FECH binds Fe2+ to PRIN9 to form heme

D'Eustachio, P., Jassal, B., Sassa, S.

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

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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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Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

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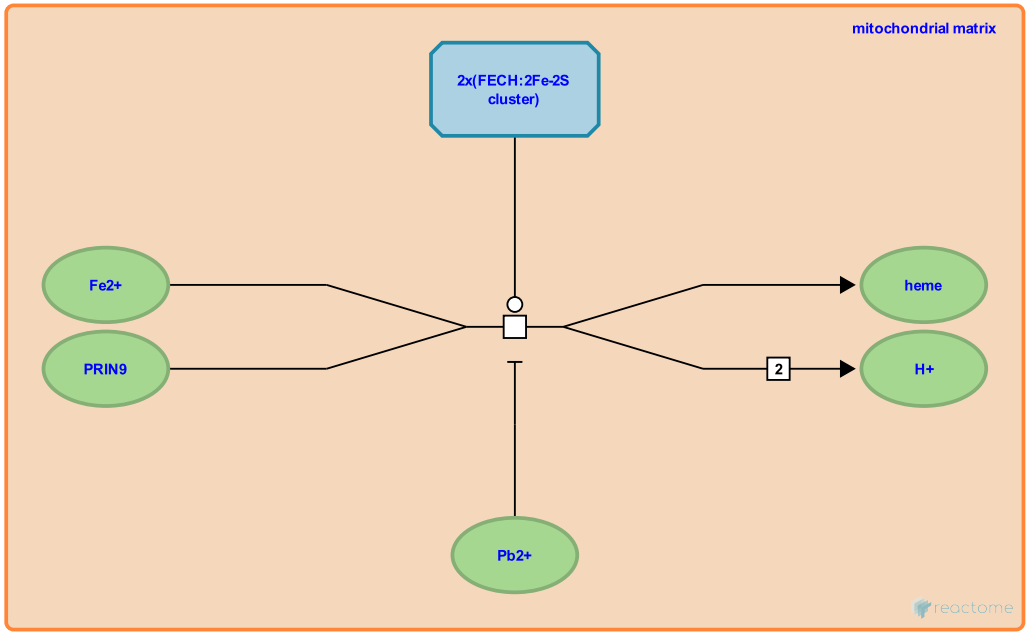
Stable identifier: R-HSA-189465

Type: transition

Compartments: mitochondrial matrix



Ferrochelatase (FECH) catalyzes the insertion of ferrous iron into protoporphyrin IX (PRIN9) to form heme. FECH is localized on the matrix surface of the inner mitochondrial membrane and this reaction takes place within the mitochondrial matrix. The enzyme functions as a homodimer with each monomer containing a nitric oxide-sensitive 2Fe-2S cluster. Enzyme deficiency is associated with erythropoietic protoporphyria in vivo, and inhibition of ferrochelatase activity is a clinically important consequence of lead poisoning (Piomelli et al. 1987).

Literature references

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Editions

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