

# Mitofusins trans-interact linking mitochondria prior to fusion

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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

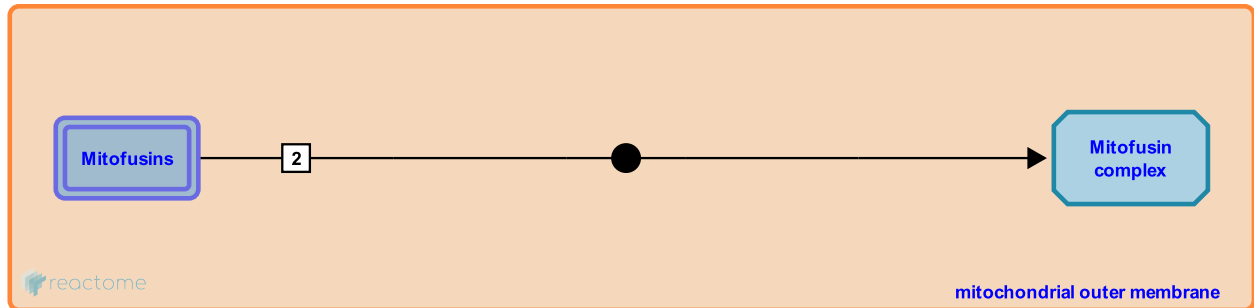
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## Mitofusins trans-interact linking mitochondria prior to fusion [↗](#)

**Stable identifier:** R-HSA-992703

**Type:** binding

**Compartments:** mitochondrial outer membrane



Mitochondria frequently fuse and divide (Bereiter-Hahn & Voth 1994); these processes affect morphology and are important for normal mitochondrial functions such as respiration, development and apoptosis. Mitofusins (MFNs) are mitochondrial GTPases that mediate mitochondrial outer membrane fusion. Mammals have two mitofusins; Mfn1-null or Mfn2-null mouse embryonic fibroblast cells show predominantly fragmented mitochondria and have greatly reduced mitochondrial fusion in vivo (Chen et al. 2003, 2005). MFNs acts in trans to bring mitochondria into close proximity prior to fusion (Koshiba et al. 2004). They also tether the endoplasmic reticulum (ER) to mitochondria, cross-linking MFNs expressed on the mitochondrial outer membrane and ER membrane (de Brito & Scorrano 2008).

### Literature references

Lombès, A., Legros, F., Château, D., Rojo, M. (2002). Membrane topology and mitochondrial targeting of mitofusins, ubiquitous mammalian homologs of the transmembrane GTPase Fzo. *J Cell Sci*, 115, 1663-74. [↗](#)

### Editions

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