

## 12-4093: Phospho-Chk1 (Ser345) (Clone: R3F9) rabbit mAb

<b>Clonality :</b>	Monoclonal
<b>Clone Name :</b>	Chk1S345-R3F9
<b>Application :</b>	FACS
<b>Reactivity :</b>	Human, Mouse, Rat
<b>Conjugate :</b>	Unconjugated
<b>Format :</b>	Purified
<b>Alternative Name :</b>	Serine/threonine-protein kinase Chk1, CHK1 checkpoint homolog, Cell cycle checkpoint kinase, Checkpoint kinase-1, CHEK1
<b>Isotype :</b>	Rabbit IgG1k
<b>Immunogen Information :</b>	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.

### Product Info

<b>Amount :</b>	20 $\mu$ l / 200 $\mu$ l
<b>Content :</b>	1X PBS, 0.02% NaN <sub>3</sub> , 50% Glycerol, 0.1% BSA
<b>Storage condition :</b>	Store at -20°C. Avoid repeated freeze and thaw cycles.

### Application Note

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.(0.5mg/ml)

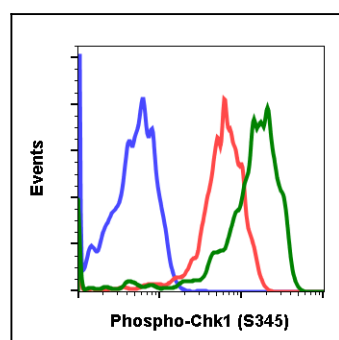


Fig-1: Flow cytometric analysis of C6 cells secondary antibody only negative control (blue) or treated with imatinib (red) or treated with pervanadate (green) using Phospho-Chk1(S345) antibody Chk1S345-R3F9 0.01 $\mu$ g/mL.

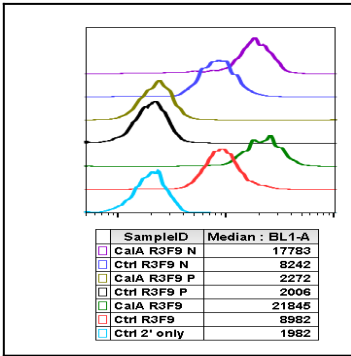


Fig 2 : Peptide blocking flow cytometric analysis of HeLa cells secondary antibody only negative control (light blue) or untreated (red) or treated with calyculin A (green) or untreated and blocked with phospho-peptide (black) or CaIA and blocked with phospho peptide (gold) or untreated and blocked with non-phospho peptide (dark blue) or CaIA and blocked with non-phospho peptide (purple) using Phospho-Chk1(S345) antibody Chk1S345-R3F9 0.01µg/mL.

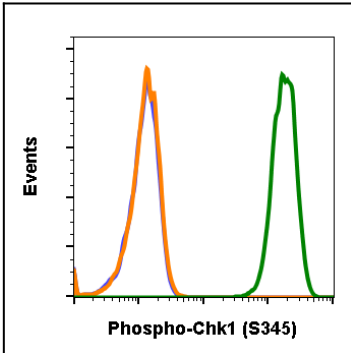


Fig-3: Chk1S345-R3F9 recognizes basal phosphorylation levels in mouse cells. Flow cytometric analysis of NIH3T3 cells secondary antibody only (blue) or 0.1 µg/mL of isotype control (orange) or of Phospho-Chk1(S345) antibody Chk1S345-R3F9 (green).

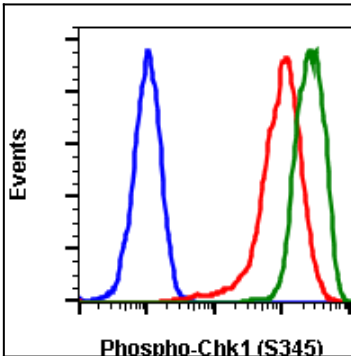


Fig-4: Flow cytometric analysis of HeLa cells secondary antibody only negative control (blue) or untreated (red) or treated with UV plus TPA (green) using Phospho-Chk1(S345) antibody Chk1S345-R3F9 1µg/mL.