Phospho-RelB (Ser552) (A7) rabbit mAb

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Applications	Detection	Clonality	Isotype
Flow Cytometry	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 Ser552 of human phospho RelB

Description: RelB contains the Rel homology Domain (RHD) shared by all members of the NF-kB family (1). It is best known for its roles in lymphoid development, DC biology, and noncanonical signaling (2). RelB is a major contributor to chromatin biology, frequently functioning as a dual transcription factor that silences sets of genes by generating silent facultative heterochromatin and activates euchromatin of others (3). RelB is required to repress immediate-response proinflammatory genes during endotoxin tolerance (4). The N-terminal leucine zipper motif of RelB, a motif unique among the NF-κB family, may associate with more diverse DNA sequences than other NF-κB members (5). RelB binds to DNA but only after forming a heterodimer with NF-kB p50 or p52 (6).
Like all NF-kB members, RelB contains an ~300-residue region known as the RHD. This region supports many of the NF-kB essential functions, such as DNA binding, dimerization, and nuclear localization (7). RelB, unlike other NF-kB members, has an N-terminal leucine zipper motif (1), a domain that can typically interact with many proteins (8). p100, the C-terminus of NF-kB2 acts as a RelB inhibitor (9), where it sequesters RelB in the cytosol to repress its activity. RelB is phosphorylated rapidly at threonine 84 and serine 552, causing cleavage at the N-terminus and its degradation by the proteasome (10). These phosphorylations are mediated by glycogen synthase kinase-3β, specific inhibition of which blocks phospho RelB phosphorylation and degradation (11). This degradation does not occur when phospho RelB is phosphorylated at serine 368, allowing its association with and stabilization by p100 (12).

References:

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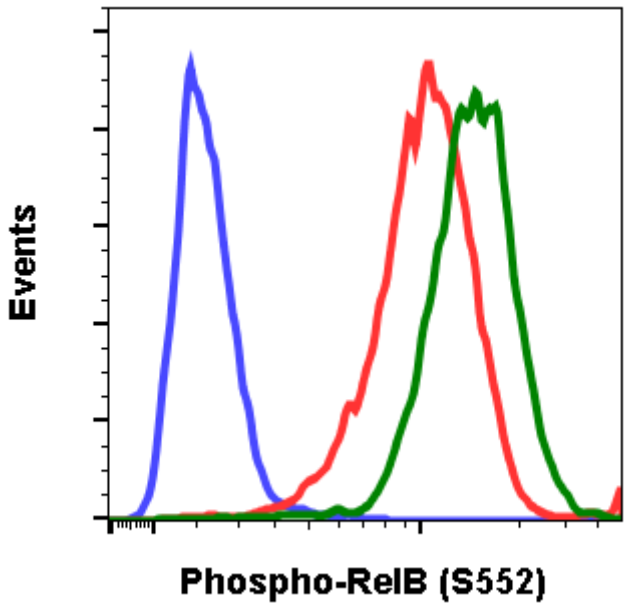
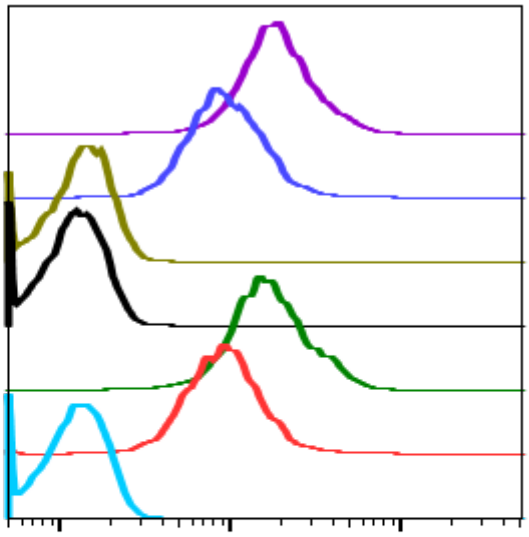
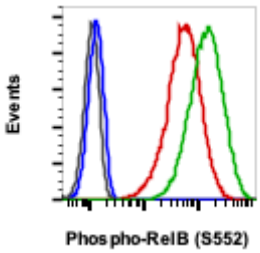


Flow cytometric analysis of NIH3T3 cells secondary antibody only negative control (blue) or untreated (red) or treated with UV + TPA (green) using 0.01 µg/mL Phospho-RelB (Ser552) antibody RelBS552-A7. Cat. #2206.



	SampleID	Median : BL1-A
	UV A7 N	18218
	Ctrl A7 N	9171
	UV A7 P	1366
	Ctrl A7 P	1206
	UV A7	16326
	Ctrl A7	8972
	Ctrl KLH	1241

Peptide blocking flow cytometric analysis of 3T3 cells isotype antibody-stained negative control (light blue) or untreated (red) or treated with UV and PMA (green) or untreated and blocked with phospho-peptide (black) or UV/PMA and blocked with phospho peptide (gold) or untreated and blocked with non-phospho peptide (dark blue) or UV/PMA and blocked with non-phospho peptide (purple) using Phospho-RelB (Ser552) antibody RelBS552-A7 0.01µg/mL. Cat. #2206.



Flow cytometric analysis of HeLa cells untreated (red) or treated with UV+TPA (green) using 0.1 µg/mL Phospho-RelB (Ser552) antibody RelBS552-A7. Cat. #2206, or concentration-matched rabbit (G9) mAb IgG isotope control #2141 for cells untreated (black) or treated with UV+TPA (blue).