# Human SerpinA1 / A1AT Protein (His Tag)

Catalog Number: 10306-H08H



### **General Information**

#### Gene Name Synonym:

A1A; A1AT; AAT; alpha1AT; MGC23330; MGC9222; PI; PI1; PRO2275; SerpinA1

#### **Protein Construction:**

A DNA sequence encoding the human SerpinA1 (NP\_000286.3) pre-protein (Met 1-Lys 418) was expressed with a C-terminal polyhistidine tag.

Source: Human

Expression Host: HEK293 Cells

**QC** Testing

Purity: > 97 % as determined by SDS-PAGE

#### **Bio Activity:**

Measured by its ability to inhibit trypsin cleavage of a fluorogenic peptide substrate, Mca-RPKPVE-Nval-WRK(Dnp)-NH2 (Anaspec, Catalog#27114) . The IC50 value is < 3.0 nM, as measured in 100 $\mu$ L reaction mixture containing 1.25 ng trypsin (Sigma, Catalog#T1426), 10  $\mu$ M substrate, 50 mM Tris, 10 mM CaCl2, 0.15 M NaCl, pH 7.5.

#### **Endotoxin:**

< 1.0 EU per µg of the protein as determined by the LAL method

#### Stability:

Samples are stable for up to twelve months from date of receipt at -70  $^{\circ}\mathrm{C}$ 

Predicted N terminal: Glu 25

### **Molecular Mass:**

The secreted mature form of recombinant human SerpinA1 consists of 405 amino acids and has a calculated molecular mass of 45.7 kDa. Due to glycosylation, the rhSerpinA1 migrates as an approximately 55-60 kDa protein in SDS-PAGE analysis under reducing conditions.

### Formulation:

Lyophilized from sterile PBS, pH 7.4

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

### **Usage Guide**

### Storage:

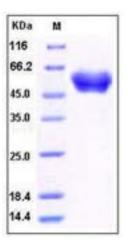
Store it under sterile conditions at  $-20^{\circ}$ C to  $-80^{\circ}$ C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

### Avoid repeated freeze-thaw cycles.

### Reconstitution:

Detailed reconstitution instructions are sent along with the products.

#### SDS-PAGE:



## **Protein Description**

SerpinA1, also known as Alpha-1 antitrypsin (AAT), is a prototype member of the Serpin superfamily of the serine protease inhibitors. This serine protease inhibitor blocks the protease, neutrophil elastase. Alpha-1 antitrypsin is mainly produced in the liver and acts as an antiprotease. Its principal function is to inactivate neutrophil elastase, preventing tissue damage. SerpinA1 (alpha1-antitrypsin), an acute phase protein and the classical neutrophil elastase inhibitor, is localized within lipid rafts in primary human monocytes in vitro. It association with monocytes is inhibited by cholesterol depleting/efflux-stimulating agents (nystatin, filipin, MbetaCD (methyl-beta-cyclodextrin) and oxidized low-density lipoprotein (oxLDL) and conversely, enhanced by free cholesterol. Furthermore, SerpinA1/monocyte association per se depletes lipid raft cholesterol as characterized by the activation of extracellular signal-regulated kinase 2, formation of cytosolic lipid droplets, and a complete inhibition of oxLDL uptake by monocytes. Previous population studies have suggested that heterozygote status for the AAT gene (SerpinA1) is a risk factor for chronic rhinosinusitis with nasal polyposis (CRSwNP). Alpha-1 antitrypsin deficiency is a recently identified genetic disease that occurs almost as frequently as cystic fibrosis. It is caused by various mutations in the SerpinA1 gene, and has numerous clinical implications. Alpha-1 antitrypsin deficiency is an inherited disease affecting the lung and liver. In the liver, alpha-1 antitrypsin deficiency may manifest as benign neonatal hepatitis syndrome; a small percentage of adults develop liver fibrosis, with progression to cirrhosis and hepatocellular carcinoma. Its most important physiologic functions are the protection of pulmonary tissue from aggressive proteolytic enzymes and regulation of pulmonary immune processes.

### References

1.Khnlein T, *et al.* (2008) Alpha-1 antitrypsin deficiency: pathogenesis, clinical presentation, diagnosis, and treatment. Am J Med. 121(1): 3-9. 2.Camelier AA, *et al.* (2008) Alpha-1 antitrypsin deficiency: diagnosis and treatment. J Bras Pneumol. 34(7): 514-27. 3.Subramaniyam D, *et al.* (2010) Cholesterol rich lipid raft microdomains are gateway for acute phase protein, SERPINA1. Int J Biochem Cell Biol. 42(9): 1562-70.

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