BMP-3B/GDF10 Protein, Mouse (HEK293, Fc)

Cat. No.:	HY-P75788
Synonyms:	Growth/differentiation factor 10; GDF-10; Bone morphogenetic protein 3B; BMP-3B
Species:	Mouse
Source:	HEK293
Accession:	P97737 (Q367-R476)
Gene ID:	14560
Molecular Weight:	Approximately 45 kDa

PROPERTIES

AA Sequence	Q W D E P R V C S R R Y L K V D F A D I G W N E W I I S P K S F D A Y Y C A G A C E F P M P K I V R P S N H A T I Q S I V R A V G I V P G I P E P C C V P D K M N S L G V L F L D E N R N A V L K V Y P N M S V E T C A C R
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	Bone Morphogenetic Protein 3 (BMP-3) is a ligand protein with pleiotropic, belongs to TGFβ family. BMP-3 is a major component of osteogenin, which has osteogenic activity[1]. BMP-3 is widely found in different animals, while the sequence in human is lowly similar to Rat (81.94%), and mouse (80.86%). BMP-3 particularly serves as a reliable biomarker for screening colorectal cancer (CRC) because BMP-3 is hypermethylated and its protein expression is significantly reduced in cancer cell lines[2]. BMP-3 also plays a suppressor role in carcinogenesis, suppresses colon tumorigenesis via ActRIIB/SMAD2-dependent and TAK1/JNK signaling pathways[2]. BMP-3 could exert two-way regulatory effects on activin signaling in distinct cell types. BMP-3 stimulates proliferation of human mesenchymal stem cells which could be blocked by TGF-β/activin receptor kinase inhibitors[3].

BMP/TGFβ signaling to involve in vascular and valvular homeostasis, which is a critical process of embryonic development[4]. And BMP/TGFβ signaling can be terminated by inhibitory SMADs including SMAD6 and SMAD7, which are activated and induced by BMP signaling and switch off BMP signaling via multiple mechanisms[5].

REFERENCES

[1]. Yang P, et al. The role of bone morphogenetic protein signaling in vascular calcification. Bone. 2020 Dec;141:115542.

[2]. Miyazawa K, et al. Regulation of TGF-β Family Signaling by Inhibitory Smads. Cold Spring Harb Perspect Biol. 2017 Mar 1;9(3):a022095.

[3]. Wen J, et al. BMP3 suppresses colon tumorigenesis via ActRIIB/SMAD2-dependent and TAK1/JNK signaling pathways. J Exp Clin Cancer Res. 2019 Oct 28;38(1):428.

[4]. Bahamonde ME, et al. BMP3: to be or not to be a BMP. J Bone Joint Surg Am. 2001;83-A Suppl 1(Pt 1):S56-62.

[5]. Stewart A, et al. BMP-3 promotes mesenchymal stem cell proliferation through the TGF-beta/activin signaling pathway. J Cell Physiol. 2010 Jun;223(3):658-66.

[6]. Daluiski A, et al. Bone morphogenetic protein-3 is a negative regulator of bone density. Nat Genet. 2001 Jan;27(1):84-8.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA