Human BMP-3 Antibody

Antigen Affinity-purified Polyclonal Goat IgG
Catalog Number: AF113

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human BMP-3 in direct ELISAs and Western blots. In direct ELISAs, less than 1% cross-reactivity with recombinant human (rh) BMP-2, rhBMP-4, rhBMP-5, rhBMP-6, and rhBMP-7 is observed. This antibody very weakly recognizes native dimeric BMP-3.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	*E. coli*-derived recombinant human BMP-3 Ser346-Arg472 Accession # P12645
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose.

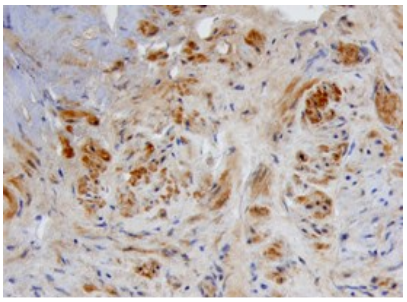
APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Human BMP-3 (Catalog # 113-BP)
Immunohistochemistry	5-15 µg/mL	See Below

DATA

Immunohistochemistry



BMP-3 in Human Prostate Cancer Tissue. BMP-3 was detected in immersion fixed paraffin-embedded sections of human prostate cancer using 15 µg/mL Goat Anti-Human BMP-3 Antigen Affinity-purified Polyclonal Antibody (Catalog # AF113) overnight at 4 °C. Tissue was stained with the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # Catalog # CTS008) and counterstained with hematoxylin (blue). View our protocol for Chromogenic IHC Staining of Paraffin-embedded Tissue Sections.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

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BACKGROUND

BMP-3, also known as osteogenin, the most abundant BMP in adult bone, is one of at least 15 structurally and functionally related BMPs, which are members of the TGF- β superfamily (1-3). BMPs were originally identified as protein regulators of cartilage and bone formation. They have since been shown to be involved in embryogenesis and morphogenesis of various tissues and organs. BMPs also regulate the growth, differentiation, chemotaxis, and apoptosis of various cell types. Similar to most other TGF-β family proteins, BMPs are highly conserved across animal species. At the amino acid sequence level, mature human and rat BMP-3 are 98% identical. BMP-3 is synthesized as a large precursor protein that is cleaved at the dibasic cleavage site (RXXR) to release the carboxy-terminal domain. Biologically active BMP-3 is a disulfide-linked homodimer of the carboxy-terminal 110 amino acid residues that contains the characteristic seven conserved cysteine residues involved in the formation of the cysteine knot and the single interchain disulfide bond (4). The role of BMP-3 in bone is contradictory since, unlike osteogenin purified from bone, recombinant BMP-3 has not shown osteogenic function (5). Several studies indicate that BMP-3 is an inhibitor of osteogenic BMPs. BMP-3 dorsalizes *Xenopus* embryos, the opposite effect of BMP-2 or 4, which cause ventralization. BMP-3 inhibits alkaline phosphatase production and induction of osteoblastic target genes in undifferentiated mesenchymal and osteogenic cell lines that have been treated with BMP-2. BMP-3 also induces the expression of TGF-β /activin responsive genes, but not BMP-responsive genes. Since the inhibitory effect is not due to direct competition with osteogenic BMPs, it has been suggested that BMP-3 activates signaling through an activin pathway, resulting in antagonism of osteogenesis induced by other BMPs.

References:

1. Chen, D. *et al.* (2004) Growth Factors **22**:233.
2. Hino, J. *et al.* (2004) Front. Biosci. **9**:1520.
3. Bahamonde, M.E. and K.M. Lyons (2001) J. Bone and Joint Surgery **83-A** (suppl 1):S156.
4. Wozney, J.M. *et al.* (1998) Science **242**:1528.
5. Daluiski, A. *et al.* (2001) Nature Genetics **27**:84.

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