Human/Mouse/Rat SP-D Alexa Fluor® 750-conjugated Antibody

Antigen Affinity-purified Polyclonal Goat IgG
Catalog Number: AF1920S
100 µg

DESCRIPTION

Species Reactivity	Human/Mouse/Rat
Specificity	Detects human SP-D in direct ELISAs and human, mouse, and rat SP-D in Western blots.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human SP-D Ala21-Phe375 (Glu22Gly) Accession # P35247
Conjugate	Alexa Fluor 750 Excitation Wavelength: 749 nm Emission Wavelength: 775 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.*

Western Blot	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

SP-D (surfactant protein-D; also PSP-D) is a 43 kDa member of the collectin family of innate immune modulators. It is constitutively secreted by alveolar lining cells and epithelium associated with tubular structures. Its principal components consist of a collagen-like region and a C-terminal carbohydrate recognition domain (CRD), a structure that further places it in a subset of an expanded group of proteins termed defense collagens (1-4). Human SP-D is synthesized as a 375 amino acid (aa) precursor. It contains a 20 aa signal sequence and a 355 aa mature region. The mature molecule is characterized by the presence of a 25 aa N-terminal linking-region, a 177 aa hydroxyproline and hydroxylysine collagen-like domain, a 46 aa coiled-coil segment, and a 106 aa, C-terminal collectin-like C-type lectin domain (CRD) (5, 6). Two additional, potential isoforms exist. One shows a 13 aa N-terminal extension, while the other combines the N-terminal extension with a deletion of aa's 206-375. Mature human SP-D shares 75% and 78% aa identity with mouse and pig SP-D, respectively. Monomeric SP-D is unusual (3). The basic form of SP-D is that of a glycosylated, disulfide-linked 150 kDa trimer that generates an α-helical coiled-coil structure linked to a "head" of three symmetrical CRDs (4, 7). Each CRD recognizes the hydroxides of one monosaccharide (4). Trimerization allows for the discrimination of monosaccharide patterns specific to microbial pathogens (7). Typically, SP-D forms a higher-order 620 kDa, X-shaped dodecamer through disulfide bonds associated with the N-terminus (8). This allows for even finer discrimination of self vs. nonself carbohydrate patterns, and facilitates binding to complex antigens (8, 9). One polymorphism, a Met11-Thr11 transition in human, apparently precludes the formation of oligomers, potentially affecting the ability of affected individuals to interact with microorganisms (9, 10). Finally, SP-D is known to bind both SIRPα and the calreticulin/CD91 complex on macrophages. When the ratio of antigen/pathogen to available CRDs is low, antigen can be bound without occupying all available CRDs. The free CRDs will bind to SIRPα, generating a signal that downmodulates the inflammatory response. When virtually all CRDs are occupied by ligand, however, free CRDs are not available for SIRPα binding. Instead, the dodecamer is depicted to undergo a structural rearrangement, exposing the N-termini of all four linked trimers. This exposed terminus is known to bind to the calreticulin/CD91 complex, an event that initiates inflammation. Thus, it would appear that SP-D allows for a graded response to environmental challenge. SP-D provides a mechanism for the clearance of small antigenic insults without the need for a damaging inflammatory response (3).

PRODUCT SPECIFIC NOTICES

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