

DESCRIPTION	
Species Reactivity	Human
Specificity	Detects human KGF in direct ELISAs and Western blots. In direct ELISAs and Western blots, this antibody does not cross-react with recombinant human (rh) FGF-3, rhFGF-4, rhFGF-5, rhFGF-6, rhFGF-9, rhFGF-10, rhFGF-11, rhFGF-13, rhFGF-16, rhFGF-17, rhFGF-18, rhFGF-19, rhFGF acidic, rhFGF basic, rmFGF-8b, rmFGF-8c, or rmFGF-15.
Source	Monoclonal Mouse IgG ₁ Clone # 29568
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant human KGF Cys32-Thr194 Accession # P21781
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

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 µg/mL	Recombinant Human KGF/FGF-7 (Catalog # 251-KG)

PREPARATION AND STORAGE	
Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <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

KGF (keratinocyte growth factor), also known as FGF-7 (fibroblast growth factor-7), is one of 22 known members of the mouse FGF family of secreted proteins that plays a key role in development, morphogenesis, angiogenesis, wound healing, and tumorigenesis (1 - 4). KGF expression is restricted to cells of mesenchymal origin. When secreted, it acts as a paracrine growth factor for nearby epithelial cells (1). KGF speeds wound healing by being dramatically upregulated in response to damage to skin or internal structures that results in high local concentrations of inflammatory mediators such as IL-1 and TNF-α. (2, 5). KGF promotes cell migration and invasion, and mediates melanocyte transfer to keratinocytes upon UVB radiation (6, 7). It has been used ectopically to avoid chemotherapy-induced oral mucositis in patients with hematological malignancies (1). Deletion of KGF affects kidney development, producing abnormally small ureteric buds and fewer nephrons (8). It also impedes hair follicle differentiation (9). The 194 amino acid (aa) KGF precursor contains a 31 aa signal sequence and, like all other FGFs, an ~120 aa β-trefoil scaffold that includes receptor- and heparin-binding sites. KGF signals only through the IIIb splice form of the tyrosine kinase receptor, FGF R2 (FGF R2-IIIb/KGF R) (10). Receptor dimerization requires an octameric or larger heparin or heparin sulfate proteoglycan (11). FGF-10, also called KGF2, shares 51% aa identity and similar function to KGF, but shows more limited expression than KGF and uses an additional receptor, FGF R2-IIIc (12). Following receptor engagement, KGF is typically degraded, while FGF-10 is recycled (12). Mature human KGF, which is active across species, shares 98% aa sequence identity with bovine, equine, ovine, and canine, 96% with mouse and porcine, and 92% with rat KGF, respectively.

References:

1. Finch, P.W. and J.S. Rubin (2006) *J. Natl. Cancer Inst.* **98**:812.
2. Werner, S. *et al.* (2007) *J. Invest. Dermatol.* **127**:998.
3. Werner, S. (1998) *Cytokine Growth Factor Rev.* **9**:153.
4. Mason, I.J. *et al.* (1994) *Mech. Dev.* **45**:15.
5. Geer, D.J. *et al.* (2005) *Am. J. Pathol.* **167**:1575.
6. Niu, J. *et al.* (2007) *J. Biol. Chem.* **282**:6001.
7. Cardinali, G. *et al.* (2005) *J. Invest. Dermatol.* **125**:1190.
8. Qiao, J. *et al.* (1999) *Development* **126**:547.
9. Guo, L. *et al.* (1996) *Genes Dev.* **10**:165.
10. de Georgi, V. *et al.* (2007) *Dermatol. Clin.* **25**:477.
11. Hsu, Y-R. *et al.* (1999) *Biochemistry* **38**:2523.
12. Belleudi, F. *et al.* (2007) *Traffic* **8**:1854.