

VCP (PT1298R) PT™ Rabbit mAb

CatalogNo: YM9140 **Recombinant** 

Key Features

Host Species

- Rabbit

Reactivity

- Human, Mouse, Rat

Applications

- WB, IHC, IF, IP, ELISA

MW

- 89kD (Calculated)
- 89kD (Observed)

Isotype

- IgG, Kappa

Recommended Dilution Ratios

IHC 1:2000-1:10000

WB 1:2000-1:10000

IF 1:200-1:1000

ELISA 1:5000-1:20000

IP 1:50-1:200

Storage

Storage* -15°C to -25°C/1 year (Do not lower than -25°C)**Formulation** PBS, 50% glycerol, 0.05% Proclin 300, 0.05% BSA

Basic Information

Clonality Monoclonal**Clone Number** PT1298R

Immunogen Information

Specificity Endogenous

| Target Information

Gene name VCP

Protein Name Transitional endoplasmic reticulum ATPase

Organism	Gene ID	UniProt ID
Human	7415 ;	P55072 ;
Mouse	269523 ;	Q01853 ;
Rat	116643 ;	P46462 ;

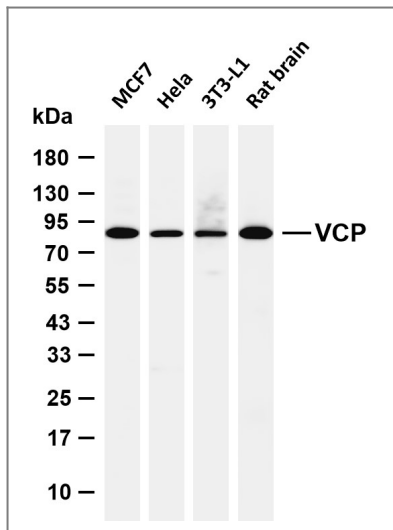
Cellular Localization Cytoplasm, cytosol . Endoplasmic reticulum . Nucleus . Cytoplasm, Stress granule . Present in the neuronal hyaline inclusion bodies specifically found in motor neurons from amyotrophic lateral sclerosis patients (PubMed:15456787). Present in the Lewy bodies specifically found in neurons from Parkinson disease patients (PubMed:15456787). Recruited to the cytoplasmic surface of the endoplasmic reticulum via interaction with AMFR/gp78 (PubMed:16168377). Following DNA double-strand breaks, recruited to the sites of damage (PubMed:22120668). Recruited to stalled replication forks via interaction with SPRTN (PubMed:23042605). Recruited to damaged lysosomes decorated with K48-linked ubiquitin chains (PubMed:27753622). Colocalizes with TIA1, ZFAND1 and G3BP1 in cytoplasmic stress granules (SGs) in response to arsenite-induced stress treatment (PubMed:29804830). .

Tissue specificity Brain,Epithelium,Fetal brain cortex,Kidney,Lymph,PCR rescued clones,Pituitary,Plate

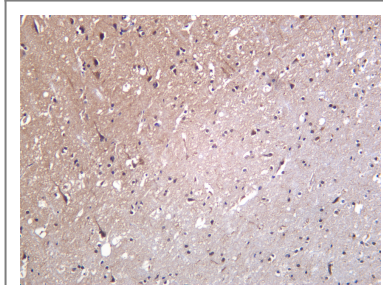
Function

Disease: Defects in VCP are the cause of inclusion body myopathy with early-onset Paget disease and frontotemporal dementia (IBMPFD) [MIM:167320]; also known as muscular dystrophy, limb-girdle, with Paget disease of bone or pagetoid amyotrophic lateral sclerosis or pagetoid neuroskeletal syndrome or lower motor neuron degeneration with Paget-like bone disease. IBMPFD features adult-onset proximal and distal muscle weakness (clinically resembling limb girdle muscular dystrophy), early-onset Paget disease of bone in most cases and premature frontotemporal dementia. Function: Necessary for the fragmentation of Golgi stacks during mitosis and for their reassembly after mitosis. Involved in the formation of the transitional endoplasmic reticulum (tER). The transfer of membranes from the endoplasmic reticulum to the Golgi apparatus occurs via 50-70 nm transition vesicles which derive from part-rough, part-smooth transitional elements of the endoplasmic reticulum (tER). Vesicle budding from the tER is an ATP-dependent process. The ternary complex containing UFD1L, VCP and NPLOC4 binds ubiquitinated proteins and is necessary for the export of misfolded proteins from the ER to the cytoplasm, where they are degraded by the proteasome. The NPLOC4-UFD1L-VCP complex regulates spindle disassembly at the end of mitosis and is necessary for the formation of a closed nuclear envelope (By similarity). Regulates E3 ubiquitin-protein ligase activity of RNF19A. PTM: Phosphorylated by tyrosine kinases in response to T-cell antigen receptor activation (By similarity). Phosphorylated upon DNA damage, probably by ATM or ATR. similarity: Belongs to the AAA ATPase family. subcellular location: Present in the neuronal hyaline inclusion bodies specifically found in motor neurons from amyotrophic lateral sclerosis patients. Present in the Lewy bodies specifically found in neurons from Parkinson disease patients. subunit: Homohexamer. Forms a ring-shaped particle of 12.5 nm diameter, that displays 6-fold radial symmetry. Part of a ternary complex containing STX5A, NSFL1C and VCP. NSFL1C forms a homotrimer that binds to one end of a VCP homohexamer. The complex binds to membranes enriched in phosphatidylethanolamine-containing lipids and promotes Golgi membrane fusion. Binds to a heterodimer of NPLOC4 and UFD1L, binding to this heterodimer inhibits Golgi-membrane fusion. Interaction with VCIP135 leads to dissociation of the complex via ATP hydrolysis by VCP. Part of a ternary complex containing NPLOC4, UFD1L and VCP. Interacts with NSFL1C-like protein p37; the complex has membrane fusion activity and is required for Golgi and endoplasmic reticulum biogenesis (By similarity). Interacts with SELS/VIMP and SYVN1, as well as with DERL1, DERL2 and DERL3; which probably transfer misfolded proteins from the ER to VCP. Interacts with SVIP. Component of a complex required to couple retrotranslocation, ubiquitination and deglycosylation composed of NGLY1, SAKS1, AMFR, VCP and RAD23B. Directly interacts with UBXD2 and RNF19A. Interacts with CASR. Interacts with UBXN6 and UBE4B.

| Validation Data



Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-VCP (PT1298R) antibody. The HRP-conjugated Goat anti-Rabbit IgG (H + L) antibody was used to detect the antibody. Lane 1: MCF7 Lane 2: HeLa Lane 3: 3T3-L1 Lane 4: Rat brain
Predicted band size: 89kDa Observed band size: 89kDa



Human brain was stained with anti-VCP (PT1298R) Rabbit antibody

Contact information

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Please scan the QR code to access additional product information:
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Rabbit mAb

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