

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human ALK-1 in direct ELISAs.
Source	Monoclonal Mouse IgG ₁ Clone # 117702
Purification	Protein A or G purified from ascites
Immunogen	Mouse myeloma cell line NS0-derived recombinant human ALK-1 Asp22-Gln118 Accession # P37023
Conjugate	Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *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.

Blockade of Receptor-ligand Interaction Optimal dilution of this antibody should be experimentally determined.

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

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.

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