

DESCRIPTION	
Source	<i>E. coli</i> -derived human FGF-3 protein Asp28-Arg212, with an N-terminal Met Accession # NP_005238
N-terminal Sequence Analysis	Met
Predicted Molecular Mass	21.1 kDa

SPECIFICATIONS	
Activity	Measured in a cell proliferation assay using NR6R-3T3 mouse fibroblast cells. Rizzino, A. <i>et al.</i> (1988) Cancer Res. 48 :4266; Thomas, K. <i>et al.</i> (1987) Methods Enzymol. 147 :120. The ED ₅₀ for this effect is 0.02-0.1 µg/mL in the presence of 1 µg/mL of heparin.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>97%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 μm filtered solution in MOPS, Na ₂ SO ₄ , TCEP and EDTA with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE		
Reconstitution	Reconstitute at 100 µg/mL in sterile, deionized water.	
Shipping	The product is shipped with polar packs. 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

Fibroblast Growth Factor 3 (FGF-3) belongs to the large FGF family which has at least 23 members (1, 2). All FGF family members are heparin-binding growth factors with a core 120 amino acid (aa) FGF domain that allows for a common tertiary structure. FGFs are expressed during embryonic development and in restricted adult tissues. They act on cells of mesodermal and neuroectodermal origin to regulate diverse physiologic functions including angiogenesis, cell growth, pattern formation, embryonic development, metabolic regulation, cell migration, neurotrophic effects and tissue repair (3, 4). Signaling receptors for FGFs are type I transmembrane receptor tyrosine kinases belonging to the Ig superfamily. Four distinct but related classes of FGF receptors, FGF R1, 2, 3, and 4, exist. Through alternative splicing, multiple isoforms for FGF R1, 2 and 3, with distinct ligand recognition profiles, are also generated (4).

The *FGF-3* gene, originally designated *int-2*, was first identified as a proto-oncogene activated in mouse mammary tumors by proviral integration (2). Amplification of this gene has also been found frequently in human tumors. Human FGF-3 cDNA predicts a 239 aa precursor protein with a 17 aa signal peptide and a 222 aa secreted mature protein with one potential N-linked glycosylation site (1). Human and mouse FGF-3 share 88% aa sequence identity. The *Xenopus* and mammalian secreted FGF-3 are processed proteolytically at both the N- and C-terminus (5). FGF-3 binds with high-affinity to the IIIb isoforms of FGF R1 and FGF R2. FGF-3 also binds the IIIc isoform of FGF R2, but with lower affinity (6). FGF-3 has been implicated in the induction of inner ear development (7). Studies have suggested that FGF-3 and FGF-8 function synergistically in otic placode induction and during neuronal development to regulate dorsoventral axis formation (8 - 10). During development, the activities of FGF-8 are regulated negatively by the sprouty family proteins and by Sef (similar expression to fg genes), a transmebrane protein that shares intracellular sequence similarities with the IL-17 receptor (10).

References:

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