Recombinant Human Endoglin/CD105

Catalog Number: 1097-EN

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived human Endoglin/CD105 protein Glu26-Gly586 Accession # Q5T9C0
N-terminal Sequence Analysis	Glu26
Predicted Molecular Mass	61 kDa

SPECIFICATIONS

SDS-PAGE	75-85 kDa, reducing conditions
Activity	Measured by its ability to inhibit BMP-10-induced alkaline phosphatase production by MC3T3-E1 mouse preosteoblast cells. The ED_{50} for this effect is 0.06-0.36 µg/mL in the presence of 100 ng/mL of Recombinant Human BMP-10 (Catalog # 2926-BP).
Endotoxin Level	<1.0 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 250 µg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
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. • 3 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 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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