

DESCRIPTION

Source *E. coli*-derived
Pro10-Ser155, with an N-terminal Ala
Accession # P03969
Produced using non-animal reagents in an animal-free laboratory.

N-terminal Sequence Analysis Ala

Predicted Molecular Mass 16.5 kDa

SPECIFICATIONS

Activity Measured in a cell proliferation assay using NR6R-3T3 mouse fibroblast cells. Rizzino, A. *et al.* (1988) *Cancer Res.* **48**:4266; Thomas, K. *et al.* (1987) *Methods Enzymol.* **147**:120.
The ED₅₀ for this effect is typically 0.1-1.5 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-HCl and NaCl. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 0.2 mg/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.
- 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, comprised of 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. Several reports indicate that a variety of forms of FGF basic are produced as a result of N-terminal extensions. These extensions apparently affect localization of FGF basic in cellular compartments but do not affect biological activity. Studies indicate that 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. The cells include fibroblasts, endothelial cells, astrocytes, oligodendrocytes, neuroblasts, keratinocytes, osteoblasts, smooth muscle cells, and melanocytes. FGF basic is chemotactic and mitogenic for endothelial cells *in vitro*. FGF basic induces neuron differentiation, survival and regeneration. FGF basic has also been shown to be crucial in modulating 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 (1 - 2).

References:

1. Coulier, F. *et al.* (1997) *J. Mol. Evol.* **44**:43.
2. Fernig, D. *et al.* (1994) *Prog. Growth Factor Res.* **5**:353.