ACVR2A/Activin RIIA Protein, Human (HEK293, Fc)

Cat. No.:	HY-P75537
Synonyms:	ACVR-2A; Activin receptor type 2A; ACTR-IIA; ACVR2
Species:	Human
Source:	HEK293
Accession:	P27037 (A20-P134)
Gene ID:	92
Molecular Weight:	60-65 kDa

PROPERTIES

Biological Activity	The enzyme activity of this recombinant protein is testing in progress, we cannot offer a guarantee yet.
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	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.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background

ACVR2A is a type II member of the TGF-β family of receptor Serine/Threonine kinases. ACVR2A is a receptor for Activin A, Activin B and Inhibin A[1][2].

The sequence of amino acids in ACVR2A proteins from different species is very stable, which leads to the conclusion that in the process of evolution, ACVR2A has been only slightly altered, and that both in humans and in animals, its function is similar.

Signaling by activins and BMPs is highly promiscuous, since apart from signaling through ALK4/7, the activin type II receptors (ACVR2A and 2B) can interact also with several type I BMP receptors (ALK1/2/3/6), which can also form complexes with the type II BMP receptor, BMPRII. Type II receptors phosphorylate and activate type I receptors which autophosphorylate, then bind and activate SMAD transcriptional regulators. ACVR2A can form complexes with different type I receptors that signal either to Smad2/3 (ALK4) or to Smad1/5/8 (ALK2, ALK3, ALK6). The different type I receptors compete for binding to ACVR2A and that this competition provides a mechanism that balances signaling between Activin A-mediated, ALK4-dependent Smad2/3 signaling, and BMP-mediated ALK2 or ALK3-dependent signaling to Smad1/5/8. In myeloma cells, BMP-6- and BMP-9-induced activation of SMAD1/5/8 through ACVR2A/ACVR2B/ALK2 is inhibited by activin A treatment[1][3].

REFERENCES

[1]. Oddrun Elise Olsen, et al. Activin A inhibits BMP-signaling by binding ACVR2A and ACVR2B. Cell Commun Signal. 2015 Jun 6;13:27.

[2]. V K Chin, et al. Inhibition of Activin A suppressed tumor necrosis factor-α secretion and improved histopathological conditions in malarial mice. Trop Biomed. 2021 Mar 1;38(1):187-204.

[3]. Szabina Szófia Szilágyi, et al. Competition between type I activin and BMP receptors for binding to ACVR2A regulates signaling to distinct Smad pathways. BMC Biol. 2022 Feb 18;20(1):50.

[4]. Heather L Hayes, et al. A Pdx-1-Regulated Soluble Factor Activates Rat and Human Islet Cell Proliferation. Mol Cell Biol. 2016 Nov 14;36(23):2918-2930.

Caution: Product has not been fully validated for medical applications. For research use only.

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