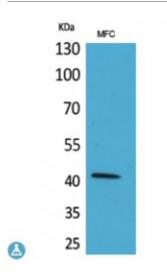


Anti-alpha-SMA antibody



Description Rabbit polyclonal to alpha-SMA.

Model STJ96485

Host Rabbit

Reactivity Human, Mouse, Rat

Applications ELISA, IHC, WB

Immunogen Synthesized peptide derived from human alpha-SMA.

Immunogen Region C-terminal

Gene ID <u>58</u>

Gene Symbol ACTA1

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

Specificity alpha-SMA Polyclonal Antibody detects endogenous levels of alpha-SMA

protein.

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

chromatography using epitope-specific immunogen.

Note For Research Use Only (RUO).

Protein Name Actin, alpha skeletal muscle Alpha-actin-1

Molecular Weight 45 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:1290MIM:102610</u>

Alternative Names Actin, alpha skeletal muscle Alpha-actin-1

Function Actins are highly conserved proteins that are involved in various types of cell

motility and are ubiquitously expressed in all eukaryotic cells.

Cellular Localization Cytoplasm, cytoskeleton.

Post-translational Oxidation of Met-46 and Met-49 by MICALs (MICAL1, MICAL2 or Modifications MICAL3) to form methionine sulfoxide promotes actin filament

depolymerization. MICAL1 and MICAL2 produce the (R)-S-oxide form. The (R)-S-oxide form is reverted by MSRB1 and MSRB2, which promote actin repolymerization . Monomethylation at Lys-86 (K84me1) regulates actin-myosin interaction and actomyosin-dependent processes. Demethylation by ALKBH4 is required for maintaining actomyosin dynamics supporting normal cleavage furrow ingression during cytokinesis and cell migration. (Microbial infection) Monomeric actin is cross-linked by V.cholerae toxins RtxA and VgrG1 in case of infection: bacterial toxins mediate the cross-link between Lys-52 of one monomer and Glu-272 of another actin monomer, resulting in formation of highly toxic actin oligomers that cause cell rounding . The toxin can be highly efficient at very low concentrations by acting on formin homology family proteins: toxic actin oligomers bind with high affinity to formins and adversely affect both nucleation and elongation abilities of formins, causing their potent inhibition in both profilin-dependent and independent manners .

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