Datasheet for ABIN1177238

anti-ERK1/2 antibody (pThr202) (PerCP-Cy5.5)

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Overview

Quantity:	50 tests
Target:	ERK1/2 (MAPK1/3)
Binding Specificity:	pThr202
Reactivity:	Human, Mouse, Rat
Host:	Mouse
Clonality:	Monoclonal
Conjugate:	This ERK1/2 antibody is conjugated to PerCP-Cy5.5
Application:	Intracellular Staining (ICS)

Product Details

Brand:	BD Phosflow™
Immunogen:	Phosphorylated Rat ERK1 (T202/Y204) Peptide
Clone:	20A
Isotype:	IgG1
Purification:	The monoclonal antibody was purified from tissue culture supernatant or ascites by affinity chromatography.

Target Details

Target:	ERK1/2 (MAPK1/3)
Alternative Name:	ERK1/2 (MAPK1/3 Products)

Target Details

Background:	The members of the Mitogen-Activated Protein Kinase (MAPK) family are components of a key signal transduction cascade that links events at the cell surface to responses in the nucleus. The signaling cascade is found in species as varied as yeast and humans, with many of the proteins being well conserved. In mammals the most widely studied members of the cascade are the Extracellular signal-Regulated Kinases, ERK1 (p44 MAPK) and ERK2 (p42 MAPK). ERK1 and ERK2 share 85% homology and are activated by extracellular signals such as growth factors, hormones, and phorbol esters. Activation occurs through a series of phosphorylations by kinases activating other kinases and eventually leading to phosphorylation of the ERKs. Growth factor stimulation leads to activation of Ras and Raf, leading to phosphorylation of MEK1 (MAPK/ERK kinase) which, in turn, activates the ERKs via dual phosphorylation. Once activated, the ERKs phosphorylate other cytoplasmic signalling molecules, cell-surface receptors, microtubule-associated proteins, and transcription factors in the nucleus. Thus, the active ERK has myriad downstream effectors that implicate it in the control of cell proliferation and differentiation, as well as regulation of the cytoskeleton. Furthermore, studies have shown that elevated ERK activity is associated with some cancers. The 20A monoclonal antibody recognizes the phosphorylated threonine 202 and tyrosine 204 (pT202/pY204) of human ERK1 and pT184/pY186 of human ERK2. The orthologous phosphorylation sites in murine ERK1 and ERK2 are T203/Y205 and T183/Y185. Synonyms: p44/42 MAPK, Extracellular signal-Regulated Kinase 1/2 (pT202/Y204)

Application Details

Application Notes:	This antibody conjugate is suitable for intracellular staining of human whole blood (using BD™ Phosflow Lyse/Fix Buffer) and peripheral blood mononuclear cells (using BD Cytofix™ Fixation Buffer or BD Phosflow™ Fix Buffer I). Any of the three BD Phosflow™ permeabilization buffers may be used.
Sample Volume:	20 µL
Restrictions:	For Research Use only

Handling

Format:	Liquid
Buffer:	Aqueous buffered solution containing BSA and ≤0.09 % sodium azide.
Preservative:	Sodium azide
Precaution of Use:	This product contains Sodium azide: a POISONOUS AND HAZARDOUS SUBSTANCE which

Handling

	should be handled by trained staff only.
Storage:	4 °C
Storage Comment:	Store undiluted at 4°C and protected from prolonged exposure to light. Do not freeze. The antibody was conjugated with PerCP-Cy5.5 under optimum conditions, and unconjugated antibody and free PerCP-Cy5.5 were removed.

Publications

Product cited in:

Kim, Hahn, Min, Yoo, Ko, Lee et al.: "Constitutive activation of extracellular signal-regulated kinase in human acute leukemias: combined role of activation of MEK, hyperexpression of extracellular signal-regulated kinase, and ..." in: **Blood**, Vol. 93, Issue 11, pp. 3893-9, (1999) (PubMed).

Sivaraman, Wang, Nuovo, Malbon: "Hyperexpression of mitogen-activated protein kinase in human breast cancer." in: **The Journal of clinical investigation**, Vol. 99, Issue 7, pp. 1478-83, (1997) (PubMed).

Treisman: "Regulation of transcription by MAP kinase cascades." in: **Current opinion in cell biology**, Vol. 8, Issue 2, pp. 205-15, (1996) (PubMed).

Cobb, Boulton, Robbins: "Extracellular signal-regulated kinases: ERKs in progress." in: **Cell regulation**, Vol. 2, Issue 12, pp. 965-78, (1992) (PubMed).

