Mouse Endoglin/CD105 Antibody

Monoclonal Rat IgG_{2A} Clone # 209721

Catalog Number: MAB13201

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse Endoglin/CD105 in ELISAs and Western blots. In Western blots, this antibody does not cross-react with recombinant human Endoglin.
Source	Monoclonal Rat IgG_{2A} Clone # 209721
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse Endoglin/CD105 Glu27-Gly581 Accession # Q8K100
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	Recombinant Mouse Endoglin/CD105 Fc Chimera (Catalog # 1320-EN) under non-reducing conditions only
Mouse Endoglin/CD105 Sandwich Immunoassay		**Reagent**
ELISA Capture	2-8 µg/mL	Mouse Endoglin/CD105 Antibody (Catalog # MAB13201)
ELISA Detection	0.1-0.4 µg/mL	Mouse Endoglin/CD105 Biotinylated Antibody (Catalog # BAF1320)
Standard		Recombinant Mouse Endoglin/CD105 Fc Chimera (Catalog # 1320-EN)

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 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.** ● 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

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-β 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-IA/ALK-3 or BMPR-IB/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 ALK-1-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 (sFlt-1), along with low placental growth factor (PlGF), are pathogenic due to anti-angiogenic activity (15).

References:

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