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ACVR1 Antibody (Center) Blocking peptide

Synthetic peptide
Catalog # BP13856c

Specification

ACVR1 Antibody (Center) Blocking peptide - Product Information

Primary Accession Q04771

ACVR1 Antibody (Center) Blocking peptide - Additional Information

Gene ID 90

Other Names
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, ACVR1, ACVRLK2

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP13856c was selected from the Center region of ACVR1. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

ACVR1 Antibody (Center) Blocking peptide - Protein Information

Name ACVR1

Synonyms ACVRLK2

Function
Bone morphogenetic protein (BMP) type I receptor that is involved in a wide variety of biological processes, including bone, heart, cartilage, nervous, and reproductive system development and regulation (PubMed:20628059, PubMed:22977237). As a type I receptor, forms heterotetrameric receptor complexes with the type II receptors AMHR2, ACVR2A or ACVR2B (PubMed:17911401). Upon binding of ligands such as BMP7 or GDF2/BMP9 to the heteromeric complexes, type II receptors transphosphorylate ACVR1 intracellular domain (PubMed:<a

href="http://www.uniprot.org/citations/25354296" target="_blank">25354296). In turn, ACVR1 kinase domain is activated and subsequently phosphorylates SMAD1/5/8 proteins that transduce the signal (PubMed:9748228). In addition to its role in mediating BMP pathway-specific signaling, suppresses TGFbeta/activin pathway signaling by interfering with the binding of activin to its type II receptor (PubMed:17911401). Besides canonical SMAD signaling, can activate non-canonical pathways such as p38 mitogen-activated protein kinases/MAPKs (By similarity). May promote the expression of HAMP, potentially via its interaction with BMP6 (By similarity).

Cellular Location
Membrane; Single-pass type I membrane protein.

Tissue Location
Expressed in normal parenchymal cells, endothelial cells, fibroblasts and tumor-derived epithelial cells

ACVR1 Antibody (Center) Blocking peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

ACVR1 Antibody (Center) Blocking peptide - Images

ACVR1 Antibody (Center) Blocking peptide - Background

Activins are dimeric growth and differentiation factorswhich belong to the transforming growth factor-beta (TGF-beta)superfamily of structurally related signaling proteins. Activinssignal through a heteromeric complex of receptor serine kinaseswhich include at least two type I (I and IB) and two type II (IIand IIB) receptors. These receptors are all transmembrane proteins,composed of a ligand-binding extracellular domain withcysteine-rich region, a transmembrane domain, and a cytoplasmicdomain with predicted serine/threonine specificity. Type Ireceptors are essential for signaling; and type II receptors arerequired for binding ligands and for expression of type Ireceptors. Type I and II receptors form a stable complex afterligand binding, resulting in phosphorylation of type I receptors bytype II receptors. This gene encodes activin A type I receptorwhich signals a particular transcriptional response in concert withactivin type II receptors. Mutations in this gene are associatedwith fibrodysplasia ossificans progressive.

ACVR1 Antibody (Center) Blocking peptide - References

Canzian, F., et al. Hum. Mol. Genet. 19(19):3873-3884(2010)Shimada, M., et al. Hum. Genet. 128(4):433-441(2010)Song, G.A., et al. J. Biol. Chem. 285(29):22542-22553(2010)Herrera, B., et al. Cancer Res. 69(24):9254-9262(2009)Jung, B., et al. PLoS ONE 4 (12), E8308 (2009) :