Human Insulin R/CD220 Alexa Fluor® 700-conjugated Antibody

Monoclonal Mouse IgG_{2B} Clone # 243523

Catalog Number: FAB15441N

100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Insulin R/CD220 in direct ELISAs and Western blots.
Source	Monoclonal Mouse IgG_{2B} Clone # 243523
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Insulin R/CD220 His28-Lys944 Accession # NP_001073285
Conjugate	Alexa Fluor 700 Excitation Wavelength: 675-700 nm Emission Wavelength: 723 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *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.*

	Recommended Concentration	Sample
Flow Cytometry	0.25-1 µg/10^6 cells	Human peripheral blood monocytes

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

The Insulin Receptor (INS R) and insulin-like growth factor-1 receptor (IGF-1 R) constitute a subfamily of receptor tyrosine kinases (1-4). The two receptors share structural similarity as well as overlapping intracellular signaling events, and are believed to have evolved through gene duplication from a common ancestral gene. INS R cDNA encodes a type I transmembrane single chain preproprotein with a putative 27 amino acid residues (aa) signal peptide. The large INS R extracellular domain is organized into two successive homologous globular domains, which are separated by a Cysteine-rich domain, followed by three fibronectin type III domains. The intracellular region contains the kinase domain sandwiched between the juxtamembrane domain used for docking insulin-receptor substrates (IRS), and the carboxy-terminal tail that contains two phosphotyrosine-binding sites. After synthesis, the single chain INS R precursor is glycosylated, dimerized and transported to the Golgi apparatus where it is processed at a furin-cleavage site within the middle fibronectin type III domain to generate the mature disulfide-linked $\alpha_2\beta_2$ tetrameric receptor. The α subunit is localized extracellularly and mediates ligand binding while the transmembrane β subunit contains the cytoplasmic kinase domain and mediates intracellular signaling. As a result of alternative splicing, two INS R isoforms (A and B) that differ by the absence or presence, respectively, of a 12 aa residue sequence in the carboxyl terminus of the α subunit exist. Whereas the A isoform is predominantly expressed in fetal tissues and cancer cells, the B isoform is primarily expressed in adult differentiated cells. Both the A and B isoforms bind insulin with high-affinity, but the A isoform has considerably higher affinity for IGF-I and IGF-II. Ligand binding induces a conformational change of the receptor, resulting in ATP binding, autophosphorylation, and subsequent downstream signaling. INS R signaling is important in metabolic regulation, but may also contribute to cell growth, differentiation and apoptosis. Mutations in the INS R gene have been linked to insulin-resistant diabetes mellitus, noninsulin-dependent diabetes mellitus and leprechaunism, an extremely rare disorder characterized by abnormal resistance to insulin that results in a variety of distinguishing characteristics, including growth delays and abnormalities affecting the endocrine system. INS R is highly conserved between species, rat INS R shares 94% and 97% aa sequence homology with the human and mouse receptor, respectively.

References:

1. Nakae, J. *et al.* (2001) Endoc. Rev. **22**:818.
2. De Meyts, P. and J. Whittaker (2002) Nature Rev. Drug Disc. **1**:769.
3. Kim, J.J. and D. Accili (2002) Growth Hormone and IGF Res. **12**:84.
4. Sciacca, L. *et al.* (2003) Endocrinology **144**:2650.

PRODUCT SPECIFIC NOTICES

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