Recombinant Canine HB-EGF Protein DataSheet

Catalog Number: GR122144

Background

Heparin-binding EGF-like growth factor (HB-EGF) is a member of the EGF family that is encoded by the HBEGF gene.[1] HB-EGF-like growth factor is synthesized as a membrane-anchored mitogenic and chemotactic glycoprotein and produced by monocytes and macrophages. It has been shown to play a role in wound healing, cardiac hypertrophy, and heart development and function.[2] First identified in the conditioned media of human macrophage-like cells, HB-EGF is an 87-amino acid glycoprotein that displays highly regulated gene expression.[3] Ectodomain shedding results in the soluble mature form of HB-EGF, which influences the mitogenicity and chemotactic factors for smooth muscle cells and fibroblasts. The transmembrane form of HB-EGF is the unique receptor for diphtheria toxin and functions in juxtacrine signaling in cells. Both forms of HB-EGF participate in normal physiological processes and in pathological processes including tumor progression and metastasis, organ hyperplasia, and atherosclerotic disease.[4] HB-EGF can bind two locations on cell surfaces: heparan sulfate proteoglycans and EGF-receptors effecting cell-to-cell interactions.[5] Recent studies indicate significant HB-EGF gene expression elevation in some human cancers as well as cancer-derived cell lines and HB-EGF plays a significant role in the development of malignant phenotypes contributing to the metastatic and invasive behaviors of tumors.[6] The proliferative and chemotactic effects of HB-EGF results from the target influence on particular cells including fibroblasts, smooth muscles cells, and keratinocytes. Both in vivo and in vitro studies of tumor formation in cancer derived cell lines indicate that expression of HB-EGF is essential for tumor development. During valve tissue development the interaction of HB-EGF with EGF receptors and heparan sulfate proteoglycans is essential for the prevention of malformation of valves due to enlargement.[7] The flow disturbance remodeling of the vascular tissues due to HB-EGF expression contributes to aortic valve disease, peripheral vascular disease, and conduit stenosis.[8]

References

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3. Jin K, et al. (2002). J. Neurosci. 22 (13): 5365–73.
4. Raab G, Klagsbrun M (1997). Biochim. Biophys. Acta. 1333 (3): F179–99.
5. Das SK, et al. (1994). Development. 120 (5): 1071–83.
6. Miyamoto S, et al. (2006). Cancer Sci. 97 (5): 341–7.
7. Iwamoto R, Mekada E (2006). Cell Struct. Funct. 31 (1): 1–14.
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Description

Source: *E coli*-derived
Compositions: Val61–His208
Accession # A0A8I3RPS3
Predicted Molecular Mass: 17 kDa (monomer)

Specifications

Activity Measured in a cell proliferation assay using Balb/3T3 mouse embryonic fibroblast cells. Rubin JS (1991) Proc Natl Acad Sci USA 88:415. The ED50 for this effect is typically 0.2-1 ng/mL.
Endotoxin Level: <1.0 EU per 1 μg of the protein by the LAL method.
Purity: >95%, by SDSPAGE under reducing conditions and visualized by silver stain.
Formulation: Lyophilized from a 0.2 μm filtered PBS with BSA as a carrier protein.

Preparation and Storage

Reconstitution: Reconstitute at 50-500 μg/mL in sterile PBS containing 1 mg/ml of human or serum albumin.
Shipping The product is shipped at ambient temperature or with wet ice. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage: Use a manual defrost freezer and avoid repeated freeze thaw cycles.

- 6 months, -20 °C as supplied.
- 3 months, -20 to -70°C under sterile conditions after reconstitution.

DECLARATION

THIS REAGENT IS FOR IN VITRO LABORATORY TESTING AND RESEARCH USE ONLY. DO NOT USE IT FOR CLINICAL DIAGNOSTICS. DO NOT USE OR INJECT IT IN HUMANS AND ANIMALS.

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