

# Basic fibroblast growth factor

Basic [fibroblast growth factor](#), also known as bFGF, [FGF2](#) or FGF- $\beta$ , is a member of the fibroblast growth factor family.

Basic fibroblast growth factor and [nerve growth factor](#) (NGF) are essential for proper development, survival, growth, and maintenance of [neurons](#) in the central and [peripheral nervous systems](#). However, because bFGF and NGF have short half-life and rapid diffusion rate, they have limited clinical efficacy.

In normal tissue, basic fibroblast growth factor is present in basement membranes and in the subendothelial extracellular matrix of blood vessels. It stays membrane-bound as long as there is no signal peptide.

It has been hypothesized that, during both wound healing of normal tissues and tumor development, the action of heparan sulfate-degrading enzymes activates bFGF, thus mediating the formation of new blood vessels, a process known as angiogenesis.

In addition, it is synthesized and secreted by human adipocytes and the concentration of bFGF correlates with the BMI in blood samples. In this study, bFGF was also shown to act on preosteoblasts – in the form of an increased proliferation – after binding to [fibroblast growth factor receptor 1](#) and activating phosphoinositide 3-kinase.

bFGF has been shown in preliminary animal studies to protect the heart from injury associated with a heart attack, reducing tissue death and promoting improved function after reperfusion.

Recent evidence has shown that low levels of FGF2 play a key role in the incidence of excessive anxiety.

Additionally, bFGF is a critical component of human embryonic stem cell culture medium; the growth factor is necessary for the cells to remain in an undifferentiated state, although the mechanisms by which it does this are poorly defined. It has been demonstrated to induce gremlin expression which in turn is known to inhibit the induction of differentiation by bone morphogenetic proteins.

It is necessary in mouse-feeder cell dependent culture systems, as well as in feeder and serum-free culture systems.

FGF2, in conjunction with BMP4, promote differentiation of stem cells to mesodermal lineages. After differentiation, BMP4 and FGF2 treated cells generally produces higher amounts of osteogenic and chondrogenic differentiation than untreated stem cells.

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see [Basic fibroblast growth factor in medulloblastoma](#).

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Fibroblast growth factors, or FGFs, are a family of growth factors, with members involved in angiogenesis, wound healing, embryonic development and various endocrine signaling pathways. The FGFs are heparin-binding proteins and interactions with cell-surface-associated heparan 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 cells and tissues.

Cai et al., demonstrated that basic [fibroblast growth factor](#) (bFGF), as a [neurotrophic factor](#), inhibited [Endoplasmic reticulum](#) (ER) stress-induced neuronal cell [apoptosis](#) and that [Oxidopamine](#) (6-hydroxydopamine 6-OHDA)-induced ER stress was involved in the progression of [Parkinson's disease](#) (PD) in rats. bFGF administration improved motor function recovery, increased [tyrosine hydroxylase](#) (TH)-positive neuron survival, and upregulated the levels of [neurotransmitters](#) in PD rats.

The 6-OHDA-induced ER stress response proteins were inhibited by bFGF treatment. Meanwhile, bFGF also increased expression of TH. The administration of bFGF activated the downstream signals PI3K/Akt and Erk1/2 in vivo and in vitro. Inhibition of the PI3K/Akt and Erk1/2 pathways by specific inhibitors partially reduced the protective effect of bFGF. This study provides new insight towards bFGF translational drug development for PD involving the regulation of ER stress <sup>1)</sup>.

To more efficiently deliver bFGF and NGF, we used a coacervate (synthesized with heparin and a biodegradable polycation at mass ratio of 500: 100). The maximal package loads of GFs in coacervate were determined by Western Blotting; release efficiency of bFGF and NGF was measured by ELISA. Additionally, we evaluated the effect of bFGF and NGF on the viability, survival, and proliferation of neurons by MTT assay, BrdU cell proliferation, and calcein staining.

Coacervate incorporated bFGF and NGF and continuously released them for at least three weeks. This enhanced the growth and proliferation of PC12 cells and SH-SY5Y cells. Moreover, co-delivery of bFGF and NGF using coacervate was more neuroprotective than free application of both factors or coacervate delivery of each GF separately.

Dual delivery of bFGF and NGF binding coacervate was neuroprotective via stimulating the growth and proliferation of neurons <sup>2)</sup>.

<sup>1)</sup>

Cai P, Ye J, Zhu J, Liu D, Chen D, Wei X, Johnson NR, Wang Z, Zhang H, Cao G, Xiao J, Ye J, Lin L. Inhibition of Endoplasmic Reticulum Stress is Involved in the Neuroprotective Effect of bFGF in the 6-OHDA-Induced Parkinson's Disease Model. *Aging Dis.* 2016 Jan 17;7(4):336-449. doi: 10.14336/AD.2016.0117. eCollection 2016 Aug. PubMed PMID: 27493838; PubMed Central PMCID: PMC4963188.

<sup>2)</sup>

Wu Y, Wang Z, Cai P, Jiang T, Li Y, Yuan Y, Li R, Khor S, Lu Y, Wang J, Chen D, Zeng Q, Zhong R, Zhang H, Lin Y, Li X, Xiao J. Dual Delivery of bFGF- and NGF-Binding Coacervate Confers Neuroprotection by Promoting Neuronal Proliferation. *Cell Physiol Biochem.* 2018 Jun 12;47(3):948-956. doi: 10.1159/000490139. [Epub ahead of print] PubMed PMID: 29895019.

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