

PAI-1 Protein, Rat, Recombinant (His)

General Information

Synonyms:	serpin peptidase inhibitor, clade E (nexin, plasminogen activator inhibitor type 1), member 1; Pai1;SERPINE1;Planh1
Protein Construction:	A DNA sequence encoding the rat SERPINE1 (NP_036752.1) (Met1-Pro402) was expressed with a polyhistidine tag at the C-terminus. Predicted N terminal: Ser 24
Species:	Rat
Expression Host:	HEK293 Cells
Accession:	A6J047
Molecular Weight:	44.2 kDa (predicted); 49 kDa (reducing conditions)

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/μg of the protein as determined by the LAL method.
Formulation:	Lyophilized from a solution filtered through a 0.22 μm 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.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry ice.

Protein Background

Plasminogen activator inhibitor 1, also known as PAI-1, Endothelial plasminogen activator inhibitor, SerpinE1 and PLANH1, is a secreted glycoprotein that belongs to the serpin family. SerpinE1 is the primary physiological inhibitor of the two plasminogen activators urokinase (uPA) and tissue plasminogen activator (tPA). Its rapid interaction with TPA may function as a major control point in the regulation of fibrinolysis. Defects in SerpinE1 are

the cause of plasminogen activator inhibitor-1 deficiency (PAI-1 deficiency) which is characterized by abnormal bleeding due to SerpinE1 defect in the plasma. High concentrations of SerpinE1 have been associated with thrombophilia which is an autosomal dominant disorder in which affected individuals are prone to develop serious spontaneous thrombosis. Studies of PAI-1 have contributed significantly to the elucidation of the protease inhibitory mechanism of serpins, which is based on a metastable native state becoming stabilised by insertion of the RCL into the central beta-sheet A and formation of covalent complexes with target proteases. Greater expression of PAI-1 has been associated with increased survival of cells and resistance to apoptosis. PAI-1 appears to influence apoptosis by decreasing cell adhesion (anoikis) as well as its effect on intracellular signaling. PAI-1, in its active state, also binds to the extracellular protein vitronectin. When in complex with its target proteases, it binds with high affinity to endocytosis receptors of the low density receptor family. The mechanisms of PAI-1 overexpression during obesity are complex, and it is conceivable that several inducers are involved at the same time at several sites of synthesis. PAI-1 is also implicated in adipose tissue development. It suggests that PAI-1 inhibitors serve in the control of atherothrombosis.

Reference

- Alessi MC, et al. (2006) PAI-1 and the metabolic syndrome: links, causes, and consequences. *Arterioscler Thromb Vasc Biol.* 26(10): 2200-7.
- Schneider DJ, et al. (2008) The effect of plasminogen activator inhibitor type 1 on apoptosis. *Thromb Haemost.* 100 (6): 1037-40.
- Dupont DM, et al. (2009) Biochemical properties of plasminogen activator inhibitor-Front Biosci. 14: 1337-61.

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