SANTA CRUZ BIOTECHNOLOGY, INC.

Factor X (CaFX-50): sc-101370

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Hemostasis following tissue injury involves the deployment of essential plasma procoagulants (Prothrombin and factors X, IX, V and VIII), which are involved in a blood coagulation cascade that leads to the formation of insoluble Fibrin clots and the promotion of platelet aggregation. Coagulation Factor X (Stuart Prower factor, FX, F10) is a vitamin K-dependent, single chain serine protease that is synthesized in the liver and circulates as an inactive precursor. The mature form of FX (FXA) is generated by Factor IX A- or Factor VII A-mediated cleavage at the tripeptide sequence, Arg-Lys-Arg, to yield a disulfide linked dimer. Together with the cofactor FVA and Ca^{2+} on the surface of platelets or endothelial cells, Factor X A coordinates as part of the prothrombinase complex, which mediates proteolysis of Prothrombin into active Thrombin. Mutations at the FX locus resulting in Factor X deficiencies can contribute to hemorrhagic diathesis.

REFERENCES

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3. Davie, E.W., et al. 1991. The coagulation cascade: initiation, maintenance, and regulation. Biochemistry 30: 10363-10370.
4. Macedo-Ribeiro, S., et al. 1999. Crystal structures of the membrane-binding C2 domain of human coagulation Factor V. Nature 402: 434-439.
5. Chambers, R.C., et al. 2000. Thrombin is a potent inducer of connective tissue growth factor production via proteolytic activation of protease-activated receptor-1. J. Biol. Chem. 275: 35584-35591.
6. Yang, Y.H., et al. 2006. Antibodies against the activated coagulation Factor X (FXA) in the antiphospholipid syndrome that interfere with the FXA inactivation by antithrombin. J. Immunol. 177: 8219-8225.
7. Todd, T., et al. 2006. Severe Factor X deficiency due to a homozygous mutation (Cys364-Arg) that disrupts a disulphide bond in the catalytic domain. Haemophilia 12: 621-624.
8. Ndonwi, M., et al. 2007. Substitution of the Gla domain in Factor X with that of Protein C impairs its interaction with Factor VII A/tissue factor: lack of comparable effect by similar substitution in Factor IX. J. Biol. Chem. 282: 15632-15644.
9. Al-Hilali, A., et al. 2007. Analysis of the novel Factor X gene mutation Glu51Lys in two families with Factor X-Riyadh anomaly. Thromb. Haemost. 97: 542-545.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.

CHROMOSOMAL LOCATION

Genetic locus: F10 (human) mapping to 13q34.

SOURCE

Factor X (CaFX-50) is a mouse monoclonal antibody raised against Factor X of human origin.

PRODUCT

Each vial contains 200 µg IgG_1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

Factor X (CaFX-50) is recommended for detection of the Gla-domain of Factor X of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for Factor X siRNA (h): sc-40405, Factor X shRNA Plasmid (h): sc-40405-SH and Factor X shRNA (h) Lentiviral Particles: sc-40405-V.

Molecular Weight of Factor X: 61 kDa.

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048.

RESEARCH USE

For research use only, not for use in diagnostic procedures.