

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived
Asn317-Arg424
Accession # O95393

N-terminal Sequence Analysis Asn317

Structure / Form Disulfide-linked homodimer

Predicted Molecular Mass 12.2 kDa (monomer)

SPECIFICATIONS

SDS-PAGE 13 kDa, reducing conditions

Activity Measured by its ability to induce alkaline phosphatase production by MC3T3-E1 mouse preosteoblast cells.
The ED₅₀ for this effect is 15-60 ng/mL.

Endotoxin Level <0.01 EU per 1 µg of the protein by the LAL method.

Purity >97%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in Acetonitrile and TFA. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 µg/mL in sterile 4 mM HCl.

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.
- 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 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). Mature human BMP-10 shares 98% amino acid sequence identity with mouse and rat BMP-10 and 49% - 63% with human BMP-9, GDF-5, -6, and -7. BMP-10 is critical for the proper development of the heart but is not expressed until after cardiac patterning or looping are completed (6-8). BMP-10 production appears at the onset of trabeculation and chamber formation and is restricted to the right atrium in the adult heart (6-8). Homozygous BMP-10 knockout mice die *in utero* due to arrested cardiac development (7). BMP-10 is required for the expression of the cardiogenic transcription factors NKX2.5 and MEF2C in developing myocardium and the growth of embryonic cardiomyocytes (7, 10). 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 over-expression (7). BMP-10 in the postnatal heart promotes increased cardiomyocyte and heart size (8). BMP-10 has been shown to signal 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 causes similar cardiac morphogenetic abnormalities (11). In dermal endothelial cells, BMP-10 induces migration, proliferation, and gene expression typically associated with ALK-1 (5).

References:

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