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Rabbit Anti-CTRP5/AP Conjugated antibody

SL11717R-AP

Product Name	Anti-CTRP5/AP
Chinese Name	碱性磷酸酶（AP）标记的补体 C1q 和 Tumour 坏死因子相关蛋白 5 抗体
Alias	C1q and tumor necrosis factor related protein 5; C1QTNF5; Complement C1q tumor necrosis factor related protein 5 precursor; LORD; C1QT5_HUMAN.
Research Area	Cell biology immunology Neurobiology Cell adhesion molecule
Immunogen Species	Rabbit
Clonality	Polyclonal
React Species	Mouse(predicted:Human,Rat,Dog,Pig,Cow,Rabbit)
Applications	IHC-P=1:100-500,IHC-F=1:100-500 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight	24kDa
Form	Lyophilized or Liquid
Concentration	1mg/ml
immunogen	KLH conjugated synthetic peptide derived from human CTRP5 (191-243aa)
Lsotype	IgG
Purification	affinity purified by Protein A
Storage Buffer	1M TBS(pH7.4) with 1% BSA, 3% Proclin300 and 50% Glycerol.
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Product Detail	**background:** Members of the C1q superfamily have diverse functions that are related to cell adhesion and basement membrane components. CTRP5 (Complement C1q tumor necrosis factor-related protein 5) is a 243 amino acid secreted and membrane-associated protein that contains a collagen-like domain and a C1q domain. CTRP5 is a short-chain collagen that is expressed in retinal pigment epithelium as well as brain, lung, liver and placenta. By forming an extracellular hexagonal lattice, CTRP5 facilitates the adhesion of basal retinal

pigment epithelium to Bruch's membrane, the innermost layer of the choroid. A mutation within the C1q domain of CTRP5 results in abnormal high molecular weight aggregate formation, which alters its structure and interactions. This mutation may result in the presentation of late-onset retinal degeneration (LORD), an autosomal dominant disorder that is characterized by punctate yellow-white deposits in the retinal fundus and night blindness.

Function:
Adipose tissue of an organism plays a major role in regulating physiologic and pathologic processes such as metabolism and immunity by producing and secreting a variety of bioactive molecules termed adipokines. One highly conserved family of adipokines is adiponectin/ACRP30 and its structural and functional paralogs, the C1q/tumor necrosis factor-alpha-related proteins (CTRPs) 1-7. Unlike adiponectin, which is expressed exclusively by differentiated adipocytes, the CTRPs are expressed in a wide variety of tissues. These proteins are thought to act mainly on liver and muscle tissue to control glucose and lipid metabolism. An analysis of the crystal structure of adiponectin revealed a structural and evolutionary link between TNF and C1q-containing proteins, suggesting that these proteins arose from a common ancestral innate immunity gene. CTRP5 has been suggested to be involved in age-related macular degeneration.

Subcellular Location:
Secreted

DISEASE:
Defects in C1QTNF5 are a cause of late-onset retinal degeneration (LORD) [MIM:605670]. LORD is an autosomal dominant disorder characterized by onset in the fifth to sixth decade with night blindness and punctate yellow-white deposits in the retinal fundus, progressing to severe central and peripheral degeneration, with choroidal neovascularization and chorioretinal atrophy.

Similarity:
Contains 1 C1q domain.
Contains 1 collagen-like domain.

Database links:
UniProtKB/Swiss-Prot: Q9BXJ0.1

Important Note:
This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

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