

## Product datasheet

# Natural Human Plasminogen protein ab62565

### Overview

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<b>Product name</b>	Natural Human Plasminogen protein
<b>Protein length</b>	Full length protein

### Description

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<b>Nature</b>	Native
<b>Source</b>	Native

### Amino Acid Sequence

<b>Species</b>	Human
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### Specifications

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Our [Abpromise guarantee](#) covers the use of **ab62565** in the following tested applications.

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

<b>Applications</b>	SDS-PAGE Functional Studies
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<b>Form</b>	Liquid
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### Preparation and Storage

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<b>Stability and Storage</b>	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles. Preservative: None Constituents: 50% Glycerol
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### General Info

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<b>Function</b>	Plasmin dissolves the fibrin of blood clots and acts as a proteolytic factor in a variety of other 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
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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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