SANTA CRUZ BIOTECHNOLOGY, INC.

TF (M-20): sc-18786

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 leading to the formation of insoluble fibrin clots and the promotion of platelet aggregation. Coagulation Factor V (Factor V, FV, proaccelerin, labile factor) is a 2,196 amino acid, single chain glycoprotein that is cleaved by Thrombin to yield an active, Ca^{2+}-dependent dimer that is essential to the blood coagulation cascade. Together with catalytic Factor Xa and Ca^{2+} on the surface of platelets or endothelial cells, Factor Va coordinates into a Prothrombinase complex, which mediates proteolysis of Prothrombin into active Thrombin. Tissue factor (TF, coagulation factor III) is a cell surface glycoprotein that enables cells to initiate blood coagulation cascades, and it functions as a high-affinity receptor for coagulation Factor VII.

REFERENCES

1. Davie, E.W., et al. 1975. Basic mechanisms in blood coagulation. Annu. Rev. Biochem. 44: 799-829.
2. Kane, W.H., et al. 1986. Cloning of a cDNA coding for human Factor V, a blood coagulation factor homologous to Factor VIII and ceruloplasmin. Proc. Natl. Acad. Sci. USA 83: 6800-6804.
3. Jenny, R.J., et al. 1987. Complete cDNA and derived amino acid sequence of human Factor V. Proc. Natl. Acad. Sci. USA 84: 4846-4850.
4. Davie, E.W., et al. 1991. The coagulation cascade: initiation, maintenance and regulation. Biochemistry 30: 10363-10370.
5. Rand, M.D., et al. 1994. Platelet coagulation Factor Va: the major secretory platelet phosphoprotein. Blood 83: 2180-2190.
6. Macedo-Ribeiro, S., et al. 1999. Crystal structures of the membrane-binding C2 domain of human coagulation Factor V. Nature 402: 434-439.

CHROMOSOMAL LOCATION

Genetic locus: F3 (mouse) mapping to 3 G1.

SOURCE

TF (M-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping within an internal region of TF of mouse origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-18786 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

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

RESEARCH USE

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

APPLICATIONS

TF (M-20) is recommended for detection of tissure factor (TF) of mouse origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for TF siRNA (m): sc-40415, TF shRNA Plasmid (m): sc-40415-SH and TF shRNA (m) Lentiviral Particles: sc-40415-V.

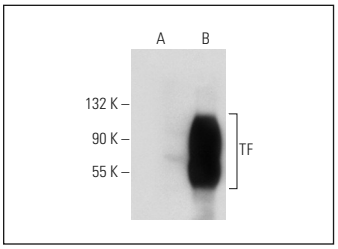
Molecular Weight of TF: 47 kDa.

Positive Controls: TF (m): 293T Lysate: sc-123995 or mouse placenta extract: sc-364247.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

DATA



TF (M-20): sc-18786. Western blot analysis of TF expression in non-transfected: sc-117752 (**A**) and mouse TF transfected: sc-123995 (**B**) 293T whole cell lysates.

SELECT PRODUCT CITATIONS

1. Albrecht, C., et al. 2010. Egr-1 deficiency in bone marrow-derived cells reduces atherosclerotic lesion formation in a hyperlipidaemic mouse model. Cardiovasc. Res. 86: 321-329.