# Mouse ALK-3 / BMPR1A Protein (His & Fc Tag)

**Catalog Number:** **50078-M03H**

Sino Biological
Biological Solution Specialist

## General Information

**Gene Name Synonym:**

1110037I22Rik; ALK3; AU045487; Bmpr; BMPR-IA

**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.

**Source:** Mouse

**Expression Host:** HEK293 Cells

## QC Testing

**Purity:** > 95 % as determined by SDS-PAGE

**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.**

**Endotoxin:**

< 1.0 EU per μg of the protein as determined by the LAL method

**Stability:**

Samples are stable for up to twelve months from date of receipt at -70 ℃

**Predicted N terminal:** Gln 24

**Molecular Mass:**

The recombinant mouse ALK3/Fc is a disulfide-linked homodimer after removal of the signal peptide. The reduced monomer consists of 376 amino acids and has a predicted molecular mass of 42 kDa. In SDS-PAGE under reducing conditions, the apparent molecular mass of rm ALK3/Fc monomer is approximately 60 kDa due to glycosylation.

**Formulation:**

Lyophilized from sterile PBS, pH 7.4

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

## Usage Guide

**Storage:**

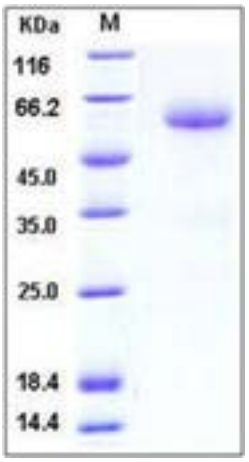
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

**Avoid repeated freeze-thaw cycles.**

**Reconstitution:**

Detailed reconstitution instructions are sent along with the products.

**SDS-PAGE:**




## Protein Description

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.

**References**

1.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. 2.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. 3.Sun J, *et al.* (2007) Deficient Alk3-mediated BMP signaling causes prenatal omphalocele-like defect. Biochem Biophys Res Commun. 360(1): 238-43.