



Recombinant Protein Technical Manual
Recombinant Human VRK1 Protein (His & GST Tag)
RPES1145

Product Data:

Product SKU: RPES1145

Size: 20µg

Species: Human

Expression host: Baculovirus-Insect Cells

Uniprot: Q99986

Protein Information:

Molecular Mass: 73 kDa

AP Molecular Mass: 65-70 kDa

Tag: N-His & GST

Bio-activity:

Purity: > 88 % as determined by reducing SDS-PAGE.

Endotoxin: < 1.0 EU per µg 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 20mM Tris, 500mM NaCl, pH 7.4, 10% gly

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

Application:

Synonyms: PCH1;PCH1A

Immunogen Information:

Sequence: Met 1-Lys 396

Background:

VRK1 is a member of the vaccinia-related kinase (VRK) family of serine/threonine protein kinases. Serine/threonine protein kinases are tumor suppressor that controls the activity of AMP-activated protein kinase family members, thereby playing a role in various processes such as cell metabolism, cell polarity, apoptosis and DNA damage response. VRK1 contains 1 protein kinase domain and localizes to the nucleus. VRK1 gene is widely expressed in human tissues and has increased expression in actively dividing cells, such as those in testis, thymus, fetal liver, and carcinomas. As a serine/threonine kinase, VRK1 phosphorylates 'Thr8' of p53/TP53 and may thereby prevent the interaction between p53/TP53 and MDM2. Defects in VRK1 are the cause of pontocerebellar hypoplasia type 1 (PCH1), also called pontocerebellar hypoplasia with infantile spinal muscular atrophy or pontocerebellar hypoplasia with anterior horn cell disease. PCH1 is characterized by an abnormally small cerebellum and brainstem, central and peripheral motor dysfunction from birth, gliosis and anterior horn cell degeneration resembling infantile spinal muscular atrophy.