

Recombinant Mouse FGF-9/FGF9 Protein (His Tag)

Catalog Number: PKSM041022

Note: Centrifuge before opening to ensure complete recovery of vial contents.

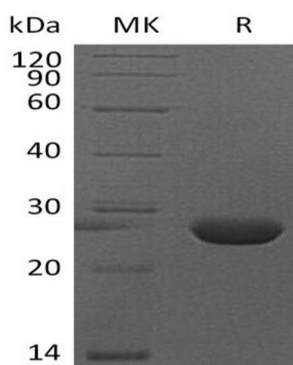
Description

Species	Mouse
Source	E.coli-derived Mouse FGF-9/FGF9 protein Met1-Ser208, with an N-terminal His
Calculated MW	24.4 kDa
Observed MW	25 kDa
Accession	P54130
Bio-activity	Not validated for activity

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Concentration	Subject to label value.
Endotoxin	< 0.01 EU per µg of the protein as determined by the LAL method.
Storage	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < -20°C.
Formulation	Supplied as a 0.2 µm filtered solution of 20mM Tris-HCl, 150mM NaCl, 5% Trehalose, 1mM EDTA, 20% Glycerol, 1mM DTT, pH 8.5.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

Fibroblast growth factor-9 (FGF-9) is an approximately 26 kDa secreted glycoprotein of the FGF family. Secreted mouse FGF-9 lacks the N-terminal 1-3 aa and shares >98% sequence identity with rat, human, equine, porcine and bovine FGF-9. FGF-9 plays an important role in the regulation of embryonic development, cell proliferation, cell differentiation and cell migration. 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. It may have a role in glial cell growth and differentiation during development, gliosis during repair and regeneration of brain tissue after damage, differentiation and survival of neuronal cells, and growth stimulation of glial tumors. Deletion of mouse FGF-9 is lethal at birth due to lung hypoplasia, and causes rhizomelia, or shortening of the proximal skeleton. An unusual constitutive dimerization of FGF 9 buries receptor interaction sites which lowers its activity, and increases heparin affinity which inhibits diffusion. 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.

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