Recombinant Mouse FGF-9

Catalog Number: 7399-F9/CF

DESCRIPTION

Source	*E. coli*-derived Met1-Ser208 Accession # NP_037084
N-terminal Sequence Analysis	Pro3
Structure / Form	Monomer
Predicted Molecular Mass	23.2 kDa

SPECIFICATIONS

SDS-PAGE	24 kDa, reducing conditions
Activity	Measured in a cell proliferation assay using Balb/3T3 mouse embryonic fibroblast cells. Rubin, J.S. *et al.* (1991) Proc. Natl. Acad. Sci. USA **88**:415. The ED_{50} for this effect is 1.5-7.5 ng/mL.
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 MOPS, Na_2SO_4 and EDTA. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 250 µg/mL in sterile, deionized water.
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-9 (fibroblast growth factor-9), also called HBGF-9 (heparin-binding growth factor-9) and GAF (glia-activating factor), is an approximately 26 kDa secreted glycoprotein of the FGF family (1-3). FGFs exhibit heparin-dependent regulation of cell proliferation, differentiation, and function, and are characterized by a core heparin-binding FGF domain of approximately 120 amino acids (aa) that exhibits a β-trefoil structure (1). FGF-9, -16 and -20 form a subfamily that shares 65-71% aa sequence identity, binds FGF R3 (IIIb), and are efficiently secreted despite having an uncleavable, bipartite signal sequence (1-3). Secreted mouse FGF-9 is a 205-207 aa protein that lacks the N-terminal 1-3 aa and shares >98% sequence identity with rat, human, equine, porcine and bovine FGF-9. In addition to FGF R3 (IIIb), FGF-9 binding to the IIIc splice forms of FGF R1, R2 and R3 are variably reported (3-5). An unusual constitutive dimerization of FGF-9 buries receptor interaction sites which lowers its activity, and increases heparin affinity which inhibits diffusion (4-6). A spontaneous mouse mutant, Eks, interferes with dimerization, resulting monomeric, diffusible FGF-9 that causes elbow and knee synostoses (joint fusions) due to FGF-9 misexpression in developing joints (6). In humans, FGF-9 mutations that lower receptor binding cause multiple synostoses syndrome (SYNS) (7). Expression in brain and kidney are reported in the adult rat (2, 8). In the mouse embryo the location and timing of FGF-9 expression affects development of the skeleton, cerebellum, lungs, heart, vasculature, digestive tract, and testes (1, 6-11). Deletion of mouse FGF-9 is lethal at birth due to lung hypoplasia, and causes rhizomelia, or shortening of the proximal skeleton (1, 10, 11). Altered FGF-9 expression or function is reported in human colon, endometrial, and ovarian cancers, correlating with progression, invasiveness, and survival (12-15).

References:

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