BMP-4 Protein, Mouse (HEK293, Fc)

Cat. No.:	HY-P74379
Synonyms:	BMP-2B; BMP-4; Bone morphogenetic protein 4; DVR4
Species:	Mouse
Source:	HEK293
Accession:	P21275 (S293-R408)
Gene ID:	12159
Molecular Weight:	Approximately 40.6 kDa

PROPERTIES

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 4 (BMP-4) is a ligand protein with pleiotropic, belongs to TGFβ family. BMP-4 involves in the vasculature circulation and can activate receptors on vascular cells[1].
BMP-4/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[4].
BMP-4 is widely found in different animals, while the sequence in human is highly similar to Rat (96.81%), and mouse (97.54%).
BMP-4 is expressed by endothelial cells (ECs) in response to hypoxia and promotes vascular SMC proliferation. Therefore it inhibits the proliferation of smooth muscle cells (SMCs) isolated from the proximal pulmonary artery while induces proliferation of SMCs isolated from distal pulmonary arteries[5].
BMP-4 appears to be a marker and driver of vascular calcification, particularly in atherosclerosis[6].
BMP-4 induces angiogenesis, endothelial cells (ECs) proliferation, and migration[7].
BMP-4 is differentially expressed in calcified atherosclerotic plaques[8], serves as the linkers between atherosclerotic vascular calcification with mechanisms of normal bone formation[9].
BMP-4 increases plaque formation via their pro-inflammatory and pro-atherogenic effects, promoting oxidative stress, endothelial dysfunction and osteogenic differentiation[3].

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]. Herrera B, et al. A rapid and sensitive bioassay for the simultaneous measurement of multiple bone morphogenetic proteins. Identification and quantification of BMP4, BMP6 and BMP9 in bovine and human serum. BMC Cell Biol. 2009 Mar 19;10:20.

[4]. Yang X, et al. Dysfunctional Smad signaling contributes to abnormal smooth muscle cell proliferation in familial pulmonary arterial hypertension. Circ Res. 2005 May 27;96(10):1053-63.

[5]. Scimeca M, et al. Plaque calcification is driven by different mechanisms of mineralization associated with specific cardiovascular risk factors. Nutr Metab Cardiovasc Dis. 2019 Dec;29(12):1330-1336.

[6]. David L, et al. Emerging role of bone morphogenetic proteins in angiogenesis. Cytokine Growth Factor Rev. 2009 Jun;20(3):203-12.

[7]. Dhore CR, et al. Differential expression of bone matrix regulatory proteins in human atherosclerotic plaques. Arterioscler Thromb Vasc Biol. 2001 Dec;21(12):1998-2003.

[8]. Demer LL, et al. Mechanism of calcification in atherosclerosis. Trends Cardiovasc Med. 1994 Jan-Feb;4(1):45-9.

[9]. Boström K, et al. Bone morphogenetic protein expression in human atherosclerotic lesions. J Clin Invest. 1993 Apr;91(4):1800-9.

[10]. Li Z, et al. BMP4 Signaling Acts via dual-specificity phosphatase 9 to control ERK activity in mouse embryonic stem cells. Cell Stem Cell. 2012 Feb 3;10(2):171-82.

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