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# ProductInformation

**Anti-Mitofusin 2 (N-terminal)**
Produced in Rabbit, Affinity Isolated Antibody

Product Number **M 6319**

## Product Description

Anti-Mitofusin 2 (N-terminal) is developed in rabbit using as immunogen a synthetic peptide corresponding to amino acid residues 38-55 of human mitofusin 2 with C-terminal added cysteine, conjugated to KLH. The corresponding sequence differs by one amino acid in both rat and mouse mitofusin 2. The antibody is affinity-purified using the immunizing peptide immobilized on agarose.

Anti-Mitofusin 2 (N-terminal) recognizes human, rat, and mouse mitofusin 2. Applications include immunoblotting (~86 kDa), immunoprecipitation, and immunofluorescence. Detection of the mitofusin 2 band by immunoblotting is specifically inhibited with the immunizing peptide.

Mitofusins (Mfn1 and Mfn2) are the mammalian homologs of the *Drosophila* protein fuzzy onion (Fzo). They are transmembrane GTPases embedded in the outer membrane of mitochondria.[1] These proteins are essential for fusion of mitochondria in mammalian cells.[2] The dynamic balance between fusion and fission determines mitochondrial morphology.[3] Mfn1 and Mfn2 form homotypic and heterotypic complexes that are functional for fusion. Mitochondrial fusion is also important for cell growth, mitochondrial membrane potential, respiration, and embryonic development. Mice deficient in either Mfn1 or Mfn2 die in mid-gestation. Mfn2 mutant embryos have a specific and severe disruption of a layer of the placenta.[4] Mitofusin 2 is broadly expressed, with highest expression in heart and skeletal muscle and is induced during myogenesis.[1, 4-5] Repression of Mfn2 causes morphological and functional fragmentation of the mitochondrial network into independent clusters and reduces mitochondrial membrane potential and glucose oxidation.

Thus, Mfn2 is essential for the maintenance of mitochondrial network and controls mitochondrial metabolism. This Mfn2-dependent regulatory mechanism is disturbed in obesity by reduced Mfn2 expression.[5] Mutations in Mitofusin 2 cause Charcot-Marie-Tooth neuropathy type 2A, a neurological disorder that results from degeneration of axons in peripheral nerves.[6]

## Reagent

The antibody is supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Antibody Concentration: ~1.0 mg/mL

## Precautions and Disclaimer

Due to the sodium azide content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazardous and safe handling practices.

## Storage/Stability

For continuous use, store at 2-8 °C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing is not recommended. Storage in "frost-free" freezers is also not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilutions should be discarded if not used within 12 hours.

## Product Profile

By immunoblotting, a working antibody concentration of 0.5-1 µg/mL is recommended using an extracts of rat and mouse brain mitochondria and a chemiluminescent detection reagent.

By indirect immunofluorescence, a working antibody concentration of 20-30 µg/mL is recommended using differentiated mouse C2 cells.

5-10 µg of the antibody immunoprecipitates mitofusin 2 from HeLa human epithelioid carcinoma cell lysate.

Note: In order to obtain the best results using various techniques and preparations, we recommend determining the optimal working dilutions by titration.

**References**


1. Rojo, M., et al., J. Cell Sci., **115**, 1663-1674 (2002).
2. Koshiba, T., et al., Science, **305**, 858-862 (2004).
3. Santel, A., and Fuller, M.T., J. Cell Sci., **114**, 867-874 (2000).
4. Chen, H., et al., J. Cell Biol., **160**, 189-200 (2003).
5. Bach, D., et al., J. Biol. Chem., **278**, 17190-17197 (2003).
6. Zuchner, S., et al., Nat. Genet., **36**, 449-51 (2004).


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