

DESCRIPTION

Species Reactivity	Human
Specificity	Detects a synthetic peptide specific for human OPA1 around amino acid 200 in Direct ELISA.
Source	Monoclonal Mouse IgG Clone # 1088319
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Synthetic Peptide Accession # O60313
Conjugate	Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunohistochemistry	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Optic Atrophy-1 (OPA1), aka Dynamin-like 120 kDa protein, mitochondrial, is a Dynamin-related GTPase required for mitochondrial fusion and regulation of apoptosis. OPA1 exists as a single-pass membrane protein in the mitochondrion inner membrane as well as in soluble forms in mitochondrion intermembrane space, and is expressed in retina, brain, testis, heart, skeletal muscles. Human OPA1 binds PARL and interacts with CHCHD3 as well as IMMT (preferentially with soluble OPA1 forms). Proteolytic processing in response to intrinsic apoptotic signals may lead to disassembly of OPA1 oligomers and release of the caspase activator cytochrome C (CYCS) into mitochondrial intermembrane space. OPA1 protein form S1 is an inactive form produced by cleavage at S1 position by metalloendopeptidase OMA1 following stress conditions that induce loss of mitochondrial membrane potential, leading to negative regulation of mitochondrial fusion. Defects in OPA1 have been linked to optic atrophy type 1 (OPA1) and dominant optic atrophy plus syndrome (DOA+).

PRODUCT SPECIFIC NOTICES

This product is provided under an agreement between Life Technologies Corporation and R&D Systems, Inc, and the manufacture, use, sale or import of this product is subject to one or more US patents and corresponding non-US equivalents, owned by Life Technologies Corporation and its affiliates. The purchase of this product conveys to the buyer the non-transferable right to use the purchased amount of the product and components of the product only in research conducted by the buyer (whether the buyer is an academic or for-profit entity). The sale of this product is expressly conditioned on the buyer not using the product or its components (1) in manufacturing; (2) to provide a service, information, or data to an unaffiliated third party for payment; (3) for therapeutic, diagnostic or prophylactic purposes; (4) to resell, sell, or otherwise transfer this product or its components to any third party, or for any other commercial purpose. Life Technologies Corporation will not assert a claim against the buyer of the infringement of the above patents based on the manufacture, use or sale of a commercial product developed in research by the buyer in which this product or its components was employed, provided that neither this product nor any of its components was used in the manufacture of such product. For information on purchasing a license to this product for purposes other than research, contact Life Technologies Corporation, Cell Analysis Business Unit, Business Development, 29851 Willow Creek Road, Eugene, OR 97402, Tel: (541) 465-8300. Fax: (541) 335-0354.