



Product Information Sheet

Polyclonal Anti-Mitofusin 2, *MFN2*

Catalogue No. PA1051

Lot No. 09G02

Ig type: rabbit IgG

Size: 100µg/vial

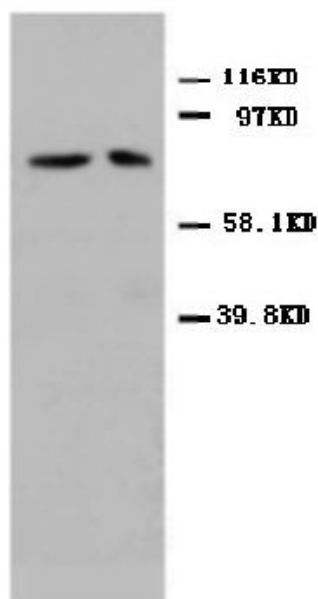
Specificity

Human, mouse, rat.

No cross reactivity with other proteins.

Recommended application

Western blot



Immunogen

A synthetic peptide corresponding to a sequence mapping at the N-terminal of human MFN2, different from the related rat and mouse sequence by single amino acids.

Purity

Immunogen affinity purified.

Application

	Concentration	Tested Species	Concluded Species	Antigen Retrieval
WB	1µg/ml	Hu, Rat	Ms	-
IHC-P	-	-	-	-
IHC-F	-	-	-	-
ICC	-	-	-	-

Other applications have not been tested.

Optimal dilutions should be determined by end user.

Contents

Each vial contains 5mg BSA, 0.9mg NaCl, 0.2mg Na₂HPO₄, 0.05mg Thimerosal, 0.05mg NaN₃.

Reconstitution

0.2ml of distilled water will yield a concentration of 500µg/ml.

To reorder contact us at:

Antagene, Inc.

Toll Free: 1(866)964-2589

email: Info@antageneinc.com

FOR RESEARCH USE ONLY. NOT FOR DIAGNOSTIC AND CLINICAL USE.

Storage

month. It can also be aliquotted and stored frozen at -20°C for longer
At -20°C for one year. After time.
reconstitution, at 4°C for one

BACKGROUND

Mitofusin 2 (MFN2) is a mitochondrial transmembrane GTPase regulating mitochondrial fusion and that the nucleotide-dependent activation of MFN2 concomitantly protects the organelle from permeability transition. It is mapped to chromosome 1 and encodes a 757-amino acid protein that contains an ATP/GTP-binding site motif. It is expressed in many tissues and cell lines such as brain and KG-1 with the highest expression in heart and skeletal muscle. This protein contains an N-terminal GTPase domain and a transmembrane domain near the C terminus. It shares 60% identity with MFN1. When stably expressed in COS-7 cells, MFN2 colocalizes with mitochondrial markers. Axonal CMT type 2A and autosomal dominant HMSN VI are caused by MFN2 and mutations in MFN2, which emphasizes its important role of mitochondrial function for both optic atrophies and peripheral neuropathies.

REFERENCE

1. Neuspiel M, Zunino R, Gangaraju S, Rippstein P, McBride H. Activated mitofusin 2 signals mitochondrial fusion, interferes with Bax activation, and reduces susceptibility to radical induced depolarization. *J Biol Chem*. 2005 Jul 1; 280(26):25060-70.
2. Nagase T, Seki N, Ishikawa K, Ohira M, Kawarabayasi Y, Ohara O, Tanaka A, Kotani H, Miyajima N, Nomura N. Prediction of the coding sequences of unidentified human genes. VI. The coding sequences of 80 new genes (KIAA0201-KIAA0280) deduced by analysis of cDNA clones from cell line KG-1 and brain. *DNA Res*. 1996 Oct 31; 3(5):321-9, 341-54.
3. Santel A, Fuller MT. Control of mitochondrial morphology by a human mitofusin. *J Cell Sci*. 2001 Mar; 114(Pt 5):867-74.
4. Zuchner S, De Jonghe P, Jordanova A, Claeys KG, Guergueltcheva V, Cherninkova S, Hamilton SR, Van Stavern G, Krajewski KM, Stajich J, Tournev I, Verhoeven K, Langerhorst CT, de Visser M, Baas F, Bird T, Timmerman V, Shy M, Vance JM. Axonal neuropathy with optic atrophy is caused by mutations in mitofusin 2. *Ann Neurol*. 2006 Feb; 59(2):276.