

BMPR1A/ALK-3 Protein, Mouse, Recombinant (His & hFc)

General Information

Synonyms:	Bmpr;bone morphogenetic protein receptor, type IA;BMPR-IA;AU045487;ALK3;1110037122Rik
Protein Construction:	A DNA sequence encoding the extracellular domain (Met 1-Arg 152) of mouse ALK3 (NP_033888.2) precursor was fused with the Fc region of human IgG1 at the C-terminus. Predicted N terminal: Gln 24
Species:	Mouse
Expression Host:	HEK293 Cells
Accession:	P36895
Molecular Weight:	42 kDa (predicted); 60 kDa (reducing condition, due to glycosylation)

QC Testing

Biological Activity:	Measured by its ability to inhibit BMP4-induced activity in MC3T3-E1 Mouse osteoblastic cells. The ED50 for this effect is typically 0.1-0.3 µg/ml in the presence of 50 ng/mL of recombinant human BMP4.
Purity:	> 95 % as determined by SDS-PAGE
Endotoxin:	< 1.0 EU/µg of the protein as determined by the LAL method.
Formulation:	Lyophilized from a solution filtered through a 0.22 µm filter, containing PBS, pH 7.4. Typically, a mixture containing 5% to 8% trehalose, mannitol, and 0.01% Tween 80 is incorporated as a protective agent before lyophilization.

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

It is recommended to store recombinant proteins at -20°C to -80°C for future use. Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry ice.

Protein Background

Activin receptor-Like Kinase 3 (ALK-3), also known as Bone Morphogenetic Protein Receptor, type IA (BMPR1A), is a type I receptor for bone morphogenetic proteins (BMPs) which belong to the transforming growth factor beta (TGF-β) superfamily. The BMP receptors form a subfamily of transmembrane serine/threonine kinases including the

type I receptors BMPR1A and BMPR1B and the type II receptor BMPR2. ALK-3/BMPR1A is expressed in the epithelium during branching morphogenesis. Deletion of BMPR1A in the epithelium with an Sftpc-cre transgene leads to dramatic defects in lung development. ALK-3 and ALK-6 share a high degree of homology, yet possess distinct signaling roles. The transforming growth factor (TGF)-beta type III receptor (TbetaRIII) enhanced both ALK-3 and ALK-6 signaling. TbetaRIII associated with ALK-3 primarily through their extracellular domains, whereas its interaction with ALK-6 required both the extracellular and cytoplasmic domains. ALK-3 plays an essential role in the formation of embryonic ventral abdominal wall, and abrogation of BMP signaling activity due to gene mutations in its signaling components could be one of the underlying causes of omphalocele at birth. The type IA BMP receptor, ALK-3 was specifically required at mid-gestation for normal development of the trabeculae, compact myocardium, interventricular septum, and endocardial cushion. Cardiac muscle lacking ALK-3 was specifically deficient in expressing TGFbeta2, an established paracrine mediator of cushion morphogenesis. Hence, ALK-3 is essential, beyond just the egg cylinder stage, for myocyte-dependent functions and signals in cardiac organogenesis.

Reference

- Gaussin V, et al. (2002) Endocardial cushion and myocardial defects after cardiac myocyte-specific conditional deletion of the bone morphogenetic protein receptor ALK3. *Proc Natl Acad Sci U S A.* 99(5): 2878-83.
- Eblaghie MC, et al. (2006) Evidence that autocrine signaling through Bmpr1a regulates the proliferation, survival and morphogenetic behavior of distal lung epithelial cells. *Dev Biol.* 291(1): 67-82.
- Sun J, et al. (2007) Deficient Alk3-mediated BMP signaling causes prenatal omphalocele-like defect. *Biochem Biophys Res Commun.* 360(1): 238-43.
- Lee NY, et al. (2009) The transforming growth factor-beta type III receptor mediates distinct subcellular trafficking and downstream signaling of activin-like kinase (ALK)3 and ALK6 receptors. *Mol Biol Cell.* 20(20): 4362-70.

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