WHAT IS FGF?

Fibroblast growth factors or FGFs are a family of growth factors involved in angiogenesis, wound healing and embryonic development. The FGFs are heparin binding proteins and interactions with cell surface associated heparin sulfate proteoglycans have been shown to be essential for FGF signal transduction. FGFs are key players in the processes of proliferation and differentiation of wide variety of cell tissues.

A detailed day-by-day study was performed in 1988 (A4, A11). Discovered only in the seventies, and also a peptide, this FGF is critical in the development of embryos, including humans. However, it is not found to be circulating in the human adult bodies.

Nerve growth factor (NGF) is a small secreted protein with is important for the growth, maintenance and survival of certain target neurons (nerve cells). It also functions as a signaling molecule. [1] [2] It is perhaps the prototypical growth factor, in that it is one of the first to be described. While “nerve growth factor” refers to a single factor, [3] ‘nerve growth factors” refers to a family of factors also known as neurotorphins. [4] Other members of the neurotropic family that are well recognized include Brain-Derived Neurotropic Factor (BDNF), Neurotropin-3 (NT-3) and Neurotropin 4/5 (NT-4/5).

EGF results in cellular proliferation, differentiation and survival.[5] EGF is a low molecular weight polypeptide first purified from the mouse submandibular gland, but since then found in many human tissues including Submandibular gland and Parotid gland. Salivary EGF which seems also regulated by dietary inorganic iodine also plays an important physiological role in the maintenance of oro-esophageal and gastric tissue integrity. The biological effects of salivary EGF include healing oral and gastro esophageal ulcers, inhibition of gastric acid secretion, stimulation of DNA synthesis as well as mucosal protection from intraluminal injurious factors such as gastric acid, bile acid, pepsin, and trypsin and to physical, chemical and bacterial agents. [6]

CTGF (connective tissue growth factor) is a cysteine rich, matrix associated, heparin binding protein. In vitro, CTGF mirrors some of the effects of TGH beta on skin fibroblasts such as simulation of extracellular heparin matrix binding proteins. CTGF has important roles in many biological processes, including cell adhesion, migration, proliferation, angiogenesis, skeletal development, and tissue wound repair, and is critically involved in fibrotic disease and several forms of cancers.

How is FGF helpful to humans?

The precise blend of oligopeptides may be seen as building blocks, without a bridge, or a director. The role of such a director is fulfilled by a growth factor known as the Fibroblast Growth Factor, (repair factor) or FGF. FGF is prolific in PESE, as well as in the human placenta. On the 11th day of the incubation cycle of a chicken egg, the embryonic tissue shows a steep increase in the FGF, with the appropriate peptides to form the solid organs and bones (A1).

FGF is responsible for building the linings in the blood vessels, creating the infrastructure for the nutrients to flow to critical areas of the brain and organs. Research credits FGF with the potential to directly affect many neuro disorders because of clear results of the ability of

FGF to affect the growth of neurites (A2). Neurites are signal senders (Axons) and signal receivers (dendrites) attached to the brain neurons.

Research (A7) has also shown clearly that new cell cultures show a dramatic increase in peptide and amino acid uptake in the presence of FGF. This result gives credence to the hypothesis that embryonic growth is influenced by a very precise mechanism, which combines unique combinations of amino acids, peptides and FGF.

Since FGF is not circulating in adults, multiple research projects on the effects of FGF serums to cure neurological disorders have been carried out.

Fundamental to the research is the fact discovered by Altman, J. in 1962 (A26) that neural STEM cells are formed by the body in response to abnormalities, and are resident in certain zones of the brain. The brain is therefore ready to repair the damage, and these cells have shown to differentiate into a wide range of neurons (A27). Neurons derived from such neural stem cells are capable of migrating to various regions of the Central Nervous System. Over a decade of work, both in vivo and ex vivo has revealed that exposure to such neural stem cells to FGF permits direct differentiation into the required neural cells (A14, A25).

REFERENCES

(1) Roberts, Pamela R, et al. Nutrition Vol. 14, No. 3, 1998
(2) Kuljis, Rodrigo O. Jour. of Neuropathology & Exp. Neur., 1994.
(3) Jiangyong Min, et al. Jour. of Neuroscience Res., 86:2984-2991 (2008)
(4) Z.Y. Zhou, et al. Neuroscience, Vol. 90, No. 4, 1493-1499, 1999
(5) Arvanitakis, Constantine. Am. Jour. of Physiology, Vol. 231, No. 1, July 1976.
(6) Kristoffer, ester S., ETC Research & Development, Oslo, Norway.

(A1) Joseph-Silverstein, Jacquelyn, et al (June 1989) Basic Fibroblast Growth Factor in the Chick Embryo: Immunolocalization to Striated Muscle Cells and Their Precursors. *The Journal of Cell Biology*, 108: 2459-2466.
(A2) Hatten, M. E., et al (1988) In Vitro Neurite Extension by Granule Neurons is Dependent upon Astroglial-Derived Fibroblast Growth Factor. *Developmental Biology*, 125:280-289.
(A4) Seed, Jennifer, et al (1988) Fibroblast Growth Factor Levels in the Whole Embryo and Limb Bud during Chick Development. *Developmental Biology*, 128:50-57.
(A11) Seed, Jennifer, et al (1988) Fibroblast Growth Factor Levels in the Whole Embryo and Limb Bud during Chick Development. *Developmental Biology*, 128:50-57.
(A14) La Spada, Albert R (December 2005) Huntington's disease and Neurogenesis: FGF-2 to the Rescue? Vol. 102.
(A24) Hagg, Theo (2005) Molecular Regulation of Adult CNS Neurogenesis: an Integrated View.
(A25) Bjugstad, K. B., et al (2001) IGF-1 and bFGF Reduce Glutaric Acid and 3- Hydroxyglutaric Acid Toxicity in Striatal Cultures.
(A26) Altman, J. (1962) Science 132:1127-1128.
(A27) Arlotta, P., et al (2003) Exp. Gerontol, 38:173-182.