

Product datasheet for **TA306219**

Tuberin (TSC2) Rabbit Polyclonal Antibody

Product data:

Product Type:	Primary Antibodies
Applications:	WB
Recommended Dilution:	WB: 1 ug/mL
Reactivity:	Human, Mouse
Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Immunogen:	TSC2 antibody was raised against a 15 amino acid peptide from near the carboxy-terminus of human TSC2.
Formulation:	PBS containing 0.02% sodium azide.
Concentration:	1ug/ul
Purification:	Affinity chromatography purified via peptide column
Conjugation:	Unconjugated
Storage:	Store at -20°C as received.
Stability:	Stable for 12 months from date of receipt.
Gene Name:	tuberous sclerosis 2
Database Link:	NP_000539 Entrez Gene 22084 Mouse Entrez Gene 7249 Human P49815



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Background:

Tuberous sclerosis complex (TSC) is an autosomal dominant tumor syndrome caused by mutations in either of the TSC1 or TSC2 tumor suppressor genes. The products of these genes form a protein complex that indirectly decreases the signaling of the mammalian Target of Rapamycin (TOR), an evolutionarily conserved serine/threonine kinase that regulates cell growth and cell cycle through its ability to integrate signals from nutrient levels and growth factors (reviewed in 1,2). TOR activity is stimulated by Rheb, a member of the Ras superfamily of G-proteins, when the GTP/GDP ratio bound to Rheb is high (3). Immunoprecipitated TSC1/TSC2 has been shown to stimulate Rheb GTPase activity in vitro (4), suggesting that the TSC1/TSC2 complex decreases the ability of Rheb to stimulate TOR activity. This is supported by experiments showing that overexpression of TSC1 and TSC2 results in a significant decrease in the GTP/GDP ratio bound to Rheb and the inhibition of cell growth (4,5). At least three isoforms of TSC2 exist.

Synonyms:

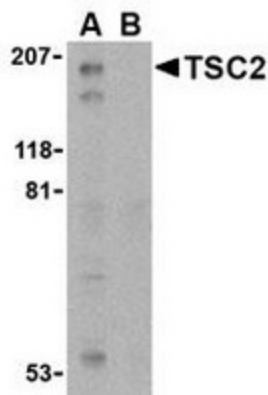
LAM; PPP1R160; TSC4

Protein Families:

Druggable Genome

Protein Pathways:

Insulin signaling pathway, mTOR signaling pathway, p53 signaling pathway

Product images:

Western blot analysis of TSC2 in L1210 cell lysate with TSC2 antibody at 1 ug/ml in the (A) absence and (B) presence of blocking peptide.