Recombinant Human ITCH/AIP4 Protein (aa 526-903)

Catalog Number: PKSH031249

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

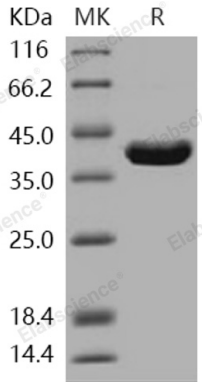
Description

Species	Human
Source	E.coli-derived Human ITCH/AIP4 protein Arg 526-Glu 903
Calculated MW	40 kDa
Observed MW	40 kDa
Accession	NP_113671.3
Bio-activity	Not validated for activity

Properties

Purity	> 98 % as determined by reducing SDS-PAGE.
Endotoxin	Please contact us for more information.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile 20mM Tris, 200mM NaCl, 10% glycerol, pH 8.0 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 98 % as determined by reducing SDS-PAGE.

Background

E3 ubiquitin-protein ligase Itchy homolog, also known as Atrophin-1-interacting protein 4, NFE2-associated polypeptide 1, NAPP1 and ITCH, is a cell membrane protein which contains oneC2 domain, oneHECT (E6AP-type E3 ubiquitin-protein ligase) domain and contains fourWW domains. ITCH acts as an E3 ubiquitin-protein ligase which accepts ubiquitin from an E2 ubiquitin-conjugating enzyme in the form of a thioester and then directly transfers the ubiquitin to targeted substrates. It catalyzes 'Lys-29'-, 'Lys-48'- and 'Lys-63'-linked ubiquitin conjugation. ITCH is involved in the control of inflammatory signaling pathways. It is an essential component of a ubiquitin-editing protein complex, comprising also TNFAIP3, TAX1BP1 and RNF11, that ensures the transient nature of inflammatory signaling pathways. ITCH promotes the association of the complex after TNF stimulation. Once the complex is formed, TNFAIP3 deubiquitinates 'Lys-63' polyubiquitin chains on RIPK1 and catalyzes the formation of 'Lys-48'-polyubiquitin chains. This leads to RIPK1 proteosomal degradation and consequently termination of the TNF- or LPS-mediated activation of NFKB1. Defects in ITCH are the cause of syndromic multisystem autoimmune disease (SMAD) which is characterized by organomegaly, failure to thrive, developmental delay, dysmorphic features and autoimmune inflammatory cell infiltration of the lungs, liver and gut.

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