10320 Camino Santa Fe, Suite G
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Anti-Aurora B Antibody
Mouse Monoclonal Antibody
Catalog # AH13604

Specification

Anti-Aurora B Antibody - Product Information

Application	**,1,3,4,**
Primary Accession	Q96GD4
Other Accession	442658
Reactivity	**Human**
Host	**Mouse**
Clonality	**Monoclonal**
Isotype	**Mouse / IgG2b**
Calculated MW	**39311**

Anti-Aurora B Antibody - Additional Information

Gene ID 9212

Other Names
AIK2; AIM-1; ARK-2; AurB; AURKB; Aurora-1; Aurora and Ipl1 like midbody associated protein 1; Aurora kinase B; Aurora-B; Aurora-related kinase 2; Aurora/IPL1-related kinase 2; IPL1; Protein phosphatase 1 regulatory subunit 48 (PPP1R48); Serine/threonine-protein kinase 12; Serine/threonine-protein kinase aurora-B; STK1; STK12; STK5

Format
200ug/ml of Ab purified from Bioreactor Concentrate by Protein A/G. Prepared in 10mM PBS with 0.05% BSA & 0.05% azide. Also available WITHOUT BSA & azide at 1.0mg/ml.

Storage
Store at 2 to 8°C.Antibody is stable for 24 months.

Precautions
Anti-Aurora B Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Anti-Aurora B Antibody - 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:11516652, PubMed:12925766, 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:11756469, PubMed:11784863, PubMed:11856369, PubMed:12689593, PubMed:14602875, PubMed:16103226, PubMed:21658950). 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 deubiquitinase 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). Phosphorylates ATXN10 which promotes phosphorylation of ATXN10 by PLK1 and may play a role in the regulation of cytokinesis and stimulating the proteasomal degradation of ATXN10

(PubMed:25666058).

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).

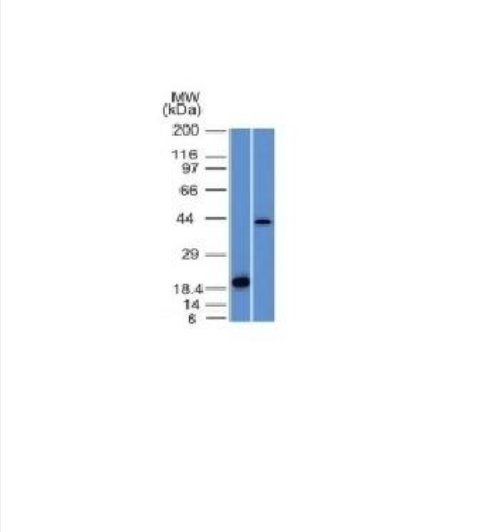
Tissue Location
High level expression seen in the thymus. It is also expressed in the spleen, lung, testis, colon, placenta and fetal liver. Expressed during S and G2/M phase and expression is up-regulated in cancer cells during M phase.

Anti-Aurora B Antibody - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Anti-Aurora B Antibody - Images



Western Blot Analysis (A) Recombinant Protein (B) Human Liver Lysate Using Aurora B Monoclonal Antibody (AURKB/1521).

Anti-Aurora B Antibody - Background

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Recognizes a protein of 39kDa, which is identified as Aurora B. The serine/threonine protein kinase aurora B (Aurora B) is a chromosomal passenger protein critical for accurate chromosome segregation, cytokinesis, protein localization to the centromere and kinetochore, correct microtubule-kinetochore attachment, and regulation of the mitotic checkpoint. Aurora B forms a tight complex with inner centrosome protein and survivin. Inactivation of any of these proteins causes similar defects in chromosome segregation. A significant overexpression of Aurora B has been found in a variety of human tumors including non-small cell lung carcinoma, astrocytoma, seminoma and carcinomas of the colon, prostate, endometrium and thyroid. The expression level of Aurora B is associated with cell proliferation and prognosis in these tumors.