

pAMPK inactivates ACACB, inhibiting malonyl-CoA synthesis

D'Eustachio, P., Gopinathrao, G., Jassal, B.

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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Reactome database release: 88

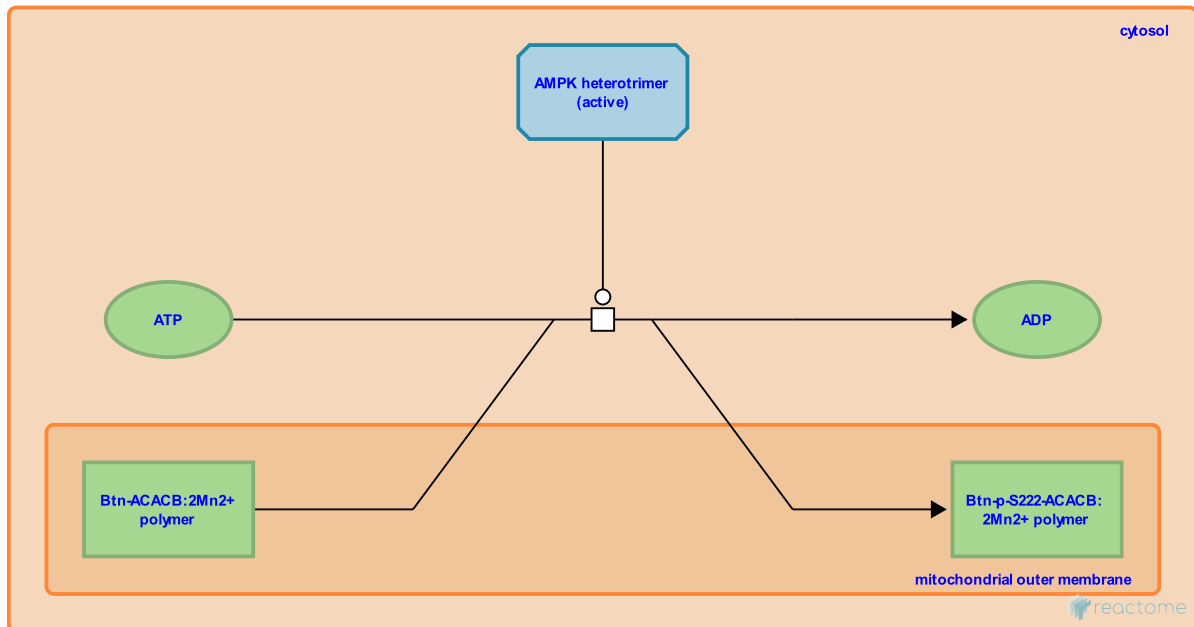
This document contains 1 reaction ([see Table of Contents](#))

pAMPK inactivates ACACB, inhibiting malonyl-CoA synthesis ↗

Stable identifier: R-HSA-200423

Type: transition

Compartments: cytosol, mitochondrial outer membrane



Acetyl-CoA carboxylase 2 (ACACB, ACC2) is involved in the regulation of mitochondrial fatty acid oxidation through the inhibition of carnitine palmitoyltransferase 1 by its product malonyl-CoA. Phosphorylated AMPK inactivates ACACB in muscle cells by phosphorylation. This results in decreased levels of malonyl CoA, contributing to the homeostasis of mitochondrial beta oxidation (Ruderman & Prentki 2004).

Literature references

Prentki, M., Ruderman, N. (2004). AMP kinase and malonyl-CoA: targets for therapy of the metabolic syndrome. *Nat Rev Drug Discov*, 3, 340-51. ↗

Editions

2007-07-30	Authored	Gopinathrao, G.
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