

Recombinant Protein Technical Manual

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

RPES1359

Product SKU: RPES1359 Size: 20µg

Expression host: Baculovirus-Insect Cells **Species**: Human

Uniprot: NP 000199.2

Molecular Mass: 72.3 kDa

AP Molecular Mass: 70 kDa

N-His & GST Tag:

Bio-activity: The specific activity was determined to be 45 nmol/min/mg using

Poly(Ala, Glu, Lys, Tyr)6:2:5:1 as substrate.

> 92 % as determined by reducing SDS-PAGE. **Purity:**

Endotoxin: < 1.0 EU per µg as determined by the LAL method.

Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles. Storage:

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.

Supplied as sterile 50mM Tris, 100mM NaCl, pH 7.4, 20% gly, 0.3mM DTT Formulation:

Reconstitution: Please refer to the printed manual for detailed information.

Application:

Synonyms: CD220;HHF5;Insulin Receptor

Immunogen Information:

Sequence: Gly 989-Ser 1382

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 mediators upon binding to insulin, including IRS1 (insulin receptor substrate 1) and phosphatidylinositol 3'-kinase (PI3K). IRS 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).