

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived
Ala317-His454
Accession # P22003

N-terminal Sequence Analysis Ala317

Predicted Molecular Mass 15.6 kDa

SPECIFICATIONS

SDS-PAGE 18-23 kDa, reducing conditions

Activity Measured by its ability to induce alkaline phosphatase production by ATDC5 mouse chondrogenic cells. Nakamura, K. *et al.* (1999) *Exp. Cell Res.* **250**:351.
The ED₅₀ for this effect is typically 0.2–1.2 µg/mL.

Endotoxin Level <1.0 EU per 1 µg of the protein by the LAL method.

Purity >95%, by SDS-PAGE under reducing conditions and visualized by silver stain.

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

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 µg/mL in 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, -70 °C under sterile conditions after reconstitution.

BACKGROUND

Bone Morphogenetic Protein-5 (BMP-5) is one of at least 15 structurally and functionally related BMPs which are members of the transforming growth factor β (TGF-β) superfamily (1). BMP-5 is synthesized as a 454 amino acid (aa) precursor protein that is cleaved at the dibasic cleavage site (RxxR) to release the 20 kDa C-terminal mature protein (2). Mature BMP-5 contains seven conserved cysteine residues involved in formation of the cysteine knot and the single interchain disulfide bond. Biologically active BMP-5 is a disulfide-linked homodimer of the C-terminal mature protein. Mature human BMP-5 shares 96% aa sequence identity with mouse and rat BMP-5. Cellular responses to BMP-5 are mediated by the formation of hetero-oligomeric complexes of type I and type II serine/threonine kinase receptors (1). BMP-5 is expressed by chondrocytes in proliferating and hypertrophic zones of bone growth plates (3). It contributes to limb development by promoting proliferation and differentiation of chondrocytes as well as apoptosis of undifferentiated mesoderm (3, 4). Genetic defects in BMP-5 which cause C-terminal truncation or loss of the proteolytic cleavage site result in multiple skeletal abnormalities, including the *short ear* phenotype in mice (5, 6). BMP-5 is also expressed by ovarian granulosa cells where it functions as an autocrine factor to promote GC proliferation and inhibit their production of progesterone (7). In the nervous system, BMP-5 promotes dendrite outgrowth and dopaminergic neuronal differentiation (8, 9). It is upregulated in oral squamous carcinoma cells and induces the apoptosis of some myeloma cell lines (10, 11).

References:

1. Chen, D. *et al.* (2004) *Growth Factors* **22**:233.
2. Celeste, A.J. *et al.* (1990) *Proc. Natl. Acad. Sci.* **87**:9843.
3. Mailhot, G. *et al.* (2008) *J. Cell. Physiol.* **214**:56.
4. Zuzarte-Luis, V. *et al.* (2004) *Dev. Biol.* **272**:39.
5. King, J.A. *et al.* (1994) *Dev. Biol.* **166**:112.
6. Ho, A.M. *et al.* (2008) *BMC Dev. Biol.* **8**:35.
7. Pierre, A. *et al.* (2005) *Biol. Reprod.* **73**:1102.
8. Beck, H.N. *et al.* (2001) *BMC Neurosci.* **2**:12.
9. Brederlau, A. *et al.* (2002) *Mol. Cell. Neurosci.* **21**:367.
10. Jin, Y. *et al.* (2001) *Oral Oncol.* **37**:225.
11. Ro, T.B. *et al.* (2004) *Oncogene* **23**:3024.