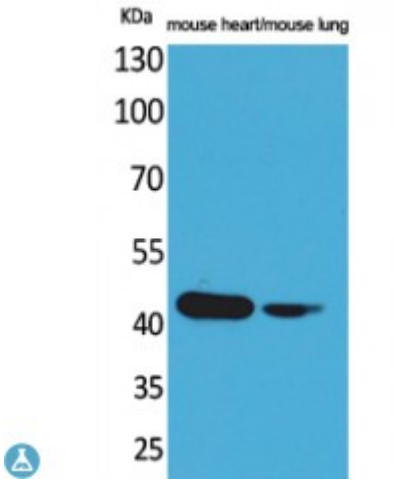
Anti-Actin-alpha cardiac muscle antibody



Description	Rabbit polyclonal to Actin-alpha cardiac muscle.

Model	STJ96475
Host	Rabbit
Reactivity	Human, Mouse, Rat
Applications	ELISA, IHC, WB
Immunogen	Synthesized peptide derived from human Actin-alpha cardiac muscle
Immunogen Region	1-80 aa, N-terminal
Gene ID	70
Gene Symbol	ACTC1
Dilution range	WB 1:500-1:2000IHC-P 1:100-300ELISA 1:20000
Specificity	Actin-alpha cardiac muscle Polyclonal Antibody detects endogenous levels of Actin-alpha cardiac muscle 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 cardiac muscle 1 Alpha-cardiac actin
Molecular Weight	42.019 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	HGNC:143OMIM:102540
Alternative Names	Actin, alpha cardiac muscle 1 Alpha-cardiac actin
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 .