

DESCRIPTION

Source *E. coli*-derived human FGF basic/FGF2/bFGF protein
Pro143-Ser288, with an N-terminal Ala
Accession # P09038

N-terminal Sequence Analysis Ala-Pro143

Predicted Molecular Mass 16.5 kDa

SPECIFICATIONS

SDS-PAGE 17 kDa, reducing conditions

Activity Measured in a cell proliferation assay using NR6R-3T3 mouse fibroblast cells. Raines, E.W. *et al.* (1985) *Methods Enzymol.* **109**:749. The ED₅₀ for this effect is 0.1-0.6 ng/mL.
The specific activity of Recombinant Human FGF basic/FGF2/bFGF is approximately 800 IU/μg, which is calibrated against recombinant human FGF basic/FGF2 basic WHO International Standard (NIBSC code: 90/712).

Endotoxin Level <0.10 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 Tris-HCl and NaCl with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100-250 μg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.

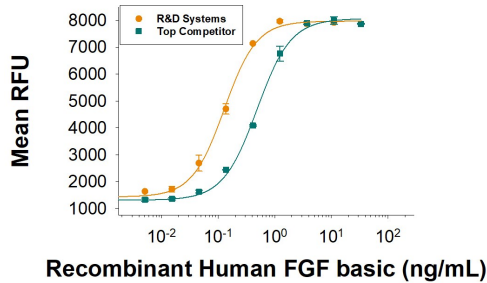
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

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.

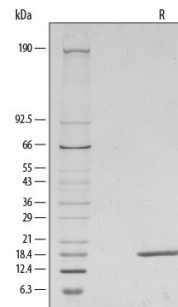
DATA

Bioactivity



Recombinant Human FGF basic/FGF2/bFGF (146 aa) (Catalog # 233-FB) stimulates cell proliferation of the NR6R-3T3 mouse fibroblast cell line. The activity is approximately 3-fold greater than the top competitor's FGF basic (146 aa).

SDS-PAGE



1 μg/lane of Recombinant Human FGF basic/FGF2/bFGF (146 aa) was resolved by SDS-PAGE with silver staining, under reducing (R) conditions, showing a band at 17 kDa.

BACKGROUND

FGF basic (also known as FGF-2 and HBGF-2) is a member of the FGF superfamily of mitogenic proteins which show 35-60% amino acid conservation. FGF acidic and basic are unique from other members of the family in that they lack classical secretory signal peptides. However, they are both readily secreted from cells by an alternative secretory pathway involving direct translocation and aided by several chaperones. FGF acidic (FGF-1) and FGF basic (FGF-2) were the first two identified FGFs, and the designations acidic and basic refer to their relative isoelectric points. The full length human FGF basic protein is 288 amino acids, but there are multiple start sites which produce various shorter forms. Further adding to the complexity, a variety of forms of FGF basic are produced as a result of N-terminal extensions. These extensions affect localization of FGF basic in cellular compartments but do not affect biological activity. FGF basic has been isolated from a number of sources, including neural tissue, adrenal cortex, pituitary gland, corpus luteum, and placenta. Binding of FGF to heparin or cell surface heparan sulfate proteoglycans is required for FGF binding with high affinity to FGF receptors. FGF basic stimulates proliferation of all cells of mesodermal origin as well as many cells of neuroectodermal, ectodermal, and endodermal origin. FGF basic also induces neuronal differentiation, survival, and regeneration, and modulates embryonic development and differentiation. These observed in vitro functions suggest FGF basic may play a role in vivo in the modulation of such normal processes as angiogenesis, wound healing and tissue repair, embryonic development and differentiation, and neuronal function and neural degeneration. Additionally, FGF basic may also participate in the development of several pathological conditions resulting from excessive cell proliferation and/or angiogenesis.

References:

1. Coulier, F. *et al.* (1997) *J. Mol. Evol.* **44**:43.
2. Chen, C.H. *et al.* (2004) *Curr. Vasc. Pharmacol.* **2**:33.
3. Mohammadi, M. *et al.* (2005) *Curr. Opin. Struct. Biol.* **15**:506.
4. Fernig, D. *et al.* (1994) *Prog. Growth Factor Res.* **5**:353.