

## 12-4123: Phospho-Chk1 (Ser317) (Clone: G1) rabbit mAb PE conjugate

<b>Clonality :</b>	Monoclonal
<b>Clone Name :</b>	Chk1S317-G1
<b>Application :</b>	FACS
<b>Reactivity :</b>	Human, Mouse
<b>Conjugate :</b>	PE
<b>Format :</b>	Conjugated
<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 Ser317 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 Chk1 at Ser317 and Ser345 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 phospho 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>	10 Tests / 100 Tests
<b>Content :</b>	1X PBS, 0.09% NaN <sub>3</sub> , 0.2% BSA
<b>Storage condition :</b>	Store at 2-8°C. Avoid repeated freeze and thaw cycles.

### Application Note

For flow cytometric staining, the suggested use of this reagent is 5  $\mu$ L per million cells or 5  $\mu$ L per 100  $\mu$ L of staining volume. It is recommended that the reagent be titrated for optimal performance for each application.

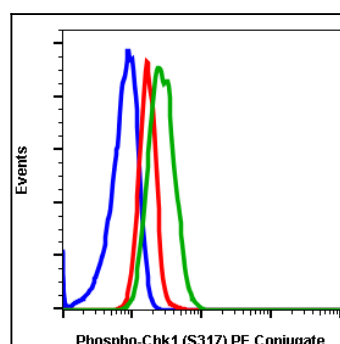


Fig-1: Flow cytometric analysis of HeLa cells unstained treated with Imatinib as negative control (blue) versus stained HeLa cells treated with Imatinib (red) or treated with pervanadate (green) using phospho-Chk1 (S317) antibody Chk1S317-G1 PE conjugate.