Revision 2

Store at -20C

#12738

Aurora A/B Substrate Antibody Sampler Kit

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-CENP-A (Ser7) Antibody	2187	20 µl	17 kDa	Rabbit
Phospho-Histone H3 (Ser10) (D2C8) XP® Rabbit mAb	3377	20 µl	17 kDa	Rabbit IgG
Phospho-Histone H3 (Ser28) Antibody	9713	20 µl	17 kDa	Rabbit
Phospho-p53 (Ser315) Antibody	2528	20 µl	53 kDa	Rabbit
Phospho-PLK1 (Thr210) (D5H7) Rabbit mAb	9062	20 µl	62 kDa	Rabbit IgG
Phospho-TACC3 (Ser558) (D8H10) XP® Rabbit mAb	8842	20 µl	140 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

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

Description

The Aurora A/B Substrate Antibody Sampler Kit provides an economical means to investigate the G2/M phase of the cell cycle. The kit contains enough primary antibody to perform two western blots per primary antibody.

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

Aurora kinases belong to a highly conserved family of mitotic serine/threonine kinases with three members identified among mammals: Aurora A, B, and C (1,2). Studies on the temporal expression pattern and subcellular localization of Aurora kinases in mitotic cells suggest an association with mitotic structure. Aurora kinase functional influences span from G2 phase to cytokinesis and may be involved in key cell cycle events such as centrosome duplication, chromosome bi-orientation and segregation, cleavage furrow positioning, and ingression (3). Aurora A is detected at the centrosomes, along mitotic spindle microtubules, and in the cytoplasm of mitotically proliferating cells. Aurora A protein levels are low during G1 and S phases and peak during the G2/M phase of the cell cycle. Phosphorylation of Aurora A at Thr288 in its catalytic domain increases kinase activity. Aurora A is involved in centrosome separation, maturation, and spindle assembly and stability. Expression of Aurora B protein also peaks during the G2/M phase of the cell cycle; Aurora B kinase activity peaks at the transition from metaphase to the end of mitosis. Aurora B associates with chromosomes during prophase prior to relocalizing to the spindle at anaphase. Aurora B regulates chromosome segregation through the control of microtubule-kinetochore attachment and cytokinesis. Expression of both Aurora A and Aurora B during the G2/M phase transition is tightly coordinated with histone H3 phosphorylation (4,5); research investigators have observed overexpression of these kinases in a variety of human cancers (2,4). Aurora C localizes to the centrosome from anaphase to cytokinesis and both mRNA and protein levels peak during G2/M phase. Although typical Aurora C expression is limited to the testis, research studies report overexpression of Aurora C is detected in various cancer cell lines (6).

Transforming acid coiled-coil (TACC) proteins are a family of proteins characterized by a common coiled-coil motif of approximately 200 amino acids at the carboxy-terminal end (7). When phosphorylated at Ser558 by Aurora A, mammalian TACC3 is localized to mitotic spindles and increases microtubule stability (8,9).

Aurora A-dependent phosphorylation of CENP-A on Ser7 during prophase is required for proper targeting of Aurora B to the inner centromere in prometaphase, proper kinetochore/microtubule attachment and proper alignment of chromosomes during mitosis (10). Aurora B also targets Ser7 on CENP-A, which in turn regulates Aurora B activity during cytokinesis (11). Aurora B phosphorylates both Ser10 and Ser28 on histone H3 in concordance with mitotic chromosome condensation (12).

Activation of p53 can lead to either cell cycle arrest and DNA repair or apoptosis (13). Aurora A phosphorylates p53 at Ser315 in a cell cycle-dependent manner leading to MDM2-mediated ubiquitination/degradation of p53 (14). Aurora A phosphorylation of Thr210 on PLK promotes mitotic entry following checkpoint-dependent cell cycle arrest (15).

Background References

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