

Anti-Caldesmon antibody



Description	Rabbit polyclonal to Caldesmon.
Model	STJ91973
Host	Rabbit
Reactivity	Human, Mouse, Rat
Applications	ELISA, IF, IHC, WB
Immunogen	Synthesized peptide derived from human Caldesmon around the non-phosphorylation site of S789.
Immunogen Region	730-810 aa
Gene ID	800
Gene Symbol	CALD1
Dilution range	WB 1:500-1:2000IHC 1:100-1:300IF 1:200-1:1000ELISA 1:5000
Specificity	Caldesmon Polyclonal Antibody detects endogenous levels of Caldesmon protein.
Tissue Specificity	High-molecular-weight caldesmon (isoform 1) is predominantly expressed in smooth muscles, whereas low-molecular-weight caldesmon (isoforms 2, 3, 4 and 5) are widely distributed in non-muscle tissues and cells. Not expressed in skeletal muscle or heart.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Note	For Research Use Only (RUO).
Protein Name	Caldesmon CDM

Molecular Weight	80 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:1441OMIM:114213
Alternative Names	Caldesmon CDM
Function	Actin- and myosin-binding protein implicated in the regulation of actomyosin interactions in smooth muscle and nonmuscle cells (could act as a bridge between myosin and actin filaments). Stimulates actin binding of tropomyosin which increases the stabilization of actin filament structure. In muscle tissues, inhibits the actomyosin ATPase by binding to F-actin. This inhibition is attenuated by calcium-calmodulin and is potentiated by tropomyosin. Interacts with actin, myosin, two molecules of tropomyosin and with calmodulin. Also play an essential role during cellular mitosis and receptor capping. Involved in Schwann cell migration during peripheral nerve regeneration .
Sequence and Domain Family	The N-terminal part seems to be a myosin/calmodulin-binding domain, and the C-terminal a tropomyosin/actin/calmodulin-binding domain. These two domains are separated by a central helical region in the smooth-muscle form.
Cellular Localization	Cytoplasm, cytoskeleton Cytoplasm, myofibril. On thin filaments in smooth muscle and on stress fibers in fibroblasts (nonmuscle).
Post-translational Modifications	In non-muscle cells, phosphorylation by CDK1 during mitosis causes caldesmon to dissociate from microfilaments. Phosphorylation reduces caldesmon binding to actin, myosin, and calmodulin as well as its inhibition of actomyosin ATPase activity. Phosphorylation also occurs in both quiescent and dividing smooth muscle cells with similar effects on the interaction with actin and calmodulin and on microfilaments reorganization. CDK1-mediated phosphorylation promotes Schwann cell migration during peripheral nerve regeneration .