

Product datasheet

Native human Plasminogen protein (FITC) ab92770

1 References

Description

Product name	Native human Plasminogen protein (FITC)
Biological activity	Activity: No plasmin activity detected with the chromogenic substrate S-2251. > 98% conversion to plasmin is observed upon activation with uPA.
Purity	> 95 % SDS-PAGE.
Expression system	Native
Protein length	Full length protein
Animal free	No
Nature	Native
Species	Human
Conjugation	FITC. Ex: 493nm, Em: 528nm

Specifications

Our **Abpromise guarantee** covers the use of **ab92770** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications	SDS-PAGE
Form	Liquid
Additional notes	Protect from light.

Preparation and Storage

Stability and Storage	Shipped on dry ice. Upon delivery aliquot and store at -80°C. Avoid freeze / thaw cycles. pH: 7.40 Constituents: 2.38% HEPES, 0.58% Sodium chloride This product is an active protein and may elicit a biological response in vivo, handle with caution.
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General Info

Function	Plasmin dissolves the fibrin of blood clots and acts as a proteolytic factor in a variety of other
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processes including embryonic development, tissue remodeling, tumor invasion, and inflammation. In ovulation, weakens the walls of the Graafian follicle. It activates the urokinase-type plasminogen activator, collagenases and several complement zymogens, such as C1 and C5. Cleavage of fibronectin and laminin leads to cell detachment and apoptosis. Also cleaves fibrin, thrombospondin and von Willebrand factor. Its role in tissue remodeling and tumor invasion may be modulated by CSPG4. Binds to cells.

Angiostatin is an angiogenesis inhibitor that blocks neovascularization and growth of experimental primary and metastatic tumors in vivo.

Tissue specificity

Present in plasma and many other extracellular fluids. It is synthesized in the liver.

Involvement in disease

Defects in PLG are a cause of susceptibility to thrombosis (THR) [MIM:188050]. It is a multifactorial disorder of hemostasis characterized by abnormal platelet aggregation in response to various agents and recurrent thrombi formation.

Defects in PLG are the cause of plasminogen deficiency (PLGD) [MIM:217090]. PLGD is characterized by decreased serum plasminogen activity. Two forms of the disorder are distinguished: type 1 deficiency is additionally characterized by decreased plasminogen antigen levels and clinical symptoms, whereas type 2 deficiency, also known as dysplasminogenemia, is characterized by normal, or slightly reduced antigen levels, and absence of clinical manifestations. Plasminogen deficiency type 1 results in markedly impaired extracellular fibrinolysis and chronic mucosal pseudomembranous lesions due to subepithelial fibrin deposition and inflammation. The most common clinical manifestation of type 1 deficiency is ligneous conjunctivitis in which pseudomembranes formation on the palpebral surfaces of the eye progresses to white, yellow-white, or red thick masses with a wood-like consistency that replace the normal mucosa.

Sequence similarities

Belongs to the peptidase S1 family. Plasminogen subfamily.

Contains 5 kringle domains.

Contains 1 PAN domain.

Contains 1 peptidase S1 domain.

Domain

Kringle domains mediate interaction with CSPG4.

Post-translational modifications

N-linked glycan contains N-acetylglucosamine and sialic acid. O-linked glycans consist of Gal-GalNAc disaccharide modified with up to 2 sialic acid residues (microheterogeneity).

In the presence of the inhibitor, the activation involves only cleavage after Arg-580, yielding two chains held together by two disulfide bonds. In the absence of the inhibitor, the activation involves additionally the removal of the activation peptide.

Cellular localization

Secreted. Locates to the cell surface where it is proteolytically cleaved to produce the active plasmin. Interaction with HRG tethers it to the cell surface.

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