

#### DESCRIPTION

**Source** *E. coli*-derived  
Pro143-Ser288, with an N-terminal Ala  
Accession # NP\_001997

**N-terminal Sequence Analysis** Ala - Pro143

**Predicted Molecular Mass** 16.0 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 ED<sub>50</sub> for this effect is typically 0.1-0.6 ng/mL.

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

**Purity** >97%, by SDS-PAGE under reducing conditions and visualized by silver stain.

**Formulation** Lyophilized from a 0.2 µm filtered solution in Tris and NaCl with BSA as a carrier protein. See Certificate of Analysis for details.

#### PREPARATION AND STORAGE

**Reconstitution** Reconstitute at 100 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.

#### BACKGROUND

FGF basic is a member of the FGF family of at least 23 related mitogenic proteins which show 35 – 60% amino acid conservation. FGF acidic and basic, unlike the other members of the family, lack signal peptides and are apparently secreted by mechanisms other than the classical protein secretion pathway. FGF basic has been isolated from a number of sources, including neural tissue, pituitary, adrenal cortex, corpus luteum, and placenta. This factor contains four cysteine residues, but reduced FGF basic retains full biological activity, indicating that disulfide bonds are not required for this activity. 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. Binding of FGF to heparin or cell surface heparan sulfate proteoglycans is necessary for binding of FGF to high affinity FGF receptors. FGF acidic and basic appear to bind to the same high affinity receptors and show a similar range of biological activities. FGF basic stimulates the proliferation of all cells of mesodermal origin and many cells of neuroectodermal, ectodermal, and endodermal origin. FGF basic induces neuron differentiation, survival, and regeneration. FGF basic also modulates embryonic development and differentiation. These observed *in vitro* functions of FGF basic 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 participate in the production of a variety of pathological conditions resulting from excessive cell proliferation and excessive 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.