Recombinant Human Activin RIIA/ACVR2A Protein (His Tag)

Catalog Number: PKSH031728

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

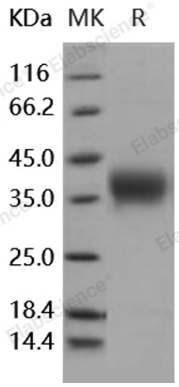
Description

Species	Human
Source	HEK293 Cells-derived Human Activin RIIA/ACVR2A protein Met 1-Pro 134, with an C-terminal His
Calculated MW	14.9 kDa
Observed MW	35-40 kDa
Accession	NP_001607.1
Bio-activity	Measured by its ability to neutralize Activin-mediated inhibition on MPC11 cell proliferation. The ED_{50} for this effect is typically 0.4-3 μg/mL in the presence of 10 ng/mL recombinant Activin A.

Properties

Purity	> 97 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile PBS, pH 7.4 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 97 % as determined by reducing SDS-PAGE.

Background

ACVR2A and ACVR2B are two activin type II receptors. ACVR2A has been shown to interact with INHBA, SYNJ2BP and ACVR1B. The bovine ACVR2A gene encodes a protein of 513 amino acids which is highly homologous (approximately 98% identity) to the rat, mouse, and human ACVR2A proteins. Inactivation of ACVR2A is a common event in prostate cancer cells suggesting it may play an important role in the development of prostate cancer. The ACVR2A gene is a putative tumor suppressor gene that is frequently mutated in microsatellite-unstable colon cancers (MSI-H colon cancers). Frameshift mutation of ACVR2A may contribute to MSI-H colon tumorigenesis via disruption of alternate TGF-beta effector pathways.

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