

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived human Pentraxin 2/SAP protein His20-Val223, with a C-terminal 10-His tag Accession # P02743
N-terminal Sequence Analysis	His20
Structure / Form	Noncovalently-linked homopentamer
Predicted Molecular Mass	25 kDa

SPECIFICATIONS

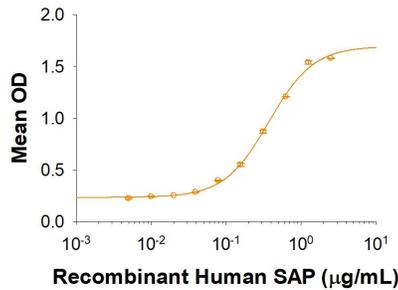
SDS-PAGE	27-33 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human Fcγ RIIA/CD32a (R167) (Catalog # 1330-CD) is immobilized at 2 µg/mL (100 µL/well), the concentration of Recombinant Human Pentraxin 2/SAP that produces 50% of the optimal binding response is 0.175-1 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in MOPS, NaCl and CaCl ₂ with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 250 µg/mL in water.
Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
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. • 3 months, ≤ -20 °C under sterile conditions after reconstitution.

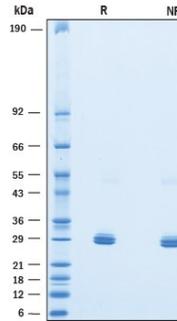
DATA

Binding Activity



When Recombinant Human Fcγ RIIA/CD32a (R167) (Catalog # 1330-CD) is immobilized at 2 µg/mL, 100 µL/well, Recombinant Human SAP binds with an ED₅₀ of 0.175-1 µg/mL.

SDS-PAGE



2 µg/lane of Recombinant Human Pentraxin 2/SAP was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 27-33 kDa.

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 pentaxin superfamily, whose members have the characteristic pentagonal discoid arrangement of five non-covalently bound subunits. Pentaxins bind to a variety of molecules in a calcium-dependent lectin-like manner through a pattern-recognition-binding site (1, 4, 5). Two subfamilies of pentaxins, the classical or short pentaxin subfamily that includes the serum C-reactive protein (CRP) and PTX2, and the fusion or long pentaxin subfamily whose members contain pentaxin-related carboxyl-terminal halves, are known (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). The gene for PTX2 has been localized to human chromosome 1 where it is closely linked to the gene for CRP.

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). It is an opsonin that interacts with all three types of human Fcγ receptors that mediate phagocytosis by human polymorphonuclear leukocytes. It has been proposed that PTX2 may function as an opsonin for a variety of ligands including autoantigens, apoptotic cells, chromatin, DNA, and micro-organisms.

References:

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