#96882

Aurora A/B Substrate Antibody Sampler



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

1 Kit (6 x 20 microliters)

Product Includes		t # Quantit	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-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
Phospho-Aurora A (Thr288)/Aurora B (Thr232)/Aurora C (Thr198) (D13A11) XP [®] Rabbit mAb	2914	20 µl	35, 40, 48 kD	a 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 includes enough antibodies to perform two western blot experiments with each 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. <i>Do not aliquot the antibodies.</i>
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 regulates 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 to 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 phosphorylati
Background References	1. Warner, S.L. et al. (2003) <i>Mol Cancer Ther</i> 2, 589-95. 2. Katayama, H. et al. (2003) <i>Cancer Metastasis Rev</i> 22, 451-64.

	 Andrews, P.D. et al. (2003) <i>Curr Opin Cell Biol</i> 15, 672-83. Pascreau, G. et al. (2003) <i>Prog Cell Cycle Res</i> 5, 369-74. Crosio, C. et al. (2002) <i>Mol Cell Biol</i> 22, 874-85. Kimura, M. et al. (1999) <i>J Biol Chem</i> 274, 7334-40. Gergely, F. et al. (2000) <i>Proc Natl Acad Sci U S A</i> 97, 14352-7. Kinoshita, K. et al. (2005) <i>J Cell Biol</i> 170, 1047-55. Schneider, L. et al. (2007) <i>J Biol Chem</i> 282, 29273-83. Kunitoku, N. et al. (2003) <i>Dev Cell</i> 5, 853-64. Zeitlin, S.G. et al. (2001) <i>J Cell Biol</i> 155, 1147-57. Goto, H. et al. (2003) <i>Nature</i> 455, 119-23. Macůrek, L. et al. (2018) <i>Cell Div</i> 13, 7.
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