Phospho-MEK1/2 (Ser217/221) (H2) rabbit mAb

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For Research Use Only. Not For Use In Diagnostic Procedures.

Applications	Detection	Clonality	Isotype
Flow Cytometry,WB	Anti-Rabbit IgG	Monoclonal	Rabbit IgGk

Format: Unconjugated

Cross Reactivity: Predicted to work with mouse, rat and other homologues.

Formulation: 1X PBS, 0.02% NaN3, 50% Glycerol, 0.1% BSA

Preparation: Protein A+G

Reactivity: Human

Recommended

Usage:

 $1\mu g/mL - 0.001\mu g/mL$. It is recommended that the reagent be titrated for optimal performance for each application. See product image legends for additional

information.

Immunogen: A synthetic phospho-peptide corresponding to residues surrounding Ser217/221 of

human phospho MEK1/2.

Description: Mitogen-activated protein kinase (MAPK) is the main building block of the

intracellular signaling network. The signals are initiated by activation of a small G protein (e.g., Ras), followed by a sequential activation of several sets of protein kinases. The extracellular signal-regulated kinase (ERK) pathway is stimulated by a large number of extracellular stimuli as well as various internal processes. This cascade regulates processes related to proliferation, differentiation, development, and oncogenic transformation. The signaling is usually initially started by activation of small G proteins (e.g. Ras), which in turn recruits to the membrane and activates the MAP3K (Raf kinase). The cascade leads to the activation of MAP kinase kinases

(MEKs). MEKs are phosphorylated at Ser218 and Ser222 in MEK1. The

phosphorylation activates MEKs. Phosphorylation of MEK at other sites regulates its activity as well. For example, phosphorylation at Ser386 by ERKs may either inhibit

ERKs activity or under certain condition, facilitate the activation of ERKs by

enhancing the MEK1 binding to Grb10 scaffold protein. Phosphorylation of MEK1 at Ser298 by p21-activating protein (PAK1) may lead to its activation whereas this can be inhibited by a feedback phosphorylation of MEK1 at Thr292 by ERKs. Protein Ser/Thr phosphatase inactivates MEK by dephosphorylation of pSer218 and

pSer222. Other phosphatases my also regulate MEK activity. Upon activation, MEKs behaves as a dual kinase and phosphorylate key Tyr and Thr residues of ERKs, thus

causing their activation.

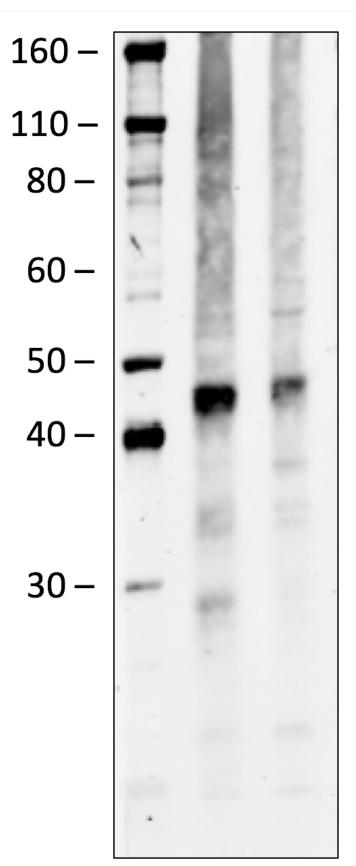
References: Raman M. and Cobb MH., (2003) Curr Biol 13:R886-888.

Kuida K., and Boucher DM. (2004) J Biochem 135:653-656.

Torii S., et al., (2004) J Biochem, 136:557-561.

Yoon S. and Seger R., (2006) Growth Factors 24:21-44. Natel A. et al., (1998) J Biol Chem 273:10475-10484.

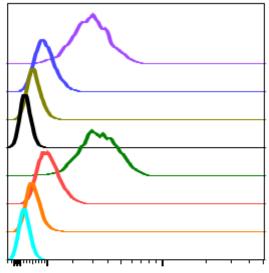




Phospho-MEK1/2 (S217/S221)

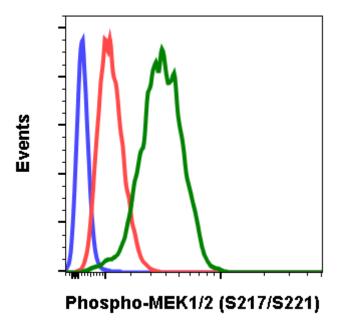
+ – UV+TPA

Western blot analysis of HeLa cell extract untreated or treated with UV+TPA using 0.1 μ g/mL Phospho-MEK1/2 (Ser217/221) antibody MEK12S217S221-H2. Cat.#2306.

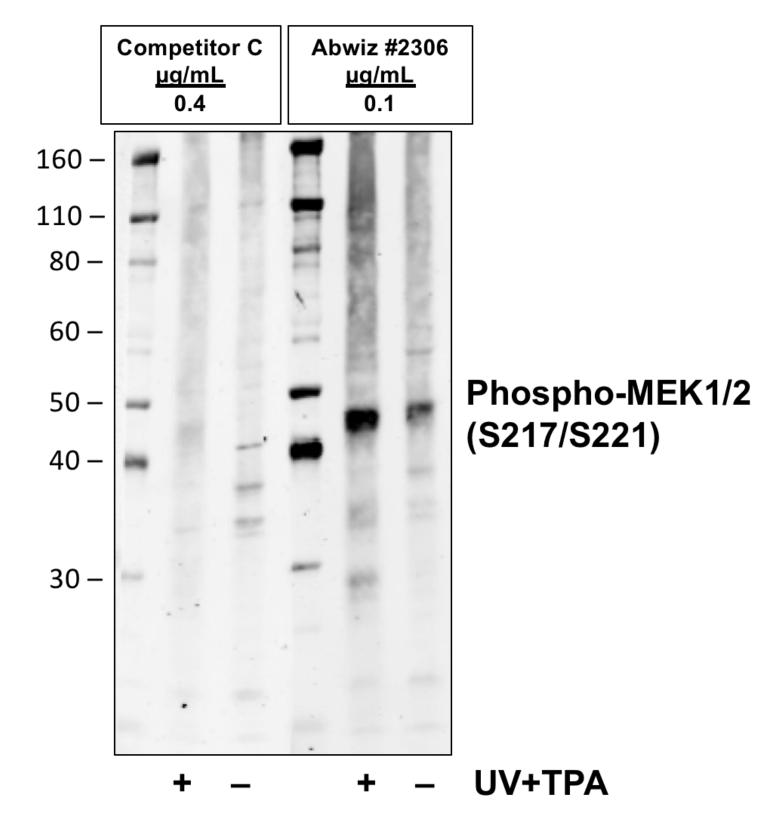


IgG	Treatment	Peptide Block	Median : BL1-A
H2	IFN	Non-phospho	2904
H2	Ctrl	Non-phospho	889
H2	IFN	Phospho	531
H2	Ctrl	Phospho	259
H2	IFN	-	3342
H2	Ctrl	-	1014
Isotype Ctrl	IFN	-	506
2' only	Ctrl	-	233

Peptide blocking flow cytometric analysis of Daudi cells secondary antibody only negative control (light blue) or stained with isotype control #2141 (orange) or untreated (red) or treated with IFN α + IL-4 + pervanadate (green) or untreated and blocked with phospho peptide (gold) or treated and blocked with phospho peptide (black) or untreated and blocked with non-phospho peptide (dark blue) or treated and blocked with non-phospho peptide (purple) using Phospho-MEK1/2 (S217/221) antibody MEK12S217S221-H2 at 0.01 µg/mL. Cat. #2306.



Flow cytometric analysis of Daudi cells secondary antibody only negative control (blue) or untreated (red) or treated with IFN α + IL-4 + pervanadate (green) using Phospho-MEK1/2 (S217/221) antibody MEK12S217S221-H2 0.01 μ g/mL. Cat. #2306.



Western blot analysis of HeLa cell extract untreated or treated with UV+TPA using 0.1 μ g/mL Phospho-MEK1/2 (Ser217/221) antibody MEK12S217S221-H2. Cat.#2306 or Company C antibody at 0.4 μ g/mL (manufacturer's recommended concentration) developed using the same exposure.