10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

BMPR1A Antibody (N-term)

Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP2004e

Specification

BMPR1A Antibody (N-term) - Product Information

Application	**WB, FC,E**
Primary Accession	P36894
Reactivity	**Human, Mouse**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Calculated MW	**60198**
Antigen Region	**1-30**

BMPR1A Antibody (N-term) - Additional Information

Gene ID 657

Other Names
Bone morphogenetic protein receptor type-1A, BMP type-1A receptor, BMPR-1A, Activin receptor-like kinase 3, ALK-3, Serine/threonine-protein kinase receptor R5, SKR5, CD292, BMPR1A, ACVRLK3, ALK3

Target/Specificity
This BMPR1A antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 1-30 amino acids from the N-terminal region of human BMPR1A.

Dilution
WB~~1:1000
FC~~1:10~50

Format
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage
Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
BMPR1A Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

BMPR1A Antibody (N-term) - Protein Information

Name BMPR1A

Synonyms ACVRLK3, ALK3

Function On ligand binding, forms a receptor complex consisting of two type II and two type I transmembrane serine/threonine kinases. Type II receptors phosphorylate and activate type I receptors which autophosphorylate, then bind and activate SMAD transcriptional regulators. Receptor for BMP2, BMP4, GDF5 and GDF6. Positively regulates chondrocyte differentiation through GDF5 interaction. Mediates induction of adipogenesis by GDF6. May promote the expression of HAMP, potentially via its interaction with BMP2 (By similarity).

Cellular Location
Cell membrane; Single-pass type I membrane protein. Cell surface {ECO:0000250|UniProtKB:P36895}

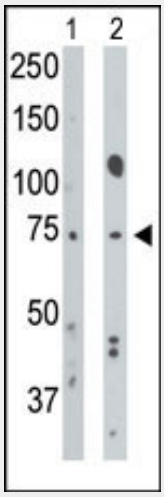
Tissue Location
Highly expressed in skeletal muscle.

BMPR1A Antibody (N-term) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

BMPR1A Antibody (N-term) - Images



The anti-BMPR1A Pab (Cat. #AP2004e) is used in Western blot to detect BMPR1A in mouse muscle tissue lysate (Lane 1) and Hela cell lysate (Lane 2).

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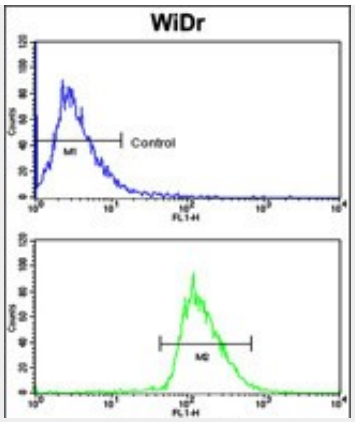

Flow cytometric analysis of WiDr cells using BMPR1A Antibody (N-term) (bottom histogram) compared to a negative control cell (top histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

BMPR1A Antibody (N-term) - Background

The bone morphogenetic protein (BMP) receptors are a family of transmembrane serine/threonine kinases that include the type I receptors BMPR1A and BMPR1B and the type II receptor BMPR2. These receptors are also closely related to the activin receptors, ACVR1 and ACVR2. The ligands of these receptors are members of the TGF-beta superfamily. TGF-betas and activins transduce their signals through the formation of heteromeric complexes with 2 different types of serine (threonine) kinase receptors: type I receptors of about 50-55 kD and type II receptors of about 70-80 kD. Type II receptors bind ligands in the absence of type I receptors, but they require their respective type I receptors for signaling, whereas type I receptors require their respective type II receptors for ligand binding. BMPRIA binds BMP4 with high-affinity in solution and is a potent BMP-4 antagonist in vitro. In adult tissues, BMPRIA is widely expressed, with the highest expression levels detected in skeletal muscle. BMPRIA is also widely expressed during embryogenesis.

BMPR1A Antibody (N-term) - References

Waite, K.A., et al., Hum. Mol. Genet. 12(6):679-684 (2003).
Zhou, X.P., et al., Am. J. Hum. Genet. 69(4):704-711 (2001).
Astrom, A.K., et al., Mamm. Genome 10(3):299-302 (1999).
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