

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human HB-EGF in direct ELISAs and Western blots. Does not cross-react with recombinant human (rh) AR, rhBTC, rhHRG- α , rhHRG- β , rhEGF, or rhTGF- α .
Source	Monoclonal Mouse IgG _{2A} Clone # 125923
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>S. frugiperda</i> insect ovarian cell line Sf21-derived recombinant human HB-EGF Asp63-Leu148 Accession # Q53H93
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	Recombinant Human HB-EGF (Catalog # 259-HE) under non-reducing conditions only

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.
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 from date of receipt, 2 to 8 °C, reconstituted. ● 6 months from date of receipt, -20 to -70 °C, reconstituted.

BACKGROUND

HB-EGF was originally purified based on its heparin-binding property and mitogenic activity on BALB-3T3 fibroblasts from the conditioned medium of the human U-937 histiocytic lymphoma cell line. The natural protein has an apparent molecular mass of 19-23 kDa and exists in multiple forms as a result of heterogeneous O-glycosylation and/or N-terminal truncation. In addition to fibroblasts, HB-EGF is also a potent mitogen for keratinocytes and smooth muscle cells but not for capillary endothelial cells. HB-EGF is produced in monocytes and macrophages. In addition, transcription of HB-EGF can be induced in vascular endothelial cells as well as aortic smooth muscle cells (SMC), suggesting that HB-EGF may have an important role in the pathogenesis of atherosclerosis.

HB-EGF is a member of the EGF family of mitogens which also include transforming growth factor- α (TGF- α), amphiregulin (AR), rat schwannoma-derived growth factor (SDGF), vaccinia growth factor (VGF), and the various ligands for the HER2/ErbB2/Neu receptor. All these cytokines are derived from transmembrane precursors that contain one or several EGF structural units in their extracellular domain. Many of these transmembrane precursors are biologically active and seem to play a role in juxtacrine stimulation of adjacent cells. The cDNA for HB-EGF encodes a 204 amino acid residue transmembrane protein that is proteolytically cleaved to generate the soluble HB-EGF. Like EGF, TGF- α , and AR; HB-EGF binds to the EGF receptor and activates the receptor tyrosine kinase. HB-EGF is reported to be a more potent SMC mitogen than EGF. It has been suggested that the differential activities found for HB-EGF compared to EGF may be mediated by the heparin-binding properties of HB-EGF. A diphtheria toxin receptor that mediates the endocytosis of the bound toxin has been cloned and found to be identical to the transmembrane HB-EGF precursor.