BMPRIB/ALK-6 Protein, Human (HEK293, Fc)

Cat. No.:	HY-P76175
Synonyms:	Bone morphogenetic protein receptor type-1B; BMPR-1B; CDw293
Species:	Human
Source:	HEK293
Accession:	O00238 (K14-R126)
Gene ID:	658
Molecular Weight:	Approximately 110 kDa.

PROPERTIES

Biological Activity	The enzyme activity of this recombinant protein is testing in progress, we cannot offer a guarantee yet.
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
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.
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

BMPRIB/ALK-6, also known as CDw293 (cluster of differentiation w293), is a member of the bone morphogenetic protein (BMP) receptor family of transmembrane serine/threonine kinases. BMPs are involved in endochondral bone formation and embryogenesis, transducing signals through the formation of heteromeric complexes of 2 types BMP receptors: type I receptors (50-55 kD) and type II receptors (70-80 kD). Type II receptors phosphorylate and activate type I receptors which autophosphorylate, then bind and activate SMAD transcriptional regulators[1]. ALK-6 is the receptor for BMP-7/OP-1 and GDF-5, positively regulates chondrocyte differentiation through GDF-5 interaction[2][3]. ALK-6 signaling is also controlled by SCUBE3, a BMP-2/BMP-4 co-receptor, recruits the BMP receptor complexes into raft microdomains, and positively modulates signaling possibly by augmenting the specific interactions between BMPs and BMP type I receptors[3]. ALK-6 involves in controlling growth, morphogenesis, and bone and teeth development through modulation of BMP signaling, is essential for chondrogenesis in vivo, while BMPR1A/ALK-3 and ALK-6 have overlapping functions during early chondrogenesis[4]. ALK-6/GDF-9/BMP-15 signals play a key role in prolificacy. ALK-6 and GDF-9 are widely expressed in 20 tissues with highest in ovary, while BMP-15 gene was expressed exclusively in ovary and pituitary[5]. The sequences of ALK-6 are highly conserved among different species, and the sequence of human shares a high similarity with rat (98.21%) and

mouse (98.21%), respectively.

REFERENCES

[1]. ten Dijke P, et al. Signaling via hetero-oligomeric complexes of type I and type II serine/threonine kinase receptors. Curr Opin Cell Biol. 1996 Apr;8(2):139-45.

[2]. Xu H, et al. [BMP7 signaling via BMPR1A, BMPR1B inhibits the proliferation of lung large carcinoma NCI-H460 cell]. Zhongguo Fei Ai Za Zhi. 2010 Jul;13(7):659-64. Chinese.

[3]. Demirhan O, et al. A homozygous BMPR1B mutation causes a new subtype of acromesomelic chondrodysplasia with genital anomalies. J Med Genet. 2005 Apr;42(4):314-7.

[4]. Lin YC, et al. SCUBE3 loss-of-function causes a recognizable recessive developmental disorder due to defective bone morphogenetic protein signaling. Am J Hum Genet. 2021 Jan 7;108(1):115-133.

[5]. Yang CX, et al. Cloning and mRNA expression levels of GDF9, BMP15, and BMPR1B genes in prolific and non-prolific goat breeds. Mol Reprod Dev. 2012 Jan;79(1):2.

[6]. Yoon BS, et al. Bmpr1a and Bmpr1b have overlapping functions and are essential for chondrogenesis in vivo. Proc Natl Acad Sci U S A. 2005 Apr 5;102(14):5062-7.

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