

## ACVR1 Protein, Rat (HEK293, hFc)

<b>Cat. No.:</b>	HY-P75538
<b>Synonyms:</b>	Activin receptor type-1; ACTR-I; SKR1; TSR-I; TSK-7L; Acvrlk2; Tgfb1
<b>Species:</b>	Rat
<b>Source:</b>	HEK293
<b>Accession:</b>	P80201 (M21-E123)
<b>Gene ID:</b>	79558
<b>Molecular Weight:</b>	Approximately 43-48 kDa

### PROPERTIES

<b>AA Sequence</b>	<p>           M E D E E P K V N P    K L Y M C V C E G L    S C G N E D H C E G    Q Q C F S S L S V N            D G F R V Y Q K G C    F Q V Y E Q G K M T    C K T P P S P G Q A    V E C C Q G D W C N            R N V T A R L P T K    G K S F P G S Q N F    H L E         </p>
<b>Biological Activity</b>	Measured by its binding ability in a functional ELISA. When Recombinant Human BMP-6 is used at 0.25 µg/mL, the concentration of Recombinant Rat ACVR1. The ED <sub>50</sub> for this effect is 4.39 µg/mL.
<b>Appearance</b>	Lyophilized powder.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution of PBS, pH 7.4.
<b>Endotoxin Level</b>	<1 EU/µg, determined by LAL method.
<b>Reconstitution</b>	It is not recommended to reconstitute to a concentration less than 100 µg/mL in ddH <sub>2</sub> O. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).
<b>Storage &amp; Stability</b>	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
<b>Shipping</b>	Room temperature in continental US; may vary elsewhere.

### DESCRIPTION

<b>Background</b>	<p>Activin A receptor type I (ACVR1) gene, also known as ALK-2, is located in chromosome 2q23-q24 and encodes for the 509 amino acid protein. The ACVR1 protein product is initially described as an activin type I receptor, and it is found to be expressed in several tissues and different human cell lines<sup>[1]</sup>.</p> <p>The sequence of amino acids in ACVR1 proteins from different species is very stable, which leads to the conclusion that in the process of evolution, ACVR1 has been only slightly altered, and that both in humans and in animals, its function is similar.</p>
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As a member of the BMP/TGF $\beta$  receptor family, the ACVR1 protein contains an extracellular N-terminal ligand-binding domain, a transmembrane (TM) domain, an intracellular glycine-serine-rich (GS) domain, and a protein kinase (PK) domain. The loop positioned in the helix-loop-helix of the GS domain contains the key residues responsible for ACVR1 activation upon phosphorylation. As a type I receptor, ACVR1 forms heterotetrameric receptor complexes with the type II receptors BMPR2, ACVR2A, and ACVR2B. Such complexes consist of two type I and two type II receptors. Upon binding of ligands to the heteromeric complexes, type II receptors transphosphorylate the GS domain of type I receptors. As a result, the kinase domain of type I receptors is activated and subsequently phosphorylates SMAD1/5/8 proteins that transduce the signal. ACVR1 is first described to bind to activin A, a member of the BMP/TGF $\beta$  family that usually triggers phosphorylation and activation of SMAD2/3 upon complex formation with type II receptors. Later, ACVR1 is also found to bind several BMPs with distinct affinities, triggering SMAD1/5/8 signalling. Besides canonical SMAD signalling, ACVR1 can activate non-canonical signalling pathways, such as p38 mitogen-activated protein kinases/MAPKs<sup>[1]</sup>.

ACVR1 is involved in a wide variety of biological processes, including bone, heart, cartilage, nervous, and reproductive system development and regulation. Moreover, ACVR1 has been extensively studied for its causal role in fibrodysplasia ossificans progressiva (FOP). ACVR1 is linked to different pathologies, including cardiac malformations and alterations in the reproductive system. More recently, ACVR1 has been experimentally validated as a cancer driver gene in diffuse intrinsic pontine glioma (DIPG)<sup>[1]</sup>.

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## REFERENCES

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- [1]. José Antonio Valer, et al. ACVR1 Function in Health and Disease. *Cells*. 2019 Oct 31;8(11):1366.
- [2]. Jing Pang, et al. ACVR1-Fc suppresses BMP signaling and chondro-osseous differentiation in an in vitro model of Fibrodysplasia ossificans progressive. *Bone*. 2016 Nov;92:29-36.
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**Caution: Product has not been fully validated for medical applications. For research use only.**

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