

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human IGFBP-4 in direct ELISAs and Western blots. In direct ELISAs and Western blots, no cross-reactivity with recombinant human (rh) IGFBP-1, rhIGFBP-2, or rhIGFBP-3 is observed.
Source	Monoclonal Mouse IgG _{2B} Clone # 82334
Purification	Protein A or G purified from ascites
Immunogen	Mouse myeloma cell line NS0-derived recombinant human IGFBP-4 Asp22-Glu258 Accession # AAA62670
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.

Neutralization Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Human IGF binding protein 4 (IGFBP-4) was isolated from human plasma based on its ability to bind immobilized IGF-I. Human IGFBP-4 cDNA encodes a 258 amino acid (aa) precursor protein with a predicted 21 aa signal peptide that is processed to generate the 237 aa mature human IGFBP-4. The human IGFBP-4 contains a potential N-linked glycosylation site and shares approximately 90% aa sequence identity with both the mouse and rat IGFBP-4. According to the nomenclature of IGFBPs defined at the 4th International Symposium of IGFs (1997, Tokyo), six high-affinity IGF binding proteins (IGFBP-1, -2, -3, -4, -5, -6) and four IGFBP-related proteins (IGFBPr-1, -2, -3, -4) have been identified. All IGFBPs have a high cysteine content and share conserved cysteine residues that are clustered in the amino- and carboxy-terminal third of the molecule. IGFBPs have been shown to either inhibit or enhance the biological activities of IGF, or act in an IGF-independent manner. Post-translational modification of IGFBPs, including phosphorylation and proteolysis, have been shown to modify the affinities of the binding proteins for IGF and may indirectly regulate IGF actions.

PRODUCT SPECIFIC NOTICES

This product is provided under an agreement between Life Technologies Corporation and R&D Systems, Inc, and the manufacture, use, sale or import of this product is subject to one or more US patents and corresponding non-US equivalents, owned by Life Technologies Corporation and its affiliates. The purchase of this product conveys to the buyer the non-transferable right to use the purchased amount of the product and components of the product only in research conducted by the buyer (whether the buyer is an academic or for-profit entity). The sale of this product is expressly conditioned on the buyer not using the product or its components (1) in manufacturing; (2) to provide a service, information, or data to an unaffiliated third party for payment; (3) for therapeutic, diagnostic or prophylactic purposes; (4) to resell, sell, or otherwise transfer this product or its components to any third party, or for any other commercial purpose. Life Technologies Corporation will not assert a claim against the buyer of the infringement of the above patents based on the manufacture, use or sale of a commercial product developed in research by the buyer in which this product or its components was employed, provided that neither this product nor any of its components was used in the manufacture of such product. For information on purchasing a license to this product for purposes other than research, contact Life Technologies Corporation, Cell Analysis Business Unit, Business Development, 29851 Willow Creek Road, Eugene, OR 97402, Tel: (541) 465-8300. Fax: (541) 335-0354.