Canine ALK-2 / ACVR1 / ALK2 Protein (His Tag)

Catalog Number: **70048-D08H**

General Information

Gene Name Synonym:

ACVR1

Protein Construction:

A DNA sequence encoding the canine ACVR1 (XP_549615.2) (Met1-Glu123) was expressed with a C-terminal polyhistidine tag.

Source: Canine

Expression Host: HEK293 Cells

QC Testing

Purity: > 95 % as determined by SDS-PAGE

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: Met 21

Molecular Mass:

The recombinant canine ACVR1 comprises 114 amino acids and has a predicted molecular mass of 13 kDa. The apparent molecular mass of the protein is approximately 19 kDa in SDS-PAGE under reducing conditions 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:

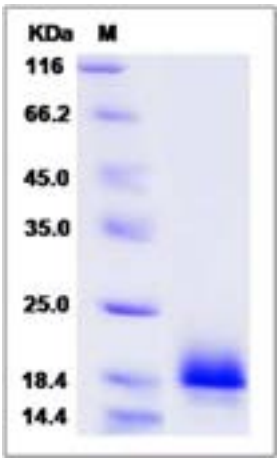
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

ALK-2, also termed as ACVR1, was initially identified as an activin type I receptor because of its ability to bind activin in concert with ActRII or ActRIIB. ALK-2 is also identified as a BMP type I receptor. It has been demonstrated that ALK-2 forms complex with either the BMP-2/7-bound BMPR-II or ACVR2A /ACVR2B. ALK-1 and ALK-2 presenting in the yeast Saccharomyces cerevisiae are two haspin homologues. Both ALK-1 and ALK-2 exhibit a weak auto-kinase activity in vitro, and are phosphoproteins in vivo. ALK-1 and ALK-2 levels peak in mitosis and late-S/G2. Control of protein stability plays a major role in ALK-2 regulation. The half-life of ALK-2 is particularly short in G1. Overexpression of ALK-2, but not of ALK-1, causes a mitotic arrest, which is correlated to the kinase activity of the protein. This suggests a role for ALK-2 in the control of mitosis. Endoglin is phosphorylated on cytosolic domain threonine residues by the TGF-beta type I receptors ALK-2 and ALK-5 in prostate cancer cells. Endoglin did not inhibit cell migration in the presence of constitutively active ALK-2. Defects in ALK-2 are a cause of fibrodysplasia ossificans progressiva (FOP).

References

1.Armes NA,*et al.* (1997) The ALK-2 and ALK-4 activin receptors transduce distinct mesoderm-inducing signals during early Xenopus development but do not co-operate to establish thresholds. Development 124(19): 3797-804.
2.Armes NA, *et al.* (1999) A short loop on the ALK-2 and ALK-4 activin receptors regulates signaling specificity but cannot account for all their effects on early Xenopus development. J Biol Chem. 274(12):7929-35.
3.Kawai S, *et al.* (2000) Mouse smad8 phosphorylation downstream of BMP receptors ALK-2, ALK-3, and ALK-6 induces its association with Smad4 and transcriptional activity.Biochem Biophys Res Commun. 271(3):682-7.