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FABP5 Human

Description: Recombinant Human Epidermal Fatty Acid Binding Protein (FABP-5) is a monodimeric, non-glycosylated, polypeptide chain containing 135 amino acids and having a total molecular mass of 15200 Daltons.

Catalog #:PRPS-424

For research use only.

Synonyms: Fatty acid-binding protein epidermal, E-FABP, Fatty acid-binding protein 5, Psoriasis-associated fatty acid-binding protein homolog, PA-FABP, FABP5, EFABP, PAFABP.

Source: Escherichia Coli.

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

Purity: Greater than 90% as determined by SDS PAGE.

Purification Method:

Two-step procedure using size exclusion chromatography before and after refolding.

Specificty:

The amino acid sequence of the recombinant human FABP5 is 100% homologous to the amino acid sequence of the human FABP-5.

Formulation:

Sterile filtered and lyophilized from 0.5 mg/ml in phosphate buffered saline.

Stability:

Store lyophilized protein at -20°C. Aliquot the product after reconstitution to avoid freeze-thaw 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:

Add 0.2 ml of dH20 and let the lyophilized pellet dissolve completely.

Introduction:

Human Fatty Epidermal Acid Binding Protein FABP also called FABP-5 is a 15 kD member of the intracellular fatty acid binding protein (FABP) family, which is known for the ability to bind fatty acids and related compounds (bile acids or retinoids). In an internal cavity. The fatty acid binding proteins aP2 (fatty acid binding protein [FABP]-4) and mal1 (EFABP) are closely related and both are expressed in adipocytes. Absence of EFABP/mal1 resulted in increased systemic insulin sensitivity in two models of obesity and insulin resistance. Adipocytes isolated from mal1-deficient mice also exhibited enhanced insulin-stimulated glucose transport capacity. In contrast, mice expressing high levels of mal1 in adipose tissue display reduced systematic insulin activity.

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