PRDX1 overoxidizes

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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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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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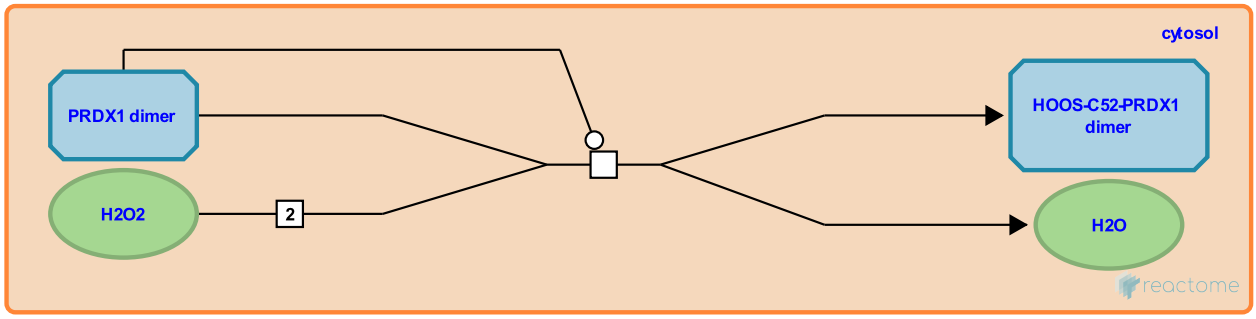
Stable identifier: R-HSA-5631885

Type: transition

Compartments: cytosol



The activity of eukaryotic PRDX1 gradually decreases with time, which is due to the overoxidation of the catalytic cysteine C52. Normally, oxidized cysteine C52-SOH is generated as a catalytic intermediate, which is subsequently reduced by thioredoxin. Occasionally, further oxidation happens, generating C52-SOOH , where the catalytic cysteine is converted to cysteine-sulfinic acid. This over-oxidation cannot be reversed by thioredoxin (Yang et al. 2002, Budanov et al. 2004). Bacterial peroxiredoxin AhpC does not undergo over-oxidation due to structural difference (Wood et al. 2003).

Literature references

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Editions

2014-12-23	Authored, Edited	Orlic-Milacic, M.
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