BMPR1A/ALK-3 Protein, Human (152a.a, HEK293, His)

Cat. No.:	HY-P75594
Synonyms:	ALK-3; BMPRIA; Bone morphogenetic protein receptor type-1A; SKR5; CD292
Species:	Human
Source:	HEK293
Accession:	P36894 (Q24-R152)
Gene ID:	657
Molecular Weight:	28-33 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	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

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[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.

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[6]. Wu XG, et al. HFE interacts with the BMP type I receptor ALK3 to regulate hepcidin expression. Blood. 2014 Aug 21;124(8):1335-43.

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