

KEAP1

Reactivity:Human Mouse Rat

Tested applications:WB IHC IF

Recommended Dilution:WB 1:500 - 1:2000 IHC 1:50 - 1:200 IF 1:50 - 1:200

Calculated MW:70kDa

Observed MW:Refer to Figures

Immunogen:

Recombinant protein of human KEAP1

Storage Buffer:

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

Synonym:

INrf2; KIAA0132; KLHL19; MGC10630; MGC11114; MGC20887; MGC4407; MGC9454

Catalog #:A1820

Antibody Type:

Polyclonal Antibody

Species:Rabbit

Gene ID:9817

Isotype:IgG

Swiss Prot:Q14145

Purity:Affinity purification

For research use only.

Background:

The nuclear factor-like 2 (NRF2) transcriptional activator binds antioxidant response elements (ARE) of target gene promoter regions to regulate expression of oxidative stress response genes. Under basal conditions, the NRF2 inhibitor INrf2 (also called KEAP1) binds and retains NRF2 in the cytoplasm where it can be targeted for ubiquitin-mediated degradation (1). Small amounts of constitutive nuclear NRF2 maintains cellular homeostasis through regulation of basal expression of antioxidant response genes. Following oxidative or electrophilic stress, KEAP1 releases NRF2, thereby allowing the activator to translocate to the nucleus and bind to ARE-containing genes (2). The coordinated action of NRF2 and other transcription factors mediates the response to oxidative stress (3). Altered expression of NRF2 is associated with chronic obstructive pulmonary disease (COPD) (4). NRF2 activity in lung cancer cell lines directly correlates with cell proliferation rates, and inhibition of NRF2 expression by siRNA enhances anti-cancer drug-induced apoptosis (5). The NRF2 repressor KEAP1 contains an amino terminal BTB/POZ domain and a carboxyl terminal KELCH domain (6,7). The KELCH domain is required for interacting with NRF2 and the BTB/POZ domain functions in binding Cul3 E3 ubiquitin ligase (8-10). Under normal conditions, the complex leads to the cytoplasmic sequestration and ubiquitin-mediated proteasomal degradation of NRF2. Electrophilic modification of KEAP1 leads to disassociation of the NRF2/KEAP1 complex. KEAP1 also targets the down regulation of NF-B activity by targeting IKK degradation (11).

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