

TGF β RI (PT1069R) PT™ Rabbit mAb

CatalogNo: YM8858 **Recombinant** 

Key Features

Host Species

- Rabbit

Reactivity

- Human, Mouse, Rat

Applications

- WB, IF, IP, ELISA

MW

- 56kD (Calculated)
55kD (Observed)

Isotype

- IgG, Kappa

Storage

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

Formulation PBS, 50% glycerol, 0.05% Proclin 300, 0.05% BSA

Recommended Dilution Ratios

WB 1:2000-1:10000

IF 1:200-1:1000

ELISA 1:5000-1:20000

IP 1:50-1:200

Basic Information

Clonality Monoclonal

Clone Number PT1069R

Immunogen Information

Specificity Endogenous

Target Information

Gene name TGFBR1 ALK5 SKR4

Protein Name TGF-beta receptor type-1

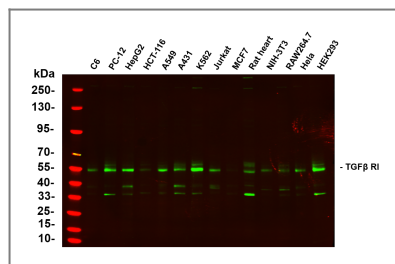
Organism	Gene ID	UniProt ID
Human	7046;	P36897;
Mouse	21812;	Q64729;
Rat		P80204;

Cellular Localization Cell membrane ; Single-pass type I membrane protein . Cell junction, tight junction . Cell surface . Membrane raft .

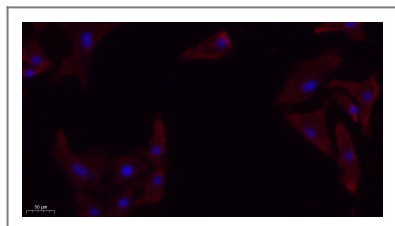
Tissue specificity Found in all tissues examined, most abundant in placenta and least abundant in brain and heart. Expressed in a variety of cancer cell lines (PubMed:25893292).

Function Catalytic activity:ATP + [receptor-protein] = ADP + [receptor-protein] phosphate.,cofactor:Magnesium or manganese.,Disease:Defects in TGFBR1 are the cause of aortic aneurysm familial thoracic type 5 (AAT5) [MIM:608967]. Aneurysms and dissections of the aorta usually result from degenerative changes in the aortic wall. Thoracic aortic aneurysms and dissections are primarily associated with a characteristic histologic appearance known as 'medial necrosis' in which there is degeneration and fragmentation of elastic fibers, loss of smooth muscle cells, and an accumulation of basophilic ground substance.,Disease:Defects in TGFBR1 are the cause of Loeys-Dietz syndrome type 1A (LDS1A) [MIM:609192]; also known as Furlong syndrome or Loeys-Dietz aortic aneurysm syndrome (LDAS). LDS1 is an aortic aneurysm syndrome with widespread systemic involvement. The disorder is characterized by arterial tortuosity and aneurysms, craniosynostosis, hypertelorism, and bifid uvula or cleft palate. Other findings include exotropia, micrognathia and retrognathia, structural brain abnormalities, intellectual deficit, congenital heart disease, translucent skin, joint hyperlaxity and aneurysm with dissection throughout the arterial tree.,Disease:Defects in TGFBR1 are the cause of Loeys-Dietz syndrome type 2A (LDS2A) [MIM:608967]. LDS2 is an aortic aneurysm syndrome with widespread systemic involvement. Physical findings include prominent joint laxity, easy bruising, wide and atrophic scars, velvety and translucent skin with easily visible veins, spontaneous rupture of the spleen or bowel, diffuse arterial aneurysms and dissections, and catastrophic complications of pregnancy, including rupture of the gravid uterus and the arteries, either during pregnancy or in the immediate postpartum period. LDS2 is characterized by the absence of craniofacial abnormalities with the exception of bifid uvula that can be present in some patients.,Function:On ligand binding, forms a receptor complex consisting of two type II and two type I transmembrane serine/threonine kinases. Type II receptors phosphorylate and activate type I receptors which autophosphorylate, then bind and activate SMAD transcriptional regulators. Receptor for TGF-beta.,PTM:Phosphorylated at basal levels in the absence of ligand binding. Activated by multiple phosphorylation, mainly in the GS region.,similarity:Belongs to the protein kinase superfamily.,similarity:Belongs to the protein kinase superfamily. TKL Ser/Thr protein kinase family. TGFB receptor subfamily.,similarity:Contains 1 GS domain.,similarity:Contains 1 protein kinase domain.,subunit:Interacts with CD109. The unphosphorylated protein interacts with FKBP1A and is stabilized in the inactive conformation. Phosphorylation of the GS region abrogates FKBP1A binding. Interacts with SMAD2 when phosphorylated on several residues in the GS region.,tissue specificity:Found in all tissues examined, most abundant in placenta and least abundant in brain and heart.,

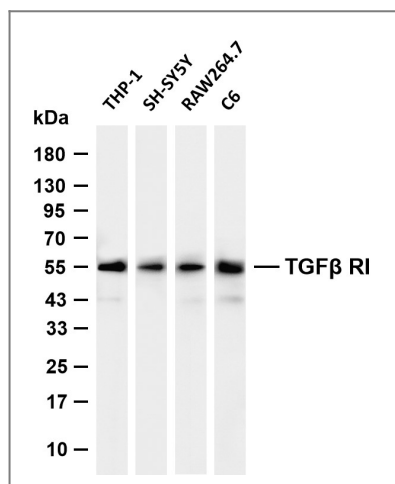
Validation Data



Various whole cell lysates were separated by 4-20% SDS-PAGE, and the primary antibody was used at 4~C, over night with a 1:5000 dilution . The Dylight 800-conjugated Goat anti-Rabbit antibody(Cat:RS23920) was used to detect the antibody. Lane1:C6- Rat glioma cells Lane2: PC-12 - Pheochromocytoma in rats Lane3: HepG2 - Human hepatocellular carcinoma Lane4: HCT-116 - Human colon cancer Lane5: A549 - Human lung adenocarcinoma Lane6: A431 - Human skin squamous cell carcinoma Lane7: K562 - Human chronic myeloid leukemia Lane8: Jurkat - Human acute T cell leukemia cells Lane9: MCF7 - Human breast cancer Lane10: Rat heart - Human neuroblastoma cells Lane11: NIH-3T3 - NIH mouse fibroblasts Lane12: RAW264.7 - Mouse mononuclear macrophage leukemia cells Lane13: HeLa - Human cervical cancer Lane14: HEK293 - Human normal embryonic kidney



Immunofluorescence analysis of A549. 1,primary Antibody(red) was diluted at 1:200(4°C overnight). 2, Goat Anti Rabbit IgG (H&L) - Alexa Fluor 594 Secondary antibody was diluted at 1:1000(room temperature, 50min).3, DAPI(blue) 10min.



Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-TGFβ RI (PT1069R) antibody. The HRP-conjugated Goat anti-Rabbit IgG (H + L) antibody was used to detect the antibody. Lane 1: THP-1 Lane 2: SH-SY5Y Lane 3: RAW264.7 Lane 4: C6 Predicted band size: 56kDa Observed band size: 55kDa

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PT™ Rabbit mAb