

DESCRIPTION

Source *E. coli*-derived
Ser32-Gln115
Accession # P01270

N-terminal Sequence Analysis Ser32

Structure / Form Monomer

Predicted Molecular Mass 9.5 kDa

SPECIFICATIONS

SDS-PAGE 9 kDa, reducing conditions

Activity Measured by its ability to induce cAMP accumulation in MC3T3-E1 mouse preosteoblast cells.
The ED₅₀ for this effect is 10-60 ng/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 PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 250 µg/mL in PBS.

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

PTH (parathyroid hormone) is a critical hormone in the regulation of Ca⁺⁺ homeostasis (1). The human PTH cDNA encodes 115 amino acids (aa) including a 25 aa signal sequence, a 6 aa propeptide, and an 84 aa mature hormone. Mature human PTH shares 70%, 73%, 88%, 87%, 86%, 86% and 85% aa identity with mouse, rat, canine, equine, bovine, porcine and feline PTH, respectively. Multiple N-terminal peptides and C-terminal peptides derived from PTH occur naturally in the circulation (1). PTH aa 32-66, called PTH (1-34) since it represents the first 34 aa of the mature hormone, reproduces all the activity of the full length mature hormone and has been used therapeutically for treatment of osteoporosis (1-3). C-terminal peptides mainly oppose the activities of PTH (1-34) and are increased in renal failure (1-3). PTH expression is mainly restricted to the parathyroid gland, with minor amounts in the thymus (4). PTH secretion is enhanced by low Ca⁺⁺ concentrations and inhibited by FGF-23 (1, 5). In normal human plasma, PTH correlates negatively with active Vitamin D and positively with ionized calcium (6). Human and other mammalian PTH will bind and stimulate human or rat PTH1R, activating adenylate cyclase and increasing cAMP production (2, 7). PTH promotes secretion of TRANCE/RANKL and periostin through PTH1R binding on osteoblasts and/or bone marrow stromal cells (8-10). TRANCE/RANKL induces differentiation of osteoclasts, which in turn promote release of Ca⁺⁺ from bone (1, 8, 9). PTH1R on osteocytes, however, allows PTH to promote bone formation and IGF-1 production (11, 12). In renal epithelium, PTH promotes conversion of Vitamin D to its active form, lowers Ca⁺⁺ excretion and increases phosphate excretion (1, 2, 9). PTH also increases hematopoietic stem cell proliferation and mobilization and induces arterial vasodilation by regulating Ca⁺⁺ influx in PTH1R-expressing arterial smooth muscle (8, 13).

References:

1. Potts, J.T. (2005) *J. Endocrinol.* **187**:311.
2. Usatii, M. *et al.* (2007) *Kidney Int.* **72**:1330.
3. Scillitani, A. *et al.* (2011) *J. Clin. Invest.* **34**:23.
4. Liu, Z. *et al.* (2010) *PLoS Genet.* **6**:e1001251.
5. Ben-Dov, I.Z. *et al.* (2007) *J. Clin. Invest.* **117**:4003.
6. Carnevale, V. *et al.* (2010) *Bone* **47**:626.
7. Schipani, E. *et al.* (1993) *Endocrinology* **132**:2157.
8. Jacome-Galarza, C.E. *et al.* (2011) *J. Bone Miner. Res.* **26**:1207.
9. Lupp, A. *et al.* (2010) *Eur. J. Endocrinol.* **162**:979.
10. Fortunati, D. *et al.* (2010) *Matrix Biol.* **29**:594.
11. O'Brien, C.A. *et al.* (2008) *PLoS ONE* **3**:e2942.
12. Lombardi, G. *et al.* (2010) *J. Endocrinol. Invest.* **33**:22.
13. Zaruba, M.M. *et al.* (2008) *Cardiovasc. Res.* **77**:722.