

## ACVR1

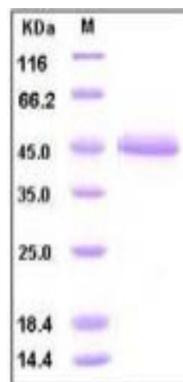
### Recombinant Human ALK-2 / ACVR1 (His & Fc Tag)

|                              |   |                  |                  |
|------------------------------|---|------------------|------------------|
| <b>Catalog No.</b>           | CRH419A-HisFc<br>CRH419B-HisFc  | <b>Quantity:</b> | 100 µg<br>200 µg |
| <b>Alternate Names:</b>      | Activin receptor type-1, Activin receptor type I, ACTR-I, Activin receptor-like kinase 2, ALK-2, Serine/threonine-protein kinase receptor R1, SKR1, TGF-B superfamily receptor type I, TSR-I  |                  |                  |
| <b>Description:</b>          | <p>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 <i>Saccharomyces cerevisiae</i> 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).</p> |                  |                  |
| <b>UniProt ID:</b>           | Q04771  |                  |                  |
| <b>Accession Number:</b>     | NP_001104537.1  |                  |                  |
| <b>Protein Construction:</b> | A DNA sequence encoding the extracellular domain (Met 1-Val 124) of human ALK2 precursor was fused with the C-terminal polyhistidine-tagged Fc region of human IgG1 at the C-terminus.  |                  |                  |
| <b>Source:</b>               | HEK293 Cells  |                  |                  |
| <b>Formulation:</b>          | Lyophilized from sterile PBS, pH 7.4<br>Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization.  |                  |                  |
| <b>Molecular Weight:</b>     | The rhALK2/Fc is a disulfide-linked homodimer after removal of the signal peptide. The monomer consists of 352 aa with a predicted MW of 39.6 kDa and migrates at ~40-45 kDa in SDS-PAGE under reducing conditions due to glycosylation.  |                  |                  |
| <b>Purity:</b>               | > 95 % as determined by SDS-PAGE.   |                  |                  |
| <b>Endotoxin Level:</b>      | < 1.0 EU per µg of the protein as determined by the LAL method  |                  |                  |
| <b>Biological Activity:</b>  | Measure by its ability to bind with human BMP2 in a functional ELISA.   |                  |                  |
| <b>Predicted N-terminal:</b> | Met 21  |                  |                  |

**Reconstitution:** **Centrifuge vial prior to opening.** Add sterile distilled water to a concentration of 0.1 mg/mL and gently pipette the solution up and down the sides of the vial. **DO NOT VORTEX.** Allow several minutes for complete reconstitution.

**Storage & Stability:** Stable for up to 1 year from date of receipt at -20°C to -80°C. After reconstitution, store working aliquots at -20°C to -80°C. **Avoid repeated freeze-thaw cycles.**

SDS-PAGE



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