Recombinant human IR

Catalog No: #AG0056

Description



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Decomption	
Product Name	Recombinant human IR
Host Species	HEK293
Purification	> 95% by Tris-Bis PAGE;> 95% by SEC-HPLC
Immunogen Description	Ser28-Lys956
Target Name	IR
Other Names	CD 220; CD220 antigen; CD220; EC 2.7.10; EC 2.7.10.1; HHF5; INSR; Insulin R; insulin receptor; InsulinR; IR
Accession No.	Uniprot:P06213-2Gene ID:3643
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GenelD	3643
Target Species	human
Calculated MW	80 KDa
Tag Info	C-Prescission-Twin Strep II-8xHis
Formulation	0.22 µm filtered solution of PBS, pH 7.4.
Storage	Aliquot and store at -80°C. Avoid repeated freeze/thaw cycles.

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 1382 amino acid (aa) human Insulin R preproprotein (B isoform) is processed by proteolysis to remove the signal peptide and produce an extracellular alpha portion (aa 28-762), and an extracellular/transmembrane/cytoplasmic beta subunit (aa 763-1382) (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 absence (IR-A) or presence (IR-B) of a 12 aa residue sequence in the carboxyl terminus of the alpha subunit exist (4). IR-A expression is highest in fetal tissues and cancer cells, while IR-B is concentrated in adult differentiated cells (2-5). 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, 6). 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 growt

Note: This product is for in vitro research use only