Recombinant Human F13a/Factor XIIIa Protein (His Tag)

Catalog Number: PKSH033713

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

SpeciesHumanSourceHEK293 Cells-derived Human	212a: Fastar VIIIa protain Ch.20 Mat722 with an C
Source HEK293 Cells-derived Human	712 a Eastar VIIIa protain Ch.20 Mat 722 with an C
	F13a;Factor XIIIa protein Gly39-Met732, with an C-
terminal His	
Calculated MW 80.3 kDa	
Observed MW 80-90 kDa	
Accession AAH27963.1	
Bio-activity Not validated for activity	
Properties	
Purity >95 % as determined by reduc	ing SDS-PAGE.
Concentration Subject to label value.	
Endotoxin < 1.0 EU per μ g of the protein	as determined by the LAL method.
Storage Store at $<$ -20°C, stable for 6 m	onths. Please minimize freeze-thaw cycles.
Shipping This product is provided as liq	uid. It is shipped at frozen temperature with blue ice/gel
packs. Upon receipt, store it it	nmediately at $< -20^{\circ}$ C.
Formulation Supplied as a 0.2 μm filtered so	lution of 50 mM NaCl, 5% Sucrose, 0.3% Histidine, pH
8.0.	
Data	
kDa MK R	
170	
130	
55	
43	
34	
26	
> 05 % as determined by reducing SDS PAGE	

> 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 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.

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