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DM2012 Monoclonal Antibody to Apolipoprotein J / Apo J - Purified

Alternate names:	40, 40, AAG4, APOJ, Aging-associated gene 4 protein, CLI, Clusterin, Complement cytolysis inhibitor, Complement-associated protein SP-40, KUB1, Ku70-binding protein 1, NA1/NA2, SP-40, Testosterone-repressed prostate message 2
Quantity:	0.1 mg
Concentration:	1.0 mg/ml
Background:	Clusterin is a 75-80 kD disulfide-linked heterodimeric protein containing about 30% of N-linked carbohydrate rich in sialic acid, but truncated forms targeted to the nucleus have also been identified. The precursor polypeptide chain is cleaved proteolytically to remove the 22-mer secretory signal peptide and subsequently between residues 227/228 to generate the alpha and beta chains. These are assembled antiparallel to give a heterodimeric molecule in which the cysteine-rich centers are linked by five disulfide bridges and are flanked by two predicted coiled-coil alpha-helices and three predicted amphipathic alpha-helices. The six sites of N-linked glycosylation are indicated as yellow spots. Across a broad range of species clusterin shows 70% to 80% of sequence homology. It is ubiquitously expressed in most mammalian tissues and can be found in plasma, milk, urine, cerebrospinal fluid and semen. It is able to bind and form complexes with numerous partners such as immunoglobulins, lipids, heparin, bacteria, complement components, paraoxonase, beta amyloid, leptin and others. Clusterin has been ascribed a plethora of functions such as phagocyte recruitment, aggregation induction, complement attack prevention, apoptosis inhibition, membrane remodelling, lipid transport, hormone transport and/or scavenging, matrix metalloproteinase inhibition. A detailed mechanism of clusterin has not been defined. One tempting hypothesis says that clusterin is an extracellular chaperone protecting cells from stress induced by degraded and misfolded protein precipitates.Clusterin is up- or downregulated on the mRNA or protein level in many pathological and clinically relevant situations including cancer, organ regeneration, infection, Alzheimer disease, retinitis pigmentosa, myocardial infarction, renal tubular damage, autoimmunity and others.
Uniprot ID:	P10909
NCBI:	NP_976084.1
GeneID:	1191
Host / Isotype:	Mouse / IgG1
Recommended Isotype Controls:	SM10P (for use in human samples), AM03095PU-N
Clone:	Hs-3
Immunogen:	The antibody was prepared against Triton X-100 Human sperm extract.

Format:

State: Lyophilized purified IgG fraction
Purification: Affinity Chromatography on Protein A-Sepharose followed by DEAE-Chromatography
Buffer System: 0.05M Phosphate buffer, 0.1M NaCl, pH 7.2
Preservatives: 15mM Sodium Azide
Reconstitution: Add 0.1 ml of deionized water and let the lyophilized pellet dissolve completely. Slight turbidity may occur after reconstitution, which does not affect activity of the antibody. In this case clarify the solution by centrifugation.

Applications:

ELISA.
Western Blot.
Immunocytochemistry.
Immunofluorescence.
Immunohistochemistry.
Other applications not tested. Optimal dilutions are dependent on conditions and should be determined by the user.

Specificity:

The anti-Clusterin Antibody, Clone Hs-3 is a Mouse monoclonal antibody against Human Clusterin.
Does not react with Cat, Dog, Bovine.

Storage:

Store lyophilized at 2-8°C for 6 months or at -20°C long term.
After reconstitution store the antibody undiluted at 2-8°C for one month or (in aliquots) at -20°C long term.
Avoid repeated freezing and thawing.
Shelf life: one year from despatch.

Product Citations:

Originator or purchased from resellers:

1. Zenkel M, Kruse FE, Jünemann AG, Naumann GO, Schlötzer-Schrehardt U. Clusterin deficiency in eyes with pseudoexfoliation syndrome may be implicated in the aggregation and deposition of pseudoexfoliative material. Invest Ophthalmol Vis Sci. 2006 May;47(5):1982-90. PubMed PMID: 16639006.
2. Schlötzer-Schrehardt U, Pasutto F, Sommer P, Hornstra I, Kruse FE, Naumann GO, et al. Genotype-correlated expression of lysyl oxidase-like 1 in ocular tissues of patients with pseudoexfoliation syndrome/glaucoma and normal patients. Am J Pathol. 2008 Dec;173(6):1724-35. doi: 10.2353/ajpath.2008.080535. Epub 2008 Oct 30. PubMed PMID: 18974306.
3. Zhang F, Sha J, Wood TG, Galindo CL, Garner HR, Burkart MF, et al. Alteration in the activation state of new inflammation-associated targets by phospholipase A2-activating protein (PLAA). Cell Signal. 2008 May;20(5):844-61. doi: 10.1016/j.cellsig.2008.01.004. Epub 2008 Jan 17. PubMed PMID: 18291623.