

Recombinant Rat AAT protein (His Tag)

Catalog Number: PDER100183

Note: Centrifuge before opening to ensure complete recovery of vial contents.

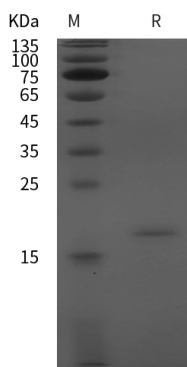
Description

Species	Rat
Source	E.coli-derived Rat AAT protein Glu25-Phe200, with an N-terminal His
Calculated MW	19.3 kDa
Observed MW	20 kDa
Accession	P17475
Bio-activity	Not validated for activity

Properties

Purity	> 95% as determined by reducing SDS-PAGE.
Endotoxin	< 10 EU/mg of the protein as determined by the LAL method
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 °C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

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, 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 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. 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.