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phospho-ERK1/2 (Thr202 + Tyr204) Recombinant Rabbit mAb

Catalog # AP100002

Product Information

Reactivity	Human, Mouse
Predicted	Rat
Host	Rabbit
Clonality	Recombinant
Calculated MW	41 KDa
Physical State	Liquid
Immunogen	KLH conjugated Synthesised phosphopeptide derived from mouse p44/42 MAPK around the phosphorylation site of Thr202/Tyr204
Epitope Specificity	FL(p-T)E(p-Y)VA
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the protein kinase superfamily. CMGCSer/Thr protein kinase family. MAP kinase subfamily. Contains 1 protein kinase domain.
SUBUNIT	Binds both upstream activators and downstream substratesin multimolecular complexes. Interacts with ADAM15, ARHGEF2, ARRB2,DAPK1 (via death domain), HSF4, IER3, IPO7, DUSP6, NISCH, SGK1, andisoform 1 of NEK2. Interacts (via phosphorylated form) with TPR(via C-terminus region and phosphorylated form); the interactionrequires dimerization of MAPK1/ERK2 and increases following EGFstimulation. Interacts (phosphorylated form) withCAV2 ('Tyr-19'-phosphorylated form); the interaction, promoted byinsulin, leads to nuclear location and MAPK1 activation. Interacts with DCC. Interacts withMORG1, PEA15 and MKNK2. MKNK2 isoform 1 binding prevents fromdephosphorylation and inactivation. The phosphorylated forminteracts with PML.
Post-translational modifications	Dually phosphorylated on Thr-183 and Tyr-185, which activatesthe enzyme. Ligand-activated ALK induces tyrosine phosphorylation. Dephosphorylated by PTPRJ at Tyr-185. Phosphorylated upon FLT3 and KIT signaling.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	The protein encoded by this gene is a member of the MAPkinase family. MAP kinases, also known as extracellularsignal-regulated kinases (ERKs), act in a signaling cascade thatregulates various cellular processes such as proliferation,differentiation, and cell cycle progression in response to avariety of extracellular signals. This kinase is activated byupstream kinases, resulting in its translocation to the nucleuswhere it phosphorylates nuclear targets. Alternatively splicedtranscript variants encoding different protein isoforms have beendescribed. [provided by RefSeq, Jul 2008].

Additional Information

Other Names	MK03_HUMAN; MAPK3; MAP kinase 3; MAPK 3; ERT2; Extracellular signal-regulated kinase 1 (ERK-1); Insulin-stimulated MAP2 kinase; MAP kinase isoform p44 (p44-MAPK); Microtubule-associated protein 2 kinase; p44-ERK1; 2.7.11.24; ERK1; PRKM3; MK01_HUMAN; MAPK1; MAP kinase 1; MAPK 1; ERT1; Extracellular signal-regulated kinase 2 (ERK-2); MAP kinase isoform p42 (p42-MAPK); Mitogen-activated protein kinase 2 (MAP kinase 2 | MAPK 2); ERK2; PRKM1; PRKM2; ERK; ERK-2; MAPK2; NS13; P42MAPK; p38; p40; p41; p41mapk; p42-MAPK; ERK-1; HS44KDAP; HUMKER1A; P44ERK1; P44MAPK; p44-MAPK; mitogen-activated protein kinase 3; ERK1 & ERK2 (phospho-T202/Y204); p-ERK1 & ERK2; phospho-ERK1 & ERK2; ERK1 & ERK2 (phospho-Thr202/Tyr204); p44/42
Dilution	Flow-Cyt=1:50-100,ICC/IF=1:50-200
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Name	phospho-ERK1/2 (Thr202 + Tyr204) Recombinant Rabbit mAb
Function	Serine/threonine kinase which acts as an essentialcomponent of the MAP kinase signal transduction pathway. MAPK1/ERK2and MAPK3/ERK1 are the 2 MAPKs which play an important role in theMAPK/ERK cascade. They participate also in a signaling cascadeinitiated by activated KIT and KITLG/SCF. Depending on the cellularcontext, the MAPK/ERK cascade mediates diverse biological functionssuch as cell growth, adhesion, survival and differentiation throughthe regulation of transcription, translation, cytoskeletalrearrangements. The MAPK/ERK cascade plays also a role ininitiation and regulation of meiosis, mitosis, and postmitoticfunctions in differentiated cells by phosphorylating a number oftranscription factors. About 160 substrates have already beendiscovered for ERKs. Many of these substrates are localized in thenucleus, and seem to participate in the regulation of transcriptionupon stimulation. However, other substrates are found in thecytosol as well as in other cellular organelles, and those areresponsible for processes such as translation, mitosis andapoptosis. Moreover, the MAPK/ERK cascade is also involved in theregulation of the endosomal dynamics, including lysosome processingand endosome cycling through the perinuclear recycling compartment(PNRC); as well as in the fragmentation of the Golgi apparatusduring mitosis. The substrates include transcription factors (suchas ATF2, BCL6, ELK1, ERF, FOS, HSF4 or SPZ1), cytoskeletal elements(such as CANX, CTTN, GJA1, MAP2, MAPT, PXN, SORBS3 or STMN1),regulators of apoptosis (such as BAD, BTG2, CASP9, DAPK1, IER3,MCL1 or PPARG), regulators of translation (such as EIF4EBP1) and avariety of other signaling-related molecules (like ARHGEF2, DCC,FRS2 or GRB10). Protein kinases (such as RAF1, RPS6KA1/RSK1,RPS6KA3/RSK2, RPS6KA2/RSK3, RPS6KA6/RSK4, SYK, MKNK1/MNK1,MKNK2/MNK2, RPS6KA5/MSK1, RPS6KA4/MSK2, MAPKAPK3 or MAPKAPK5) andphosphatases (such as DUSP1, DUSP4, DUSP6 or DUSP16) are othersubstrates which enable the propagation the MAPK/ERK signal toadditional cytosolic and nuclear targets, thereby extending thespecificity of the cascade. Mediates phosphorylation of TPR inrespons to EGF stimulation. May play a role in the spindle assemblycheckpoint. Phosphorylates PML and promotes its interaction withPIN1, leading to PML degradation (By similarity). Acts as a transcriptional repressor. Binds to a[GC]AAA[GC] consensus sequence. Repress the expression ofinterferon gamma-induced genes. Seems to bind to the promoter ofCCL5, DMP1, IFIH1, IFITM1, IRF7, IRF9, LAMP3, OAS1, OAS2, OAS3 andSTAT1. Transcriptional activity is independent of kinase activity.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.