Anti-AIP4 antibody

Description	Rabbit polyclonal to AIP4.

Model	STJ91517
Host	Rabbit
Reactivity	Human, Mouse
Applications	ELISA, WB
Immunogen	Synthesized peptide derived from human AIP4 around the non-phosphorylation site of Y420.
Immunogen Region	360-440 aa
Gene ID	83737
Gene Symbol	ITCH
Dilution range	WB 1:500-1:2000ELISA 1:40000
Specificity	AIP4 Polyclonal Antibody detects endogenous levels of AIP4 protein.
Tissue Specificity	Widely expressed.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Note	For Research Use Only (RUO).
Protein Name	E3 ubiquitin-protein ligase Itchy homolog Itch Atrophin-1-interacting protein 4 AIP4 HECT-type E3 ubiquitin transferase Itchy homolog NFE2-associated polypeptide 1 NAPP1
Molecular Weight	103 kDa

Clonality	Polyclonal
Conjugation	Unconjugated
Isotype	IgG
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Concentration	1 mg/ml
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:13890OMIM:606409
Alternative Names	E3 ubiquitin-protein ligase Itchy homolog Itch Atrophin-1-interacting protein 4 AIP4 HECT-type E3 ubiquitin transferase Itchy homolog NFE2-associated polypeptide 1 NAPP1
Function	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. It is involved in the control of inflammatory signaling pathways. 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. 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 proteasomal degradation and consequently termination of the TNF- or LPS-mediated activation of NFKB1. Ubiquitinates RIPK2 by 'Lys-63'-linked conjugation and influences NOD2-dependent signal transduction pathways. Regulates the transcriptional activity of several transcription factors, and probably plays an important role in the regulation of immune response. Ubiquitinates NFE2 by 'Lys-63' linkages and is implicated in the control of the development of hematopoietic lineages. Critical regulator of T-helper (TH2) cytokine development through its ability to induce JUNB ubiquitination and degradation . Ubiquitinates SNX9. Ubiquitinates CXCR4 and HGS/HRS and regulates sorting of CXCR4 to the degradative pathway. It is involved in the negative regulation of MAVS-dependent cellular antiviral responses. Ubiquitinates MAVS through 'Lys-48'-linked conjugation resulting in MAVS proteasomal degradation. Ubiquitinates MAP3K7 through 'Lys-48'-linked conjugation . Involved in the regulation of apoptosis and reactive oxygen species levels through the ubiquitination and proteasomal degradation of TXNIP. Mediates the antiapoptotic activity of epidermal growth factor through the ubiquitination and proteasomal degradation of p15 BID. Targets DTX1 for lysosomal degradation and controls NOTCH1 degradation, in the absence of ligand, through 'Lys-29'-linked polyubiquitination. Ubiquitinates BRAT1 and this ubiquitination is enhanced in the presence of NDFIP1 .
Cellular Localization	Cell membrane. Cytoplasm Nucleus. Associates with endocytic vesicles. May be recruited to exosomes by NDFIP1.
Post-translational Modifications	On T-cell activation, phosphorylation by the JNK cascade on serine and threonine residues surrounding the PRR domain accelerates the ubiquitination and degradation of JUN and JUNB. The increased ITCH catalytic activity due to phosphorylation by JNK1 may occur due to a conformational change disrupting the interaction between the PRR/WW motifs domain and the HECT

domain and, thus exposing the HECT domain . Phosphorylation by FYN reduces interaction with JUNB and negatively controls JUN ubiquitination and degradation. Ubiquitinated; autopolyubiquitination with 'Lys-63' linkages which does not lead to protein degradation.