

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Serpin A1/ α 1-Antitrypsin in direct ELISAs and Western blots. In Western blots, no cross-reactivity with recombinant human Serpin A3, A4, A5, C1, F1, recombinant mouse Serpin D1, or F2 is observed.
Source	Monoclonal Mouse IgG _{2A} Clone # 202808
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Serpin A1/ α 1-Antitrypsin Glu25-Lys418 Accession # P01009
Conjugate	Alexa Fluor 700 Excitation Wavelength: 675-700 nm Emission Wavelength: 723 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.
Immunocytochemistry	Optimal dilution of this antibody should be experimentally determined.
Immunohistochemistry	Optimal dilution of this antibody should be experimentally determined.
Immunoprecipitation	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

Serpin A1 is the archetypal member of the Serpin superfamily of the serine protease inhibitors (1). As one of the most abundant proteinase inhibitors in the circulation, it is synthesized in the liver and secreted into the bloodstream with the major function to protect tissues against neutrophil elastase. A severe Serpin A1 deficiency leads to several clinical complications such as pulmonary emphysema, juvenile hepatitis, cirrhosis, and hepatocellular carcinoma (2). The deficiency is caused by point mutations in naturally occurring Serpin A1 variants (over 70 are known). For example, the Z variant (Glu342 to Lys) forms intracellular inclusion bodies, is not secreted, and leads to a severe Serpin A1 deficiency (3).

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