Activation, myristoylation of BID and translocation to mitochondria

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This is just an excerpt of a full-length report for this pathway. To access the complete report, please download it at the Reactome Textbook.

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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


Reactome database release: 82
This document contains 1 pathway and 4 reactions (see Table of Contents)

https://reactome.org
BID may promote cell death by activating BAX and BAK while inactivating anti-apoptotic proteins. The engagement of cell surface receptors activates the caspase-8, a heterodimer, that cleaves BID in its amino terminal region. This particular event may act as a link between Extrinsic (caspase 8/10 dependent) and Intrinsic (Bcl-2 inhibitable) pathways although some evidences from mouse genetic experiments suggest the contrary. It has been suggested that the death signals from the extrinsic or death receptor pathway may get amplified by the mechanisms of intrinsic pathway and that this functional loop may be enabled by the molecules like tBID (truncated BID).

Cleavage of BID to tBID can also be achieved by Granzyme B. The truncated protein is myristoylated and translocates to mitochondria.

**Literature references**


Caspase-8 activates BID by cleavage

**Location:** Activation, myristoylation of BID and translocation to mitochondria

**Stable identifier:** R-HSA-139898

**Type:** transition

**Compartments:** cytosol

The caspase 8-mediated cleavage of cytosolic, inactive p22 BID at internal Asp sites yields a major p15 and minor p13 and p11 fragments. After myristoylation, tBID translocates to mitochondria as an integral membrane protein.

**Followed by:** Myristoylation of tBID by NMT1

**Literature references**


**Editions**

2005-04-27 Authored Gopinathrao, G.
**Granzyme-B activates BID by cleavage**

**Location:** Activation, myristolyation of BID and translocation to mitochondria

**Stable identifier:** R-HSA-139893

**Type:** transition

**Compartments:** cytosol

GZMB (granzyme B) cleaves BID to produce a p15 truncated form of BID (tBID) (Alimonti et al. 2001).

**Followed by:** Myristoylation of tBID by NMT1

**Literature references**

Myristoylation of tBID by NMT1

Location: Activation, myristoylation of BID and translocation to mitochondria

Stable identifier: R-HSA-141367

Type: transition

Compartments: cytosol

After proteolytic activation, tBID is myristoylated by NMT-1 at an exposed glycine. N-myristoylation may enable the activated tBID to associate with the lipid components of the mitochondrial membrane.

Preceded by: Caspase-8 activates BID by cleavage, Granzyme-B activates BID by cleavage

Followed by: Translocation of tBID to mitochondria

Literature references


Editions

2004-11-08 Authored Gopinathrao, G.
Translocation of tBID to mitochondria

Location: Activation, myristoylation of BID and translocation to mitochondria

Stable identifier: R-HSA-139920

Type: transition

Compartments: cytosol, mitochondrial outer membrane

N-myristoylation targets tBID to the mitochondrial outer membrane (Zha et al. 2000).

Preceded by: Myristoylation of tBID by NMT1

Literature references

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