ProductInformation

Monoclonal Anti-Human C-Reactive Protein
Clone CRP-8
Mouse Ascites Fluid
Product No. C 1688
Lot 053H4834

Monoclonal Anti-Human C-Reactive Protein (mouse IgG1 isotype) is derived from the CRP-8 hybridoma produced by the fusion of mouse myeloma cells and splenocytes from BALB/c mice immunized with purifed human C-reactive protein. The isotype is determined using the Sigma ImmunoType™ Kit (Sigma Stock No. ISO-1) and by a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents (Sigma Stock No. ISO-2). The product is provided as ascites fluid with 0.1% sodium azide (see MSDS)* as a preservative.

Specificity
Monoclonal Anti-Human C-Reactive Protein (CRP) recognizes an epitope located on the 24 kD subunit of denatured and reduced CRP in an immunoblotting technique. It does not cross react with human serum amyloid P component (SAP), human haptoglobin, human α-1-acid glycoprotein and human IgG, nor with CRP from Limulus. The product displays its reactivity against CRP (native and denatured-reduced) in ELISA, dot-blot and immunoblotting.

Description
C-Reactive Protein (CRP) is a major acute phase reactant synthesized primarily in the liver hepatocytes.[1] It is a pentraxin (cyclic pentameric protein) compound of five identical nonglycosylated subunits of 206 amino acids each (m.w. 24 kD), that are bound noncovalently to form the physiologic CRP molecule (m.w. 117.5 kD). CRP was discovered on the basis of its Ca^{++} ion-dependent binding to the C-polysaccharide of pneumococcal cell wall that occurs via the Ca^{++}-dependent site for phosphorylcholine (PC), on each of the subunits of CRP. The CRP molecule has a striking homology with Serum Amyloid P Component (SAP), and regions of sequence homology with the nucleoplasmin/B23 family of proteins.[2] The precise biological function of CRP is not known. CRP mediates activities associated with preimmune nonspecific host resistance. It is opsonic, an initiator of the classical complement cascade and an activator of monocytes/macrophages.[3] Cleavage of CRP by neutrophil-derived proteases leads to the production of peptides that have immunomodulating actions. CRP also binds to several nuclear components including chromatin, histones and snRNP, suggesting that it may play a role as a scavenger during cell necrosis. In addition, CRP binds, through the Ca^{++}-dependent PC-binding site, to many other body components including phospholipids, lecithin, sphingomyelin, a variety of monophosphate esters, low density lipoprotein,[4] fibronectin and to the basement membrane protein laminin. CRP binding to polycations has also been demonstrated, but this binding does not appear to occur through the PC-binding site on CRP. In humans, normal blood levels of CRP are less than 200 ng/ml, but rise several hundredfold during the first 6-24 hrs of an acute inflammation or after tissue injury.

Uses
Monoclonal Anti-Human C-Reactive Protein may be used for the localization of C-Reactive Protein and SAP using various immunochemical assays such as ELISA, dot-blot and immunoblot.

Titer: 1:6,000
Titer was determined by indirect ELISA using freshly prepared human CRP at 10 µg/ml for coating.

In order to obtain best results in different techniques or preparations, it is recommended that each individual user determine their optimal working dilutions by titration assay.

Storage
For continuous use, store at 2-8 °C. For extended storage, the solution may be frozen in working aliquots. Repeated freezing and thawing is **not** recommended. Storage in "frost-free" freezers is **not** recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use.

* Due to the sodium azide content a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazardous and safe handling practices.

References

1. Gewurz, H., et al., Adv. Int. Med., **27**, 345-372 (1982).
2. DuClos, T.W., et al., J. Immunol., **145**, 3869-3875 (1990).
3. Tseng, Y., et al., Hybridoma, **7**, 185-191 (1988).
4. Nunomura, W. and Hatakeyama, M., Hokkaido J. Med. Sci., **65**, 474-480 (1990).