Revision 1

#9917 Store at -20°C

Cell Cycle/Checkpoint Antibody Sampler Kit

1 Kit (6 x 20 microliters)

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-cdc2 (Tyr15) (10A11) Rabbit mAb	4539	20 µl	34 kDa	Rabbit
Phospho-Chk1 (Ser345) (133D3) Rabbit mAb	2348	20 µl	56 kDa	Rabbit IgG
Phospho-Chk2 (Thr68) (C13C1) Rabbit mAb	2197	20 µl	62 kDa	Rabbit IgG
Phospho-Rb (Ser807/811) (D20B12) XP® Rabbit mAb	8516	20 µl	110 kDa	Rabbit IgG
Phospho-Rb (Ser795) Antibody	9301	20 µl	110 kDa	Rabbit
Phospho-p53 (Ser15) (16G8) Mouse mAb	9286	20 µl	53 kDa	Mouse IgG1
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat
Anti-mouse IgG, HRP-linked Antibody	7076	100 µl		Horse

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

The Cell Cycle/Checkpoint Antibody Sampler Kit provides a fast and economical means of evaluating multiple proteins involved in the cell cyle and checkpoint control. The kit contains enough primary and secondary antibody to perform four Western blot experiments.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Background

The cell division cycle demands accuracy to avoid the accumulation of genetic damage. This process is controlled by molecular circuits called "checkpoints" that are common to all eukaryotic cells (1). Checkpoints monitor DNA integrity and cell growth prior to replication and division at the G1/S and G2/M transitions, respectively. The cdc2-cyclin B kinase is pivotal in regulating the G2/M transition (2,3). Cdc2 is phosphorylated at Thr14 and Tyr15 during G2-phase by the kinases Wee1 and Myt1, rendering it inactive. The tumor suppressor protein retinoblastoma (Rb) controls progression through the late G1 restriction point (R) and is a major regulator of the G1/S transition (4). During early and mid G1-phase, Rb binds to and represses the transcription factor E2F (5). The phosphorylation of Rb late in G1-phase by CDKs induces Rb to dissociate from E2F, permitting the transcription of S-phase-promoting genes. *In vitro*, Rb can be phosphorylated at multiple sites by cdc2, cdk2, and cdk4/6 (6-8). DNA damage triggers both the G2/M and the G1/S checkpoints. DNA damage activates the DNA-PK/ATM/ATR kinases, which phosphorylate Chk at Ser345 (9), Chk2 at Thr68 (10) and p53 (11). The Chk kinases inactivate cdc25 via phosphorylation at Ser216, blocking the activation of cdc2.

Background References

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