

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived		
	Human BMP-10 Propeptide (Ser20 - Arg313) & (Pro23 - Arg313) Accession # O95393	RR	6-His tag
	N-terminus		C-terminus
N-terminal Sequence Analysis	Ser20 & Pro23		
Predicted Molecular Mass	34.6 kDa		

SPECIFICATIONS

SDS-PAGE	42-50 kDa, reducing conditions
Activity	Measured by its ability to inhibit BMP-10-induced alkaline phosphatase production by MC3T3-E1 mouse preosteoblast cells. The ED ₅₀ for this effect is typically 0.75-3 µg/mL in the presence of 100 ng/mL of rhBMP-10.
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 250 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

BMP-10, along with BMP-9, GDF-5, -6, and -7, belongs to a subgroup of sequence related TGF-β superfamily proteins that signal through heterodimeric complexes composed of type I and type II BMP receptors (1 - 3). Proteolytic removal of the propeptide from the 60 kDa proprotein yields a 12 kDa mature BMP-10 which forms disulfide-linked non-glycosylated homodimers (4, 5). In transfectants, BMP-10 is secreted as a cleaved mature dimer, an uncleaved proform dimer, and an uncleaved proform monomer (4). The propeptide of human BMP-10 shares 82% amino acid sequence identity with mouse and rat proBMP-10 and 19% - 34% with the propeptides of human BMP-9, GDF-5, -6, and -7. BMP-10 is critical for the proper development of the heart and first appears at the onset of trabeculation and chamber formation (6 - 8). Homozygous BMP-10 knockout mice die *in utero* due to arrested cardiac development (7). BMP-10 is required for maintaining expression of the cardiogenic transcription factors NKX2.5 and MEF2C in developing myocardium and promoting the growth of embryonic cardiomyocytes (7, 9, 10). The BMP-10 mediated proliferation of these cells requires Notch signaling (11). NKX2.5 itself negatively regulates BMP-10 expression in cardiac myocytes (10). Multiple human congenital heart defects result from mutations in NKX2.5 and require BMP-10 expression (10). In mice, genetic knockout of ErbB leads to a similar phenotype but appears not to involve BMP-10, and knockout of the calcium channel subunit FKBP12 induces BMP-10 overexpression (7). BMP-10 in the postnatal heart promotes increased cardiomyocyte and heart size (8). BMP-10 has been shown to induce signaling through ALK-1, BMPR-IA, BMPR-IB, and BMPR-II in transfectants and non-cardiac cell lines (4, 5). A functional BMP-10 receptor in the heart has not yet been identified, although deletion of BMPR-IA or BMP-10 causes similar cardiac morphogenetic abnormalities (12).

References:

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