Recombinant Human Insulin Receptor/INSR Protein (His & GST Tag)

Catalog No. PKSH030373

Note: Centrifuge before opening to ensure complete recovery of vial contents.

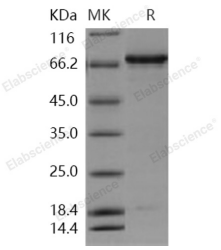
Description

Synonyms	CD220;HHF5;Insulin Receptor
Species	Human
Expression Host	Baculovirus-Insect Cells
Sequence	Gly 989-Ser 1382
Accession	NP_000199.2
Calculated Molecular Weight	72.3 kDa
Observed molecular weight	70 kDa
Tag	N-His-GST
Bioactivity	The specific activity was determined to be 45 nmol/min/mg using Poly(Ala, Glu, Lys, Tyr)6:2:5:1 as substrate.

Properties

Purity	> 92 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < - 20°C.
Formulation	Supplied as sterile 50mM Tris, 100mM NaCI, pH 7.5, 25% glycerol, 1mM TCEP,0.5mM GSH
Reconstitution	Not Applicable

Data



> 92 % as determined by reducing SDS-PAGE.

Background

INSR (Insulin receptor), also known as CD220, is a transmembrane receptor that is activated by insulin. INSR belongs to theprotein kinase superfamily, and exists as a tetramer consisting of two alpha subunits and two beta subunits linked by disulfide bonds. The alpha and beta subunits are encoded by a single INSR gene, and the beta subunits pass through the cellular membrane. As the receptor for insulin with tyrosine-protein kinase activity, INSR associates with downstream

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mediators upon binding to insulin, including IRS1 (insulin receptor substrate 1) and phosphatidylinositol 3'-kinase (PI3K). IRS-1 binding and phosphorylation eventually leads to an increase in the high affinity glucose transporter (Glut4) molecules on the outer membrane of insulin-responsive tissues. INSR isoform long and isoform short are expressed in the peripheral nerve, kidney, liver, striated muscle, fibroblasts and skin, and is found as a hybrid receptor with IGF1R which also binds IGF1 in muscle, heart, kidney, adipose tissue, skeletal muscle, hepatoma, fibrobasts, spleen and placenta. Defects in Insulin Receptor/INSR are the cause of Rabson-Mendenhall syndrome (Mendenhall syndrome), insulin resistance (Ins resistance), leprechaunism (Donohue syndrome), and familial hyperinsulinemic hypoglycemia 5 (HHF5). It may also be associated with noninsulin-dependent diabetes mellitus (NIDDM).

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