DAPK1 Protein, Human (sf9, His-GST)

Cat. No.:	HY-P76858
Synonyms:	Death-associated protein kinase 1; DAP kinase 1; DAPK
Species:	Human
Source:	Sf9 insect cells
Accession:	P53355-1 (M1-L363)
Gene ID:	1612
Molecular Weight:	Approximately 64 kDa

PROPERTIES

Biological Activity	The specific activity was determined to be 20 nmol/min/mg using synthetic R11-S6-Peptide as substrate.
Appearance	Solution.
Formulation	Supplied as a 0.2 μm filtered solution of 20 mM Tris, 500 mM NaCl, pH 8.0, 10% gly
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	DAPK1 protein, a calcium/calmodulin-dependent serine/threonine kinase, intricately participates in multiple cellular signaling pathways, regulating cell survival, apoptosis, and autophagy. Its versatile role includes orchestrating both type I apoptotic and type II autophagic cell death signals, the former being caspase-dependent and the latter characterized by the accumulation of autophagic vesicles and being caspase-independent. DAPK1 engages in phosphorylation events with diverse substrates: it inhibits the catalytic activity, nuclear localization, and cellular function of PIN1, enhances stress fiber formation in endothelial cells by phosphorylating TPM1, and significantly decreases the binding of STX1A to STXBP1. Additionally, it phosphorylates PRKD1 to regulate JNK signaling under oxidative stress and modulates autophagy by phosphorylating BECN1, disrupting its interaction with BCL2 and BCL2L1. DAPK1 also phosphorylates TSC2, disrupting the TSC1-TSC2 complex and stimulating mTORC1 activity in a growth factor-dependent pathway. In the context of extrasynaptic NMDA receptors, DAPK1 acts as a signaling amplifier, mediating brain damage in stroke by inducing injurious Ca(2+) influx through NMDA receptor channels. Furthermore, DAPK1 collaborates with DAPK3 to phosphorylate RPL13A upon interferon-gamma activation, leading to transcript-selective translation inhibition. Notably, isoform 2 of DAPK1 lacks the ability to induce apoptosis but retains the capacity to induce membrane blebbing.

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

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