

Product datasheet

Anti-CYLD antibody ab60266

1 Image

Overview

Product name	Anti-CYLD antibody
Description	Goat polyclonal to CYLD
Host species	Goat
Tested applications	Suitable for: ELISA, WB
Species reactivity	Reacts with: Mouse, Rat Predicted to work with: Horse, Cow, Dog, Human, Chimpanzee, Orangutan 
Immunogen	Synthetic peptide: C-DSQQSKSKNPWYIDE , corresponding to internal sequence amino acids 361-375 of Mouse CYLD Run BLAST with Run BLAST with
Positive control	Brain lysate.

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
Storage buffer	Preservative: 0.02% Sodium Azide Constituents: 0.5% BSA, Tris buffered saline, pH 7.3
Purity	Immunogen affinity purified
Purification notes	Purified from goat serum by ammonium sulphate precipitation followed by antigen affinity chromatography using the immunizing peptide.
Clonality	Polyclonal
Isotype	IgG

Applications

Our [Abpromise guarantee](#) covers the use of **ab60266** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
ELISA		
WB		
Application notes	<p>Peptide ELISA: antibody detection limit dilution 1/32000.</p> <p>WB: Use at a concentration of 0.5 - 1.5 µg/ml. Detects a band of approximately 120 kDa (predicted molecular weight: 107 kDa).</p> <p>Not yet tested in other applications.</p> <p>Optimal dilutions/concentrations should be determined by the end user.</p>	
Target		
Function	<p>Protease that specifically cleaves 'Lys-63'-linked polyubiquitin chains. Has endodeubiquitinase activity. Plays an important role in the regulation of pathways leading to NF-kappa-B activation. Contributes to the regulation of cell survival, proliferation and differentiation via its effects on NF-kappa-B activation. Negative regulator of Wnt signaling. Inhibits HDAC6 and thereby promotes acetylation of alpha-tubulin and stabilization of microtubules. Plays a role in the regulation of microtubule dynamics, and thereby contributes to the regulation of cell proliferation, cell polarization, cell migration, and angiogenesis. Required for normal cell cycle progress and normal cytokinesis. Inhibits nuclear translocation of NF-kappa-B. Plays a role in the regulation of inflammation and the innate immune response, via its effects on NF-kappa-B activation. Dispensable for the maturation of intrathymic natural killer cells, but required for the continued survival of immature natural killer cells. Negatively regulates TNFRSF11A signaling and osteoclastogenesis.</p>	
Tissue specificity	<p>Detected in fetal brain, testis, and skeletal muscle, and at a lower level in adult brain, leukocytes, liver, heart, kidney, spleen, ovary and lung. Isoform 2 is found in all tissues except kidney.</p>	
Involvement in disease	<p>Defects in CYLD are the cause of familial cylindromatosis (FCYL) [MIM:132700]; also known as Ansell-Spiegler cylindromas or turban tumor syndrome or dermal eccrine cylindromatosis. CYLD is an autosomal dominant and highly tumor type-specific disorder. The tumors (known as cylindromas because of their characteristic microscopic architecture) are believed to arise from or recapitulate the appearance of the eccrine or apocrine cells of the skin that secrete sweat and scent respectively. Cylindromas arise predominantly in hairy parts of the body with approximately 90% on the head and neck. The development of a confluent mass which may ulcerate or become infected has led to the designation 'turban tumor syndrome'. The skin tumors show differentiation in the direction of hair structures, hence the synonym trichoepithelioma.</p> <p>Defects in CYLD are the cause of multiple familial trichoepithelioma type 1 (MFT1) [MIM:601606]; also known as epithelioma adenoides cysticum of Brooke (EAC) or hereditary multiple benign cystic epithelioma or Brooke-Fordyce trichoepitheliomas. MFT1 is an autosomal dominant dermatosis characterized by the presence of many skin tumors predominantly on the face. Since histologic examination shows dermal aggregates of basaloid cells with connection to or differentiation toward hair follicles, this disorder has been thought to represent a benign hamartoma of the pilosebaceous apparatus. Trichoepitheliomas can degenerate into basal cell carcinoma.</p> <p>Defects in CYLD are the cause of Brooke-Spiegler syndrome (BRSS) [MIM:605041]. BRSS is an autosomal dominant disorder characterized by the appearance of multiple skin appendage tumors such as cylindroma, trichoepithelioma, and spiradenoma. These tumors are typically located in the head and neck region, appear in early adulthood, and gradually increase in size and</p>	

number throughout life.

Sequence similarities

Belongs to the peptidase C67 family.

Contains 3 CAP-Gly domains.

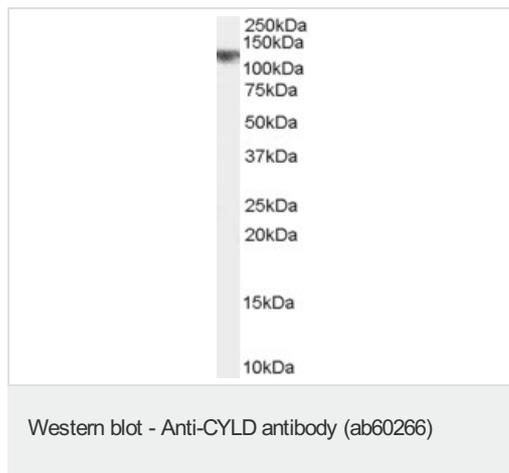
Post-translational modifications

Phosphorylated on several serine residues by IKKA and/or IKKB in response to immune stimuli. Phosphorylation requires IKKKG. Phosphorylation abolishes TRAF2 deubiquitination, interferes with the activation of Jun kinases, and strongly reduces CD40-dependent gene activation by NF-kappa-B.

Cellular localization

Cytoplasm. Cytoplasm > perinuclear region. Cytoplasm > cytoskeleton. Cell membrane. Detected at the microtubule cytoskeleton during interphase. Detected at the midbody during telophase.

Images



ab60266 antibody (0.5µg/ml) staining of CYLD in Rat Brain lysate
(35µg protein in RIPA buffer)

Predicted band size: 107 kDa

Observed band size: 120 kDa

[why is the actual band size different from the predicted?](#)

Primary incubation was 1 hour. Detected by chemiluminescence.

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