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F13A1 Antibody (monoclonal) (M02)

Mouse monoclonal antibody raised against a full length recombinant F13A1.
Catalog # AT1976a

Product Information

Application	E
Primary Accession	P00488
Other Accession	BC027963
Reactivity	Human
Host	mouse
Clonality	monoclonal
Isotype	IgG
Clone Names	M1
Calculated MW	83268

Additional Information

Gene ID	2162
Other Names	Coagulation factor XIII A chain, Coagulation factor XIIIa, Protein-glutamine gamma-glutamyltransferase A chain, Transglutaminase A chain, F13A1, F13A
Target/Specificity	F13A1 (AAH27963, 1 a.a. ~ 732 a.a) full-length recombinant protein with GST tag. MW of the GST tag alone is 26 KDa.
Dilution	E~~N/A
Format	Clear, colorless solution in phosphate buffered saline, pH 7.2 .
Storage	Store at -20°C or lower. Aliquot to avoid repeated freezing and thawing.
Precautions	F13A1 Antibody (monoclonal) (M02) is for research use only and not for use in diagnostic or therapeutic procedures.

Background

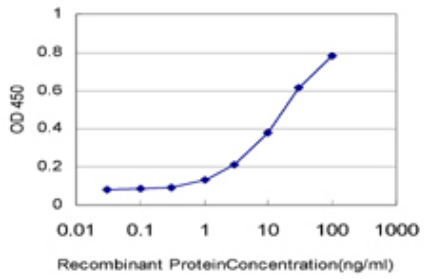
This gene encodes the coagulation factor XIII A subunit. Coagulation factor XIII is the last zymogen to become activated in the blood coagulation cascade. Plasma factor XIII is a heterotetramer composed of 2 A subunits and 2 B subunits. The A subunits have catalytic function, and the B subunits do not have enzymatic activity and may serve as plasma carrier molecules. Platelet factor XIII is comprised only of 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. It also crosslinks alpha-2-plasmin inhibitor, or fibronectin, to the alpha chains of fibrin. 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.

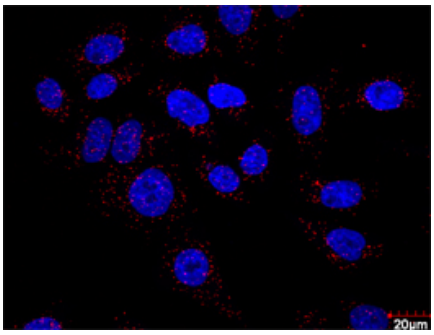
References

A genetic association study of maternal and fetal candidate genes that predispose to preterm prelabor rupture of membranes (PROM). Romero R, et al. Am J Obstet Gynecol, 2010 Jul 29. PMID 20673868.Maternal genes and facial clefts in offspring: a comprehensive search for genetic associations in two population-based cleft studies from Scandinavia. Jugessur A, et al. PLoS One, 2010 Jul 9. PMID 20634891.Variation at the NFATC2 Locus Increases the Risk of Thiazolinedinedione-Induced Edema in the Diabetes REduction Assessment with ramipril and rosiglitazone Medication (DREAM) Study. Bailey SD, et al. Diabetes Care, 2010 Jul 13. PMID 20628086.Allele-allele interaction within the F13A1 gene: a risk factor for ischaemic heart disease in Spanish population. Carreras-Torres R, et al. Thromb Res, 2010 Sep. PMID 20553949.Study of 18 functional hemostatic polymorphisms in mucocutaneous bleeding disorders. Ant?n AI, et al. Ann Hematol, 2010 Nov. PMID 20532885.

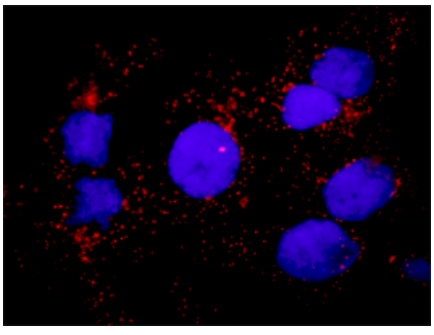
Images



Detection limit for recombinant GST tagged F13A1 is approximately 0.3ng/ml as a capture antibody.



Proximity Ligation Analysis of protein-protein interactions between HSPB1 and F13A1 HeLa cells were stained with anti-HSPB1 rabbit purified polyclonal 1:100 and anti-F13A1 mouse monoclonal antibody 1:50. Each red dot represents the detection of protein-protein interaction complex, and nuclei were counterstained with DAPI (blue).



Proximity Ligation Analysis of protein-protein interactions between HSPB1 and F13A1. Huh7 cells were stained with anti-HSPB1 rabbit purified polyclonal 1:1200 and anti-F13A1 mouse monoclonal antibody 1:50. Each red dot represents the detection of protein-protein interaction complex, and nuclei were counterstained with DAPI (blue).