Human SerpinG1 / C1 inhibitor / C1IN Protein (His Tag)

Catalog Number: **10995-H08H**

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General Information

Gene Name Synonym:

C1IN; C1INH; C1NH; HAE1; HAE2; C1INH; C1nh

Protein Construction:

A DNA sequence encoding the human SERPING1 (NP_000053.2) precursor (Met 1-Ala 500) was expressed with a polyhistidine tag at the C-terminus.

Source: Human

Expression Host: Human Cells

QC Testing

Purity: > 95 % as determined by SDS-PAGE

Bio Activity:

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.

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 ℃

Predicted N terminal: Asn 23

Molecular Mass:

The secreted recombinant human SERPING1 consists of 489 amino acids and predictes a molecular mass of 54.3 kDa. In SDS-PAGE under reducing conditions, it migrates with the apparent molecular mass of 110 kDa due to glycosylation.

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:

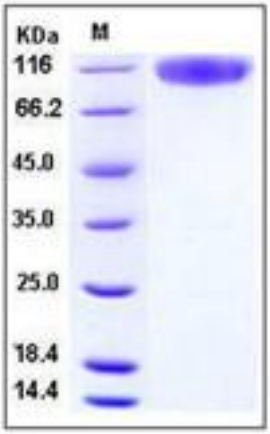
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

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 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.

References

1.Davis AE 3rd. *et al.* (2004) Biological effects of C1 inhibitor. Drug News Perspect. 17(7): 439-46.
2.Cicardi M, *et al.* (2005) C1 inhibitor: molecular and clinical aspects. Springer Semin Immunopathol. 27(3): 286-98.
3.Wouters D, *et al.* (2008) C1 inhibitor: just a serine protease inhibitor? New and old considerations on therapeutic applications of C1 inhibitor. Expert Opin Biol Ther. 8(8): 1225-40.