

Serpin A1 Protein, Rat, Recombinant (His)

General Information

Synonyms:	SerpinA1;serpin peptidase inhibitor, clade A (α -1 antiproteinase, antitrypsin), member 1; serpin peptidase inhibitor, clade A (alpha-1 antiproteinase, antitrypsin), member 1
Protein Construction:	A DNA sequence encoding the rat SERPINA1 (NP_071964.2) (Met1-Arg411) was expressed with a polyhistidine tag at the C-terminus. Predicted N terminal: Glu 25
Species:	Rat
Expression Host:	HEK293 Cells
Accession:	P17475
Molecular Weight:	45.2 kDa (predicted)

QC Testing

Biological Activity:	Activity testing is in progress. It is theoretically active, but we cannot guarantee it. If you require protein activity, we recommend choosing the eukaryotic expression version first.
Purity:	> 95 % as determined by SDS-PAGE.
Endotoxin:	< 1.0 EU/ μ g of the protein as determined by the LAL method.
Formulation:	Lyophilized from a solution filtered through a 0.22 μ m filter, containing PBS, pH 7.4. Typically, a mixture containing 5% to 8% trehalose, mannitol, and 0.01% Tween 80 is incorporated as a protective agent before lyophilization.

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

It is recommended to store recombinant proteins at -20°C to -80°C for future use. Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Shipping:

In general, Lyophilized powders are shipping with blue ice.

Protein Background

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. Its 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 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.

Reference

Khnlein T,et al.(2008) Alpha-1 antitrypsin deficiency: pathogenesis, clinical presentation, diagnosis, and treatment. Am J Med. 121(1): 3-9.

Camelier AA,et al.(2008) Alpha-1 antitrypsin deficiency: diagnosis and treatment. J Bras Pneumol. 34(7): 514-27.

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Kilty SJ,et al.(2010) Polymorphisms in the SERPINA1 (Alpha-1-Antitrypsin) gene are associated with severe chronic rhinosinusitis unresponsive to medical therapy. Am J Rhinol Allergy. 24(1): e4-9.

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Tel:781-999-4286 E_email:info@targetmol.com Address:34 Washington Street,Wellesley Hills,MA 02481