

DESCRIPTION

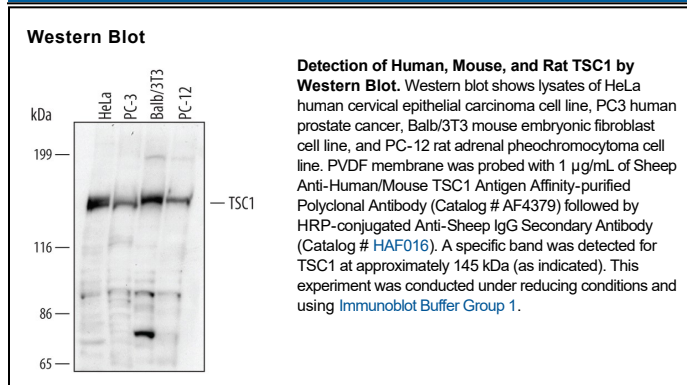
Species Reactivity	Human/Mouse/Rat
Specificity	Detects human, mouse, and rat TSC1 in direct ELISAs and Western blots.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	<i>E. coli</i> -derived recombinant human TSC1 Asp156-Thr300 Accession # Q92574
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	1 µg/mL	See Below
Intracellular Staining by Flow Cytometry	0.25 µg/mL	Jurkat human acute T cell leukemia cell line fixed with paraformaldehyde and permeabilized with saponin
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

TSC1 (Tuberous sclerosis 1), or hamartin, is a tumor suppressor which interacts with tumor suppressor TSC2 (tuberin) to form a cytoplasmic heterodimer. Mutations in either hamartin or tuberin are responsible for tuberous sclerosis (TSC), an autosomal dominant disease characterized by renal dysfunction, seizures, developmental delays, benign hamartomas and low grade neoplasms predominantly affecting the CNS, kidney, lung, skin, and heart. The TSC1/TSC2 complex suppresses cell growth by inhibiting mTOR, with TSC1 acting to inhibit the ubiquitination of TSC2, leading to increased cellular levels of TSC2 and thus enhancing its catalytic activity as a GTPase-activating protein for Rheb. TSC1 and TSC2 are also involved in the G2/M transition of the cell cycle through their interactions with CDK1 and cyclin B1. TSC1 has also been shown to interact with F-actin and ERM (Ezrin-Radixin-Moesin) proteins, implying a role in the modulation of cell adhesion and morphology.