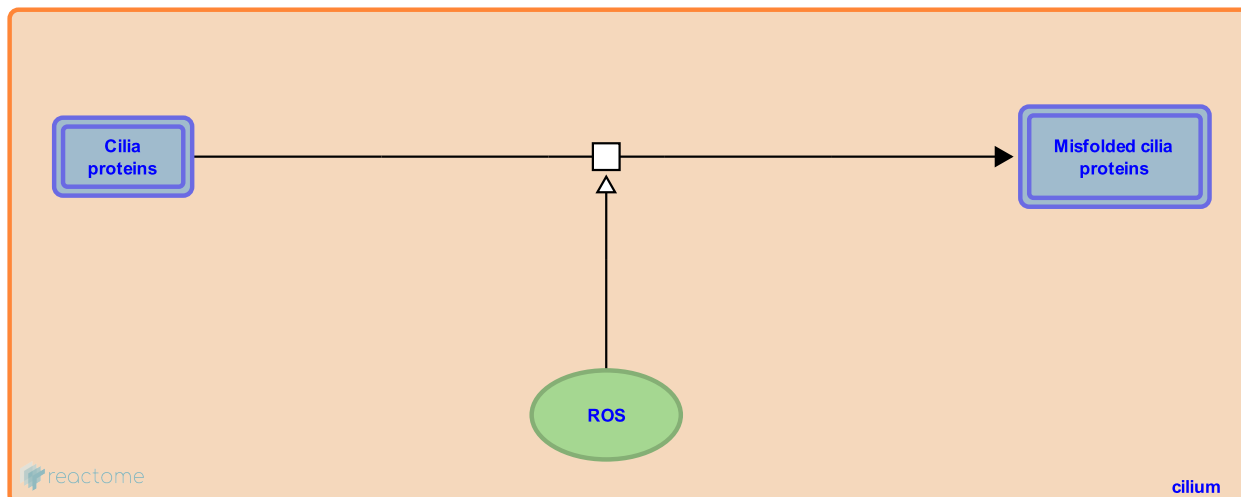
ROS positively regulates misfolding of cilia proteins [↗](#)

Stable identifier: R-HSA-9639698

Type: transition

Compartments: cilium

Inferred from: [ROS positively regulates misfolding of cilia proteins \(Mus musculus\)](#)



Accumulation of excessive reactive oxygen species (ROS) within cells results in oxidative stress. This stress can trigger proteins misfolding and make them dysfunctional. Cilia proteins are damaged when subjected to oxidative stress and may be targeted to the autophagy machinery. This results in the shortening of the cilium (Lam HC et al. 2013, Kim JI et al. 2013). Experiments leading to this finding were performed in mice.

Literature references

Jang, HS., Lipschutz, JH., Kim, JI., Noh, MR., Park, KM., Kim, J. (2013). Reduction of oxidative stress during recovery accelerates normalization of primary cilia length that is altered after ischemic injury in murine kidneys. *Am. J. Physiol. Renal Physiol.*, 304, F1283-94. [↗](#)

Sathirapongsasuti, JF., Ryter, SW., Cervo, M., Lam, HC., Shan, B., Cloonan, SM. et al. (2013). Histone deacetylase 6-mediated selective autophagy regulates COPD-associated cilia dysfunction. *J. Clin. Invest.*, 123, 5212-30. [↗](#)

Editions

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