KEAP1 Protein, Human (P.pastoris, His)

Cat. No.:	HY-P701249
Synonyms:	Kelch-like ECH-associated protein 1; Cytosolic inhibitor of Nrf2; KEAP1; KEAP1
Species:	Human
Source:	P. pastoris
Accession:	Q14145 (M1-C624)
Gene ID:	9817
Molecular Weight:	71.7kDa

PROPERTIES

Appearance	Lyophilized powder
Formulation	Lyophilized from a 0.22 μm filtered solution of 20 mM Tris-HCl, 0.5 M NaCl, 6% Trehalose, pH 8.0.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	KEAP1, as the substrate-specific adapter within the BCR (BTB-CUL3-RBX1) E3 ubiquitin ligase complex, intricately regulates the cellular response to oxidative stress by orchestrating the ubiquitination of NFE2L2/NRF2. Serving as a crucial sensor for oxidative and electrophilic stress, KEAP1, under normal conditions, facilitates the ubiquitination and subsequent degradation of NFE2L2/NRF2, a transcription factor essential for the expression of numerous cytoprotective genes. When confronted with oxidative stress, distinct electrophile metabolites induce non-enzymatic covalent modifications on highly reactive cysteine residues in KEAP1, effectively dampening the ubiquitin ligase activity of the BCR(KEAP1) complex. This disruption promotes the nuclear accumulation of NFE2L2/NRF2 and triggers the expression of phase II detoxifying enzymes. Furthermore, selective autophagy leads to the sequestration of KEAP1 in inclusion bodies through its interaction with SQSTM1/p62, resulting in the inactivation of the BCR(KEAP1) complex and the activation of NFE2L2/NRF2. Notably, the BCR(KEAP1) complex extends its ubiquitin ligase activity to substrates like SQSTM1/p62, BPTF, and PGAM5, modulating their degradation via the proteasome. The ubiquitin ligase activity of the BCR(KEAP1) complex faces inhibition in response to oxidative stress and electrophile metabolites such as sulforaphane, as these metabolites react with reactive cysteine residues in KEAP1, leading to the non-enzymatic covalent modifications that incapacitate the complex. Moreover, selective autophagy contributes to the inactivation of the BCR(KEAP1) complex through the interaction between KEAP1 and SQSTM1/p62, promoting the sequestration of the complex in inclusion bodies and facilitating its degradation.

Caution: Product has not been fully validated for medical applications. For research use only.

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