ACVRL1/ALK1 Protein, Mouse (HEK293, Fc)

Cat. No.:	HY-P7481
Synonyms:	rMuActivin Receptor-like Kinase 1, C-Fc; ALK-1; ACVRL1; Activin Receptor-like Kinase 1
Species:	Mouse
Source:	HEK293
Accession:	Q61288 (D23-P119)
Gene ID:	11482
Molecular Weight:	55-60 kDa

PROPERTIES

AA Sequence	D L A K P S K L V N C T C E S P H C K R P F C Q G S W C T V V L V R E Q G R H P Q V Y R G C G S L N Q E L C L G R P T E F L N H H C C Y R S F C N H N V S L M L E A T Q T P S E E P E V D A H L P
Biological Activity	The enzyme activity of this recombinant protein is testing in progress, we cannot offer a guarantee yet.
Appearance	Lyophilized powder.
Formulation	Lyophilized after extensive dialysis against PBS, pH 7.4.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	ALK-1, also known as ACVRL1, is a type I receptor for TGF-β superfamily with 2 ligands, BMP9 and BMP10. ALK-1 is predominantly expressed in endothelial cells and plays a critical role in regulating angiogenesis[1][2]. Mature human ALK-1 shares 89% amino acid sequence identity with mouse and rat ALK-1. While, mouse ALK-1 shares 96% aa sequence identity with rat ALK-1 protein. ALK-1 is able to bind to TGF-β1 or activins in the presence of either TβR-II or activin type II receptors, respectively. However, ALK-1 does not elicit a specific transcriptional response. Thus, ALK-1 has been considered an “orphan” receptor. ALK-1 is a type I receptor that mediates signaling of BMP9 (bone morphogenetic protein) and BMP10, proteins in the TGF-β

superfamily. Signaling through ALK-1 results in phosphorylation of the intracellular Smad 1/5/8 cascade which activates proangiogenic transcription factors such as ID1 and ID3. ALK-1 binds to TGF-β1 and phosphorylates Smad1 and Smad5. Overexpression of ALK-1 in HepG2 cells inhibits the ALK5-mediated TGF-β1 response. The balance between ALK-1 and ALK5 may be crucial for controlling the properties of endothelium during angiogenesis[1]. BMP9/BMP10/ALK-1 signaling controlled the specific gene expression program and survival of Kupffer cells (KCs) through a Smad4-dependent pathway. Functionally, the loss of ALK-1 resulted in impaired capture of L. monocytogenes and overwhelming disseminated infections [2].

ALK-1 is expressed in blood vessels during embryogenesis and adult stages. In addition, mutations of the ALK-1 gene have been linked to the type II hereditary hemorrhagic telangiectasia[1]. ALK-1 inhibits BMP9-mediated Id-1 expression in human umbilical vein endothelial cells. In a chick chorioallantoic membrane assay, ALK-1 reduces VEGF-, FGF-, and BMP10-mediated vessel formation. In addition, ALK1 reduces tumor burden in mice receiving orthotopic grafts of MCF7 mammary adenocarcinoma cells[3].

REFERENCES

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[3]. Dianne Mitchell, et al. ALK1-Fc inhibits multiple mediators of angiogenesis and suppresses tumor growth. Mol Cancer Ther. 2010 Feb;9(2):379-88.

[4]. Dongxing Zhu, et al. BMP-9 regulates the osteoblastic differentiation and calcification of vascular smooth muscle cells through an ALK1 mediated pathway. J Cell Mol Med. 2015 Jan;19(1):165-74.

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