Human ERK2 / MAPK1 / MAPK2 Protein (GST Tag)

Catalog Number: **10030-H09B**

Sino Biological
Biological Solution Specialist

General Information

Gene Name Synonym:

ERK; ERK-2; ERK2; ERT1; MAPK2; p38; p40; p41; p41mapk; p42-MAPK; P42MAPK; PRKM1; PRKM2

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.

Source: Human

Expression Host: Baculovirus-Insect Cells

QC Testing

Purity: > 98 % as determined by SDS-PAGE

Bio Activity:

No Kinase Activity

Endotoxin:

< 1.0 EU per μg of the protein as determined by the LAL method

Stability:

Samples are stable for up to twelve months from date of receipt at -70 ℃

Predicted N terminal: Met

Molecular Mass:

The recombinant human ERK2/GST chimera consists of 585 amino acids and predicts a molecular mass of 67 kDa as estimated in SDS-PAGE under reducing conditions.

Formulation:

Lyophilized from sterile 50mM Tris, 100mM NaCl, 0.5mM PMSF, 10% Glycerol, pH 8.0

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:

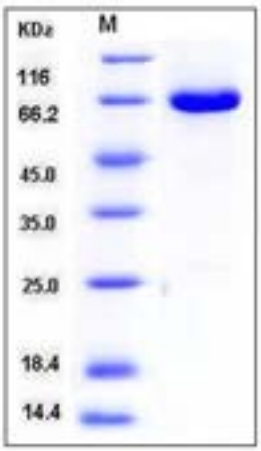
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

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 are 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 a number of transcription factors such as ELK1. MAPK1 acts as a transcriptional repressor which 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 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.

References

1.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. 2.Jivan A, *et al.* (2010) Reconstitution of the Nuclear Transport of the MAP Kinase ERK2. Methods Mol Biol. 661: 273-85. 3.Yu CG, *et al.* (2010) Involvement of ERK2 in traumatic spinal cord injury. J Neurochem. 113(1): 131-42.

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