# TGF β Receptor II Rabbit mAb

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| **Catalog No** | YP-rAb-17921 |
| **Isotype** | IgG |
| **Reactivity** | Human,Mouse,Rat |
| **Applications** | WB,IHC,IF,IP,ELISA |
| **Gene Name** | TGFBR2 |
| **Protein Name** | TGF-beta receptor type-2 |
| **Purification Process** | Protein A |
| **Specificity** | Endogenous |
| **Formulation** | PBS, 50% glycerol, 0.05% Proclin 300, 0.05%BSA |
| **Source** | Monoclonal, Rabbit,IgG |
| **Dilution** | IHC 1:200-1:500; WB 1:1000-1:5000; IF 1:200-1:1000; ELISA 1:5000-1:20000; IP 1:50-1:200, Note: For IHC, we suggest antigen retrieval with TE buffer pH 9.0 |
| **Concentration** | 0.5 mg/ml |
| **Purity** | ≥90% |
| **Storage Stability** | -15° C to -25° C/1 year(Do not lower than -25° C) |
| **Synonyms** | TGFBR2 ; TGF-beta receptor type-2 ; TGFR-2 ; TGF-beta type II receptor ; Transforming growth factor-beta receptor type II ; TGF-beta receptor type II ; TbetaR-II |
| **Observed Band** | 65kD |
| **Calculated Molecular Weight** | 65kD |
| **Cell Pathway** | Membrane |
| **Tissue Specificity** | Cerebellum,Colon,Epithelium,Glial cell,Liver, |
| **Function** | 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 |

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ICO-IP检测 | 切片 | 染色 | 免疫组化 | 免疫荧光 | 透射电镜全套
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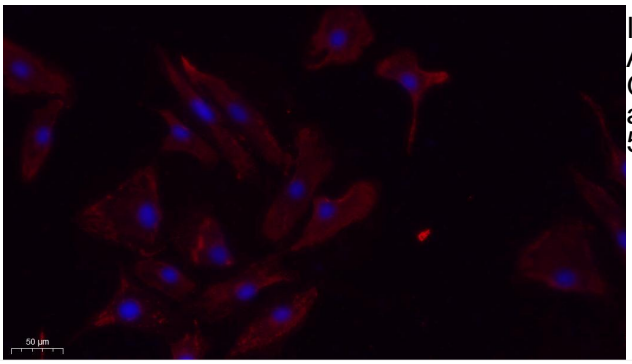
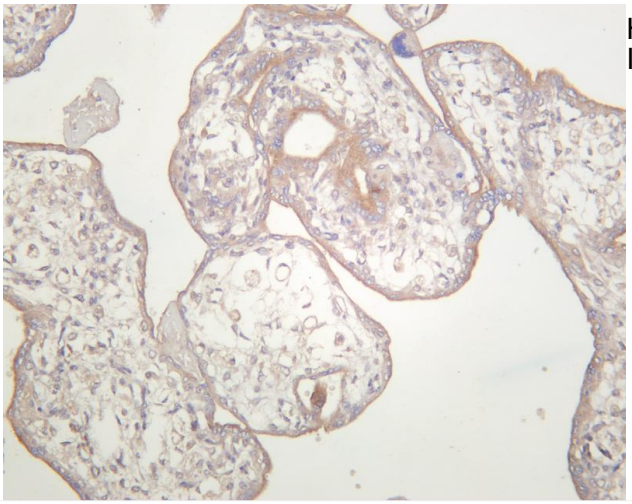
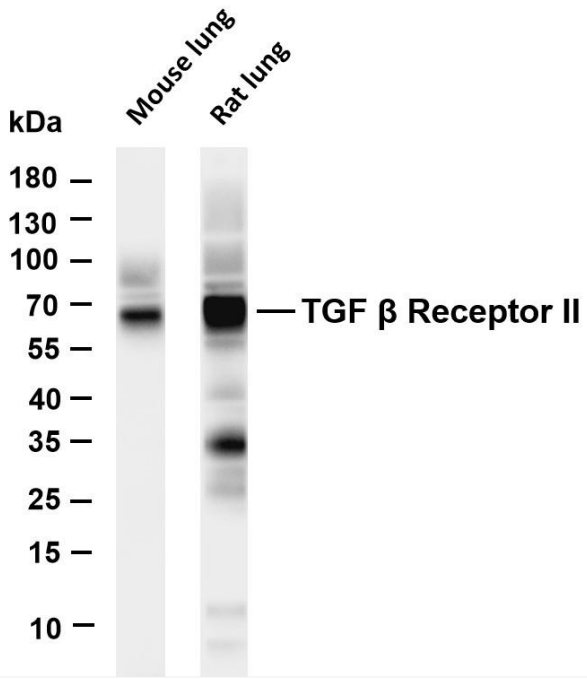
| | cause of hereditary non-polyposis colorectal cancer type 6 (HNPCC6) [MIM:190182]. Mutations in more than one gene locus can be involved alone or in combination in the production of the HNPCC phenotype (also called Lynch syndrome). Most families with clinically recognized HNPCC have mutations in either 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 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., |
| **Background** | 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], |
| **matters needing attention** | Avoid repeated freezing and thawing! |
| **Usage suggestions** | This product can be used in immunological reaction related experiments. For more information, please consult technical personnel. |



Immunofluorescence analysis of A549. 1,primary Antibody(red) was diluted at 1:200(4℃ 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.



Human placenta was stained with anti-TGF β Receptor II rabbit antibody




Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-TGF β Receptor II antibody. The HRP-conjugated Goat anti-Rabbit IgG (H + L) antibody was used to detect the antibody. Lane 1: Mouse lung Lane 2: Rat lung Predicted band size: 65kDa Observed band size: 65kDa