Human C-Reactive Protein/CRP Antibody

Antigen Affinity-purified Polyclonal Sheep IgG
Catalog Number: AF1707

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human C-Reactive Protein/CRP in direct ELISAs and Western blots. In Western blots, approximately 35% cross-reactivity with recombinant mouse CRP and 10% cross-reactivity with recombinant rat CRP is observed.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human C-Reactive Protein/CRP Phe17-Pro224 Accession # P02741
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

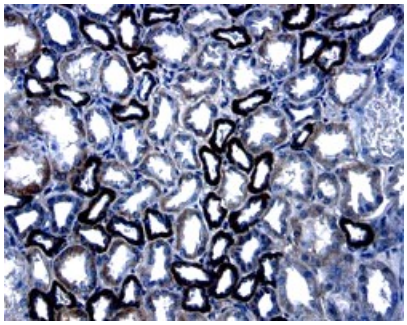
APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Human C-Reactive Protein/CRP (Catalog # 1707-CR)
Immunohistochemistry	5-15 µg/mL	See Below

DATA

Immunohistochemistry



C-Reactive Protein/CRP in Human Kidney Cancer Tissue. C-Reactive Protein/CRP was detected in immersion fixed paraffin-embedded sections of human kidney cancer tissue using 1.7 µg/mL Human C-Reactive Protein/CRP Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1707) overnight at 4 °C. Tissue was stained with the Anti-Sheep HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS019) and counterstained with hematoxylin (blue). Specific labeling was localized to epithelial cells in tubules. View our protocol for Chromogenic IHC Staining of Paraffin-embedded Tissue Sections.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month from date of receipt, 2 to 8 °C, reconstituted. ● 6 months from date of receipt, -20 to -70 °C, reconstituted.

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BACKGROUND

CRP is a member of the pentraxin family of proteins that are characterized by a cyclic pentameric structure. Human CRP gene encodes a 224 amino acids precursor. The mature human CRP protein has 206 amino acids that are non-covalently linked to form the pentameter. Human CRP shares 71% and 64% amino acid sequence homology with mouse and rat respectively.

CRP, synthesized by hepatocytes, is a major acute phase serum protein in human. IL-6, IL-1 and glucocorticoids are the major inducer of the CRP gene. In response to infection, inflammation or tissue damage, the level of CRP in human serum can increase 1,000-fold within 24-48 hours. It will come back to base level of less than 1 µg/mL very fast. Human CRP is an acute-phase serum protein that plays a role in the first line in host innate host defense. Like other pentraxins, CRP exhibits Ca^{++} - dependent binding to ligands. Phosphocholine (PCh), a constituent of many bacterial and fungal walls, is a principal ligand of CRP. CRP also binds to the membrane of injured cells, membrane and nuclear components of necrotic and apoptotic cells. Upon binding with the ligands, CRP is recognized by C1q and initiates the activation of complement cascade. Ligand bound CRP also binds to Fcγ RI and Fcγ RIIa on phagocytes and activates phogocytotic responses. In addition to phogocytosis, CRP also can induce production of hydrogen peroxide and inflammatory cytokines, such as IL-1, IL-6 and TNF-α by monocytes. With these functions, human CRP is an important serum protein for anti-bacterial pathogen and clearance of damaged and apoptotic cells. However, in mouse, CRP is expressed at very low level and is not an acute phase reactant. Serum amyloid P component (SAP), another pentraxin, is the major acute phase serum protein in mice. It has been shown that high levels of CRP in humans is associated with an increased risk of cardiovascular diseases.

References:

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2. Volanakis, J.E. (2001) Molecular Immunology **38**:189.
3. Bharadwaj, D. *et al.* (1999) J. Experimental Medicine **190**:585.
4. Ballou S.P. and G. Lozanski (1992) Cytokine **4**:361.
5. Danesh, J. *et al.* (2004) N. Engl. J. Med. **350**:1387.