

Anti-Troponin I (cardiac) Antibody

Our Troponin I (cardiac) rabbit polyclonal primary antibody from PhosphoSolutions is produced in-hou
Catalog # AN1588

Specification

Anti-Troponin I (cardiac) Antibody - Product Information

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

Anti-Troponin I (cardiac) Antibody - 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 isoform antibody, Troponin I type 3 cardiac antibody, troponin I cardiac 3 antibody, Troponin I 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).

Format

Neat 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) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

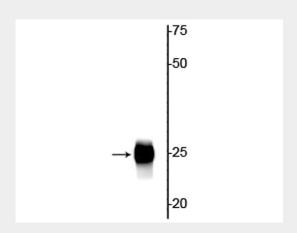
Shipping Blue Ice

Anti-Troponin I (cardiac) Antibody - Protocols

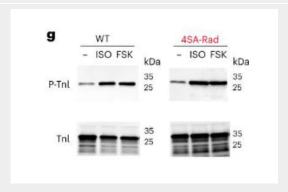
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Anti-Troponin I (cardiac) Antibody - Images

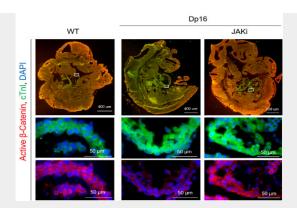


Western blot of 20 ug of mouse heart lysate showing specific immunolabeling of the ~25 kDa cardiac troponin I protein.



Western blots showing specific labeling of S23/S24 Tnl, cat. p2010-2324 (upper) and Tnl; cat. 2010-Tnl (lower) in protein lysates of mouse cardiomyocytes. Image from publication CC-BY-4.0. PMID: 36424916





Examination of cardiac troponin I (cTnI) (cat. 2010-TNI, green) and active β -Catenin (red) levels in embryos at E9.5 by immunofluorescence (IF) analysis. Pregnant mice were treated daily with vehicle or the JAKi (10 mg/kg body weight/day, i.p.) beginning with day 6.5 post-conception. Embryos were harvested at E9.5 for IF analysis. Image from publication. CC-BY-4.0

Anti-Troponin I (cardiac) Antibody - 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).