

Alexa Fluor® 488 anti-ERK1/2 Phospho (Thr202/Tyr204)

Catalog # / Size: 2447540 / 100 tests
2447535 / 25 tests

Clone: 6B8B69

Isotype: Mouse IgG2a, κ

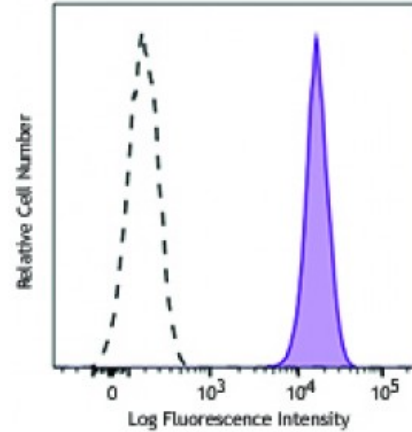
Immunogen: Synthetic peptide (TGFLT*EY*VATRC) conjugated to KLH.

Reactivity: Human, Mouse

Preparation: The antibody was purified by affinity chromatography and conjugated with Alexa Fluor® 488 under optimal conditions.

Formulation: Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide and 0.2% (w/v) BSA (origin USA).

Concentration: Lot-specific



Human peripheral blood lymphocytes were stimulated with (filled histogram) or without (open histogram) Cell Activation Cocktail (without Brefeldin A) for 15 minutes, then fixed with Fixation Buffer, permeabilized with True-Phos™ Perm Buffer, and int

Applications:

Applications: Flow Cytometry

Recommended Usage: Each lot of this antibody is quality control tested by intracellular flow cytometry . For flow cytometric staining, the suggested use of this reagent is 5 microL per million cells or 5 microL per 100 microL of whole blood. It is recommended that the reagent be titrated for optimal performance for each application.

* Alexa Fluor® 488 has a maximum emission of 519 nm when it is excited at 488 nm.

Application Notes: Clone 6B8B69 was found to strongly cross-react with mouse ERK1/2 Phospho (Thr202/Tyr204) when tested in-house on C57BL/6 mouse splenocytes.

Description: ERK1/2 are members of mitogen-activated kinases (MAPKs) of serine/threonine protein kinases. ERK1/2 can be activated by a range of extracellular stimuli, such as mitogen, growth factors, neurotransmitters, chemokines, and cytokines, through receptor tyrosine kinases (RTK), G protein-coupled receptors (GPCRs), or protein kinase C (PKC). Upon stimulation, ERK1/2 are phosphorylated by the upstream kinase MEK on residues Thr202 and Tyr204 and in turn phosphorylate many other downstream molecules that are involved in a range of cellular processes such as cell proliferation, differentiation, motility and cell death.

Antigen References:

1. Futran AS, *et al.* 2013. *Curr. Biol.* 23:R972.
2. Mendoza MC, *et al.* 2011. *Trends Biochem. Sci.* 36:320.
3. Chambard JC, *et al.* 2007. *Biochim. Biophys. Acta.* 1773:1299.
4. Roux PP,