Human Endoglin/CD105
Alexa Fluor® 350-conjugated Antibody

Monoclonal Mouse IgG_1 Clone # 166707

Catalog Number: FAB10971U

100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Endoglin/CD105.
Source	Monoclonal Mouse IgG_1 Clone # 166707
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Endoglin/CD105 Glu26-Gly586 Accession # Q5T9B9
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2 mg/mL 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	0.25-1 µg/10^6 cells	U937 human histiocytic lymphoma cell line

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.

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% aa identity in 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). Human Endoglin cDNA encodes 658 amino acids (aa) including a 25 aa signal sequence, a 561 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). An isoform with a 14 aa cytoplasmic domain (S-endoglin) can oppose effects of long (L) Endoglin (6, 7). The human Endoglin ECD shares 65-72% aa identity with mouse, rat, bovine, porcine and canine Endoglin. 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 BMP7 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 a 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 (sFLT1), along with low placental growth factor (PlGF), are pathogenic due to antiangiogenic activity (15).

References:

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Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956
USA TEL 800 343 7475 Canada TEL 855 668 8722 China TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449

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USA TEL 800 343 7475 Canada TEL 855 668 8722 China TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449