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NFE2L1 Antibody (C-term) Blocking peptide
Synthetic peptide
Catalog # BP10927b

Specification

NFE2L1 Antibody (C-term) Blocking peptide - Product Information

Primary Accession Q14494

NFE2L1 Antibody (C-term) Blocking peptide - Additional Information

Gene ID 4779

Other Names
Nuclear factor erythroid 2-related factor 1, NF-E2-related factor 1, NFE2-related factor 1, Locus control region-factor 1, Nuclear factor, erythroid derived 2, like 1, Transcription factor 11, TCF-11, Transcription factor HBZ17, Transcription factor LCR-F1, NFE2L1, HBZ17, NRF1, TCF11

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

NFE2L1 Antibody (C-term) Blocking peptide - Protein Information

Name NFE2L1

Function
[Endoplasmic reticulum membrane sensor NFE2L1]: Endoplasmic reticulum membrane sensor that translocates into the nucleus in response to various stresses to act as a transcription factor (PubMed:20932482, PubMed:24448410). Constitutes a precursor of the transcription factor NRF1 (By similarity). Able to detect various cellular stresses, such as cholesterol excess, oxidative stress or proteasome inhibition (PubMed:20932482). In response to stress, it is released from the endoplasmic reticulum membrane following cleavage by the protease DDI2 and translocates into the nucleus to form the transcription factor NRF1 (By similarity). Acts as a key sensor of cholesterol excess: in excess cholesterol conditions, the endoplasmic reticulum membrane form of the protein directly binds cholesterol via its CRAC motif, preventing cleavage and release of the transcription factor NRF1, thereby allowing expression of genes promoting cholesterol removal, such as CD36 (By similarity). Involved in proteasome homeostasis: in response to proteasome inhibition, it is released from the endoplasmic reticulum membrane, translocates to the nucleus and activates expression of genes encoding proteasome subunits (PubMed:<a href="http://www.uniprot.org/citations/20932482"

target="_blank">20932482).

Cellular Location
[Endoplasmic reticulum membrane sensor NFE2L1]: Endoplasmic reticulum membrane; Single-pass type II membrane protein. Endoplasmic reticulum membrane; Single-pass type III membrane protein. Note=In normal conditions, probably has a single-pass type II membrane protein topology, with the DNA-binding domain facing the endoplasmic reticulum lumen (PubMed:24448410). Following cellular stress, it is rapidly and efficiently retrotranslocated to the cytosolic side of the membrane, a process dependent on p97/VCP, to have a single-pass type III membrane protein topology with the major part of the protein facing the cytosol (PubMed:24448410). Retrotranslocated proteins are normally rapidly degraded by the proteasome and active species do not accumulate (PubMed:24448410). However, retrotranslocated protein NFE2L1 escapes degradation and is cleaved at Leu-104 by DDI2, releasing the protein from the endoplasmic reticulum membrane and forming the transcription factor NRF1 that translocates into the nucleus (PubMed:24448410)

NFE2L1 Antibody (C-term) Blocking peptide - Protocols

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

- Blocking Peptides

NFE2L1 Antibody (C-term) Blocking peptide - Images

NFE2L1 Antibody (C-term) Blocking peptide - Background

This gene encodes a protein that is involved in globingene expression in erythrocytes. Confusion has occurred inbibliographic databases due to the shared symbol of NRF1 for thisgene, NFE2L1, and for 'nuclear respiratory factor 1' which has anofficial symbol of NRF1.

NFE2L1 Antibody (C-term) Blocking peptide - References

Steffen, J., et al. Mol. Cell 40(1):147-158(2010)Radhakrishnan, S.K., et al. Mol. Cell 38(1):17-28(2010)Wang, W., et al. J. Biol. Chem. 282(34):24670-24678(2007)Newman, J.R., et al. Science 300(5628):2097-2101(2003)Toki, T., et al. Oncogene 14(16):1901-1910(1997)