

## **Product** Data Sheet

## MST1/STK4 Protein, Human (sf9, His)

Cat. No.: HY-P74742

Synonyms: Serine/threonine-protein kinase 4; MST1/N; MST1/C; STK4; KRS2

Species:

Sf9 insect cells Source: Accession: Q13043 (E2-F487)

Gene ID: 6789

**Molecular Weight:** Approximately 58 kDa

PROPERTIES	
Biological Activity	The specific activity was determined to be >150 nmol/min/mg using MBP as substrate.
Appearance	Solution.
Formulation	Supplied as a 0.2 μm filtered solution of 20 mM Tris, 500 mM NaCl, pH 7.4, 10% glycerol.
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

MST1/STK4 protein is a stress-activated, pro-apoptotic kinase with diverse cellular functions, including a critical role in the Hippo signaling pathway. Following caspase-cleavage, MST1/STK4 enters the nucleus, inducing chromatin condensation and internucleosomal DNA fragmentation, contributing to apoptosis. In the Hippo pathway, MST1/STK4, along with its regulatory protein SAV1, forms a kinase cascade that phosphorylates and activates LATS1/2-MOB1 complex. This, in turn, phosphorylates and inactivates the YAP1 oncoprotein and WWTR1/TAZ, regulating genes involved in proliferation, cell death, and cell migration. MST1/STK4 is essential for repressing hepatocyte proliferation, inhibiting facultative adult liver stem cells, and preventing tumor formation. Additionally, it plays a role in histone modification during apoptosis, phosphorylates various substrates like FOXO3, MOBKL1A, MOBKL1B, RASSF2, TNNI3, and FOXO1, influencing diverse cellular processes such as transcription, cell death initiation, and signaling pathways like PKB/AKT1 and AR. Notably, MST1/STK4 acts as an inhibitor of PKB/AKT1, and its phosphorylation of SIRT1 promotes p53-dependent transcription and apoptosis in response to DNA damage. Furthermore, it intersects with PKB/AKT1 signaling to suppress AR activity by antagonizing the formation of AR-chromatin complexes.

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 $\label{lem:caution:Product} \textbf{Caution: Product has not been fully validated for medical applications. For research use only.}$ 

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