BMPR1A/ALK-3 Protein, Mouse (129a.a, HEK293, His-Fc)

Cat. No.:	HY-P7483
Synonyms:	Bone morphogenetic protein receptor type-1A; ALK-3; SKR5; CD292; ACVRLK3; BMPR-IA
Species:	Mouse
Source:	HEK293
Accession:	P36895 (Q24-R152)
Gene ID:	12166
Molecular Weight:	55-60 kDa

PROPERTIES

AA Sequence	Q N L D S M L H G T G M K S D L D Q K K P E N G V T L A P E D T L P F L K C Y C S G H C P D D A I N N T C I T N G H C F A I I E E D D Q G E T T L T S G C M K Y E G S D F Q C K D S P K A Q L R R T I E C C R T N L C N Q Y L Q P T L P P V V I G P F F D G S I R H H H H H H
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 20 mM PB, 150 mM NaCl, 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-3 (BMPR1A; ACVRLK3) is the receptor bone morphogenetic protein (BMP) type I receptors, for BMP2, BMP4, GDF5 and GDF6. Among BMP type I receptors, ALK-2 and 3 are widely expressedin tissues, while ALK-1 is more selectively expressed in endothelial cells (ECs)[1]. Hepcidin, the main regulator of iron metabolism, is synthesized and released by hepatocytes in response to increased body iron concentration and inflammation. BMP/ALK/SMAD pathway controls hepcidin expression, while BMP type I receptors ALK-2 and ALK-3 are responsible for iron-dependent hepcidin upregulation and basal hepcidin expression, respectively, to avoid low hepcidin which causes iron overload or high hepcidin levels which induce iron-

restricted erythropoiesis[2]. ALK-3 positively regulates chondrocyte differentiation through GDF5 interaction and mediates induction of adipogenesis by GDF6[3]. ALK-3 protein shows function for the initiation of chondrogenesis, for regulating differentiation along the chondrogenic lineage, and for endochondral bone formation[5]. Components of BMP signaling have been implicated in both pathogenesis of pulmonary arterial hypertension (PAH) and endothelial-mesenchymal transition (EndoMT), and BMPR1A is key to maintain endothelial identity and to prevent excessive EndoMT. BMPR1A-ID2/ZEB1-TGFBR2 signaling axis could serve as a potential novel target for PAH and other EndoMT-related vascular disorders[4].

REFERENCES

[1]. Yang P, et al. The role of bone morphogenetic protein signaling in vascular calcification. Bone. 2020 Dec;141:115542.

[2]. Traeger L, et al. HFE and ALK3 act in the same signaling pathway. Free Radic Biol Med. 2020 Nov 20;160:501-505.

[3]. Miyazawa K, et al. Regulation of TGF-β Family Signaling by Inhibitory Smads. Cold Spring Harb Perspect Biol. 2017 Mar 1;9(3):a022095.

[4]. Lee HW, et al. BMPR1A Promotes ID2-ZEB1 Interaction to Suppress Excessive Endothelial to Mesenchymal Transition. Cardiovasc Res. 2022 Sep 27:cvac159.

[5]. Jing J, et al. Bmpr1a Signaling in Cartilage Development and Endochondral Bone Formation. Vitam Horm. 2015;99:273-91.

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