Recombinant Human PTPN11 Protein (His Tag)

Catalog Number: PDEH100916

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

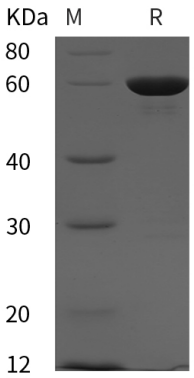
Description

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



SDS-PAGE analysis of Human PTPN11 proteins, 2 μg/lane of Recombinant Human PTPN11 proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 60 kDa.

Background

Toll-free: 1-888-852-8623
Web: www.elabscience.com
Tel: 1-832-243-6086
Email: techsupport@elabscience.com
Fax: 1-832-243-6017

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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