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[Marker Primary Antibody]

Rabbit Anti-phospho-P38 MAPK (Thr180)/Cy3 Conjugated antibody

Catalog Number: SL5476R-Cy3

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	Anti-phospho-P38 MAPK (Thr180)/Cy3
Immunogen:	KLH conjugated Synthesised phosphopeptide derived from human MAPK14 around the phosphorylation site of Thr180
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Applications:	Flow-Cyt=1μg/Test IF=1:100-500 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Human,Mouse,Rat(predicted:Chicken,Dog,Pig,Horse,Rabbit)
Background:	background: p38 is a 38 kDa Stress Activated Protein Kinase/Map Kinase (SAPK/MAPK) that is fully activated by dual phosphorylation on threonine 180 and tyrosine 182, within the activation loop. p38 MAPK plays a critical role in the initiation of G2 delay after ultraviolet radiation; gene knock out studies have also revealed a critical role for p38 in cardiac remodeling. Downstream targets of p38 include the transcription factors ELK1 and ATF2 and the kinases MAPKAPK2 and MAPKAPK5. p38 MAPK plays a role in the production of IL6 and is thought to stabilize erythropoietin production during hypoxic stress. It is activated by environmental stress, many proinflammatory cytokines and lipopolysaccharide. Dual phosphorylation by MAP2K3 and MAP2K6 is required for activation of p38 MAPK. It interacts with MAX, Cdc25B, Cdc25C and binds to the kinase interaction domain in the protein tyrosine phosphatase PTPRR; this interaction

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	retains p38 MAPK in the cytoplasm. **Function:** Serine/threonine kinase which acts as an essential component of the MAP kinase signal transduction pathway. MAPK14 is one of the four p38 MAPKs which play an important role in the cascades of cellular responses evoked by extracellular stimuli such as proinflammatory cytokines or physical stress leading to direct activation of transcription factors. Accordingly, p38 MAPKs phosphorylate a broad range of proteins and it has been estimated that they may have approximately 200 to 300 substrates each. Some of the targets are downstream kinases which are activated through phosphorylation and further phosphorylate additional targets. RPS6KA5/MSK1 and RPS6KA4/MSK2 can directly phosphorylate and activate transcription factors such as CREB1, ATF1, the NF-kappa-B isoform RELA/NFKB3, STAT1 and STAT3, but can also phosphorylate histone H3 and the nucleosomal protein HMGN1. RPS6KA5/MSK1 and RPS6KA4/MSK2 play important roles in the rapid induction of immediate-early genes in response to stress or mitogenic stimuli, either by inducing chromatin remodeling or by recruiting the transcription machinery. On the other hand, two other kinase targets, MAPKAPK2/MK2 and MAPKAPK3/MK3, participate in the control of gene expression mostly at the post-transcriptional level, by phosphorylating ZFP36 (tristetraprolin) and ELAVL1, and by regulating EEF2K, which is important for the elongation of mRNA during translation. MKNK1/MNK1 and MKNK2/MNK2, two other kinases activated by p38 MAPKs, regulate protein synthesis by phosphorylating the initiation factor EIF4E2. MAPK14 interacts also with casein kinase II, leading to its activation through autophosphorylation and further phosphorylation of TP53/p53. In the cytoplasm, the p38 MAPK pathway is an important regulator of protein turnover. For example, CFLAR is an inhibitor of TNF-induced apoptosis whose proteasome-mediated degradation is regulated by p38 MAPK phosphorylation. In a similar way, MAPK14 phosphorylates the ubiquitin ligase SIAH2, regulating its activity towards EGLN3. MAPK14 may also inhibit the lysosomal degradation pathway of autophagy by interfering with the intracellular trafficking of the transmembrane protein ATG9. Another function of MAPK14 is to regulate the endocytosis of membrane receptors by different mechanisms that impinge on the small GTPase RAB5A. In addition, clathrin-mediated EGFR internalization induced by inflammatory cytokines and UV irradiation depends on MAPK14-mediated phosphorylation of EGFR itself as well as of RAB5A effectors. Ectodomain shedding of transmembrane proteins is regulated by p38 MAPKs as well. In response to inflammatory stimuli, p38 MAPKs phosphorylate the

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	membrane-associated metalloprotease ADAM17. Such phosphorylation is required for ADAM17-mediated ectodomain shedding of TGF-alpha family ligands, which results in the activation of EGFR signaling and cell proliferation. Another p38 MAPK substrate is FGFR1. FGFR1 can be translocated from the extracellular space into the cytosol and nucleus of target cells, and regulates processes such as rRNA synthesis and cell growth. FGFR1 translocation requires p38 MAPK activation. In the nucleus, many transcription factors are phosphorylated and activated by p38 MAPKs in response to different stimuli. Classical examples include ATF1, ATF2, ATF6, ELK1, PTPRH, DDIT3, TP53/p53 and MEF2C and MEF2A. The p38 MAPKs are emerging as important modulators of gene expression by regulating chromatin modifiers and remodelers. The promoters of several genes involved in the inflammatory response, such as IL6, IL8 and IL12B, display a p38 MAPK-dependent enrichment of histone H3 phosphorylation on 'Ser-10' (H3S10ph) in LPS-stimulated myeloid cells. This phosphorylation enhances the accessibility of the cryptic NF-kappa-B-binding sites marking promoters for increased NF-kappa-B recruitment. Phosphorylates CDC25B and CDC25C which is required for binding to 14-3-3 proteins and leads to initiation of a G2 delay after ultraviolet radiation. Phosphorylates TIAR following DNA damage, releasing TIAR from GADD45A mRNA and preventing mRNA degradation. The p38 MAPKs may also have kinase-independent roles, which are thought to be due to the binding to targets in the absence of phosphorylation. Protein O-Glc-N-acylation catalyzed by the OGT is regulated by MAPK14, and, although OGT does not seem to be phosphorylated by MAPK14, their interaction increases upon MAPK14 activation induced by glucose deprivation. This interaction may regulate OGT activity by recruiting it to specific targets such as neurofilament H, stimulating its O-Glc-N-acylation. Required in mid-fetal development for the growth of embryo-derived blood vessels in the labyrinth layer of the placenta. Also plays an essential role in developmental and stress-induced erythropoiesis, through regulation of EPO gene expression. Isoform MXI2 activation is stimulated by mitogens and oxidative stress and only poorly phosphorylates ELK1 and ATF2. Isoform EXIP may play a role in the early onset of apoptosis. Phosphorylates S100A9 at 'Thr-113'. **Subunit:** Binds to a kinase interaction motif within the protein tyrosine phosphatase, PTPRR (By similarity). This interaction retains MAPK14 in the cytoplasm and prevents nuclear accumulation. Interacts with SPAG9 and GADD45A. Interacts with CDC25B, CDC25C, DUSP1, DUSP10, DUSP16, NP60, FAM48A and TAB1. Interacts with casein kinase II subunits

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CSNK2A1 and CSNK2B.
Subcellular Location: Cytoplasm. Nucleus.
Tissue Specificity: Brain, heart, placenta, pancreas and skeletal muscle. Expressed to a lesser extent in lung, liver and kidney.
Post-translational modifications: Dually phosphorylated on Thr-180 and Tyr-182 by the MAP2Ks MAP2K3/MKK3, MAP2K4/MKK4 and MAP2K6/MKK6 in response to inflammatory citokines, environmental stress or growth factors, which a ctivates the enzyme. Dual phosphorylation can also be mediated by TAB1-mediated autophosphorylation. TCR engagement in T-cells also leads to Tyr-323 phosphorylation by ZAP70. Dephosphorylated and inactivated by DUPS1, DUSP10 and DUSP16.
Acetylated at Lys-53 and Lys-152 by KAT2B and EP300. Acetylation at Lys-53 increases the affinity for ATP and enhances kinase activity. Lys-53 and Lys-152 are deacetylated by HDAC3.
Ubiquitinated. Ubiquitination leads to degradation by the proteasome pathway.
Similarity: Belongs to the protein kinase superfamily. CMGC Ser/Thr protein kinase family. MAP kinase subfamily.
Contains 1 protein kinase domain.
Database links:
Entrez Gene: 1432 Human
Entrez Gene: 26416 Mouse
Entrez Gene: 81649 Rat
Entrez Gene: 403856 Dog
GenBank: NM_001315 Human
GenBank: NM_139012 Human
GenBank: NM_011951 Mouse
GenBank: NM_031020 Rat
Omim: 600289 Human
SwissProt: O02812 Dog
SwissProt: Q16539 Human
SwissProt: P47811 Mouse
SwissProt: P70618 Rat
Unigene: 485233 Human
Unigene: 311337 Mouse
Unigene: 88085 Rat
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Mitogen-activated protein kinase p38 (p38 MAPK, phosphorylated pERK) is involved in various physiological processes such as cell growth, proliferation,

	differentiation, death, and functional synchronization between cells. P-p38MAPK is a member of the mitogen-activated protein kinase family, and numerous studies have shown that p38 has widespread roles in energy metabolism. p38 is involved in energy metabolism in tissues and organs such as adipose tissue, skeletal muscle, pancreatic islet cells, and liver. p38 MAPK: as a convergence point of the cellular signal transduction system, it is a signal transduction pathway that is ubiquitous in cells. Extracellular physical stress factors such as high osmotic pressure, heat shock, ultraviolet light, as well as cell factors and endotoxin lipopolysaccharide (LPS), can all activate this pathway, inducing biological effects such as intracellular protein synthesis and secretion, cell differentiation, and apoptosis. p38 MAPK can also interact with other intracellular signaling pathways and is a convergence point or common pathway of the intracellular signal transduction system. Once activated, p38 MAPK can phosphorylate serine and threonine sites of some transcription factors such as CREB, activating factor-1 (ATF-1), ATF-2, and activating protein-1 (AP-1), activating these transcription factors and thereby regulating the expression of target genes. After phosphorylation (at serine sites), p38 can directly activate transcription factors and participate in the body's stress response.

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