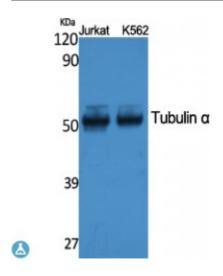
Anti-Tubulin alpha antibody



Description Rabbit polyclonal to Tubulin alpha.

Model STJ96416

Host Rabbit

Reactivity Human, Mouse, Rat

Applications ELISA, IHC, WB

Immunogen Synthesized peptide derived from human Tubulin alpha.

Immunogen Region C-terminal

Gene ID <u>7846</u>

Gene Symbol TUBA1A

Dilution range WB 1:500-1:2000IHC 1:100-1:300ELISA 1:20000

Specificity Tubulin alpha Polyclonal Antibody detects endogenous levels of Tubulin

alpha protein.

Tissue Specificity Expressed at a high level in fetal brain.

Purification The antibody was affinity-purified from rabbit antiserum by affinity-

chromatography using epitope-specific immunogen.

Note For Research Use Only (RUO).

Protein Name Tubulin alpha-1A chain Alpha-tubulin 3 Tubulin B-alpha-1 Tubulin alpha-3

chain Detyrosinated tubulin alpha-1A chain

Molecular Weight 50 kDa

Clonality Polyclonal

Conjugation Unconjugated

Isotype IgG

Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Concentration 1 mg/ml

Storage Instruction Store at -20°C, and avoid repeat freeze-thaw cycles.

Database Links <u>HGNC:20766OMIM:602529</u>

Alternative Names Tubulin alpha-1A chain Alpha-tubulin 3 Tubulin B-alpha-1 Tubulin alpha-3

chain Detyrosinated tubulin alpha-1A chain

Function Tubulin is the major constituent of microtubules. It binds two moles of GTP,

one at an exchangeable site on the beta chain and one at a non-exchangeable

site on the alpha chain.

Cellular Localization Cytoplasm, cytoskeleton.

Post-translational Modifications Some glutamate residues at the C-terminus are polyglutamylated, resulting in polyglutamate chains on the gamma-carboxyl group. Polyglutamylation plays a key role in microtubule severing by spastin (SPAST). SPAST preferentially recognizes and acts on microtubules decorated with short polyglutamate tails: severing activity by SPAST increases as the number of glutamates per tubulin rises from one to eight, but decreases beyond this glutamylation threshold. Some glutamate residues at the C-terminus are monoglycylated but not polyglycylated due to the absence of functional TTLL10 in human. Monoglycylation is mainly limited to tubulin incorporated into axonemes (cilia and flagella). Both polyglutamylation and monoglycylation can coexist on the same protein on adjacent residues, and lowering glycylation levels increases polyglutamylation, and reciprocally. The precise function of monoglycylation is still unclear (Probable). Acetylation of alpha chains at Lys-40 is located inside the microtubule lumen. This modification has been correlated with increased microtubule stability, intracellular transport and ciliary assembly. Methylation of alpha chains at Lys-40 is found in mitotic microtubules and is required for normal mitosis and cytokinesis contributing to genomic stability. Nitration of Tyr-451 is irreversible and interferes with normal dynein intracellular distribution. Undergoes a tyrosination/detyrosination cycle, the cyclic removal and re-addition of a Cterminal tyrosine residue by the enzymes tubulin tyrosine carboxypeptidase (TTCP) and tubulin tyrosine ligase (TTL), respectively. Tubulin alpha-1A chain: Tyrosination promotes microtubule interaction with CAP-Gly domaincontaining proteins such as CLIP1, CLIP2 and DCTN1. Tyrosination regulates the initiation of dynein-dynactin motility via interaction with DCTN1, which brings the dynein-dynactin complex into contact with microtubules. In neurons, tyrosinated tubulins mediate the initiation of retrograde vesicle transport . Detyrosinated tubulin alpha-1A chain: Detyrosination is involved in metaphase plate congression by guiding chromosomes during mitosis: detyrosination promotes interaction with CENPE, promoting pole-proximal transport of chromosomes toward the equator. Detyrosination increases microtubules-dependent mechanotransduction in dystrophic cardiac and skeletal muscle. In cardiomyocytes, detyrosinated microtubules are required to resist to contractile compression during contraction: detyrosination promotes association with desmin (DES) at force-generating sarcomeres, leading to

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