**Human Cardiac Troponin I peptide ab47002**

### Description

<table>
<thead>
<tr>
<th>Product name</th>
<th>Human Cardiac Troponin I peptide</th>
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<tbody>
<tr>
<td>Animal free</td>
<td>No</td>
</tr>
<tr>
<td>Nature</td>
<td>Synthetic</td>
</tr>
<tr>
<td>Species</td>
<td>Human</td>
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### Specifications

Our Abpromise guarantee covers the use of ab47002 in the following tested applications. The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

*Applications*  Blocking - Blocking peptide for Anti-Cardiac Troponin I antibody (ab47003)

*Form*  Liquid

*Additional notes*
- First try to dissolve a small amount of peptide in either water or buffer. The more charged residues on a peptide, the more soluble it is in aqueous solutions.
- If the peptide doesn't dissolve try an organic solvent e.g. DMSO, then dilute using water or buffer.
- Consider that any solvent used must be compatible with your assay. If a peptide does not dissolve and you need to recover it, lyophilise to remove the solvent.
- Gentle warming and sonication can effectively aid peptide solubilisation. If the solution is cloudy or has gelled the peptide may be in suspension rather than solubilised.
- Peptides containing cysteine are easily oxidised, so should be prepared in solution just prior to use.

### Preparation and Storage

*Stability and Storage*  Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles. Information available upon request.

### General Info

*Function*  Troponin I is the inhibitory subunit of troponin, the thin filament regulatory complex which confers calcium-sensitivity to striated muscle actomyosin ATPase activity.
Involvement in disease

Defects in TNNI3 are the cause of cardiomyopathy familial hypertrophic type 7 (CMH7) [MIM:613690]. Familial hypertrophic cardiomyopathy is a hereditary heart disorder characterized by ventricular hypertrophy, which is usually asymmetric and often involves the interventricular septum. The symptoms include dyspnea, syncope, collapse, palpitations, and chest pain. They can be readily provoked by exercise. The disorder has inter- and intrafamilial variability ranging from benign to malignant forms with high risk of cardiac failure and sudden cardiac death.

Defects in TNNI3 are the cause of cardiomyopathy familial restrictive type 1 (RCM1) [MIM:115210]. RCM1 is an heart muscle disorder characterized by impaired filling of the ventricles with reduced diastolic volume, in the presence of normal or near normal wall thickness and systolic function.

Defects in TNNI3 are the cause of cardiomyopathy dilated type 2A (CMD2A) [MIM:611880]. Dilated cardiomyopathy is a disorder characterized by ventricular dilation and impaired systolic function, resulting in congestive heart failure and arrhythmia. Patients are at risk of premature death.

Defects in TNNI3 are the cause of cardiomyopathy dilated type 1FF (CMD1FF) [MIM:613286]. Dilated cardiomyopathy is a disorder characterized by ventricular dilation and impaired systolic function, resulting in congestive heart failure and arrhythmia. Patients are at risk of premature death.

Sequence similarities

Belongs to the troponin I family.

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