

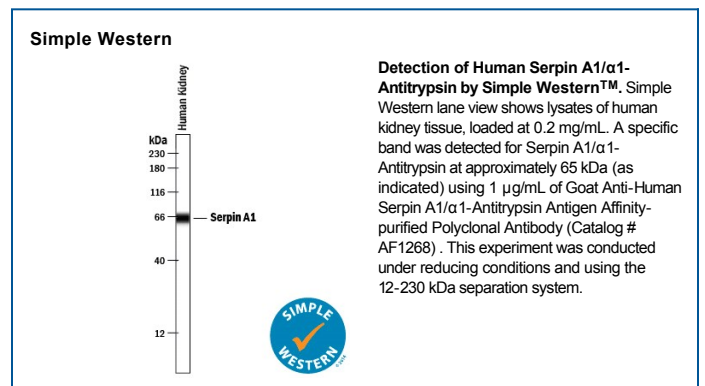
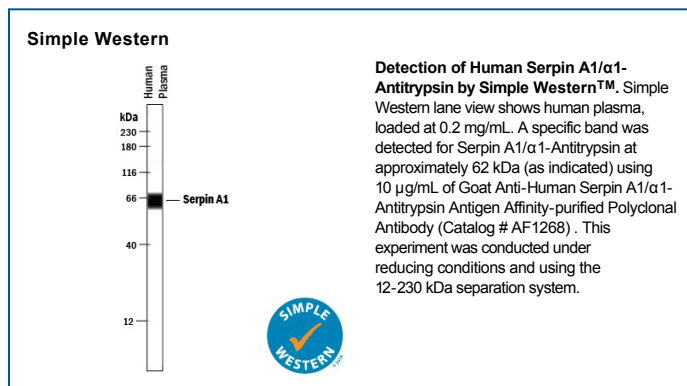
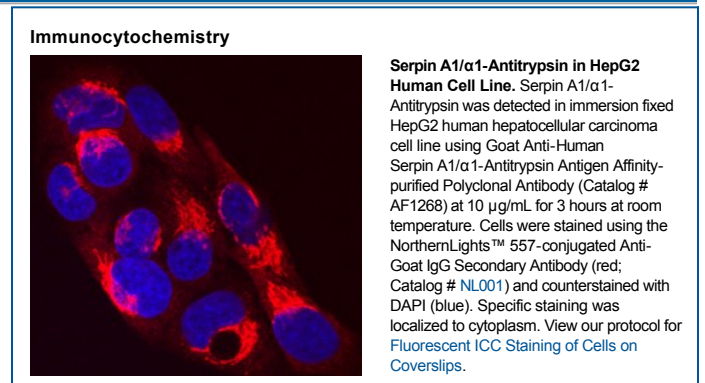
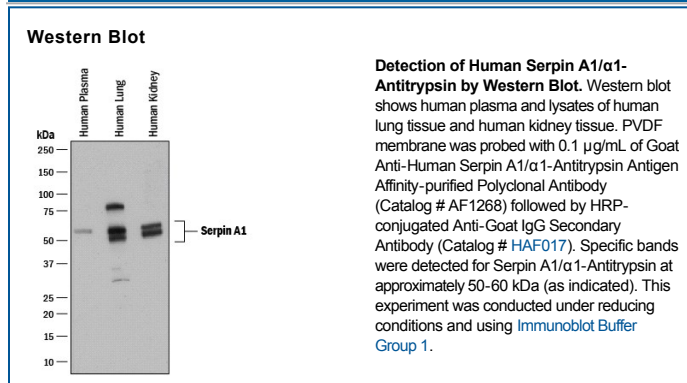
DESCRIPTION	
Species Reactivity	Human
Specificity	Detects human Serpin A1/ α 1-Antitrypsin in direct ELISAs and Western blots. In direct ELISAs, less than 5% cross-reactivity with recombinant human (rh) Serpin A3, rhSerpin A5, rhSerpin A6, rhSerpin A7, rhSerpin A11, and rhSerpin A12 is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Serpin A1/ α 1-Antitrypsin Glu25-Lys418 Accession # P01009
Formulation	Lyophilized from a 0.2 μ m 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 μ m filtered solution in PBS.

APPLICATIONS

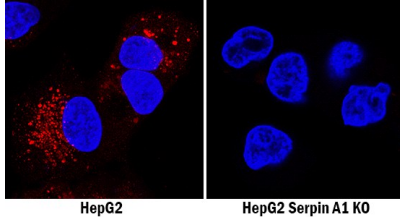
Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	0.1 μ g/mL	See Below
Immunocytochemistry	5-15 μ g/mL	See Below
Immunoprecipitation	25 μ g/mL	Conditioned cell culture medium spiked with Recombinant Human Serpin A1/ α 1-Antitrypsin (Catalog # 1268-PI), see our available Western blot detection antibodies
Simple Western	10 μ g/mL	See Below
Knockout Validated	Serpin A1 is specifically detected in HepG2 human hepatocellular carcinoma parental cell line but is not detectable in Serpin A1 knockout HepG2 cell line.	

DATA



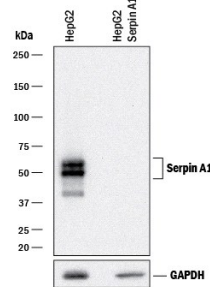
Knockout Validated



Serpin A1/ α 1-Antitrypsin Specificity is Shown by Immunocytochemistry in Knockout Cell Line.

Serpin A1/ α 1-Antitrypsin was detected in immersion fixed HepG2 human hepatocellular carcinoma cell line but is not detected in Serpin A1/ α 1-Antitrypsin knockout (KO) HepG2 cell line using Goat Anti-Human Serpin A1/ α 1-Antitrypsin Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1268) at 9 μ g/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Goat IgG Secondary Antibody (red; Catalog # NL001) and counterstained with DAPI (blue). Specific staining was localized to cytoplasm. View our protocol for [Fluorescent ICC Staining of Cells on Coverslips](#).

Knockout Validated



Western Blot Shows Human Serpin A1/ α 1-Antitrypsin Specificity by Using Knockout Cell Line. Western blot shows lysates of HepG2 human hepatocellular carcinoma parental cell line and Serpin A1/ α 1-Antitrypsin HepG2 knockout cell line (KO). PVDF membrane was probed with 0.25 μ g/mL of Goat Anti-Human Serpin A1/ α 1-Antitrypsin Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1268) followed by HRP-conjugated Anti-Goat IgG Secondary Antibody (Catalog # HAF017). Specific bands were detected for Serpin A1/ α 1-Antitrypsin at approximately 45-55 kDa (as indicated) in the parental HepG2 cell line, but is not detectable in knockout HepG2 cell line. GAPDH (Catalog # AF5718) is shown as a loading control. This experiment was conducted under reducing conditions and using [Immunoblot Buffer Group 1](#).

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 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

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).

References:

1. Silverman, G.A. *et al.* (2001) *J. Biol. Chem.* **276**:33293.
2. Barbour, K.W. *et al.* (2002) *Genomics* **80**:515.
3. Lomas, D.A. *et al.* (2002) *Biochem. Soc. Trans.* **30**:89.