Coenzyme Q10 supplementation increases blood concentrations but shows limited and inconsistent effects on exercise performance: A systematic review and meta-analysis

Hengzhi Deng[1], Tianyu Song[1], Mingyue Yin[2], Kai Xu[2], Yuming Zhong [3], Norsyazmi bin Mohd[1], Mohamed Nashrudin bin Naharudin[1], Ashril Yusof[1], Xiaohan Fan[1*]

[1]Faculty of Sports and Exercise Science, University Malaya, Kuala Lumpur, Malaysia

[2]School of Athletic Performance, Shanghai University of Sport, Shanghai, China

[3]School of Medical and Health Sciences, Edith Cowan University, Joondalup, Australia

***Corresponding Author:** Xiaohan Fan Universiti Malaya, Jalan Universiti, 50603 Kuala Lumpur, Malaysia. Phone:(60)122488566, Email address: fanxiaohan5@gmail.com

Running head: CoQ10 raises blood levels, small performance gains

This peer-reviewed article has been accepted for publication but not yet copyedited or typeset, and so may be subject to change during the production process. The article is considered published and may be cited using its DOI
10.1017/S0007114525106211
The British Journal of Nutrition is published by Cambridge University Press on behalf of The Nutrition Society

Email address and contact details of co-author:

Hengzhi Deng: denghengzhi66@gmail.com

Tianyu Song: tianyusong123@outlook.com

Mingyue Yin: mingyue0531@sus.edu.cn

Kai Xu: 2221152066@sus.edu.cn

Yuming Zhong: 2221111051@sus.edu.cn

Norsyazmi bin Mohd: syazmimohd@um.edu.my

Mohamed Nashrudin bin Naharudin: nashrudin@um.edu.my

Ashril Yusof: ashril@um.edu.my

Key Points

- CoQ10 supplementation consistently raises blood CoQ10 levels but yields small, heterogeneous effects on exercise performance.
- Subjective fatigue tended to decrease with supplementation, but findings were inconsistent.
- Under chronic supplementation, potential benefits were observed for aerobic endurance exercise and trained individuals, though these results were not consistent across analyses.
- No stable dose–response relationships for supplementation duration or consistent improvements in anaerobic or strength outcomes have been demonstrated under chronic supplementation conditions.
- Preliminary evidence suggests that daily doses of approximately 90–100 mg may represent a practical starting point for chronic CoQ10 supplementation, though optimal dosing requires further investigation.

Abstract

Coenzyme Q10 (CoQ10) is biologically plausible as an ergogenic aid through roles in mitochondrial energy production and antioxidant defence, yet findings from randomised trials are inconsistent. This review included 24 studies from 6 databases published up to November 2025, assessing effects of CoQ10 on exercise performance, subjective fatigue, and circulating CoQ10 levels in healthy adults. Randomised trials comparing CoQ10 with placebo were synthesised using a three-level model. Risk of bias was assessed with RoB2 and certainty judged with GRADE.

Supplementation consistently increased blood CoQ10, indicating robust biochemical responsiveness. In contrast, performance effects were small and inconsistent. In primary analyses, chronic supplementation showed a small benefit, whereas acute supplementation showed no benefit. After excluding outliers, the chronic effect was no longer stable and the acute effect remained trivial. All subgroup analyses were restricted to chronic supplementation. Within this context, aerobic endurance was significant in primary analyses but became borderline after outlier exclusion, while anaerobic and strength outcomes showed little change. Evidence for reduced subjective fatigue was suggestive and became more consistent after outlier exclusion. Benefits in trained individuals were unstable and became consistent only after outlier exclusion. No stable dose–response pattern emerged for supplementation dosage or duration. Heterogeneity and moderate-to-high risk of bias reduced certainty.

Collectively, CoQ10 reliably elevates circulating levels but provides at most modest and context-dependent benefits for exercise performance, largely under chronic use. Overall certainty is very low to low. Well-controlled randomised trials that standardise formulation, dose, and duration and examine sex-specific and endurance-related responses are needed.

Key Words: Coenzyme Q10; Exercise performance; Blood CoQ10; Antioxidant supplementation; Meta-analysis.

1. Introduction

While structured training is the primary driver of physiological adaptation, the strategic use of nutritional supplements has gained increasing attention as a means to enhance exercise performance, support recovery, and mitigate exercise-induced physiological stress. Among these, coenzyme Q10 (CoQ10) is biologically plausible because it cycles between oxidised (ubiquinone) and reduced (ubiquinol) forms, thereby supporting mitochondrial electron transport [1] and antioxidant defence [2], with high tissue distribution in mitochondria-rich membranes [3,4]. These properties suggest potential to support energy production and redox balance during exercise, particularly when reactive oxygen species increase with sustained or intense activity [5–7].

However, findings from human trials are inconsistent. Some studies have reported benefits for aerobic capacity [8,9], anaerobic power [10,11], and strength [12,13], while others have found no performance benefits [14–16]. Heterogeneity in design, formulation, dose, duration, participant characteristics, and outcome selection likely contributes to these discrepancies.

Prior evidence syntheses offer only partial coverage. Most meta-analyses focused on biomarkers of muscle damage, oxidative stress, or inflammation rather than exercise performance [17–20]. A 2024 meta-analysis included only eight trials that reported power-based performance outcomes but did not detect clear ergogenic effects [21]. Other reviews were narrative or athlete-focused [22–25] and did not provide quantitative estimates or omitted eligible studies [9–13,16,26–34]. To date, no analysis has comprehensively quantified CoQ10 effects across multiple performance domains while formally addressing design heterogeneity.

Accordingly, this review synthesises randomised controlled trials in healthy adults to estimate the effects of CoQ10 on exercise performance. Acute and chronic supplementation were compared, and performance outcomes were grouped as aerobic endurance, anaerobic performance, muscular strength, and muscular endurance. Subjective fatigue and circulating CoQ10 were evaluated as secondary outcomes to contextualise potential mechanisms. Exploratory analyses examined whether participant and intervention characteristics, including sex, training status, age, formulation, dose, and duration, moderated the effects, and sought to identify the conditions under which use may be most effective.

2. Methods

The systematic review adhered to the 2020 Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement [35]. The completed PRISMA 2020 checklist is available in **Electronic Supplementary Material Appendix S1**. Additionally, this review protocol was preregistered with the Open Science Framework (OSF; registration ID: osf.io/f4y3j) on June 27, 2025. All extracted datasets and R code used for the analyses have also been deposited in the same OSF project to enhance transparency and reproducibility.

2.1 Eligibility Criteria

For this systematic review and meta-analysis, studies were included if they met the following inclusion criteria: (1) randomised controlled trials published as full-text articles in peer-reviewed journals; (2) investigated the effects of CoQ10 supplementation; (3) included at least one measure of exercise performance derived from a structured exercise test; (4) involved original research on human subjects; and (5) included healthy adults participants without known clinical conditions (≥18 years of age). Studies were excluded if they met any of the following criteria: (1) CoQ10 was co-administered with other active substances exclusively in the experimental group; (2) the experimental group underwent additional exercise training not equally applied to the control group; (3) animal or in vitro studies; (4) no exercise performance outcomes reported; (5) not original research articles (e.g., reviews, trial protocols, conference abstracts); (6) not published in English; or (7) lacked sufficient methodological detail to assess eligibility.

2.2 Data Sources and Search

We searched the Cochrane Library, PubMed, SciELO, SPORTDiscus-EBSCO, and Web of Science database using the following terms: concept 1 ("Coenzyme Q10" OR "CoQ10" OR "Co-Q10" OR "Ubiquinone" OR "Ubiquinol" OR "ubidecarenone" OR "Coenzyme Q-10") and concept 2 ("exercise" OR "physical performance" OR "aerobic capacity" OR "aerobic exercise" OR "anaerobic exercise" OR "endurance" OR "fatigue" OR "training" OR "maximal oxygen uptake" OR "VO2max" OR "sports performance" OR "muscle function"). Searches were conducted on 1 June 2025 and updated on 12 November 2025, with no restrictions on

publication year or language and no additional filters applied.

We did not search trial registries (e.g., ClinicalTrials.gov/WHO ICTRP), grey literature, or preprints. This decision was made a priori to reduce the risk of including unverified, duplicate, or non-peer-reviewed data, and to ensure that all evidence used for quantitative synthesis was traceable to peer-reviewed randomised controlled trials with stable methods and outcomes.

2.3 Data Extraction

All retrieved titles and abstracts were downloaded into Microsoft Excel spreadsheets and EndNote (21) and manually cross-referenced to identify duplicates. Article screening was performed independently by two researchers (D.H.Z and S.T.Y), and discrepancies were resolved by consensus.

Data extracted including:

1) Study characteristics: authors, publication year, study design (parallel or crossover).

2) Participant characteristics: sample size, sex, age, training status, and health status.

3) Intervention characteristics: type of CoQ10 (ubiquinone or ubiquinol), dosage, supplementation duration and timing relative to exercise.

4) Exercise testing protocols.

5) Primary outcomes related to exercise performance: VO_{2max} or VO_{2peak}, time to exhaustion, anaerobic power (e.g., peak or mean power), muscular strength (e.g., 1RM, bench press), muscular endurance (e.g., push-up test, reps to failure), and sport-specific performance tests (e.g., sprint time, jump height, agility tests).

6) Secondary outcomes: self-reported fatigue scores, including Rating of Perceived Exertion (RPE), Visual Analog Scale (VAS) for fatigue, and other validated fatigue questionnaires; and circulating CoQ10 concentrations.

If the data of interest were not reported in the study, we sent an email to the corresponding author to request the data. For studies that did not receive a response, we used the WebPlotDigitizer program (https://apps.automeris.io/wpd/) to extract the data of interest from the original figures reported in the paper [36]. It is worth noting that one study exhibited a slight discrepancy in the reported image data [37]. As no response was received from the corresponding author after inquiry, the relevant data from a previous meta-analysis were adopted as a reference [21].

2.4 Quality and Risk of Bias Assessment

The quality of the articles was assessed using the Physiotherapy Evidence Database (PEDro) scale. Given the specific features of nutritional supplementation trials, we adopted a previously recommended modification [38], adding an item on the effectiveness of placebo blinding: "Did the study assess the effectiveness of placebo blinding?" This addition was intended to capture risks arising when taste, smell or capsule appearance may compromise allocation masking in supplement studies. As item 1 was not scored, the total score ranged from 0 to 11. Articles with a total score of 10–11 were rated as excellent, 7–9 as good, 5–6 as fair, and articles with less than 5 points were rated as poor methodological quality. PEDro scoring was performed at the study level, as it reflects overall methodological rigour rather than outcome-specific sources of bias.

In parallel, risk of bias was assessed using the Cochrane Collaboration Risk of Bias Tool 2 (RoB-2) [39], which evaluates five domains: (i) bias arising from the randomisation process; (ii) bias due to deviations from intended interventions; (iii) bias due to missing outcome data; (iv) bias in measurement of the outcome; and (v) bias in selection of the reported result.

Both assessments were conducted independently by two authors (D.H.Z. and S.T.Y.), with disagreements resolved through discussion. RoB-2 was assessed separately for each outcome category (exercise performance, subjective fatigue, and blood CoQ10 concentration) because multiple outcomes were often reported within the same trials and outcome-specific issues can differ. For subgroup analyses within exercise performance (e.g., by exercise type age, or sex), the same RoB-2 ratings were applied across subgroups, because these subgroup divisions

originated from the same trials and thus shared identical study-level design and implementation features relevant to risk of bias. This approach maintains methodological consistency and prevents artificial inflation of precision that could arise from redundant re-ratings of the same studies. It also ensures that the risk-of-bias assessment can directly inform outcome-specific downgrading decisions regarding certainty of evidence.

The modified PEDro scale and RoB-2 were used complementarily: PEDro provided a quantitative index of overall methodological quality, whereas RoB-2 offered a domain-specific appraisal of internal validity at the outcome level. Inter-rater reliability was quantified at two levels. For item-level agreement, we used Cohen's kappa [40] for binary PEDro items and weighted kappa for the three-category RoB-2 ratings [41]. Alongside kappa statistics, we calculated raw percentage agreement for all categorical items as a direct measure of consensus. A value of ≥80% was considered to indicate acceptable inter-rater reliability [42]. For the PEDro total score, which is continuous, we used the intraclass correlation coefficient (ICC) [43]. Kappa values were interpreted using conventional thresholds (<0.20 = slight, 0.21–0.40 = fair, 0.41–0.60 = moderate, 0.61–0.80 = substantial, >0.80 = almost perfect) [41], and ICC values were interpreted as <0.50 = poor, 0.50–0.75 = moderate, 0.75–0.90 = good, >0.90 = excellent [44]. The corresponding reliability statistics, including raw agreement percentages, are presented together with the quality assessment results.

2.5 Statistical Analysis

2.5.1 Data Extraction, Synthesis and Effect Measures

In this study, the effects of CoQ10 supplementation on the primary outcome of exercise performance and secondary outcomes of subjective fatigue and blood CoQ10 concentrations were analyzed by comparing between-group differences (CoQ10 vs. placebo) or within-group pre–post changes in the CoQ10 supplementation arm. All effect size calculations followed the Cochrane Handbook for Systematic Reviews of Interventions (Version 6.5, 2024) [45].

Given the generally small samples of included studies, effects were expressed as standardised mean differences (Hedges' g) with small-sample correction [46]. When studies reported pre- and post-values, we derived g from change scores. When only post-intervention

values were available, we used between-group differences for parallel trials and paired differences for crossover trials, while preserving within-subject dependency [47,48].

Many trials did not report the required correlations. In the primary analyses, we assumed r = 0.50 and applied this consistently to all pre–post and within-person calculations for both parallel and crossover designs to ensure comparability [47].

Effect sizes were interpreted using standard thresholds: trivial (<0.20), small (0.20–0.49), medium (0.50–0.79), large (≥0.80) [49].

For subjective fatigue and blood CoQ10 concentrations, data extraction and synthesis followed the same approach as for performance outcomes, using between-group differences in change scores as the primary effect measure. Given the expected physiological stability of blood CoQ10 in placebo groups, within-group changes in the CoQ10 supplementation arm were also analyzed as supplementary sensitivity analyses (**Appendix S2**).

Detailed computational formulas and step-by-step procedures are provided in **Appendix S3**.

2.5.2 Meta-Analysis and Heterogeneity

We conducted a three-level meta-analysis following the framework of Assink & Wibbelink (2016) methods and recommendations [50] to account for dependent effect sizes within studies [51,52]. This approach partitions total variance into sampling (level 1), within-study (level 2), and between-study (level 3) components, thereby preserving multiple correlated outcomes without double counting [53]. Model parameters were estimated using restricted maximum likelihood (REML) and cross-checked with maximum likelihood (ML) to ensure stability.

Because the primary aim of this meta-analysis was to evaluate the effects of CoQ10 supplementation on objective exercise performance, we only included studies that reported at least one physical performance outcome (e.g., aerobic capacity, muscular strength). Although subjective fatigue measures and blood CoQ10 concentration were extracted and analyzed as secondary outcomes when available in the included studies, trials that exclusively assessed perceived fatigue and/or blood CoQ10 concentration without accompanying performance data

were excluded to maintain consistency with the primary research objective.

Tests of model coefficients and corresponding confidence intervals (CI) were based on the t-distribution [54]. Prediction intervals (PI) were also computed to characterise expected variation in future studies [55,56]. For heterogeneity, we used the most widely recommended and commonly reported metric, I^2, to assess and report [50,57]. 0% to 25% (low heterogeneity), 25% to 50% (moderate heterogeneity), 50% to 75% (high heterogeneity), and 75% to 100% (fairly high heterogeneity) [47]. To assess the adequacy of statistical power to detect true effects and the possibility of type II errors due to insufficient power, we performed power analyses for both primary and secondary outcomes using the *metameta* package [58].

2.5.3 Moderators and Subgroup Analysis

To explore sources of heterogeneity, moderator and meta-regression analyses were conducted for both categorical and continuous variables [59]. Typically, at least 10 studies are required for each meta-regression and at least 5 studies are required for each subgroup to ensure adequate statistical power [60,61]. The *metameta* package was also used to perform power analysis for each subgroup [58].

To determine whether acute (single dose) versus chronic (≥1 week) supplementation significantly moderated the overall effect, we performed an initial subgroup analysis comparing these two protocols within the complete dataset. Given the distinct pharmacokinetic profiles of acute and chronic dosing and the predominance of chronic protocols in the available literature, all subsequent moderator and subgroup analyses were restricted to chronic studies. The moderators examined included 1) sex; 2) training status; 3) age; 4) CoQ10 type; 5) exercise category; 6) CoQ10 dosage; and 7) supplementation duration.

For age-based subgroup analysis, participants were categorized as young adults (18–35 years), middle-aged adults (36–59 years), and older adults (≥60 years). This classification was informed by mean age ranges commonly reported in the literature and reflects physiologically distinct life stages, particularly in relation to baseline CoQ10 status, mitochondrial function, and susceptibility to oxidative stress. These age-related differences may influence both the absorption and biological efficacy of CoQ10 supplementation, justifying stratified analysis

across age groups (The details were discussed in **section 4.4**). Based on previous participant categorization frameworks, participants were classified as untrained and trained (recreationally active, trained/developmental, well-trained/national level, elite/international level, and world-class) [62]. For consistency, studies explicitly reporting the oxidised form were classified as ubiquinone, whereas those reporting the reduced form were classified as ubiquinol. Trials that did not specify formulation were labelled as unspecified CoQ10.

To better explore the potential ergogenic effects of CoQ10 and to reduce the instability of the computational model due to the small sample size, performance outcomes were grouped into four physiological domains based on the primary potential energy system or neuromuscular mechanism [63,64]: 1) Strength/Power: Efforts lasting ≤10 seconds, characterized by maximal intensity and primarily relying on the ATP–phosphocreatine system (e.g., vertical jump height, 1–3 RM lifts, maximal short sprints); 2) Anaerobic Performance: Continuous efforts lasting >10 seconds and ≤60 seconds, predominantly dependent on anaerobic glycolysis (e.g., 30-s Wingate test, repeated sprint ability); 3) Muscular Endurance: Sustained submaximal contractions performed to volitional exhaustion or failure, typically involving repeated or continuous efforts targeting a specific muscle group, and generally lasting from ~30 seconds to several minutes depending on protocol (e.g., bench press to failure at submaximal load, repeated knee extensions to exhaustion); 4) Aerobic Endurance: Continuous efforts exceeding ~2–3 minutes (typically >10 minutes) where oxidative metabolism provides the predominant energy supply, even when significant anaerobic contributions occur in early phases (e.g., 3-min all-out test, VO_2max ramp protocols, time to exhaustion treadmill tests).

To assess the relationship shapes of continuous variables analysis (CoQ10 dosage and supplementation duration), we attempted to fit a linear, quadratic, or cubic function and compared the models, selecting the model with the lowest bias-corrected Akaike information criterion [65,66]. Additionally, to account for varied supplementation protocols across studies and enhance analysis precision, supplementation duration was standardised to weeks rather than days for meta-regression analysis, allowing for more meaningful dose-response relationship assessment across the included studies.

All regression models were performed using the *metafor* package and later visualized with the *ggplot2* and *orchaRd* packages [67].

2.5.4 Risk of Publication Bias and Sensitivity Analysis

The contour-enhanced funnel plot [68], along with Egger's asymmetry test [69,70], was employed to assess publication bias (tests were only conducted when $k \geq 10$) [71], with $p > 0.05$ indicating no risk of publication bias. Funnel plot symmetry was evaluated through both visual inspection and Egger's test, allowing for a combined subjective and statistical assessment of potential small-study effects and publication bias.

As the main analyses were based on a three-level meta-analytic framework, some existing bias-correction methods (e.g., trim-and-fill, selection models, PET–PEESE) cannot be directly implemented within this structure. Nevertheless, for categories in which publication bias was detected in the three-level analyses, we additionally applied trim-and-fill method under a conventional two-level random-effects model (each study contributing one independent effect size) to conduct a sensitivity analysis [72]. The adjusted pooled effect sizes were compared with the original estimates to evaluate the robustness of the findings.

Sensitivity analyses included: 1) varying assumed correlation coefficients (r): a relatively low value ($r = 0.2$) and a relatively high value ($r = 0.8$) [73]; 2) leave-one-out analyses; 3) exclusion of outliers identified via Cook's distance, hat values (exceeded three times their respective means) and studentized residuals (the absolute value exceeded 3) [74,75]; and 4) exclusion of digitized or borrowed values. Among these, outlier exclusion was applied not only to the overall models but also across all moderator and meta-regression analyses to ensure robustness of subgroup inferences, whereas the remaining sensitivity checks were conducted for the primary pooled effects.

2.6 Certainty of the Evidence

Certainty of the evidence was evaluated using the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) approach, which classifies evidence as "high", "moderate", "low", or "very low" [76]. Risk-of-bias ratings from the RoB-2 assessment

directly informed the GRADE downgrading decisions, alongside considerations of inconsistency, indirectness, imprecision, and publication bias (**Appendix S4)**. GRADE assessments were independently conducted by one reviewer (D.H.Z) and verified by a second (F.X.H).

3 Results

3.1 Studies Retrieved

The initial search yielded 2,871 publications: 2,821 from the primary database search and 50 from an updated search conducted five months later. After title/abstract and full-text screening, 24 studies were included in the meta-analysis, resulting in a total of 131 effect sizes (k = 131) as some studies contributed multiple outcomes. A summary of the search process is shown in **Fig 1**.

3.2 Characteristics of Included Studies

Most of the included studies involved long-term interventions, during which participant withdrawal was reported. The effective sample size per study ranged from 6 to 98 participants, yielding a total pooled sample of 641 individuals (n = 641). Of these, 418 were male and 66 were female. However, the gender of 157 participants could not be determined due to incomplete reporting in some studies [8,15,33]. One study did not report the age of the participants [33], and the mean age of the participants in the remaining studies ranged from 18 to 63.8. Furthermore, fourteen studies recruited trained participants, nine studies recruited untrained participants, and one study recruited both trained and untrained participants [15].

Of the 24 studies included in the meta-analysis, 22 investigated the effects of long-term CoQ10 supplementation, three employed acute supplementation protocols, and one examined both acute and long-term administration [15]. With respect to the formulation of CoQ10, seven studies utilized the reduced form (ubiquinol), five employed the oxidised form (ubiquinone), and the remaining 12 did not specify the chemical form; these were thus classified as CoQ10 of unspecified formulation.

Furthermore, five studies evaluated subjective fatigue as an outcome measure, while thirteen reported changes of blood CoQ10 concentrations. A detailed summary of study characteristics is provided in **Table 1**.

3.3 Primary Analysis

For overall exercise performance (k = 131; n = 641), CoQ10 supplementation yielded a small, unstable improvement (Hedges' g = 0.13; 95% CI: [–0.01, 0.27]; p = 0.06; Low GRADE), suggesting limited practical benefit when all protocols are combined. Moderate heterogeneity (I^2 = 37%, primarily at level 3 = 47%) and low statistical power (7%) further indicate inconsistent effects across studies.

When stratified by intake duration, chronic supplementation (k = 108; n = 585) demonstrated a statistically significant small effect (g = 0.15; 95% CI: [0.01, 0.29]; PI: [-0.43, 0.72]; p = 0.045; I^2 = 42%; Low GRADE), while acute supplementation (k = 23; n = 97) produced a negligible and non-significant effect (g = 0.04; 95% CI: [–0.21, 0.28]; p = 0.76; I^2 = 0; Moderate GRADE). This suggests that CoQ10 may require sustained intake to exert measurable ergogenic effects, consistent with the time needed for tissue saturation and mitochondrial incorporation.

CoQ10 showed a trend toward reducing subjective fatigue (k = 17; n = 141; g = −0.21; 95% CI: [−0.44, 0.02]; I^2 = 5%; p = 0.07; Low GRADE), suggesting a potential subjective benefit that warrants further investigation, though the effect did not reach statistical significance. In contrast, CoQ10 significantly increased blood CoQ10 levels (k = 16; n = 258; g =2.03; 95% CI: [1.50, 2.56]; PI: [0.35, 3.71]; p < 0.001; Low GRADE certainty; power = 97%), confirming robust biochemical responsiveness. However, high heterogeneity (I^2 = 76%) reflects variability in supplementation protocols (e.g., dose, formulation, duration). Importantly, the large biomarker response contrasts sharply with the small and inconsistent performance effects, indicating that elevated circulating CoQ10 does not necessarily translate into functional improvements in exercise capacity.

For more details, please refer **Fig 2**.

3.4 Moderator Analysis

3.4.1 Categorical variables

Sex-based subgroup analysis revealed a statistically significant difference ($p = 0.049$; $I^2 = 37\%$). The pooled effect size among male participants was negligible ($k = 79$; $n = 329$; $g = 0.03$; 95% CI: [–0.16, 0.21]; $p = 0.75$, Low GRADE), whereas larger but imprecise effect was observed among females ($k = 2$; $n = 48$; $g = 0.93$; 95% CI: [0.11, 1.74]; $p = 0.03$; PI [–0.05, 1.90]; Very low GRADE). This apparent sex difference should be interpreted cautiously, as it was driven by a small number of studies with wide confidence intervals and may reflect hormonal, metabolic, or sampling variability.

Regarding training status, no significant between-group difference was found ($p = 0.73$; $I^2 = 43\%$). A small, borderline-significant effect was observed in trained individuals ($k = 70$; $n = 382$; $g = 0.20$; 95% CI: [−0.01, 0.41]; $p = 0.07$, Low GRADE), while effects in untrained ($g = 0.07$) and mixed-training samples ($g = 0.06$) were trivial and non-significant. This pattern may reflect greater sensitivity to marginal performance gains in trained populations or more standardised exercise protocols, though statistical power remained low.

Similarly, age group did not moderate the effects of supplementation ($p = 0.54$; $I^2 = 31\%$). A small, non-significant benefit was observed in older adults ($k = 6$; $n = 27$; $g = 0.32$; 95% CI: [–0.09, 0.73]; $p = 0.12$, Very low GRADE), with lower effect sizes observed in young and middle-aged participants.

Subgroup differences by CoQ10 formulation were statistically significant ($p < 0.01$; $I^2 = 31\%$). A moderate and statistically significant effect was observed in studies using unspecified formulations ($k = 48$; $n = 250$; $g = 0.32$; 95% CI: [0.15, 0.49]; PI: [-0.14, 0.78]; $p < 0.001$; $I^2 = 51\%$; Very low GRADE). In contrast, studies using ubiquinol reported no significant effects ($g = 0.07$; $p = 0.54$; Low GRADE), while ubiquinone was associated with a small but significant negative effect ($g = −0.37$; $p = 0.04$; Very low GRADE). Notably, unspecified formulations outperformed the more bioavailable ubiquinol, an unexpected finding that suggests potential confounding by study design or dosing protocols and warrants cautious interpretation.

For exercise types, aerobic endurance showed a small benefit (k = 44; n = 485; g = 0.24; 95% CI: [0.06, 0.43]; PI: [-0.39, 0.88]; p = 0.01, Very low GRADE), while anaerobic performance, muscular endurance, and strength/power were minimally affected and not significant. The difference between aerobic and anaerobic performance was statistically significant (p = 0.03), indicating that CoQ10 may preferentially support oxidative metabolism over high-intensity, glycolytic activities.

For more details, please refer to **Fig 3**.

3.4.2 Continuous variables

Linear meta-regression of supplementation duration (**Fig 4, top**) indicated a positive but non-significant slope (k = 108; n = 585; $\beta_1 = 0.04$; p = 0.12; $I^2 = 39\%$). The cubic model (**Fig 4, bottom**) also showed no significant non-linear trend (all p > 0.05; $I^2 = 40\%$). Thus, within the studied range, longer supplementation periods did not clearly translate into greater performance benefits. Likewise, dosage showed no significant linear relationship (k = 107; n = 565; $\beta_1 = 0.0003$; p = 0.76; $I^2 = 43\%$), and the cubic model identified no robust curvilinear association (all p > 0.05; $I^2 = 40\%$; **Appendix S5**).

Finally, to explore potential synergistic effects, an interactive dosage × duration model was examined. Data of dosage and duration showed weak-to-moderate negative correlation (r = –0.31 to –0.45), and neither main effects nor the interaction term reached significance (all p > 0.05; **Appendix S6**), providing no conclusive evidence of synergistic dose–time effects.

3.5 Risk of Bias and Quality of Methods

RoB-2 assessments were conducted separately for overall exercise performance, subjective fatigue, and blood CoQ10 concentration.

For overall exercise performance, most trials were judged at some concerns or high risk, primarily due to inadequate reporting of the randomisation process and, to a lesser extent, selective reporting. In contrast, risk was generally low for deviations from intended interventions, missing outcome data, and outcome measurement. Inter-rater reliability between assessors was high across all outcomes (Kappa (k) = 0.79–1.00; raw agreement 83–100%),

exceeding the predefined threshold of 80% and confirming consistency in the evaluation process.

For subjective fatigue, among the four trials assessed, two were rated high risk overall and two showed some concerns. The principal weaknesses were selective reporting (frequently high risk) and incomplete reporting of randomisation (often some concerns). Deviations from intended interventions, missing data, and outcome measurement were largely low risk. Inter-rater agreement for this outcome was variable due to the small sample size (raw agreement 75–100%; Kappa (k) varied widely across domains), with lower agreement observed in the randomisation and deviations domains.

For blood CoQ10 concentration, overall risk was moderate to high, similar to exercise performance. The most frequent issues were inadequate reporting of the randomisation process (limited detail on sequence generation and allocation concealment) and selective reporting. Domains relating to outcome measurement and missing data were predominantly low risk, though some pockets of high risk were present. Inter-rater reliability for this outcome was strong (Kappa (k) = 0.83–1.00; raw agreement 92–100%), supporting the stability of ratings.

In summary, while measurement and follow-up procedures were generally sound, weaknesses in trial design and reporting (particularly randomisation and outcome selection) limit confidence in the body of evidence and should be considered when interpreting pooled effects. For further details, including the assessment results of chronic supplementation subgroup for exercise performance, please see **Appendix S7**.

Funnel plots and Egger's test indicated potential publication bias for several categories, including overall exercise performance, chronic CoQ10 supplementation, changes in blood CoQ10 levels, studies involving mixed/unknown sex, trained participants, unspecified CoQ10 formulations, and aerobic endurance outcomes. In contrast, no evidence of publication bias was observed for the remaining categories ($p > 0.05$) An exploratory trim-and-fill analysis using a conventional two-level random-effects model produced bias-adjusted pooled effects that were consistent with the corresponding primary estimates, suggesting that publication bias is unlikely to have materially influenced the overall conclusions. For more details, please refer

to **Appendix S8** and **S9.**

The mean PEDro score for all studies was 6.91, indicating overall fair-to-good methodological quality. Inter-rater reliability for the total PEDro score was moderate (ICC = 0.56; 95% CI 0.22–0.78). Item-level agreement exceeded 80% for all PEDro items, denoting acceptable consensus despite some low kappa values attributable to category prevalence effects. For more information including per-item agreement statistics, please refer to **Appendix S10**.

The complete evaluation process, raw data, and detailed rationale for RoB-2 and PEDro can be found at osf.io/f4y3j.

3.6 Sensitivity Analysis

For exercise performance, results were generally robust across sensitivity checks, including variation of the assumed correlation coefficient (r = 0.2 and 0.8), refitting with maximum likelihood model rather than restricted maximum likelihood model and exclusion of statistical outliers. Within the overall subgroup framework, excluding statistical outliers and removing digitized or borrowed values rendered the apparent benefit of long-term supplementation non-significant. Consistently, when restricting the dataset to long-term supplementation, the pooled effect was attenuated to borderline non-significance (g = 0.14; 95% CI [–0.01, 0.30]; p = 0.07), likely due to reduced sample size and consequently lower statistical power [77,78] (**Appendix S2**). In the leave-one-out analysis limited to long-term studies, no single study unduly influenced the findings, with pooled effects ranging from 0.09 to 0.18 and corresponding p-values from 0.06 to 0.22 (**Appendix S11**). After exclusion of outliers, between-study heterogeneity for this outcome was eliminated (I^2 = 0%; **Appendix S2**).

Regarding subjective fatigue, the findings are quite sensitive to model selection. Although subjective fatigue was not significant under the default specification (r = 0.5; restricted maximum likelihood), statistical significance emerged when r was set to 0.8 or when models were estimated by maximum likelihood (p = 0.04; **Appendix S2**). Following outlier exclusion, CoQ10 was associated with a reduction in subjective fatigue (g = –0.19; 95% CI [–0.39, –0.01]; p = 0.048) and heterogeneity decreased to I^2 = 0% (**Appendix S2**).

For blood CoQ10 concentration, outlier exclusion did not resolve residual heterogeneity for this outcome (I^2 = 48%), and the remaining sensitivity checks did not materially alter the direction of the pooled effect (**Appendix S2**).

In moderator analyses, outlier removal yielded notable changes: 1) In the sex subgroup, the effect for mixed/unknown sex became non-significant (g = 0.21; 95% CI: [–0.02, 0.44]; p = 0.07), and the female subgroup was entirely excluded; 2) Among training levels, the effect in trained participants became significant (g = 0.19; 95% CI: [0.01, 0.38]; p = 0.049), while the mixed group was excluded; 3) In the age subgroup, no statistical changes were observed, except that the older adult group was fully excluded due to outlier influence; 4) In terms of CoQ10 formulation, oxidised CoQ10 (ubiquinone) no longer showed a significant negative effect on performance (g = –0.51; 95% CI: [–1.34, 0.33]; p = 0.23); 5) Finally, within exercise modality, the effect on aerobic endurance was attenuated and became borderline significant (g = 0.17; 95% CI: [–0.01, 0.35]; p = 0.05) (**Appendix S12**).

In the meta-regression analysis, removal of outliers did not substantially alter the linear or cubic non-linear associations for supplementation duration, nor the linear association for the dose × duration interaction. No significant trends were observed in any of these models (**Appendix S6 and S13**). However, after excluding outliers (k = 15), a significant non-linear relationship emerged between dosage and effect size (**Fig 5**; $\beta 2$ = 0.001, p = 0.047; I^2 = 31%). The model identified two local extrema at 146.56 mg/day (g = -0.22) and 259.44 mg/day (g = 0.45), with an inflection point at 203 mg/day (g = 0.11). Importantly, the only dosage range demonstrating statistically robust effects (95% CI lower bound consistently > 0) was 90–97.1 mg/day (green shaded region), suggesting this as a potential evidence-based therapeutic window. Higher dosages, including the theoretical peak at 259 mg/day, showed insufficient statistical stability for reliable recommendations.

4. Discussion

This meta-analysis provides a comprehensive quantitative synthesis of the effects of CoQ10 supplementation on exercise performance, subjective fatigue, and circulating CoQ10 levels. Overall, CoQ10 supplementation produced a small and inconsistent improvement in

performance, a modest reduction in subjective fatigue, and a large, robust increase in blood CoQ10 concentrations. Collectively, these findings suggest that while CoQ10 supplementation effectively elevates systemic levels, the translation of these biochemical changes into functional performance benefits remains limited and context-dependent. Below, we interpret these findings in light of plausible biological mechanisms and existing evidence.

4.1 Effects of CoQ10 on Overall Exercise Performance: Acute vs. Chronic Supplementation

The observation that chronic rather than acute supplementation is associated with small performance gains aligns with the known pharmacokinetics of CoQ10. Sustained intake is typically required to replenish tissue stores, particularly within skeletal muscle mitochondria, where CoQ10 supports ATP production via the electron transport chain [(3)]. With oral supplementation, plasma concentrations rise gradually and generally reach steady-state after about two to three weeks of continuous use [(79)]. By contrast, a single dose is often insufficient to overcome limited absorption and distribution to metabolically active tissues, especially in the absence of co-ingested fats or sustained-release formulations.

Moreover, longer-term intake allows CoQ10 to exert its antioxidant effects, reduce exercise-induced oxidative stress and improve mitochondrial efficiency, mechanisms that require time to induce meaningful physiological adaptations [(80)]. These changes are unlikely to occur following an acute or short-term dose. Nevertheless, the small effect size and wide prediction interval underscore substantial variability among studies, suggesting that the ergogenic response to CoQ10 is influenced by multiple contextual factors such as exercise type, training status, and baseline CoQ10 levels. These findings reinforce the view that chronic supplementation is a prerequisite for any measurable effect but that its practical significance remains uncertain.

4.2 Chronic Supplementation

4.2.1 Sex-specific effects of long-term CoQ10 supplementation on exercise performance

Female participants exhibited larger but less precise performance effects compared with males, consistent with possible differences in baseline CoQ10 status [81,82] or hormonal modulation of mitochondrial function [83]. However, this interpretation remains tentative, as only two studies included women, and the female subgroup was removed after outlier exclusion due to insufficient data. The limited sample size and wide confidence intervals preclude firm conclusions, highlighting the need for adequately powered, sex-stratified trials to better elucidate whether and to what extent biological sex moderates the effects of CoQ10 supplementation on exercise performance.

4.2.2 Influence of Training Status on the Effects of CoQ10 Supplementation

Training status appeared to moderate the ergogenic effects of CoQ10 supplementation. Trained individuals typically exhibit greater mitochondrial density, oxidative enzyme activity, and metabolic turnover during exercise, conditions under which CoQ10's role in electron transport and ATP resynthesis becomes more physiologically relevant [80,84]. Consequently, CoQ10 supplementation may offer greater benefits when mitochondrial demand and oxidative stress are elevated [85].

In contrast, untrained individuals likely experience lower oxidative stress and possess a larger adaptive reserve [85], which diminishes the physiological need for additional CoQ10. Moreover, greater variability in physical capacity and motivation among untrained participants may introduce higher between-study heterogeneity, potentially obscuring subtle ergogenic effects [86]. Overall, these differences in responsiveness underscore the importance of considering participants' training status when evaluating the efficacy of mitochondrial-targeted nutritional interventions.

4.2.3 Influence of Age on the Efficacy of CoQ10 Supplementation

Although comprehensive data on age-related changes in CoQ10 concentrations across different human tissues remain limited, existing evidence suggests that endogenous CoQ10 biosynthesis peaks around the age of 25 and declines progressively thereafter [87,88]. Additionally, brain ageing, which typically begins around the age of 40, has been associated with mitochondrial dysfunction, including impaired ATP production and increased generation of ROS, both of which may affect CoQ10 turnover and utilisation efficiency [89,90]. According to the World Health Organization (WHO), individuals aged 60 years and older are classified as older adults, a stage at which endogenous CoQ10 synthesis is often markedly reduced [91]. Notably, some studies have reported elevated plasma concentrations of oxidised CoQ10 in older adults; however, this may reflect reduced cellular uptake or impaired redox cycling, rather than improved functional CoQ10 status [92,93]. These physiological and biochemical factors underscore the importance of evaluating age-specific responses to CoQ10 supplementation and form the rationale for the age stratification used in this analysis.

Subgroup analyses stratified by age revealed very small effect sizes in young and middle-aged adults that did not reach statistical significance, whereas effect estimates in older adults were slightly higher, but the results were inconsistent and imprecise. These age-related variations can be explained by several physiological mechanisms. First, ageing is associated with mitochondrial dysfunction, including reduced electron transport chain efficiency and lower endogenous CoQ10 synthesis in tissues such as skeletal muscle and cardiac muscle [79,80]. As a result, older individuals may benefit more from exogenous CoQ10 supplementation due to their greater baseline deficiency. This could partially explain the relatively larger effect size observed prior to outlier removal. However, the limited number of studies in older populations (only six effect sizes) severely restricts statistical power and undermines the stability of this subgroup's estimate.

Second, young and middle-aged adults generally have higher baseline mitochondrial function and antioxidant capacity [94,95], which may make additional CoQ10 supplementation less impactful unless these individuals are exposed to significant oxidative stress or have low CoQ10 status due to training demands or dietary insufficiency. Moreover, because metabolic

demand and redox balance during exercise vary with age, the extent to which CoQ10 can affect performance may differ accordingly.

4.2.4 Influence of CoQ10 Formulation on Exercise Performance

Although ubiquinol, the reduced and more bioavailable form of CoQ10, achieves higher plasma concentrations than ubiquinone when administered at equivalent doses [96], this advantage did not translate into superior performance effects. The inconsistency may be attributed to heterogeneity in study design, dosing duration, or participant characteristics [97].

The apparent advantage of "unspecified" formulations likely reflects reporting ambiguity rather than genuine efficacy differences. Similarly, the unexpected negative association observed for ubiquinone prior to outlier exclusion is difficult to reconcile biologically and may be explained by methodological heterogeneity or small-study effects rather than a true detrimental effect. Given the limited number of trials explicitly reporting the formulation type, these findings highlight the need for transparent reporting and standardisation of CoQ10 products in future research.

4.2.5 Influence of Exercise Type on the Effects of CoQ10 Supplementation

CoQ10 may offer greater benefits for aerobic endurance training than for anaerobic performance or strength training outcomes. This pattern is physiologically plausible given CoQ10 primary role in mitochondrial oxidative phosphorylation, which is more critical for sustained aerobic energy production than for short-duration, high-intensity efforts that rely predominantly on glycolytic and phosphagen pathways [3]. During prolonged endurance exercise, CoQ10 facilitates electron transfer in the mitochondrial respiratory chain, supports ATP resynthesis, and helps mitigate exercise-induced oxidative stress [80]. This mechanistic rationale is supported by previous trials reporting improvements in aerobic capacity, reduced lactate accumulation, and increased time-to-exhaustion following CoQ10 supplementation [8,9].

In contrast, the negligible effects observed in anaerobic and strength/power outcomes likely reflect the lower dependence of these performance domains on mitochondrial ATP production and the limited capacity of CoQ10 to influence short-term energy systems or

neuromuscular force generation. This finding aligns with prior evidence showing no significant ergogenic effects of CoQ10 on maximal power output or sprint performance in resistance-trained individuals [15,98].

4.2.6 Influence of Supplementation Duration and Dosage

Across models, supplementation duration did not show a consistent time–response gradient. This is compatible with a scenario in which tissue availability plateaus once basic stores are replenished [79], such that longer exposure does not necessarily yield proportional performance gains. Heterogeneity in adherence, baseline CoQ10 status, and training load likely further blunts any monotonic temporal signal.

For dosage, a non-linear pattern emerged following influence diagnostics, suggesting that ergogenic responses may depend on thresholds and potential saturation rather than increase proportionally with dose. This is biologically plausible because CoQ10 absorption depends on formulation and lipid co-ingestion, involves lymphatic transport, and appears subject to limited by solubility and saturable uptake [99,100]. Additional factors such as differences in redox state (ubiquinone versus ubiquinol), carrier matrices (for example, emulsified or solubilised forms), and interindividual variability in gastrointestinal absorption and mitochondrial incorporation may also contribute to nonmonotonic dose–effect patterns even when circulating CoQ10 concentrations rise [80,101].

In the two-dimensional meta-regression model, no significant main effects or interaction between duration and dose were observed either before or after outlier removal, which is consistent with the absence of a simple monotonic gradient. Nevertheless, the non-linear signal for dose was dependent on outlier handling and was derived from a relatively small number of effect sizes. Study dose ranges may also covary with other methodological features such as exercise modality, intervention duration, or product type. Therefore, these findings should be regarded exploratory, and future trials with standardised dosage ranges and formulations are needed to determine whether a reproducible therapeutic dose window exists.

4.3 Effects of CoQ10 Supplementation on Subjective Fatigue

The overall pattern of findings suggests that CoQ10 supplementation may help reduce subjective fatigue during exercise, although the strength of this effect remains uncertain. Mechanistically, CoQ10's role as a mitochondrial electron carrier supports ATP resynthesis and promotes more efficient oxidative phosphorylation, which can help attenuate peripheral fatigue signals and lower subjective effort [80]. Additionally, its antioxidant properties may reduce exercise-induced oxidative stress in muscle fibres, helping preserve contractile function and delay the onset of exertion-related discomfort [11,25]. Notably, despite differences in statistical significance thresholds, the consistent direction of the effect supports a biologically plausible anti-fatigue benefit of CoQ10 supplementation. However, the magnitude of the observed effect is small and heterogeneous across exercise types and participant characteristics. Given that subjective fatigue is influenced by multiple physiological and psychological factors, CoQ10's impact should be regarded as supportive rather than determinative.

4.4 Effects of CoQ10 Supplementation on Blood CoQ10 Concentrations

The present analysis confirmed that oral CoQ10 supplementation substantially elevates circulating CoQ10 levels. This finding aligns with its known pharmacokinetics, whereby ingested CoQ10 is incorporated into chylomicrons and transported into the bloodstream, resulting in marked increases in plasma concentrations within hours of ingestion [79,82].

However, the physiological relevance of these increases remains uncertain. Although elevated plasma CoQ10 is commonly interpreted as a marker of compliance and systemic availability, evidence suggests that tissue uptake, particularly into skeletal muscle mitochondria, is much more limited. For example, Cooke et al. (2008) [15] and Zhou et al. (2005) [34] both reported no appreciable increases in intramuscular CoQ10 despite significant rises in circulating levels. This dissociation may reflect regulatory mechanisms that limit tissue accumulation even when plasma concentrations are elevated.

Moreover, although the large effect size confirms the bioavailability of supplementation, the high between-study heterogeneity highlights variability in formulation characteristics, dosing regimens, and analytical methods. These factors complicate interpretation of the

relationship between biochemical and functional outcomes. Taken together, while increased blood CoQ10 demonstrates effective absorption, it should not be assumed to directly translate to higher mitochondrial content or improved exercise performance. Clarifying these distinctions is essential to understanding the true ergogenic potential of CoQ10.

4.5 Practical Applications

Collectively, our findings indicate that CoQ10 supplementation may hold particular promise for endurance-type athletes or individuals with elevated oxidative demands or low baseline CoQ10 status, whereas its benefits for strength, power or high-intensity anaerobic performance appear negligible. Given the modest but consistent increases in circulating CoQ10 and the observed small performance gains in aerobic conditions, an approximate dose of 90–100 mg per day may be considered a practical starting point for long-term supplementation, pending further high-quality dose-ranging studies.

However, practitioners should remain mindful that: (1) the evidence for a clear dose–response is currently lacking and tissue uptake remains uncertain; (2) elevated plasma CoQ10 does not guarantee functional improvements in muscle mitochondria; (3) athlete-specific factors such as training status, sex, exercise type and baseline nutritional or CoQ10 status modulate responsiveness; and (4) cost-benefit and formulation quality (absorption, bioavailability) must be weighed before routine use in athletic populations. If cost and supply allow, the reduced form ubiquinol may be a preferable choice, given its higher bioavailability and more favourable absorption characteristics compared with ubiquinone.

In practice, endurance coaches or sports nutrition practitioners may choose to implement CoQ10 supplementation over a sustained period (for example, four weeks or longer), monitor subjective fatigue and performance trends, and reassess its continued use in the context of individual response, economy and training phase.

4.6 Future Directions

Future research should prioritize large, rigorously designed randomised controlled trials that stratify participants by sex, age, and training level to clarify population-specific effects. Standardising supplementation protocols, including dose, formulation, duration, and co-ingestion strategies, is essential to reduce heterogeneity and improve comparability across studies. In addition, future trials should incorporate direct measures of skeletal muscle CoQ10 content, mitochondrial function, and oxidative stress biomarkers to better elucidate mechanistic pathways linking supplementation to performance outcomes. Finally, given the current uncertainty surrounding optimal dosing regimens, well-designed dose-ranging trials are needed to determine whether threshold or saturation effects account for the inconsistent ergogenic benefits observed to date.

4.7 Strengths and Limitations

A key strength of this meta-analysis is its comprehensive and quantitative integration of existing evidence using rigorous multi-level models, moderator analyses, and sensitivity analyses. Unlike prior reviews [22–24], which provided exclusively narrative syntheses or qualitative summaries, our study offers the first detailed meta-analytic estimates of CoQ10's effects across multiple domains, including performance outcomes, subjective fatigue, and circulating biomarkers. By accounting for methodological heterogeneity and potential moderators such as sex, age, training status, supplementation duration, dosage, and formulation type, our findings highlight that the ergogenic potential of CoQ10 is likely context-dependent rather than universally beneficial. This provides a more nuanced perspective than earlier reports, which often presented overly general conclusions regarding its efficacy.

Moreover, while Talebi et al. (2024) conducted a meta-analysis [21], their focus was limited to a single outcome (mean power), preventing broader conclusions about endurance, fatigue perception, or biochemical adaptations. In contrast, our multi-dimensional approach integrates a wider array of performance indices, enhancing ecological validity and practical relevance. Extensive sensitivity analyses, including leave-one-out, estimator variation, and outlier

exclusion, further strengthen the robustness and transparency of our conclusions.

Nevertheless, several limitations merit consideration. At the study level, although 24 trials were included, many subgroups had small sample sizes, and protocols varied considerably in dosage, duration, and outcome measurement. The limited reporting of muscle tissue CoQ10 levels further restricts mechanistic interpretation. Moreover, the included trials exhibited moderate-to-high risk of bias, particularly in randomisation and selective reporting domains, which may affect the reliability of individual study estimates. At the meta-analysis level, high between-study variability and the absence of consistent dose–response relationships suggest that unmeasured confounders or methodological heterogeneity may have influenced pooled effects. Furthermore, this review was restricted to peer-reviewed, English-language sources and did not search trial registries, grey literature, or preprints to reduce duplicate records and ensure data quality. However, publication bias and language bias may persist, as null or negative findings are often underrepresented in published literature. While small-study effects were assessed using contour-enhanced funnel plots and Egger's tests, the overall certainty should be interpreted considering these potential limitations.

Overall, this meta-analysis advances the field by providing a comprehensive and critical evaluation of CoQ10 supplementation in exercise contexts, while highlighting key methodological and conceptual gaps that should inform the design of future high-quality trials.

5. Conclusion

In conclusion, this meta-analysis confirms that CoQ10 supplementation reliably elevates blood concentrations but yields only modest, context-dependent ergogenic effects. Potential benefits seem greatest with chronic use in aerobic endurance settings, yet the instability of the findings and the absence of a clear dose–response relationship mean that its role as a consistent performance enhancer remains uncertain and warrants further rigorous investigation.

Acknowledgments

No financial assistance was received in support of this study by any of the authors.

H.D. conceptualised the study, designed the methodology, performed the statistical analysis and completed the draft of the manuscript; **T.S.** and **X.F.** conducted article screening and formatting; **M.Y.**, **K.X.**, **Y.Z.** and **N.B.M.** contributed to article screening and data collection; **M.N.B.N.**, **A.Y.** and **X.F.** reviewed and proofread the manuscript. All authors contributed to the interpretation of the results, revised the manuscript and approved the final version.

All authors declare no conflict of interest.

The authors wish to express their highest respect to the editors and reviewers for their insightful suggestions. We also sincerely appreciate all researchers whose studies were included in this meta-analysis.

Reference

1. Littarru GP & Tiano L (2010) Clinical aspects of coenzyme Q10: An update. *Nutrition* **26**, 250–254.
2. Bentinger M, Brismar K, & Dallner G (2007) The antioxidant role of coenzyme Q. *Mitochondrion* **7**, S41–S50.
3. Crane FL (2001) Biochemical functions of coenzyme Q10. *Journal of the American College of Nutrition* **20**, 591–598.
4. Hargreaves IP (2014) Coenzyme Q10 as a therapy for mitochondrial disease. *The International Journal of Biochemistry & Cell Biology* **49**, 105–111.
5. Merry TL & Ristow M (2016) Mitohormesis in exercise training. *Free Radical Biology and Medicine* **98**, 123–130.
6. Powers SK & Jackson MJ (2008) Exercise-induced oxidative stress: Cellular mechanisms and impact on muscle force production. *Physiological Reviews* **88**, 1243–1276.
7. Reid MB (2016) Redox interventions to increase exercise performance. *The Journal of Physiology* **594**, 5125–5133.
8. Alf D, Schmidt ME, & Siebrecht SC (2013) Ubiquinol supplementation enhances peak power production in trained athletes: a double-blind, placebo controlled study. *J Int Soc Sports Nutr* **10**, 24.

9. Kunching S, Nararatwanchai T, Chalermchai T, Wongsupasawat K, Siti prapaporn P, & Thipsiriset A (2022) The effects of ubiquinol supplementation on clinical parameters and physical performance of trained men.

10. Ebrahimi M & and Ladary MR (2016) Effect of CoQ10 on anaerobic performance in elite wrestlers. *International Journal of Wrestling Science* **6**, 86–89.

11. Mizuno K, Tanaka M, Nozaki S, Mizuma H, Ataka S, Tahara T *et al.* (2008) Antifatigue effects of coenzyme Q10 during physical fatigue. *Nutrition* **24**, 293–299.

12. Mohammadi M, Naderi A, Siavoshi H, Mohammadi F, & Abadi MP (2020) The effect of short-term use of Coenzyme Q10 supplementation on selected physical and physiological characteristics of young male elite wrestlers. *Revista Brasileira de Nutriçao Esportiva* **14**, 53–65.

13. Moreno-Fernandez J, Puche-Juarez M, Toledano JM, Chirosa I, Chirosa LJ, Pulido-Moran M *et al.* (2023) Ubiquinol short-term supplementation prior to strenuous exercise improves physical performance and diminishes muscle damage. *Antioxidants* **12**, 1193.

14. Bloomer RJ, Canale RE, McCarthy CG, & Farney TM (2012) Impact of oral ubiquinol on blood oxidative stress and exercise performance. *Oxidative Medicine and Cellular Longevity* **2012**, 465020.

15. Cooke M, Iosia M, Buford T, Shelmadine B, Hudson G, Kerksick C *et al.* (2008) Effects of acute and 14-day coenzyme Q10 supplementation on exercise performance in both trained and untrained individuals. *J Int Soc Sports Nutr* **5**, 8.

16. Mieszkowski J, Kochanowicz A, Brzezińska P, Kochanowicz M, Żołądkiewicz K, Stankiewicz B *et al.* (2024) Ubiquinone (coenzyme Q-10) supplementation influences exercise-induced changes in serum 25(OH)D3 and the methyl-arginine metabolites: A double-blind randomized controlled trial. *Antioxidants* **13**, 760.

17. Akbari A, Mobini GR, Agah S, Morvaridzadeh M, Omidi A, Potter E *et al.* (2020) Coenzyme Q10 supplementation and oxidative stress parameters: a systematic review and meta-analysis of clinical trials. *Eur J Clin Pharmacol* **76**, 1483–1499.

18. Dabbaghi Varnousfaderani S, Musazadeh V, Ghalichi F, Kavyani Z, Razmjouei S, Faghfouri AH *et al.* (2023) Alleviating effects of coenzyme Q10 supplements on biomarkers of inflammation and oxidative stress: results from an umbrella meta-analysis. *Front Pharmacol* **14**. doi:10.3389/fphar.2023.1191290.

19. Qu H & Qu Y (2025) Can coenzyme Q10 supplementation reduce exercise-induced muscle damage and oxidative stress in athletes? A systematic review and meta-analysis.

Complementary Therapies in Clinical Practice **60**, 102001.

20. Sedaghat A, Samadi M, Shirvani H, Sepandi M, & Tahmasebi W (2022) Coenzyme Q10 supplementation and oxidative stress parameters: An updated systematic review and meta-analysis of randomized controlled clinical trials.

21. Talebi S, Pourgharib Shahi MH, Zeraattalab-Motlagh S, Asoudeh F, Ranjbar M, Hemmati A *et al.* (2024) The effects of coenzyme Q10 supplementation on biomarkers of exercise-induced muscle damage, physical performance, and oxidative stress: A GRADE-assessed systematic review and dose-response meta-analysis of randomized controlled trials. *Clinical Nutrition ESPEN* **60**, 122–134.

22. Bian Z & Wei L (2025) The role of coenzyme Q10 in exercise tolerance and muscle strength. *Archives of Physiology and Biochemistry* **0**, 1–20.

23. Drobnic F, Lizarraga MA, Caballero-García A, & Cordova A (2022) Coenzyme Q10 supplementation and its impact on exercise and sport performance in humans: A recovery or a performance-enhancing molecule? *Nutrients* **14**, 1811.

24. Fernandes MSDS, Fidelis DEDS, Aidar FJ, Badicu G, Greco G, Cataldi S *et al.* (2023) Coenzyme Q10 Supplementation in Athletes: A Systematic Review. *Nutrients* **15**, 3990.

25. Sarmiento A, Diaz-Castro J, Pulido-Moran M, Kajarabille N, Guisado R, & Ochoa JJ (2016) Coenzyme Q10 supplementation and exercise in healthy humans: A systematic review. *Current Drug Metabolism* **17**, 345–358.

26. Deichmann RE, Lavie ,Carl J., & and Dornelles AC (2012) Impact of coenzyme Q-10 on parameters of cardiorespiratory fitness and muscle performance in older athletes taking statins. *The Physician and Sportsmedicine* **40**, 88–95.

27. Gharahdaghi N, Shabkhiz F, Azarboo E, & Keyhanian A (2013) The effects of daily coenzyme Q10 supplementation on VO2max , vVO2max and intermittent exercise performance in soccer players. *Life Science Journal* **10**.

28. Laaksonen R, Fogelholm M, Himberg J-J, Laakso J, & Salorinne Y (1995) Ubiquinone supplementation and exercise capacity in trained young and older men. *Eur J Appl Physiol* **72**, 95–100.

29. Malm C, Svensson M, Ekblom B, & Sjödin B (1997) Effects of ubiquinone-10 supplementation and high intensity training on physical performance in humans. *Acta Physiologica Scandinavica* **161**, 379–384.

30. Orlando P, Silvestri ,Sonia, Galeazzi ,Roberta, Antonicelli ,Roberto, Marcheggiani ,Fabio, Cirilli ,Ilenia *et al.* (2018) Effect of ubiquinol supplementation on biochemical and oxidative stress indexes after intense exercise in young athletes. *Redox Report* **23**,

136–145.

31. Porter DA, Costill DL, Zachwieja JJ, Krzeminski K, Fink WJ, Wagner E *et al.* (1995) The effect of oral coenzyme Q10 on the exercise tolerance of middle-aged, untrained men. *International Journal of Sports Medicine* **16**, 421–427.

32. Thar PP, Likitwattanasade T, & Srikuea R (2025) Post-workout supplementation with CoQ10 and sports drink on exercise performance and muscle recovery after exercise in normal and overweight males. *Sports Medicine and Health Science*. doi:10.1016/j.smhs.2025.02.005.

33. Ylikoski T, Piirainen J, Hanninen O, & Penttinen J (1997) The effect of coenzyme Q10 on the exercise performance of cross-country skiers. *Molecular Aspects of Medicine* **18**, 283–290.

34. Zhou S, Zhang Y, Davie A, Marshall-Gradisnik S, Hu H, Wang J *et al.* (2005) Muscle and plasma coenzyme Q10 concentration, aerobic power and exercise economy of healthy men in response to four weeks of supplementation. *The Journal of Sports Medicine and Physical Fitness* **45**, 337–346.

35. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD *et al.* (2021) The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *BMJ* **372**, n71.

36. Premaratne S, Newman J, Hobbs S, Garnham A, & Wall M (2020) Meta-analysis of direct surgical versus endovascular revascularization for aortoiliac occlusive disease. *Journal of Vascular Surgery* **72**, 726–737.

37. Gökbel H, Gül I, Belviranl M, & Okudan N (2010) The effects of coenzyme Q10 supplementation on performance during repeated bouts of supramaximal exercise in sedentary men. *The Journal of Strength & Conditioning Research* **24**, 97.

38. Grgic J (2018) Caffeine ingestion enhances Wingate performance: a meta-analysis. *European Journal of Sport Science* **18**, 219–225.

39. Sterne JAC, Savović J, Page MJ, Elbers RG, Blencowe NS, Boutron I *et al.* (2019) RoB 2: a revised tool for assessing risk of bias in randomised trials. *BMJ* **366**, l4898.

40. Cohen J (1960) A coefficient of agreement for nominal scales. *Educational and Psychological Measurement* **20**, 37–46.

41. Landis JR & Koch GG (1977) The measurement of observer agreement for categorical data. *Biometrics* **33**, 159–174.

42. McHugh ML (2012) Interrater reliability: the kappa statistic. *Biochemia Medica* **22**, 276–282.

43. Shrout PE & Fleiss JL (1979) Intraclass correlations: Uses in assessing rater reliability. *Psychological Bulletin* **86**, 420–428.

44. Koo TK & Li MY (2016) A guideline of selecting and reporting intraclass correlation coefficients for reliability research. *Journal of Chiropractic Medicine* **15**, 155–163.

45. Higgins J, Thomas J, Chandler J, Cumpston M, Li T, Page MJ *et al.* (2019) *Cochrane handbook for systematic reviews of interventions*. 2nd ed. Newark: John Wiley & Sons, Incorporated.

46. Hedges LV (1983) A random effects model for effect sizes. *Psychological Bulletin* **93**, 388–395.

47. Cumpston M, Li T, Page MJ, Chandler J, Welch VA, Higgins JP *et al.* (2019) Updated guidance for trusted systematic reviews: a new edition of the Cochrane Handbook for Systematic Reviews of Interventions. *Cochrane Database Syst Rev* **2019**, ED000142.

48. Hedges LV, Shymansky JA, & Woodworth G (1989) A pratical guide to modern methods of meta-analysis. *ERIC* **ISBN-0-87355-081-1**, 80.

49. Cohen J (2013) *Statistical power analysis for the behavioral sciences*. 2nd ed. New York: Routledge.

50. Assink M & Wibbelink C (2016) Fitting three-level meta-analytic models in R: A step-by-step tutorial. *The Quantitative Methods for Psychology* **12**. doi:10.20982/tqmp.12.3.p154.

51. Kadlec D, Sainani KL, & Nimphius S (2023) With great power comes great responsibility: Common errors in meta-analyses and meta-regressions in strength & conditioning research. *Sports Med* **53**, 313–325.

52. Van den Noortgate W, López-López JA, Marín-Martínez F, & Sánchez-Meca J (2013) Three-level meta-analysis of dependent effect sizes. *Behav Res* **45**, 576–594.

53. Cheung MW-L (2019) A guide to conducting a meta-analysis with non-independent effect sizes. *Neuropsychol Rev* **29**, 387–396.

54. Jukic I, Castilla AP, Ramos AG, Van Hooren B, McGuigan MR, & Helms ER (2023) The acute and chronic effects of implementing velocity loss thresholds during resistance training: A systematic review, meta-analysis, and critical evaluation of the literature. *Sports Med* **53**, 177–214.

55. Borg DN, Impellizzeri FM, Borg SJ, Hutchins KP, Stewart IB, Jones T *et al.* (2024) Meta-analysis prediction intervals are under reported in sport and exercise medicine. *Scandinavian Journal of Medicine & Science in Sports* **34**, e14603.

56. IntHout J, Ioannidis JPA, Rovers MM, & Goeman JJ (2016) Plea for routinely presenting prediction intervals in meta-analysis. *BMJ Open* **6**, e010247.

57. Nakagawa S, Noble DWA, Senior AM, & Lagisz M (2017) Meta-evaluation of meta-analysis: ten appraisal questions for biologists. *BMC Biol* **15**, 18.

58. Quintana DS (2023) A guide for calculating study-level statistical power for meta-analyses. *Advances in Methods and Practices in Psychological Science* **6**, 25152459221147260.

59. Hopkins WG (2018) Improving meta-analyses in sport and exercise science. *Sportscience* **22**, 11–19.

60. Deeks JJ, Higgins JP, Altman DG, & Group on behalf of the CSM (2019) Analysing data and undertaking meta-analyses. *In Cochrane Handbook for Systematic Reviews of Interventions*, pp. 241–284. John Wiley & Sons, Ltd.

61. Ruppar T (2020) Meta-analysis: How to quantify and explain heterogeneity? *European Journal of Cardiovascular Nursing* **19**, 646–652.

62. McKay AKA, Stellingwerff T, Smith ES, Martin DT, Mujika I, Goosey-Tolfrey VL *et al.* (2022) Defining training and performance caliber: A participant classification framework. *Int J Sports Physiol Perform* **17**, 317–331.

63. McArdle WD, Katch FI, & Katch VL (2010) *Exercise physiology: Nutrition, energy, and human performance*. Lippincott Williams & Wilkins.

64. Riebe D, Ehrman JK, Liguori G, & Magal M (2018) *ACSM's guidelines for exercise testing and prescription*. American College of Sports Medicine.

65. Harrell FE (2015) General aspects of fitting regression models. *In Regression Modeling Strategies: With Applications to Linear Models, Logistic and Ordinal Regression, and Survival Analysis*, pp. 13–44 [Harrell Jr Frank E, editor]. Cham: Springer International Publishing.

66. Zuur AF, Ieno EN, Walker N, Saveliev AA, & Smith GM (2009) *Mixed effects models and extensions in ecology with R*. New York, NY: Springer.

67. Wickham H (2011) ggplot2. *WIREs Computational Statistics* **3**, 180–185.

68. Peters JL, Sutton AJ, Jones DR, Abrams KR, & Rushton L (2008) Contour-enhanced meta-analysis funnel plots help distinguish publication bias from other causes of

asymmetry. *Journal of Clinical Epidemiology* **61**, 991–996.

69. Egger M, Smith GD, Schneider M, & Minder C (1997) Bias in meta-analysis detected by a simple, graphical test. *BMJ* **315**, 629–634.

70. Fernández-Castilla B, Declercq L, Jamshidi L, Beretvas SN, Onghena P, & Noortgate WV den (2021) Detecting selection bias in meta-analyses with multiple outcomes: A simulation study. *The Journal of Experimental Education*.

71. Sterne JAC, Sutton AJ, Ioannidis JPA, Terrin N, Jones DR, Lau J *et al.* (2011) Recommendations for examining and interpreting funnel plot asymmetry in meta-analyses of randomised controlled trials. *BMJ* **343**, d4002.

72. Duval S & Tweedie R (2000) Trim and fill: A simple funnel-plot–based method of testing and adjusting for publication bias in meta-analysis. *Biometrics* **56**, 455–463.

73. Deng H, Song T, Yin M, Xu K, Zhong Y, Liu P *et al.* (2025) Does one shot work? The acute impact of a single taurine dose on exercise performance: A meta-analytic review. *Scandinavian Journal of Medicine & Science in Sports* **35**, e70123.

74. Atkinson AC, Cook RD, & Weisberg S (1983) Residuals and Influence in Regression. *In Biometrics*, p. 818.

75. Viechtbauer W & Cheung MW-L (2010) Outlier and influence diagnostics for meta-analysis. *Research Synthesis Methods* **1**, 112–125.

76. Schünemann HJ, Higgins JP, Vist GE, Glasziou P, Akl EA, Skoetz N *et al.* (2019) Completing 'Summary of findings' tables and grading the certainty of the evidence. 375–402.

77. Sun X, Briel M, Walter SD, & Guyatt GH (2010) Is a subgroup effect believable? Updating criteria to evaluate the credibility of subgroup analyses. *BMJ* **340**, c117.

78. Wang R, Lagakos SW, Ware JH, Hunter DJ, & Drazen JM (2007) Statistics in medicine — Reporting of subgroup analyses in clinical trials. *New England Journal of Medicine* **357**, 2189–2194.

79. Bhagavan HN & and Chopra RK (2006) Coenzyme Q10: Absorption, tissue uptake, metabolism and pharmacokinetics. *Free Radical Research* **40**, 445–453.

80. Littarru GP & Tiano L (2007) Bioenergetic and antioxidant properties of coenzyme Q10: recent developments. *Mol Biotechnol* **37**, 31–37.

81. Gutierrez-Mariscal FM, Yubero-Serrano ,Elena M., Villalba ,Jose M., & and Lopez-Miranda J (2019) Coenzyme Q10: From bench to clinic in aging diseases, a translational review. *Critical Reviews in Food Science and Nutrition* **59**, 2240–2257.

82. Miles MV, Horn PS, Morrison JA, Tang PH, DeGrauw T, & Pesce AJ (2003) Plasma coenzyme Q10 reference intervals, but not redox status, are affected by gender and race in self-reported healthy adults. *Clinica Chimica Acta* **332**, 123–132.

83. Ventura-Clapier R, Moulin M, Piquereau J, Lemaire C, Mericskay M, Veksler V *et al.* (2017) Mitochondria: a central target for sex differences in pathologies. *Clinical Science* **131**, 803–822.

84. Holloszy JO & Coyle EF (1984) Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology* **56**, 831–838.

85. Niklowitz P, Sonnenschein A, Janetzky B, Andler W, & Menke T (2007) Enrichment of coenzyme Q10 in plasma and blood cells: defense against oxidative damage. *Int J Biol Sci* **3**, 257–262.

86. Braakhuis AJ & Hopkins WG (2015) Impact of Dietary Antioxidants on Sport Performance: A Review. *Sports Med* **45**, 939–955.

87. Hernández-Camacho JD, Bernier M, López-Lluch G, & Navas P (2018) Coenzyme Q10 supplementation in aging and disease. *Front Physiol* **9**. doi:10.3389/fphys.2018.00044.

88. Kalén A, Appelkvist E-L, & Dallner G (1989) Age-related changes in the lipid compositions of rat and human tissues. *Lipids* **24**, 579–584.

89. Mantle D, Heaton RA, & Hargreaves IP (2022) Coenzyme Q10, ageing and the nervous system: An overview. *Antioxidants* **11**, 2.

90. Peters R (2006) Ageing and the brain. *Postgraduate Medical Journal* **82**, 84–88.

91. Ravaglia G, Forti P, Maioli F, Scali RC, Boschi F, Cicognani A *et al.* (1996) Coenzyme Q10 plasma levels and body composition in elderly males. *Archives of Gerontology and Geriatrics* **22**, 539–543.

92. Del Pozo-Cruz J, Rodríguez-Bies E, Ballesteros-Simarro M, Navas-Enamorado I, Tung BT, Navas P *et al.* (2014) Physical activity affects plasma coenzyme Q10 levels differently in young and old humans. *Biogerontology* **15**, 199–211.

93. Wada H, Goto H, Hagiwara S-I, & Yamamoto Y (2007) Redox status of coenzyme Q10 is associated with chronological age. *Journal of the American Geriatrics Society* **55**, 1141–1142.

94. Cenini G, Lloret A, & Cascella R (2019) Oxidative stress in neurodegenerative diseases: From a mitochondrial point of view. *Oxidative Medicine and Cellular Longevity* **2019**, 2105607.

95. Short KR, Bigelow ML, Kahl J, Singh R, Coenen-Schimke J, Raghavakaimal S *et al.* (2005) Decline in skeletal muscle mitochondrial function with aging in humans. *Proceedings of the National Academy of Sciences* **102**, 5618–5623.

96. Langsjoen PH & Langsjoen AM (2014) Comparison study of plasma coenzyme Q10 levels in healthy subjects supplemented with ubiquinol versus ubiquinone. *Clin Pharmacol Drug Dev* **3**, 13–17.

97. Zhang Y, Liu J, Chen X-Q, & Oliver Chen C-Y (2018) Ubiquinol is superior to ubiquinone to enhance Coenzyme Q10 status in older men. *Food Funct* **9**, 5653–5659.

98. Östman B, Sjödin A, Michaëlsson K, & Byberg L (2012) Coenzyme Q10 supplementation and exercise-induced oxidative stress in humans. *Nutrition* **28**, 403–417.

99. López-Lluch G, Del Pozo-Cruz J, Sánchez-Cuesta A, Cortés-Rodríguez AB, & Navas P (2019) Bioavailability of coenzyme Q10 supplements depends on carrier lipids and solubilization. *Nutrition* **57**, 133–140.

100. Mantle D & Dybring A (2020) Bioavailability of coenzyme Q10: An overview of the absorption process and subsequent metabolism. *Antioxidants* **9**, 386.

101. Pravst I, Rodríguez Aguilera JC, Cortes Rodriguez AB, Jazbar J, Locatelli I, Hristov H *et al.* (2020) Comparative bioavailability of different coenzyme Q10 formulations in healthy elderly individuals. *Nutrients* **12**, 784.

102. Bonetti A, Solito F, Carmosino G, Bargossi A, & Fiorella pier luigi (2000) Effect of ubidecarenone oral treatment on aerobic power in middle-aged trained subjects. *The Journal of sports medicine and physical fitness* **40**, 51–7.

103. Braun B, Clarkson PM, Freedson PS, & Kohl RL (1991) Effects of coenzyme Q10 supplementation on exercise performance, VO2max, and lipid peroxidation in trained cyclists. doi:10.1123/ijsn.1.4.353.

104. Weston SB, Zhou S, Weatherby RP, & Robson SJ (1997) Does exogenous coenzyme Q10 affect aerobic capacity in endurance athletes. *Int J Sport Nutr* **7**, 197–206.

105. Yanprechaset T, Kanungsukkasem V, & Kaimuk P (2015) The role of coenzyme Q10 and circuit resistance training on energy expenditure at rest and during submaximal treadmill walking in 40-60 year-old overweight women. *Journal of Health Research* **29**, 37–45.

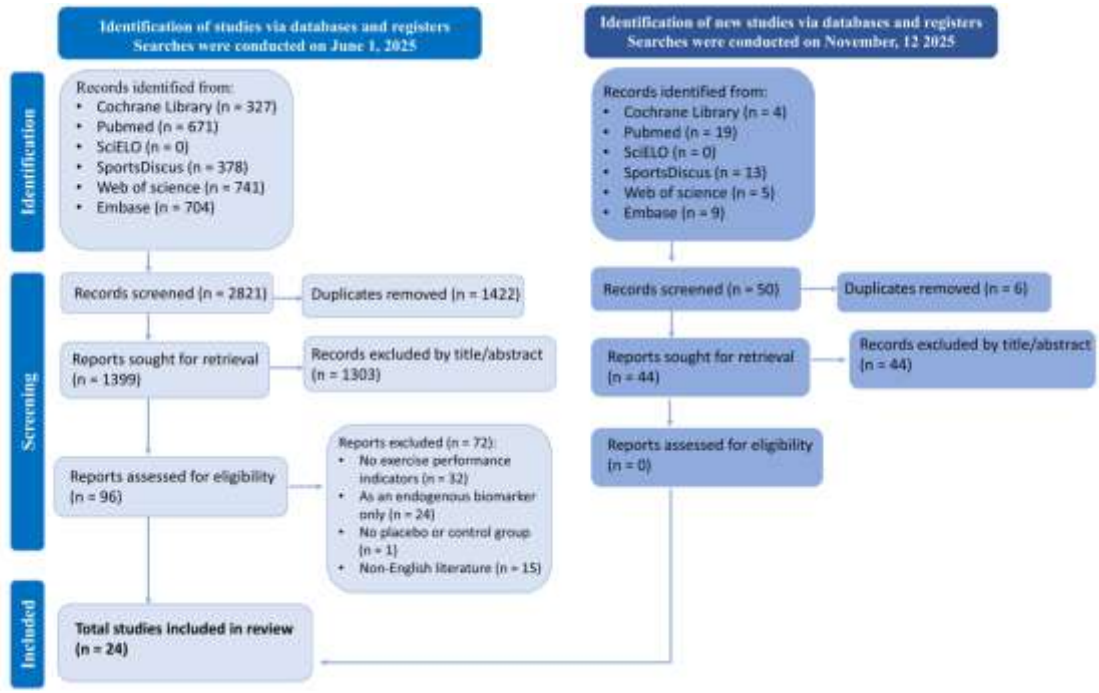

Figure. 1 PRISMA flow diagram for included and excluded studies

Notes: Flow of records through identification, screening, eligibility, and inclusion for the review and meta-analysis. Boxes report numbers at each stage and main reasons for exclusion at full-text screening. Searches were conducted on 1 June 2025 and updated on 12 November 2025.

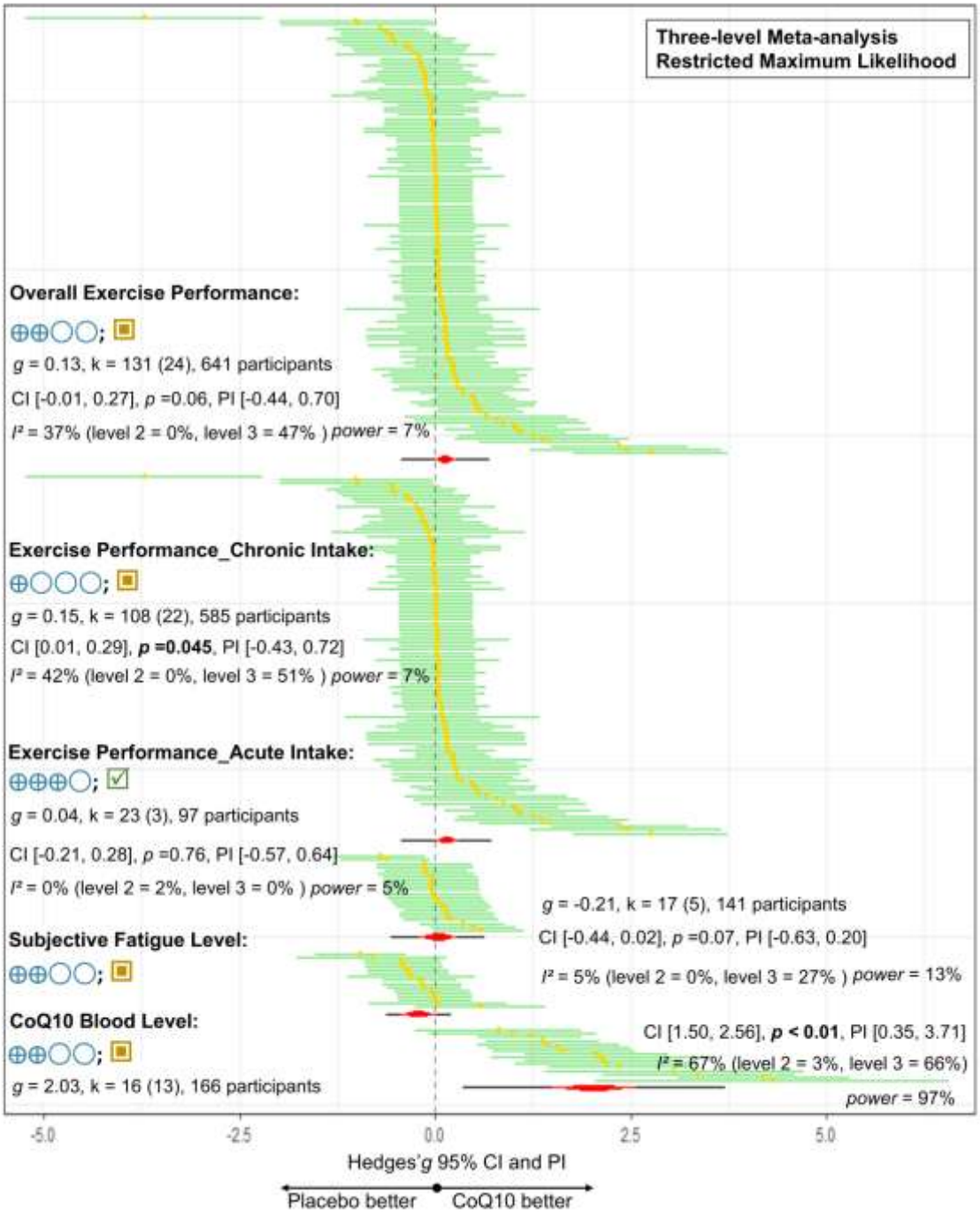

Figure 2. Primary pooled effect sizes of CoQ10 supplementation on exercise performance (Acute and Chronic), subjective fatigue, and blood CoQ10

Notes: Three-level random-effects estimates are shown as standardised mean differences (Hedges' g) with 95% confidence intervals, 95% prediction intervals and P values. Positive values favour CoQ10 for performance and blood CoQ10. Negative values favour CoQ10 for subjective fatigue (lower perceived exertion). Labels report k (number of effects), n (number of participants), I^2 (heterogeneity), and statistical power for each outcome. GRADE ratings (Blue Circles) indicate the certainty of evidence for each pooled estimate. RoB-2 (squares; green--low risk, yellow--some concerns, and red-- high risk) denotes domain-level risk-of-bias summaries for each subgroup.

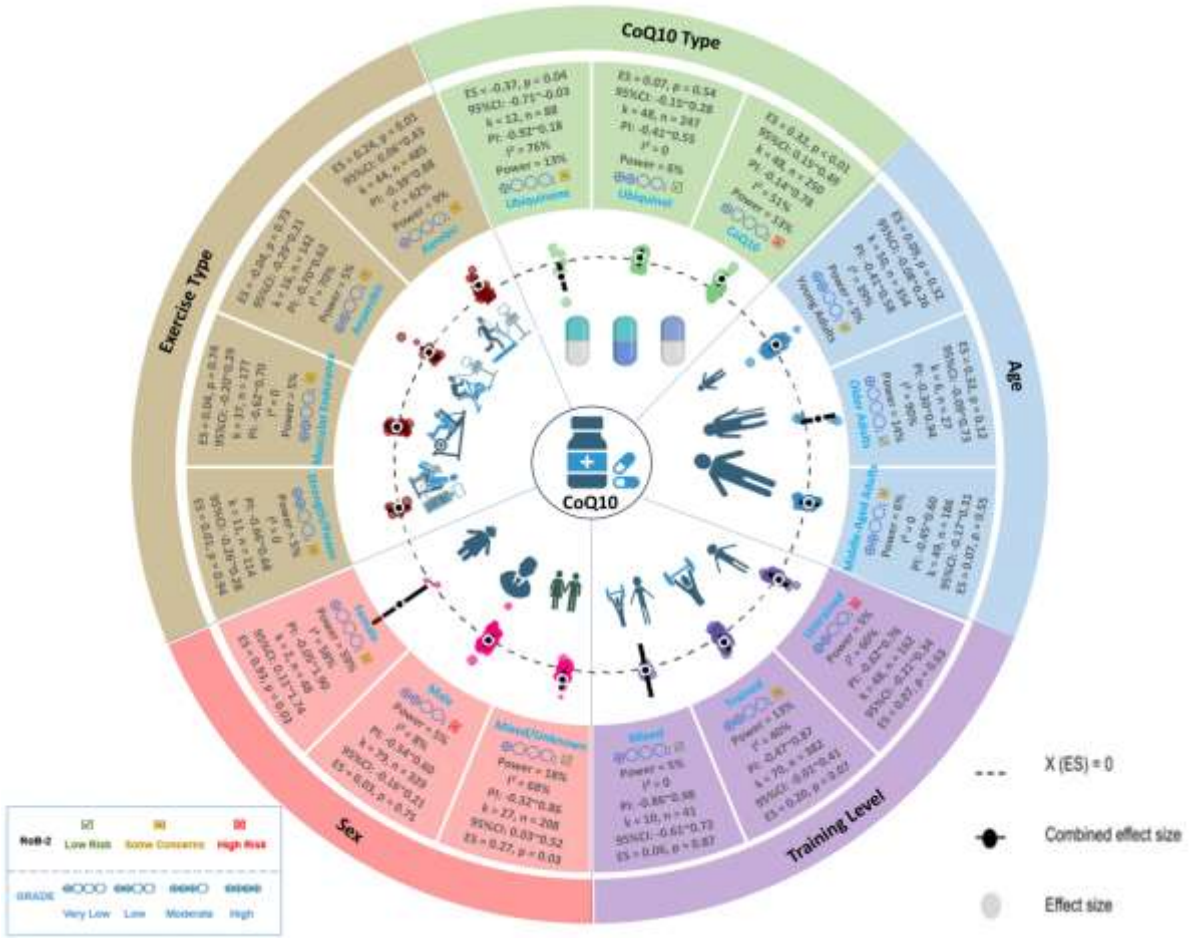

Figure 3. Moderator analyses under chronic supplementation

Notes: Subgroup effects for sex, training status, age, Coenzyme Q10 (CoQ10) type, and exercise domain are displayed as Hedges' g with 95% confidence intervals, 95% prediction intervals and P values. Labels report k (number of effects), n (number of participants), I^2 (heterogeneity), and statistical power for each outcome. GRADE ratings (blue circles) indicate the certainty of evidence for each pooled estimate. RoB-2 (squares; green--low risk, yellow--some concerns, and red-- high risk) denotes domain-level risk-of-bias summaries for each subgroup. Analyses were pre-specified and restricted to chronic trials to reflect pharmacokinetic differences and the predominance of chronic protocols.

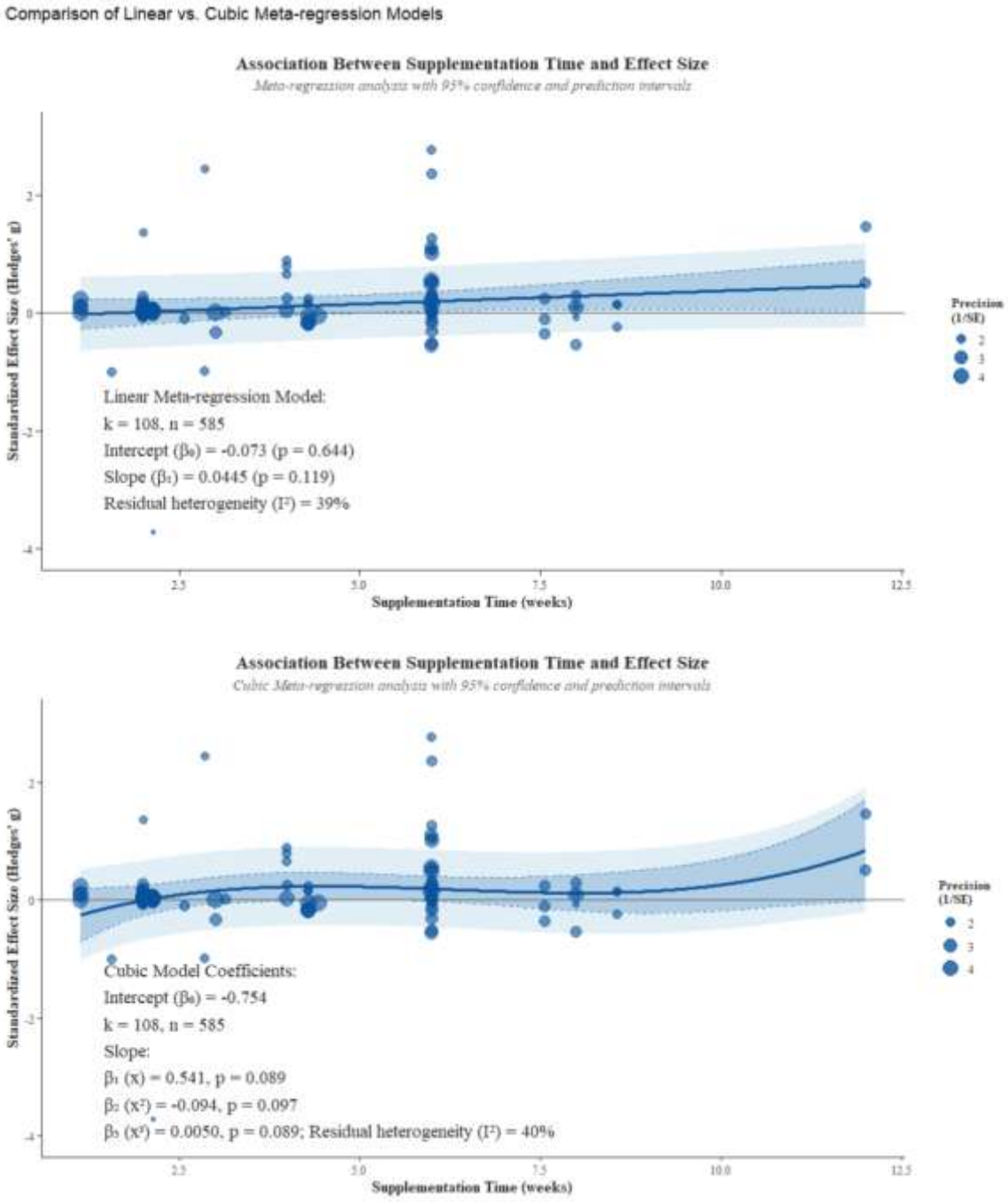

Figure 4. Meta-regression of chronic supplementation duration on exercise performance

Notes: Predicted effects (Hedges' g) across supplementation duration (weeks) under linear and cubic models, with 95% confidence bands (light blue shaded area) and prediction intervals (outermost blue dotted lines). The solid line shows the fitted regression line. Each point represents a separate effect size (k), which size varies according to the weight. k (number of effects), n (number of participants), β_0 (intercept), slopes (β_1, β_2, β_3), I^2 (heterogeneity). P values refer to the statistical significance of the association between supplementation duration and effect size. No statistically significant dose–duration relationship was detected.

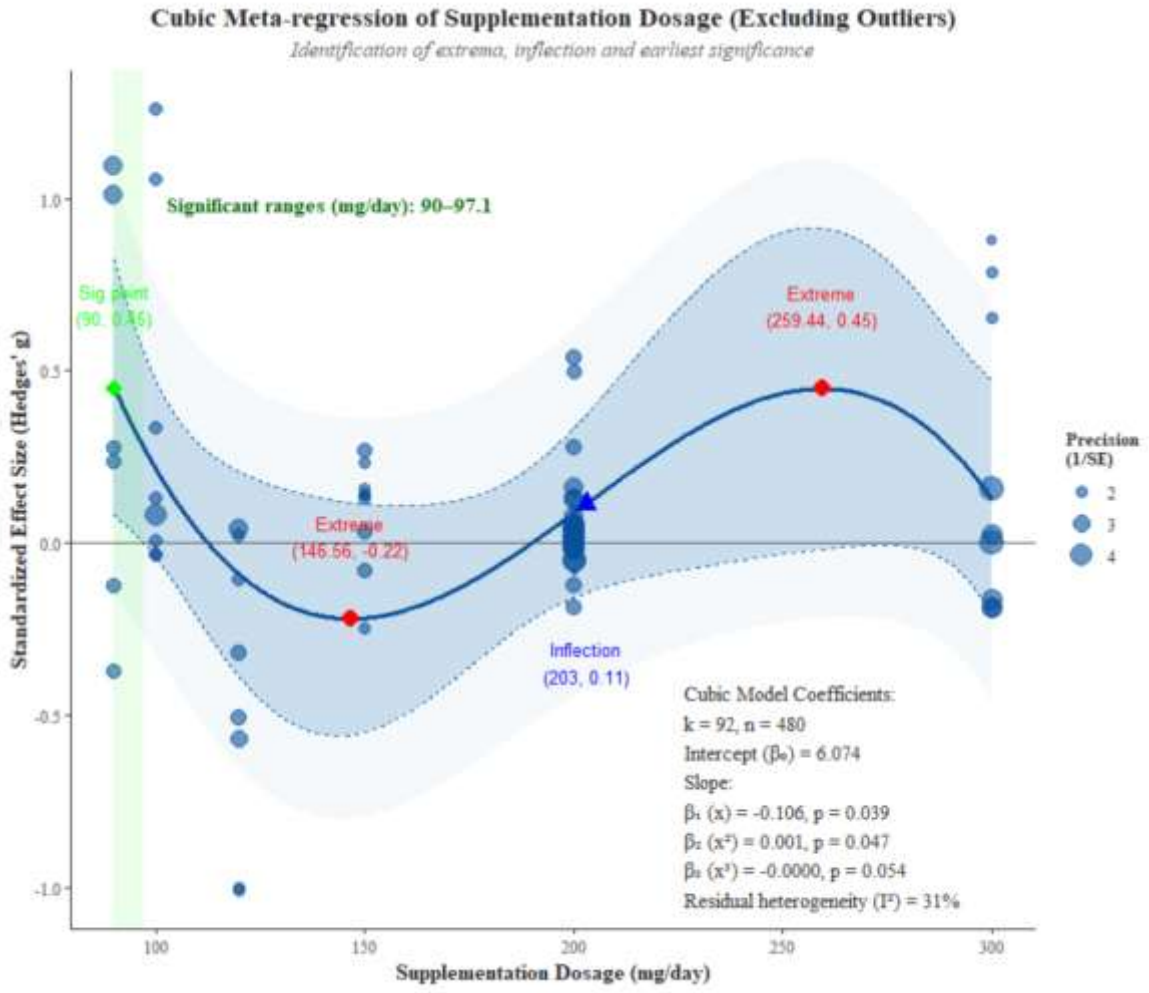

Figure 5. Cubic meta-regression of chronic supplementation dosage on exercise performance after outlier exclusion

Notes: After excluding outliers, predicted effects (Hedges' g) across daily dose (mg/day) under cubic models, with 95% confidence bands (light blue shaded area) and prediction intervals (outermost blue dotted lines). The solid line shows the fitted regression line. Each point represents a separate effect size (k), which size varies according to the weight. k (number of effects), n (number of participants), β_0 (intercept), slopes (β_1, β_2, β_3), I^2 (heterogeneity). The first significant point (Sig point), the maximum and minimum values within the model range (Extreme), dose efficiency inflection point (Inflection point). P values refer to the statistical significance of the association between supplementation dose and effect size. Green shaded regions mark dose ranges that reached nominal significance in the primary model (90-97.1 mg/day). Results are exploratory and should be interpreted cautiously.

Table 1. Summary and results of the studies reviewed assessing the effect of coenzyme Q10 supplementation on exercise performance.

Study; Design	Exercise protocol	Sample; Mean age (y), body weight (kg); Training status	Dosage of CoQ10 and PLA; Form; Duration (post-test timepoints);	Outcomes	Statistical significance
Alf et al. (2013) RDB Parallel	Graded exercise test on cycling ergometer	100 (53 males and 47 females) and divided into 2 groups (50/group, but PLA group withdrew 2); 19.9 ± 2.3, 78 ± 19.7; Elite athletes	CoQ10: 300 mg/day OL; PLA: 300 mg/day lactose and MCT oil; Capsule; 42 days (21st day, 42nd day test)	Pre- and post- 1 and post 2 peak power output (W/kg): OL: 3.70 ± 0.56, 3.81 ± 0.53 and 4.08 ± 0.48 vs PLA: 3.64 ± 0.49, 3.75 ± 0.49 and 3.94 ± 0.47	Performance: Yes ↑ (p = 0.03)
Bloomer et al. (2012) RDB Crossover	Graded exercise test on treadmill; Cycle sprint test on ergometer	15 (10 males, 5 females); 42.7 ± 10.4; 75.2 ± 16.3; Physically active	CoQ10: 300 mg/day OL; PLA: MCT oil, beeswax, soy lecithin, ascorbyl palmitate; Capsule; 30 days (28th day running test, 30th day cycling test), 21-day washout between CoQ10 and PLA	Time of running (s): OL: 1324.5 ± 268.40 vs PLA: 1317.3 ± 240.13 RPE of cycling (3 timepoints; 6-20): OL: 11.4 ± 1.55, 13.9 ± 2.32, 19.5 ± 0.77 vs PLA: 11.9 ± 1.16, 14.3 ± 2.32, 19.7 ± 0.04 Peak power of cycling (W): OL: 875 ± 245.16 vs PLA: 914.7 ± 221.53 Mean power of cycling (W): OL: 483.3 ± 136.33 vs PLA: 511.0 ± 142.91 Total work of cycling (kJ): OL: 4833.1 ± 1361.74 vs PLA: 5109.8 ± 1428.74 Pre- and post- blood CoQ10 (μg·mL^{-1}) OL: 0.98 ± 0.39 and 2.33 ± 1.63 vs PLA: 1.02 ± 0.43 and 0.99 ± 0.35	Performance: No (p > 0.05); RPE: No (p > 0.05) Blood CoQ10: Yes ↑ (p = 0.02)
Bonetti et al. (2000)	Graded exercise test on cycling	28 males and divided into 2 groups (14/group, but CoQ10 group	CoQ10: 100 mg/day ONE; PLA: NS but similar	Pre- and post-VO_{2peak} (mL·min^{-1}·kg^{-1}): ONE: 54.6 ± 8.0 and 54.7 ± 8.1 vs PLA:	Performance: No

RDB Parallel	ergometer	withdrew 3, PLA group withdrew 2); CoQ10 group: 40.6 ± 6.0, 68.0 ± 3.8, PLA group: 40.1 ± 7.4, 67.1 ± 5.8; Well-trained (Cycling 2314 ± 311 km/month)	appearance and taste; Capsule; 56 days (20 hours after last supplementation test)	53.0 ± 8.5 and 57.7 ± 7.8 Pre- and post- blood CoQ10 ($\mu g \cdot mL^{-1}$): ONE: 0.78 ± 0.16 and 2.23 ± 1.25 vs PLA: 0.85 ± 0.31 and 1.03 ± 0.35	($p > 0.05$); Blood CoQ10: Yes ↑ ($p < 0.02$)
Braun et al. (1991) RDB Parallel	Graded exercise test on cycling ergometer	12 males and divided into 2 groups (6/group, but CoQ10 each group withdrew 1) 21.9 ± 1.8, 72.6 ± 8.3; Collegiate cyclists (Cycling 4.1 ± 3.2 years and 248 ± 62.7 miles/week)	CoQ10: 200 mg/day CoQ10; PLA: Cornmeal and similar appearance; Capsule; 56 days (56^{th} day test)	Pre- and post-total work of cycling (kJ): CoQ10: 232 ± 87.21 and 274 ± 96.15 vs PLA: 253 ± 55.90 and 302 ± 60.37 Pre- and post-VO_{2peak} ($L \cdot min^{-1}$) CoQ10: 4.38 ± 0.69 and 4.67 ± 0.74 vs PLA: 4.54 ± 0.38 and 4.77 ± 0.42 Pre- and post- blood CoQ10 ($\mu g \cdot mL^{-1}$): CoQ10: 0.84 ± 0.13 and 1.51 ± 0.20 vs PLA: 0.81 ± 0.11 and 0.78 ± 0.11	Performance: No ($p > 0.05$); Blood CoQ10: Yes ↑ ($p < 0.01$)
Cooke et al. (2008) RDB Parallel	Isokinetic knee extension endurance test; 30-s Wingate test; Graded exercise test on treadmill	41 (gender NS) and divided into 2 groups (CoQ10 group 21, PLA group 20); 21.6 ± 7.6, 73.5 ± 17; 22 aerobically trained, 19 untrained	CoQ10: 200 mg/day CoQ10; PLA: Dextrose and similar appearance; Fast-melt tablet; 14 days (1^{st} day within 60 min ingestion and after 14^{th} day test)	Pre-, acute and post- extensor peak power (N/m): CoQ10: 132 ± 42, 127 ± 37 and 132 ± 34 vs PLA: 110 ± 38, 110 ± 34 and 107 ± 35 Pre-, acute and post- flexor peak power (N/m): CoQ10: 72 ± 28, 75 ± 28 and 73 ± 24 vs PLA: 65 ± 26, 66 ± 28 and 65 ± 28 Pre-, acute and post- extensor total work (N/m): CoQ10: 4047 ± 1218, 3955 ± 1114 and 4073 ± 1116 vs PLA: 3391 ± 992, 3352 ± 936 and 3242 ± 921	Performance: No ($p > 0.05$); Blood CoQ10: Yes, only with CoQ10 group between pre- and post- test ↑ ($p < 0.01$); Muscle CoQ10: No ($p > 0.05$)

Pre-, acute and post- flexor total work (N/m): CoQ10: 2213 ± 847, 2203 ± 877 and 2230 ± 822 vs PLA: 1962 ± 891, 1996 ± 963 and 1866 ± 839

Pre-, acute and post- extensor average power (W): CoQ10: 117 ± 36, 115 ± 34 and 117 ± 33 vs PLA: 95 ± 30, 95 ± 27 and 95 ± 30

Pre-, acute and post- flexor average power (W): CoQ10: 61 ± 25, 62 ± 26 and 62 ± 24 vs PLA: 52 ± 25, 54 ± 27 and 52 ± 26

Pre-, acute and post- peak power of Wingate test (W): CoQ10: 1162 ± 436, 1169 ± 421 and 1209 ± 420 vs PLA: 1013 ± 417, 1079 ± 416 and 1069 ± 400

Pre-, acute and post- mean power of Wingate test (W): CoQ10: 592 ± 210, 594 ± 209 and 603 ± 211 vs PLA: 535 ± 181, 545 ± 180 and 548 ± 181

Pre-, acute and post- VO_{2amx} ($mL \cdot min^{-1} \cdot kg^{-1}$): CoQ10: 42.3 ± 9.1, 42.9 ± 8.5 and 42.1 ± 8.5 vs PLA: 43.9 ± 9.0, 45.5 ± 7.9 and 43.4 ± 9.6

Pre-, acute and post- TTE (min): CoQ10: 12.9 ± 2.4, 13.1 ± 2.8 and 13.3 ± 2.3 vs PLA: 13.6 ± 2.3, 13.8 ± 2.1 and 13.7 ± 2.4

				Pre- and post- blood CoQ10 ($\mu g \cdot mL^{-1}$): CoQ10: 0.61 ± 0.46 and 2.34 ± 0.33 vs PLA: 0.75 ± 0.47 and 0.70 ± 0.34 Pre- and post- muscle CoQ10 ($\mu g \cdot mg^{-1}$ muscle): CoQ10: 1.19 ± 2.05 and 1.25 ± 1.46 vs PLA: 1.60 ± 1.54 and 1.38 ± 1.28	
Deichmann et al. (2012) RDB Crossover	Graded exercise test on treadmill; Leg extension test	20 (15 males and 5 females), but withdrew 1 female; 63.6 ± 8.2, NS; Older athletes (Training 5 times/weeks)	CoQ10: 200 mg/day CoQ10; PLA: 200 mg/day starch and similar appearance; Capsule; 42 days (42^{nd} day test)	Pre- and post- change of VO_2 at ANT ($ml.kg^{-1}.min^{-1}$): CoQ10: 2.34 ± 0.80 vs PLA: -0.59 ± 1.20 Pre- and post- change of time to ANT (s): CoQ10: 40.26 ± 15.70 vs PLA: 0.58 ± 16.50 Pre- and post- change of VO_{2max} ($ml.kg^{-1}.min^{-1}$): CoQ10: 1.13 ± 3.6 vs PLA: 0.35 ± 3.8 Pre- and post- change of leg extension repetitions (times): CoQ10: 3.78 ± 5.00 vs PLA: 1.73 ± 2.90	Performance: Yes, only with CoQ10 vs PLA on time to ANT and leg extension repetitions ↑ ($p < 0.05$)
Ebrahimi & and Ladary (2016) RDB Crossover	Handgrip dynamometer; Chin-up; Illinois agility run test; 35 m sprint; RAST	14 males; 20.4 ± 3.0, 73.4 ± 11.6; Elite wrestlers	CoQ10: 200 mg ONE; PLA: Starch and similar appearance; Capsule; Acute ingestion before 2-hour test, and with 1 week wash out	Handgrip test (kg): ONE: 55.80 ± 8.19 vs PLA: 56.80 ± 8.44 Chin-up test (repetitions): ONE: 19.86 ± 7.56 vs PLA: 18.57 ± 10.01 35-meter run test (s): ONE: 5.34 ± 0.26 vs PLA: 5.57 ± 0.35 Illinois Agility Run test (s): ONE: 17.92 ± 0.73 vs PLA: 18.53 ± 1.06 RAST peak power (W): ONE: 554.25 ±	Performance: Yes, only with agility test, 35 m sprint and mean and minimum power of RAST ↑ ($p < 0.05$)

				111.67 vs PLA: 510.86 ± 121.88 RAST mean power (W): ONE: 425.39 ± 71.87 vs PLA: 386.92 ± 75.90 RAST minimum power (W): ONE: 322.11 ± 61.92 vs PLA: 284.41 ± 61.51	
Gharahdaghi et al. (2013) RDB Parallel	Graded exercise test on treadmill; RAST; Hoff football test	16 males and divided into 2 groups (CoQ10 group 10, PLA group 6); 21.9 ± 1.6, 67.1 ± 6.6; Soccer player (at least 2 years trained experience)	CoQ10: 300 mg/day OL; PLA: 300 mg/day wheat flour and similar appearance; Capsule; 28 days (28th day test)	Pre- and post-VO_{2max} ($mL \cdot min^{-1} \cdot kg^{-1}$): OL: 52.27 ± 3.21 and 53.63 ±2.54 vs PLA: 51.28 ± 1.88 and 50.80 ± 2.24 Pre- and post-RAST peak power (W): OL: 498.81 ± 119.57 and 557.54 ± 99.17 vs PLA: 535.5 ± 111.28 and 506.2 ± 59.30 Pre- and post-Hoff football test (m): OL: 1374.4 ± 95.13 and 1421.5 ±72.61 vs PLA: 1361.66 ± 30.76 and 1342.16 ± 35.55	Performance: Yes, only with post- vs pre- on VO_{2max} and Hoff football test ↑ ($p < 0.05$)
Gökbel et al. (2010) RDB Crossover	5 * 30 s Wingate tests with 2 minutes interval	15 males; 19.9 ± 0.9, 72.1 ± 12.9; Healthy, nonsmoker, and sedentary	CoQ10: 100 mg/day CoQ10; PLA: 100 mg/day glucose and similar appearance; Capsule; 56 days (56th day test), 28-day washout between CoQ10 and PLA	Average power output Between CoQ10 group and PLA group (W): 19.51 ± 224.01	NS
Kunching et al. (2022) RDB Parallel	1RM bench press; Push up; Vertical jump; 40-yard sprint;	29 males and divided into 2 groups (CoQ10 group 15, PLA group 14); CoQ10 group: 23.5 ± 8.1, 70.1 ± 5.4, PLA group: 22.8 ± 8.6, 66.7 ± 4.9;	CoQ10: 200 mg/day OL; PLA: 200 mg/day maltodextrin and similar appearance; Capsule; 42 days (42nd day test)	Pre- and post- 1RM bench press (kg): OL: 84.2 ± 10.07 and 83.3 ± 8.91 vs PLA: 79.1 ± 10.48 and 80.1 ± 10.10 Pre- and post- push up (repetitions/2min): OL: 57.4 ± 12.78	Performance: Yes, only with OL vs PLA on VO_{2max} ↑ ($p < 0.05$)

	1.5 mile running test	Healthy trained		and 56.5 ± 12.39 vs PLA: 52.6 ± 15.00 and 53.4 ± 13.94 Pre- and post- jump height (cm): OL: 37.7 ± 2.71 and 38.3 ± 2.32 vs PLA: 39.1 ± 1.87 and 39.0 ± 2.62 Pre- and post- 40-yard sprint time (s): OL: 7.1 ± 1.16 and 7.2 ± 0.77 vs PLA: 6.5 ± 1.12 and 6.6 ± 0.75 Pre- and post- VO_{2amx} ($mL \cdot min^{-1} \cdot kg^{-1}$): OL: 42.1 ± 1.55 and 42.4 ± 1.16 vs PLA: 42.8 ± 1.50 and 42.3 ± 1.50	
Laaksonen et al. (1995) RDB Crossover	Graded exercise test on cycling ergometer with TTE	19 males (11 young and 8 older); Young: 28.4 ± 13.53, 72.8 ± 11.84 Older: 63.8 ± 10.09, 70.0 ± 16.59; Healthy trained (All have experience in marathon/triathlon)	CoQ10: 120 mg/day ONE; PLA: NS; Capsule; 42 days (42^{nd} day test), 28-day washout between CoQ10 and PLA	VO_{2max} ($mL \cdot min^{-1} \cdot kg^{-1}$): Young: ONE: 59.00 ± 12.34 vs PLA: 58.50 ± 9.31, older: ONE: 33.70 ± 11.24 vs PLA: 37.20 ± 7.93 TTE (min): Young: ONE: 82.10 ± 6.16 vs PLA: 85.70 ± 5.59, older: ONE: 77.20 ± 9.85 vs PLA: 82.90 ± 10.16 Pre- and post- blood CoQ10 ($\mu mol \cdot L^{-1}$): Young: CoQ10: 0.92 ± 0.28 and 1.98 ± 0.90 vs PLA: 1.05 ± 0.40 and 0.97 ± 0.45, older: CoQ10: 1.32 ± 0.49 and 3.49 ± 0.90 vs PLA: 1.23 ± 0.37 and 1.26 ± 0.43	Performance: NO ($p > 0.05$); Blood CoQ10: Yes, only with ONE vs PLA and older vs young ↑ ($p < 0.05$)
Malm et al. (1997) RDB Parallel	30-s Wingate test; 10 * 10 s cycling sprints;	18 males and divided into 2 groups (CoQ10 group 9, PLA group 9); CoQ10 group: 27.8 ± 4.8, 79.6 ± 8.8,	CoQ10: 120 mg/day ONE; PLA: equivalent but NS; NS; 22 days (11^{th}, $15^{th,}$ and 20^{th} day	Pre- and 20^{th} day post- power of 30-s Wingate test ($W \cdot kg^{-1}$): ONE: 9.03 ± 0.90 and 8.01 ± 0.90 vs PLA: 9.16 ± 0.78 and 9.04 ± 0.84	Performance: Yes, only with the change between pre- and

	Cycling VO_2max test; Running Vo_{2max} test	PLA group: 22.8 ± 2.0, 76.5 ± 3.6; Healthy	test 30-s Wingate and 10 * 10 cycling sprints; 18th day test cycling VO_2max; 22nd day test running Vo_{2max})	Changes between pre- and 11th, 15th and 20th (3 timepoints) post- average power of 10 * 10 s cycling sprints ($W \cdot kg^{-1}$): ONE: -0.1 ± 0.27 vs PLA: 0.17 ± 0.24, ONE: -0.92 ± 0.36 vs PLA: 0.23 ± 0.21, ONE: 0.76 ± 0.27 vs PLA: 0.14 ± 0.21 Pre- and 18th day post- cycling Vo_{2max} ($mL \cdot min^{-1}$): ONE: 4705 ± 675 and 4649 ± 630 vs PLA: 4438 ± 789 and 4365 ± 633 Pre- and post- 22nd day post- running Vo_{2max} ($mL \cdot min^{-1}$): ONE: 4789 ± 654 and 4669 ± 834 vs PLA: 4621 ± 576 and 4577 ± 633 Pre- and 18th day post- RPE at 75% submaximal cycling (0-20): ONE: 14.9 ± 0.5 and 14.2 ± 0.4 vs PLA: 15.4 ± 0.2 and 15.0 ± 0.2	15th, 20th post- average power of 10 * 10 s cycling sprints ↑ ($p < 0.05$); RPE: No ($p > 0.05$)
Mieszkowski et al. (2024) RDB Parallel	Graded exercise test on treadmill	28 males and divided into 2 groups (CoQ10 group 14, PLA group 14); CoQ10 group: 20.1 ± 1.1, 77.7 ± 7.1, PLA group: 20.3 ± 1.6, 79.9 ± 7.8; Healthy physically active (Attend recreational sports 2~3 times/week)	CoQ10: 300 mg/day ONE; PLA: Rice-flavoured gelatine and similar appearance; Capsule; 21 days (21st day test)	Pre- and post- VO_{2amx} ($mL \cdot min^{-1} \cdot kg^{-1}$): ONE: 57.2 ± 8.0 and 55.9 ± 7.9 vs PLA: 51.3 ± 9.4 and 52.8 ± 7.5 Pre- and post- blood CoQ10 ($\mu g \cdot mL^{-1}$): ONE: 0.90 ± 0.32 and 2.49 ± 1.18 vs PLA: 0.85 ± 0.18 and 0.83 ± 0.17	Performance: No ($p > 0.05$); Blood CoQ10: Yes ↑ ($p < 0.05$)
Mizuno et al. (2008) RDB	2 * 10 s maximum velocity test on	17 (8 males and 9 females); 37.5 ± 9.9, 58.3 ± 11.3; Healthy	CoQ10: 100 mg/day and 300 mg/day CoQ10; PLA: MCT oil and glyceryl	First maximum velocity (rpm): 100 mg/day CoQ10: 74.20 ± 21.02 vs 300 mg/day CoQ10: 76.43 ± 21.66 vs PLA:	Performance: Yes, only with 300mg/day vs

Crossover	cycling ergometer		fatty acid esters with similar appearance; Capsule; 8 days (8th day, after taking an additional 3 capsules before each test), with 4-week wash out across three conditions	74.52 ± 22.93 Second maximum velocity (rpm): 100 mg/day CoQ10: 72.29 ± 23.57 vs 300 mg/day CoQ10: 74.84 ± 21.02 vs PLA: 69.75 ± 21.33 Blood CoQ10 across three conditions (μg·mL^{-1}): 100 mg/day CoQ10: 1.96 ± 0.79 vs 300 mg/day CoQ10: 3.33 ± 1.10 vs PLA: 0.54 ± 0.18 Baseline, fatigue, recovery SFS (0-100): 100 mg/day CoQ10: 38.00 ± 20.05 vs 300 mg/day CoQ10: 34.01 ± 16.81 vs PLA: 41.95 ± 18.67; 100 mg/day CoQ10: 72.30 ± 15.87 vs 300 mg/day CoQ10: 63.42 ± 17.05 vs PLA: 78.13 ± 11.91; 100 mg/day CoQ10: 56.89 ± 17.97 vs 300 mg/day CoQ10: 50.82 ± 20.54 vs PLA: 59.69 ± 21.48	PLA on change of velocity ↑ ($p < 0.05$); Blood CoQ10: Yes ↑ ($p < 0.01$); SFS: Yes, 300 mg/day vs PLA during fatigue and recovery phases ↓ ($p < 0.05$)
Mohammadi et al. (2020) RNS Parallel	RAST; Cooper test; Curl-up test; Press-up test; Chin-up test	20 males and divided into 2 groups (CoQ10 group 10 PLA group 10); CoQ10 group: 18.0 ± 1.0, 81.0 ± 1.0, PLA group: 18.5 ± 1.0, 28.75 ± 1.0; Young trained wrestlers (Trained 12 ± 9 years)	CoQ10: 100 mg/day CoQ10; PLA: Wheat flour and food coloring with similar appearance; Capsule; 42 days (42nd day test)	Pre- and post- change of RAST maximum power (W): CoQ10: 2.62 ± 186.49 vs PLA: 1.13 ± 198.43 Pre- and post- change of RAST average power (W): CoQ10: 2.60 ± 208.26 vs PLA: 9.58 ± 160.50 Pre- and post- change of RAST minimum power (W): CoQ10: 2.76 ± 226.67 vs PLA: 9.58 ± 160.50 Pre- and post- change of VO_{2amx}	Performance: Yes, except curl-up and chin-up test on post vs pre of CoQ10 group ↑, except chin- up test on post-test of CoQ10 vs PLA ↑

				($mL \cdot min^{-1} \cdot kg^{-1}$): CoQ10: 6.51 ± 4.06 vs PLA: 2.60 ± 1.11 Pre- and post- change of curl-up test (times): CoQ10: 7.20 ± 1.59 vs PLA: 3.70 ± 4.20 Pre- and post- change of press-up test (times): CoQ10: 7.20 ± 4.18 vs PLA: 6.20 ± 9.53 Pre- and post- change of chin-up test (times): CoQ10: 6.30 ± 2.59 vs PLA: 4.70 ± 5.95	($p < 0.05$)
Moreno-Fernandez et al. (2023) RDB Parallel	Circuit weight training and test	100 males and divided into 2 groups (50/group, but each group withdrew 16); CoQ10 group: 38.9 ± 9.9, 76.8 ± 9.9, PLA group: 38.2 ± 8.5, 76.3 ± 14.1; Healthy and well-trained firemen	CoQ10: 200 mg/day OL; PLA: Canola oil, diglycerol monooleate, beeswax, and soy lecithin with similar appearance; Capsule; 14 days (14^{th} day test and repeat the test after 24 hours)	Bench press maximum speed power (8 timepoints) (m/s): OL: 0.85 ± 1.98, 0.76 ± 1.98, 0.74 ± 2.33, 0.64 ± 2.16, 0.79 ± 1.75, 0.75 ± 1.98, 0.70 ± 1.87, 0.61 ± 1.87 vs PLA: 0.76 ± 1.52, 0.72 ± 1.98, 0.72 ± 1.98, 0.61 ± 2.10, 0.75 ± 1.63, 0.70 ± 2.33, 0.67 ± 2.45, 0.59 ± 2.45 Bench press maximum force (8 timepoints) (N): OL: 372.50 ± 761.35, 376.52 ± 696.39, 397.77 ± 631.49, 391.70 ± 696.39, 384.62 ± 513.42, 379.55 ± 601.99, 378.54 ± 413.12, 369.43 ± 513.47 vs PLA: 383.60 ± 861.52, 385.63 ± 661.00, 376.52 ± 531.20, 370.45 ± 460.35, 362.35 ± 537.09, 368.42 ± 542.92, 383.60 ± 607.88, 363.36 ± 537.03	Performance: Yes, OL vs PLA on overall conditions ↑ ($p < 0.05$); SFS: No ($p > 0.05$)

				Bench press power (8 timepoints) (W): OL: 759.15 ± 2506.61, 774.39 ± 1884.39, 759.15 ± 2151.03, 740.85 ± 2222.18, 719.51 ± 1919.90, 728.66 ± 1777.74, 682.93 ± 1688.88, 689.02 ± 1546.60 vs PLA: 728.66 ± 1866.60, 719.51 ± 1279.95, 719.51 ± 1866.66, 667.68 ± 1617.74, 664.63 ± 1386.66, 655.49 ± 1404.44, 679.88 ± 1635.52, 634.15 ± 1386.66 Average load (4 timepoints) (kg): OL: 37.62 ± 48.16, 38.76 ± 69.91, 37.62 ± 40.93, 38.55 ± 47.00 vs PLA: 37.93 ± 47.00, 37.83 ± 66.94, 36.07 ± 43.70, 36.07 ± 47.58 Average repetitions (4 timepoints) (times): OL: 17.55 ± 31.78, 17.85 ± 30.61, 18.65 ± 30.03, 18.15 ± 29.45 vs PLA: 16.40 ± 30.32, 16.60 ± 33.24, 18.00 ± 31.20, 17.75 ± 32.65 SFS (0-10): OL: 7.34 ± 8.51, 7.96 ± 11.60, 7.51 ± 8.92, 7.99 ± 8.22 vs PLA: 7.68 ± 8.92, 8.04 ± 9.62, 7.58 ± 7.70, 8.01 ± 8.05	
Orlando et al. (2018) RDB Crossover	Submaximal endurance treadmill test at 85% HRmax	21 males; 26 ± 5, NS; Healthy and trained rugby athletes	CoQ10: 200 mg/day OL; PLA: Soya lecithin, MCT oil with similar appearance; Capsule;	Pre- and post- average speed (km/h): OL: 10.62 ± 3.44 and 10.63 ± 3.35 vs PLA: 10.53 ± 3.57 and 10.74 ± 4.39 Pre- and post- time of 75% maximum	Performance: No ($p > 0.05$)

			30 days (30th day test)	speed (s): OL: 34.33 ± 27.58 and 33.96 ± 19.83 vs PLA: 35.49 ± 18.80 and 36.20 ± 23.95	
Östman et al. (2012) RDB Parallel	Graded exercise test on cycling ergometer; 3 * 10 min maximal ergometer cycling; 15 * 3 min ergometer cycling * 2; 60-minutes treadmill running	25 males and divided into 2 groups (CoQ10 group 13, PLA group 12, but each group withdrew 1); CoQ10 group: 28.2 ± 8.8, 71.0 ± 6.6, PLA group: 25.3 ± 5.7, 76.0 ± 8.7; Healthy and moderately trained (Trained 3-5 times/week)	CoQ10: 90 mg/day CoQ10; PLA: Soy oil with similar appearance; Capsule; 56 days (Threshold testing 3-5 days before 56th day, and specific exercise testing 1-2 days after 56th day)	Pre- and post- change of VO_{2amx} ($L \cdot min^{-1}$): CoQ10: -0.2 ± 0.27 vs PLA: -0.1 ± 0.25 Pre- and post- change of maximal workload (W): CoQ10: 10 ± 71.57 vs PLA: 19 ± 72.77 Pre- and post- change of workload at lactate threshold (W): CoQ10: 6.8 ± 19.16 vs PLA: 0.5 ± 31.21 Pre- and post- change of cycling sprint mean power output (W): CoQ10: 40 ± 108.93 vs PLA: 10 ± 99.91 Change of pre-and post- 1st RPE of 15 * 3 cycling (6-20): CoQ10: -0.90 ± 1.14 vs PLA: -1.6 ± 1.19 Change of pre-and post- 2nd RPE of 15 * 3 cycling (6-20): CoQ10: -2.1 ± 1.32 vs PLA: -1.7 ± 1.26 Change of pre-and post- RPE of 60-minutes treadmill running (6-20): CoQ10: -2.5 ± 2.35 vs PLA: -2.6 ± 3.00	Performance: No ($p > 0.05$); RPE: No ($p > 0.05$)
Porter et al. (1995) RSB Parallel	Graded exercise test on cycling ergometer	15 males and divided into 2 groups (CoQ10 group 8, PLA group 7); 44.7 ± 2.0, NS; Healthy	CoQ10: 150 mg/day CoQ10; PLA:1 g glucose; Tablet; 60 days (30th and 60th day test)	Pre- and post- 1 month, 2 months VO_{2amx} ($L \cdot min^{-1}$): CoQ10: 2.97 ± 0.51 and 2.97 ± 0.45, 3.05 ± 0.42 vs PLA: 2.97 ± 0.50 and 2.89 ± 0.45, 2.98 ± 0.42	Performance: No ($p > 0.05$); Blood CoQ10:

				Pre- and post- 1 month, 2 months LT ($L \cdot min^{-1}$): CoQ10: 2.04 ± 0.34 and 2.17 ± 0.34, 2.02 ± 0.34 vs PLA: 2.13 ± 0.40 and 2.17 ± 0.37, 2.20 ± 0.26 Pre- and post- 1 month, 2 months maximal workload (W): CoQ10: 243 ± 42.43 and 243 ± 42.43, 246 ± 50.91 vs PLA: 249 ± 37.04 and 243 ± 34.39, 246 ± 37.04 Pre- and post- 1 month, 2 months blood CoQ10 ($\mu g \cdot mL^{-1}$): CoQ10: 0.72 ± 0.34, 0.87 ± 0.57 and 1.08 ± 0.79 vs PLA: 0.91 ± 0.29, 0.71 ± 0.24 and 0.70 ± 0.29	Yes ↑ ($p < 0.05$)
Thar et al. (2025) RNS Crossover	Resistance exercise; 3MOAT	42 males and divided into 2 groups (Normal group 21, overweight group 21); Normal group: 26.3 ± 2.7, 58.8 ± 7.7, Overweight group: 26.6 ± 2.2, 73.5 ± 5.5; Moderate physical activity level	CoQ10: 300 mg OL; PLA: Empty capsule; Capsule; Acute ingestion before 2-hour test, and with 2 weeks wash out	Resistance exercise volume (kg): Normal: OL: 1658.01 ± 671.00 vs PLA: 1560.61 ± 616.88; Overweight: OL: 1777.06 ± 573.6 vs PLA: 1679.65 ± 573.59 3MOAT peak power (w): Normal: OL: 231.58 ± 126.32 vs PLA: 236.84 ± 126.32; Overweight: OL: 273.68 ± 157.90 vs PLA: 268.42 ± 147.37 3MOAT critical power (w): Normal: OL: 106.78 ± 57.63 vs PLA: 94.92 ± 52.54; Overweight: OL: 133.90 ± 54.24 vs PLA: 127.12 ± 59.32	Performance: Yes, only with OL vs PLA for both normal group and overweight group on resistance exercise ↑, and OL vs PLA for normal group on critical power ↑ ($p < 0.05$)
Weston et al. (1997)	Graded exercise test on cycling	20 males and divided into 2 groups (CoQ10 group 10, PLA group 10,	CoQ10: 1 mg/kg/day CoQ10; PLA: Lactose and acriflavine	Pre- and post-VO_{2peak} ($L \cdot min^{-1}$) CoQ10: 4.57 ± 0.31 and 4.68 ± 0.25 vs PLA:	Performance: No ($p > 0.05$);

RDB Parallel	ergometer	but CoQ10 group withdrew 2); CoQ10 group: 24.8 ± 8.8, 70.8 ± 4.8, PLA group: 25.6 ± 8.2, 70.7 ± 9.2; Healthy and well-trained road cyclists (At least trained 2 years)	with similar appearance; Capsule; 28 days (28th day test)	4.60 ± 0.35 and 4.62 ± 0.41 Pre- and post- blood CoQ10 (μg·mL^{-1}): CoQ10: 0.91 ± 0.37 and 1.97 ± 0.76 vs PLA: 0.72 ± 0.13 and 0.71 ± 0.25	Blood CoQ10: Yes ↑ ($p < 0.05$)
Yanprechaset et al. (2015) RNS Parallel	Graded exercise test on treadmill	48 overweight females and divided into 4 groups (12/group); CoQ10: 50.1 ± 5.82, 62.6 ± 6.12, Con: 48.7 ± 3.57, 60.7 ± 5.57, CoQ10 + Exercise: 50.9 ± 4.1, 64 ± 4.89, Exercise: 52.3 ± 6.23, 60.8 ± 5.79; Healthy but overweight	CoQ10: 200 mg/day CoQ10; PLA: NS; Soft gel; 84 days (84th test)	Pre- and post- VO_{2amx} (mL·min^{-1}·kg^{-1}): CoQ10: 30.4 ± 1.8 and 33.6 ± 2.6 vs Con: 33.4 ± 3.2 and 32.3 ± 3.4 vs CoQ10 + Exercise: 31.6 ± 1.5 and 34.6 ± 1.7 vs Exercise: 32.5 ± 2 and 34.5 ± 2.4 Pre- and post- blood CoQ10 (μmol·L^{-1}): CoQ10: 0.23 ± 0.09 and 0.90 ± 0.44 vs CoQ10 + Exercise: 0.30 ± 0.12 and 0.94 ± 0.18 vs Con: 0.24 ± 0.07 and 0.43 ± 0.12 vs Exercise: 0.25 ± 0.1 and 0.53 ± 0.19	Performance: Yes, only with post- VO_{2amx} of CoQ10, exercise and combined group vs Con group ↑ ($p < 0.05$); Blood CoQ10: Yes ↑ ($p < 0.05$)
Ylikoski et al. (1997) RDB Crossover	Graded exercise test on a specific ski pole treadmill	25 subjects, gender NS and withdrew 7; NS, NS; National cross-country skier	CoQ10: 90 mg/day CoQ10; PLA:1 g glucose; Capsule; 42 days (42nd day test), and with 6 weeks wash out	Pre- and post- change of VO_{2amx} between CoQ10 group and PLA group (mL·min^{-1}·kg^{-1}): 1.6 ± 2.81 Pre- and post- change of ANT between CoQ10 group and PLA group (mL·min^{-1}·kg^{-1}): 2.4 ± 2.27 Pre- and post- change of AET between CoQ10 group and PLA group (mL·min^{-1}·kg^{-1}): 2.6 ± 2.27 Pre- and post- blood CoQ10 (μg·mL^{-1}):	Performance: Yes, all indicators ↑ ($p < 0.05$); Blood CoQ10: Yes, only with CoQ10 vs baseline ↑ ($p < 0.05$)

				Baseline: 0.81 ± 0.24 and CoQ10: 2.78 ± 2.02 vs PLA: 0.86 ± 0.24	
Zhou et al. (2005) RSB Crossover	Exercise economy test at 50 W, 100 W and 150 W; Graded exercise test on cycling ergometer	7 males and withdrew 1; 29.7 ± 7.2, 77.5 ± 13.0; Healthy and recreational cyclists	CoQ10: 150 mg/day CoQ10; PLA: Starch with similar appearance; Capsule; 14 days (14^{th} day exercise and blood test, and one more blood and muscle test on 28^{th} day)	Pre- and post- VO_2 at 50 W ($mL \cdot min^{-1}$): CoQ10: 1116.71 ± 95.1 and vs 1090.78 ± 69.16 vs PLA: 1116.71 ± 95.1and 1121.04 ± 108.07 Pre- and post- VO_2 at 100 W ($mL \cdot min^{-1}$): CoQ10: 1531.70 ± 38.91 and 1479.83 ± 30.26 vs PLA: 1531.70 ± 38.91 and 1536.02 ± 21.62 Pre- and post- VO_2 at 150 W ($mL \cdot min^{-1}$): CoQ10: 2007.21 ± 211.81 and 1994.24 ± 181.55 vs PLA: 2007.21 ± 211.81 and 2002.88 ± 207.49 Pre- and post- VO_{2amx} ($L \cdot min^{-1}$): CoQ10: 2.65 ± 0.54 and 2.75 ± 0.51 vs PLA: 2.65 ± 0.54 and 2.80 ± 0.55 Pre- and post- blood CoQ10 ($\mu g \cdot mL^{-1}$): CoQ10: 0.77 ± 0.34 and 2.52 ± 1.17 vs PLA: 0.83 ± 0.43 and 0.77 ± 0.34 Pre- and post- muscle CoQ10 of CoQ10 group ($\mu g \cdot g^{-1}$ dry weight): 207 ± 39 and 220 ± 46	Performance: No ($p > 0.05$); RPE (No data): No ($p > 0.05$); Blood CoQ10: Yes, only at stage 2 ↑ ($p < 0.05$) Muscle CoQ10: No ($p > 0.05$)

Notes: ***AET***, aerobic threshold; ***ANT***, anaerobic threshold; ***CoQ10***, Coenzyme Q10; ***Con***: Control group; ***g***, grams; ***kg***, kilogram; ***LT***, lactate threshold; ***L***,liter; ***MCT*****:** medium-chain triacylglycerol**;** ***min***, minutes; ***NS***, Not specified; ***ONE***, ubiquinone; ***OL***, ubiquinol; ***PLA***, placebo; ***RDB***, randomised double-blind trial; ***RSB***, randomised single-blind trial; ***s,*** seconds; **SFS**, subjective fatigue sensation; ***RAST***, Running-based anaerobic sprint test; ***TTE***, time to exhaustion; ***h***, hour; ***μg·mL*$^{-1}$,** microgram/ milliliter; ***W***, watt; ***3MAOT***, three-minute all-out test; **↑,** represents significantly improved**; ↓,** represents significantly decreased.