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

Cat. No.:	HY-P76176
Synonyms:	Bone morphogenetic protein receptor type-1B; BMPR-1B; CDw293
Species:	Mouse
Source:	HEK293
Accession:	P36898 (K14-K126)
Gene ID:	12167
Molecular Weight:	Approximately 39.4 kDa.

PROPERTIES

AA Sequence	K K E D G E S T A P T P R P K I L R C K C H H H C P E D S V N N I C S T D G Y C F T M I E E D D S G M P V V T S G C L G L E G S D F Q C R D T P I P H Q R R S I E C C T E R N E C N K D L H P T L P P L K D R D F V D G P I H H K
Biological Activity	Measured by its ability to inhibit rhBMP-4-induced alkaline phosphatase production by ATDC5 mouse chondrogenic cells. The ED_{50} for this effect is 0.1346 μg/mL in the presence of 30 ng/mL of rhBMP-4. Corresponding to a specific activity is 7.429×10^3 U/mg.
Appearance	Lyophilized powder
Formulation	Lyophilized from a 0.2 μm filtered solution of 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	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 mouse shares a high similarity with human (98.2%) and rat (99.0%), 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.

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[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.

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[7]. Renault L, et al. BMPR1A and BMPR1B Missense Mutations Cause Primary Ovarian Insufficiency. J Clin Endocrinol Metab. 2020 Apr 1;105(4):dgz226.

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