

Clusterin Antibody
Catalog # ASC10434**Specification**

Clusterin Antibody - Product Information

Application	WB, IHC-P, IF, E
Primary Accession	P10909
Other Accession	NP_001822 , 1191
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Application Notes	Clusterin antibody can be used for the detection of Clusterin by Western blot at 0.5 - 1 µg/mL. Antibody can also be used for immunohistochemistry starting at 5 µg/mL. For immunofluorescence start at 20 µg/mL.

Clusterin Antibody - Additional InformationGene ID **1191****Other Names**

Clusterin Antibody: CLI, AAG4, APOJ, CLU1, CLU2, KUB1, SGP2, APO-J, SGP-2, SP-40, TRPM2, TRPM-2, NA1/NA2, CLI, Clusterin, Aging-associated gene 4 protein, Apo-J, clusterin

Target/Specificity

Clusterin antibody was raised recombinant human Clusterin isoform 1.

Reconstitution & Storage

Clusterin antibody can be stored at 4°C for three months and -20°C, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.

Precautions

Clusterin Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Clusterin Antibody - Protein Information**Name** CLU ([HGNC:2095](#))**Function**

[Isoform 1]: Functions as extracellular chaperone that prevents aggregation of non native proteins (PubMed:11123922, PubMed:19535339). Prevents stress-induced aggregation of blood plasma proteins (PubMed:11123922, PubMed:12176985, PubMed:12176985, PubMed:12176985).

<http://www.uniprot.org/citations/17260971> target="_blank">17260971, PubMed:19996109). Inhibits formation of amyloid fibrils by APP, APOC2, B2M, CALCA, CSN3, SNCA and aggregation-prone LYZ variants (in vitro) (PubMed:12047389, PubMed:17407782, PubMed:17412999). Does not require ATP (PubMed:11123922). Maintains partially unfolded proteins in a state appropriate for subsequent refolding by other chaperones, such as HSPA8/HSC70 (PubMed:11123922). Does not refold proteins by itself (PubMed:11123922). Binding to cell surface receptors triggers internalization of the chaperone-client complex and subsequent lysosomal or proteasomal degradation (PubMed:21505792). Protects cells against apoptosis and against cytolysis by complement (PubMed:2780565). Intracellular forms interact with ubiquitin and SCF (SKP1-CUL1-F-box protein) E3 ubiquitin-protein ligase complexes and promote the ubiquitination and subsequent proteasomal degradation of target proteins (PubMed:20068069). Promotes proteasomal degradation of COMMD1 and IKBKB (PubMed:20068069). Modulates NF-kappa-B transcriptional activity (PubMed:12882985). A mitochondrial form suppresses BAX- dependent release of cytochrome c into the cytoplasm and inhibit apoptosis (PubMed:16113678, PubMed:17689225). Plays a role in the regulation of cell proliferation (PubMed:19137541). An intracellular form suppresses stress-induced apoptosis by stabilizing mitochondrial membrane integrity through interaction with HSPA5 (PubMed:22689054). Secreted form does not affect caspase or BAX-mediated intrinsic apoptosis and TNF-induced NF-kappa-B-activity (PubMed:24073260). Secreted form act as an important modulator during neuronal differentiation through interaction with STMN3 (By similarity). Plays a role in the clearance of immune complexes that arise during cell injury (By similarity).

Cellular Location

[Isoform 1]: Secreted. Note=Can retrotranslocate from the secretory compartments to the cytosol upon cellular stress. [Isoform 6]: Cytoplasm. Note=Keeps cytoplasmic localization in stressed and unstressed cell.

Tissue Location

Detected in blood plasma, cerebrospinal fluid, milk, seminal plasma and colon mucosa. Detected in the germinal center of colon lymphoid nodules and in colon parasympathetic ganglia of the Auerbach plexus (at protein level). Ubiquitous. Detected in brain, testis, ovary, liver and pancreas, and at lower levels in kidney, heart, spleen and lung.

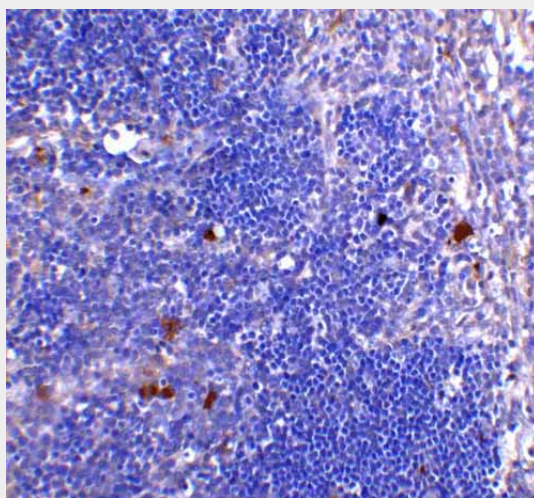
Clusterin Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

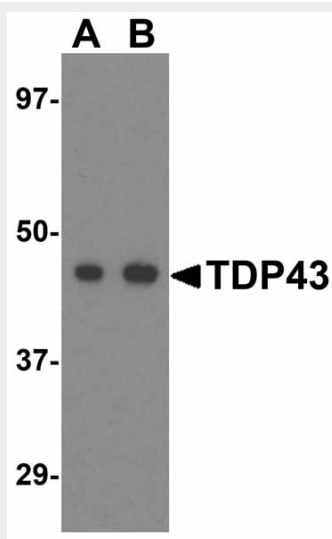
- [Western Blot](#)
- [Blocking Peptides](#)

- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

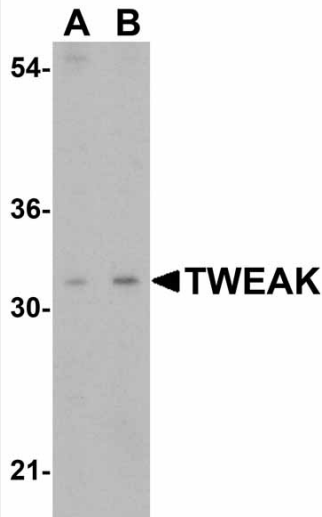
Clusterin Antibody - Images



Immunohistochemistry of CD4 in human thymus tissue with CD4 antibody at 5 μ g/ml.



Western blot analysis of TDP43 in L1210 cell lysate with TDP43 antibody at (A) 0.5 and (B) 1 μ g/mL.



Western blot analysis of TWEAK in HeLa cell lysate with TWEAK antibody at (A) 1 and (B) 2 µg/mL.

Clusterin Antibody - Background

Clusterin Antibody: Clusterin, also known as Apolipoprotein J (ApoJ), is a ubiquitous multifunctional glycoprotein that can interact with a broad spectrum of molecules such as complement components, various receptors, and the Alzheimer's b-amyloid peptide. Clusterin expression is increased in Alzheimer's disease brain tissue and clusterin-immunoreactive amyloid plaques are found associated with phospho-tau-positive dystrophic neurites and it has been suggested that clusterin facilitates the conversion of diffuse b-amyloid deposits into amyloid and enhances tau phosphorylation in neurites around these plaques. Other reports show that clusterin expression is decreased in proliferating cells and is upregulated in quiescent and senescent cells, suggesting that it may also play a role in aging and tumorigenesis suppression. Clusterin exists in at least two distinct isoforms.

Clusterin Antibody - References

- Calero M, Rostagno A, Frangione B, et al. Clusterin and Alzheimer's disease. *Subcell. Biochem.* 2005; 38:273-98.
- Martin-Rehrmann MD, Hoe HS, Capuani EM, et al. Association of apolipoprotein J-positive β -amyloid plaques with dystrophic neurites in Alzheimer's disease brain. *Neurotox. Res.* 2005; 7:231-42.
- Trougakos IP and Gonos ES. Clusterin/Apolipoprotein J in human aging and cancer. *Int. J. Biochem. Cell Biol.* 2002; 34:1430-48.
- Martinon F and Tschopp J. NLRs join TLRs as innate sensors of pathogens. *TRENDS Imm.* 2005; 26:447-54.