



Recombinant Protein Technical Manual

Recombinant Mouse KIRREL3/NEPH2 Protein (His Tag)(Active)

RPES2763

Product Data:

Product SKU: RPES2763

Size: 20µg

Species: Mouse

Expression host: HEK293 Cells

Uniprot: Q8BR86

Protein Information:

Molecular Mass: 57.5 kDa

AP Molecular Mass: 65 kDa

Tag: C-His

Bio-activity: Measured by the ability of the immobilized protein to support the adhesion of MS1 mouse pancreatic islet endothelial cells. When cells are added to mouse KIRREL3 coated plates (15 µg/mL, 100 µL/well), > 40% will adhere specifically after 90 minutes at 37

Purity: > 96 % as determined by SDS-PAGE

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

Storage: Lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.

Shipping: This product is provided as lyophilized powder which is shipped with ice packs.

Formulation: Lyophilized from sterile PBS, pH 7.4

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

Application:

Synonyms: 1500010O20Rik;2900036G11Rik;mKIAA1867;NEPH2;SST4

Immunogen Information:

Sequence: Met 1-Ala 535

Background:

Kin of IRRE-like protein 3 (KIRREL3) also known as nin of irregular chiasm-like protein 3 or nephrin-like protein 2 (NEPH2) is a member of the nephrin-like protein family of transmembrane proteins, which includes NEPH1 (KIRREL) and NEPH3 (KIRREL2). KIRREL3/NEPH2 is expressed in fetal and adult brain, and also in podocytes of kidney glomeruli. The cytoplasmic domains of KIRREL3/NEPH2 interact with the C-terminus of podocin, also expressed in the podocytes, cells involved in ensuring size- and charge-selective ultrafiltration. Mutations in KIRREL3/NEPH2 are associated with mental retardation autosomal dominant type 4. KIRREL3/NEPH2 expression is turned on in migrating neurogenesis of the pontine nucleus (PN) neurons only after they enter the presumptive nuclear region. KIRREL3/NEPH2 knockdown disrupted the nuclear organization of PN presumably by changing the migratory behavior of PN neurons inside the nuclear region. Moreover, overexpression of the cytoplasmic region of KIRREL3, which can sequester intracellular signaling of endogenous KIRREL3, resulted in similar phenotypes. Overall, these results suggest KIRREL3 is involved in the neurogenesis of the PN through the control of neuronal migration inside the nucleus.