

Phospho-MEK1/2 (Ser221) (D3) rabbit mAb

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#2276

Store at: -20°C

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, Mouse

Recommended

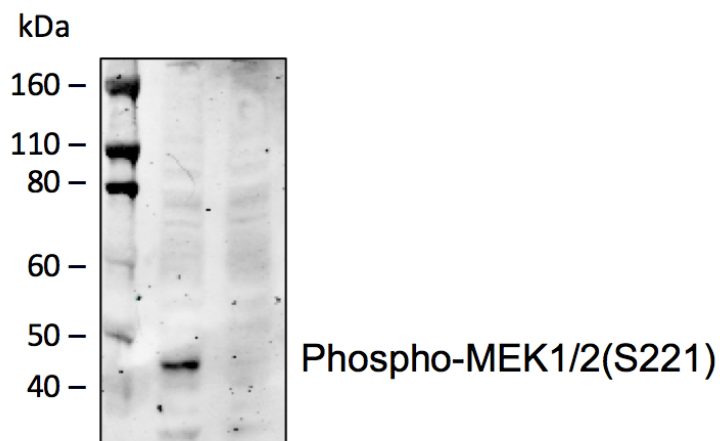
Usage: 1µg/mL ? 0.001µg/mL. It is recommended that the reagent be titrated for optimal performance for each application. See product image legends for additional information.

Immunogen: A synthetic phospho-peptide corresponding to residues surrounding Ser221 of human phospho MEK1/2.

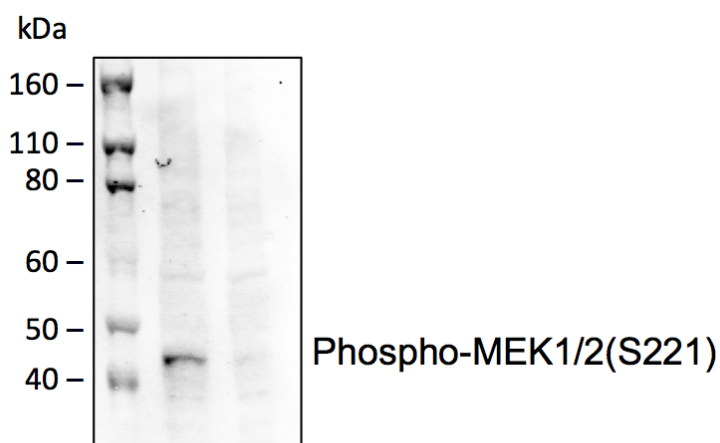
Description: Mitogen-activated protein kinase (MAPK) is the main building block of the intracellular signaling network. The signals are initiated by activation of a small G protein (e.g., Ras), followed by a sequential activation of several sets of protein kinases. The extracellular signal-regulated 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 may 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:

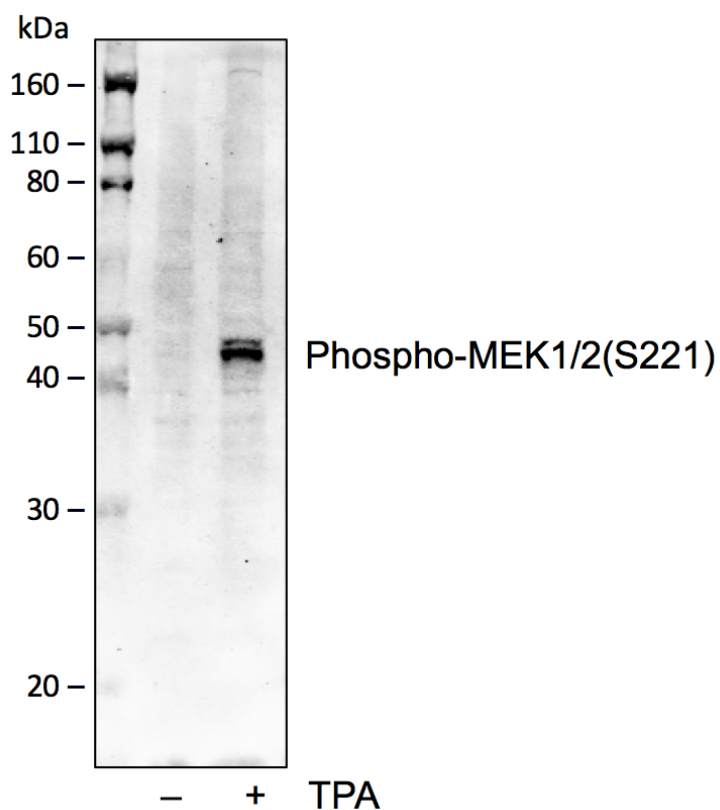
Raman M. and Cobb MH., (2003) Curr Biol 13:R886-888.
Kuida K., and Boucher DM. (2004) J Biochem 135:653-656.
Torii S., et al., (2004) J Biochem. 136:557-561.



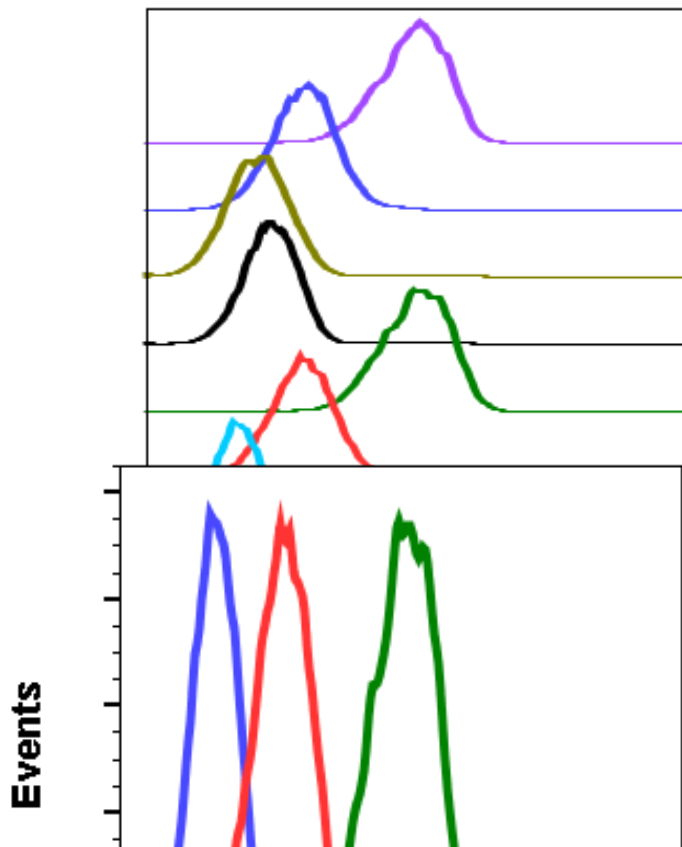
Western blot analysis of HT1080 cell extract untreated or treated with TPA using 0.1 μ g/mL Phospho-MEK1/2 (Ser221) antibody MEK12S221-D3. Cat.#2276.



Western blot analysis of C2C12 cell extract untreated or treated with TPA using 0.1 μ g/mL Phospho-MEK1/2 (Ser221) antibody MEK12S221-D3. Cat.#2276.



Western blot analysis of NIH3T3 cell extract untreated or treated with TPA using 0.1 μ g/mL Phospho-MEK1/2 (Ser221) antibody MEK12S221-D3. Cat.#2276.



Peptide blocking flow cytometric analysis of HeLa cells secondary antibody only negative control (light blue) or treated with imatinib (red) or pervanadate (green) or imatinib and blocked with phospho peptide (gold) or pervanadate and blocked with phospho peptide (black) or imatinib and blocked with non-phospho peptide (dark blue) or pervanadate and blocked with non-phospho peptide (purple) using Phospho-MEK1/2 (S221) antibody MEK12S221-D3 at 0.1 $\mu\text{g/mL}$. Cat. #2276.

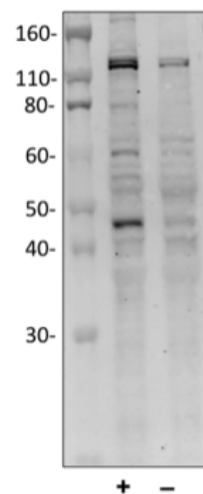
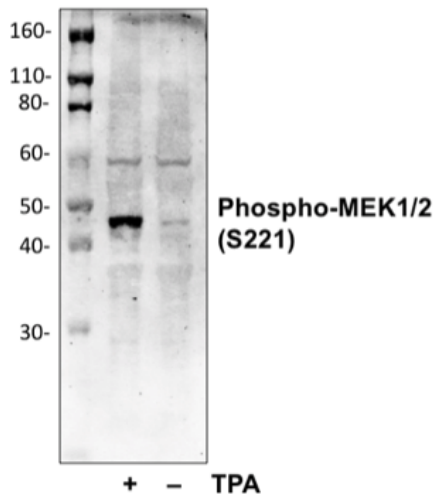
Flow cytometric analysis of HeLa cells secondary antibody only negative control (blue) or treated with imatinib (red) or with pervanadate (green) using Phospho-MEK1/2 (S221) antibody MEK12S221-D3 0.1 $\mu\text{g/mL}$. Cat. #2276.

Abwiz Cat. #2276

0.1 $\mu\text{g/mL}$

Company A

0.005 $\mu\text{g/mL}$
(recommended conc.)



Western blot analysis of A549 cell extract untreated or treated with TPA using 0.1 $\mu\text{g/mL}$ Phospho-MEK1/2 (Ser221) antibody MEK12S221-D3 Cat. #2276 or Company A antibody at 0.005 $\mu\text{g/mL}$ (manufacturer's recommended concentration).