Mouse Endoglin/CD105 Fluorescein-conjugated Antibody

Monoclonal Rat IgG_{2A} Clone # 209701

Catalog Number: FAB1320F

100 TESTS, 25 TESTS

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse Endoglin/CD105 in direct ELISAs and Western blots. In Western blots, this antibody shows 100% cross-reactivity with recombinant human Endoglin/CD105.
Source	Monoclonal Rat IgG_{2A} Clone # 209701
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse Endoglin/CD105 Glu21-Gly581 (predicted) Accession # NP_031958
Conjugate	Fluorescein Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm (FITC)
Formulation	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

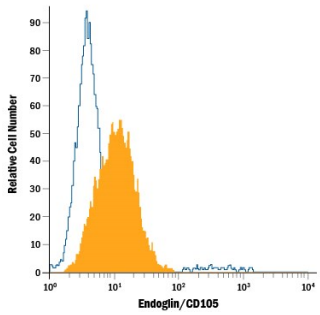
APPLICATIONS

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
Flow Cytometry	10 µL/10^6 cells	See Below

DATA

Flow Cytometry



Detection of Endoglin/CD105 in MS-1 Mouse Cell Line by Flow Cytometry. MS-1 mouse pancreatic islet endothelial cell line was stained with Rat Anti-Mouse Endoglin/CD105 Fluorescein-conjugated Monoclonal Antibody (Catalog # FAB1320F, filled histogram) or isotype control antibody (Catalog # IC006F, open histogram). View our protocol for Staining Membrane-associated Proteins.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Protect from light. Do not freeze.** ● 12 months from date of receipt, 2 to 8 °C as supplied.

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BACKGROUND

Endoglin (CD105) is a 90 kDa type I transmembrane glycoprotein of the Zona Pellucida (ZP) family of proteins (1-3). Endoglin and Betaglycan/TβRIII are type III receptors for TGF beta superfamily ligands, sharing 71% amino acid (aa) identity within the transmembrane (TM) and cytoplasmic domains. Endoglin is highly expressed on proliferating vascular endothelial cells, chondrocytes, and syncytiotrophoblasts of term placenta, with lower amounts on hematopoietic, mesenchymal, and neural crest stem cells, activated monocytes, and lymphoid and myeloid leukemic cells (2-5). Mouse Endoglin cDNA encodes 653 aa including a 26 aa signal sequence, a 555 aa extracellular domain (ECD) with an orphan domain and a two-part ZP domain, a TM domain, and a 47 aa cytoplasmic domain (1-3). A mouse isoform with a 35 aa cytoplasmic domain (S-Endoglin) can oppose effects of long (L) Endoglin (6, 7). The mouse Endoglin ECD shares 69%, 84%, 62%, 63%, and 66% aa identity with human, rat, bovine, porcine, and canine Endoglin, respectively. Endoglin homodimers interact with TGF-β1 and TGF-β3 (but not TGF-β2) but only after binding TβRII (8). Similarly, they interact with Activin-A and BMP-7 via Activin type IIA or B receptors, and with BMP-2 via BMPR-1A/ALK-3 or BMPR-1B/ALK-6 (9). BMP-9, however, is reported to bind Endoglin directly (10). Endoglin modifies ligand-induced signaling in multiple ways. For example, expression of Endoglin can inhibit TGF-β1 signals but enhance BMP-7 signals in the same myoblast cell line (11). In endothelial cells, Endoglin inhibits TβRI/ALK5 but enhances ALK1-mediated activation (12). Deletion of mouse Endoglin causes lethal vascular and cardiovascular defects, and human Endoglin haploinsufficiency can cause the vascular disorder, hereditary hemorrhagic telangiectasia type I (13, 14). These abnormalities confirm the essential function of Endoglin in differentiation of smooth muscle, angiogenesis, and neovascularization (2-4, 12-14). In preeclampsia of pregnancy, high levels of proteolytically generated soluble Endoglin and VEGF R1 (sFlt-1), along with low Placental Growth Factor (PlGF), are pathogenic due to anti-angiogenic activity (15).

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