

DESCRIPTION

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| Species Reactivity | Human |
| Specificity | Detects human Insulin R/CD220 in direct ELISAs. |
| Source | Monoclonal Mouse IgG _{2B} Clone # 243524 |
| 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 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 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

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| 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. <ul style="list-style-type: none"> ● 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-I 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 α₂β₂ 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.

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