Recombinant Human FGF-17

Catalog Number: 319-FG/CF

DESCRIPTION

Source	*E. coli*-derived Thr23-Thr216, with and without an N-terminal Met Accession # O60258
N-terminal Sequence Analysis	Met & Thr23
Predicted Molecular Mass	22.6 kDa

SPECIFICATIONS

SDS-PAGE	21.5 & 22.6 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 ED_{50} for this effect is 100-500 ng/mL, in the presence of 1 µg/mL heparin, in a fluorometric assay using the redox sensitive dye, Resazurin (Catalog # AR002) and 15-60 ng/mL, in the presence of 1 µg/mL heparin, when measured by ^{3}H-thymidine incorporation.
Endotoxin Level	<0.10 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 100 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	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 3 months, 2 to 8 °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 human or mouse FGF-17 cDNA encodes a cleavable 22 aa signal sequence and a 194 secreted mature protein (1). Mature human FGF-17 shares 99% aa identity with mouse, rat, porcine and canine FGF-17. 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 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 humans, down-regulation of FGF-17 expression has been associated with Dandy-Walker cerebellar malformation (10). FGF-17 is also expressed in adult bovine ovarian follicles and the human prostate, and its expression is increased by both benign hypertrophy and cancer of the prostate (11-13). FGF-8, FGF-17, and FGF-18 are also abnormally expressed in many human leukemic cell lines and can enhance growth of cancer cells (14).

References:

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