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Murine Anti-Factor VIII

Clone 8011

Factor VIII (FVIII) is a heterodimer consisting of a heavy chain (ranging in mass from 90 to 200 kDa) bound via metal ions to a light chain (80 kDa). In plasma, FVIII circulates in an inactive form bound to von Willebrand factor. Following activation by factor Xa or thrombin, factor VIIIa can function as cofactor for the enzyme factor IXa in the activation of factor X in the presence of phospholipid and Ca^{2+}. Absent or defective FVIII is the cause of the X-linked recessive bleeding disorder hemophilia A. Mab HFVIII-8011 (also known as 2A9)[1] recognizes the C1 domain of FVIII, inhibits FVIII activation and binding to VWF and phospholipids[2], and displays subnanomolar binding to FVIII by surface plasmon resonance[2].

Description

Antibody Source:	mouse monoclonal, IgG_{2a}
Antigen Species Bound:	human
Specificity:	FVIII C1 domain (residues 2157-2164, 2091-2092)[2]
Immunogen:	B-domain deleted recombinant human FVIII

Formulation and Storage

Purity:	Purified by protein G affinity chromatography from serum-free cell culture supernatant.
Product Formulation:	Lyophilized from a ≥1 mg/ml solution in 20 mM NaH_2PO_4 0.15 M NaCl, 1.0% (w/v) mannitol, pH 7.4. Concentration determined by absorbance measurement at 280 nm and using an extinction coefficient of 1.4 ($\varepsilon_{0.1\%}$).
Reconstitution:	Reconstitute with deionized water.
Storage:	Store lyophilized or reconstituted and aliquoted material at -20° C for prolonged periods. Avoid freeze-thaw cycles. Alternatively, add 0.02% (w/v) sodium azide to reconstituted solution and and store at 4° C.
Country of origin:	USA
Size Options:	0.1 mg or 0.5 mg

Applications

Working Concentration:	Approximately 1-5 µg/ml. Researcher should titer antibody in specific assay.
ELISA:	Binds immobilized human FVIII.
Immunoblotting:	Not recommended.
Inhibition:	Weakly inhibitory in Bethesda assay[2].
Affinity Constant (apparent K_D):	K_D = 0.9 nM, (k_{dis} = 2.2x10^{-4} sec^{-1})[2]

References

[1] R.J. Summers, S.L. Meeks, J.F. Healey, H.C. Brown, E.T. Parker, C.L. Kempton, C.B. Doering, P. Lollar. Factor VIII A3 domain substitution N1922S results in hemophilia A due to domain-specific misfolding and hyposecretion of functional protein. (2011). *Blood*. 117(11):3190-3198.

[2] G. Batsuli, W. Deng, J. F. Helaey, E.T. Parker, W. H. Baldwin, C. Cox, B. Nguyen, J. Kahle, C. Konigs, R. Li, P. Lollar, S.L. Meeks. High affinity, noninhibiotry pathogenic C1 domain antibodies are present in patients with hemophilia A and inhibitors. (2016). *Blood.* 128(16):2055-2067.

[3] J.M. Stewart, A.F. Tarantal, W.J. Hawthorne, E.J. Salvaris, P.J. O'Connell, M.B. Nottle, A.J.F. d'Apice, P.J. Cowan, M. Kearns-Jonker. Rhesus monkeys and baboons develop factor VIII inhibitors in response to porcine endothelial cells or islets. (2014). *Xenotransplantation.* 21(4):341–352.