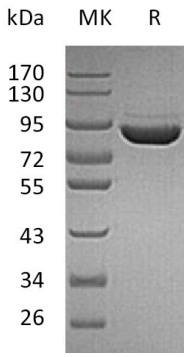
Product Name: Recombinant Human F13A (C-6His)
Catalog #: PHH0612

Summary

Name	Coagulation Factor XIII A Chain/F13a
Purity	Greater than 95% as determined by reducing SDS-PAGE
Endotoxin level	<1 EU/μg as determined by LAL test.
Construction	Recombinant Human Coagulation Factor XIII A Chain is produced by our Mammalian expression system and the target gene encoding Gly39-Met732 is expressed with a 6His tag at the C-terminus.
Accession #	AAH27963.1
Host	Human Cells
Species	Human
Predicted Molecular Mass	80.3 KDa
Formulation	Supplied as a 0.2 μm filtered solution of 20 mM Tris-HCl, 5% Sucrose, 10% Glycerol, 0.02% Tween 80, pH8.0.
Shipping	The product is shipped on dry ice/polar packs. Upon receipt, store it immediately at the temperature listed below.
Stability&Storage	Store at ≤ -70℃, stable for 6 months after receipt. Store at ≤ -70℃, stable for 3 months under sterile conditions after opening. Please minimize freeze-thaw cycles.
Reconstitution	

SDS-PAGE image



Background

Alternative Names	Coagulation Factor XIII A Chain; Coagulation Factor XIIIa; Protein-Glutamine Gamma-Glutamyltransferase A Chain; Transglutaminase A Chain; F13A1; F13A
Background	Coagulation factor XIII is the last zymogen to become activated in the blood

coagulation cascade. Plasma factor XIII is a heterotetramer composed of 2 A subunits and 2 B subunits. The A subunits have catalytic function, and the B subunits do not have enzymatic activity and may serve as plasma carrier molecules. Platelet factor XIII is composed of just 2 A subunits, which are identical to those of plasma origin. Upon cleavage of the activation peptide by thrombin and in the presence of calcium ion, the plasma factor XIII dissociates its B subunits and yields the same active enzyme, factor XIIIa, as platelet factor XIII. This enzyme acts as a transglutaminase to catalyze the formation of gamma-glutamyl-epsilon-lysine crosslinking between fibrin molecules, thus stabilizing the fibrin clot. Factor XIII deficiency is classified into two categories: type I deficiency, characterized by the lack of both the A and B subunits; and type II deficiency, characterized by the lack of the A subunit alone. These defects can result in a lifelong bleeding tendency, defective wound healing, and habitual abortion.

Note

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