Product Name: TGFBR2 (Phospho-Tyr284) Rabbit Polyclonal Antibody
Catalog #: APRab06096

Summary

Production Name	TGFBR2 (Phospho-Tyr284) Rabbit Polyclonal Antibody
Description	Rabbit Polyclonal Antibody
Host	Rabbit
Application	WB
Reactivity	Human,Mouse,Rat

Performance

Conjugation	Unconjugated
Modification	Phospho Antibody
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
Buffer	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Purification	Affinity purification

Immunogen

Gene Name	TGFBR2
Alternative Names	TGF-beta receptor type-2 (TGFR-2) (EC 2.7.11.30) (TGF-beta type II receptor) (Transforming growth factor-beta receptor type II) (TGF-beta receptor type II) (TbetaR-II)
Gene ID	7048.0
SwissProt ID	P37173.

Application

Dilution Ratio	WB 1:500-2000
Molecular Weight	62kD

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Background

catalytic activity:ATP + [receptor-protein] = ADP + [receptor-protein] phosphate.,cofactor:Magnesium or manganese.,disease:Defects in TGFBR2 are a cause of esophageal cancer [MIM:133239].,disease:Defects in TGFBR2 are the cause of aortic aneurysm familial thoracic type 3 (AAT3) [MIM:610380]. 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' or 'Erdheim cystic medial necrosis' in which there is degeneration and fragmentation of elastic fibers, loss of smooth muscle cells, and an accumulation of basophilic ground substance. AAT3 is an autosomal dominant disorder with reduced penetrance and variable expression.,disease:Defects in TGFBR2 are the cause of hereditary non-polyposis colorectal cancer type 6 (HNPCC6) [MIM:190182]. Mutations in more than one gene locus can be involved alone or in combination in the production of the HNPCC phenotype (also called Lynch syndrome). Most families with clinically recognized HNPCC have mutations in either MLH1 or MSH2 genes. HNPCC is an autosomal, dominantly inherited disease associated with marked increase in cancer susceptibility. It is characterized by a familial predisposition to early onset colorectal carcinoma (CRC) and extra-colonic cancers of the gastrointestinal, urological and female reproductive tracts. HNPCC is reported to be the most common form of inherited colorectal cancer in the Western world, and accounts for 15% of all colon cancers. Cancers in HNPCC originate within benign neoplastic polyps termed adenomas. Clinically, HNPCC is often divided into two subgroups. Type I: hereditary predisposition to colorectal cancer, a young age of onset, and carcinoma observed in the proximal colon. Type II: patients have an increased risk for cancers in certain tissues such as the uterus, ovary, breast, stomach, small intestine, skin, and larynx in addition to the colon. Diagnosis of classical HNPCC is based on the Amsterdam criteria: 3 or more relatives affected by colorectal cancer, one a first degree relative of the other two; 2 or more generation affected; 1 or more colorectal cancers presenting before 50 years of age; exclusion of hereditary polyposis syndromes. The term "suspected HNPCC" or "incomplete HNPCC" can be used to describe families who do not or only partially fulfill the Amsterdam criteria, but in whom a genetic basis for colon cancer is strongly suspected. HNPCC6 is a type of colorectal cancer complying with the clinical criteria of HNPCC, except that the onset of cancer was beyond 50 years of age in all cases.,disease:Defects in TGFBR2 are the cause of Loeys-Dietz syndrome type 1B (LDS1B) [MIM:610168]. 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 exotropy, 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 TGFBR2 are the cause of Loeys-Dietz syndrome type 2B (LDS2B) [MIM:610380]; formerly Marfan syndrome type 2. 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

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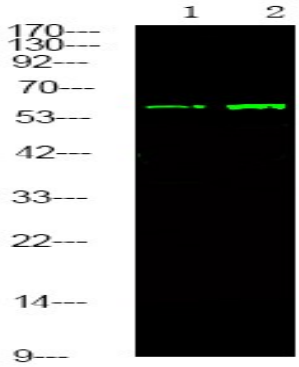
transmembrane serine/threonine kinases. Type II receptors phosphorylate and activate type I receptors which autophosphorylate, then bind and activate SMAD transcriptional regulators. Receptor for TGF-beta.,PTM:Phosphorylated on a Ser/Thr residue in the cytoplasmic domain.,similarity:Belongs to the protein kinase superfamily.,similarity:Belongs to the protein kinase superfamily. TKL Ser/Thr protein kinase family. TGFB receptor subfamily.,similarity:Contains 1 protein kinase domain.,subunit:Binds to DAXX. Interacts with TCTEX1D4.,

Research Area

Image Data



Western Blot analysis of 1 A549 cell, 2 LPS 100ng/mL 30min treated ,using primary antibody at 1:1000 dilution. Secondary antibody was diluted at 1:10000

Note

For research use only.