

bs-11717R**[Primary Antibody]****CTRP5 Rabbit pAb****Bioss**
ANTIBODIES

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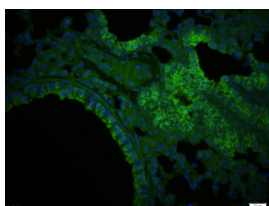
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— DATASHEET —

Host: Rabbit	Isotype: IgG	Applications: IHC-P (1:100-500)
Clonality: Polyclonal		IHC-F (1:100-500)
GeneID: 114902	SWISS: Q9BXJ0	IF (1:100-500)
Target: CTRP5		Reactivity: Mouse (predicted: Human, Rat, Rabbit, Pig, Cow, Dog)
Immunogen: KLH conjugated synthetic peptide derived from human CTRP5: 191-243/243.		
Purification: affinity purified by Protein A		Predicted MW.: 24 kDa
Concentration: 1mg/ml		Subcellular Location: Secreted
Storage: 0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.		
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.		

— VALIDATION IMAGES —

Paraformaldehyde-fixed, paraffin embedded (Mouse lung); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (CTRP5) Polyclonal Antibody, Unconjugated (bs-11717R) at 1:200 overnight at 4°C, followed by a conjugated Goat Anti-Rabbit IgG antibody (bs-0295G-FITC) for 90 minutes, and DAPI for nuclei staining.

— SELECTED CITATIONS —

- **[IF=6.268]** Miyagishima, Kiyoharu J.. et al. AMPK modulation ameliorates dominant disease phenotypes of CTRP5 variant in retinal degeneration. Commun Biol. 2021 Dec;4(1):1-16 ICC ;Material. 34887495