

KEAP1 Antibody (N-Terminus)
Goat Polyclonal Antibody
Catalog # ALS15404**Specification**

KEAP1 Antibody (N-Terminus) - Product Information

Application	WB, IHC
Primary Accession	Q14145
Reactivity	Human, Monkey, Pig, Horse, Bovine, Dog
Host	Goat
Clonality	Polyclonal
Calculated MW	70kDa KDa

KEAP1 Antibody (N-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. Reported variants represent identical protein: NP_036421.2, NP_987096.1

Reconstitution & Storage

Store at -20°C. Minimize freezing and thawing.

Precautions

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

KEAP1 Antibody (N-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: 15601839, PubMed: 15983046, PubMed: 37339955). 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: 15601839, PubMed: 15601839).

<http://www.uniprot.org/citations/16006525> target="_blank">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:16006525, PubMed:17127771, PubMed:18251510, PubMed:19489739, 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.

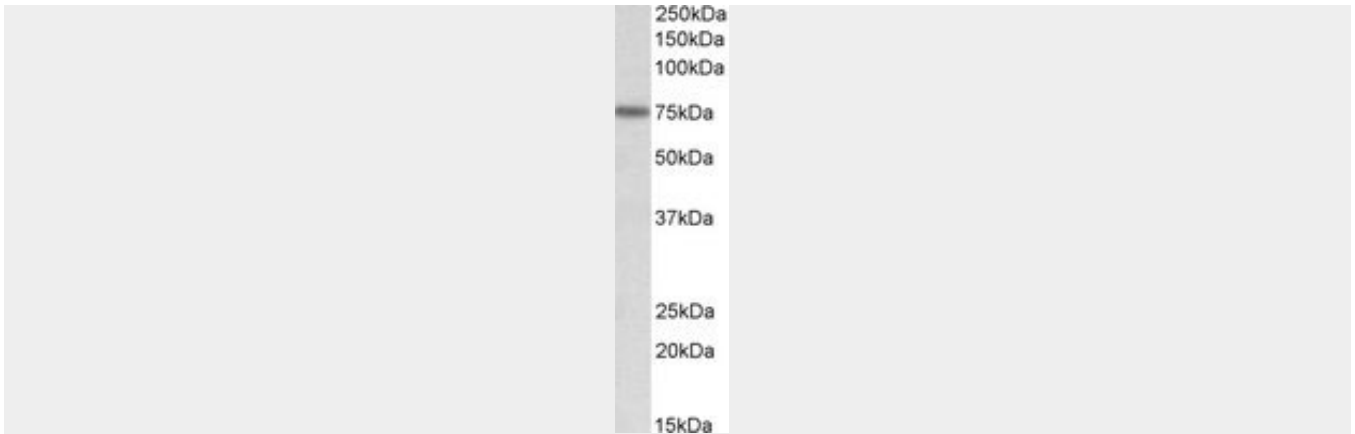
KEAP1 Antibody (N-Terminus) - Protocols

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

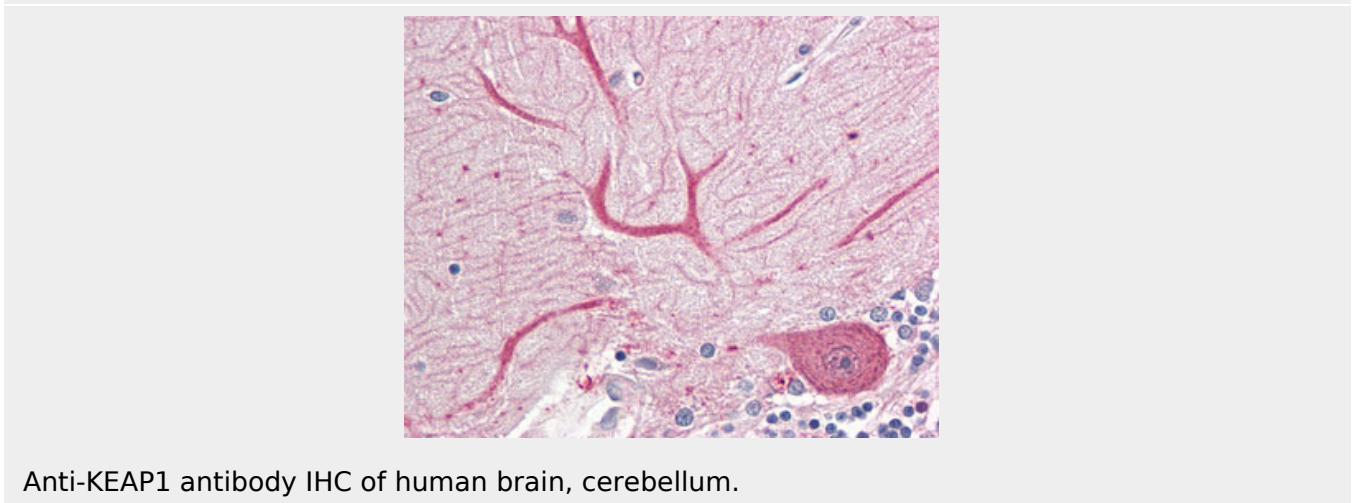
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

KEAP1 Antibody (N-Terminus) - Images





KEAP1 antibody (0.2 ug/ml) staining of NIH3T3 lysate (35 ug protein/ml in RIPA buffer).



Anti-KEAP1 antibody IHC of human brain, cerebellum.

KEAP1 Antibody (N-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 (N-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).