# abcam

# Product datasheet

# Recombinant Human TGFBI protein ab86218

## 1 Image

**Description** 

Product name Recombinant Human TGFBI protein

Purity > 95 % SDS-PAGE.

purified by using conventional chromatography techniques.

Expression system Escherichia coli

Protein length Protein fragment

Animal free No

Nature Recombinant

**Species** Human

**Sequence** MGTVMDVLKGDNRFSMLVAAIQSAGLTETLNREGVYTVFA

PTNEAFRALP

PRERSRLLGDAKELANILKYHIGDEILVSGGIGALVRLKSLQ

**GDKLEVSL** 

KNNVVSVNKEPVAEPDIMATNGVVHVITNVLQPPA

Amino acids 502 to 636

**Specifications** 

Our Abpromise guarantee covers the use of ab86218 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

**Preparation and Storage** 

**Stability and Storage** Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.

pH: 8.00

Constituent: 0.242% Tris

**General Info** 

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#### **Function**

Binds to type I, II, and IV collagens. This adhesion protein may play an important role in cell-collagen interactions. In cartilage, may be involved in endochondral bone formation.

#### **Tissue specificity**

Highly expressed in the corneal epithelium.

#### Involvement in disease

Defects in TGFBI are the cause of epithelial basement membrane corneal dystrophy (EBMD) [MIM:121820]; also known as Cogan corneal dystrophy or map-dot-fingerprint type corneal dystrophy. EBMD is a bilateral anterior corneal dystrophy characterized by grayish epithelial fingerprint lines, geographic map-like lines, and dots (or microcysts) on slit-lamp examination. Pathologic studies show abnormal, redundant basement membrane and intraepithelial lacunae filled with cellular debris. Although this disorder usually is not considered to be inherited, families with autosomal dominant inheritance have been identified.

Defects in TGFBI are the cause of corneal dystrophy Groenouw type 1 (CDGG1) [MIM:121900]; also known as corneal dystrophy granular type. Inheritance is autosomal dominant. Corneal dystrophies show progressive opacification of the cornea leading to severe visual handicap. Defects in TGFBI are the cause of corneal dystrophy lattice type 1 (CDL1) [MIM:122200]. Inheritance is autosomal dominant.

Defects in TGFBI are a cause of corneal dystrophy Thiel-Behnke type (CDTB) [MIM:602082]; also known as corneal dystrophy of Bowman layer type 2 (CDB2).

Defects in TGFBI are the cause of Reis-Buecklers corneal dystrophy (CDRB) [MIM:608470]; also known as corneal dystrophy of Bowman layer type 1 (CDB1).

Defects in TGFBI are the cause of lattice corneal dystrophy type 3A (CDL3A) [MIM:608471]. CDL3A clinically resembles to lattice corneal dystrophy type 3, but differs in that its age of onset is 70 to 90 years. It has an autosomal dominant inheritance pattern.

Defects in TGFBI are the cause of Avellino corneal dystrophy (ACD) [MIM:607541]. ACD could be considered a variant of granular dystrophy with a significant amyloidogenic tendency. Inheritance is autosomal dominant.

#### Sequence similarities

Contains 1 EMI domain.
Contains 4 FAS1 domains.

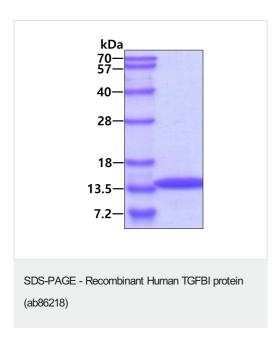
# Post-translational modifications

Gamma-carboxyglutamate residues are formed by vitamin K dependent carboxylation. These residues are essential for the binding of calcium.

#### **Cellular localization**

Secreted > extracellular space > extracellular matrix. May be associated both with microfibrils and with the cell surface.

### **Images**



15% SDS Page analysis of ab86218 (3µg).

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