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Recombinant Human PTPN11 protein (His Tag)

Catalog Number: PDEH100916

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

Description

Species Human

Source E.coli-derived Human PTPN11 protein Thr2-Ala521, with an N-terminal His

 Calculated MW
 57.1 kDa

 Observed MW
 60 kDa

 Accession
 Q06124

Bio-activity Not validated for activity

Properties

Purity > 95% as determined by reducing SDS-PAGE.

Endotoxin < 10 EU/mg of the protein as determined by the LAL method

Storage Generally, 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.

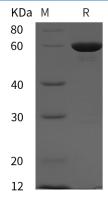
ShippingThis product is provided as lyophilized powder which is shipped with ice packs.FormulationLyophilized from a 0.2 μm filtered solution in PBS with 5% Trehalose and 5%

Mannitol.

Reconstitution It is recommended that sterile water be added to the vial to prepare a stock solution of

0.5 mg/mL. Concentration is measured by UV-Vis.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

SHP-2 (PTPN11) is a ubiquitously expressed, nonreceptor protein tyrosine phosphatase (PTP). It participates in signaling events downstream of receptors for growth factors, cytokines, hormones, antigens, and extracellular matrices in the control of cell growth, differentiation, migration, and death. Activation of SHP-2 and its association with Gab1 is critical for sustained Erk activation downstream of several growth factor receptors and cytokines. In addition to its role in Gab1-mediated Erk activation, SHP-2 attenuates EGF-dependent PI3 kinase activation by dephosphorylating Gab1 at p85 binding sites. SHP-2 becomes phosphorylated at Tyr542 and Tyr580 in its carboxy terminus in response to growth factor receptor activation. These phosphorylation events are thought to relieve basal inhibition and stimulate SHP-2 tyrosine phosphatase activity. Mutations in the corresponding gene result in a pair of clinically similar disorders (Noonan syndrome and LEOPARD syndrome) that may result from abnormal MAPK regulation.

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