Datasheet

Cat. No. PH2045

Recombinant Human FGF9

Information

Accession #	PH2045
Alternate Names	Fibroblast Growth Factor 9
Source	Met1 & Ala2
Protein sequence	
M.Wt	23 kDa
Appearance	Solution protein
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. - 3 years from date of receipt, -20 to -70°C as supplied.
Concentration	0. 2 mg/mL
Formulation	Dissolved in sterile PBS buffer.
Reconstitution	We recommend that this vial be briefly centrifuged prior to opening to bring the contents to the bottom. This solution can be diluted into other aqueous buffers.
Biological Activity	
Shipping Condition	Shipping with dry ice.
Handling	Centrifuge the vial prior to opening.
Usage	For Research Use Only! Not to be used in humans.

Quality Control

Purity	> 95%, determined by SDS-PAGE.
Endotoxin	＜1.0 EU per 1 ug of the protein by the LAL method.

Description

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 prolifer ation, differentiation, and function, and are characterized by a core heparin-binding FGF domain of approximately 120 amino acids (aa) that exhibits a beta -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].

Reference

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