DESCRIPTION

Source
Mouse myeloma cell line, NS0-derived human Insulin R/CD220 protein
His28-Arg750 (α subunit) & Ser751-Lys944 with a C-terminal 10-His tag (β subunit)
Accession # P06213-2

N-terminal Sequence Analysis
His28 (α subunit) & Ser751 (β subunit)

Structure / Form
Tetramer; disulfide-linked homodimer of disulfide-linked heterodimers (α & β)

Predicted Molecular Mass
82.9 kDa (α subunit), 22.9 kDa (β subunit)

SPECIFICATIONS

SDS-PAGE
122-135 kDa and 33-43 kDa, reducing conditions

Activity
Measured by its binding ability in a functional ELISA. When 15 ng/mL of biotinylated recombinant human Insulin is added to serially diluted Recombinant Human Insulin R/CD220, the concentration of Recombinant Human Insulin R/CD220 that produces 50% of the optimal binding response is 0.03-0.15 μg/mL.

Endotoxin Level
<0.10 EU per 1 μg of the protein by the LAL method.

Purity
>90%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation
Lyophilized from a 0.2 ml Tetramer; disulfide >90%, by SDS-PAGE filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution
Reconstitute at 100 μg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.

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

Stability & Storage
Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

The Insulin Receptor (gene name INSR, designated CD220) is a type I transmembrane glycoprotein in the Insulin/IGF Receptor family of receptor tyrosine kinases that share structural similarity and overlapping intracellular signaling events (1-3). The 1370 amino acid (aa) human Insulin R preproprotein (A isoform) is processed by proteolysis to remove the signal peptide and produce an extracellular α portion (aa 28-750), and an extracellular/transmembrane/cytoplasmic β subunit (aa 751-1370) (4). The extracellular domain (ECD) contains two homologous globular domains separated by a cysteine-rich domain and followed by three fibronectin type III domains. The intracellular region contains insulin-receptor substrate (IRS) docking sites, the kinase domain, and a phosphotyrosine-containing linker region. The human Insulin R ECD shares 96% aa sequence identity with mouse, rat, equine and canine Insulin R. As a result of alternative splicing, two INSR isoforms that differ by the presence (IR-A) or absence (IR-B) of a 12 aa residue sequence in the carboxyl terminus of the α subunit exist (4). IR-A and IR-B may homodimerize, or heterodimerize with the IGF-I receptor (1, 3, 4). All receptor combinations bind insulin, IGF-I or IGF-II, but with differing affinities; for example, IR-A has considerably higher affinity for IGF-II as compared to IR-B (2-5). This system allows fine tuning of signaling pathways according to the concentrations of insulin, IGF-I and IGF-II, and expression of receptor subunits on the cell surface (2, 3). Insulin R signaling regulates glucose uptake and metabolism, but also contributes to cell growth, differentiation and apoptosis (2, 3, 5). Mutations in the Insulin R gene have been linked severe insulin resistance (type A and Rabson-Mendenhall syndrome) that may include type II diabetes mellitus and, rarely, leprechaunism (Donohue syndrome) that also includes growth delays and endocrine system abnormalities (1, 7).

REFERENCES: