Recombinant Protein Technical Manual

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

RPES3476

Product Data:

Product SKU: RPES3476

Size: 20µg

Species: Human

Expression host: E. coli

Uniprot: NP_113671.3

Protein Information:

Molecular Mass: 40 kDa

AP Molecular Mass: 40 kDa

Tag:

Bio-activity:

Purity: > 98 % as determined by reducing SDS-PAGE.

Endotoxin: Please contact us for more information.

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℃ 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

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

Application:

Synonyms: ADMFD;AIF4;AIP4;dJ468O1.1;NAPP1

Immunogen Information:

Sequence: Arg 526-Glu 903

Background:

E3 ubiquitin-protein ligase Itchy homolog, also known as Atrophin-interacting protein 4, NFE2-associated polypeptide 1, NAPP1 and ITCH, is a cell membrane protein which contains one C2 domain, one HECT (E6AP-type E3 ubiquitin-protein ligase) domain and contains four WW 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.