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KEAP1 Antibody (C-Terminus)
Rabbit Polyclonal Antibody
Catalog # ALS15665

Specification

KEAP1 Antibody (C-Terminus) - Product Information

Application	**IHC, IF, WB**
Primary Accession	Q14145
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**70kDa KDa**

KEAP1 Antibody (C-Terminus) - Additional Information

Gene ID 9817

Other Names
Kelch-like ECH-associated protein 1, Cytosolic inhibitor of Nrf2, INrf2, Kelch-like protein 19, KEAP1, INRF2, KIAA0132, KLHL19

Target/Specificity
Human KEAP1. At least two isoforms of KEAP1 are known to exist.

Reconstitution & Storage
Long term: -20°C; Short term: +4°C. Avoid repeat freeze-thaw cycles.

Precautions
KEAP1 Antibody (C-Terminus) is for research use only and not for use in diagnostic or therapeutic procedures.

KEAP1 Antibody (C-Terminus) - Protein Information

Name KEAP1 {ECO:0000303|PubMed:14585973, ECO:0000312|HGNC:HGNC:23177}

Function
Substrate-specific adapter of a BCR (BTB-CUL3-RBX1) E3 ubiquitin ligase complex that regulates the response to oxidative stress by targeting NFE2L2/NRF2 for ubiquitination (PubMed:14585973, PubMed:15379550, PubMed:15572695, PubMed:15983046, PubMed:15601839). KEAP1 acts as a key sensor of oxidative and electrophilic stress: in normal conditions, the BCR(KEAP1) complex mediates ubiquitination and degradation of NFE2L2/NRF2, a transcription factor regulating expression of many cytoprotective genes (PubMed:15601839, PubMed:16006525). In response

to oxidative stress, different electrophile metabolites trigger non-enzymatic covalent modifications of highly reactive cysteine residues in KEAP1, leading to inactivate the ubiquitin ligase activity of the BCR(KEAP1) complex, promoting NFE2L2/NRF2 nuclear accumulation and expression of phase II detoxifying enzymes (PubMed:19489739, PubMed:16006525, PubMed:17127771, PubMed:18251510, PubMed:29590092). In response to selective autophagy, KEAP1 is sequestered in inclusion bodies following its interaction with SQSTM1/p62, leading to inactivation of the BCR(KEAP1) complex and activation of NFE2L2/NRF2 (PubMed:20452972). The BCR(KEAP1) complex also mediates ubiquitination of SQSTM1/p62, increasing SQSTM1/p62 sequestering activity and degradation (PubMed:28380357). The BCR(KEAP1) complex also targets BPTF and PGAM5 for ubiquitination and degradation by the proteasome (PubMed:15379550, PubMed:17046835).

Cellular Location
Cytoplasm. Nucleus. Note=Mainly cytoplasmic (PubMed:15601839). In response to selective autophagy, relocalizes to inclusion bodies following interaction with SQSTM1/p62 (PubMed:20452972).

Tissue Location
Broadly expressed, with highest levels in skeletal muscle.

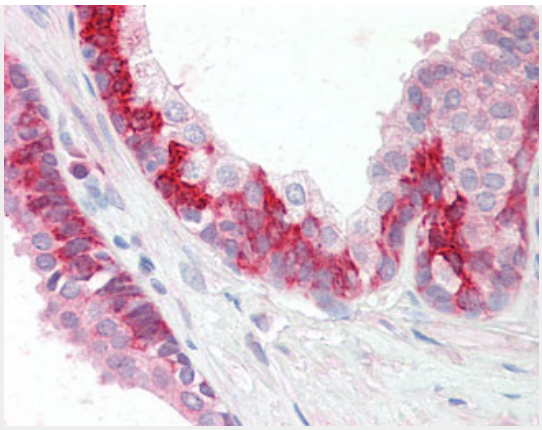
KEAP1 Antibody (C-Terminus) - Protocols

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

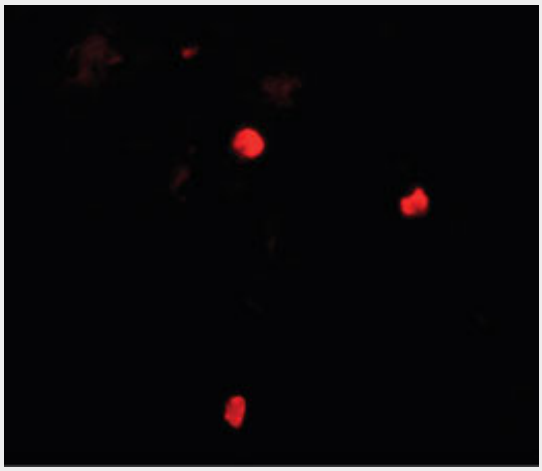
- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

KEAP1 Antibody (C-Terminus) - Images

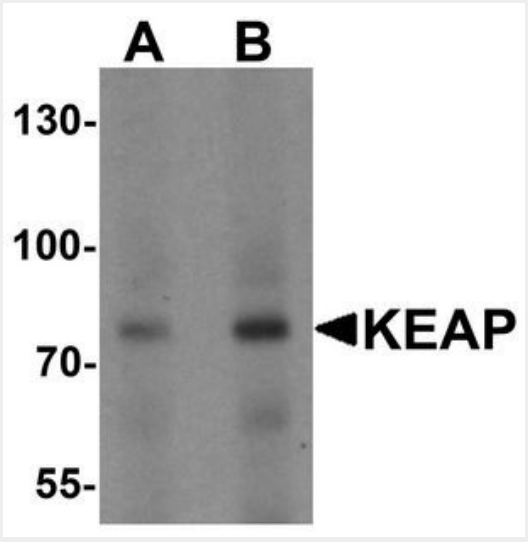
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Anti-KEAP1 antibody IHC staining of human prostate.



Immunofluorescence of KEAP1 in human lung tissue with KEAP1 antibody at 20 ug/ml.



Western blot analysis of KEAP1 in human lung tissue lysate with KEAP1 antibody at (A) 1 and (B)...

KEAP1 Antibody (C-Terminus) - Background

Acts as a substrate adapter protein for the E3 ubiquitin ligase complex formed by CUL3 and RBX1 and targets NFE2L2/NRF2 for ubiquitination and degradation by the proteasome, thus resulting in the suppression of its transcriptional activity and the repression of antioxidant response

element-mediated detoxifying enzyme gene expression. Retains NFE2L2/NRF2 and may also retain BPTF in the cytosol. Targets PGAM5 for ubiquitination and degradation by the proteasome.

KEAP1 Antibody (C-Terminus) - References

Dhakshinamoorthy S.,et al.Submitted (MAR-2001) to the EMBL/GenBank/DDBJ databases.
Nagase T.,et al.DNA Res. 2:167-174(1995).
Ohara O.,et al.Submitted (DEC-2008) to the EMBL/GenBank/DDBJ databases.
Ota T.,et al.Nat. Genet. 36:40-45(2004).
Grimwood J.,et al.Nature 428:529-535(2004).