

DESCRIPTION

Source *E. coli*-derived
Thr23-Thr216, with an N-terminal Met
Accession # P63075

N-terminal Sequence Analysis Met

Predicted Molecular Mass 22.7 kDa

SPECIFICATIONS

SDS-PAGE 23 kDa, reducing conditions

Activity Measured in a cell proliferation assay using NR6R-3T3 mouse fibroblast cells. Rizzino, A. *et al.* (1988) Cancer Res. **48**:4266; Thomas, K. *et al.* (1987) Methods Enzymol. **147**:120.
The ED₅₀ for this effect is 150-750 ng/mL, in the presence of 10 µg/mL heparin.

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 MOPS, (NH₄)₂SO₄, DTT and EDTA. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 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

FGF-17 is a member of the fibroblast growth factor (FGF) family. FGFs play multiple roles in biological functions, including angiogenesis, mitogenesis, cell differentiation and wound repair. FGFs share 30-70% amino acid (aa) identity in a conserved, approximately 120 amino acid core domain (1-3). The mouse or human FGF-17 cDNA encodes a cleavable 22 aa signal sequence and a 194 secreted mature protein (1). Mature mouse FGF-17 shares 100%, 99%, 99%, 97%, and 97% aa identity with rat, human, porcine, canine and equine FGF-17, respectively. The FGF domain of FGF-17 shares the most aa identity with FGF-8 (75%) and FGF-18 (64%). These three FGFs constitute a subfamily that overlaps in some areas of expression and function (1-5). All are reported to bind and signal through FGF R4 and the "c" splice forms of FGF R1-3 (6, 7). During embryogenesis, FGF-17 plays an organizing and inducing role in the patterning at the midbrain/hindbrain junction, and is also expressed in hindgut, parts of the developing skeleton, tail bud, major arteries, and heart (2-5). In many of these areas, it is expressed along with FGF-8, but slightly later (2-6). Unlike FGF-8 and FGF-18, deletion of FGF-17 produces viable mice. However, FGF-17^{-/-} mice show abnormalities in the dorsal frontal cortex, midbrain and cerebellum, manifested in some cases by ataxia, auditory defects, and abnormal social behavior (1, 4, 5, 8, 9). In the adult, FGF-17 is expressed in ovarian follicles and the prostate, and its expression is increased by both benign hypertrophy and cancer of the prostate (10-12). FGF-8, FGF-17, and FGF-18 are also abnormally expressed in many leukemic cell lines and can enhance growth of cancer cells (13).

References:

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