

DESCRIPTION

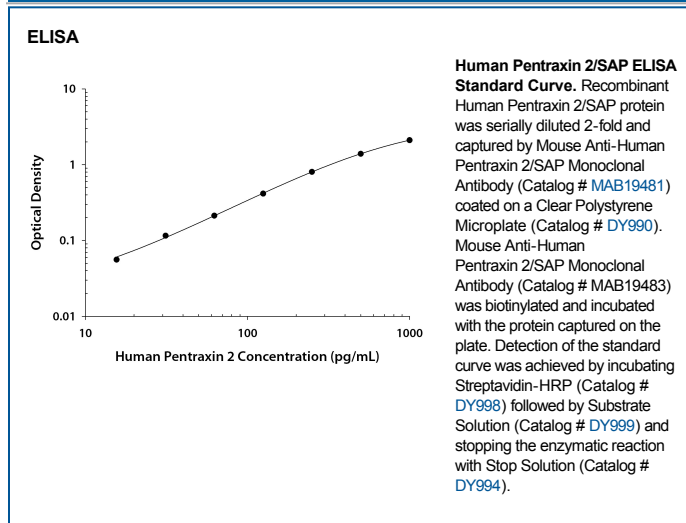
Species Reactivity	Human
Specificity	Detects human Pentraxin 2/SAP in direct ELISAs.
Source	Recombinant Monoclonal Mouse IgG ₁ Clone # 910135R
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Pentraxin 2/SAP His20-Val233 Accession # P02743
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

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.

ELISA	This antibody functions as an ELISA detection antibody when paired with Mouse Anti-Human Pentraxin 2/SAP Monoclonal Antibody (Catalog # MAB19481). <i>This product is intended for assay development on various assay platforms requiring antibody pairs. We recommend the Human Pentraxin 2/SAP DuoSet ELISA Kit (Catalog # DY1948-05) for convenient development of a sandwich ELISA.</i>
--------------	---

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Pentraxin 2 (PTX2), also known as Serum Amyloid P Component (SAP), is a secreted serum glycoprotein that is a universal non-fibrillar component of amyloid deposits. These extracellular deposits of insoluble protein fibrils are the result of protein misfolding and can lead to tissue damage and disease (1, 2). PTX2 belongs to the pentraxin (pentaxin) superfamily, whose members have the characteristic pentagonal discoid arrangement of five non-covalently bound subunits. Pentraxins bind to a variety of molecules in a calcium-dependent lectin-like manner through a pattern-recognition-binding site (1, 4, 5). There are two known subfamilies of pentraxins, the classical or short pentraxin subfamily that includes the serum C-reactive Protein (CRP) and PTX2, and the fusion or long pentraxin subfamily whose members contain pentraxin-related carboxyl-terminal halves (1).

PTX2 and CRP share approximately 50% amino acid sequence identity (2, 5). They are produced and secreted by liver hepatocytes and circulates in plasma. Mouse PTX2 is a major acute-phase protein whose plasma concentrations increase dramatically during an acute phase response (2). In human where CRP is the major acute-phase protein, the plasma concentration of human PTX2 remains relatively constant in response to tissue-damage (2, 5).

PTX2 associates ubiquitously with all amyloid deposits that are implicated in a diverse range of diseases including Alzheimer's and prion diseases, type 2 diabetes and various systemic amyloidoses (3, 6, 7). As a non-fibrillar component, PTX2 regulates the solubility of amyloid fibrils and protects them from degradation by proteolytic enzymes and phagocytic cells. In addition to its role in the pathogenesis of amyloidoses, PTX2 also has an important physiological function in innate immunity (8).

References:

1. Goodman, A. *et al.* (1996) *Cytokine Growth Factor Rev.* **7**:191.
2. Steel, D. and A. Whitehead (1994) *Immunol. Today* **15**:81.
3. Hirschfield, G.M. and P.N. Hawkins (2003) *Int. J. Biochem. Cell Biol.* **35**:1608.
4. Emsley, J. *et al.* (1994) *Nature* **367**:338.
5. Mantzouranis, E. *et al.* (1985) *J. Biol. Chem.* **260**:7752.
6. Botto, M. *et al.* (1997) *Nature Medicine* **3**:855.
7. Pepys, M. *et al.* (2002) *Nature* **417**:254.
8. Bharadwaj, D. *et al.* (2001) *J. Immunol.* **166**:6735.