Phospho-Aurora A (Thr288)/Aurora B (Thr232)/Aurora C (Thr198) (CC12) rabbit mAb

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Catalog: #2386 **Store at:** -20ºC

For Research Use Only. Not For Use In Diagnostic Procedures.

Applications	Detection	Clonality	Isotype
Flow Cytometry	Anti-Rabbit IgG	Monoclonal	Rabbit IgGk

Format: Unconjugated

Cross Reactivity: Predicted to work with mouse, rat and other homologues.

Formulation: 1X PBS, 0.02% NaN3, 50% Glycerol, 0.1% BSA

Preparation: Protein A+G

Reactivity: Human

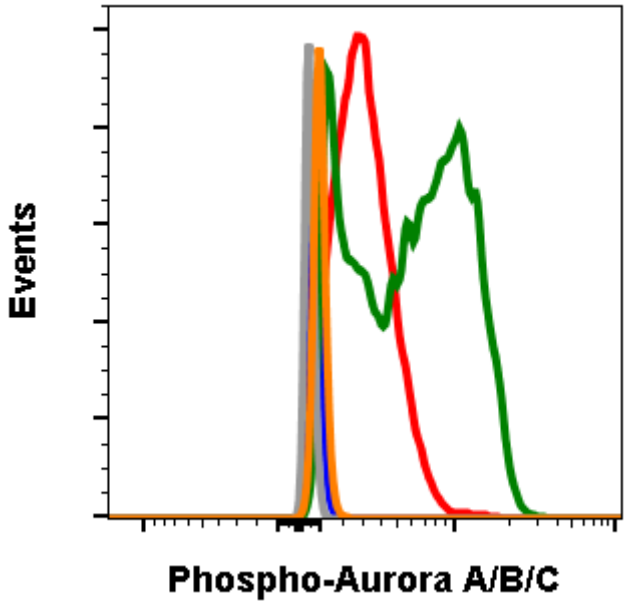
Recommended Usage: 1µg/mL – 0.001µg/mL. It is recommended that the reagent be titrated for optimal performance for each application. See product image legends for additional information.

Immunogen: A synthetic phospho-peptide corresponding to residues surrounding human Aurora A (Thr288)/Aurora B (Thr232)/Aurora C (Thr198)

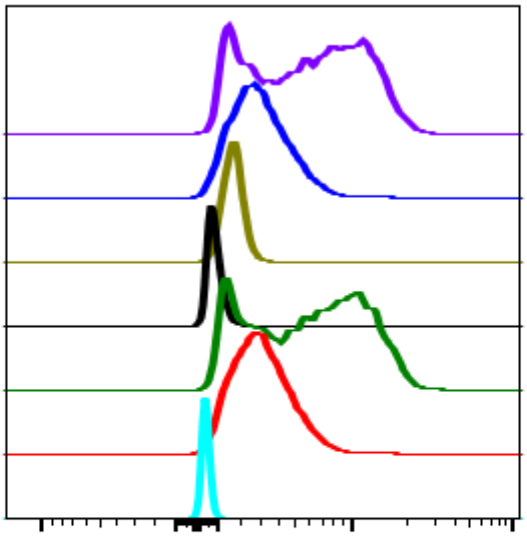
Description: Aurora kinases (serine/threonine kinases) are essential requirement for the onset and progression of mitosis. These kinases share a similar protein structure as well as kinase activity, however each kinase display distinct cellular and subcellular localization. Each Aurora member is phosphorylated at specific residues upon co-factor binding during mitosis. Aurora kinases acquire active kinase conformations due to the activation loop. The active kinase conformation is acquired upon auto-phosphorylation through an intermolecular (trans)-reaction within Aurora kinase domain. Aurora Kinase A (Aurora A) is involved in G2/M transition. AuroraA promotes centrosome maturation and mitotic spindle assembly, whereas AuroraB and AuroraC act as chromosome-passenger complex proteins. They play a crucial role in chromosomal binding to kinetochores and segregation of chromosomes. Aurora B is widely distributed in the cell, while AuroraC is expressed mainly in the meiotically-active germ cells. Aurora kinases are auto-phosphorylated into active forms at conserved threonine residues (i.e. the Thr288 (AurA), Thr232 (AurB) and Thr195 (AurC) residues). AuroraA auto-phosphorylation is initiated by several co-factors acting at different steps of mitosis. AroraB and AruroaC auto-phosphorylation are mediated by survivin and Borealin proteins.

References:

Hochegger H, et al., (2013) Open Biol. 3:120185.
Carmena M, et al., (2009) Curr Opin Cell Biol 21:796–805. □
Bolanos-Garcia VM. (2005) Int J Biochem Cell Biol. 37:1572–1577.
Kimmins S, et al., (2007) Mol Endocrinol. 2007;21(3):726–739.
Vader G, and Lens SMA. (2008) Biochim Biophys Acta. 1786:60–72.



Flow cytometric analysis of HeLa cells secondary antibody only negative control (blue) or untreated (grey) or treated with nocodazole (orange) using 0.5 µg/mL of isotype control Cat. #2141 or untreated (red) or treated (green) using Phospho-Aurora A (Thr288)/Aurora B (Thr232)/Aurora C (Thr198) antibody AuroraABC-CC12 at 0.5 µg/mL. Cat. #2386.



	IgG	Treatment	Peptide Block	Median : BL1-A
□	CC12	Nocod	Non-phos.	6223
□	CC12	Control	Non-phos.	2827
□	CC12	Nocod	Phospho	1637
□	CC12	Control	Phospho	717
□	CC12	Nocod	-	6294
□	CC12	Control	-	2948
□	2' only	Control	-	355

Peptide blocking flow cytometric analysis of HeLa cells unstained cells negative control (light blue) or untreated (red) or treated with nocodazole (green) or untreated and blocked with phospho-peptide (black) or nocodazole and blocked with phospho peptide (gold) or untreated and blocked with non-phospho peptide (dark blue) or nocodazole and blocked with non-phospho peptide (purple) using Phospho-Aurora A (Thr288)/Aurora B (Thr232)/Aurora C (Thr198) antibody AuroraABC-CC12 at 0.5 µg/mL. Cat. #2386.