

DESCRIPTION

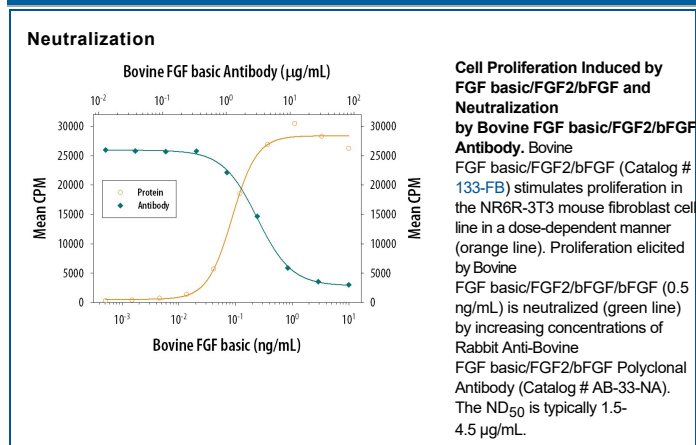
Species Reactivity	Bovine
Specificity	Detects bovine FGF basic/FGF2/bFGF in direct ELISAs and Western blots. In direct ELISAs, approximately 5% cross-reactivity with FGF acidic and recombinant human (rh) β -ECGF is observed and less than 1% cross-reactivity with rhFGF-4, rhFGF-5, rhFGF-6, rhFGF-7, recombinant mouse (rm) FGF-8b, rmFGF-8c, rhFGF-9, rhFGF-10, rmFGF-15, rhFGF-17, and rhFGF-18 is observed. Neutralizes the biological activity of bovine FGF basic/FGF2/bFGF and will also neutralize the biological activity of recombinant human FGF basic/FGF2/bFGF.
Source	Polyclonal Rabbit IgG
Purification	Protein A or G purified
Immunogen	Bovine brain-derived FGF basic/FGF2/bFGF
Endotoxin Level	<0.10 EU per 1 μ g of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 μ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

	Recommended Concentration	Sample
Western Blot	1 μ g/mL	Bovine FGF basic/FGF2/bFGF (Catalog # 133-FB)
Neutralization	Measured by its ability to neutralize FGF basic/FGF2/bFGF-induced proliferation in the NR6R-3T3 mouse fibroblast cell line. Rizzino, A. et al. (1988) Cancer Res. 48 :4266. The Neutralization Dose (ND ₅₀) is typically 1.5-4.5 μ g/mL in the presence of 0.5 ng/mL Bovine FGF basic/FGF2/bFGF.	

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 1 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	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <ul style="list-style-type: none"> 12 months from date of receipt, -20 to -70 °C as supplied. 1 month, 2 to 8 °C under sterile conditions after reconstitution. 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

FGF basic is a member of the FGF family, currently comprised of seven related mitogenic proteins which show 35-55% 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.