

Product Information

sigma-aldrich.com

3050 Spruce Street, Saint Louis, MO 63103 USA

Tel: (800) 521-8956 (314) 771-5765 Fax: (800) 325-5052 (314) 771-5757

email: techservice@sial.com sigma-aldrich.com

Anti-phospho-p38 (pThr¹⁸⁰/pTyr¹⁸²)

produced in rabbit, affinity isolated antibody

Catalog Number **P1491**

Synonyms: Anti- SAPK/MAPK; Anti- Stress Activated Protein Kinase/MAP Kinase

Product Description

Anti-phospho-p38 (pThr¹⁸⁰/pTyr¹⁸²) is produced in rabbit using as immunogen a synthetic phosphopeptide derived from the region of p38 containing Thr¹⁸⁰ and Tyr¹⁸². The antibody is purified by epitope-specific affinity chromatography. The antibody is preadsorbed using: (i) a non-phosphopeptide corresponding to the site of phosphorylation to remove any reactivity with non-phosphorylated p38 enzymes, (ii) a JNK-derived peptide phosphorylated at Thr¹⁸³ and Tyr¹⁸⁵ within the activation loop.

The antibody recognizes the endogenous, active forms of p38 α , β , γ (43 kDa), and detects human and rat p38. Mouse, dog, monkey, and carp (100% homologous) p38 have not been tested, but are expected to react. The antibody is used in immunoblotting and immunostaining applications.

p38 is a mitogen-activated protein kinase (MAPK) member of the stress-activated protein kinases (SAPK). It is known also as drug-binding protein 1 (CSBP1), MXI2 and CSAID-binding protein.¹ The p38 signaling pathway plays a pivotal role in cell proliferation, differentiation, control of inflammatory responses, and apoptosis. p38 kinase plays a critical role in the initiation of a G2 delay after ultraviolet radiation.^{2,3}

p38 is activated by numerous cytokines and growth factors, endotoxic lipopolysaccharide, osmotic and environmental stress, and UV irradiation. More recently, it has been shown to be activated during ischemia/ reperfusion.⁴ *In vitro*, p38 binds and phosphorylates CDC25B and CDC25C, which is required for binding to 14-3-3 proteins. *In vivo*, inhibition of p38 prevents both phosphorylation of CDC25B at Ser³⁰⁹ and 14-3-3 binding after ultraviolet radiation. Regulation of CDC25B phosphorylation by p38 is a critical event for initiating the G2/M checkpoint after ultraviolet radiation.

The dually phosphorylated form of p38, Thr¹⁸⁰ and Tyr¹⁸², is a High Osmolarity Glycerol response kinase (HOG), a 43 kDa endogenous, active form of p38 α , β , and γ required for growth under high osmolarity conditions. This phosphorylated form is present in a variety of cells following treatment with a broad range of extracellular stimuli, including UV B irradiation of human skin, and ischemia.^{5,6}

Reagents

Supplied as a solution in Dulbecco's phosphate buffered saline (without Mg²⁺ and Ca²⁺), pH 7.3, with 50% glycerol, 1.0 mg/mL BSA (IgG and protease free) and 0.05% sodium azide.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

Store at -20 °C. Due to the presence of 50% glycerol the antibody will remain in solution. For extended storage, centrifuge the vial briefly before opening and prepare working aliquots. To ensure accurate dilutions mix gently, remove excess solution from pipette tip with clean absorbent paper, pipette slowly. The antibody is stable for at least six months when stored appropriately. Working dilutions should be discarded if not used within 12 hours.

Product Profile

One vial is sufficient for 10 immunoblots.

Immunoblotting: a recommended working dilution 1:1000 is determined using extracts from HEK 293 cells treated with UV irradiation.

Anti-phospho-p38 (pThr¹⁸⁰/pTyr¹⁸²) recognizes 43 kDa proteins corresponding to endogenous active forms p38 α , β , γ .

Data demonstrates that only phosphopeptide corresponding to the region containing Thr¹⁸⁰ and Tyr¹⁸² blocks the antibody signal, confirming the specificity of Anti-phospho-p38 (pThr¹⁸⁰/pTyr¹⁸²) for this phosphorylated residue.

Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working concentration by titration test.

References

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3. Bulavin, P.D., et al., Initiation of a G2/M checkpoint after ultraviolet radiation requires p38 kinase. *Nature*, **411**, 102-107 (2001).
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5. Pfundt, R., et al., *In situ* demonstration of phosphorylated c-jun and p38 MAP kinase in epidermal keratinocytes following ultraviolet B irradiation of human skin. *J. Pathol.*, **193**, 248-255 (2001).
6. Weinbrenner, C., et al., Phosphorylation of tyrosine 182 of p38 mitogen-activated protein kinase correlates with the protection of preconditioning in the rabbit heart. *J. Mol. Cell Cardiol.* **29**, 2383-2391 (1997).

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