

ERK2 Protein, Human, Recombinant (GST)

General Information

Synonyms:	ERK-2;PRKM2;Mitogen-activated protein kinase 1;MAP kinase 2;ERK2;ERT1;MAPK1;MAPK 1;MAPK 2;MAP kinase 1;PRKM1;p42-MAPK
Protein Construction:	A DNA sequence encoding the human ERK2 (NP_002736.3) (Met 1-Ser 360) was fused with the GST tag at the N-terminus. Predicted N terminal: Met
Species:	Human
Expression Host:	Baculovirus Insect Cells
Accession:	P28482-1
Molecular Weight:	67 kDa (predicted); 67 kDa (reducing conditions)

QC Testing

Biological Activity:	No Kinase Activity
Purity:	> 98 % as determined by SDS-PAGE
Endotoxin:	< 1.0 EU/µg of the protein as determined by the LAL method.
Formulation:	Lyophilized from a solution filtered through a 0.22 µm filter, containing 50 mM Tris, 100 mM NaCl, 0.5 mM PMSF, 10% Glycerol, pH 8.0. Typically, a mixture containing 5% to 8% trehalose, mannitol, and 0.01% Tween 80 is incorporated as a protective agent before lyophilization.

Preparation and Storage

Reconstitution:	Reconstituted with sterile deionized water to 0.25 mg/mL. Reconstitution conditions may vary depending on the lot.
Stability & Storage:	It is recommended to store recombinant proteins at -20°C to -80°C for future use. Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots. <small>Actual storage temperature shall be subject to the COA.</small>
Shipping:	In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry ice.

Protein Background

MAP kinases, also known as extracellular signal-regulated kinases (ERKs), act as an integration point for multiple biochemical signals and are involved in a wide variety of cellular processes such as proliferation, differentiation, transcription regulation, and development. ERK is a versatile protein kinase that regulates many cellular functions. Growing evidence suggests that extracellular signal-regulated protein kinase 1/2 (ERK1/2) plays a crucial role in promoting cell death in a variety of neuronal systems, including neurodegenerative diseases. It is believed that

the magnitude and the duration of ERK1/2 activity determine its cellular function. Activation of ERK1/2 is implicated in the pathophysiology of spinal cord injury (SCI). ERK2 signaling is a novel target associated with the deleterious consequences of spinal injury. ERK-2, also known as mitogen-activated protein kinase 1 (MAPK1), is a member of the protein kinase superfamily and MAP kinase subfamily. MKP-3 is a dual-specificity phosphatase exclusively specific to MAPK1 for its substrate recognition and dephosphorylating activity. The activation of MAPK1 requires its phosphorylation by upstream kinases. Upon activation, MAPK1 translocates to the nucleus of the stimulated cells, where it phosphorylates nuclear targets. MAPK1 is involved in both the initiation and regulation of meiosis, mitosis, and postmitotic functions in differentiated cells by phosphorylating some transcription factors such as ELK1. MAPK1 acts as a transcriptional repressor that represses the expression of interferon gamma-induced genes. Transcriptional activity is independent of kinase activity. The nuclear-cytoplasmic distribution of ERK2 is regulated in response to various stimuli and changes in a cell context. Furthermore, the nuclear flux of ERK2 occurs by several energy- and carrier-dependent and -independent mechanisms. ERK2 has been shown to translocate into and out of the nucleus by facilitated diffusion through the nuclear pore, interacting directly with proteins within the nuclear pore complex, as well as by karyopherin-mediated transport. ERK2 interacts with the PDE4 catalytic unit by binding to a KIM (kinase interaction motif) docking site located on an exposed beta-hairpin loop and an FQF (Phe-Gln-Phe) specificity site located on an exposed alpha-helix. These flank a site that allows phosphorylation by ERK, the functional outcome of which is orchestrated by the N-terminal UCR1/2 (upstream conserved region 1 and 2) modules. Cancer Immunotherapy Immune Checkpoint Immunotherapy Targeted Therapy

Reference

- Houslay MD, et al. (2003) The role of ERK2 docking and phosphorylation of PDE4 cAMP phosphodiesterase isoforms in mediating cross-talk between the cAMP and ERK signalling pathways. *Biochem Soc Trans.* 31(Pt 6): 1186-90.
- Jivan A, et al. (2010) Reconstitution of the Nuclear Transport of the MAP Kinase ERK2. *Methods Mol Biol.* 661: 273-85.
- Yu CG, et al. (2010) Involvement of ERK2 in traumatic spinal cord injury. *J Neurochem.* 113(1): 131-42.
- Subramaniam S, et al. (2010) ERK and cell death: ERK1/2 in neuronal death. *FEBS J.* 277(1): 22-9.

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