10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Anti-Troponin I (cardiac) Ser23/24 Antibody

Our Anti-Troponin I (cardiac) Ser23/24 rabbit polyclonal phosphospecific primary antibody from Phosp
Catalog # AN1589

Product Information

Application	WB
Primary Accession	P48787
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	24259

Additional Information

Gene ID	21954
Other Names	cardiac muscle antibody, Cardiac troponin I antibody, Cardiac Troponin I antibody, cardiomyopathy dilated 2A (autosomal recessive) antibody, Cardiomyopathy familial hypertrophic 7 included antibody, CMD1FF antibody, CMD2A antibody, CMH7 antibody, cTnI antibody, Familial hypertrophic cardiomyopathy 7 antibody, MGC116817 antibody, RCM1 antibody, Tn1 antibody, Tni antibody, TNN I3 antibody, TNNC 1 antibody, TNNC1 antibody, TNNI3 antibody, TNNI3_HUMAN antibody, Troponin I antibody, Troponin I cardiac antibody, Troponin I cardiac muscle antibody, Troponin I cardiac muscle isoform antibody, Troponin I type 3 cardiac antibody, troponin I cardiac 3 antibody, TroponinI antibody, Troponin I type 3 (cardiac) antibody
Target/Specificity	Troponin I (cTnI) is 1 of 3 subunits, along with troponin C (TnC) and troponin T (TnT) of troponin complex found in cardiac muscle. cTnI binds to actin in thin myofilaments to hold the troponin-tropomyosin complex in place. Phosphorylation of cardiac isoform of TnI at serines 22,23 in the unique amino-terminal end molecule decreases the calcium sensitivity of the sarcomere, promotes calcium dissociation from troponin C and by extension enhances rates of cross-bridge cycling and diastolic relaxation (Noland, Jr. et al., 1995; Noland et al., 1989). In addition, studies using reconstituted fibers and mutational analysis have shown that PKC phosphorylation of TnI (largely at Ser-43) inhibits the actin-cross bridge reaction and reduces the Ca++ dependent actomyosin ATPase rate as well as the calcium sensitivity of force generation (Noland, Jr. and Kuo, 1991). Phosphorylation at Thr-144 (mediated by several PKC isoforms) reduces maximal tension development and cross-bridge cycling rates (Sumandea et al., 2008). Importantly, changes in the phosphorylation at each of these sites have been shown to be stage-specific with regard to cardiac disease progression (Walker et al., 2010).
Dilution	WB~~1:1000
Format	Antigen Affinity Purified from Pooled Serum

Storage	Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	Anti-Troponin I (cardiac) Ser23/24 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.
Shipping	Blue Ice

Background

Troponin I (cTnI) is 1 of 3 subunits, along with troponin C (TnC) and troponin T (TnT) of troponin complex found in cardiac muscle. cTnI binds to actin in thin myofilaments to hold the troponin-tropomyosin complex in place. Phosphorylation of cardiac isoform of TnI at serines 22,23 in the unique amino-terminal end molecule decreases the calcium sensitivity of the sarcomere, promotes calcium dissociation from troponin C and by extension enhances rates of cross-bridge cycling and diastolic relaxation (Noland, Jr. et al., 1995; Noland et al., 1989). In addition, studies using reconstituted fibers and mutational analysis have shown that PKC phosphorylation of TnI (largely at Ser-43) inhibits the actin-cross bridge reaction and reduces the Ca++ dependent actomyosin ATPase rate as well as the calcium sensitivity of force generation (Noland, Jr. and Kuo, 1991). Phosphorylation at Thr-144 (mediated by several PKC isoforms) reduces maximal tension development and cross-bridge cycling rates (Sumandea et al., 2008). Importantly, changes in the phosphorylation at each of these sites have been shown to be stage-specific with regard to cardiac disease progression (Walker et al., 2010).