Tuberin/TSC2 (Phospho Ser1254) Rabbit pAb

CatalogNo: YP1538

Key Features

Host Species	Reactivity	Applications
• Rabbit	• Human,Mouse,Rat	• WB

MW	Isotype
• 200kD (Observed)	• IgG

Recommended Dilution Ratios

WB 1:1000-2000

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 phosho peptide around human Tuberin(Ser1254)
Specificity	This antibody detects endogenous levels of Human Mouse Rat Tuberin/TSC2 (phospho-Ser1254).The name of modified sites may be influenced by many factors, such as species (the modified site was not originally found in human samples) and the change of protein sequence (the previous protein sequence is incomplete, and the protein sequence may be prolonged with the development of protein sequencing technology). When naming, we will use the "numbers" in historical reference to keep the sites consistent with the reports. The antibody binds to the following modification sequence (lowercase letters are modification sites):SLsVP

Target Information

Gene name TSC2 TSC4

Protein Name Tuberin/TSC2 (Ser1254)

Organism	Gene ID	UniProt ID
Human	7249;	P49815;
Mouse		Q61037;
Rat	24855;	P49816;

Cellular Localization Cytoplasm. Membrane; Peripheral membrane protein. At steady state found in association with membranes.

Tissue specificity Liver, brain, heart, lymphocytes, fibroblasts, biliary epithelium, pancreas, skeletal muscle, kidney, lung and placenta.

Function Alternative products:Additional isoforms seem to exist. Experimental confirmation may be lacking for some isoforms,Disease:Defects in TSC2 are a cause of lymphangioleiomyomatosis (LAM) [MIM:606690]. LAM is a progressive and often fatal lung disease characterized by a diffuse proliferation of abnormal smooth muscle cells in the lungs. It affects almost exclusively young women and can occur as an isolated disorder or in association with tuberous sclerosis complex.,Disease:Defects in TSC2 are the cause of tuberous sclerosis complex (TSC) [MIM:191100]. The molecular basis of TSC is a functional impairment of the tuberin-hamartin complex. TSC is an autosomal dominant multi-system disorder that affects especially the brain, kidneys, heart, and skin. TSC is characterized by hamartomas (benign overgrowths predominantly of a cell or tissue type that occurs normally in the organ) and hamartias (developmental abnormalities of tissue combination). Clinical symptoms can range from benign hypopigmented macules of the skin to profound mental retardation with intractable seizures to premature death from a variety of disease-associated causes.,Function:Implicated as a tumor suppressor. May have a function in vesicular transport, but may also play a role in the regulation of cell growth arrest and in the regulation of transcription mediated by steroid receptors. Interaction between TSC1 and TSC2 may facilitate vesicular docking. Specifically stimulates the intrinsic GTPase activity of the Ras-related protein RAP1A and RAB5. Suggesting a possible mechanism for its role in regulating cellular growth. Mutations in TSC2 leads to constitutive activation of RAP1A in tumors.,online information:TSC2 mutation db,PTM:Phosphorylation at Ser-1387, Ser-1418 or Ser-1420 does not affect interaction with TSC1.,similarity:Contains 1 Rap-GAP domain.,subcellular location:At steady state found in association with membranes.,subunit:Interacts with TSC1 and HERC1; the interaction with TSC1 stabilizes TSC2 and prevents the interaction with HERC1. May also interact with the adapter molecule RABEP1. The final complex contains TSC2 and RABEP1 linked to RAB5 (Probable). Interacts with HSPA1 and HSPA8.,tissue specificity:Liver, brain, heart, lymphocytes, fibroblasts, biliary epithelium, pancreas, skeletal muscle, kidney, lung and placenta.,

Validation Data

Contact information

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