

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human and bovine FGF basic/FGF2/bFGF in direct ELISAs and Western blots. In direct ELISAs, approximately 75% cross-reactivity with recombinant mouse FGF basic/FGF2/bFGF is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Bovine brain-derived FGF basic/FGF2/bFGF
Conjugate	Alexa Fluor 594 Excitation Wavelength: 590 nm Emission Wavelength: 617 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.

Neutralization	Optimal dilution of this antibody should be experimentally determined.
Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunohistochemistry	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

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.

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