

Anti-Coagulation factor X/F10 Antibody (1Q361)

Product Details

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| Ig Type: | Mouse IgG1 |
| Reactivity: | Human |
| Conjugation: | Unconjugated |
| Clone: | 1Q361 |
| Purification: | Protein A |

Applications

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| Verified Activity: | Immunochemical staining of human FX in human cirrhosis with mouse monoclonal antibody (1:60, formalin-fixed paraffin embedded sections). |
| Application: | IHC-P |
| Recommended | IHC-P: 1:50-1:200 |

Properties

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| Stability & Storage: | Store at 2°C-8°C for 1 month. Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles. Preservative-Free. |
| Shipping: | Shipping with blue ice. |

Antigen Details

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| Immunogen: | Recombinant Protein: Human Coagulation Factor X / F10 protein (TMPY-01101) |
| Antigen Species: | Human |
| Synonyms: | coagulation factor X;FXA;coagulation factor 10;FX |
| Biology Area: | Serine Proteases and Regulators |

Research Background

Coagulation factor X, also known as FX, F10, Eponym Stuart-Prower factor, and thrombokinase, is an enzyme of the coagulation cascade. It is one of the vitamin K-dependent serine proteases, and plays a crucial role in the coagulation cascade and blood clotting, as the first enzyme in the common pathway of thrombus formation. Factor X deficiency is one of the rarest of the inherited coagulation disorders. FX deficiency among the most severe of the rare coagulation defects, typically including hemarthroses, hematomas, and umbilical cord, gastrointestinal, and central nervous system bleeding. Factor X is synthesized in the liver as a mature heterodimer formed from a single-chain precursor, and vitamin K is essential for its synthesis. Factor X is activated into factor Xa (FXa) by both factor IX (with its cofactor, factor VIII in a complex known as intrinsic Xase) and factor VII (with its cofactor, tissue factor in a complex known as extrinsic Xase) through cleaving the activation propeptide. As the first member of the final common pathway or thrombin pathway, FXa converts prothrombin to thrombin in the presence of factor Va, Ca²⁺, and phospholipid during blood clotting and cleaves prothrombin in two places (an arg-thr and then an arg-ile bond). This process is optimized when factor Xa is complexed with activated cofactor V in the prothrombinase complex. Inborn deficiency of factor X is very uncommon, and may present with epistaxis (nose bleeds), hemarthrosis (bleeding into joints) and gastrointestinal blood loss. Apart from congenital deficiency, low factor X levels may occur occasionally in a number of disease states. Furthermore, factor X deficiency may be seen in amyloidosis, where factor X is adsorbed to the amyloid fibrils in the vasculature.

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Tel:781-999-4286 E_mail:info@targetmol.com Address:34 Washington Street,Wellesley Hills,MA 02481