

Recombinant Human F13a/Factor XIIIa Protein (His Tag)

Catalog No. PKSH033713

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

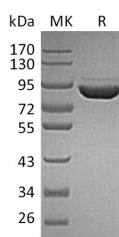
Description

Synonyms	Coagulation Factor XIII A Chain;Coagulation Factor XIIIa;Protein-Glutamine Gamma-Glutamyltransferase A Chain;Transglutaminase A Chain;F13A1;F13A
Species	Human
Expression Host	HEK293 Cells
Sequence	Gly39-Met732
Accession	AAH27963.1
Calculated Molecular Weight	80.3 kDa
Observed molecular weight	80-90 kDa
Tag	C-His
Bioactivity	Not validated for activity

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	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < -20°C.
Formulation	Supplied as a 0.2 µm filtered solution of 50 mM NaCl, 5% Sucrose, 0.3% Histidine, pH 8.0.
Reconstitution	Not Applicable

Data



> 95 % as determined by reducing SDS-PAGE.

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

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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.