

## DESCRIPTION

<b>Species Reactivity</b>	Rat
<b>Specificity</b>	Detects rat C-Reactive Protein/CRP in direct ELISAs and Western blots. In Western blots, approximately 20% cross-reactivity with recombinant mouse CRP is observed and 10% cross-reactivity with recombinant human CRP is observed.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Antigen Affinity-purified
<b>Immunogen</b>	Mouse myeloma cell line NS0-derived recombinant rat C-Reactive Protein/CRP His20-Ser230 Accession # P48199
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

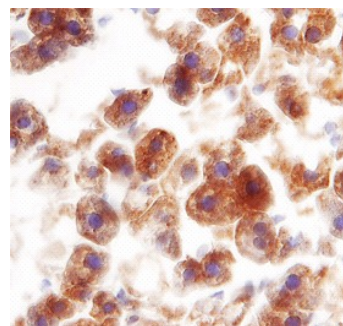
## APPLICATIONS

**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.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Western Blot</b>	0.1 µg/mL	Recombinant Rat C-Reactive Protein/CRP (Catalog # 1744-CR)
<b>Immunohistochemistry</b>	5-15 µg/mL	See Below

## DATA

### Immunohistochemistry



#### C-Reactive Protein/CRP in Rat Liver.

C-Reactive Protein/CRP was detected in perfusion fixed frozen sections of rat liver using Goat Anti-Rat C-Reactive Protein/CRP Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1744) at 5 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). Specific labeling was localized to the cytoplasm of hepatocytes. View our protocol for [Chromogenic IHC Staining of Frozen Tissue Sections](#).

## PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.2 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>1 month from date of receipt, 2 to 8 °C, reconstituted.</li> <li>6 months from date of receipt, -20 to -70 °C, reconstituted.</li> </ul>

## BACKGROUND

C-Reactive Protein (CRP) is a member of the pentraxin family of proteins that are characterized by a cyclic pentameric structure. The rat CRP gene encodes a 230 amino acid (aa) precursor with a signal peptide of 19 aa and the mature polypeptide of 211 aa. Rat CRP shares 64% and 70% aa sequence homology with human and mouse CRP respectively. Human, mouse and rabbit CRP are non-glycosylated proteins, and the units are non-covalently linked to form the pentamer. In contrast, rat CRP is a glycoprotein and contains a covalently linked dimer in the pentamer. CRP exhibits Ca<sup>++</sup>-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, the 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 phagocytotic responses. In addition to phagocytosis, CRP also induces the production of hydrogen peroxide and inflammatory cytokines, such as IL-1, IL-6 and TNF-α. In human and rabbits, CRP is an important acute-phase protein that plays a role in the first line of host innate defense. The level of plasma CRP at basal conditions in human and rabbits is very low, and can increase 1,000-fold within 24-48 hours in response to infection, inflammation or tissue damage. In rats, CRP exists at a high level at basal conditions and only increases about 2-fold in response to injury. CRP is not a typical acute-phase protein in rat and is a minor component in response to injury. In mice, CRP is expressed at very low levels and is not an acute phase reactant. Serum amyloid P component (SAP), another pentraxin, is an acute phase serum protein in mice.

## References:

1. Mohammad, R. *et al.* (1992) J. Biol. Chem. **267**:2947.
2. Sambasivam, H. *et al.* (1993) J. Biol. Chem. **268**:10007.
3. Padilla, N.D. *et al.* (2003) Immunology **109**:564.
4. Volanakis, J.E. (2001) Molecular Immunology **38**:189.