

DESCRIPTION

Source *Spodoptera frugiperda*, Sf 21 (baculovirus)-derived human FGF-9 protein
Met1-Ser208 and Ala2-Ser208
Accession # P31371

N-terminal Sequence Analysis Met1 & Ala2

Predicted Molecular Mass 23 kDa

SPECIFICATIONS

SDS-PAGE Multiple bands between 22-29 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 ED₅₀ for this effect is 1-5 ng/mL.

Endotoxin Level <1.0 EU per 1 µg of the protein by the LAL method.

Purity >97%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in MOPS, Na₂SO₄ and EDTA. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 µg/mL in sterile PBS.

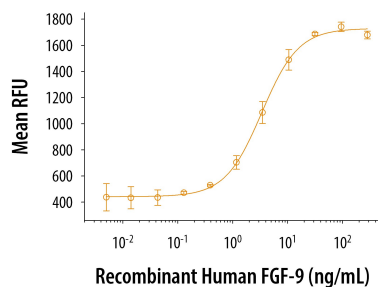
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

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.

DATA

Bioactivity



Bioactivity of Human FGF-9
Recombinant human FGF-9 (Catalog # 273-F9/CF) induces proliferation in the Balb/3T3 mouse embryonic fibroblast cell line. The ED₅₀ for this effect is 1-5 ng/mL.

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 human FGF-9 is a 205-207 aa protein that lacks the N-terminal 1-3 aa and shares >98% sequence identity with mouse, rat, 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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