

RELM a Mouse

Description: Mouse RELM-alpha Recombinant produced in E.Coli is a monomeric, non-glycosylated, polypeptide chain containing 88 amino acids and having a molecular mass of 10 kDa. The Mouse RELM-alpha is purified by proprietary chromatographic techniques.

Synonyms: Resistin-like alpha, RELMalpha, Cysteine-rich secreted protein FIZZ1, Parasite-induced macrophage novel gene 1 protein, Cysteine-rich secreted protein A12-gamma, RELM-a.

Source: Escherichia Coli.

Physical Appearance: Sterile Filtered White lyophilized (freeze-dried) powder.

Amino Acid Sequence: MDETIEIIVE NKVKELLANP ANYPSTVTKT LSCTSVKTMN
RWASCPAGMT ATGCACGFAC GSWEIQSGDT CNCLCLLDVW TTARCCQLS.

Purity: Greater than 98% as determined by SDS-PAGE & RP-HPLC.

Formulation:

Sterile filtered and lyophilized from 0.5mg/ml in 10mM sodium phosphate buffer, pH 7.5.

Stability:

Store lyophilized protein at -20°C. Aliquot the product after reconstitution to avoid repeated freezing/thawing cycles. Reconstituted protein can be stored at 4°C for a limited period of time; it does not show any change after two weeks at 4°C.

Usage:

NeoBiolab's products are furnished for LABORATORY RESEARCH USE ONLY. The product may not be used as drugs, agricultural or pesticidal products, food additives or household chemicals.

Solubility:

At 0.1mg/ml of deionized sterile water.

Introduction:

Bronchoalveolar lavage fluid from mice with experimentally induced allergic pulmonary inflammation contains a novel 9.4 kDa cysteine-rich secreted protein, RELM-alpha (FIZZ1, found in inflammatory zone). RELM-alpha is a secreted protein that has a restricted tissue distribution with highest levels in adipose tissue stroma. Murine RELM-alpha (FIZZ1) is the founding member of a new gene family including two other murine genes expressed, respectively, in intestinal crypt epithelium (RELM-beta) and white adipose tissue (Resistin), and two related human genes. RELMalpha inhibits the differentiation of 3T3-L1 preadipocytes into adipocytes but has no effect on proliferation of 3T3-L1 preadipocytes. RELMalpha is able to form heterooligomers with resistin but not RELMbeta. Since RELMalpha is expressed by adipose tissue and it is a secreted factor, our findings suggest that RELMalpha may be involved in the control of the adipogenesis as well as in the process of muscle differentiation. In the lung, RELM-alpha is induced by hypoxia and was renamed as hypoxia-induced mitogenic factor (HIMF). HIMF strongly activated Akt phosphorylation. The phosphatidylinositol 3-kinase (PI3K) inhibitor LY294002 (10 micromol/L) inhibited HIMF-activated Akt phosphorylation. It also inhibited HIMF stimulated RPSM proliferation. Thus, the PI3K/Akt pathway, at least in part, mediates the proliferative effect of HIMF. Further

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studies showed that HIMF had angiogenic and vasoconstrictive properties. HIMF increased pulmonary arterial pressure and vascular resistance. Further studies suggest that HIMF regulates apoptosis and may participate in lung alveolarization and maturation.



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