

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human ALK-1 in direct ELISAs and Western blots. Shows less than 1% cross-reactivity with recombinant mouse ALK-1, recombinant human (rh) Activin R1A, or rhActivin R1B.
Source	Monoclonal Mouse IgG _{2A} Clone # 117720
Purification	Protein A or G purified from ascites
Immunogen	Mouse myeloma cell line NS0-derived recombinant human ALK-1 Asp22-Gln118 Accession # P37023
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.

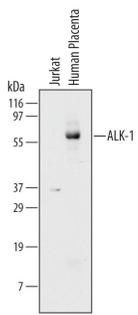
APPLICATIONS

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	2 µg/mL	See Below
Simple Western	40 µg/mL	See Below

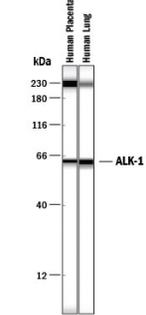
DATA

Western Blot



Detection of Human ALK-1 by Western Blot.
Western blot shows lysates of Jurkat human acute T cell leukemia cell line and human placenta tissue. PVDF membrane was probed with 2 µg/mL of Mouse Anti-Human ALK-1 Monoclonal Antibody (Catalog # MAB370) followed by HRP-conjugated Anti-Mouse IgG Secondary Antibody (Catalog # HAF007). A specific band was detected for ALK-1 at approximately 65 kDa (as indicated). This experiment was conducted under reducing conditions and using Immunoblot Buffer Group 1.

Simple Western



Detection of Human ALK-1 by Simple Western™.
Simple Western lane view shows lysates of human placenta tissue and human lung tissue, loaded at 0.2 mg/mL. A specific band was detected for ALK-1 at approximately 63 kDa (as indicated) using 40 µg/mL of Mouse Anti-Human ALK-1 Monoclonal Antibody (Catalog # MAB370). This experiment was conducted under reducing conditions and using the 12-230 kDa separation system.

Non-specific interaction with the 230 kDa Simple Western standard may be seen with this antibody.



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. <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

Transforming Growth Factor beta (TGF-β) superfamily ligands exert their biological activities via binding to heteromeric receptor complexes of two types (I and II) of serine/threonine kinases. Type II receptors are constitutively active kinases that phosphorylate type I receptors upon ligand binding. In turn, activated type I kinases phosphorylate downstream signaling molecules including the various smads. Transmembrane proteoglycans, including the type III receptor (betaglycan) and endoglin, can bind and present some of the TGF-β superfamily ligands to type I and II receptor complexes and enhance their cellular responses. Seven type I receptors (also termed activin receptor-like kinase (ALK)) and five type II receptors have been isolated from mammals. ALK-2, -3, -4, -5, and -6 are also known as Activin R1A, BMPR-1A, Activin R1B, TGF-β R1, and BMPR-1B, respectively, reflecting their ligand preferences. Evidence suggests that TGF-β1, TGF-β3 and an unknown ligand present in serum can activate chimeric ALK-1. ALK-1 shares with other type I receptors a cysteine-rich domain with conserved cysteine spacing in the extracellular region, and a glycine- and serine-rich domain (the GS domain) preceding the kinase domain. ALK-1 is expressed highly in endothelial cells and other highly vascularized tissues. The expression patterns of ALK-1 parallels that of endoglin. Mutations in ALK-1 as well as in endoglin are associated with hereditary hemorrhagic telangiectasia (HHT), suggesting a critical role for ALK-1 in the control of blood vessel development or repair. Human and mouse ALK-1 share approximately 71% amino acid sequence identity in their extracellular regions.

References:

1. ten Dijke, P. *et al.* (1993) *Oncogene* **8**:2879.
2. ten Dijke, P. *et al.* (1994) *Science* **264**:101.
3. Lux, A. *et al.* (1999) *J. Biol. Chem.* **274**:9984.