

Aurora-B (ARK/STK12) Antibody (Center) Blocking peptide Synthetic peptide Catalog # BP7240c

Specification

Aurora-B (ARK/STK12) Antibody (Center) Blocking peptide - Product Information

Primary Accession

<u>Q96GD4</u>

Aurora-B (ARK/STK12) Antibody (Center) Blocking peptide - Additional Information

Gene ID 9212

Other Names

Aurora kinase B, Aurora 1, Aurora- and IPL1-like midbody-associated protein 1, AIM-1, Aurora/IPL1-related kinase 2, ARK-2, Aurora-related kinase 2, STK-1, Serine/threonine-protein kinase 12, Serine/threonine-protein kinase 5, Serine/threonine-protein kinase aurora-B, AURKB, AIK2, AIM1, AIRK2, ARK2, STK1, STK12, STK5

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Aurora-B (ARK/STK12) Antibody (Center) Blocking peptide - Protein Information

Name AURKB

Function

Serine/threonine-protein kinase component of the chromosomal passenger complex (CPC), a complex that acts as a key regulator of mitosis (PubMed:11516652, PubMed:12925766, PubMed:14610074, PubMed:14722118, PubMed:29449677). The CPC
complex has essential functions at the centromere in ensuring correct chromosome alignment and
segregation and is required for chromatin-induced microtubule stabilization and spindle assembly
(PubMed:12925766,
PubMed:14610074,
PubMed:14722118,
PubMed:14722118,
PubMed:14610074,
PubMed:14722118,
PubMed:26829474).
Involved in the bipolar attachment of spindle microtubules to kinetochores and is a key regulator



for the onset of cytokinesis during mitosis (PubMed: 15249581). Required for central/midzone spindle assembly and cleavage furrow formation (PubMed:12458200, PubMed:12686604). Key component of the cytokinesis checkpoint, a process required to delay abscission to prevent both premature resolution of intercellular chromosome bridges and accumulation of DNA damage: phosphorylates CHMP4C, leading to retain abscission-competent VPS4 (VPS4A and/or VPS4B) at the midbody ring until abscission checkpoint signaling is terminated at late cytokinesis (PubMed:22422861, PubMed:24814515). AURKB phosphorylates the CPC complex subunits BIRC5/survivin, CDCA8/borealin and INCENP (PubMed:11516652, PubMed:12925766, PubMed:14610074). Phosphorylation of INCENP leads to increased AURKB activity (PubMed: 11516652, PubMed:12925766, PubMed:14610074). Other known AURKB substrates involved in centromeric functions and mitosis are CENPA, DES/desmin, GPAF, KIF2C, NSUN2, RACGAP1, SEPTIN1, VIM/vimentin, HASPIN, and histone H3 (PubMed:11784863, PubMed:12689593, PubMed:14602875, PubMed:11856369, PubMed:16103226, PubMed:21658950, PubMed:11756469). A positive feedback loop involving HASPIN and AURKB contributes to localization of CPC to centromeres (PubMed:21658950). Phosphorylation of VIM controls vimentin filament segregation in cytokinetic process, whereas histone H3 is phosphorylated at 'Ser-10' and 'Ser-28' during mitosis (H3S10ph and H3S28ph, respectively) (PubMed:11784863, PubMed:11856369). AURKB is also required for kinetochore localization of BUB1 and SGO1 (PubMed: 15020684, PubMed:17617734). Phosphorylation of p53/TP53 negatively regulates its transcriptional activity (PubMed:20959462). Key regulator of active promoters in resting B- and T-lymphocytes: acts by mediating phosphorylation of H3S28ph at active promoters in resting B-cells, inhibiting RNF2/RING1B-mediated ubiquitination of histone H2A and enhancing binding and activity of the USP16 deubiguitinase at transcribed genes (By similarity). Acts as an inhibitor of CGAS during mitosis: catalyzes phosphorylation of the N-terminus of CGAS during the G2-M transition, blocking CGAS liquid phase separation and activation, and thereby preventing CGAS-induced autoimmunity (PubMed:33542149). Phosphorylates KRT5 during anaphase and telophase (By similarity).

Cellular Location

Nucleus. Chromosome. Chromosome, centromere. Chromosome, centromere, kinetochore. Cytoplasm, cytoskeleton, spindle. Midbody. Note=Localizes on chromosome arms and inner centromeres from prophase through metaphase and then transferring to the spindle midzone and midbody from anaphase through cytokinesis (PubMed:20929775). Colocalized with gamma tubulin in the midbody (PubMed:17726514). Proper localization of the active, Thr-232- phosphorylated form during metaphase may be dependent upon interaction with SPDYC (PubMed:20605920). Colocalized with SIRT2 during cytokinesis with the midbody (PubMed:17726514). Localization (and



probably targeting of the CPC) to the inner centromere occurs predominantly in regions with overlapping mitosis-specific histone phosphorylations H3pT3 and H2ApT12 (PubMed:20929775).

Tissue Location

High level expression seen in the thymus. It is also expressed in the spleen, lung, testis, colon, placenta and fetal liver. Expressed during S and G2/M phase and expression is up-regulated in cancer cells during M phase.

Aurora-B (ARK/STK12) Antibody (Center) Blocking peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

<u>Blocking Peptides</u>

Aurora-B (ARK/STK12) Antibody (Center) Blocking peptide - Images

Aurora-B (ARK/STK12) Antibody (Center) Blocking peptide - Background

Chromosomal segregation during mitosis as well as meiosis is regulated by kinases and phosphatases. The Aurora kinases associate with microtubules during chromosome movement and segregation. STK12 (Aurora kinase B) localizes to microtubules near kinetochores, specifically to the specialized microtubules called K-fibers, and Aurora kinase A localizes to centrosomes

Aurora-B (ARK/STK12) Antibody (Center) Blocking peptide - References

Kimura, M., et al., Biochem. Biophys. Res. Commun. 316(3):930-936 (2004).Yasui, Y., et al., J. Biol. Chem. 279(13):12997-13003 (2004).Lampson, M.A., et al., Nat. Cell Biol. 6(3):232-237 (2004).Wheatley, S.P., et al., J. Biol. Chem. 279(7):5655-5660 (2004).Honda, R., et al., Mol. Biol. Cell 14(8):3325-3341 (2003).