KEAP1 Protein, Human (sf9, His-GST)

Cat. No.:	HY-P75899
Synonyms:	Kelch-like ECH-associated protein 1; Cytosolic inhibitor of Nrf2; KEAP1; KEAP1
Species:	Human
Source:	Sf9 insect cells
Accession:	Q14145 (Q2-C624)
Gene ID:	9817
Molecular Weight:	Approximately 109 kDa

PROPERTIES

Appearance	Solution.
Formulation	Supplied as a 0.2 μm filtered solution of 20 mM Tris, 500 mM NaCl, 10% Glycerol, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	N/A
Storage & Stability	Stored at -80°C for 1 year. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles.
Shipping	Shipping with dry ice

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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