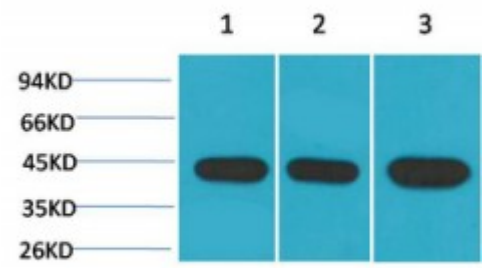
Anti-alpha-SMA antibody



Description	Mouse monoclonal to alpha-SMA.

Model	STJ97381
Host	Mouse
Reactivity	Human, Mouse, Rat
Applications	IHC, WB
Immunogen	Synthetic Peptide
Gene ID	59
Gene Symbol	ACTA2
Dilution range	WB 1:5000-50000IHC 1:1000-2000
Specificity	The antibody detects endogenous alpha-SMA protein.
Purification	The antibody was affinity-purified from mouse ascites by affinity-chromatography using epitope-specific immunogen.
Clone ID	1E+12
Note	For Research Use Only (RUO).
Protein Name	Actin, aortic smooth muscle Alpha-actin-2 Cell growth-inhibiting gene 46 protein Actin, aortic smooth muscle, intermediate form
Clonality	Monoclonal
Conjugation	Unconjugated
Isotype	IgG1

Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:130OMIM:102620
Alternative Names	Actin, aortic smooth muscle Alpha-actin-2 Cell growth-inhibiting gene 46 protein Actin, aortic smooth muscle, intermediate form
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 Modifications	Oxidation of Met-46 and Met-49 by MICALs (MICAL1, MICAL2 or 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 .