

bs-11336R**[Primary Antibody]****C1QA Rabbit pAb****Bioss**
ANTIBODIES

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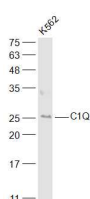
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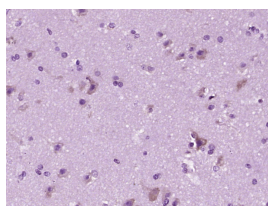
400-901-9800

— DATASHEET —

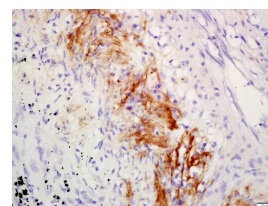
Host: Rabbit	Isotype: IgG	Applications: WB (1:500-2000)
Clonality: Polyclonal		IHC-P (1:50-200)
GeneID: 712	SWISS: P02745	IHC-F (1:50-200)
Target: C1QA		IF (1:100-500)
Immunogen: KLH conjugated synthetic peptide derived from human C1QA: 61-160/245.		Reactivity: Human (predicted: Mouse, Rat)
Purification: affinity purified by Protein A		
Concentration: 1mg/ml		Predicted MW.: 24 kDa
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.		Subcellular Location: Secreted
Background: C1q, a subcomponent of the classical complement pathway, is composed of nine subunits that mediate classical complement activation and thereby play an important role in the immune response. Six of these subunits are disulfide-linked dimers of chains A and B, while three of these subunits, designated C1q-A through C1q-C, are disulfide-linked dimers of chain C. The presence of receptors for C1q on effector cells modulates its activity, which may be antibody-dependent or independent. Macrophages are the primary source of C1q, while anti-inflammatory drugs as well as cytokines differentially regulate expression of the mRNA as well as the protein. However, its ability to modulate the interaction of platelets with collagen and immune complexes suggests C1q influences homeostasis as well as other immune activities, and perhaps thrombotic complications resulting from immune injury. Defects in C1q-A, C1q-B and C1q-C cause inactivation of the classical pathway, leading to a rare genetic disorder characterized by lupus-like symptoms.		

— VALIDATION IMAGES —

Sample: K562(Human) Cell Lysate at 30 ug
 Primary: Anti-C1Q (bs-11336R) at 1/1000 dilution
 Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution Predicted band size: 24 kD
 Observed band size: 25 kD



Paraformaldehyde-fixed, paraffin embedded (human brain glioma); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (C1Q) Polyclonal Antibody, Unconjugated (bs-11336R) at 1:400 overnight at 4°C, followed by operating according to SP Kit(Rabbit) (sp-0023) instructions and DAB staining.



Tissue/cell: human lung carcinoma; 4% Paraformaldehyde-fixed and paraffin-embedded; Antigen retrieval: citrate buffer (0.01M, pH 6.0), Boiling bathing for 15min; Block endogenous peroxidase by 3% Hydrogen peroxide for 30min; Blocking buffer (normal goat serum, C-0005) at 37°C for 20 min; Incubation: Anti-C1Q Polyclonal Antibody, Unconjugated(bs-11336R) 1:200, overnight at 4°C, followed by conjugation to the secondary antibody(SP-0023) and DAB(C-0010) staining

— SELECTED CITATIONS —

- **[IF=7.109]** Wu Yutong. et al. Reduced osteoclast-derived apoptotic bodies in bone marrow characterizes the

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pathological progression of osteoporosis. CELL DEATH DISCOV. 2023 Apr;9(1):1-9 WB ;Mouse. 37185334

- **[IF=5.156]** Liu, Tao. et al. Microarray-based analysis of renal complement components reveals a therapeutic target for lupus nephritis. Arthritis Res Ther. 2021 Dec;23(1):1-15 IHC ;Human. 34433493