Prevalence and Predictors of Cardiac Troponin Elevations Following Exercise: a Systematic Review, Meta-analysis, and Meta-regression

RUNNING TITLE: Exercise and Cardiac Troponins

Jesse O. Stekelenburg[a,b *]

Kristian Berge[c,d *]

Sylvan L.J.E. Janssen[a]

Torbjørn Omland[c,d]

Peder L. Myhre[d,e]

Paul D. Thompson[f]

Vincent L. Aengevaeren[a,g]

Thijs M.H. Eijsvogels[a]

[*]Equal author contributions

Affiliations:

[a]Department of Medical BioSciences, Exercise Physiology research group, Radboud university medical center, Nijmegen, The Netherlands.

[b]Department of Circulation and Medical Imaging, Faculty of Medicine and Health Sciences, Norwegian University of Science and Technology, Trondheim, Norway.

[c]Department of Cardiology, Akershus University Hospital, Lørenskog, Norway.

[d]K.G. Jebsen Center for Cardiac Biomarkers, Institute of Clinical Medicine, University of Oslo, Oslo, Norway.

[e]University of Oslo, Institute of Clinical Medicine, Oslo, Norway.

[f]Department of Cardiology, Hartford Hospital, Hartford, Connecticut, USA.

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[g]Department of Cardiology, Radboud University Medical Center, Nijmegen, The Netherlands.

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Address for correspondence:

Dr Thijs Eijsvogels, Department of Medical BioSciences (928), Radboud University Medical Center, P.O. Box 9101, 6500 HB Nijmegen, The Netherlands, Tel +31 24 36 13 674, Fax +31 24 36 16413, E-mail: Thijs.Eijsvogels@radboudumc.nl

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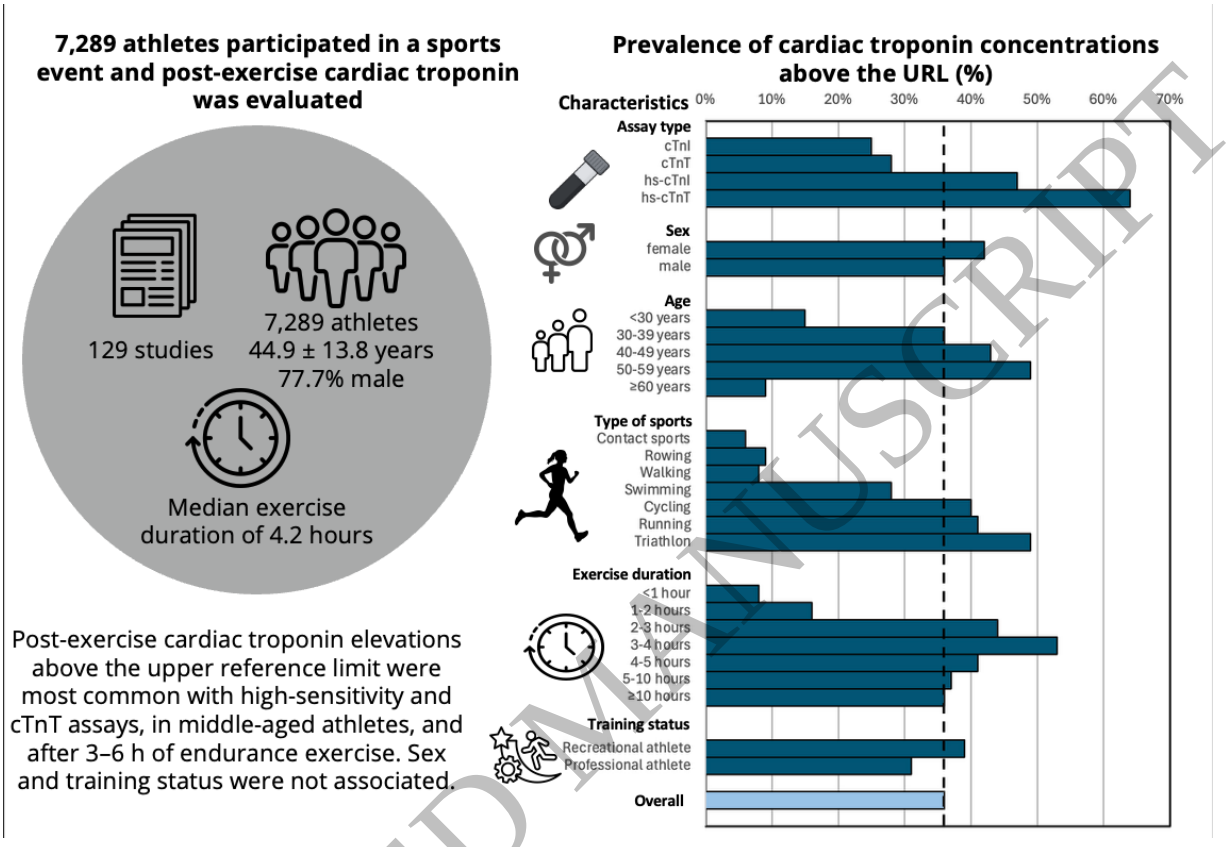

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ABSTRACT

Aims: Myocardial injury is defined by cardiac troponin (cTn) elevations exceeding the upper reference limit (URL) and serves as a diagnostic criterion for myocardial infarction. However, exercise can also elevate cTn, complicating interpretation. We aimed to determine the prevalence and predictors of cTn elevations following exercise.

Methods: We conducted a random-effects meta-analysis of post-exercise cTn elevations above the assay-specific URL. Associations between prespecified covariates and the prevalence of elevated post-exercise cTn were assessed using multilevel mixed-effects meta-regression and cubic spline models.

Results: We identified 4,312 articles and included 129. The cohort consisted of 7,289 athletes (78% male; mean age 44.9 years; median exercise duration 4.2 hours). Overall, 36% (95%CI 31–42%) of athletes demonstrated cTn concentrations above the URL following exercise, with extreme between-study heterogeneity (range 2–98%). The prevalence was higher with high-sensitivity *versus* non-high-sensitivity assays (58% *versus* 26%), cTnT *versus* cTnI assays (45% *versus* 30%), and in middle-aged individuals. Sex and training status were not associated with elevations. Endurance sports, including triathlon (49%), cycling (40%), and running (41%), showed the highest prevalence, while contact sports (6%) and walking (8%) showed the lowest. The prevalence was non-linearly associated with exercise duration, with a peak (52%) after 3–6 hours of exercise.

Conclusions: cTn elevations are common following exercise, most often with high-sensitivity assays and cTnT measurements, particularly in endurance sports lasting 3–6 hours. Clinicians should consider assay type and exercise characteristics rather than sex and training status when evaluating post-exercise cTn elevations in athletes.

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Lay summary

Cardiac troponins are blood biomarkers that are used to assess the presence of damage to the heart. Exercise can raise cardiac troponin levels in recreational athletes without symptoms of heart problems. We aimed to determine the prevalence and predictors of cardiac troponin elevations following exercise, and present the following findings:

- Exercise leads to cardiac troponin elevations with abnormal values in more than one in three athletes.
- These elevations were most common with high-sensitivity assays, cTnT measurements, in middle aged athletes, and after endurance sports with a duration of 3-6 hours.
- Sex and training status did not influence the likelihood of post-exercise troponin elevations.

PROSPERO registration number: CRD42023384920

Keywords: Myocardial injury, cardiac troponin, athletes, endurance exercise, biomarkers, sports.

INTRODUCTION

Cardiac troponins (cTn) play a key role in cardiomyocyte contraction, and circulating concentrations remain low under normal conditions.[1] However, cTn elevations — which are defined as concentrations above the 99th percentile upper reference limit (URL) — are diagnostic for myocardial injury and, if combined with evidence of acute myocardial ischemia, for acute myocardial infarction.[2] Exercise can also cause transient increases in cTn concentrations, which may lead to diagnostic ambiguity in individuals evaluated for suspected myocardial infarction or other cardiovascular diseases linked to myocardial injury in the post-exercise setting.[3] Recent studies have identified associations between exercise-induced cTn elevations,

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occult coronary artery disease,[4] and adverse cardiovascular events,[5] suggesting that such elevations in some circumstances may reflect subclinical cardiovascular disease. A better understanding of the prevalence and predictors of exercise-induced cTn elevations is therefore warranted.

Previous studies have reported a wide range of predictors of exercise-induced cTn release, including exercise intensity, exercise duration, type of exercise, age, sex, body composition, blood pressure, and the presence of cardiovascular diseases.[6,7] However, it remains largely unknown how these factors are related to the prevalence of cTn elevations. Previous meta-analyses indicated that 46–70% of athletes exhibited cTn elevations post-exercise; yet studies included in these analyses only focused on runners,[8,9] lacked direct comparison of cardiac troponin isoforms,[9-11] or did not harmonize between different assay types.[12] cTnT and cTnI differ in their biochemical properties[13] and post-exercise kinetics.[14] Stratifying these isoforms may therefore reveal meaningful differences in post-exercise cTn elevations. Recent research has measured post-exercise cTn concentrations across various sports,[5,15-19] with better registration of exercise characteristics and utilizing newer, more accurate high-sensitivity (hs) cTn assays. These emerging insights are key to improving our understanding of cTn elevations following exercise.

This meta-analysis aimed to determine the prevalence of cTn elevations after exercise and to identify predictors of such elevations. To our knowledge, this is the largest comprehensive systematic review and meta-analysis on cTn elevations following exercise and the first to include and categorize both cTnT and cTnI, as well as to distinguish between hs and non-hs assays.

METHODS

This systematic review and meta-analysis was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA)[20] and Meta-analysis of Observational Studies in

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Epidemiology (MOOSE) checklist.[21] The protocol of this meta-analysis was registered in the international Prospective Register of Systematic Reviews (PROSPERO; CRD42023384920).

Data sources and search strategy

A systematic literature search was conducted using the search terms and variations of "Troponin" and "Exercise" alone and in combination (**Supplemental Table 1**) across the PubMed, Embase (Ovid), and SPORTDiscus databases from their inception until December 2023. We also searched the reference lists of original articles and other systematic reviews for potentially relevant citations to ensure that we did not miss relevant studies. Prior to screening, duplicates were identified and removed using a de-duplication method by Bramer *et al.*[22] Study selection was performed independently by two researchers (J.O.S. and K.B.) using Covidence systematic review software (Veritas Health Innovation, Melbourne, Australia).[23] Potential publications were screened based on titles and abstracts before full-text articles were retrieved and reviewed. Disagreements and discrepancies were resolved by consensus or by adjudication by a third researcher (T.M.H.E.).

Study selection

We included studies that measured cTn in athletes following exercise. Studies were eligible if: (1) exercise was performed under competitive conditions, and (2) exercise consisted of an endurance component lasting at least 10 minutes, including mixed modality exercise, and (3) blood samples were collected within 12 hours post-exercise. Studies were excluded if they: (1) did not report the prevalence of cTn elevations, (2) were non-original research or previously published cohorts, (3) included fewer than 10 athletes or participants younger than 18 years, or (4) were not published in English.

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Quality assessment and risk of bias

The risk of bias was assessed by J.O.S. and K.B using a modified Newcastle-Ottawa Scale (NOS)[24] (**Supplemental File 1**). In case of discrepancies, consensus was reached by consulting a third researcher (T.M.H.E.). Studies were scored on (1) cohort representativeness, (2) exposure assessment, and (3) outcomes, using a 0- to 10-point scale, where 0–3 indicated high, 4–7 intermediate, and 8–10 low risk of bias.

Data extraction

Data were extracted by J.O.S. and K.B and recorded in a standardized form, including the prevalence of cTn elevations above the assay-specific 99th percentile URL, assay characteristics (non-hs *or* hs), cTn isoform (cTnI *or* cTnT), age, sex, body mass index (BMI), history of cardiovascular disease, training status, sport type, duration of exercise, timing of blood sampling after exercise cessation, and data for risk of bias assessment. The classification of athletes as recreational or professional/highly trained according to the definitions and terminology used in was derived from the reporting in the original studies, as no individual participant data on peak oxygen consumption, years of practice or competition level reached was available to conduct a harmonized approach. In studies where the prevalence of cTn elevations was presented solely in graphical format, data were extracted from the figures using WebPlotDigitizer (version 4.7, Automeris).[25] In studies with missing data, the corresponding authors were contacted via e-mail. A unique cohort was defined as a unique group of participants within a study (e.g., different sports events or participant groups). For studies reporting prevalences of cTn elevations for more than one cTn assay within the same unique cohort, each assay-specific prevalence was included as a separate assay cohort in the meta-analysis.

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Statistical analyses

A meta-analysis was conducted using a random-effects model to account for heterogeneity between studies. Restricted maximum likelihood (REML) was used to estimate between-study variance, due to high heterogeneity in prevalence of cTn elevations and study sample sizes.[26] Publication bias was assessed by visually inspecting funnel plots for asymmetry, and small-study effects were formally tested using Egger's test. Study-level prevalences were transformed using the Freeman–Tukey double-arcsine transformation prior to pooling, which stabilizes variance and ensures valid confidence interval estimation for prevalences near 0 or 1. To account for the non-independence of estimates from cohorts contributing data across multiple assay types, prevalences of cTn elevations and associations with covariates were estimated using multilevel mixed-effects meta-analysis, with cohort as the clustering variable. Crude overall and subgroup prevalences were illustrated with forest plots, with 95% confidence intervals (CIs) calculated on the transformed scale and back-transformed to the prevalence metric for presentation. The 95% prediction interval for the overall pooled estimate was calculated to characterize the expected range of prevalences across different sport contexts. Covariates were selected *a priori* based on previous studies identifying them as key determinants of exercise-induced cardiac troponin release.[6] Wald's test was used to assess differences in Freeman–Tukey transformed prevalences between subgroups and to test associations between covariates and prevalence. For exercise duration and age, both linear and non-linear (restricted cubic spline models using 3–5 knots) associations were evaluated, and the best model fit was assessed based on the Akaike information criterion, and models were compared using the likelihood-ratio test.[27] Multivariable models were adjusted for cTn assay generation (non-hs *versus* hs), cTn isoform (I *versus* T), baseline prevalence of cTn elevations, and time-to-sampling after exercise cessation. To generate comparable estimates across all included studies, median and interquartile ranges were converted to mean and standard deviation using a recommended formula.[28] All statistical analyses were performed using Stata

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Software (version 18, Stata Corp., College Station, TX, USA). A two-sided P-value of <0.05 was considered statistically significant.

Sensitivity analysis

A sensitivity analysis was performed by restricting the meta-analysis to cohorts excluding athletes with known cardiovascular disease to assess whether underlying cardiovascular disease influenced the prevalence of post-exercise cTn elevations. In addition, a sensitivity analysis was performed excluding cohorts from studies with a high risk of bias.

RESULTS

After removing duplicates, the search identified 4,312 studies that were potentially eligible for inclusion in the meta-analysis. Three additional references were identified through snowballing.[29-31] Following title and abstract screening, 269 full-text articles were evaluated. Three studies could not be retrieved,[32-34] and 137 studies were excluded. This resulted in a final inclusion of 129 studies (**Figure 1**).

Study and population characteristics

Of the 129 studies, 33 (26%) reported prevalences of elevated troponins for more than one athlete cohort, resulting in 193 unique cohorts. Among these cohorts, 19 reported prevalences for more than one assay type within the same cohort, yielding a total of 218 prevalence estimates. Among these, hs-cTnI was reported in 24 cohorts (11%), cTnI in 82 cohorts (38%), hs-cTnT in 54 cohorts (25%), and cTnT in 58 cohorts (27%). Study sizes ranged from 10 to 1,002 athletes (**Supplemental Table 2**). The meta-analysis included 7,289 unique athletes with a mean age of 44.9 ± 13.8 years, of whom 77.7% were males, and a mean BMI

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of 24.2 ± 3.0 kg/m^2. Athletes with cardiovascular diseases were excluded in 123 assay cohorts, included in 38, and not reported in 57. A total of 10,061 post-exercise cTn measurements were assessed, of which 2,880 (28.6%) were measured using a hs-cTnT assay, 1,727 (17.2%) with a hs-cTnI assay, 2,282 (22.7%) with a cTnT assay, and 3,172 (31.5%) with a cTnI assay. The types of exercise studied included running (n=121), cycling (n=21), triathlon (n=12), contact sports (n=11), long-distance walking (n=8), swimming (n=3), and rowing (n=2). Among the 92 studies reporting exercise duration, the median exercise duration was 4.2 [IQR 2.3–9.0] hours, ranging from 10 minutes to 48 hours. Blood samples were collected within one hour after exercise cessation in 93.5% of cases, whereas the remaining studies collected samples ranging from 1.7 to 4 hours post-exercise.

Quality assessment

An overview of the quality scores for each study is presented in **Supplemental Table 3**. Out of the 218 assay cohorts, 63 (28.9%) were rated as having a low risk for bias, 143 (65.6%) an intermediate risk, and 12 (5.5%) a high risk of bias. The minimum and maximum scores were 3 and 10 points, respectively. Assessment of publication bias for the association between exercise exposure and prevalence of cTn elevations showed a symmetrical distribution in the funnel plot (**Supplemental Figure 1**), and Egger's test did not indicate significant small-study effects (p = 0.12), suggesting minimal publication bias.

Prevalence of cTn elevations

The prevalence of cTn elevations under resting conditions (n=179 assay cohorts) was 2.8% (95%CI 2.0–3.6%). Post-exercise, the overall prevalence was 36.2% (95%CI 30.6–41.7%) (**Figure 2**), ranging from 0%

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(n=29 assays) to 100% (n=11 assays) at the individual study level (**Supplemental Table 2**), resulting in a wide prediction interval ranging from 1.7–97.7%.

The type of assay had a significant impact on the prevalence of cTn elevations, with hs-assays detecting cTn elevations more frequently than non-hs-assays (57.8% vs 26.0%, p<0.001), and cTnT detecting elevations more frequently than cTnI (44.6% vs 29.8%, p<0.001). These differences remained highly significant after adjustment for pre-exercise prevalence of cTn elevations (p<0.001 for high-sensitivity vs conventional assays and p<0.003 for troponin I vs troponin T assays). The respective prevalences across assay types were 25.1% (95%CI 18.8–31.5%) for non-hs-cTnI, 28.4% (95%CI 20.0–36.8%) for non-hs-cTnT, 47.3% (95%CI 32.7–61.8%) for hs-cTnI, and 63.8% (95%CI 51.6–75.9%) for hs-cTnT (**Figure 2**).

There were no significant differences in cTn elevations between assay cohorts composed predominantly (>50% of athletes) of females (n=25) (41.9% [95%CI 24.0–59.7%]) vs males (n=167) (35.7% [95%CI 29.4–42.0%]) in unadjusted analysis (p=0.50) or after adjustment (p=0.45).

The prevalence of post-exercise cTn elevations varied significantly across age groups (p<0.001), with the youngest and oldest athletes showing the lowest prevalences (**Figure 2**). This pattern remained statistically significant in our adjusted analysis (p<0.001). When age was modelled as a continuous variable, a significant non-linear association was observed (overall p=0.014, p for non-linearity=0.006), with prevalence peaking in athletes aged approximately 40–50 years and declining thereafter (**Figure 3**). This association remained statistically significant in fully adjusted analysis of cTn elevations (p=0.004).

No significant difference was found between studies of professional/highly trained athletes (31.1% [95%CI 22.4–39.7%]) and recreational athletes (38.6% [95%CI 30.2–47.0%]) in unadjusted (p=0.23) or adjusted analysis (p=0.34).

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The type and duration of sport influenced the prevalence of post-exercise cTn elevations (**Figure 2**). Endurance sports were associated with the highest prevalences, 49.4% (95%CI 25.7–73.0%) for triathlon, 40.4% (95%CI 25.9–54.8%) for cycling, and 41.4% (95%CI 34.5–48.5%) for running. Contact sports (6.1% [95%CI 0.5–11.7%]) and long-distance walking (7.6% [95%CI 4.9–10.2%] reported lower prevalences. Prevalences for sports represented by fewer than five unique cohorts were 8.6% (95%CI 0.0–18.1%) for rowing (n=2) and 27.8% (95%CI 0.0–65.6%) for swimming (n=3). These differences were statistically significant across sport categories in adjusted analyses (p=0.003). There was a non-linear relationship between exercise duration and prevalence of cTn elevations (p for non-linearity <0.001), and the best model fit was a restricted cubic spline model with four knots. Based on this model, the highest prevalence of cTn elevations (~52%) was observed after exercise bouts lasting 4–5 hours, and gradually declining with longer exercise durations (**Figure 4**). Higher prevalences were observed for cTnT versus cTnI across the full exercise duration spectrum (**Supplemental Figure 4**). Peak prevalence reached 92.0% (95%CI 90.6–93.3%) in the subset of studies using hs-cTnT assays with exercise duration of 3–6 hours (n=9) (**Supplemental Figure 5**).

A sensitivity analysis restricted to assay cohorts excluding athletes with cardiovascular disease yielded similar prevalence estimates of cTn elevations as the main analysis (**Supplemental Figure 2**), and exclusion of assay cohorts from studies with a high risk of bias did not change the prevalence estimates (**Supplemental Figure 3**).

DISCUSSION

This systematic review and meta-analysis of 129 studies, including >7,000 athletes and >10,000 evaluations of post-exercise cTn concentrations, assessed the prevalence of cTn elevations above the 99th percentile URL following exercise, and examined subject characteristics most frequently associated with cTn

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elevations (**Graphical Abstract**). We found that 36% of participants exhibited elevated cTn concentrations post-exercise. The prevalence of cTn elevations was higher with hs-assays than non-hs assays, with cTnT than cTnI, and in middle-aged individuals. A non-linear association was observed between exercise duration and the prevalence of elevated post-exercise cTn concentrations, with the highest rates occurring after 3–6 hours of exercise. Individuals engaging in endurance sports more frequently exhibited elevated cTn concentrations, with prevalences reaching up to 93% when measured using an hs-cTnT assay following exercise bouts lasting 3–6 hours. Findings from this meta-analysis highlight that cTn concentrations are frequently elevated following exercise and emphasize the influence of assay type and exercise characteristics, factors that could improve the clinical interpretation of cTn elevations in athletes.

Interpretation of prevalence estimates

The prevalence of cTn elevations was 36%, which is lower than the findings of previous meta-analyses ranging from 46–70%.[8-12] This discrepancy is likely attributable to the broader scope of our approach, as we included all sport types rather than focusing solely on endurance sports,[8-11] and included studies using both non-hs and hs cTn assays.[8,9,11] Accordingly, we were able to determine the prevalence across distinct strata, allowing for more accurate estimates for the expected prevalence of cTn elevations for specific settings (i.e., dependent on assay type and exercise characteristics). It is important to highlight that prevalence estimates varied widely across study contexts, ranging from near zero in cohorts using conventional assays during lower-intensity exercise to more than 90% in cohorts using hs-cTnT assays following prolonged endurance exercise. These findings indicate that the pooled prevalence should be interpreted as an average across heterogeneous settings rather than as a typical or generalizable estimate. Subgroup- and context-specific estimates may be more clinically informative for the interpretation of post-exercise cTn elevations.

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Impact of troponin isoform and assay characteristics

cTn elevations were more prevalent with cTnT than cTnI. The difference between cTn isoforms may relate to the 99th percentile URL applied according to the manufacturers' package inserts. Data from the Universal Sample Bank indicated that the URLs are not equivalent across assays and should be increased for cTnT and decreased for cTnI,[35] which could attenuate the observed difference between assay types. Alternatively, the presence of troponin isoforms within the early releasable pool, i.e. cTn fragments located in the cytosol or loosely bound to the sarcomere of cardiomyocytes, is smaller for cTnI (2–4% of total cTn) than cTnT (6–8% of total cTn).[13] Exercise-induced increases in cardiomyocyte membrane permeability[36] may accordingly allow greater leakage of unbound cTnT versus cTnI from the early releasable pool into the circulation.[37,38] Another possible explanation is that cTnT assays are more prone to cross-reactivity with skeletal muscle troponin T than cTnI assays.[39,40] As exercise may cause skeletal muscle injury, part of the observed increase in post-exercise cTnT concentrations could theoretically originate from a non-cardiac source (i.e., skeletal muscle). It is important to emphasize, however, that the contribution of such cross-reactivity to post-exercise cTn concentrations is believed to be limited, if any.[6,41] Finally, the higher prevalence of cTn elevations in hs- *versus* non-hs-assays may be related to their greater precision and higher sensitivity, permitting the establishment of a 99th percentile URL and jointly leading to a better discrimination between normal and elevated values.[42] Taken together, these observations underscore the importance of assay type when interpreting post-exercise cTn concentrations.

Impact of exercise characteristics

The type of exercise influenced the prevalence of post-exercise cTn elevations. High prevalences were observed in cyclists, runners, and triathletes, whereas lower prevalences were found in contact sports, swimmers, and long-distance walkers. Sport-specific differences in exercise intensity and duration likely

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underlie these findings, as these exercise characteristics are well-recognized contributors to the magnitude of exercise-induced cTn elevations.[5,6,43] This was elegantly illustrated by a study randomizing 15 athletes to perform 60 minutes of maximal exertion in running, cycling, and swimming.[44] No differences in post-exercise hs-cTnT concentrations were found across sport disciplines, suggesting that exercise type serves as a proxy for intensity, with comparable cTn elevations observed at peak effort given similar duration. The relatively low prevalence of elevated cTn concentrations among rowers and swimmers, was likely attributable to their relatively shorter exercise duration,[44-47] whereas the lower prevalence among long-distance walkers likely reflects a lower exercise intensity,[5,48-51] compared to other endurance sports such as running.[43]

Exercise exposure for 3 to 6 hours was associated with the highest prevalence of cTn elevations. These findings align with previous observations that increasing running distance, and thus exercise time, correlates with higher cTn concentrations.[50,52] Transitioning from marathon distance to ultra-endurance events appears to result in a lower prevalence of post-exercise cTn elevations,[53-55] but these estimates should be interpreted with caution given the smaller number of contributing cohorts in extreme exercise settings. Taken together, the exercise-induced increase in cardiac workload, defined by the interplay of exercise intensity and exercise duration, is likely the key driver of the magnitude of cTn elevations as exercise modalities such as marathon running or mountain cycling produced the highest prevalence of cTn elevations.[43,56]

Impact of athlete characteristics

Sex and training status did not significantly influence the prevalence of post-exercise cTn elevations. While this may appear inconsistent with previous studies,[6] heterogeneity in the definitions of training status, limited use of sex-specific URLs, and residual confounding by exercise characteristics may have attenuated

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detectable associations. Alternatively, exercise characteristics such as exercise intensity and exercise duration are recognized as the strongest predictors of the magnitude of exercise-induced cTn elevations,[6,57] and may outperform athlete characteristics. Future studies with pooled individual athlete-level data are therefore warranted, as present analyses only evaluated the role of sex and exercise characteristics using cohort level data (i.e. >50% male/female, average exercise intensity and duration).

We found a non-linear association between age and post-exercise cTn elevations, with the highest prevalence in middle-aged individuals. These findings align with previous studies identifying age as a significant predictor for exercise-induced cTn elevations. A potential explanation for the higher prevalence in middle-aged individuals *versus* younger and older athletes may relate to age-specific differences in exercise duration (i.e. typically longer in middle-aged to older athletes) and exercise intensity (i.e. typically higher in younger and middle-aged athletes). It is unlikely that middle-aged individuals are more vulnerable to elevated cTn as previous studies showed that athletes' cardiovascular health status may affect cTn elevations,[58] which is strongly age-dependent. The effect of age on cTn release is, therefore, likely driven by underlying exercise characteristics.

Clinical implications

The findings of this meta-analysis can aid the clinical interpretation of cTn elevations following exercise and emphasize the relevance of assay type and exercise characteristics, whereas sex and training status appear to have limited influence. Our outcomes also raise questions on the long-term clinical impact of elevated cTn concentrations in athletes. Data from large-scale studies in patients[59,60] and individuals from the general population[61] found that cTn elevations were consistently associated with adverse health outcomes. A study among long-distance walkers also found that post-exercise cTn concentrations were prognostic of adverse health outcomes during follow-up,[5] but it remains unknown whether these findings can be

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extrapolated to younger athletes and those with low-risk profiles. Large prospective studies in representative athletic cohorts are therefore eagerly anticipated.[62]

Strength and limitations

A major strength of this study is the large number of included studies (n=129), yielding a meta-analytic cohort of 7,289 athletes and 10,061 cTn assessments. However, several limitations should be considered. First, there was significant variation across the studies in the timing of blood sample collection after exercise cessation, ranging from immediately to 12 hours post-exercise. To mitigate this variability, we included only the first post-exercise blood sample in studies with multiple samples and added the time-to-sampling after exercise cessation to our statistical models. Second, cTnI assays lack standardization, as each manufacturer uses unique monoclonal antibodies, making direct comparison between cTnI assays difficult. In contrast, all cTnT assays are produced by a single manufacturer, ensuring more consistency. Nevertheless, we used assay-specific 99th percentile URLs to harmonize data across platforms. Third, sex-specific URLs are recommended by the IFCC when using hs-cTn assays.[63] However, only one cohort in our meta-analysis reported the prevalence of cTn elevations based on sex-specific URLs and found similar prevalences between males and females.[15]

Conclusions

Exercise leads to cTn elevations in a significant proportion of athletes, which is important to consider when evaluating individuals for possible myocardial infarction or other causes of myocardial injury. Post-exercise cTn elevations are more likely to occur when measured using cTnT, analyzed with hs assays, in middle aged athletes, and following vigorous-intensity endurance sports lasting 3–6 hours.

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CONFLICT OF INTEREST

J.O.S., S.L.J.E.J., P.D.T., V.L.A. and T.M.H.E report no conflict of interest. K.B. received speaker honoraria from Boehringer Ingelheim, Novartis, Amgen, and AstraZeneca. T.O. has received honoraria and/or research support from Abbott Laboratories, CardiNor, ChromaDex Novo Nordisk, Novartis, Roche Diagnostics,

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AUTHORS' CONTRIBUTIONS

J.O.S. and K.B. contributed equally to this work. J.O.S. drafted the manuscript and K.B. performed the statistical analyses. J.O.S. developed the search strategy. J.O.S., K.B., and T.M.H.E. defined the study selection criteria and the variables for data extraction. J.O.S. and K.B. performed the study selection. J.O.S. extracted the data, which was independently verified by K.B. J.O.S. and K.B. performed the risk of bias assessments. All authors contributed to the interpretation of the data. S.L.J.E.J., T.O., P.L.M., P.D.T., V.L.A., and T.M.H.E. critically reviewed the manuscript for important intellectual content. All authors read and approved the final version of the manuscript. T.M.H.E. is the guarantor of the work and accepts full responsibility for the integrity of the study, the analyses, and the decision to publish.

DATA AVAILABILITY STATEMENT

The harmonized dataset including assay and cohort level data underlying the meta-analyses and meta-regression analyses that we conducted will be shared upon reasonable request to the corresponding author.

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FIGURE LEGENDS

Figure 1. PRISMA flow chart depicting the number of papers that were identified, screened, and included. Preferred reporting items for systematic reviews and meta-analyses flow diagram of study selection process are presented.

Figure 2. Forest plot for the prevalence of elevated cTn post-exercise, stratified for assay type, sex, type of sport, duration of exercise, training status and risk of bias. The overall post-exercise prevalence of elevated cTn was 36% (95% CI [31–42%]). For each variable, the blue square represents the prevalence, and the black horizontal line represent the 95% CI. The blue diamond represents the pooled estimate and its 95% CI.

Figure 3. Relationship between age and prevalence of post-exercise elevated cTn. A restricted cubic spline model with 4 knots was used, as the association was non-linear (p for non-linearity =0.006), with prevalence peaking in athletes aged approximately 40–50 years and declining thereafter. Green shaded areas represent the corresponding 95% CI. The number of cohorts per age category was 31 (<30 years), 57 (30-39 years), 86 (40-49 years), 18 (50-59 years), and 7 (≥60 years)

Figure 4. Relationship between duration of exercise and prevalence of post-exercise elevated cTn. A restricted cubic spline model with 4 knots was used, as the association was non-linear (p for non-linearity<0.001). The highest prevalence of elevated cTn was observed after exercise lasting approximately 3–6 hours, with the prevalence close to 55%. Green shaded areas represent the corresponding 95% CI. The number of cohorts per duration category was 14 (<1 hour), 22 (1–2 hours), 13 (2–3 hours), 16 (3–4 hours), 23 (4–5 hours), 24 (5–10 hours), and 25 (10-24 hours). Studies with exercise duration ≥24 hours were excluded from this figure.

Graphical abstract. Post-exercise cardiac troponin concentrations above the upper reference limit were most prevalent when high-sensitivity and cTnT assays were used, in middle-aged athletes, and among vigorous-intensity endurance sports lasting 3 to 6 hours. Sex and training status were not associated with the likelihood of cardiac troponin elevations.

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FIGURE 1

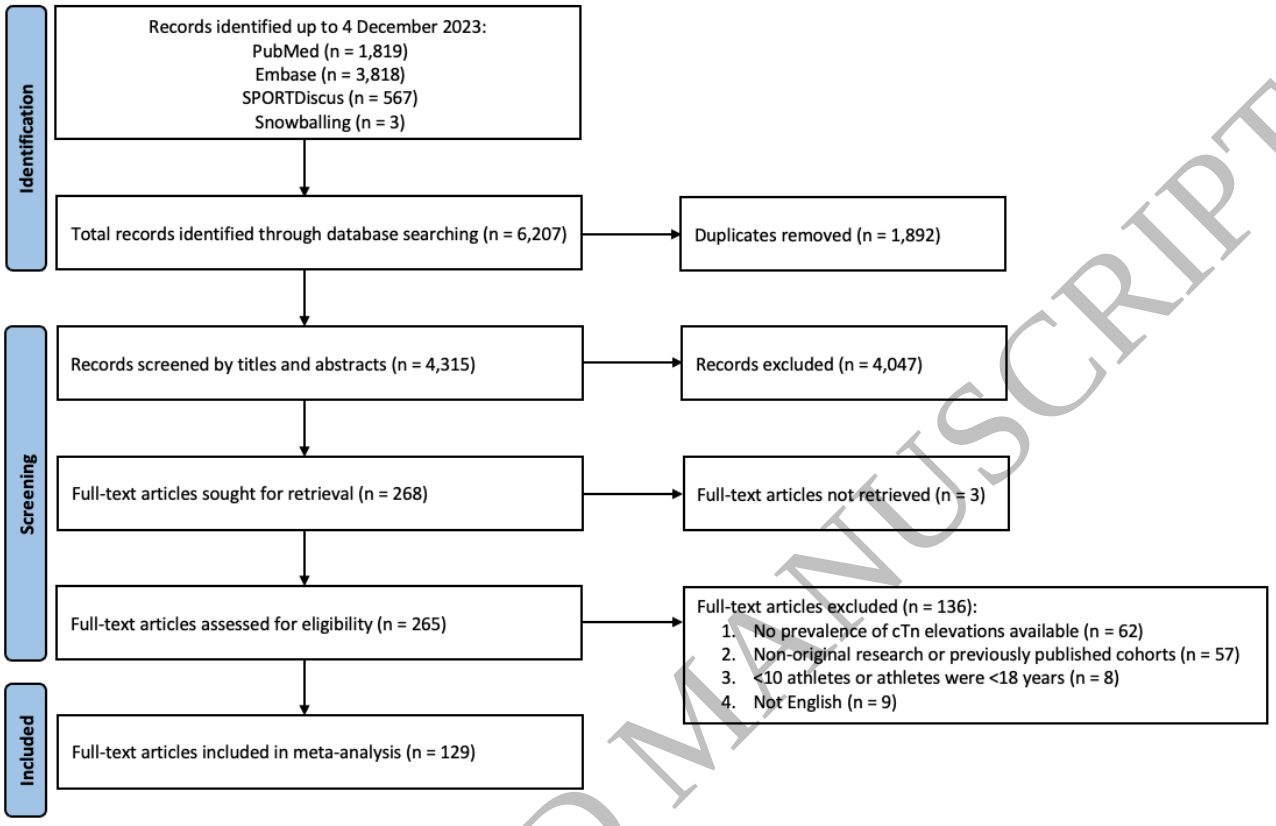

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FIGURE 2

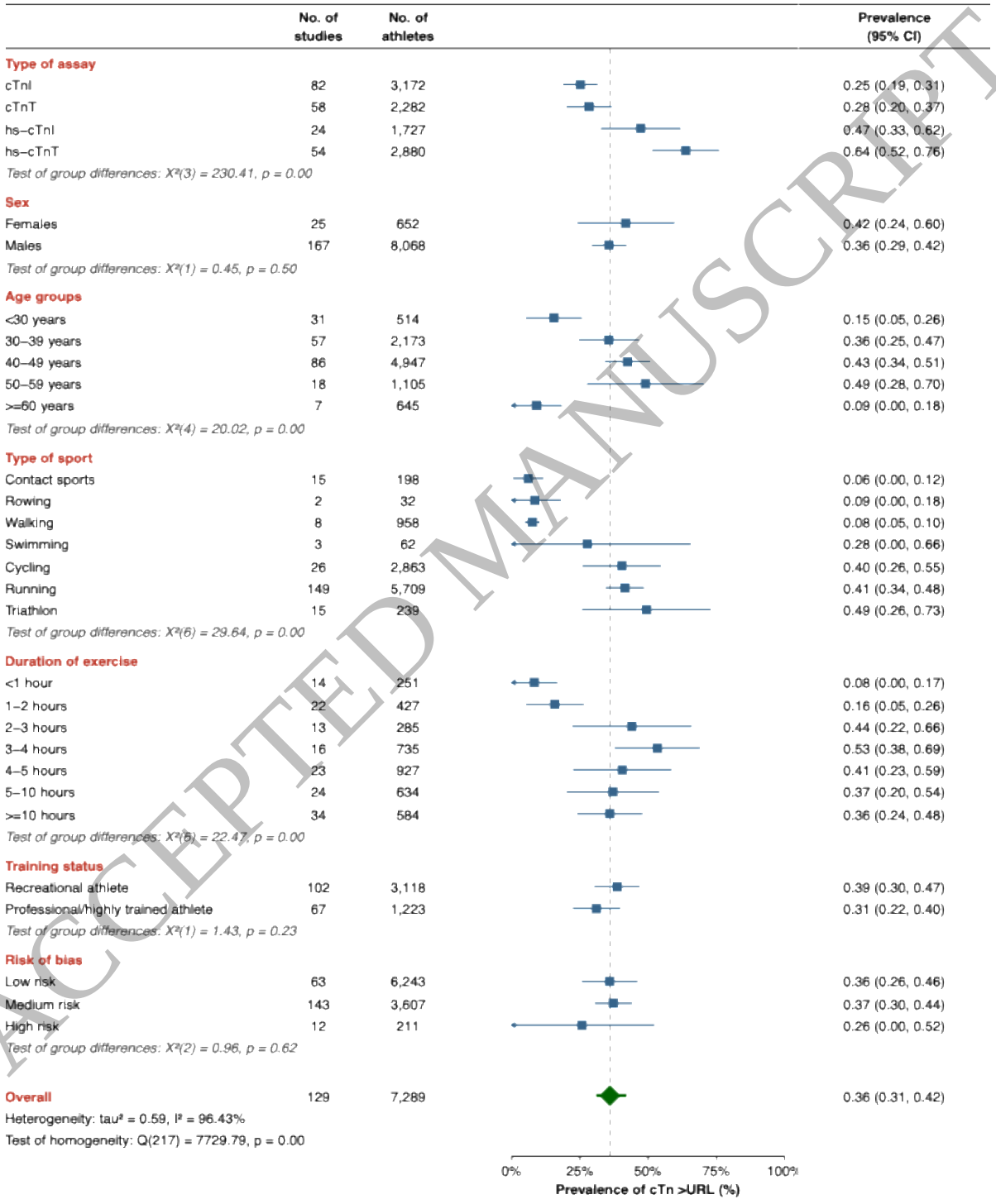

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FIGURE 3

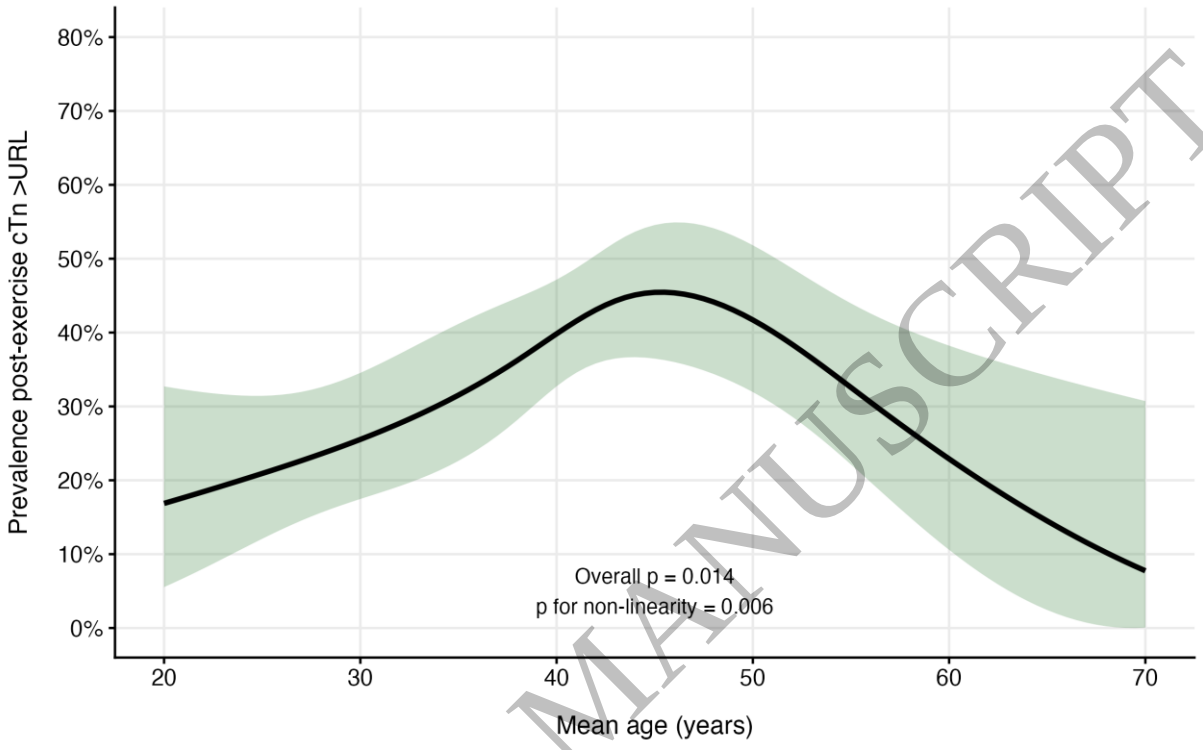

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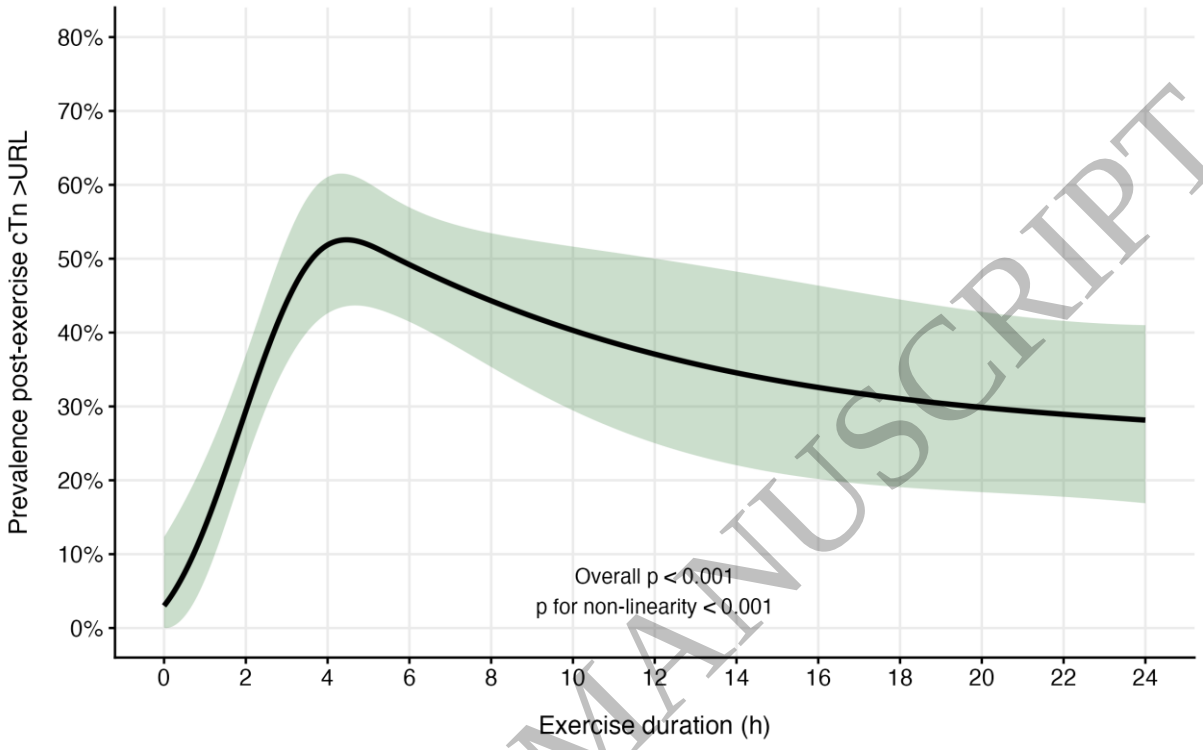
FIGURE 4



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