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Activin Receptor Type IA (ACVR1) Antibody (Center N99) Blocking peptide

Synthetic peptide
Catalog # BP7101c

Specification

Activin Receptor Type IA (ACVR1) Antibody (Center N99) Blocking peptide - Product Information

Primary Accession Q04771

Activin Receptor Type IA (ACVR1) Antibody (Center N99) 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 AP7101c was selected from the Center region of human 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.

Activin Receptor Type IA (ACVR1) Antibody (Center N99) 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: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

Activin Receptor Type IA (ACVR1) Antibody (Center N99) Blocking peptide - Protocols

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

- Blocking Peptides

Activin Receptor Type IA (ACVR1) Antibody (Center N99) Blocking peptide - Images

Activin Receptor Type IA (ACVR1) Antibody (Center N99) Blocking peptide - Background

Activins are dimeric growth and differentiation factors which belong to the transforming growth factor-beta (TGF-beta) superfamily of structurally related signaling proteins. Activins signal through a heteromeric complex of receptor serine kinases which include at least two type I (I and IB) and two type II (II and IIB) receptors. These receptors are all transmembrane proteins, composed of a ligand-binding extracellular domain with cysteine-rich region, a transmembrane domain, and a cytoplasmic domain with predicted serine/threonine specificity. Type I receptors are essential for signaling; and type II receptors are required for binding ligands and for expression of type I receptors. Type I and II receptors form a stable complex after ligand binding, resulting in phosphorylation of type I receptors by type II receptors. ACVR1 is an activin A type I receptor which signals a particular transcriptional response in concert with activin type II receptors.

Activin Receptor Type IA (ACVR1) Antibody (Center N99) Blocking peptide - References

Casagrandi, D., et al., Mol. Hum. Reprod. 9(4):199-203 (2003).Welt, C.K., Curr Opin Obstet Gynecol 14(3):317-323 (2002).Schneider-Kolsky, M.E., et al., Placenta 23(4):294-302 (2002).Chapman, S.C., et al., Mol. Endocrinol. 15(4):668-679 (2001).Schulte, K.M., et al., Horm. Metab. Res. 32(10):390-400 (2000).