

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human, mouse, and rat OPA1 in Western blots.
Source	Monoclonal Rabbit IgG Clone # 1284B
Purification	Protein A or G purified from cell culture supernatant
Immunogen	A synthetic peptide made to an internal region within amino acid residues 500-600 of human OPA1 Accession # O60313
Conjugate	Alexa Fluor 532 Excitation Wavelength: 534 nm Emission Wavelength: 553 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.

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+).

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