

# Hamartin Antibody

Rabbit mAb

Catalog # AP92337

## Product Information

<b>Application</b>	WB, IHC, IF, FC, ICC, IHF
<b>Primary Accession</b>	<a href="#">Q92574</a>
<b>Reactivity</b>	Human
<b>Clonality</b>	Monoclonal
<b>Other Names</b>	Hamartin; kiaa0243; LAM; TSC; Tsc1; Tuberous sclerosis 1;
<b>Isotype</b>	Rabbit IgG
<b>Host</b>	Rabbit
<b>Calculated MW</b>	129767

## Additional Information

<b>Dilution</b>	WB 1:1000~1:5000 IHC 1:50~1:200 ICC/IF 1:50~1:200 FC 1:200
<b>Purification</b>	Affinity-chromatography
<b>Immunogen</b>	A synthesized peptide derived from human Hamartin
<b>Description</b>	In complex with TSC2, inhibits the nutrient-mediated or growth factor-stimulated phosphorylation of S6K1 and EIF4EBP1 by negatively regulating mTORC1 signaling. Seems not to be required for TSC2 GAP activity towards RHEB. Implicated as a tumor suppressor. Involved in microtubule-mediated protein transport, but this seems to be due to unregulated mTOR signaling.
<b>Storage Condition and Buffer</b>	Rabbit IgG in phosphate buffered saline , pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol. Store at +4°C short term. Store at -20°C long term. Avoid freeze / thaw cycle.

## Protein Information

<b>Name</b>	TSC1 {ECO:0000303   PubMed:9242607, ECO:0000312   HGNC:HGNC:12362}
<b>Function</b>	Non-catalytic component of the TSC-TBC complex, a multiprotein complex that acts as a negative regulator of the canonical mTORC1 complex, an evolutionarily conserved central nutrient sensor that stimulates anabolic reactions and macromolecule biosynthesis to promote cellular biomass generation and growth (PubMed: <a href="#">12172553</a> , PubMed: <a href="#">12271141</a> , PubMed: <a href="#">12906785</a> , PubMed: <a href="#">15340059</a> , PubMed: <a href="#">24529379</a> , PubMed: <a href="#">28215400</a> ). The TSC-TBC complex acts as a GTPase-activating protein (GAP) for the small GTPase RHEB, a direct activator of the protein kinase activity of mTORC1 (PubMed: <a href="#">12906785</a> , PubMed: <a href="#">15340059</a> , PubMed: <a href="#">24529379</a> ). In absence of nutrients, the TSC-TBC complex inhibits mTORC1, thereby preventing phosphorylation of ribosomal protein S6 kinase (RPS6KB1 and RPS6KB2) and EIF4EBP1 (4E-BP1) by the mTORC1 signaling (PubMed: <a href="#">12271141</a> , PubMed: <a href="#">24529379</a> , PubMed: <a href="#">28215400</a> ,

PubMed:[33215753](#)). The TSC-TBC complex is inactivated in response to nutrients, relieving inhibition of mTORC1 (PubMed:[12172553](#), PubMed:[24529379](#)). Within the TSC-TBC complex, TSC1 stabilizes TSC2 and prevents TSC2 self-aggregation (PubMed:[10585443](#), PubMed:[28215400](#)). Acts as a tumor suppressor (PubMed:[9242607](#)). Involved in microtubule-mediated protein transport via its ability to regulate mTORC1 signaling (By similarity). Also acts as a co-chaperone for HSP90AA1 facilitating HSP90AA1 chaperoning of protein clients such as kinases, TSC2 and glucocorticoid receptor NR3C1 (PubMed:[29127155](#)). Increases ATP binding to HSP90AA1 and inhibits HSP90AA1 ATPase activity (PubMed:[29127155](#)). Competes with the activating co-chaperone AHSA1 for binding to HSP90AA1, thereby providing a reciprocal regulatory mechanism for chaperoning of client proteins (PubMed:[29127155](#)). Recruits TSC2 to HSP90AA1 and stabilizes TSC2 by preventing the interaction between TSC2 and ubiquitin ligase HERC1 (PubMed:[16464865](#), PubMed:[29127155](#)).

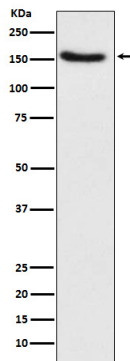
### Cellular Location

Lysosome membrane; Peripheral membrane protein. Cytoplasm, cytosol  
Note=Recruited to lysosomal membranes in a RHEB-dependent process in absence of nutrients (PubMed:[24529379](#)). In response to nutrients, the complex dissociates from lysosomal membranes and relocates to the cytosol (PubMed:[24529379](#)).

### Tissue Location

Highly expressed in skeletal muscle, followed by heart, brain, placenta, pancreas, lung, liver and kidney (PubMed:[9242607](#)). Also expressed in embryonic kidney cells (PubMed:[9242607](#)).

## Images



Western blot analysis of Hamartin expression in HeLa cell lysate.

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