

BGH3 Rabbit pAb

CatalogNo: YN2221

Key Features

Host Species

- Rabbit

Reactivity

- Human, Mouse

Applications

- WB, ELISA

MW

- 75kD (Observed)

Isotype

- IgG

Recommended Dilution Ratios

WB 1:500-2000**ELISA 1:5000-20000**

Storage

Storage*

-15°C to -25°C/1 year (Do not lower than -25°C)

Formulation

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Basic Information

Clonality

Polyclonal

Immunogen Information

Immunogen

Synthesized peptide derived from human protein . at AA range: 230-310

Specificity

BGH3 Polyclonal Antibody detects endogenous levels of protein.

Target Information

Gene name

TGFB1 BIGH3

Protein Name	Transforming growth factor-beta-induced protein ig-h3 (Beta ig-h3) (Kerato-epithelin) (RGD-containing collagen-associated protein) (RGD-CAP)		
	Organism	Gene ID	UniProt ID
	Human	7045;	Q15582;
	Mouse		P82198;
Cellular Localization	Secreted . Secreted, extracellular space, extracellular matrix . May be associated both with microfibrils and with the cell surface (PubMed:8077289). .		
Tissue specificity	Highly expressed in the corneal epithelium (PubMed:27609313, PubMed:8077289). Expressed in heart, placenta, lung, liver, skeletal muscle, kidney and pancreas (PubMed:8077289).		
Function	<p>Disease: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).,Disease: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.,Disease: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.,Disease:Defects in TGFBI are the cause of corneal dystrophy lattice type 1 (CDL1) [MIM:122200]. Inheritance is autosomal dominant.,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.,Disease: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.,Disease: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).,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.,induction:By TGF-beta.,PTM:Gamma-carboxyglutamate residues are formed by vitamin K dependent carboxylation. These residues are essential for the binding of calcium.,similarity:Contains 1 EMI domain.,similarity:Contains 4 FAS1 domains.,subcellular location:May be associated both with microfibrils and with the cell surface.,tissue specificity:Highly expressed in the corneal epithelium.,</p>		

| Validation Data

| Contact information

Orders: order.cn@immunoway.com
Support: support.cn@immunoway.com
Telephone: 400-8787-807(China)
Website: <http://www.immunoway.com.cn>
Address: 2200 Ringwood Ave San Jose, CA 95131 USA



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BGH3 Rabbit pAb

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