

DESCRIPTION

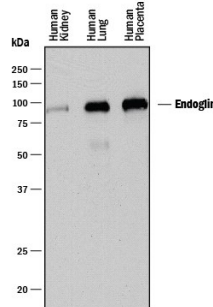
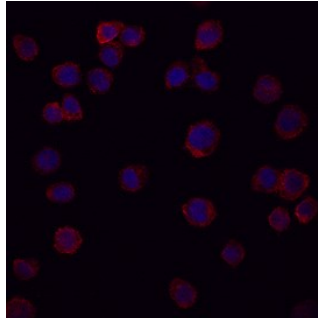
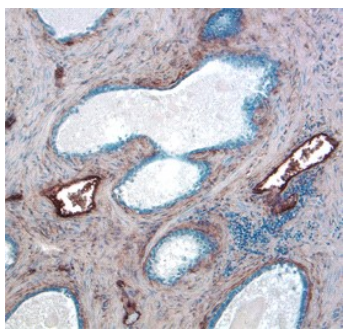
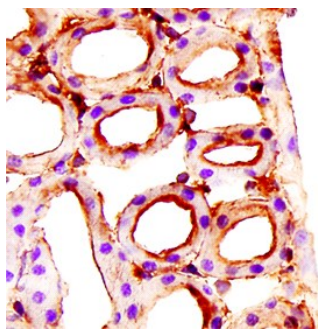
Species Reactivity	Human
Specificity	Detects human Endoglin in direct ELISAs and Western blots. In direct ELISAs, less than 15% cross-reactivity with recombinant mouse Endoglin and recombinant rat Endoglin is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Endoglin/CD105 Glu26-Gly586 Accession # Q5T9B9
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 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	0.25 µg/mL	See Below
Flow Cytometry	0.25 µg/10 ⁶ cells	U937 human histiocytic lymphoma cell line
Immunocytochemistry	5-15 µg/mL	See Below
Immunohistochemistry	5-15 µg/mL	See Below
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	

DATA

<p>Western Blot</p>  <p>Detection of Human Endoglin/CD105 by Western Blot. Western blot shows lysates of human kidney tissue, human lung tissue, human placenta tissue. PVDF membrane was probed with 0.25 µg/mL of Goat Anti-Human Endoglin/CD105 Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1097) followed by HRP-conjugated Anti-Goat IgG Secondary Antibody (Catalog # HAF017). A specific band was detected for Endoglin/CD105 at approximately 90 kDa (as indicated). This experiment was conducted under reducing conditions and using Immunoblot Buffer Group 1.</p>	<p>Immunocytochemistry</p>  <p>Endoglin/CD105 in U937 Human Cell Line. Endoglin/CD105 was detected in immersion fixed U937 human histiocytic lymphoma cell line using Goat Anti-Human Endoglin/CD105 Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1097) at 10 µg/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Goat IgG Secondary Antibody (red; Catalog # NL001) and counterstained with DAPI (blue). View our protocol for Fluorescent ICC Staining of Cells on Coverslips.</p>
<p>Immunohistochemistry</p>  <p>Endoglin/CD105 in Human Prostate. Endoglin/CD105 was detected in immersion fixed paraffin-embedded sections of human prostate using Goat Anti-Human Endoglin/CD105 Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1097) at 1.7 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). View our protocol for Chromogenic IHC Staining of immersion fixed paraffin-embedded Tissue Sections.</p>	<p>Immunohistochemistry</p>  <p>Endoglin/CD105 in Mouse Kidney. Endoglin/CD105 was detected in immersion fixed paraffin-embedded sections of mouse kidney using Goat Anti-Human Endoglin/CD105 Antigen Affinity-purified Polyclonal Antibody (Catalog # AF1097) at 15 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). Specific staining was localized to plasma membranes in tubules. View our protocol for Chromogenic IHC Staining of Paraffin-embedded Tissue Sections.</p>

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

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 including a 25 amino acid (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 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 antiangiogenic activity (15).

References:

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