abcam

Product datasheet

Anti-Insulin Affibody® Molecule ab31906

3 References 2 Images

Overview

Product name Anti-Insulin Affibody® Molecule

Tested applications Suitable for: IHC-Fr, IHC-P

Species reactivity Reacts with: Mouse, Rat, Human

Immunogen Recombinant full length protein corresponding to Insulin.

General notes ab31906 is a recombinant protein produced in E. coli.

What are Affibody Molecules?

Affibody® affinity ligands are small, simple proteins composed of a three-helix bundle based on the scaffold of one of the IgG-binding domains of Protein A. Protein A is a surface protein from the bacterium Staphylococcus aureus. This scaffold has excellent features as an affinity ligand and can be designed to bind with high affinity to any given target protein. The domain consists of 58 amino acids, 13 of which are randomized to generate Affibody[®] libraries with a large number of ligand variants. Thus, the libraries consist of a multitude of protein ligands with an identical backbone and variable surface-binding properties. The current Affibody[®] libraries contains billions of variants. In function, Affibody® molecules mimic antibodies, nature's own binders to an infinite number of antigens. Compared to antibodies, the most striking dissimilarity of Affibody[®] molecules is the small size. Affibody[®] molecules have a molecular weight of 14 kDa, compared to the molecular weight of antibodies, which is 150 kDa. In spite of its small size, the binding site of Affibody® molecules is similar to that of an antibody. The advantages of Affibody[®] molecules over antibodies are · their small size · the simple structure of the molecules \cdot its robust physical properties \cdot its ability to fold correctly intracellularly \cdot the fast and cost-efficient production in bacteria · the possibility to produce Affibody® molecules through chemical synthesis · the possibility to couple Affibody[®] molecules in multimeric

This Anti-Insulin Affibody[®] Molecule is modified with a unique C-terminal cysteine for directed single-point chemical modification, facilitating labelling with fluorescent dyes, biotin or coupling to matrices. However, tail-to-tail dimers are spontaneously generated via a disulphide bridge between the C-terminal cysteines. Prior to coupling via the C-terminal the Affibody[®] Molecule needs to be reduced to expose the reactive cysteine residue. Recommended reducing condition is 20mM DTT at a pH above 7.5 and incubation at room temperature for 2 hours. Remove excess DTT by passage through a desalting column, not by dialysis. Not yet tested in other applications. Optimal dilutions/concentrations should be determined by the end user.

THIS AFFIBODY[®] MOLECULE REQUIRES CONJUGATION TO A SUITABLE LABEL BEFORE USE. PLEASE REFER TO THE "PROTOCOLS" LINK BELOW.

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Properties

Form Liquid

Storage instructions Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C long

term.

Storage buffer pH: 7.40

Constituent: 0.079% Ammonium bicarbonate

Purification notes ab31906 is >98% pure, as determined by SDS-PAGE (Coomassie blue staining) and RP-HPLC

analysis.

Function Insulin decreases blood glucose concentration. It increases cell permeability to monosaccharides,

amino acids and fatty acids. It accelerates glycolysis, the pentose phosphate cycle, and glycogen

synthesis in liver.

Involvement in disease Defects in INS are the cause of familial hyperproinsulinemia (FHPRI) [MIM:176730].

Defects in INS are a cause of diabetes mellitus insulin-dependent type 2 (IDDM2) [MIM:125852]. IDDM2 is a multifactorial disorder of glucose homeostasis that is characterized by susceptibility to ketoacidosis in the absence of insulin therapy. Clinical fetaures are polydipsia, polyphagia and polyuria which result from hyperglycemia-induced osmotic diuresis and secondary thirst. These derangements result in long-term complications that affect the eyes, kidneys, nerves, and blood

vessels.

Defects in INS are a cause of diabetes mellitus permanent neonatal (PNDM) [MIM:606176]. PNDM is a rare form of diabetes distinct from childhood-onset autoimmune diabetes mellitus type

1. It is characterized by insulin-requiring hyperglycemia that is diagnosed within the first months of

life. Permanent neonatal diabetes requires lifelong therapy.

Defects in INS are a cause of maturity-onset diabetes of the young type 10 (MODY10)

[MIM:613370]. MODY10 is a form of diabetes that is characterized by an autosomal dominant mode of inheritance, onset in childhood or early adulthood (usually before 25 years of age), a primary defect in insulin secretion and frequent insulin-independence at the beginning of the

disease.

Sequence similarities Belongs to the insulin family.

Cellular localization Secreted.

Applications

The Abpromise guarantee

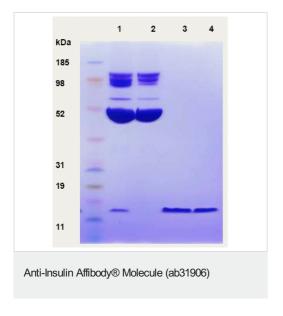
Our **Abpromise guarantee** covers the use of ab31906 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

| Application | Abreviews | Notes |
|-------------|-----------|--|
| IHC-Fr | | Use at an assay dependent concentration. |
| IHC-P | | Use at an assay dependent concentration. |
| AP | | Use at an assay dependent concentration. |

To demonstrate the binding capacity and specificity of the Anti-Insulin Affibody® molecule, 1.5 ml of five times diluted human serum spiked with insulin was injected on a column with 0.4 ml SulfoLink® Coupling gel with immobilized Anti-Insulin Affibody® molecule.

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Eluted and flow-through fractions were analyzed by an SDS-PAGE analysis:

Lane 1: human serum spiked with insulin

Lane 2: flowthrough fraction

Lane 3: eluted fraction

Lane 4: human insulin standard

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Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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