

**VCP Antibody**  
**Rabbit Polyclonal Antibody**  
**Catalog # ABV10575****Specification**

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**VCP Antibody - Product Information**

Application	WB, IP
Primary Accession	<a href="#">P55072</a>
Other Accession	<a href="#">NP_009057.1</a>
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	89322

**VCP Antibody - Additional Information****Gene ID 7415**

Application & Usage	<b>Western blotting (1:500 - 1:2000) and immunoprecipitation. However, the optimal concentrations should be determined individually. The antibody recognizes the 97 kDa VCP from samples of human and mouse origins. HeLa and NIH3T3 cell lysates can be used as positive controls. Reactivity to other species has not been tested.</b>
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**Other Names**

VCP, Valosin-Containing Protein, TERA, Transitional Endoplasmic Reticulum ATPase, p97, IBMPFD

**Target/Specificity**

VCP

**Antibody Form**

Liquid

**Appearance**

Colorless liquid

**Formulation**

100 µl affinity purified rabbit polyclonal antibody in phosphate-buffered saline (PBS) containing 30% glycerol, 1% BSA, and 0.02% thimerosal.

**Handling**

The antibody solution should be gently mixed before use.

**Reconstitution & Storage**

-20 °C

## Background Descriptions

### Precautions

VCP Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

## VCP Antibody - Protein Information

### Name VCP

### 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 UFD1, 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- UFD1-VCP complex regulates spindle disassembly at the end of mitosis and is necessary for the formation of a closed nuclear envelope. Regulates E3 ubiquitin-protein ligase activity of RNF19A. Component of the VCP/p97-AMFR/gp78 complex that participates in the final step of the sterol-mediated ubiquitination and endoplasmic reticulum-associated degradation (ERAD) of HMGCR. Mediates the endoplasmic reticulum- associated degradation of CHRNA3 in cortical neurons as part of the STUB1-VCP-UBXN2A complex (PubMed:<a href="http://www.uniprot.org/citations/26265139" target="\_blank">26265139</a>). Involved in endoplasmic reticulum stress-induced pre-emptive quality control, a mechanism that selectively attenuates the translocation of newly synthesized proteins into the endoplasmic reticulum and reroutes them to the cytosol for proteasomal degradation (PubMed:<a href="http://www.uniprot.org/citations/26565908" target="\_blank">26565908</a>). Involved in clearance process by mediating G3BP1 extraction from stress granules (PubMed:<a href="http://www.uniprot.org/citations/29804830" target="\_blank">29804830</a>, PubMed:<a href="http://www.uniprot.org/citations/34739333" target="\_blank">34739333</a>). Also involved in DNA damage response: recruited to double-strand breaks (DSBs) sites in a RNF8- and RNF168-dependent manner and promotes the recruitment of TP53BP1 at DNA damage sites (PubMed:<a href="http://www.uniprot.org/citations/22020440" target="\_blank">22020440</a>, PubMed:<a href="http://www.uniprot.org/citations/22120668" target="\_blank">22120668</a>). Recruited to stalled replication forks by SPRTN: may act by mediating extraction of DNA polymerase eta (POLH) to prevent excessive translesion DNA synthesis and limit the incidence of mutations induced by DNA damage (PubMed:<a href="http://www.uniprot.org/citations/23042605" target="\_blank">23042605</a>, PubMed:<a href="http://www.uniprot.org/citations/23042607" target="\_blank">23042607</a>). Together with SPRTN metalloprotease, involved in the repair of covalent DNA-protein cross- links (DPCs) during DNA synthesis (PubMed:<a href="http://www.uniprot.org/citations/32152270" target="\_blank">32152270</a>). Involved in interstrand cross-link repair in response to replication stress by mediating unloading of the ubiquitinated CMG helicase complex (By similarity). Mediates extraction of PARP1 trapped to chromatin: recognizes and binds ubiquitinated PARP1 and promotes its removal (PubMed:<a href="http://www.uniprot.org/citations/35013556" target="\_blank">35013556</a>). Required for cytoplasmic retrotranslocation of stressed/damaged mitochondrial outer-membrane proteins and their subsequent proteasomal degradation (PubMed:<a href="http://www.uniprot.org/citations/16186510" target="\_blank">16186510</a>, PubMed:<a href="http://www.uniprot.org/citations/21118995" target="\_blank">21118995</a>). Essential for the maturation of ubiquitin-containing autophagosomes and the clearance of ubiquitinated protein by autophagy (PubMed:<a href="http://www.uniprot.org/citations/20104022" target="\_blank">20104022</a>, PubMed:<a href="http://www.uniprot.org/citations/27753622" target="\_blank">27753622</a>). Acts as a negative regulator of type I interferon production by

interacting with RIGI: interaction takes place when RIGI is ubiquitinated via 'Lys-63'-linked ubiquitin on its CARD domains, leading to recruit RNF125 and promote ubiquitination and degradation of RIGI (PubMed:<a href="http://www.uniprot.org/citations/26471729" target="\_blank">26471729</a>). May play a role in the ubiquitin-dependent sorting of membrane proteins to lysosomes where they undergo degradation (PubMed:<a href="http://www.uniprot.org/citations/21822278" target="\_blank">21822278</a>). May more particularly play a role in caveolins sorting in cells (PubMed:<a href="http://www.uniprot.org/citations/21822278" target="\_blank">21822278</a>, PubMed:<a href="http://www.uniprot.org/citations/23335559" target="\_blank">23335559</a>). By controlling the steady-state expression of the IGF1R receptor, indirectly regulates the insulin-like growth factor receptor signaling pathway (PubMed:<a href="http://www.uniprot.org/citations/26692333" target="\_blank">26692333</a>).

### Cellular Location

Cytoplasm, cytosol. Endoplasmic reticulum. Nucleus. Cytoplasm, Stress granule. Note=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).

### VCP Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### VCP Antibody - Images

### VCP Antibody - Background

Valosin containing protein (VCP), also designated TERA (for transitional endoplasmic reticulum ATPase) or p97, is a member of the AAA family of ATPases, which are involved in a variety of cellular activities. VCP is the mammalian homolog of *Saccharomyces cerevisiae* Cdc48, a protein essential for the completion of mitosis in yeast. VCP is thought to be involved in a variety of membrane functions and in the regulation of the cell cycle. It associates with ubiquitinated I $\kappa$ B- $\alpha$ s as well as with the 26S Proteasome, indicating a potential role for VCP in the proteasome-mediated degradation of I $\kappa$ B- $\alpha$ .