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Exercise-induced cardiac troponin release: do we need to worry?

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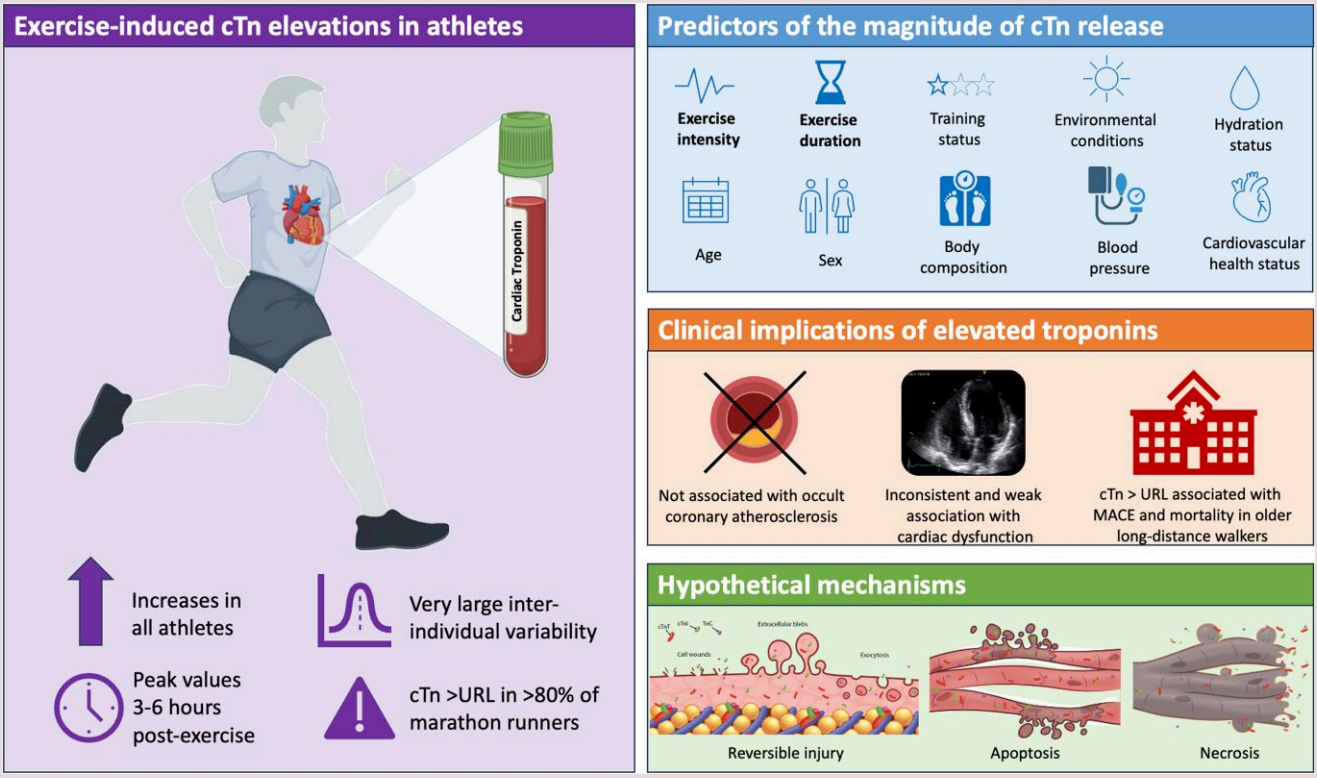
Graphical Abstract



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Cardiac troponins (cTn), either T (cTnT) or I (cTnI), are the preferred biomarkers for the diagnosis of myocardial infarction because of their excellent diagnostic performance. The detection of a typical rise and fall pattern is integrated in accelerated diagnostic pathways using high-sensitivity assays to triage patients with chest pain in the Emergency Department. The interpretation of elevated cTn concentrations in athletes can, however, be complicated as recent engagement in endurance exercise also produces transient cTn elevations. For example, marathon runners have a median hs-cTnT and hs-cTnI concentration of 30–50 ng/L post-race, with >80% of athletes having concentrations that exceed the diagnostic cutoffs.[1] This raises the question whether exercise-induced cTn elevations are a physiological or pathophysiological phenomenon, or maybe a combination thereof.

cTn concentrations typically peak at 3–6 h after exercise and show considerable inter-individual variation between athletes. Previous studies have identified personal and exercise-related factors that are associated with the magnitude of exercise-induced cTn elevations, including age, sex, body composition, cardiovascular health status, blood pressure, training experience, exercise intensity and exercise duration.[1] The intensity and duration of exercise are the strongest predictors, reflecting the magnitude of the exercise-induced workload of the heart. Nevertheless, the predictive value of these joint factors together remained low, only explaining around a third of post-exercise cTn concentrations.

There is ongoing debate as to the clinical relevance of exercise-induced cTn elevations. Data from population-based studies have shown that minor cTn elevations have predictive value for adverse health outcomes. A higher post-exercise cTn concentration may, therefore, be indicative of subclinical disease and/or cardiac vulnerability. Recently, the TREAT study showed no differences in coronary artery disease between athletes with very high vs. very low post-exercise cTn concentrations.[2] Other studies reported inconsistent but weak associations between post-exercise cTn concentrations and cardiac dysfunction.[1] Finally, a prospective study among older long-distance walkers found that post-exercise cTn concentrations above the upper reference limit (URL) were independently predictive of the composite outcome of major adverse cardiovascular events and mortality (Hazard Ratio: 2.5, 95% confidence interval: 1.3–4.8).[3] The high age [61 (54–69) years] and prevalence of cardiovascular risk factors and diseases (40%) of the study population are, however, different from the typical endurance athlete, highlighting the need for other studies to confirm or challenge this observation.

It is thought that the mechanisms of cTn release can be due to (i) reversible injury attributable to cell wounds, cytoplasmic blebbing, or extracellular vesicle release; (ii) apoptosis; and (iii) myocardial necrosis.[4] Only few studies explored the potential cTn release mechanisms in athletes. In one study, exercise led to compromised cardiomyocyte integrity as shown by cardiac magnetic resonance, and the magnitude of this effect was positively associated with post-exercise cTn concentrations.[5] These findings suggest that degraded cTn forms may escape from the cytosol into the circulation,[6] possibly reflecting a form of reversible injury. Nevertheless, the presence of some irreversible injury cannot be ruled out as contemporary MRI protocols have insufficient spatial resolution to quantify acute micro-injury. Further mechanistic studies are warranted.

Whether athletes and physicians 'can run away' from elevated post-exercise cTn concentrations depends on the clinical context and whether this is accompanied by possible cardiac symptoms. The general observation that endurance exercise produces cTn elevations in all athletes and that it might be caused by leakage of small cTn forms into the circulation, offers some reassurance in those without symptoms. However, prospective data on cTn and clinical outcomes in patients and in the general population consistently show positive associations between cTn concentrations and the risk of adverse health outcomes. Future exercise studies, especially in young and low-risk athletes with long-term follow-up on health outcomes, should point out what exactly to do in whom.

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Data availability

No data were generated or analysed for this manuscript.

Appendix

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