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SYSTEMATIC REVIEW

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Exercise improves sperm quality and pregnancy outcome by attenuating oxidative stress: a systematic review and meta-analysis

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Abstract

Background: Male fertility contributes to the life quality of couples. Although exercise has been reported to mitigate the declining trend in sperm quality, the available reports are conflicting, and the precise influence on male fertility indices warrants further investigation. Hence, this study evaluated the effect of exercise on sperm quality, pregnancy outcomes, sex hormones, and markers of oxidative stress and inflammation in men. **Methods**: This is a systematic review and meta-analysis that evaluated exercise interventions, including aerobic, resistance, and combined modalities, on conventional and advanced semen parameters, and pregnancy and live birth rates. **Results**: Exercise considerably improved ejaculate volume, sperm count, motility, morphology, and sperm DNA integrity. Also, exercise enhanced seminal total antioxidant capacity (TAC) and decreased seminal reactive oxygen species, malondialdehyde, and pro-inflammatory cytokines levels. More so, pregnancy and live birth rates increased in the exercise group when compared with the control. Nonetheless, exercise did not show any considerable effect on testosterone, luteinizing hormone (LH), and follicle-stimulating hormone (FSH) levels. The observed effects varied by exercise type and duration, with moderate aerobic exercise showing beneficial effects. Moderate aerobic exercise enhances sperm quality and improves pregnancy outcomes by attenuating oxidative stress and inflammation. **Conclusions**: These findings show that moderate aerobic exercise is a promising non-pharmacological strategy in male infertility management. However, more clinical trials are required to optimize exercise protocols for reproductive benefits. **The PROSPERO Registration**: https://www.crd.york.ac.uk/PROSPERO/view/CRD42024622166, CRD42024622166.

Keywords

Exercise; Infertility; Live birth; Male infertility; Pregnancy; Testosterone

1. Introduction

Infertility is the failure of a couple to get pregnant spontaneously despite a year of regular, unprotected intercourse [1]. About one in six people of reproductive age have infertility issues globally [2]. Although it is wrongly perceived as a condition caused by female pathology, male factors account for about 50% of the cases, either alone or in combination with female factors [3, 4]. While the prevalence of male infertility varies across the globe, it constitutes a major public health burden across high-, middle-, and low-income countries alike [5]. Previous studies have reported a declining trend in sperm quality across the globe, suggesting an increase in the prevalence of male infertility [6, 7].

Causes of male infertility include varicocele [8], cryptorchidism [9], testicular torsion/detorsion [10, 11], infections of the reproductive tract [12], hormonal imbalances, problems with sperm production, obstructions of ejaculatory duct and vas deferens, genetic disorders, and exposure to environmental toxicants (like heavy metal, pesticides, and irradiation) [13–15]. Additionally, lifestyle choices have a significant effect on male fertility. Smoking [16], excessive alcohol intake [17], use of illicit drugs [18, 19], psychological stress [20], and sedentary lifestyle [21] may impair sperm quality, contributing to the global burden of infertility.

There is increasing evidence linking physical activity/exercise with sperm quality. Physical activity maintains a healthy body weight, which is vital as obesity

is linked to low testosterone levels and impaired sperm production [22, 23]. Also, exercise enhances natural cellular antioxidant levels, thus protecting sperm cells from damage [22]. However, there are conflicting reports. Some studies revealed that moderate-to-vigorous activity results in increased total sperm count, concentration [24, 25], semen volume, and sperm normal morphology [26], while some studies show that physical activity was not associated with semen quality [27, 28]. On the contrary, long-distance runners were observed to have reduced semen volume and sperm motility and morphology during training [29].

Despite the conflicting reports, there are scanty meta-analyses that have pooled the available data in the literature for a robust evaluation. Although the available meta-analyses on exercise and semen quality reveal the potential benefits of exercise on sperm quality [30, 31], these studies did not explore the likely impact on pregnancy outcomes and the associated mechanisms. Therefore, the present study investigated the impact of exercise on sperm quality and pregnancy outcomes. The impact on male sex hormones, oxidative stress, and pro-inflammatory cytokines was also explored.

2. Materials and methods

2.1 Study protocol and measured outcomes

This systematic review and meta-analysis was prospectively registered on PROSPERO (CRD42024622166) and synthesized data from prior publications examining the effects of physical activity or exercise on semen quality parameters, male sex hormones, pregnancy outcomes, and oxidative stress markers. We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [32]. The PRISMA check list is provided as a **Supplementary material 1**.

Primary outcomes included exercise-induced changes in semen volume, sperm count, concentration, total and progressive motility, normal morphology, DNA fragmentation, and pregnancy and live birth rate. Secondary outcomes included levels of total and free testosterone, follicle-stimulating hormone (FSH), luteinizing hormone (LH), sex hormone-binding globulin (SHBG), cortisol, reactive oxygen species (ROS), malondialdehyde (MDA), total antioxidant capacity (TAC), interleukin (IL)-8, IL-6, IL-1β, and tumor necrosis factor (TNF)-α.

2.2 Eligibility criteria

This study addresses the research question: "Does exercise improve semen quality and pregnancy outcomes?". It was structured according to the Population, Intervention, Comparator, Outcome, and Study designs (PICO) framework.

2.2.1 Inclusion criteria

Studies were included if they met all of the following PICO-based criteria:

i. Population: Males of reproductive age.

ii. Intervention: Effects of exercise on semen quality and pregnancy outcomes.

iii. Comparator: Age-matched controls with comparable sociodemographic characteristics, but no exercise involvement.

iv. Outcomes: Reported sperm parameters (semen volume, sperm count, concentration, total motility, progressive motility, normal morphology, DNA fragmentation), pregnancy outcomes (pregnancy rate, live birth rate), male sex hormones (free and total testosterone, FSH, LH, SHBG), cortisol, oxidative stress markers (ROS, MDA, TAC), and inflammatory cytokines (IL-8, IL-6, IL-1β, TNF-α) as means with standard deviations (SDs) or data allowing SD derivation.

v. Study design: Controlled cross-sectional or longitudinal studies addressing the effect of exercise on semen quality, reproductive hormones, markers of oxidative stress and inflammation, and pregnancy outcomes.

2.2.2 Exclusion criteria

Studies were excluded for any of the following reasons:

i. Population: No participant age reported.

ii. Intervention: Combined exercise with unspecified lifestyle changes, drugs, or *in vitro*/animal studies.

iii. Comparator: Lacked non-exercising control group.

iv. Outcomes: No quantifiable data (means/SDs or derivable) for variables of interest; self-reported reproductive outcomes.

v. Study design: Case studies, reviews, commentaries, letters, or editorials.

Additional exclusions included theses, conference abstracts, retracted papers, and preprints. No language restrictions were applied.

2.3 Search strategies

All authors performed a comprehensive search of Cochrane, Embase, PubMed, Scopus, and Web of Science databases up to 30 March 2025. Search terms combined Medical Subject Headings (MeSH) and Boolean operators: ("sperm" OR "semen" OR "sperm count" OR "sperm concentration" OR "sperm motility" OR "semen volume" OR "sperm DNA" OR "DNA fragmentation index (DFI)") AND ("pregnancy" OR "live birth") AND ("testosterone" OR "luteinizing hormone" OR "LH" OR "follicle-stimulating hormone" OR "FSH" OR "sex hormone-binding globulin" OR "SHBG" OR "cortisol") AND ("oxidative stress" OR "ROS" OR "reactive oxygen species" OR "MDA" OR "malondialdehyde" OR "TAC" OR "total antioxidant capacity") AND ("IL-1β" OR "IL-6" OR "IL-8" OR "TNF-α" OR "cytokine").

Eligible studies were compiled, and citation chasing was used to identify additional relevant papers. Five investigators (CAA, PJA, VJA, AEA, TMA) screened abstracts and full texts for eligibility; disagreements were arbitrated by REA.

2.4 Quality assessment and data extraction

Quality of evidence (QoE), risk of bias (RoB), and certainty of evidence (CoE) were evaluated using the Cochrane RoB tool and Grading of Recommendations Assessment, Development and Evaluation (GRADE) Working Group guidelines, as detailed in prior work [14, 32]. Assessments were performed by five investigators (BMA, AAO, OAA, CAA, DTM), with REA

resolving disputes. Relevant data were extracted by BMA, AAO, OAA, CAA, and DTM.

2.5 Statistical analysis

CAA and REA conducted meta-analyses in Review Manager (RevMan) version 5.4.1 (Cochrane Informatics and Technology (IT) Services, London, UK). Random-effects models were applied for significant heterogeneity ($p < 0.1$ or $I^2 > 50\%$); fixed-effects models for low heterogeneity ($p \geq 0.1$ or $I^2 \leq 50\%$). Publication bias was inspected via funnel plots. Subgroup analyses targeted longitudinal studies; sensitivity analyses excluded the highest-weighted study, those with high RoB in ≥1 domain, or low CoE. Statistical significance was set at $p < 0.05$. Results are presented as standardized mean difference (SMD) or weighted mean difference (WMD) with 95% confidence interval (CI) and p values.

3. Results

3.1 Study identification and inclusion

A total of 37,472 articles were collected. After screening out the duplicates and those that were not eligible, only 16 articles [33–48] were included (Fig. 1).

3.2 Study characteristics

All included studies were controlled studies; six were cross-sectional, seven were non-randomized controlled longitudinal studies, and three were randomized controlled trials (RCTs). Four of the studies were from the USA, two from Australia, one each from Egypt and Colombia, two from Italy, and three each from Iran and Spain. Other details of the included studies are in **Supplementary Table 1** (Ref. [33–48]).

3.3 Assessment of the quality of the studies

All the included studies had good QoE (≥5/10) (Table 1, Ref, [33–48]). Three of the included studies [33, 34, 36] had unclear RoB on random sequence generation, allocation concealment, and incomplete outcome data domains, one had unclear risk on allocation concealment [35] and random sequence generation [39] only, and one on allocation concealment and incomplete outcome data [37]. Three of the studies [42, 44, 48] had high RoB on at least one domain, while others [40, 41, 43, 45–47] had low RoB on all domains (Fig. 2A, Ref. [33–48]). Overall, there was <75% low RoB on the random sequence generation and allocation concealment domain each, 75% on the incomplete outcome data domain, <100% but >75% on the selective reporting domain, and 100% on the blinding of participants/personnel, blinding of outcome assessment, and other bias domains each (Fig. 2B).

In addition, there was high CoE in all eligible studies except in one [37] that had low CoE (Table 2, Ref. [33–48]).

3.4 Quantitative analysis

3.4.1 Semen volume

Exercise significantly increased semen volume when compared with the control (−0.48 [−0.82, −0.15] 0.004), but there was inter-study heterogeneity (I^2 = 91%; χ^2 p < 0.00001) (Fig. 3, Ref. [33–48]). Publication bias was observed (**Supplementary Fig. 1**).

The subgroup analysis of longitudinal studies also showed a significant increase in semen volume in subjects who exercised when compared with the control counterpart (−0.13 [0.23, −0.04] 0.007), and there was no inter-study heterogeneity (I^2 = 29%; χ^2 p = 0.19). Subgroup analysis of the moderate aerobic exercise studies revealed that exercise significantly improved semen volume (−0.59 [−1.06, −0.12] 0.01), but there was also an inter-study heterogeneity (I^2 = 93%; χ^2 p < 0.00001). In addition, the sensitivity analysis revealed that exercise involvement significantly increased semen volume in comparison with the control (−0.66 [−1.22, −0.11] 0.02), but there was inter-study heterogeneity (I^2 = 91%; χ^2 p < 0.00001) (Fig. 3).

3.4.2 Sperm count

Fourteen studies were examined for the influence of exercise on sperm count, consisting of 438 controls and 430 subjects in the exercise group. Sperm count significantly increased in the exercise group in comparison with the control group (−8.22 [−13.50, −2.94] 0.002). However, there was inter-study heterogeneity (I^2 = 73%; χ^2 p < 0.00001) (Fig. 4, Ref. [33–48]). There was a significant publication bias (**Supplementary Fig. 2**).

Subgroup analysis of longitudinal studies of sperm count among subjects who exercised showed an insignificant increase in comparison with the control group (−4.69 [−9.41, 0.02] 0.05) and there was inter-study heterogeneity (I^2 = 79%; χ^2 p < 0.0001). Subgroup analysis of the moderate aerobic exercise studies showed that exercise significantly improved sperm count (−6.80 [−12.54, −1.07] 0.02), but there was also an inter-study heterogeneity (I^2 = 80%; χ^2 p < 0.00001). Also, the sensitivity analysis revealed that exercise significantly increased sperm concentration (−8.30 [−15.80, −0.81] 0.03), but there was inter-study heterogeneity (I^2 = 56%; χ^2 p = 0.01) (Fig. 4).

3.4.3 Sperm concentration

The effect of exercise on sperm concentration was assessed in fifteen studies with 593 and 595 subjects in the control and exercise group, respectively. There were no significant changes in sperm concentration (−0.24 [−5.77, 5.30] 0.93), but there was inter-study heterogeneity (I^2 = 78%; χ^2 p < 0.00001) (Fig. 5, Ref. [33–48]). Publication bias was observed (**Supplementary Fig. 3**).

However, subgroup analysis of longitudinal studies revealed that there was a significant increase in sperm concentration in exercising subjects compared with the control counterpart (−2.80 [−4.77, −0.82] 0.005) and there was no inter-study heterogeneity (I^2 = 9%; χ^2 p = 0.36), while subgroup analysis of the moderate aerobic exercise studies revealed that exercise did not significantly alter sperm concentration (2.37 [−7.25, 11.99] 0.63), but there was also inter-study heterogeneity (I^2 = 77%; χ^2 p < 0.00001). The sensitivity analysis revealed that sperm concentration was not significantly altered (8.42 [−4.06, 20.90] 0.19), but there was inter-study heterogeneity (I^2 = 81%; χ^2 p < 0.00001) (Fig. 5).

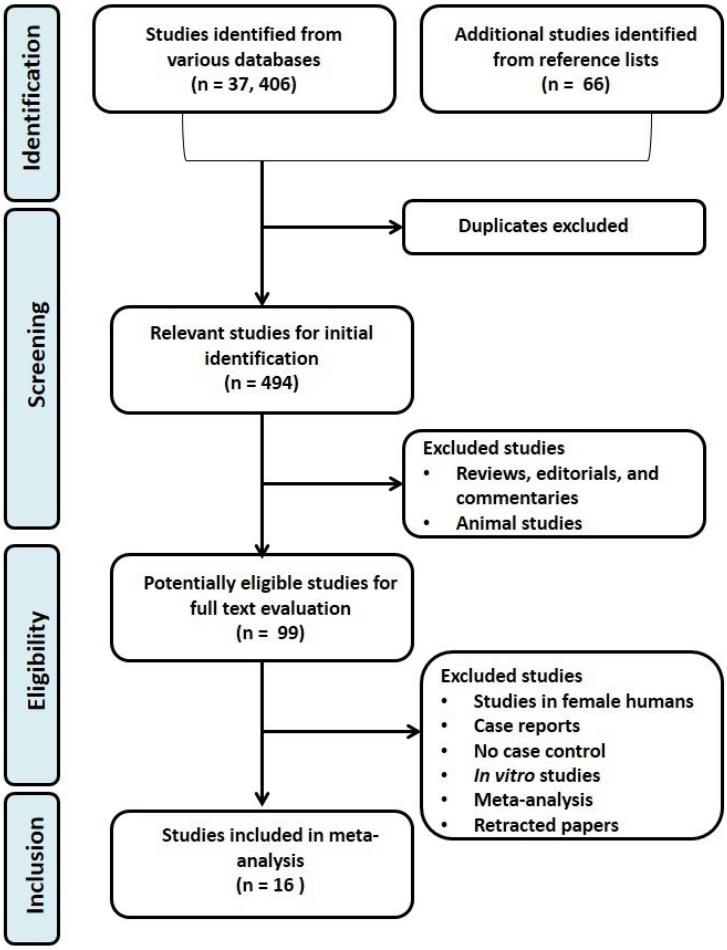


FIGURE 1. PRISMA flowchart for the strategic identification, screening, and inclusion of eligible studies.

TABLE 1. Assessment of the quality of evidence of the eligible studies.

Study	Study design	Study size	Method of measuring exposure	Method of measuring outcome	Analysis with adjustment	Total
Arce *et al.* [33], 1993	2	0	2	2	0	5/10
Bagatell and Bremner [34], 1990	2	0	2	2	0	6/10
De Souza *et al.* [36], 1994	2	0	2	2	0	6/10
Denham *et al.* [35], 2015	2	0	2	2	2	8/10
Fahrner and Hackney [37], 1997	2	0	2	2	0	6/10
Ismail *et al.* [38], 2023	2	0	2	2	0	6/10
Lalinde *et al.* [39], 2017	2	0	2	2	0	6/10
Lovell *et al.* [40], 2012	2	0	2	2	0	6/10
Luigi *et al.* [41], 2001	2	1	2	2	0	7/10
Maleki and Tartibian [42]	2	2	2	2	0	8/10
Maleki *et al.* [44], 2017	2	2	2	2	0	8/10
Maleki *et al.* [43], 2013	2	2	2	2	0	8/10
Montano *et al.* [45], 2022	2	2	2	2	0	8/10
Rosety *et al.* [46], 2017	2	1	2	2	0	7/10
Rosety-Rodriguez *et al.* [47], 2014	2	1	2	2	0	7/10
Vaamonde *et al.* [48], 2012	2	0	2	2	0	6/10

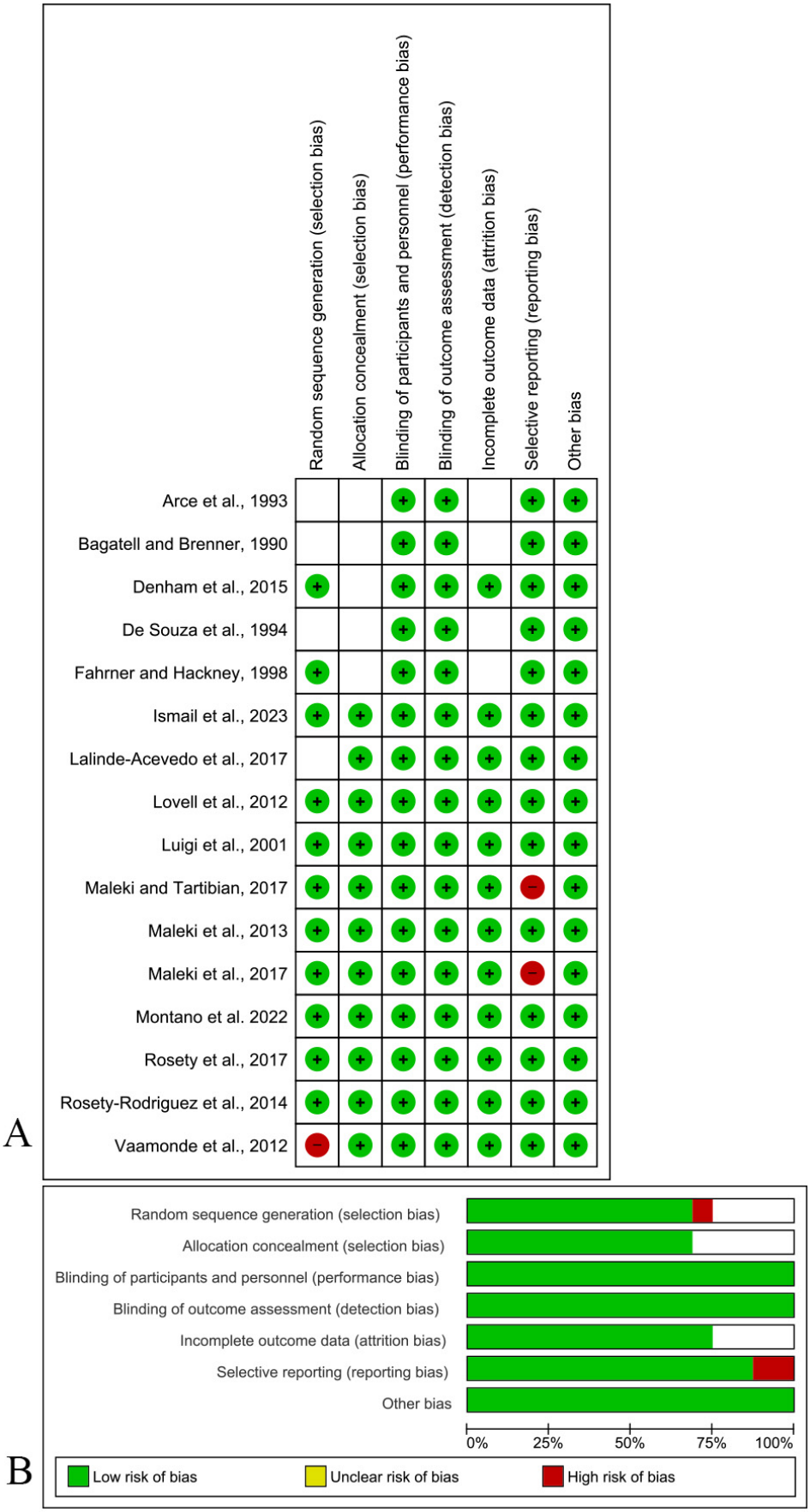


FIGURE 2. The risk of bias (RoB) of the included studies [33–48]; RoB summary showing each risk of bias item for each included study (A) and each risk of bias item presented as percentages across all included studies (B). +: Low RoB; −: High RoB.

TABLE 2. Assessment of certainty of evidence of the eligible studies.

Study	Initial Rating	Downgrading?	Upgrading?	Confidence in body of evidence
Arce *et al.* [33], 1993	High	1 (imprecision)	1 (consistency)	High
Bagatell and Bremner [34], 1990	High	1 (imprecision)	1 (consistency)	High
De Souza *et al.* [36], 1994	High	1 (imprecision)	2 (consistency, dose-response)	High
Denham *et al.* [35], 2015	High	-	-	High
Fahrner and Hackney [37], 1997	High	2 (unexplained inconsistency, indirectness)	-	Low
Ismail *et al.* [38], 2023	High	-	-	High
Lalinde *et al.* [39], 2017	High	-	1 (rare outcome)	High
Lovell *et al.* [40], 2012	High	-	-	High
Luigi *et al.* [41], 2001	High	-	-	High
Maleki and Tartibian [42]	High	1 (risk of bias)	1 (rare outcome)	High
Maleki *et al.* [44], 2017	High	1 (risk of bias)	1 (rare outcome)	High
Maleki *et al.* [43], 2013	High	-	-	High
Montano *et al.* [45], 2022	High	-	-	High
Rosety *et al.* [46], 2017	High	-	-	High
Rosety-Rodriguez *et al.* [47], 2014	High	-	-	High
Vaamonde *et al.* [48], 2012	High	-	-	High

3.4.4 Sperm total motility

Sperm total motility was assessed in six studies to evaluate the effect of exercise in 304 control subjects and 316 subjects who engaged in exercise. Exercise showed no significant impact on sperm total motility (−5.64 [−15.53, 4.24] 0.26), but inter-study heterogeneity was significant (I^2 = 96%; χ^2 p < 0.00001) (Fig. 6, Ref. [33–48]). Publication bias was also noted (**Supplementary Fig. 4**).

The subgroup analysis of longitudinal studies showed an insignificant increase in sperm total motility among the exercise-group subjects when compared with the control group (−6.07 [−12.22, 0.09] 0.05), and there was inter-study heterogeneity (I^2 = 84%; χ^2 p = 0.0002). Subgroup analysis of studies on the moderate aerobic exercise demonstrated that exercise considerably increased sperm total motility (−10.20 [−18.47, −1.93] 0.02), but there was also inter-study heterogeneity (I^2 = 93%; χ^2 p < 0.00001). The sensitivity analysis showed that exercise had no significant impact on sperm total motility (−5.38 [−19.19, 8.43] 0.45), but there was inter-study heterogeneity (I^2 = 96%; χ^2 p < 0.00001) (Fig. 6).

3.4.5 Sperm progressive motility

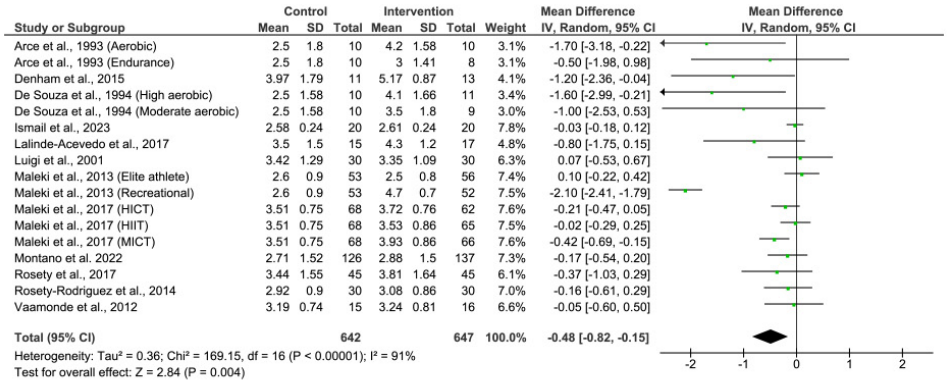
Twelve studies were evaluated for the outcome of exercise on sperm progressive motility, comprising 480 control subjects and subjects who exercised. The global analysis showed no significant changes, reflecting that exercise does not influence sperm progressive motility (−0.32 [−5.24, 4.59] 0.90). Nonetheless, inter-study heterogeneity was significant (I^2 = 89%; χ^2 p < 0.00001) (Fig. 7, Ref. [33–48]). Publication bias was also significant (**Supplementary Fig. 5**).

On the contrary, the subgroup analysis of longitudinal studies demonstrated a substantial increase in sperm progressive motility in subjects who participated in exercise when compared with subjects in the control group (−4.85 [−9.46, −0.25] 0.04), and there was inter-study heterogeneity (I^2 = 87%; χ^2 p < 0.00001). Subgroup analysis of the moderate aerobic exercise studies revealed that exercise did not considerably alter sperm progressive motility (−2.22 [−7.89, 3.45] 0.44), but there was also an inter-study heterogeneity (I^2 = 90%; χ^2 p < 0.00001). Then again, the sensitivity analysis revealed that exercise involvement did not alter the sperm progressive motility (2.33 [−4.53, 9.19] 0.51), but there was an inter-study heterogeneity (I^2 = 88%; χ^2 p < 0.00001) (Fig. 7).

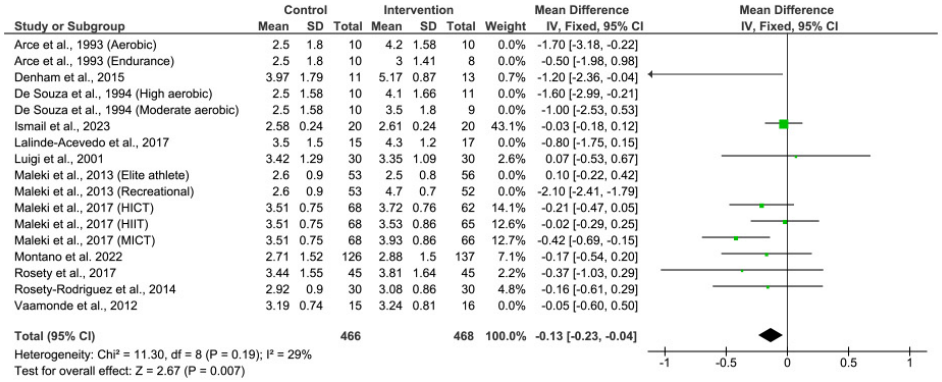
3.4.6 Sperm normal morphology

Since sperm morphology was evaluated by the eligible studies using different criteria (as stated in **Supplementary Table 1**), SMD was used. The effect of exercise on sperm normal morphology was assessed in the intervention group, which revealed that there was a marginal increase in comparison with the control subjects (−0.24 [−0.50, 0.02] 0.07). Likewise, there was inter-study heterogeneity (I^2 = 79%; χ^2 p < 0.00001) (Fig. 8, Ref. [33–48]). Also, a significant publication bias is shown (**Supplementary Fig. 6**).

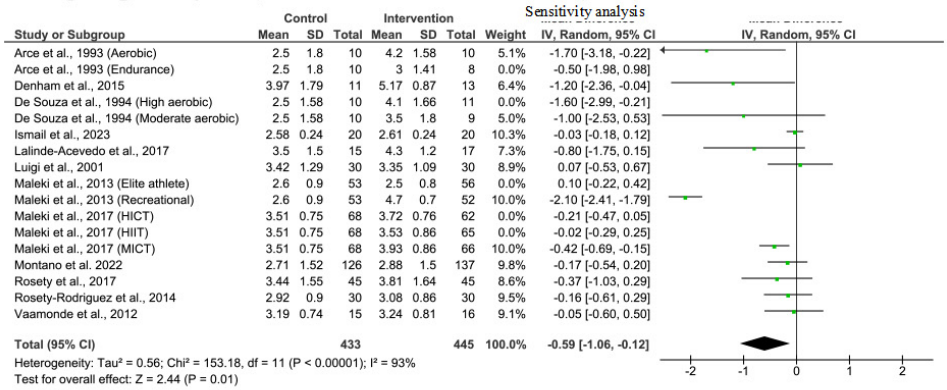
Furthermore, the subgroup analysis of longitudinal studies proved that sperm normal morphology was significantly increased in subjects who exercised when compared with the control counterpart (−0.35 [−0.48, −0.22] <0.00001), inter-study heterogeneity was significant (I^2 = 44%; $\chi^2 p$ = 0.08). Subgroup analysis of the moderate aerobic exercise studies showed that exercise significantly improved sperm normal morphology (−0.41 [−0.64, −0.17] 0.0006), but there was also an inter-study heterogeneity (I^2 = 58%; χ^2 p = 0.006). In



Subgroup analysis (Longitudinal studies)



Subgroup analysis (Moderate aerobic exercise)



Sensitivity analysis

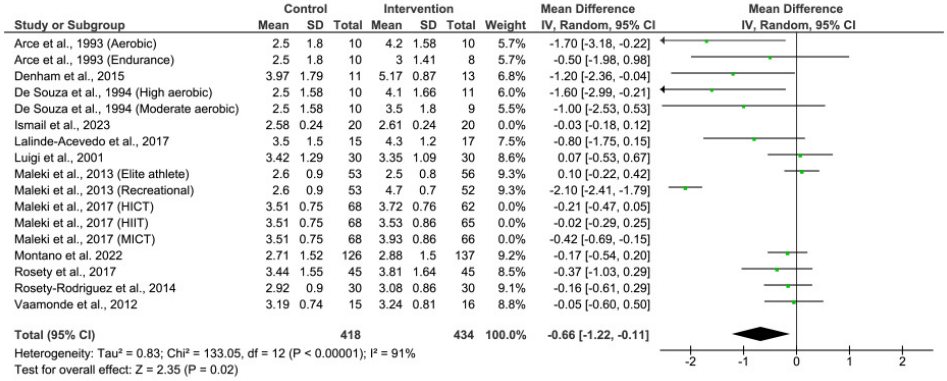
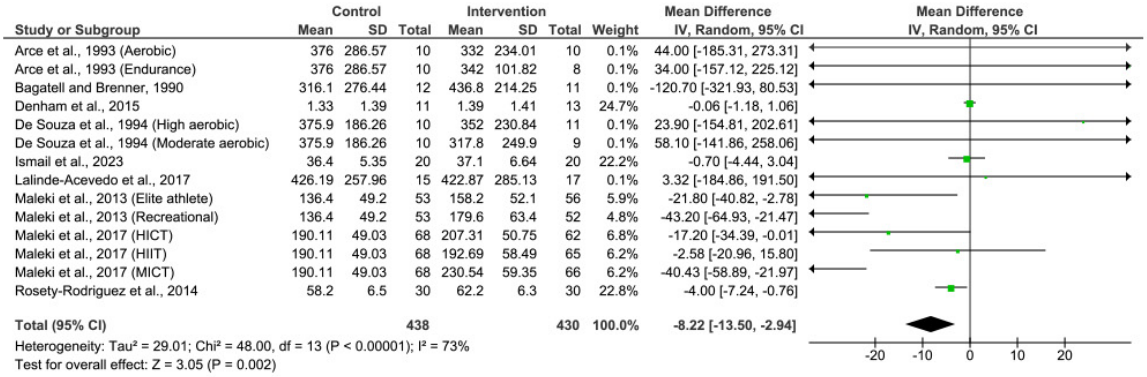
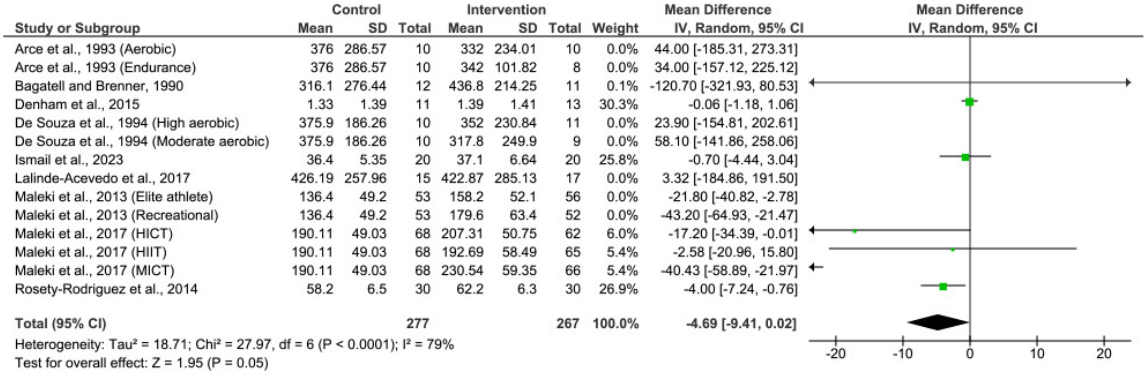


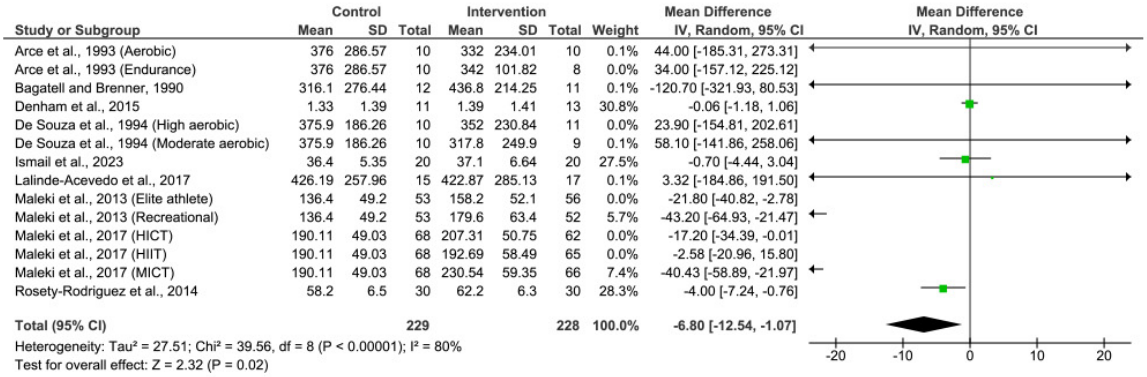
FIGURE 3. Effect of exercise on semen volume [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval; HICT: High-intensity continuous exercise; HIIT: High-intensity interval exercise; MICT: Moderate-intensity continuous exercise.



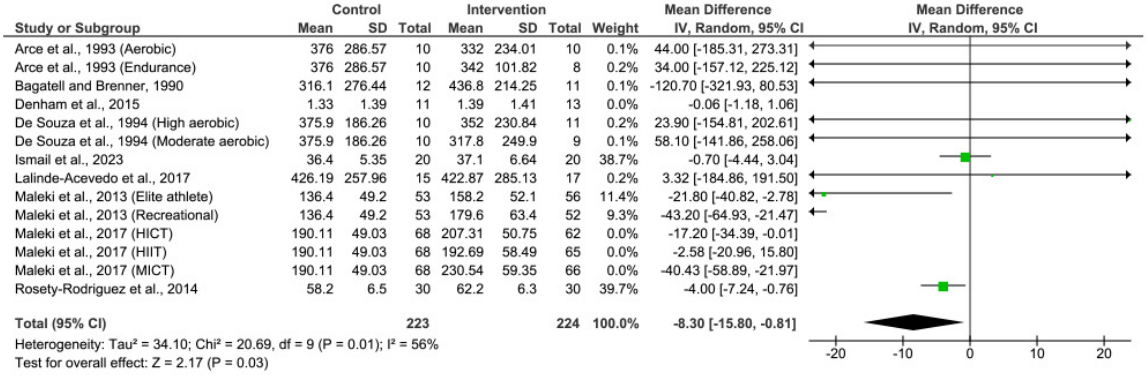
Subgroup analysis (Longitudinal studies)



Subgroup analysis (Moderate aerobic exercise)



Sensitivity analysis



F I G U R E 4. Effect of exercise on sperm count [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval; HICT: High-intensity continuous exercise; HIIT: High-intensity interval exercise; MICT: Moderate-intensity continuous exercise.

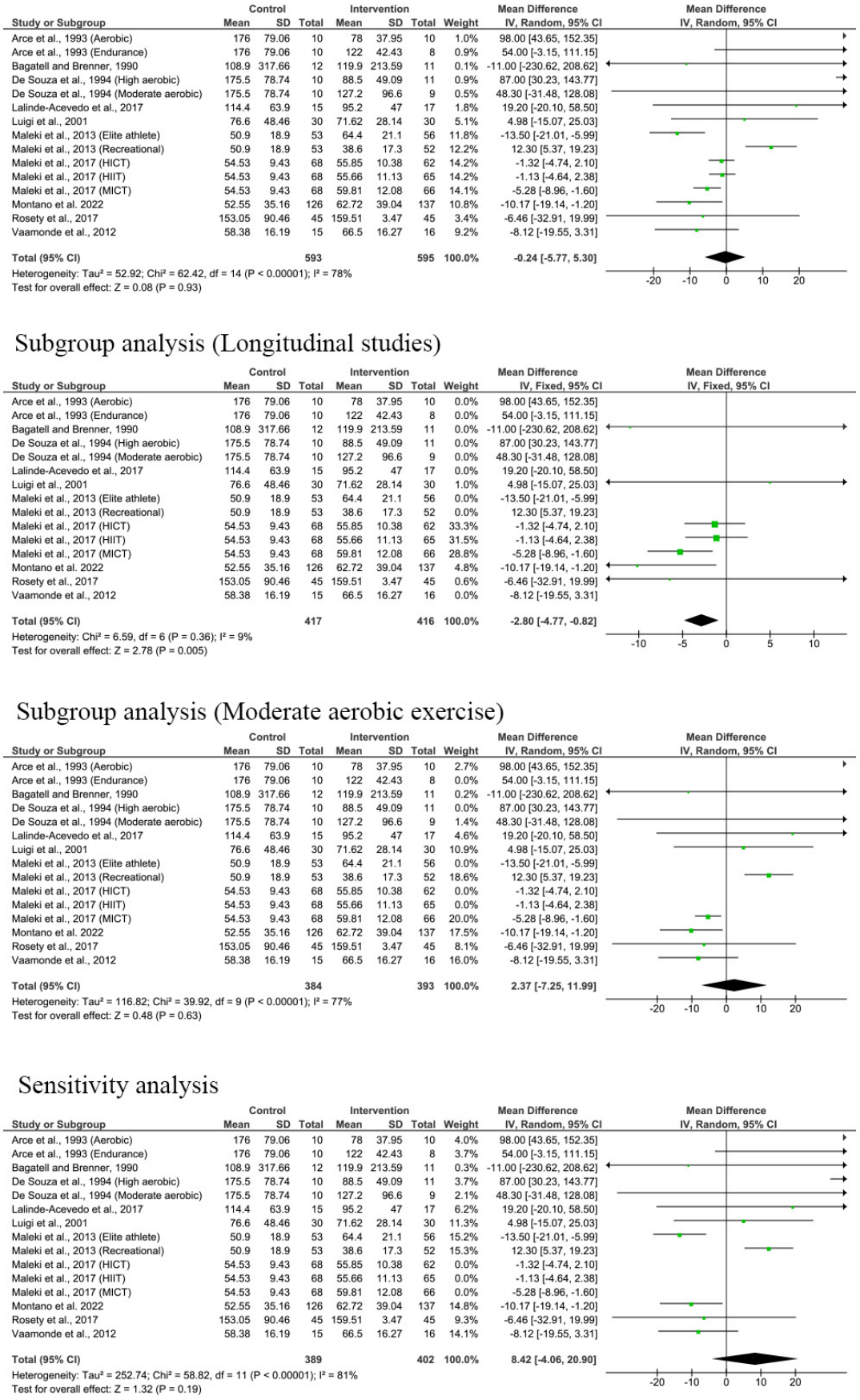
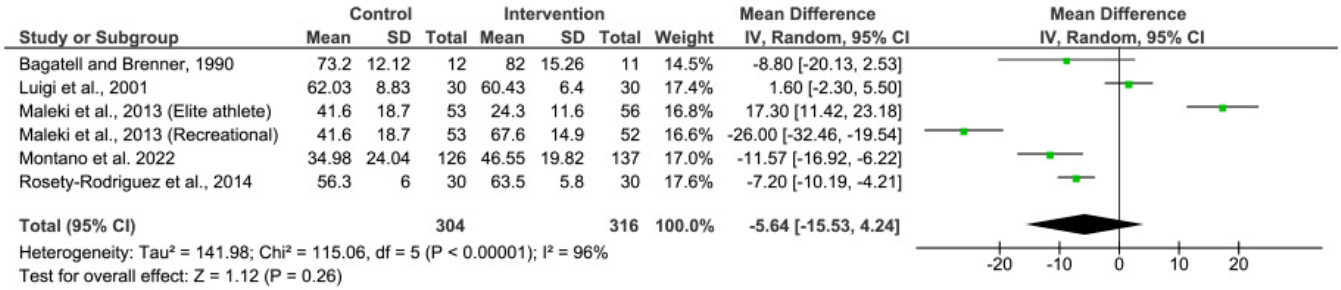
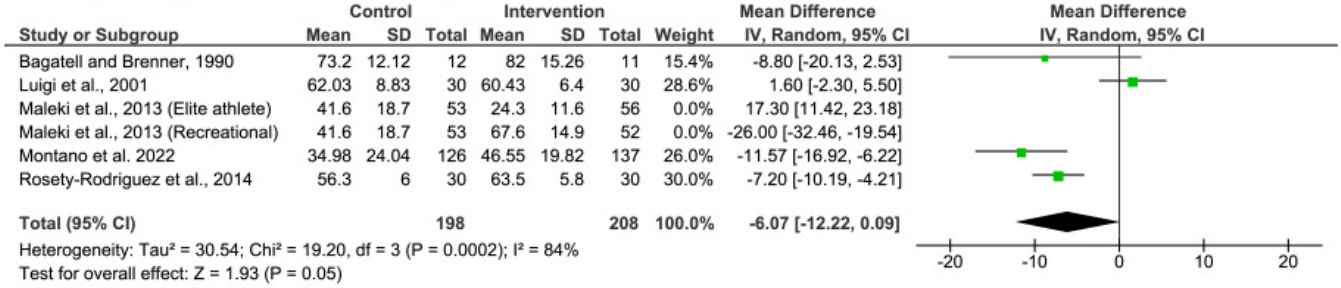


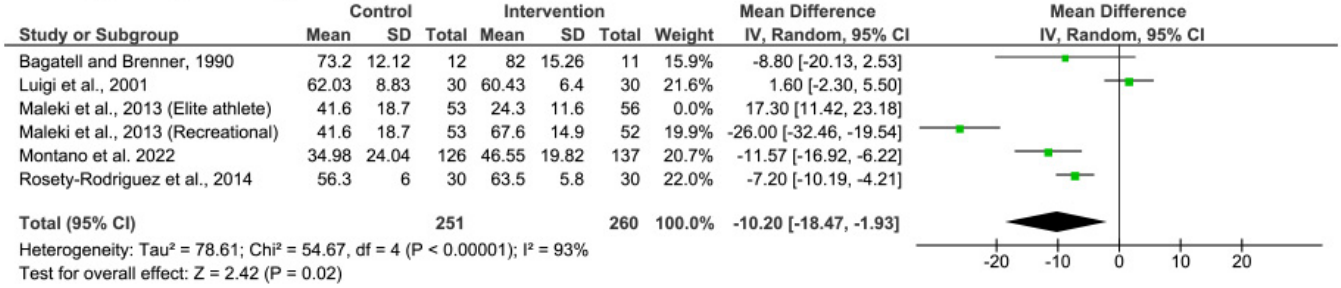
FIGURE 5. Effect of exercise on sperm concentration [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval; HICT: High-intensity continuous exercise; HIIT: High-intensity interval exercise; MICT: Moderate-intensity continuous exercise.



Subgroup analysis (Longitudinal studies)



Subgroup analysis (Moderate aerobic exercise)



Sensitivity analysis

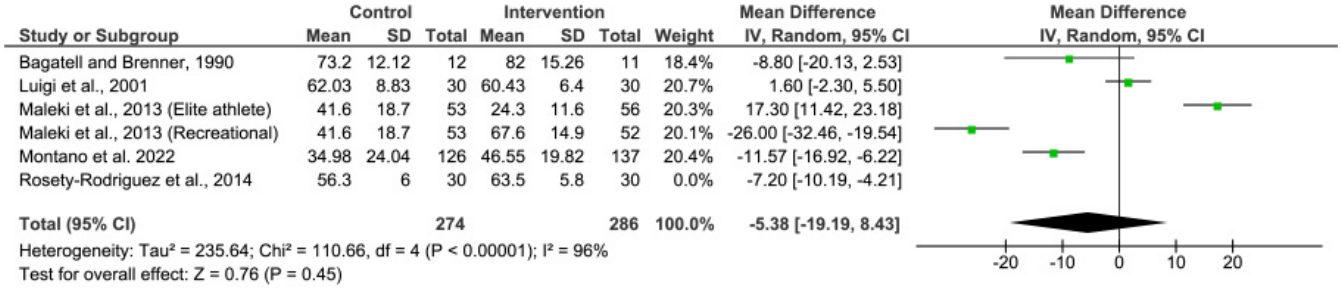
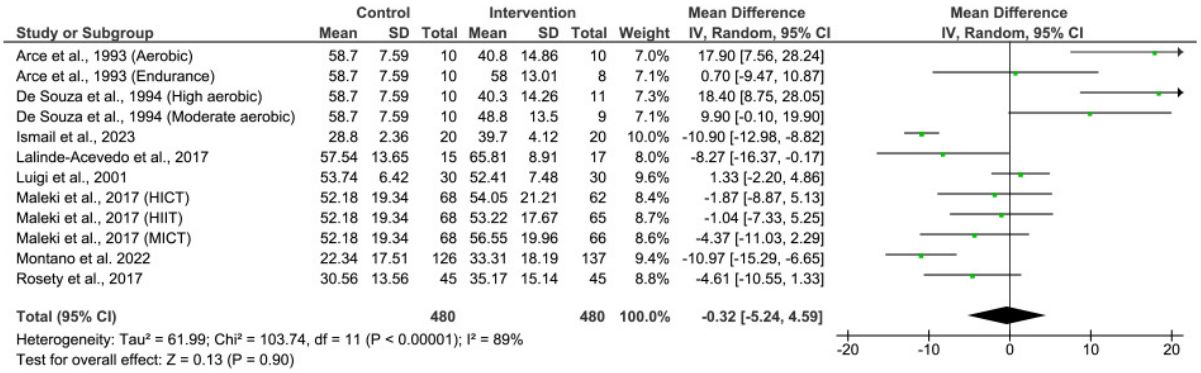
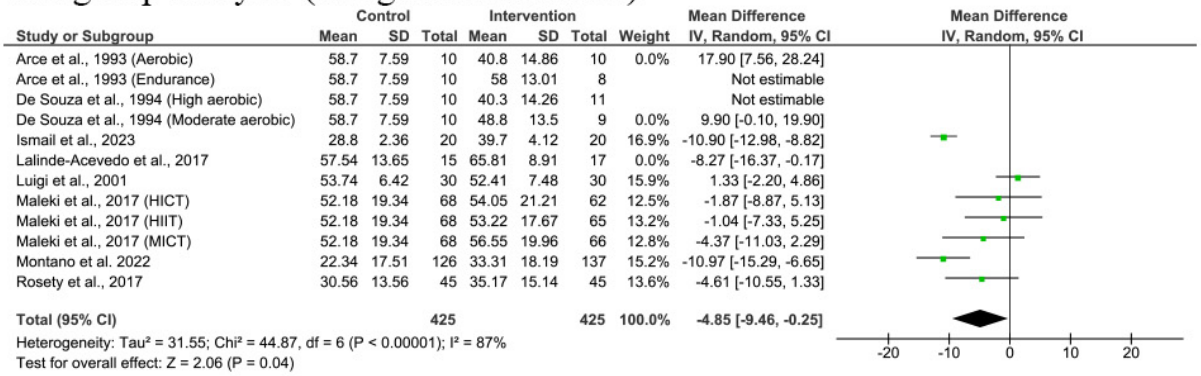


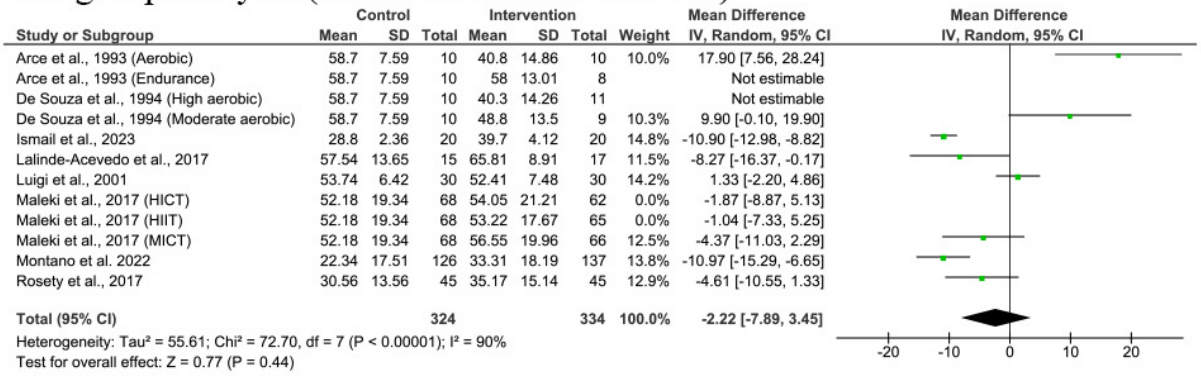
FIGURE 6. Effect of exercise on sperm total motility [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval.



Subgroup analysis (Longitudinal studies)



Subgroup analysis (Moderate aerobic exercise)



Sensitivity analysis

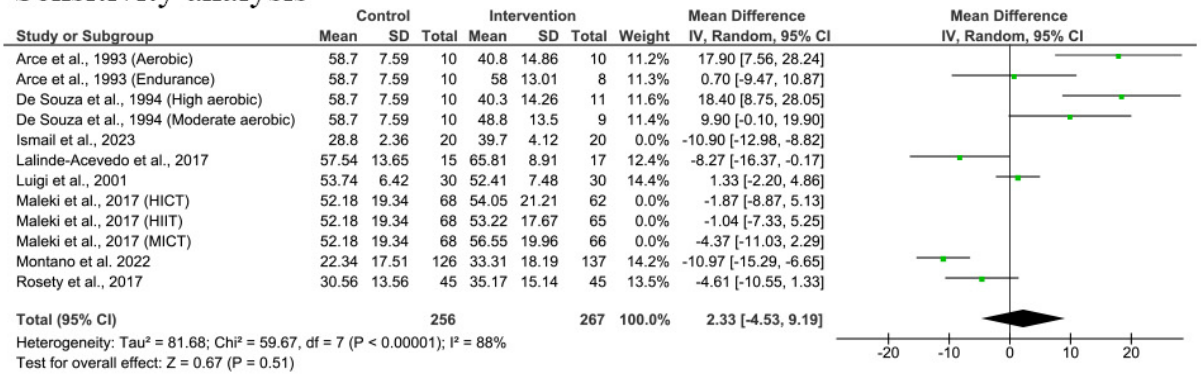
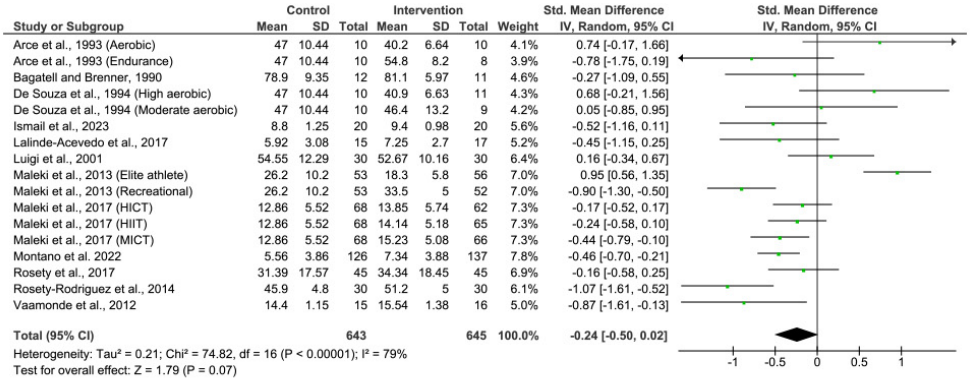
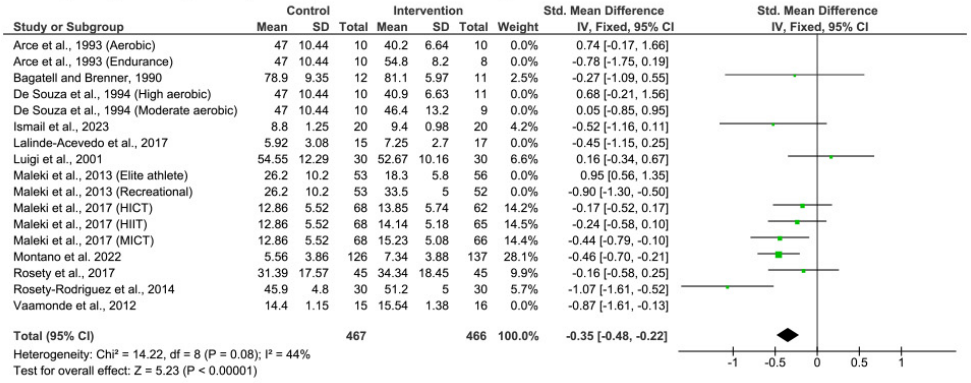


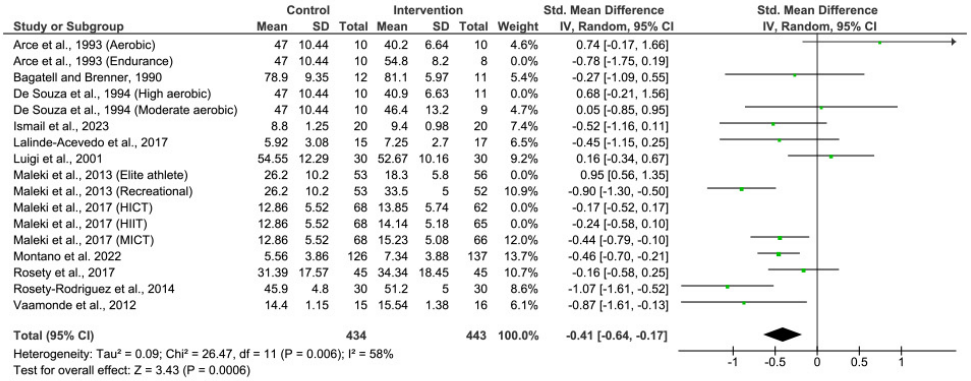
FIGURE 7. Effect of exercise on sperm progressive motility [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval; HICT: High-intensity continuous exercise; HIIT: High-intensity interval exercise; MICT: Moderate-intensity continuous exercise.



Subgroup analysis (Longitudinal studies)



Subgroup analysis (Moderate aerobic exercise)



Sensitivity analysis

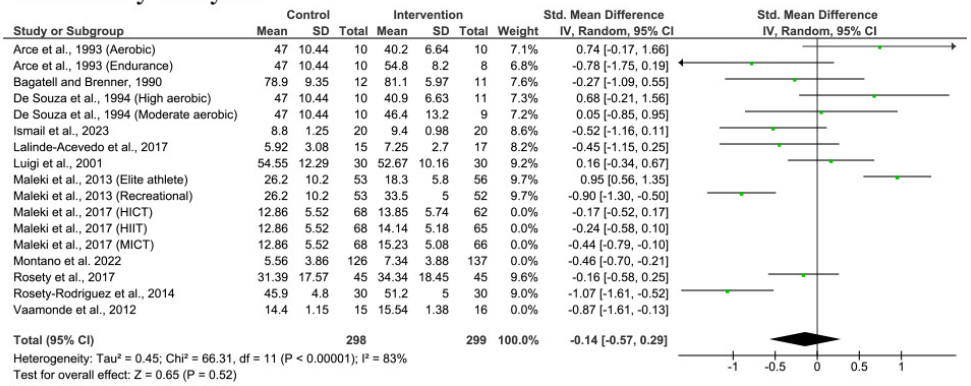


FIGURE 8. Effect of exercise on sperm normal morphology [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval; HICT: High-intensity continuous exercise; HIIT: High-intensity interval exercise; MICT: Moderate-intensity continuous exercise.

contrast, the sensitivity analysis demonstrated that there was no considerable impact of exercise on sperm normal morphology (−0.14 [−0.57, 0.29] 0.52) but with an inter-study heterogeneity (I^2 = 83%; χ^2 $p < 0.00001$) (Fig. 8).

3.4.7 Sperm DNA fragmentation

The influence of exercise on sperm DNA fragmentation was assessed in five studies incorporating 239 control and 230 subjects in the exercise group. The global analysis showed that sperm DNA fragmentation significantly increased in the subjects of the control group when compared with the subjects who exercised (0.60 [0.28, 0.93] 0.0003), but there was inter-study heterogeneity (I^2 = 62%; χ^2 p = 0.03) (Fig. 9, Ref. [33–48]). There was a significant publication bias (**Supplementary Fig. 7**).

Additionally, the subgroup analysis of longitudinal studies revealed that there was a substantial increase in sperm DNA fragmentation in the control group compared to those who exercised (0.71 [0.51, 0.90] <0.00001) and there was no inter-study heterogeneity (I^2 = 13%; χ^2 p = 0.33). Subgroup analysis of the moderate aerobic exercise studies did not show any significant changes in sperm DNA fragmentation (0.50 [−0.15, 1.15] 0.13), but there was also an inter-study heterogeneity (I^2 = 75%; χ^2 p = 0.02). The sensitivity analysis revealed that there were no significant changes in sperm DNA fragmentation in both groups (0.38 [−0.92, 1.68] 0.57), but there was inter-study heterogeneity (I^2 = 86%; χ^2 p = 0.007) (Fig. 9).

3.4.8 Total testosterone

Twelve reports were studied for the effect of exercise on total testosterone. There were 194 subjects in the control group and 191 subjects who exercised. Exercise had no significant impact on total testosterone when compared with the control (−0.27 [−0.95, 0.42] 0.45), but inter-study heterogeneity was significant (I^2 = 88%; χ^2 $p < 0.00001$) (Fig. 10, Ref. [33–48]). There was a significant publication bias (**Supplementary Fig. 8**).

No significant increase was seen in total testosterone in the subgroup analysis of longitudinal studies in subjects who exercised when compared with the control counterpart (−0.51 [−1.16, 0.15] 0.13), and there was substantial inter-study heterogeneity (I^2 = 83%; χ^2 $p < 0.0001$). Subgroup analysis of the moderate aerobic exercise studies revealed that exercise did not significantly affect total testosterone level (−0.70 [−1.76, 0.36] 0.20), but there was also an inter-study heterogeneity (I^2 = 91%; χ^2 $p < 0.00001$). In addition, the sensitivity analysis demonstrated that exercise had no considerable influence on total testosterone (−0.17 [−0.86, 0.51] 0.62), but also with

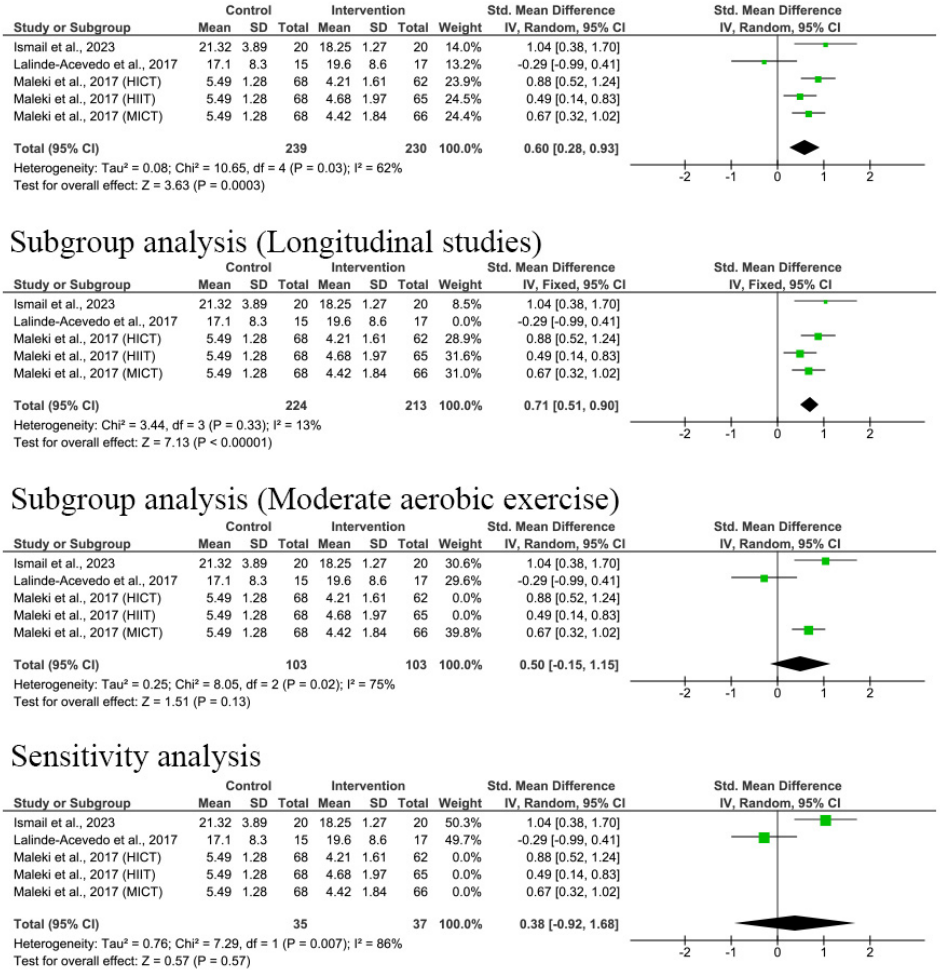
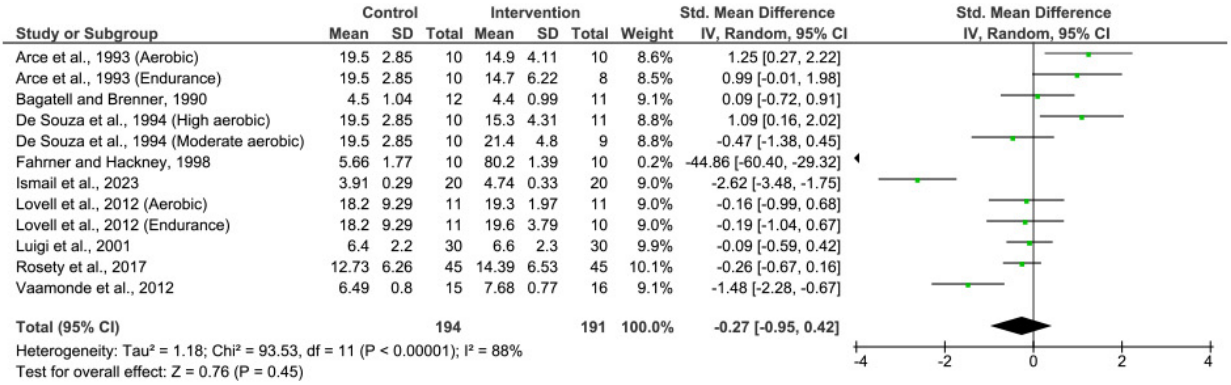
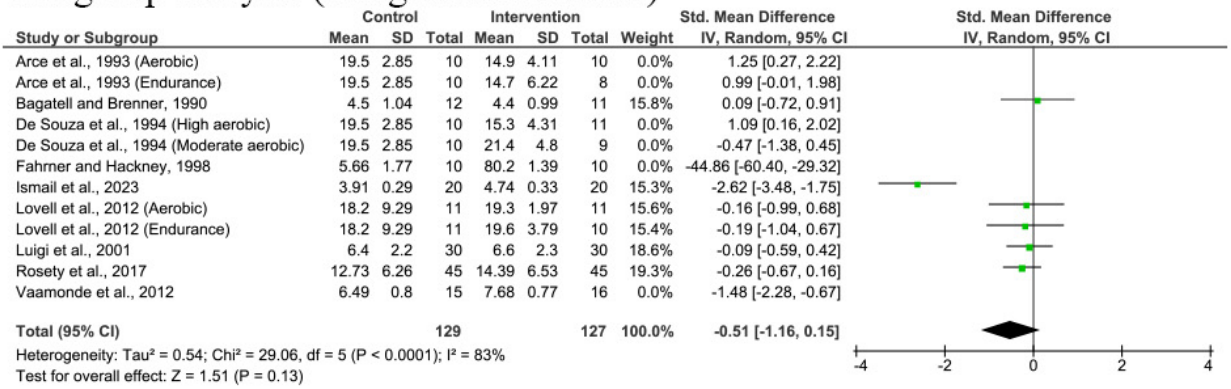


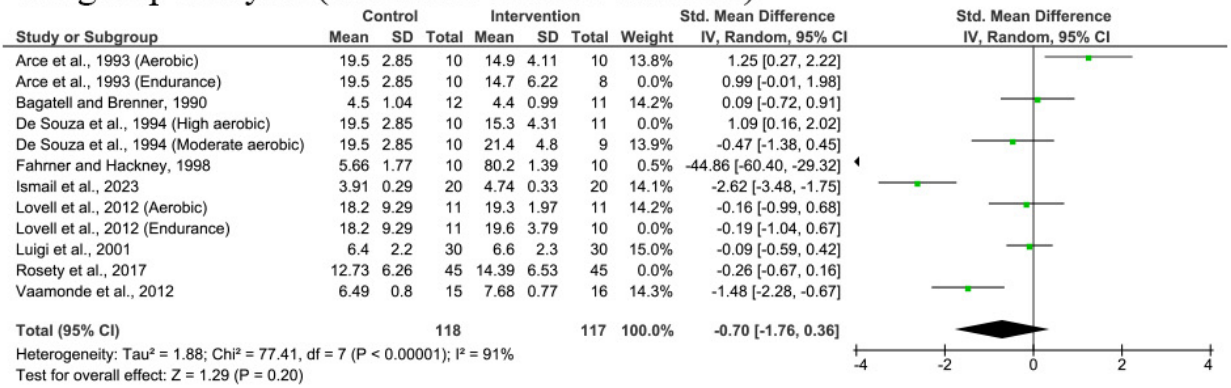
FIGURE 9. Effect of exercise on sperm DNA fragmentation [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval; HICT: High-intensity continuous exercise; HIIT: High-intensity interval exercise; MICT: Moderate-intensity continuous exercise.



Subgroup analysis (Longitudinal studies)



Subgroup analysis (Moderate aerobic exercise)



Sensitivity analysis

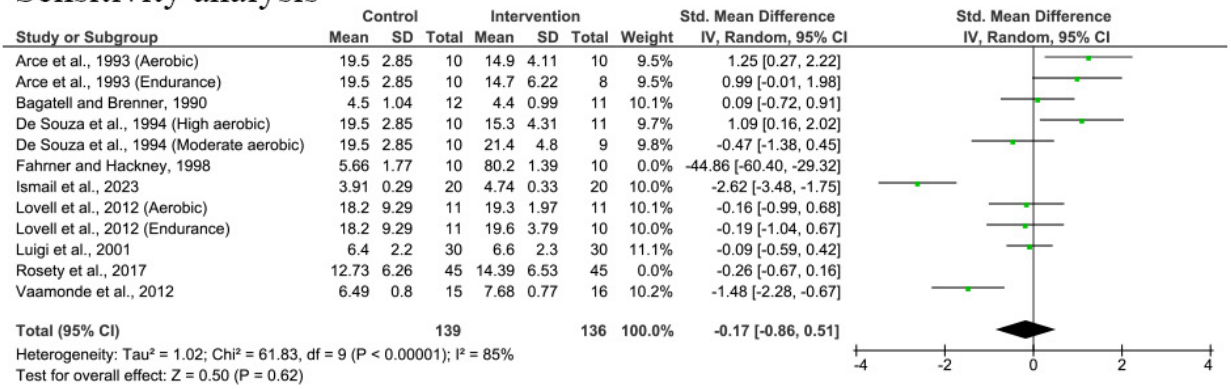


FIGURE 10. Effect of exercise on total testosterone [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval.

inter-study heterogeneity (I^2 = 85%; χ^2 $p < 0.00001$) (Fig. 10).

3.4.9 Free testosterone

A control and intervention group consisting of 114 and 110 subjects, respectively, from nine studies, were assessed for the effect of exercise on free testosterone. Exercise had no significant impact on free testosterone when both groups were compared (0.00 [−0.61, 0.61] 1.00), but there was inter-study heterogeneity (I^2 = 78%; χ^2 $p < 0.0001$) (Fig. 11, Ref. [33–48]). There was no significant publication bias (**Supplementary Fig. 9**).

The subgroup analysis of longitudinal studies also showed no noteworthy increase in free testosterone in subjects who exercised when compared with the control group (−0.00 [−0.36, 0.35] 0.98) and there was no inter-study heterogeneity (I^2 = 0%; χ^2 p = 0.42). Subgroup analysis of the moderate aerobic exercise studies also revealed that exercise did not considerably affect free testosterone level (−0.32 [−1.10, 0.46] 0.42), but there was also inter-study heterogeneity (I^2 = 81%; χ^2 $p < 0.0001$). More so, the sensitivity analysis revealed exercise had no considerable impact on free testosterone (0.24 [−0.35, 0.82] 0.42); however, inter-study heterogeneity was significant (I^2 = 66%; χ^2 p = 0.008) (Fig. 11).

3.4.10 Follicle-stimulating hormone

The effect of exercise on follicle-stimulating hormone was assessed in nine studies, including 152 controls and 150 subjects in the exercise group. Exercise had no substantial influence on FSH when both groups were compared (−0.21 [−0.60, 0.19] 0.31), but there was inter-study heterogeneity (I^2 = 62%; χ^2 p = 0.007) (Fig. 12, Ref. [33–48]). There was a significant publication bias (**Supplementary Fig. 10**).

Conversely, the subgroup analysis of longitudinal studies showed a significant increase in FSH in the control group than those who exercised (0.37 [0.07, 0.67] 0.02), and there was no inter-study heterogeneity (I^2 = 4%; χ^2 p = 0.35). In contrast, subgroup analysis of the moderate aerobic exercise studies revealed that exercise did not significantly alter FSH (−0.17 [−0.64, 0.31] 0.49), but there was an inter-study heterogeneity (I^2 = 69%; χ^2 p = 0.003). The sensitivity analysis also demonstrated that the effect of exercise on FSH was comparable across both groups (−0.30 [−0.81, 0.21] 0.25), but with inter-study heterogeneity (I^2 = 65%; χ^2 p = 0.009) (Fig. 12).

3.4.11 Luteinizing hormone

Nine studies were evaluated for the influence of exercise on LH, consisting of 152 controls and 150 subjects in the exercise group. The global analysis revealed no significant impact of exercise on LH among those who exercised when compared with the control group (−0.05 [−0.28, 0.18] 0.66), and there was no inter-study heterogeneity (I^2 = 15%; χ^2 p = 0.31) (Fig. 13, Ref. [33–48]). There was no significant publication bias (**Supplementary Fig. 11**).

The subgroup analysis of longitudinal studies also showed no significant effect of exercise on LH in subjects who exercised when compared with the control counterpart (0.15 [−0.15, 0.45] 0.33), and there was no inter-study heterogeneity (I^2 = 0%; χ^2 p = 0.65). Subgroup analysis of the moderate aerobic exercise studies showed that exercise did not significantly alter LH (−0.06 [−0.30, 0.19] 0.66) and there was no inter-study heterogeneity (I^2 = 27%; χ^2 p = 0.66). In addition, the sensitivity analysis revealed that exercise involvement did not significantly alter LH in comparison with the control (−0.21 [−0.49, 0.08] 0.16), and there was no inter-study heterogeneity (I^2 = 2%; χ^2 p = 0.41) (Fig. 13).

3.4.12 Prolactin and estrogen

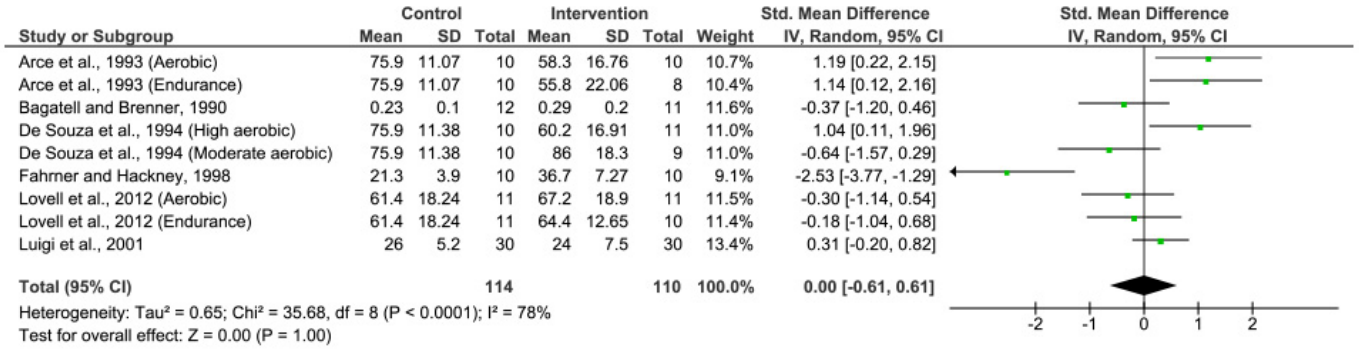
Five studies were assessed for the effect of exercise on prolactin, including 70 controls and 68 subjects in the exercise group. For the effect of exercise on estrogen, four studies were assessed, including 95 controls and 93 subjects in the exercise group. Exercise did not affect prolactin levels when compared with the control (−0.25 [−0.91, 0.41] 0.45), but there was inter-study heterogeneity (I^2 = 69%; χ^2 p = 0.01) (Fig. 14A, Ref. [33–48]). There was a significant publication bias (**Supplementary Fig. 12**). Subgroup analysis of the moderate aerobic exercise studies showed that prolactin was not significantly affected by exercise (−0.07 [−0.95, 0.81] 0.88), but there was also an inter-study heterogeneity (I^2 = 75%; χ^2 p = 0.02). Also, the sensitivity analysis revealed that exercise involvement significantly increased prolactin levels in comparison with the control (−0.51 [−0.97, −0.05] 0.03), but there was no inter-study heterogeneity (I^2 = 0%; χ^2 p = 0.41) (Fig. 14A).

Exercise also did not have any effect on estrogen levels when compared with the control (0.23 [−0.06, 0.52] 0.12), and there was no inter-study heterogeneity (I^2 = 11%; χ^2 p = 0.34) (Fig. 14B). There was a significant publication bias (**Supplementary Fig. 13**). In addition, the subgroup analysis of longitudinal studies for estrogen did not reveal any significant alteration in subjects who exercised when compared with the control counterpart (0.13 [−0.38, 0.65] 0.61), but there was inter-study heterogeneity (I^2 = 60%; χ^2 p = 0.12). Subgroup analysis of the moderate aerobic exercise studies revealed that exercise did not significantly alter oestrogen level (0.19 [−0.11, 0.49] 0.22), and there was also no inter-study heterogeneity (I^2 = 25%; χ^2 p = 0.26). However, the sensitivity analysis for estrogen levels revealed that exercise involvement did not affect levels of estrogen in comparison with the control (0.09 [−0.31, 0.49] 0.65) and there was no inter-study heterogeneity (I^2 = 19%; χ^2 p = 0.29) (Fig. 14B).

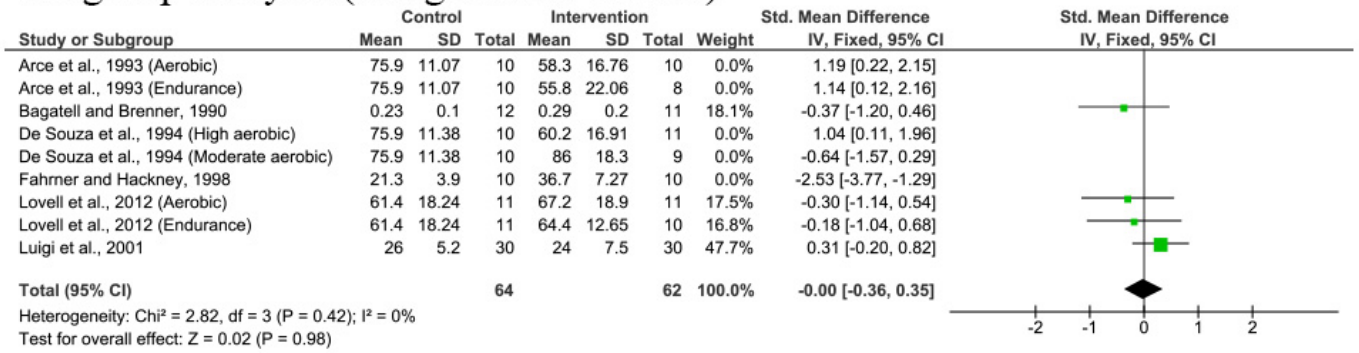
3.4.13 Sex hormone-binding hormone

Four studies were assessed for the effect of exercise on sex hormone-binding globulin, including 44 controls and 42 subjects in the exercise group. Exercise did not significantly increase SHBG when compared with the control (−0.02 [−0.44, 0.41] 0.94), and there was no inter-study heterogeneity (I^2 = 0%; χ^2 p = 0.97) (Fig. 15, Ref. [33–48]). There was a significant publication bias (**Supplementary Fig. 14**).

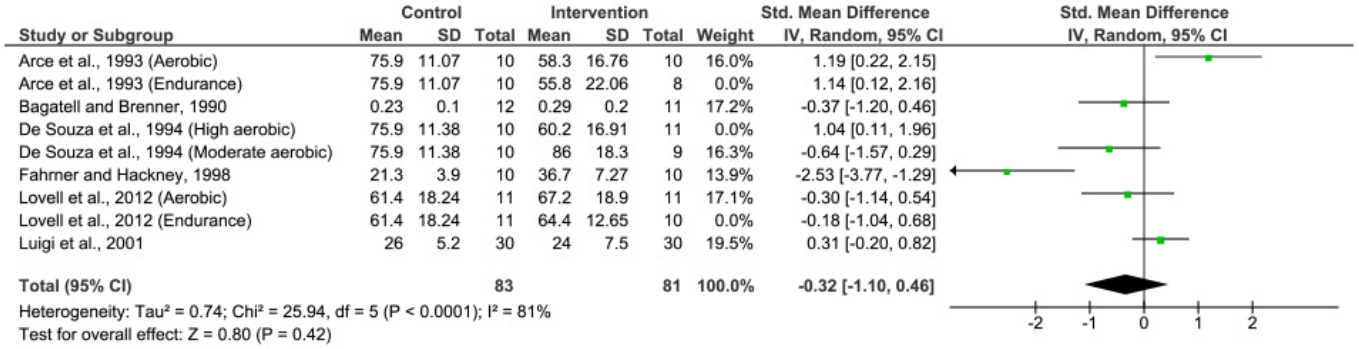
The subgroup analysis of longitudinal studies also showed that exercise did no significantly alter SHBG (0.04 [−0.44, 0.52] 0.87) and there was no inter-study heterogeneity (I^2 = 0%; χ^2 p = 0.99). Subgroup analysis of the moderate aerobic exercise studies revealed that exercise did not considerably alter SHBG (−0.05 [−0.53, 0.44] 0.85), and there was also no inter-study heterogeneity (I^2 = 0%; χ^2 p = 0.91). In addition, the sensitivity analysis revealed that exercise involvement



Subgroup analysis (Longitudinal studies)



Subgroup analysis (Moderate aerobic exercise)



Sensibility analysis

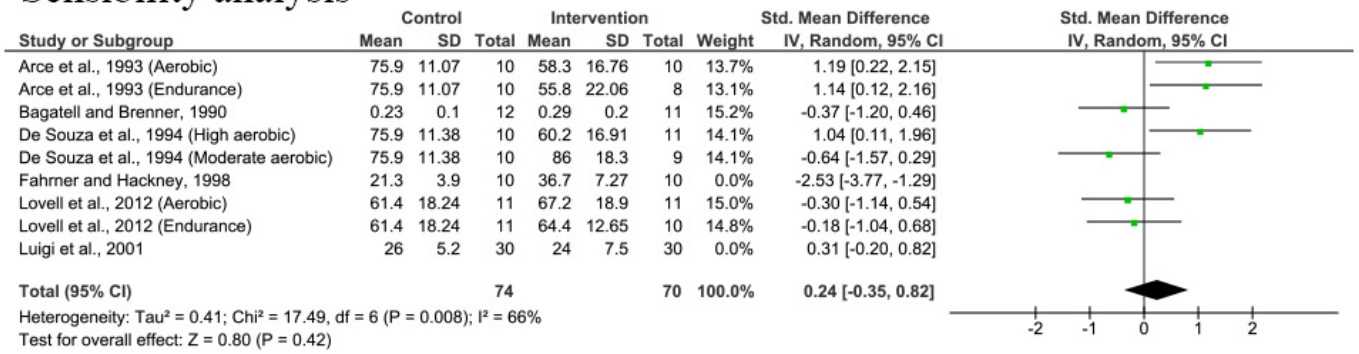
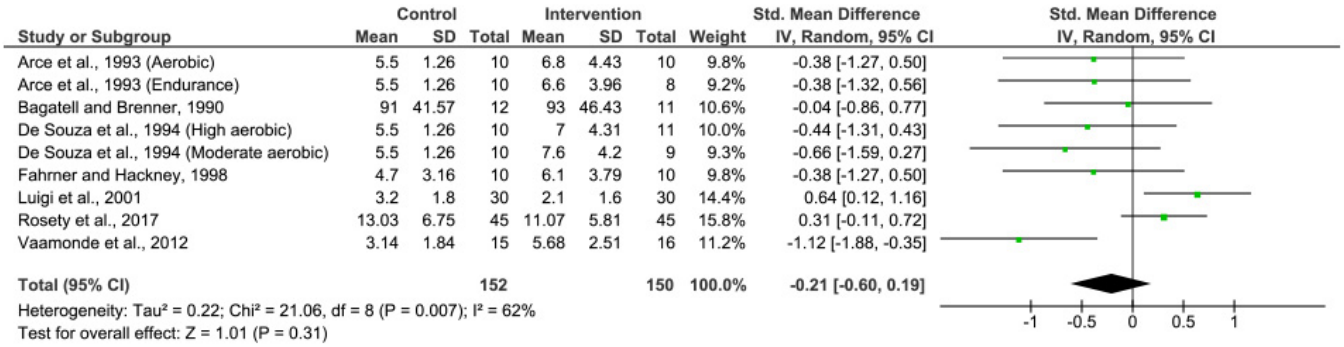
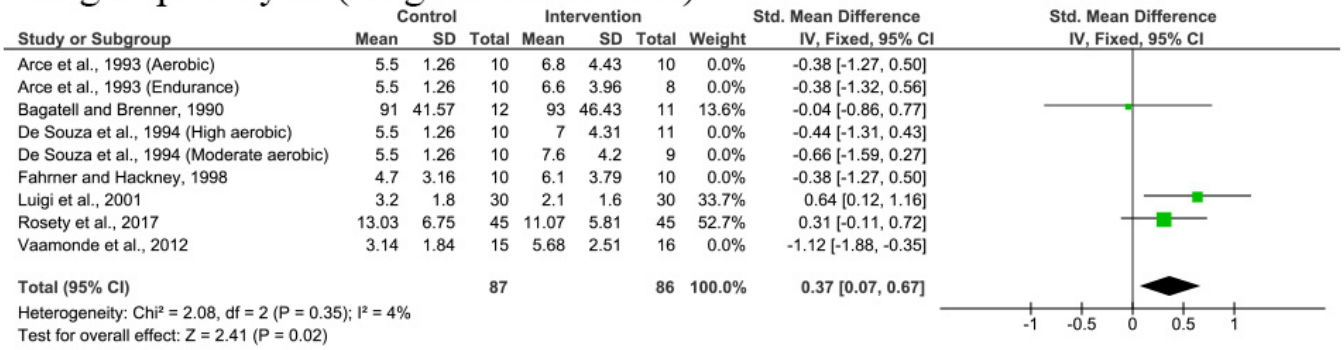


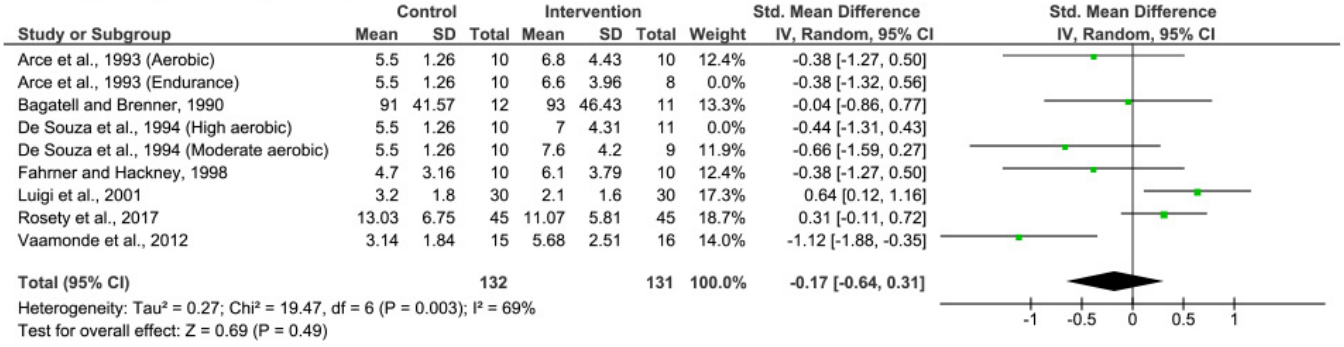
FIGURE 11. Effect of exercise on free testosterone [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval.



Subgroup analysis (longitudinal studies)



Subgroup analysis (Moderate aerobic exercise)



Sensitivity analysis

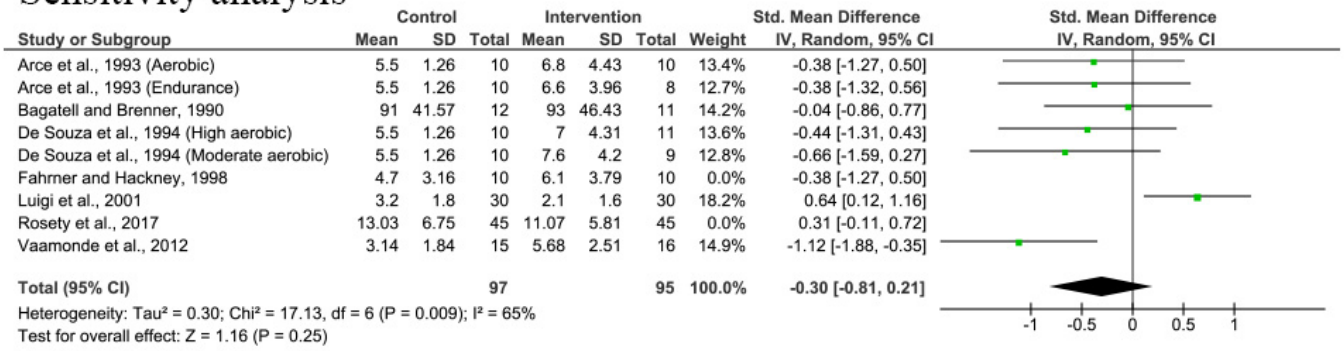
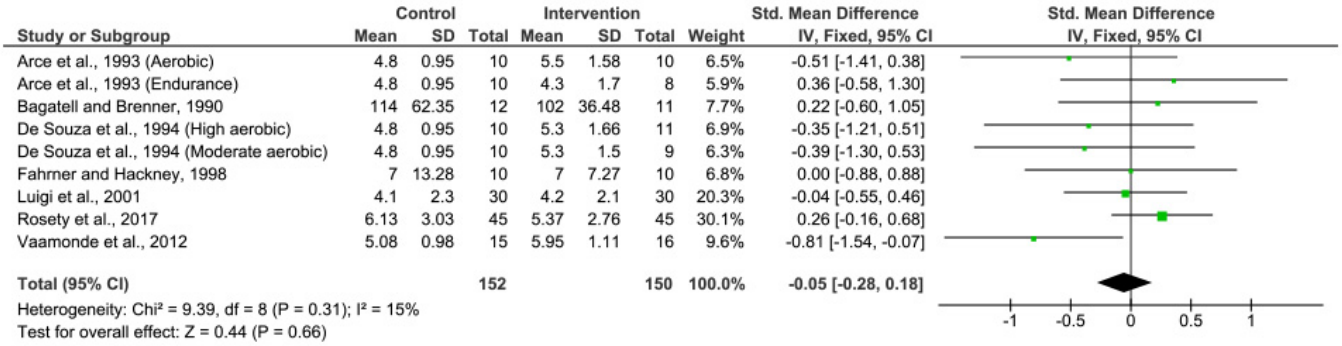
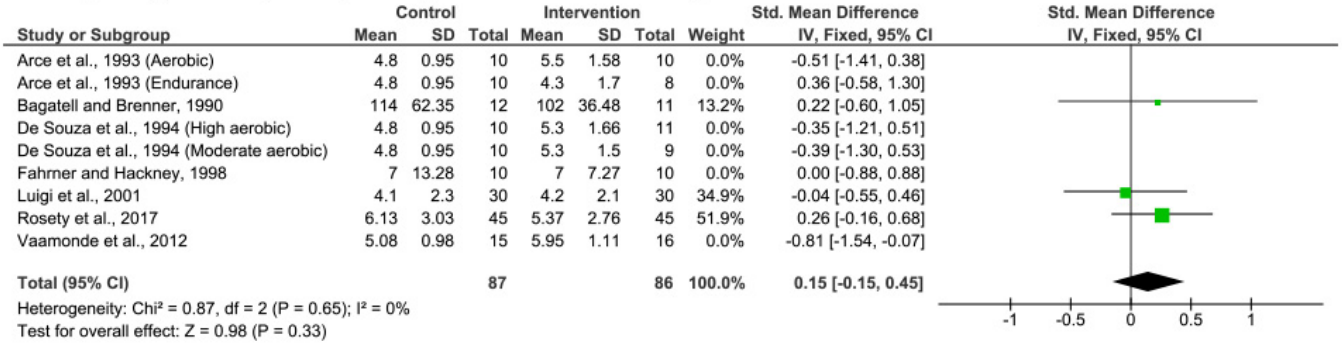


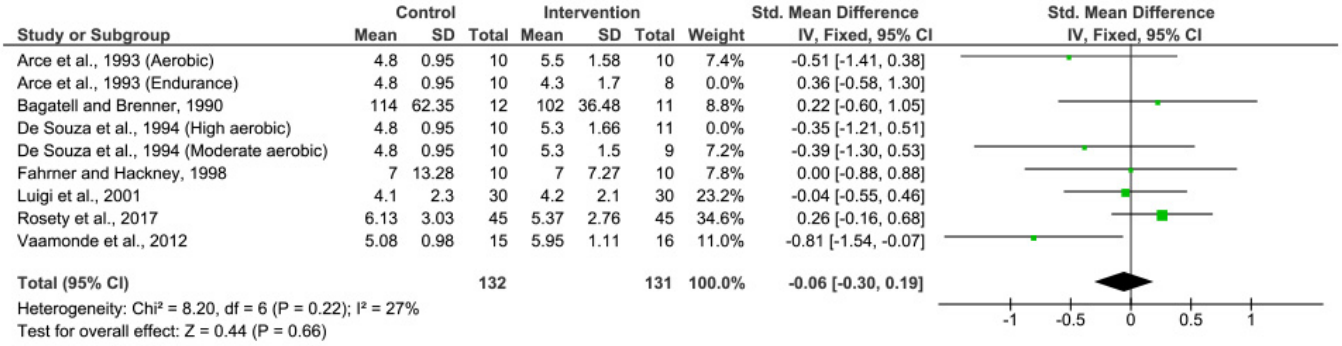
FIGURE 12. Effect of exercise on follicle-stimulating hormone [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval.



Subgroup analysis (Longitudinal studies)



Subgroup analysis (Moderate aerobic exercise)



Sensitivity analysis

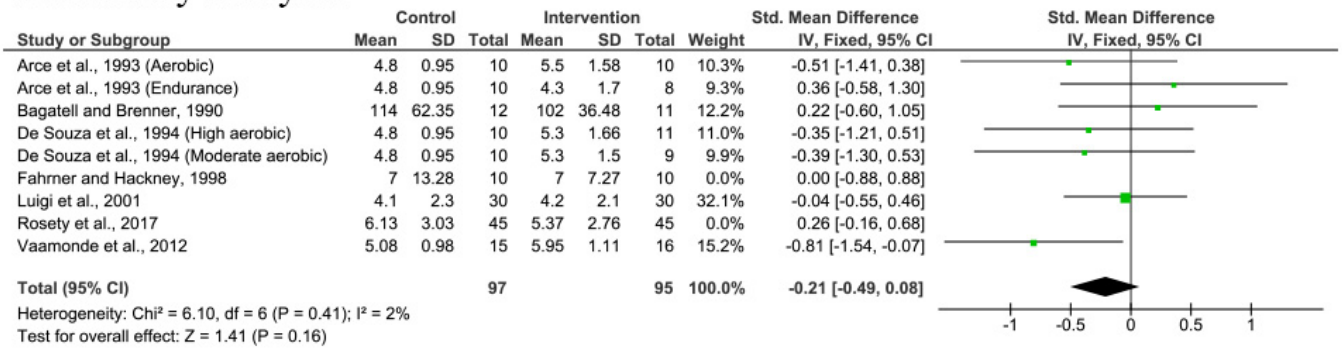


FIGURE 13. Effect of exercise on luteinizing hormone [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval.

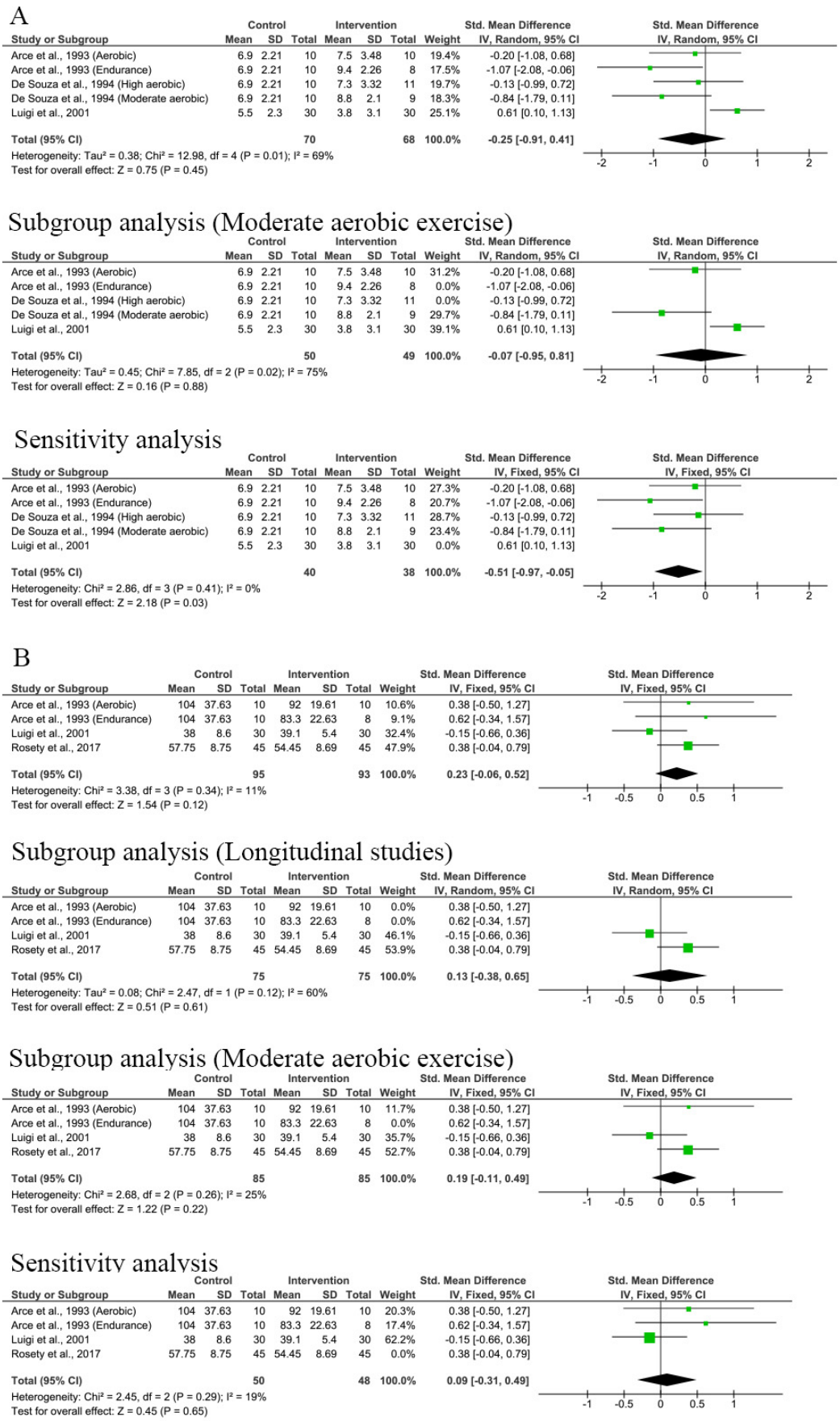


FIGURE 14. Effect of exercise on (A) prolactin and (B) oestrogen [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval.

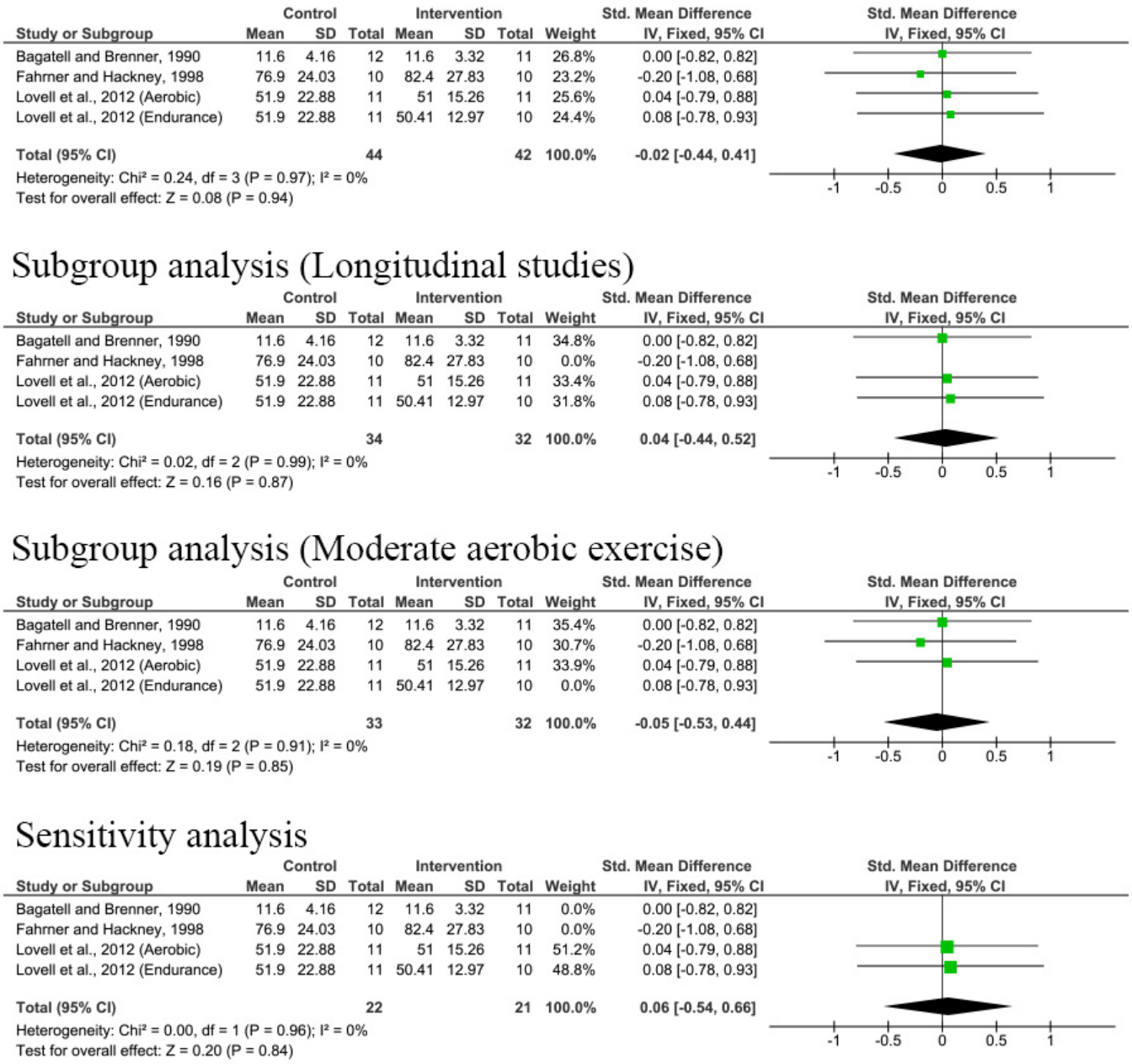


FIGURE 15. Effect of exercise on sex hormone-binding hormone [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval.

did not significantly increase SHBG in comparison with the control counterpart (0.06 [−0.54, 0.66] 0.84) and there was no inter-study heterogeneity (I^2 = 0%; χ^2 p = 0.96) (Fig. 15).

3.4.14 Seminal ROS

Five studies were assessed for the effect of exercise on seminal fluid reactive oxygen species (ROS), including 310 control and 297 subjects in the exercise group. There was no significant impact of exercise on seminal ROS (72.96 [−165.65, 311.58] 0.55), but there was inter-study heterogeneity (I^2 = 98%; χ^2 p < 0.00001) (Fig. 16, Ref. [33–48]). There was a significant publication bias (**Supplementary Fig. 15**).

There was a substantial reduction in the seminal ROS in subjects who exercised when compared with the control counterpart in the subgroup analysis of longitudinal studies (202.58 [5.09, 400.07] 0.04), and there was inter-study heterogeneity (I^2 = 95%; χ^2 p < 0.00001). Subgroup analysis of the moderate aerobic exercise studies revealed that exercise significantly reduced seminal ROS (137.79 [80.67, 194.90] <0.00001), and there was also no inter-study heterogeneity (I^2 = 0%; χ^2 p = 0.56). On the contrary, the sensitivity analysis revealed that exercise involvement did not significantly increase seminal ROS in comparison with the control (−123.15 [−670.96, 424.66] 0.66), but there was an inter-study heterogeneity (I^2 = 99%; χ^2 p < 0.00001) (Fig. 16).

3.4.15 Seminal MDA

An assessment of five studies was carried out on the effect of exercise on seminal MDA concentration, including 310 control and 301 subjects in the exercise group. Exercise did not significantly affect seminal MDA concentration when compared with the control (0.02 [−0.17, 0.22] 0.81), but there

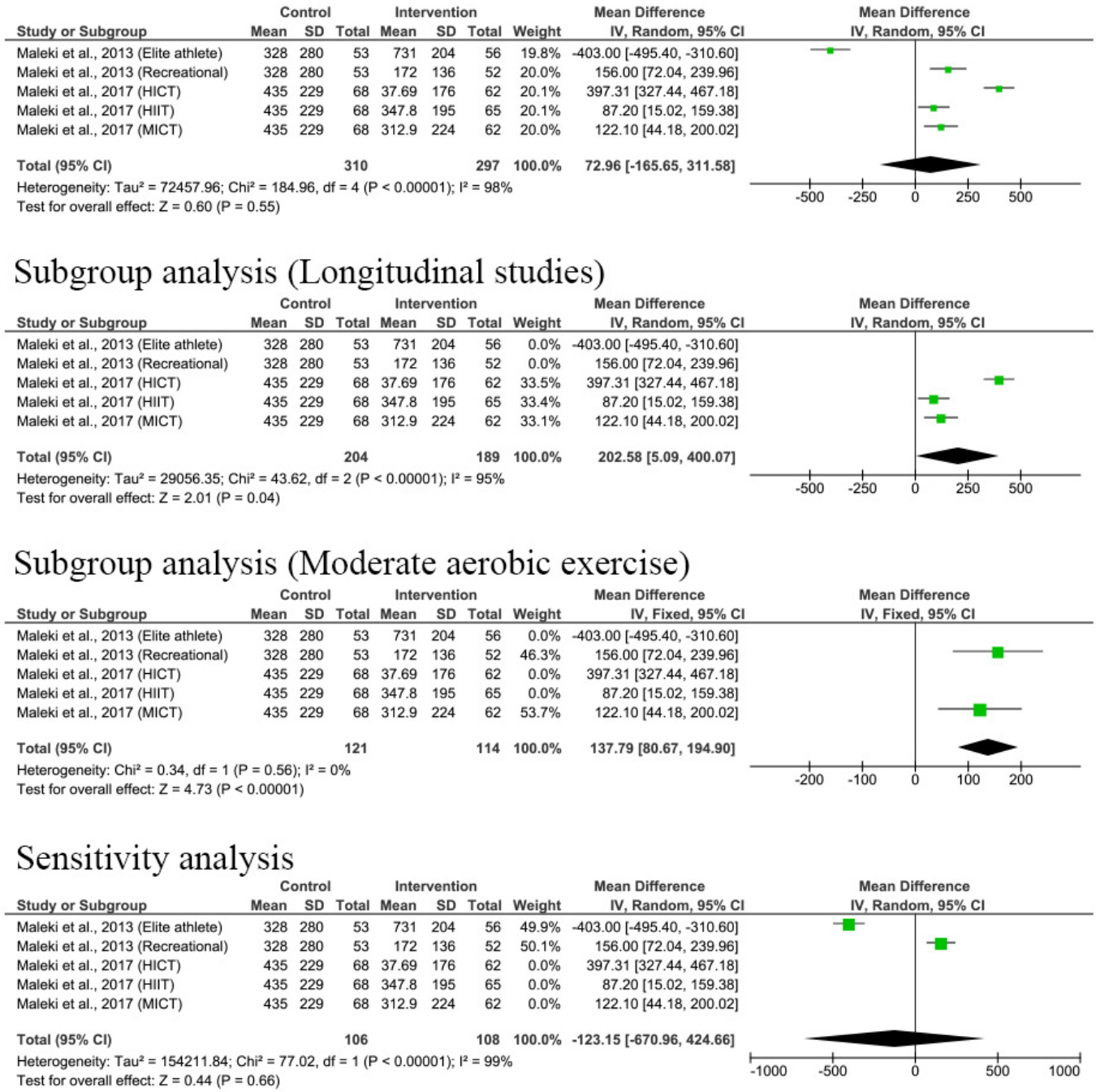


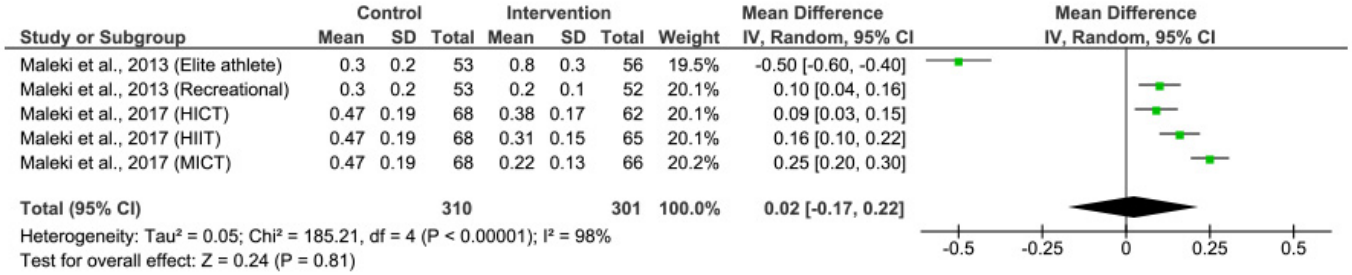
FIGURE 16. Effect of exercise on seminal fluid reactive oxygen species (ROS) [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval; HICT: High-intensity continuous exercise; HIIT: High-intensity interval exercise; MICT: Moderate-intensity continuous exercise.

was inter-study heterogeneity (I^2 = 98%; χ^2 p < 0.00001) (Fig. 17, Ref. [33–48]). There was a significant publication bias (**Supplementary Fig. 16**).

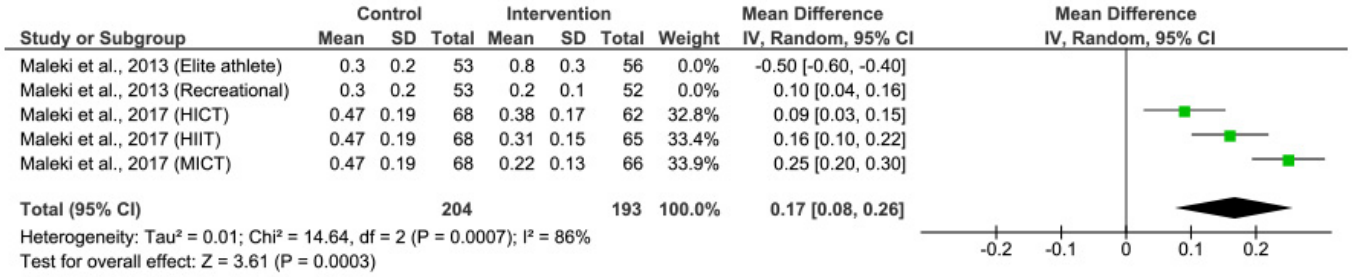
However, the subgroup analysis of longitudinal studies showed a considerably higher seminal MDA concentration in subjects who were in the control group in comparison with those who exercised (0.17 [0.08, 0.26] 0.0003), and there was also inter-study heterogeneity (I^2 = 86%; χ^2 p = 0.0007). Subgroup analysis of the moderate aerobic exercise studies also revealed that exercise markedly reduced seminal MDA (0.18 [0.03, 0.32] 0.02), but there was also an inter-study heterogeneity (I^2 = 92%; χ^2 p = 0.0003). The sensitivity analysis revealed that exercise involvement did not significantly increase seminal MDA concentration in comparison with the control (−0.20 [−0.79, 0.39] 0.51), but there was an inter-study heterogeneity (I^2 = 99%; χ^2 p < 0.00001) (Fig. 17).

3.4.16 Seminal TAC

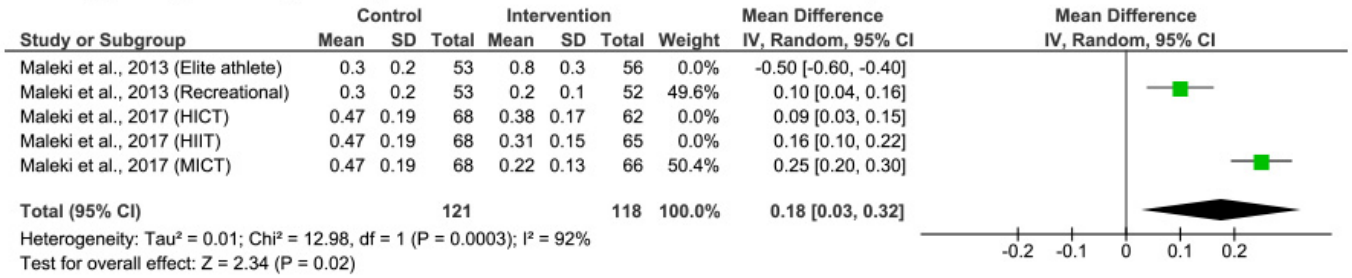
Six studies were assessed for the effect of exercise on seminal TAC, including 436 controls and 438 subjects in the exercise group. Seminal TAC was significantly higher in the subjects who exercised compared with those in the control group (−1.71 [−2.85, −0.58] 0.003), and there was inter-study heterogeneity



Subgroup analysis (Longitudinal studies)



Subgroup analysis (Moderate aerobic exercise)



Sensitivity analysis

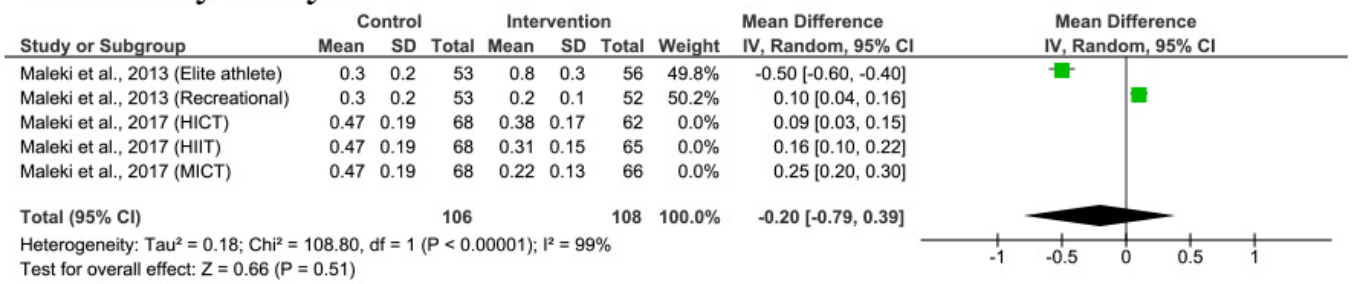


FIGURE 17. Effect of exercise on seminal fluid malondialdehyde (MDA) concentration [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval; HICT: High-intensity continuous exercise; HIIT: High-intensity interval exercise; MICT: Moderate-intensity continuous exercise.

(I^2 = 98%; χ^2 p < 0.00001) (Fig. 18, Ref. [33–48]). There was a significant publication bias (**Supplementary Fig. 17**).

The subgroup analysis of longitudinal studies showed that exercise had a marginal positive impact on seminal TAC in subjects who exercised when compared with the control counterpart (−0.29 [−0.59, 0.02] 0.06); inter-study heterogeneity was significant (I^2 = 73%; χ^2 p = 0.01). Subgroup analysis of the moderate aerobic exercise studies demonstrated that exercise significantly improved seminal TAC (−5.86 [−8.43, −3.29] <0.00001), but there was also inter-study heterogeneity (I^2 = 99%; χ^2 p < 0.00001). In contrast, the sensitivity analysis revealed that exercise involvement did not significantly improve seminal TAC in comparison with the control (−10.69 [−34.32, 12.95] 0.38), but there was an inter-study heterogeneity (I^2 = 100%; χ^2 p < 0.00001) (Fig. 18).

3.4.17 Cortisol

A total of five studies were assessed for the effect of exercise on serum cortisol, consisting of 57 controls and 57 subjects in the exercise group. Exercise did not significantly impact serum cortisol when compared with the control (−0.32 [−1.02, 0.38] 0.37), but there was inter-study heterogeneity (I^2 = 69%; χ^2

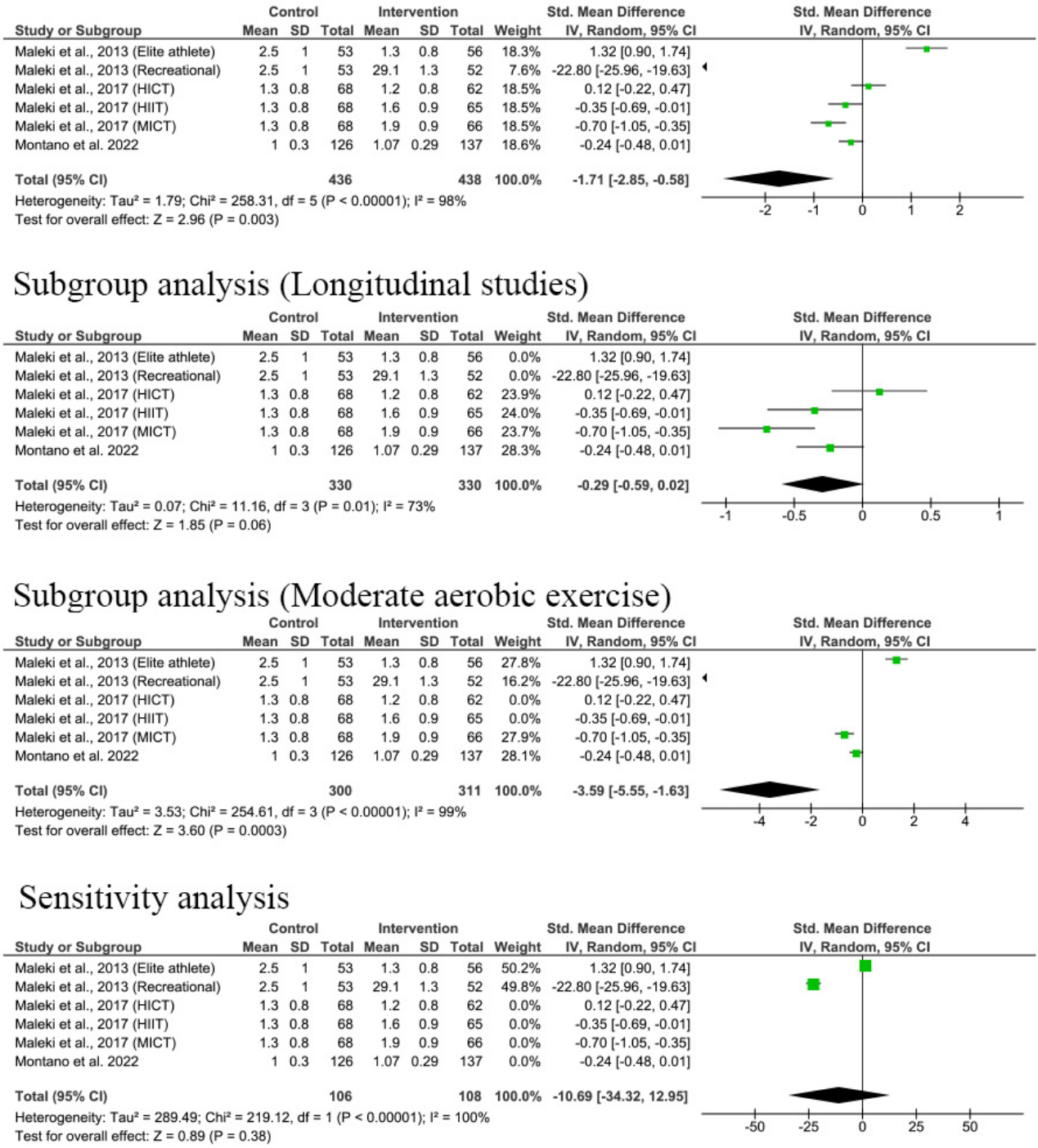


FIGURE 18. Effect of exercise on seminal fluid total antioxidant capacity (TAC) [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval; HICT: High-intensity continuous exercise; HIIT: High-intensity interval exercise; MICT: Moderate-intensity continuous exercise.

$p = 0.01$) (Fig. 19, Ref. [33–48]). There was no significant publication bias (**Supplementary Fig. 18**).

Subgroup analysis of the moderate aerobic exercise studies revealed that cortisol level was not significantly altered by exercise (−0.33 [−1.23, 0.57] 0.47), but there was also an inter-study heterogeneity ($I^2 = 77\%$; χ^2 $p = 0.005$). In addition, the sensitivity analysis revealed that exercise involvement did not significantly increase serum cortisol in comparison with the control (−0.30 [−1.09, 0.50] 0.46), but there was an inter-study heterogeneity ($I^2 = 59\%$; χ^2 $p = 0.09$) (Fig. 19).

3.5 Qualitative analysis of inflammatory markers and pregnancy outcomes

It was observed that exercise attenuated pro-inflammatory cytokines (IL-1β, IL-6, IL-8, and TNF-α) when compared with the control group [44]. Also, exercise resulted in increased pregnancy rate as well as live birth rate when compared with the control [42].

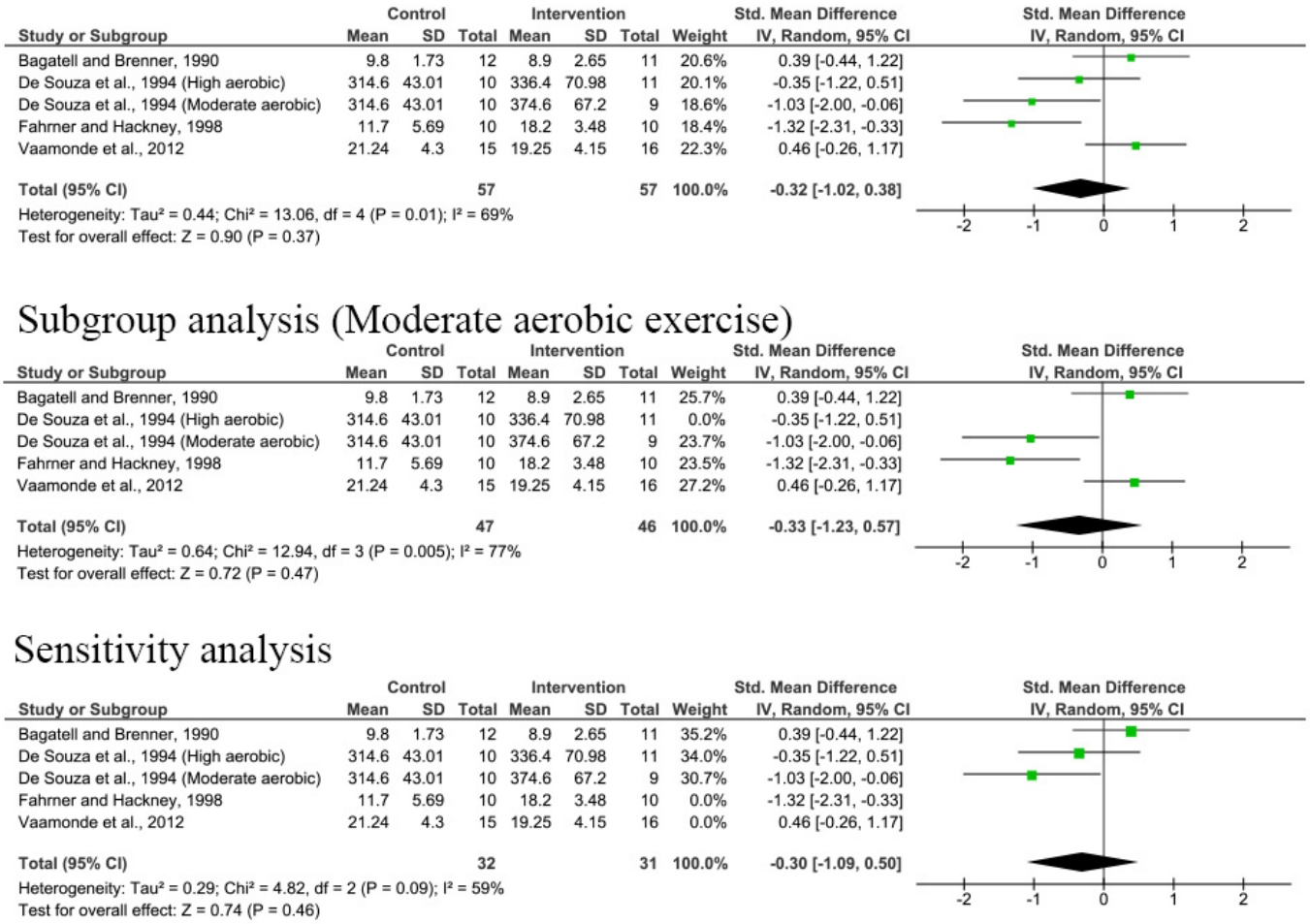


FIGURE 19. Effect of exercise on serum cortisol [33–48]. SD: Standard deviation; IV: Inverse variance; CI: Confidence interval.

4. Discussion

Findings from this study demonstrate that exercise improves sperm quality, especially semen volume, sperm count, concentration, progressive motility, and normal morphology. In addition, exercise reduced sperm DNA damage and increased pregnancy rate and live birth rate. These are associated with increased TAC and reduced ROS, MDA, and pro-inflammatory cytokines.

Conventional semen analysis assesses fundamental physiological sperm variables, such as semen volume, sperm count, concentration, motility, and morphology, to screen for male infertility, while advanced analyses, like DNA fragmentation and oxidative stress markers, give deeper insights into sperm function and sperm fertilization capacity. Sperm DNA fragmentation promotes sperm damage, and elevated sperm DNA fragmentation is associated with reduced pregnancy and increased adverse fertility outcomes [49, 50]. This study shows that exercise significantly improves basic sperm parameters and sperm DNA. This agrees with a previous study that reported reduced sperm DNA fragmentation in men who are on an exercise regimen and antioxidants [51]. Also, it agrees with previous studies that demonstrated improved sperm DNA in men on a weight loss program [52, 53]. However, this does not agree with the study of Håkonsen and his colleagues [54], who did not observe any significant improvement in sperm chromatin structure in men undergoing weight control and exercise. Although studies reporting the impact of exercise on pregnancy rate and live birth rate in couples whose husbands were on an exercise regimen are scanty, the present findings that exercise improved pregnancy rate and live birth rates may be attributed to the improvement of sperm quality in men who are engaged in exercise since sperm DNA integrity is positively correlated with improved pregnancy rate and live birth rate [49, 50]. This agrees with an earlier study that reported increased pregnancy rate and live birth rates [52], but not with another that showed no association between exercise and clinical pregnancy or live birth rates [24]. The observed variation may be due to the type, intensity, and duration of the exercise regimen.

In an attempt to explore the role of male sex hormones and stress in the link between exercise and sperm quality, the impact of exercise on testosterone, LH, FSH, SHBG, oestrogen, prolactin, and cortisol levels was investigated. SHBG regulates the bioavailability of testosterone [55], which, at an optimal level, maintains spermatogenesis and sperm protein expression, thus enhancing sperm quality [56]. LH stimulates the Leydig cells to produce testosterone, while FSH promotes Sertoli cell function that is essential for sperm maturation [57]. Elevated oestrogen disrupts hormonal balance, impair-

ing spermatogenesis and lowering sperm quality [58], while elevated prolactin suppresses gonadotropin-releasing hormone (GnRH), LH, FSH, and testosterone, leading to hypogonadism and poor semen quality [59]. Overall, physiological levels of these hormones are essential for spermatogenesis, sperm quality, and overall fertility potential. Remarkably, the present study shows that exercise does not significantly alter male sex hormones and cortisol levels. Although exercise was found to increase prolactin level following a global analysis of the included studies, subgroup analysis of the moderate aerobic exercise revealed no significant changes in the prolactin level; this suggests that the increased prolactin level may be due to the type and intensity of the exercise engaged in.

Sperm DNA fragmentation is mediated by seminal oxidative stress [60, 61]. In addition, seminal ROS level and MDA generation are positively correlated with adverse pregnancy outcomes [62]. Cellular antioxidant levels and TAC influence incident oxidative stress [63], thus modulating sperm DNA fragmentation and pregnancy outcomes. The present study showed that exercise elevated seminal TAC and reduced seminal ROS and MDA. This infers that exercise, especially moderate aerobic exercise, improves sperm quality and pregnancy outcomes by downregulating seminal ROS and MDA generation and enhancing TAC. Oxidative stress is a cause and consequence of inflammation [64]; thus, seminal oxidative stress may promote seminal inflammation and *vice versa*. The present findings that exercise reduces pro-inflammatory cytokines in association with improved sperm quality and pregnancy outcomes, and attenuated oxidative stress, suggest that exercise protects sperm cells by downregulating cytokine accumulation via suppression of seminal oxidative stress.

Despite the substantial facts presented in the present study, it has some limitations. First, there are scanty RCTs that were eligible for inclusion. This possibly affected the quality of the included studies and the study outcome. Also, most of the analyses showed publication bias. This may be due to the varying degree of exercise intensity and duration, study design, and different methods of determining the endpoints. Nonetheless, this study seems to be the first meta-analysis on the effect of exercise on semen quality and pregnancy outcomes with an exploration of the possible role of inflammation and oxidative stress.

5. Conclusions

In conclusion, exercise improves sperm quality and pregnancy outcomes by downregulating seminal inflammation and oxidative stress. This may be independent of testosterone and cortisol. However, well-designed RCTs should be conducted to investigate the role of graded aerobic and resistance exercises with varying durations on sperm quality. This will offer a useful tool in the non-pharmacological and non-surgical management of male infertility.

AVAILABILITY OF DATA AND MATERIALS

The data used to support the findings of the present study are available from the corresponding author upon request.

AUTHOR CONTRIBUTIONS

REA—Conceptualized and designed the study, supervised the study, validated the data, participated in fund acquisition, developed the methodology, participated in the investigation, participated in project administration, wrote the original draft, reviewed, edited and approved the final draft. BMA and CAA—developed the methodology, participated in the investigation, participated in project administration, wrote the original draft, reviewed, edited and approved the final draft. AAO, OAA, PJA, DTM, VJA, AEA, LBO, FFA—participated in the investigation, participated in project administration, reviewed, edited and approved the final draft. TMA—participated in fund acquisition, developed the methodology, participated in the investigation, participated in project administration, wrote the original draft, reviewed, edited and approved the final draft. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

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CONFLICT OF INTEREST

The authors have no conflicts of interest. The authors declare no conflict of interest. Roland Eghoghosoa Akhigbe is serving as one of the Editorial Board members of this journal. We declare that Roland Eghoghosoa Akhigbe had no involvement in the peer review of this article and has no access to information regarding its peer review. Full responsibility for the editorial process for this article was delegated to MS.

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found, in the online version, at https://oss.jomh.org/files/article/208272345768071987 2/attachment/Supplementary%20material.zip.

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