

## Product datasheet

# Native rat Fibrinogen protein ab93072

### Description

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<b>Product name</b>	Native rat Fibrinogen protein
<b>Biological activity</b>	Specific activity: >90% Clottable.
<b>Purity</b>	> 95 % SDS-PAGE. ab93072 is prepared from fresh Rat plasma using several chromatographic steps. Plasminogen depleted by lysine affinity chromatography.
<b>Expression system</b>	Native
<b>Protein length</b>	Full length protein
<b>Animal free</b>	No
<b>Nature</b>	Native
<b>Species</b>	Rat

### Specifications

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Our **Abpromise guarantee** covers the use of **ab93072** 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
<b>Form</b>	Liquid
<b>Additional notes</b>	Thaw protein in a 37°C water bath. Aliquot and freeze the unused portion. Keep fibrinogen at 25-37°C, aliquot and flash freeze unused portion.

### Preparation and Storage

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<b>Stability and Storage</b>	Shipped on dry ice. Upon delivery aliquot and store at -80°C. Avoid freeze / thaw cycles. pH: 7.40 Constituent: 0.588% Sodium citrate This product is an active protein and may elicit a biological response in vivo, handle with caution.
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### General Info

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<b>Function</b>	Fibrinogen has a double function: yielding monomers that polymerize into fibrin and acting as a
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cofactor in platelet aggregation.

**Tissue specificity**

Plasma.

**Involvement in disease**

Defects in FGA are a cause of congenital afibrinogenemia (CAFBN) [MIM:202400]. This is a rare autosomal recessive disorder characterized by bleeding that varies from mild to severe and by complete absence or extremely low levels of plasma and platelet fibrinogen. Note=The majority of cases of afibrinogenemia are due to truncating mutations. Variations in position Arg-35 (the site of cleavage of fibrinopeptide a by thrombin) leads to alpha-dysfibrinogenemias.

Defects in FGA are a cause of amyloidosis type 8 (AMYL8) [MIM:105200]; also known as systemic non-neuropathic amyloidosis or Ostertag-type amyloidosis. AMYL8 is a hereditary generalized amyloidosis due to deposition of apolipoprotein A1, fibrinogen and lysozyme amyloids. Viscera are particularly affected. There is no involvement of the nervous system. Clinical features include renal amyloidosis resulting in nephrotic syndrome, arterial hypertension, hepatosplenomegaly, cholestasis, petechial skin rash.

**Sequence similarities**

Contains 1 fibrinogen C-terminal domain.

**Domain**

A long coiled coil structure formed by 3 polypeptide chains connects the central nodule to the C-terminal domains (distal nodules). The long C-terminal ends of the alpha chains fold back, contributing a fourth strand to the coiled coil structure.

**Post-translational modifications**

The alpha chain is not glycosylated.

Forms F13A-mediated cross-links between a glutamine and the epsilon-amino group of a lysine residue, forming fibronectin-fibrinogen heteropolymers.

About one-third of the alpha chains in the molecules in blood were found to be phosphorylated.

Conversion of fibrinogen to fibrin is triggered by thrombin, which cleaves fibrinopeptides A and B from alpha and beta chains, and thus exposes the N-terminal polymerization sites responsible for the formation of the soft clot. The soft clot is converted into the hard clot by factor XIIIa which catalyzes the epsilon-(gamma-glutamyl)lysine cross-linking between gamma chains (stronger) and between alpha chains (weaker) of different monomers.

Phosphorylation sites are present in the extracellular medium.

**Cellular localization**

Secreted.

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**Please note:** All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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