Phospho-Chk1 (Ser345) (R3F9) rabbit mAb

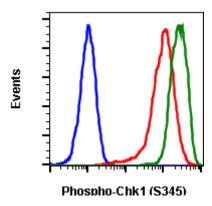
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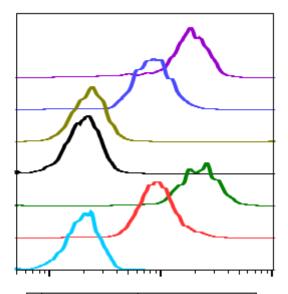
For Research Use Only. Not For Use In Diagnostic Procedures.

Applications Flow Cytometry	Detection Anti-Rabbit IgG	Clonality Monoclonal	lsotype Rabbit lgGk
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,Rat		
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 Ser345 of human phospho Chk1		
Description:	The act of DNA damaged response and cell cycle checkpoints requires the activation of four protein kinases that form the canonical ATR-Chk1 and ATM-Chk2 pathways. ATR activation requires the generation of structures containing single strand DNA (ssDNA) adjacent to double strand DNA (dsDNA). Such ssDNA is coated with replication protein A complex and attracts ATR (1,2). The accumulation of ATR to damage sites results in initial activation of ATR. ATR phosphorylates proteins at the ssDNA which are called checkpoint regulators. The accumulation and phosphorylation of these checkpoint regulators further stimulates the catalytic activity of ATR. ATR-induced Chk1 phosphorylation likely occurs at the sites of DNA damage on chromatin (3-5). The activated ATR phosphorylates Ser317 and Ser345 of phospho Chk1 in its C-terminal regulatory domain. Phospho Chk1 is critical for DNA damage checkpoint activation, replication control, and cell viability (6-8). Functionally, ATR-mediated phosphorylation elevates Chk1 catalytic activity. The N-terminal catalytic domain of Chk1 adopts an open kinase conformation and the deletion of C-terminal domain increases Chk1 catalytic activity.		
References:	 Caprelli ML, et al. (2013) Cell Cycle, 12: 916-22. Capasso H, et al. (2002) J. Cell Sci. 115: 4555-64. Carrassa L, et al. (2011) Cell Cycle 10: 2121-8. Chen MS, et al. (2003) Mol. Cell Biol. 23: 7488-97. Ciccia A, et al. Mol. Cell 40: 179-204. Cimprich CA, (2014) Oncogene 33: 3351-60. Niida H, et al. (2007) Mol. Cell Biol. 27: 2572-81. 		



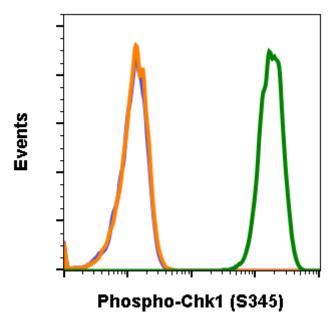


Flow cytometric analysis of Hela cells secondary antibody only negative control (blue) or untreated (red) or treated with UV plus TPA (green) using Phospho-Chk1(S345) antibody Chk1S345-R3F9 1µg/mL. Cat. #2191.

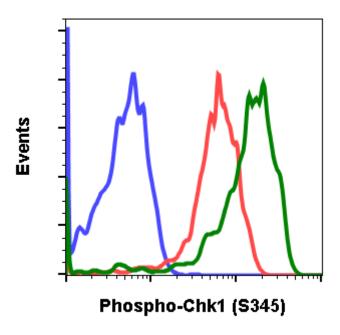


SampleID	Median : BL1-A
CalA R3F9 N	17783
Ctrl R3F9 N	8242
CalA R3F9 P	2272
Ctrl R3F9 P	2006
CalA R3F9	21845
Ctrl R3F9	8982
Ctrl 2' only	1982

Peptide blocking flow cytometric analysis of Hela cells secondary antibody only negative control (light blue) or untreated (red) or treated with calyculin A (green) or untreated and blocked with phospho-peptide (black) or CalA and blocked with phospho peptide (gold) or untreated and blocked with non-phospho peptide (dark blue) or CalA and blocked with non-phospho peptide (purple) using Phospho-Chk1(S345) antibody Chk1S345-R3F9 $0.01\mu g/mL$. Cat. #2191.



Chk1S345-R3F9 recognizes basal phosphorylation levels in mouse cells. Flow cytometric analysis of NIH3T3 cells secondary antibody only (blue) or $0.1~\mu g/mL$ of isotype control Cat. #2141 (orange) or of Phospho-Chk1(S345) antibody Chk1S345-R3F9 (green) Cat. #2191.



Flow cytometric analysis of C6 cells secondary antibody only negative control (blue) or treated with imatinib (red) or treated with pervanadate (green) using Phospho-Chk1(S345) antibody Chk1S345-R3F9 $0.01\mu g/mL$. Cat. #2191.