Genorise® Recombinant Canine Cardiac Troponin T Protein DataSheet

Catalog #: GR122011

Background

Cardiac Troponin T (TnT), is a protein which is encoded by the *TNNT2* gene.[1] Cardiac TnT is the tropomyosin-binding subunit of the troponin complex, which is located on the thin filament of striated muscles and regulates muscle contraction in response to alterations in intracellular calcium ion concentration. Cardiac TnT is a 35.9 kDa protein composed of 298 amino acids.[2] Cardiac TnT is the largest of the three troponin subunits (cTnT, troponin I (TnI), troponin C (TnC)) on the actin thin filament of cardiac muscle. The structure of TnT is asymmetric; the globular C-terminal domain interacts with tropomyosin (Tm), TnI and TnC, and the N-terminal tether which strongly binds Tm. The N-terminal region of TnT is alternatively spliced, accounting for multiple isoforms observed in cardiac muscle.[3] As part of the Troponin complex, the function of cTnT is to regulate muscle contraction. The N-terminal region of TnT that strongly binds actin most likely moves with Tm and actin during strong myosin crossbridge binding and force generation. This region is likely involved in the transduction of cooperativity down the thin filament. The C-terminal region of TnT constitutes part of the globular troponin complex domain, and participates in employing the calcium sensitivity of strong myosin crossbridge binding to the thin filament. Mutations in this gene have been associated with familial hypertrophic cardiomyopathy as well as with restrictive[4] and dilated cardiomyopathy. Transcripts for this gene undergo alternative splicing that results in many tissue-specific isoforms, however, the full-length nature of some of these variants has not yet been determined. Mutations of this gene may be associated with mild or absent hypertrophy and predominant restrictive disease, with a high risk of sudden cardiac death.[4] Advancement to dilated cardiomyopathy may be more rapid in patients with TNNT2 mutations than in those with myosin heavy chain mutations.[5]

References

1. Townsend PJ, et al. (1994). *Genomics* **21** (2): 311–6.
2. Zong, N. C. et al. (2013). *Circulation Research* **113** (9): 1043–53.
3. Anderson PA, et al (1991). *Circulation Research* **69** (5).
4. Revera M, et al. (2007). *Cardiovascular Journal of Africa* **18** (3): 146–53.
5. Fujino N, et al. (2002). *The American Journal of Cardiology* **89** (1): 29–33.

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Description

Source: *E coli* derived
Components: Met1–Lys275
Accession # A0A8I3PBC8
Predicted Molecular Mass: 31 kDa

Specifications

SDS-PAGE: 43 kDa, reducing conditions
Purity: >95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation: Lyophilized from a 0.2 μm filtered PBS with BSA as carrier protein.

Preparation and Storage

Reconstitution: Reconstitute at 50-500 μg/mL in sterile PBS with 0.1% BSA.
Shipping: The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage: Use a manual defrost freezer and avoid repeated freeze thaw cycles.

- 6 months from date of receipt, -20 to -70°C as supplied.
- 3 months, -20 to -70°C under sterile conditions after reconstitution.

DECLARATION

THIS REAGENT IS FOR IN VITRO LABORATORY TESTING AND RESEARCH USE ONLY. DO NOT USE IT FOR CLINICAL DIAGNOSTICS. DO NOT USE OR INJECT IT IN HUMANS AND ANIMALS.

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