

Recombinant Human SerpinG1/C1IN Protein (His Tag)

Catalog Number:PKSH031316



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

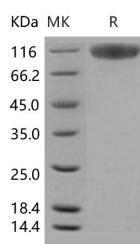
Description

Synonyms	Plasma Protease C1 Inhibitor;C1 Inh;C1Inh;C1 Esterase Inhibitor;C1-Inhibiting Factor;Serpin G1;SERPING1;C1IN;C1NH;HAE1;HAE2
Species	Human
Expression Host	HEK293 Cells
Sequence	Met 1-Ala 500
Accession	NP_000053.2
Calculated Molecular Weight	54.3 kDa
Observed molecular weight	110 kDa
Tag	C-His
Bioactivity	Measured by its ability to inhibit recombinant human complement component C1s (Catalog # 2060-SE) cleavage of a colorimetric peptide substrate, N-carbobenzyloxy-Lys-ThioBenzyl ester (Z-K-SBzl). The IC50 is < 3 nM.

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per µg 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 sterile PBS, pH 7.4 Normally 5 % - 8 % trehalose, mannitol and 0.01 % Tween80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

Plasma protease C1 inhibitor, also known as C1-inhibiting factor, C1-INH, C1 esterase inhibitor, SERPING1 and C1IN, is a serine proteinase inhibitor (serpin) that regulates activation of both the complement and contact systems. By its C-terminal part (serpin domain), characterized by three beta-sheets and an exposed mobile reactive loop, C1-INH binds, and blocks the activity of its target proteases. The N-terminal end (nonserpin domain) confers to C1-INH the capacity to bind

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lipopolysaccharides and E-selectin. Owing to this moiety, C1-INH intervenes in regulation of the inflammatory reaction. The heterozygous deficiency of C1-INH results in hereditary angioedema (HAE). Owing to its ability to modulate the contact and complement systems and the convincing safety profile, plasma-derived C1 inhibitor is an attractive therapeutic protein to treat inflammatory diseases other than HAE. Deficiency of C1 inhibitor results in hereditary angioedema, which is characterized by recurrent episodes of localized angioedema of the skin, gastrointestinal mucosa or upper respiratory mucosa. C1 inhibitor may prove useful in a variety of other diseases including septic shock, reperfusion injury, hyperacute transplant rejection, traumatic and hemorrhagic shock, and the increased vascular permeability associated with thermal injury, interleukin-2 therapy and cardiopulmonary bypass.

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