Recombinant Mouse BMPRIA/ALK-3 Protein (His &Fc Tag)

Catalog Number: PKSM040937

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

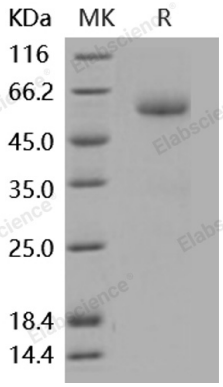
Description

Species	Mouse
Source	HEK293 Cells-derived Mouse BMPRIA/ALK-3 protein Met 1-Arg 152, with an C-terminal His & Fc
Calculated MW	42 kDa
Observed MW	60 kDa
Accession	NP_033888.2
Bio-activity	Measured by its ability to inhibit BMP4-induced activity in MC3T3-E1 Mouse osteoblastic cells. The ED_{50} for this effect is typically 0.1-0.3 μg/ml in the presence of 50 ng/mL of recombinant human BMP4.

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile PBS, pH 7.4 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 95 % as determined by reducing SDS-PAGE.

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 (TGFB) 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. 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.

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