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Rabbit Anti-phospho-TGF beta Receptor II (Tyr336) antibody

SL18064R

Product Name:	phospho-TGF beta Receptor II (Tyr336)
Chinese Name:	磷酸化转移生长因子β受体2抗体
Alias:	TGF beta Receptor II (phospho Tyr336); p-TGFβ RII (Tyr 336);p-TGFβ RII (Tyr336) AAT3; FAA3; LDS1B; LDS2B; MFS2; RIIC; TAAD2; TbetaR II; TbetaR-II; TGF beta receptor type II; TGF beta receptor type IIB; TGF beta type II receptor; TGF-beta receptor type II; TGF-beta receptor type-2; TGF-beta type II receptor; TGFB R2; TGFbeta - RII; TGFbeta RII; TGFBR2; TGFR-2; TGFR2_HUMAN; Transforming growth factor beta receptor II; Transforming growth factor beta receptor type II; Transforming growth factor beta receptor type IIC; Transforming growth factor, beta receptor II (70/80kDa); Transforming growth factor-beta receptor type II.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,
Applications:	ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800ICC=1:100-500IF=1:100-500（Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	62kDa
Cellular localization:	The cell membrane
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthesised phosphopeptide derived from human TGF beta Receptor II around the phosphorylation site of Tyr336:QE(p-Y)LT
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized

	antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
PubMed:	PubMed
Product Detail:	This gene encodes a member of the Ser/Thr protein kinase family and the TGFB receptor subfamily. The encoded protein is a transmembrane protein that has a protein kinase domain, forms a heterodimeric complex with another receptor protein, and binds TGF-beta. This receptor/ligand complex phosphorylates proteins, which then enter the nucleus and regulate the transcription of a subset of genes related to cell proliferation. Mutations in this gene have been associated with Marfan Syndrome, Loeys-Deitz Aortic Aneurysm Syndrome, and the development of various types of tumors. Alternatively spliced transcript variants encoding different isoforms have been characterized. [provided by RefSeq, Jul 2008] **Function:** Transmembrane serine/threonine kinase forming with the TGF-beta type I serine/threonine kinase receptor, TGFBR1, the non-promiscuous receptor for the TGF-beta cytokines TGFB1, TGFB2 and TGFB3. Transduces the TGFB1, TGFB2 and TGFB3 signal from the cell surface to the cytoplasm and is thus regulating a plethora of physiological and pathological processes including cell cycle arrest in epithelial and hematopoietic cells, control of mesenchymal cell proliferation and differentiation, wound healing, extracellular matrix production, immunosuppression and carcinogenesis. The formation of the receptor complex composed of 2 TGFBR1 and 2 TGFBR2 molecules symmetrically bound to the cytokine dimer results in the phosphorylation and the activation of TGFRB1 by the constitutively active TGFBR2. Activated TGFBR1 phosphorylates SMAD2 which dissociates from the receptor and interacts with SMAD4. The SMAD2-SMAD4 complex is subsequently translocated to the nucleus where it modulates the transcription of the TGF-beta-regulated genes. This constitutes the canonical SMAD-dependent TGF-beta signaling cascade. Also involved in non-canonical, SMAD-independent TGF-beta signaling pathways. **Subcellular Location:** Cell membrane. **Tissue Specificity:** Phosphorylated on a Ser/Thr residue in the cytoplasmic domain. **DISEASE:** Defects in TGFBR2 are the cause of hereditary non-polyposis colorectal cancer type 6 (HNPCC6) [MIM:614331]. 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. Defects in TGFBR2 are a cause of esophageal cancer (ESCR) [MIM:133239]. 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.
Defects in TGFBR2 are the cause of Loeys-Dietz syndrome type 2B (LDS2B) [MIM:610380]. 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. Note=TGFBR2 mutations Cys-460 and His-460 have been reported to be associated with thoracic aortic aneurysms and dissection (TAAD). This phenotype, also known as thoracic aortic aneurysms type 3 (AAT3), is distinguised from LDS2B by having aneurysms restricted to thoracic aorta. As individuals carrying these mutations also exhibit descending aortic disease and aneurysms of other arteries (PubMed:16027248), they have been considered as LDS2B by the OMIM resource.

Similarity:
Belongs to the protein kinase superfamily.
TKL Ser/Thr protein kinase family. TGFB receptor subfamily.
Contains 1 protein kinase domain.

SWISS:
P37173

Gene ID:

	7048 **Database links:** Entrez Gene: 7048 Human Entrez Gene: 21813 Mouse Omim: 190182 Human SwissProt: P37173 Human SwissProt: Q62312 Mouse Unigene: 604277 Human Unigene: 82028 Human Unigene: 172346 Mouse **Important Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.