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Highlights

- This crossover study examined the acute effects of masturbation-induced orgasm performed 30 min before maximal exercise in well-trained male athletes.
- Post-orgasmic sexual activity produced small, transient increases in HR, testosterone, and cortisol, without impairing athletic performance.
- Modest improvements were observed in exercise duration and mean handgrip strength compared with abstinence.
- No significant alterations were found in lactate accumulation, inflammation (CRP, IL-6), or muscle damage markers (CK, LDH, Mb).
- These findings indicate that short-interval sexual activity before exercise does not hinder performance and may reflect transient sympathetic–neuroendocrine activation rather than fatigue.

Sexual activity before exercise influences physiological response and sports performance in high-level trained men athletes.

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Abstract

Background: The influence of sexual activity prior to exercise on athletic performance remains controversial. While pre-competition abstinence is commonly advised, scientific evidence on its physiological impact is limited and inconsistent.

Methods: A randomized crossover study was conducted in 21 well-trained male athletes (age 22 ± 1 y) to compare the acute effects of masturbation-induced orgasm versus sexual abstinence performed 30 min before testing. Each participant completed an incremental cycling test and an isometric handgrip strength test under both conditions. Blood samples were analyzed for muscle damage (CK, LDH, Mb), inflammatory (CRP, IL-6), and hormonal (testosterone, cortisol, LH) markers.

Results: Compared with abstinence, the post-masturbation condition resulted in a longer exercise duration (+3.2%, $p < 0.01$) and higher heart rate ($p < 0.001$), accompanied by a small increase in mean handgrip strength ($p < 0.05$). Lower plasma LDH levels ($p < 0.001$) indicated reduced muscle stress. Testosterone and cortisol concentrations were significantly higher (both $p < 0.001$), whereas inflammatory markers (CRP, IL-6) showed no significant change.

Conclusions: Masturbation 30 min before exercise elicited mild sympathetic and hormonal activation without detrimental effects on performance or muscle damage. These findings suggest that pre-exercise sexual activity does not impair athletic capacity in trained men, challenging the long-standing myth of mandatory abstinence before competition.

Keywords: sexual activity; exercise performance; testosterone; cortisol; muscle damage biomarkers.

Abbreviations

- Abstinence Condition (ABST)
- Body Mass Index (BMI)
- Changes in plasma volume (%ΔPV)
- Creatine Kinase (CK)
- C-reactive protein (CRP)
- Effect size (ES)
- Exercise-Induced Muscle Damage (EIMD)
- Heart Rate (HR)
- Hour (h)
- Hypothalamic-Pituitary-Adrenal (HPA)
- Hypothalamic-Pituitary-Gonadal (HPG)
- Interleukin 6 (IL-6)
- Kilogram (kg)
- Lactate Dehydrogenase (LDH)
- Luteinizing Hormone (LH)
- Meters (m)
- Minutes (min)
- Myoglobin (Mb)
- Post-Activation Potentiation (PAP)
- Ratings of Perceived Exertion (RPE)
- Revolutions per minute (rpm)
- Sexual Activity Condition (SACT)
- Strengthening The Reporting of Observational Studies in Epidemiology (STROBE)
- Total Time (T)
- Watts (w)
- World Anti-Doping Agency (WADA)

1. Introduction

Sexual activity and physical exercise are two key components of human well-being. A healthy sexual lifestyle has been associated with better physical fitness and quality of life [1], while regular physical activity is known to improve sexual health and function [2]. However, whether sexual activity immediately preceding exercise may influence athletic performance remains a subject of debate [2–9].

Pre-competition sexual activity (i.e., intercourse, masturbation, or orgasm) could potentially affect sports performance through several physiological and psychological pathways: (a) modulation of central and autonomic nervous system activity [10,11]; (b) alterations in psychological state, including motivation, aggression, or concentration [12]; (c) transient hormonal changes such as increased testosterone or catecholamines [13]; (d) disturbances in sleep quality and recovery [4] and (e) alterations in energy metabolism and substrate use [14]. Sexual activity represents a mild-to-moderate physical stressor, inducing increases in heart rate (HR) and blood pressure that may reach 60–70% of maximal exercise values [15]. Cardiovascular responses during arousal and orgasm resemble those elicited by moderate aerobic exertion [16–20]. Thus, sexual activity may acutely activate the sympathetic nervous system in a manner comparable to a brief warm-up [21].

Despite this physiological overlap, scientific findings remain inconsistent. Most studies have shown that sexual activity 8–12 hour (h) before exercise does not impair physical [22,23], aerobic [24,25] or overall exercise performance [26].

However, when the interval between sexual activity and exercise was reduced to less than 2 h, results diverged: several studies found no detrimental effects [22–24,27], whereas others reported reduced lower- or upper-limb strength when sexual activity occurred 12–24 h before testing [28–

30]. A minor transient decline in performance was observed only when exercise followed sexual activity within 2 h, disappearing after adequate recovery [25,31]. The recent meta-analysis conducted by Zavorsky et al. [26] confirmed that pre-exercise sexual activity, regardless of type or timing, exerts negligible effects on aerobic capacity, muscular strength, or power output, aligning with prior systematic reviews [23].

From a neuroendocrine perspective, physical activity itself triggers acute increases in circulating steroid hormones such as testosterone and cortisol, reflecting activation of the hypothalamic–pituitary–gonadal (HPG) and hypothalamic–pituitary–adrenal (HPA) axes [32]. In trained or overreached athletes, chronic stress may blunt testosterone production and elevate cortisol levels, shifting the anabolic–catabolic balance [33–35]. Testosterone supports muscle anabolism, strength, and aggressiveness, whereas cortisol promotes catabolism, hyperglycemia, and cardiovascular activation [35,36]. Experimental data indicate that masturbation-induced orgasm can acutely raise testosterone levels following periods of abstinence [37–39], though other studies report no changes [40,41] or even transient reductions during prolonged sexual stimulation [17]. These discrepancies may depend on timing, type of sexual activity, or baseline hormonal status. Furthermore, masturbation appears to increase both testosterone and cortisol, suggesting a brief sympathetic–adrenal activation without persistent hormonal imbalance [42–44].

Beyond hormonal responses, both physical exercise and sexual activity induce mechanical and metabolic stress on skeletal muscle. Exercise-induced muscle damage (EIMD) involves sarcomere disruption, transient inflammation, and elevations of circulating biomarkers such as creatine kinase (CK), lactate dehydrogenase (LDH), and myoglobin (Mb) [45–47]. Sexual activity also provokes neuromuscular activation and metabolic demands that may, theoretically, modulate these processes via neuroendocrine–immune interactions [48,49]. However, no previous study has

simultaneously assessed biomarkers of muscle damage, inflammation, and hormonal response in the immediate post-orgasmic period before exercise.

Taken together, the literature suggests that the effect of sexual activity on performance depends primarily on the recovery interval, the type of activity (coitus vs masturbation), and the specific physiological parameters analyzed. Nevertheless, data on short post-orgasmic intervals (<1 h) in well-trained athletes are virtually nonexistent. Therefore, the present study aimed to examine acute physical performance (time to exhaustion, applied power, isometric handgrip strength), functional variables (HR, lactate concentration), muscle damage (CK, LDH, Mb), inflammatory (C-reactive protein [CRP], interleukin 6 [IL-6]), and hormonal responses (testosterone, cortisol, Testosterone / Cortisol ratio, luteinizing hormone [LH]), after masturbation-induced orgasm compared with sexual abstinence performed 30 minutes (min) before maximal incremental exercise. We hypothesized that post-orgasmic activation would not impair, and might transiently enhance, selected performance and hormonal parameters in well-trained men.

2. Material and Methods

2.1 Ethical Considerations

The study was approved by the Ethics Committee of the Valladolid East Health Area of the University Clinical Hospital of Valladolid, Spain (approval code: CEIM PI-23-3048; 13 April 2023). All procedures complied with the Declaration of Helsinki [50] and the European Union General Data Protection Regulation (EU GDPR 2016/679). Participants were assured of complete anonymity and confidentiality, particularly regarding any sensitive information related to sexual behavior. No financial incentives were provided to participants. The study was not preregistered in a public database due to its non-interventional, physiological design.

2.2 Subjects

Healthy, physically active adult males were recruited via posters and announcements displayed at the Faculty of Health Sciences (University of Valladolid) and the High-Performance Training Center (CAEP-Soria, Spain). Interested volunteers received written information describing the study's aims, procedures, benefits, and potential risks. All participants provided written informed consent and retained a signed copy. Recruitment took place from January to June 2024.

Twenty-one well-trained male athletes participated (age: 22.0 ± 1.4 years; body mass: 74.5 ± 7.4 kilograms (kg); height: 1.79 ± 0.04 meters (m); body mass index (BMI): 23.2 ± 1.9 kg/m^2). Participants competed at regional, national, or international levels, including basketball (n = 5), volleyball (n = 3), long-distance running (n = 7), boxing (n = 2), and judo (n = 4), with a mean training experience of 8.0 ± 2.9 years and weekly volume of 4.3 ± 0.7 days · $week^{-1}$ for 2.7 ± 0.6 h · day^{-1}.

Inclusion criteria were: (a) age 18–25 years; (b) regular participation in competitive sports for at least three years; (c) no history of cardiovascular, endocrine, or psychiatric disorders; and (d) no current medication or supplementation affecting hormonal or inflammatory responses. All participants identified as heterosexual to ensure a standardized response to the audiovisual sexual stimulus used in the protocol.

Exclusion criteria included: (a) use of antibiotics, anti-inflammatory, or anabolic drugs; (b) history of hormonal, psychological, or sexual dysfunction; (c) consumption of alcohol, caffeine, or recreational drugs 48 h before testing; and (d) sleep deprivation (< 7 h · $night^{-1}$) in the preceding 48 h.

Participants were instructed to maintain their regular diet and training routine throughout the study, refrain from alcohol, and replicate their usual pre-competition meal and hydration on both testing days.

The sample size was calculated *a priori* using G*Power 3.1 software [51] or a crossover design (two conditions, $\alpha = 0.05$, power = 0.95, effect size = 0.8), based on expected within-subject changes in serum testosterone. This analysis indicated a minimum of 18 participants; 21 were finally included to account for potential dropouts. The number of subjects is consistent with previous studies investigating similar physiological responses [29,52].

The study adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines [53]. Of the 46 athletes initially screened, 25 were excluded (9 not meeting inclusion criteria, 5 declining participation, and 4 reporting the use of prohibited substances). An additional 7 were removed during follow-up (5 for non-protocol sexual activity and 2 for missing the second test day). The final sample therefore comprised 21 participants (Figure 1).

2.3 Experimental design

The study followed a randomized crossover design with repeated measures, in which each participant completed both the experimental (sexual activity condition; SACT) and control (sexual abstinence; ABST) conditions, in a counterbalanced and randomized order. To minimize learning effects, all subjects attended a familiarization session one week before testing to practice the exercise protocols.

Testing was conducted on two separate days, one week apart, at the same time of day (starting at 9:00 a.m.) under identical laboratory conditions to minimize circadian variation. The order of

conditions was randomly assigned using a computerized random number generator, resulting in approximately half the participants starting with SACT and half with ABST.

SACT: Participants were instructed to perform masturbation to orgasm in a private room, under unmonitored conditions, 30 min before the start of the exercise tests. A standardized pornographic audiovisual stimulus (heterosexual content, 15 min duration) was provided to ensure comparable sexual arousal across participants. After achieving orgasm, participants remained seated and watched a 15-min neutral documentary film to standardize post-orgasmic relaxation and return to baseline sympathetic tone. The exact time of orgasm was self-reported anonymously via an anonymized electronic message sent directly to the investigator's secure device to ensure accurate timing of data collection.

ABST: In the abstinence condition, participants refrained from any sexual activity (intercourse, masturbation, or petting) for seven days before testing. On the testing morning, they watched only the 15-min neutral documentary film under identical laboratory conditions, ensuring visual and temporal control without sexual stimulation.

All exercise tests (incremental cycling test and isometric handgrip strength test) were performed in the same order and under identical environmental conditions (21–23°C; 40–50% relative humidity) across both sessions. Figure 2 summarizes the experimental sequence.

2.4 Anthropometric parameters

Body mass and stature were measured with participants wearing light clothing and no shoes, using a digital scale (Healthometer, Neosho, MO, USA) and a wall-mounted stadiometer (Heightronics, QuickMedical, Issaquah, WA, USA), respectively. Measurements were taken in the morning,

before testing, following standard anthropometric procedures. BMI was calculated as body mass (kg) divided by height squared (m^2).

2.5 Physical variables

2.5.1 Hand grip strength test

Handgrip strength was assessed using a Jamar hand dynamometer (Lafayette Instrument, Lafayette, IN, USA), with a measurement range up to 90 kg force in 2 kg increments. The test followed the standardized procedure described by Bohannon [54]. Participants performed a warm-up consisting of arm and wrist mobility exercises followed by 10 continuous compressions of a rubber ball. The dynamometer handle was adjusted to hand size and maintained at the same position for all sessions. Tests were performed in a standing position with the elbow fully extended and the dynamometer held beside the hip. Participants executed a maximal isometric contraction lasting 3 seconds, avoiding elbow flexion or body swing. The dominant hand was tested three times with 30 seconds rest between attempts, and both the best (Handgrip_Best) and mean (Handgrip_Mean) values were used for analysis.

2.5.2 Incremental exercise test

Participants performed the incremental cycling test in a temperature-controlled room (21 ± 1 °C; 60 ± 5 % relative humidity). After a 3-min warm-up at 25 watts (W) and 60 revolutions per minute (rpm) [55], the test began at 140 W and 70 rpm and increased by 35 W every min until volitional exhaustion. The test was performed on a braked cycle ergometer (Kettler Axiom P2, GmbH & Co. KG, Ense-Parsit, Germany). HR was continuously monitored using a chest-strap transmitter (Polar H10, Polar Electro, Finland) and recorded on a wrist device (Polar Vantage V3, Polar Electro, Finland).

The following variables were recorded: maximum power, total time (T), peak HR, and relative power (Relative power = Maximum power/body mass). Maximal effort was confirmed when at least two of the following criteria were met: (i) HR > 90 % of age-predicted maximum, (ii) rating of perceived exertion (RPE) > 8 on the Borg CR-10 scale, or (iii) volitional exhaustion. Verbal encouragement was provided uniformly across participants.

2.5.3 Rate of perceived exertion

Perceived exertion was evaluated using the Borg CR-10 scale [56], which ranges from 0 ("rest") to 10 ("maximal effort"). Participants were familiarized with the scale during the preliminary session and asked to report their RPE immediately after each test and before blood sampling.

2.5.4 Blood lactate analysis

Capillary blood lactate concentration was determined only during the incremental test. Samples were collected from the earlobe at rest (pre-test) and within one min after exhaustion (post-test). The ΔLactate value was calculated as the difference between post-test and pre-test concentrations [57] . After cleaning and drying the area, a sterile lancet (Accu-Chek Softclix Pro, Germany) was used for puncture. The first drop of blood was discarded, and 10 µL of capillary blood was collected in non-heparinized microcapillaries using Lactate Scout SENSORS (SensLab GmbH, Leipzig, Germany). Lactate concentration was analyzed by spectrophotometry using a Lactate Scout SPORT analyzer (SensLab GmbH, Leipzig, Germany). Care was taken to avoid excessive pressure during sampling to prevent hemolysis.

2.6 Blood withdrawal and analyses

Blood samples were collected following World Anti-Doping Agency (WADA) regulations for collection and transportation [58]. Venous blood was drawn immediately (< 5 min) after the

incremental exercise test, corresponding to approximately 60 min post-orgasm in the SACT condition, with participants seated and resting for 5 min to standardize posture and minimize hydrostatic variation. All sessions were conducted at the same time of day (10:00 a.m. ± 30 min) to control circadian influences.

Blood was obtained by venipuncture from the antecubital vein using a 10 mL syringe equipped with a 2.5 cm needle and transferred into EDTA-treated Vacutainer® tubes, which were kept on ice until processing. Samples were centrifuged at 2000 × g for 10 min at 4 °C, and plasma and serum aliquots were stored at −80 °C until analysis.

Changes in plasma volume (%ΔPV) were calculated using the method of Van Beaumont [59], and analytical marker values were corrected for plasma volume shifts using the following formula:

Corrected value = Uncorrected value × [(100 + %ΔPV) / 100] [60].

Sample preparation followed procedures previously validated in our laboratory [34,46,47].

2.6.1 Muscle biomarkers

Serum concentrations of CK and LDH were determined using coupled enzymatic reactions on an automated analyzer (Hitachi 917, Hitachi Ltd., Tokyo, Japan) at 37 °C. Mb was measured using a commercially available ELISA kit (Myoglobin ELISA, Merck KGaA, Darmstadt, Germany) according to the manufacturer's instructions. Intra- and inter-assay coefficients of variation were below 6% for all assays.

2.6.2 Hormonal Response

Serum LH, testosterone, and cortisol concentrations were analyzed using automated chemiluminescent immunoassays (ACS 180 System, Chiron Diagnostics, Leverkusen, Germany).

Testosterone and cortisol were quantified using Cobas® immunoassays (testosterone II, ref. 05200067 190; cortisol, ref. 11875116 122; Cobas Asset Management, Madrid, Spain), with analytical ranges of 0.087–52.0 nM and 0.001–1.75 µM, respectively. LH was determined using a commercial ELISA kit (IBL International GmbH, Hamburg, Germany) with a range of 1.27–200 mIU/mL.

Intra- and inter-assay coefficients of variation were 6.3% for LH, 5.6% and 6.6% for testosterone, and 4.5% and 6.4% for cortisol. Hormonal ratios were calculated from total (not free) serum concentrations [61].

2.6.3 Inflammatory parameters

Serum IL-6 and CRP were quantified using commercial ELISA kits (IL-6: ab178013; CRP: BMS288INST; Invitrogen, ThermoFisher Scientific, Waltham, MA, USA), with sensitivities of <2 pg/mL and <10 pg/mL, respectively. All samples were analyzed in duplicate and within the same batch to minimize inter-assay variability. Coefficients of variation were below 8% for both assays.

2.6.4 Statistical analysis

All statistical analyses were conducted using STATA version 15.0 (StataCorp, College Station, TX, USA). Data are presented as mean ± standard deviation, and statistical significance was accepted at $p \leq 0.05$.

Given the two-period crossover design, within-subject comparisons were analyzed using two-way repeated-measures ANOVA (Condition × Time) models, with both factors (Condition: SACT vs. ABST; Time: pre- vs. post-exercise) treated as intra-subject variables. When a significant

interaction (Condition × Time) or main effect of Condition was detected, paired post hoc t-tests (Bonferroni-adjusted) were applied to locate differences.

To verify the integrity of the crossover design, a preliminary two-way ANOVA (Condition × Sequence) tested for potential order or carryover effects, revealing no significant interactions ($p > 0.10$). Therefore, data from both sequences were pooled for analysis.

Normality of residuals was examined using the Shapiro–Wilk test, and skewed variables (CK, LDH, IL-6, cortisol) were $\log_{10}$-transformed. When normality could not be achieved, non-parametric Wilcoxon signed-rank tests were applied to Δ-values (post − pre). The direction and magnitude of effects were consistent across transformed and untransformed analyses.

Given the exploratory purpose and limited sample size, no global correction for multiple comparisons was applied; instead, interpretation emphasized practical relevance through effect sizes (ES) (Cohen's *dz*), classified as trivial (0.0–0.2), small (0.2–0.6), moderate (0.6–1.2), large (1.2–2.0), and very large (2.0–4.0) [62]. This approach balances type I and type II error risk in small exploratory samples.

A post hoc sensitivity analysis confirmed that, with $n = 21$, $\alpha = 0.05$, and within-subject correlation $\rho = 0.5$, the study had 95% power to detect a moderate-to-large within-subject effect ($dz \geq 0.65$) in physiological or biochemical outcomes [63].

Descriptive and inferential plots were generated to visualize individual variability and condition-dependent trends. Boxplots and individual trajectories were used to inspect potential order effects. Statistical plots were produced in STATA, and final visual layouts were formatted in Tableau (Salesforce, Seattle, USA).

3. Results

Results of the maximal incremental exercise test under ABST and SACT conditions are shown in Figure 3. Significant within-subject differences were found for test duration ($p = 0.006$; ES = 0.31, small) and HR ($p < 0.001$; ES = 0.73, moderate), both higher in the SACT condition. No significant differences were detected for relative power, maximum power, blood lactate, or RPE ($p > 0.05$ for all; Panels B, C, E, and F).

For handgrip strength, a significant within-subject difference was observed for Hand Grip Mean ($p = 0.027$; ES = 0.22, small), with higher values in the SACT condition. No significant difference was found for Hand Grip Best ($p = 0.994$; ES = 0.00, trivial; Panel G).

Figure 4 shows the results for muscle damage biomarkers. A significant difference was identified for LDH ($p < 0.001$; ES = 0.47, small), which was higher in ABST compared with SACT. No significant differences were found for CK ($p = 0.96$; ES = 0.00, trivial) or Mb ($p = 0.291$; ES = 0.05, trivial). In both conditions, post-exercise values of all markers were higher than baseline (data not shown).

No additional significant differences were observed in other biochemical variables analyzed such as hormonal response (Figure 5) or inflammatory parameters (Figure 6).

4. Discussion

4.1. Main findings

This randomized crossover study aimed to explore whether masturbation-induced orgasm, performed 30 min before maximal exercise, modulates physiological, biochemical, and performance responses in well-trained men. Compared with sexual abstinence, the post-orgasmic condition elicited statistically significant, yet small improvements in total exercise time and mean handgrip strength, without detrimental changes in lactate accumulation, perceived exertion, or

inflammatory markers. A significant increase in HR was observed, along with moderate elevations in testosterone and cortisol and a significant reduction in LDH, with non-significant trends for CK and Mb.

Taken together, these findings suggest that a single post-orgasmic episode does not compromise subsequent exercise performance, nor does it increase physiological stress. Rather, the data indicate a short-lived shift in neuroendocrine tone and autonomic balance consistent with sympathetic arousal followed by partial parasympathetic rebound. These results fill a critical gap in the literature, where evidence from field observations [22–24,28,30,39,52] has been inconsistent, often due to uncontrolled timing, lack of standardization of sexual stimuli, and absence of biochemical verification. By implementing strict crossover control, standardized audiovisual stimuli, and identical time-of-day testing, the present study reduces many of these confounding factors and provides mechanistic insight into a topic long dominated by anecdote and myth.

Nevertheless, the magnitude of observed changes was small and must be interpreted with caution. Performance effects did not exceed trivial-to-small ES, suggesting that any influence of sexual activity on exercise performance operates within narrow physiological margins, likely related to transient arousal modulation rather than ergogenic enhancement.

4.2. Physiological interpretation

Sexual arousal and orgasm are characterized by a rapid activation of the sympathetic nervous system, marked by increases in HR, blood pressure, and catecholamine secretion [39,64,65] This acute adrenergic discharge resembles the transient cardiovascular priming typically observed

before exertion. The significant elevation in HR recorded in our participants during the post-orgasmic condition indirectly supports this interpretation.

From a mechanistic standpoint, this transient sympathetic excitation could facilitate oxygen transport and neuromuscular readiness at exercise onset. However, unlike a voluntary warm-up, sexual arousal is accompanied by an intense neuroendocrine cascade involving dopamine, oxytocin, prolactin, and serotonin [66,67], which may variably modulate attention, motivation, and fatigue perception. While sympathetic activation might prime cardiovascular readiness, excessive arousal could also provoke premature fatigue or attentional disruption through overactivation of the HPA axis.

Therefore, the physiological state following orgasm is best described as a *homeostatic oscillation*: an initial sympathetic surge facilitating alertness and cardiovascular perfusion, followed by a parasympathetic rebound aimed at restoring equilibrium. Studies by Krüger et al. [11] and Exton et al. [17]. demonstrated marked increases in HR and blood pressure during orgasm, followed by a rapid parasympathetic recovery within approximately 10–20 min. In our protocol, exercise began 30 min post-orgasm, likely coinciding with this transitional phase in which residual adrenergic tone remains sufficient to enhance readiness without compromising performance stability.

Still, it must be emphasized that this interpretation is inferential. We did not directly measure HR variability, catecholamines, or sympathetic nerve activity. Consequently, the mechanistic link between sexual activation and exercise performance remains speculative and should be tested through future studies integrating direct autonomic indices and neuroendocrine markers.

4.3. Muscle performance and psychophysiological factors

The small improvement in mean handgrip strength and relative power observed post-orgasm (ES ≤ 0.3, small) could be interpreted through the framework of post-activation potentiation (PAP). PAP describes the transient enhancement of muscle force following a conditioning contraction, mediated by increased phosphorylation of myosin light chains and heightened motor unit excitability [68–70]. Although sexual activity involves rhythmic pelvic and abdominal muscle contractions, the intensity and neural recruitment patterns differ markedly from voluntary resistance contractions. Thus, the "PAP-like" effect described here must be viewed as an analogy, not a physiological equivalence [21]. However, this analogy should be treated cautiously, as electromyography activity and motor unit recruitment were not directly assessed in the present study.

Nevertheless, the sympathetic arousal accompanying sexual activity may induce a neural "priming" effect analogous to PAP by increasing motoneuron excitability and central drive. The 30 min recovery window used in our study appears critical, as it falls within the window where potentiation can dominate over fatigue [71]. Shorter intervals (≤15 min) might result in overactivation and premature fatigue, while longer intervals (>3–6 h) would dissipate any potentiating influence. This temporal specificity could partly explain the heterogeneity in prior findings, where studies using 2–12 h delays often reported neutral or detrimental effects [24,28–30].

It is also essential to consider the psychological component. Orgasm induces transient alterations in neurotransmitters (dopamine, serotonin, endorphins, endocannabinoids) that can affect motivation, confidence, and pain perception [66]. The absence of RPE differences between conditions suggests that these neuromodulators were insufficient to alter conscious effort perception during maximal exertion. Yet, subtle changes in attentional focus, motivation, or self-

efficacy could have contributed to the small performance enhancement. Expectancy bias—participants believing that sexual activity might hinder or help—represents another plausible modulator. Although crossover counterbalancing minimized this effect, complete blinding to condition was inherently impossible given the nature of the stimulus. The use of standardized audiovisual stimuli and blinded data collection minimized but did not eliminate this bias. Future work should incorporate validated psychometric tools to quantify mood, motivation, and perceived readiness.

In sum, while neuromuscular potentiation and psychological readiness both provide plausible explanatory models, their relative contributions remain uncertain. What is clear is that sexual activity 30 min before exercise did not provoke measurable fatigue, suggesting that the recovery interval was sufficient to offset any transient depletion of autonomic or energetic reserves.

4.4. Hormonal and metabolic responses

The concomitant rise in testosterone and cortisol following masturbation supports the notion of a transient neuroendocrine stress response integrating the HPG and HPA axes [36,38–40,42], consistent with the acute sympathetic–adrenal activation typical of sexual and exercise stressors. Sympathetic activation stimulates adrenal and testicular secretion, preparing the organism for acute physical or emotional challenge. The present study's blood samples—collected after exercise—reflect the integrated effect of pre-exercise sexual arousal plus exercise-induced stress, making interpretation complex but ecologically valid.

The moderate increases in both hormones likely represent an acute eustress adaptation rather than a chronic anabolic–catabolic imbalance. Testosterone elevations of this magnitude (ES ≈ 0.5–0.6) are within the range reported after brief high-intensity exercise, while cortisol rises similarly to

those seen in controlled stress paradigms [32,36]. This parallel activation implies enhanced energetic availability (via gluconeogenesis and lipolysis) coupled with heightened motivational drive and central nervous system excitability. Given that both increases were small-to-moderate in magnitude, the concurrent rise of cortisol does not necessarily negate the anabolic potential of testosterone; rather, it reflects coordinated mobilization of substrates for performance demands.

Nevertheless, these effects are transient. Prior work has shown that testosterone and cortisol normalize within 60–120 min post-orgasm [37,40]. Hence, hormonal effects likely acted indirectly—through transient arousal and central drive—rather than via direct anabolic mechanisms. The absence of significant changes in CRP and IL-6 further reinforces that the hormonal perturbation did not extend into inflammatory or recovery-related pathways.

A key limitation, however, is that only total hormone concentrations were measured. Free fractions, which represent bioactive availability, may respond differently. Furthermore, without parallel assessment of sympathetic markers (e.g., epinephrine, norepinephrine), our ability to separate endocrine from autonomic contributions remains limited. Future studies should adopt an integrated neuroendocrine approach combining plasma catecholamines, HR variability metrics, and free hormone indices to better define the nature of this short-term adaptation.

4.5. Muscle damage and inflammatory markers

To our knowledge, this is the first study to evaluate biochemical markers of EIMD following pre-exercise sexual activity. The significant reduction in LDH, with non-significant changes in CK and Mb, indicates no evidence of increased muscle strain. Whether the LDH difference reflects true physiological modulation or normal biological variability remains uncertain.

However, the interpretation of LDH reductions warrants caution. LDH is a non-specific cytosolic enzyme whose plasma concentration depends not only on muscle fiber disruption but also on tissue perfusion, redox status, and plasma volume variation. Although we corrected for %ΔPV using the Van Beaumont method, minor hemodynamic differences between sessions cannot be excluded. Furthermore, LDH isoforms originate from both skeletal and cardiac muscle; and are also influenced by hepatic and erythrocytic metabolism, thus, attributing changes exclusively to skeletal damage is speculative. It is plausible that the mild sympathetic activation before exercise improved muscle perfusion and reduced ischemic transients, leading to lower leakage of cytosolic enzymes. Alternatively, the observed difference may reflect random day-to-day biological variability rather than a causal effect of sexual activity.

The absence of increases in CK and Mb, two well-validated EIMD biomarkers [45,46], further reinforces that the exercise stimulus used—though maximal—did not provoke substantial structural damage. Given that sexual activity itself involves brief isometric and rhythmic contractions, it is unlikely to have induced any pre-existing muscle microtrauma that would confound post-exercise readings. This aligns with reports from Zavorsky et al. [24,27] showing no deleterious impact of pre-competition sex on strength recovery or soreness.

The absence of changes in CRP and IL-6 suggests that systemic inflammation remained unaltered. IL-6 typically rises in proportion to metabolic stress and glycogen depletion [72], and CRP responds to more prolonged or tissue-damaging stimuli. Since both remained stable, the data that pre-exercise sexual activity neither amplified inflammatory cascades nor interfered with recovery. This is consistent with the hypothesis that the transient endocrine and autonomic activation induced by orgasm constitutes a controlled physiological stressor—sufficient to prime

cardiovascular and neuroendocrine systems without crossing the threshold into systemic inflammation.

Nevertheless, several uncertainties remain. First, inflammatory mediators exhibit diurnal variation; although testing was conducted at the same time of day, intra-individual fluctuations may persist. Second, the 30min interval between orgasm and exercise may have coincided with partial parasympathetic recovery, attenuating stress-induced cytokine release. Finally, our biomarker panel was limited to CRP and IL-6; inclusion of IL-1β, tumour necrosis factor α, or oxidative stress markers (Malondialdehyde, Ferric Reducing Ability of Plasma and Total Antioxidant Capacity) would provide a more comprehensive picture of redox–inflammatory crosstalk. Future research combining biochemical, electrophysiological, and autonomic (HR variability, baroreflex) measures could elucidate whether the apparent attenuation of muscle damage reflects true physiological protection, improved perfusion, or a simple statistical artifact.

4.6. Strengths and limitations

The principal strength of this study lies in its methodological rigor and experimental control within an otherwise anecdotal field of research. We employed a randomized crossover design, allowing each participant to serve as his own control and thereby reducing interindividual noise—a key limitation in most prior works. The use of standardized audiovisual stimuli (neutral vs erotic) and strict timing (30 min post-orgasm) eliminated major confounders such as emotional variability, expectancy, and circadian influence. Moreover, the simultaneous assessment of performance, cardiovascular, hormonal, and inflammatory parameters provides a multidimensional understanding of the post-orgasmic physiological state.

A further strength is the novelty of the biochemical approach: no previous study has simultaneously examined LDH, CK, Mb, CRP, and IL-6 within a controlled post-orgasmic context. This allows, for the first time, a quantitative discussion of whether sexual activity modifies redox–inflammatory homeostasis or muscle integrity.

However, several limitations must be acknowledged. The sample size, although statistically justified (power = 0.95; ES = 0.8), remains small for generalization. The cohort was composed exclusively of healthy, well-trained young men (basketball players, volleyball players, long-distance runners, boxers, and judokas); extrapolation to women, sedentary individuals, or older adults is not warranted. Furthermore, although the crossover design strengthens internal validity, its short washout period (1 week) might not fully exclude residual psychological conditioning effects. Moreover, sexual activity was restricted to masturbation-induced orgasm rather than partnered intercourse—a necessary compromise to preserve internal validity but one that limits ecological generalizability.

From a methodological standpoint, psychological constructs such as motivation, aggression, anxiety, or pre-competitive tension were not formally assessed. Given the well-documented psychophysiological interplay between sexual arousal and emotional state [12,64], this represents a missed opportunity to disentangle cognitive from somatic contributors to performance. Additionally, only total hormone concentrations were analyzed, not their free fractions, which would more accurately reflect biological availability. Similarly, autonomic modulation was inferred from HR rather than directly quantified via HR variability or catecholamines, limiting mechanistic precision.

Despite these limitations, the internal validity of the study is strong. The within-subject, counterbalanced design minimizes confounding, and the consistency of directionality across

multiple endpoints strengthens the plausibility of observed effects. In essence, the data are robust enough to justify the central conclusion—that acute sexual activity before exercise does not impair physiological or performance outcomes—while acknowledging the modest scope of inference.

4.7. Practical implications and future directions

From a performance standpoint, the findings have both applied and conceptual relevance. Masturbation-induced orgasm performed approximately 30 min before maximal exercise did not impair any measure of strength, endurance, or metabolic stress and may even confer mild autonomic and psychological readiness benefits. For athletes and coaches, this implies that pre-competition sexual activity, when moderate and followed by adequate recovery, is unlikely to hinder performance—a conclusion that challenges decades of anecdotal abstinence dogma.

However, these results should not be misinterpreted as evidence of an ergogenic strategy. The small magnitude and high interindividual variability of effects, combined with the psychological and ethical complexity of sexual behavior, preclude any recommendation for deliberate pre-competition stimulation. Moreover, ethical and privacy considerations limit the feasibility of systematically prescribing or manipulating sexual activity in applied sport contexts. Moreover, partnered intercourse may elicit distinct neuroendocrine and metabolic profiles—particularly through oxytocin and prolactin pathways—whose implications for recovery and sleep remain unexplored.

Future investigations should therefore adopt a multidimensional and sex-inclusive framework, incorporating both male and female athletes, and extending the observation window to different recovery intervals (e.g., 1 h, 6 h, 12 h post-orgasm). Measurement of HR variability, plasma catecholamines, and free testosterone–cortisol ratio would help delineate the autonomic and

endocrine contributions to performance modulation. Including validated psychological metrics (motivation, mood state, perceived readiness) would also clarify whether sexual activity influences output primarily through central or peripheral pathways.

From a broader perspective, this line of research contributes to a more nuanced understanding of human integrative physiology, where sexual behavior represents not merely a psychological variable but a potent natural modulator of neuroendocrine and autonomic systems. The evidence presented here suggests that, when appropriately timed, sexual activity may form part of a normal pre-competition behavioral repertoire without adverse physiological consequences. This does not refute the traditional emphasis on discipline and focus but rather reframes it within an evidence-based physiological context.

5. Conclusion

This randomized crossover study addressed a critical evidence gap by examining the acute effects of masturbation-induced orgasm performed 30 min before maximal exercise in well-trained men. The results demonstrate that post-orgasmic activation does not impair, and may transiently modulate, selected physiological and performance parameters within normal physiological limits.

Specifically, the post-orgasmic condition elicited small, short-lived increases in HR, testosterone, and cortisol, accompanied by modest improvements in total exercise time and mean handgrip strength, without detrimental alterations in lactate accumulation, perceived exertion, inflammatory biomarkers (CRP, IL-6), or muscle damage indices (CK, LDH, Mb). These findings suggest that acute sexual activity induces a brief neuroendocrine and sympathetic activation that enhances cardiovascular readiness while preserving muscular and metabolic stability.

Overall, this controlled laboratory evidence refutes the traditional belief that pre-competition sexual activity compromises athletic performance, showing instead that, when adequate recovery (~30 min) is allowed, it neither harms nor meaningfully enhances exercise capacity. The physiological response appears to reflect a transient, adaptive homeostatic activation rather than an ergogenic or fatiguing influence.

Highlights

- This crossover study examined the acute effects of masturbation-induced orgasm performed 30 min before maximal exercise in well-trained male athletes.
- Post-orgasmic sexual activity produced small, transient increases in HR, testosterone, and cortisol, without impairing athletic performance.
- Modest improvements were observed in exercise duration and mean handgrip strength compared with abstinence.
- No significant alterations were found in lactate accumulation, inflammation (CRP, IL-6), or muscle damage markers (CK, LDH, Mb).
- These findings indicate that short-interval sexual activity before exercise does not hinder performance and may reflect transient sympathetic–neuroendocrine activation rather than fatigue.

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Disclosure Statement

The authors report there are not competing interest to declare

Data Availability

The original contributions presented in the study are included in the article; further inquiries can be directed to the corresponding author

Consent

All participants have given informed consent to participate in the research

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work the author(s) used ChatGPT. Artificial intelligence tools were not used to generate, analyze, or interpret data. Limited use of AI-based language assistance was applied for English editing under full human supervision, and all content was critically reviewed and approved by the authors. After using this tool/service, the authors reviewed and edited the content as needed and takes full responsibility for the content of the published article.

Author Contributions

D.F.-L.: (corresponding author) served as study coordinator, conceived and designed research, analyzed data, interpreted results of experiments, prepared figures, drafted manuscript and approved final version of manuscript; J.S.-C., E.R., M.G. and J.M.-A.: drafted manuscript and edited and revised manuscript; G.S.: data analysis and interpretation, provided comments on the manuscript, and revised manuscript; J.M.I.V.: analyzed data, prepared figures and revised manuscript. All authors have read and agreed to the published version of the manuscript.

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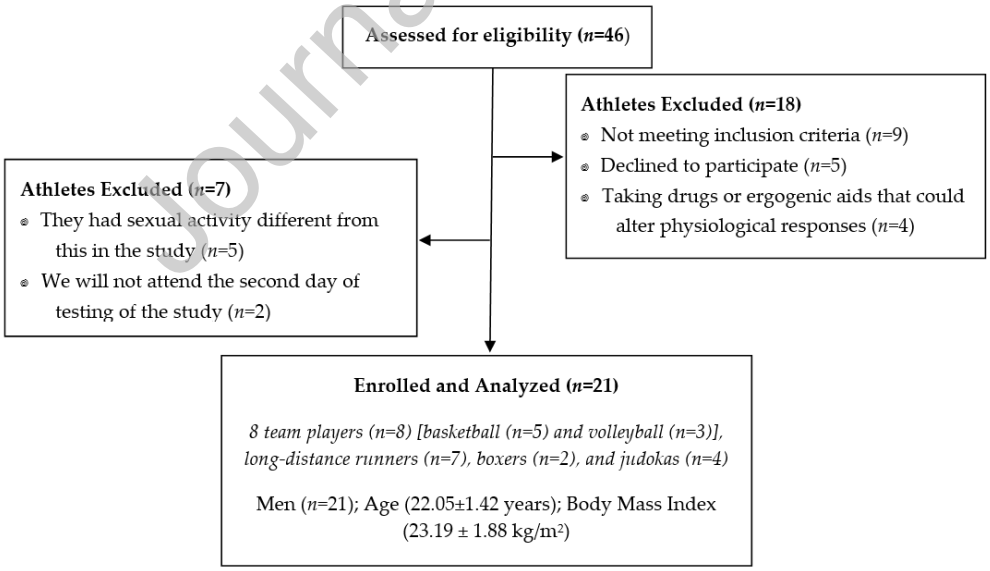

Figure 1. Flow diagram for selection of participants according STROBE criteria.

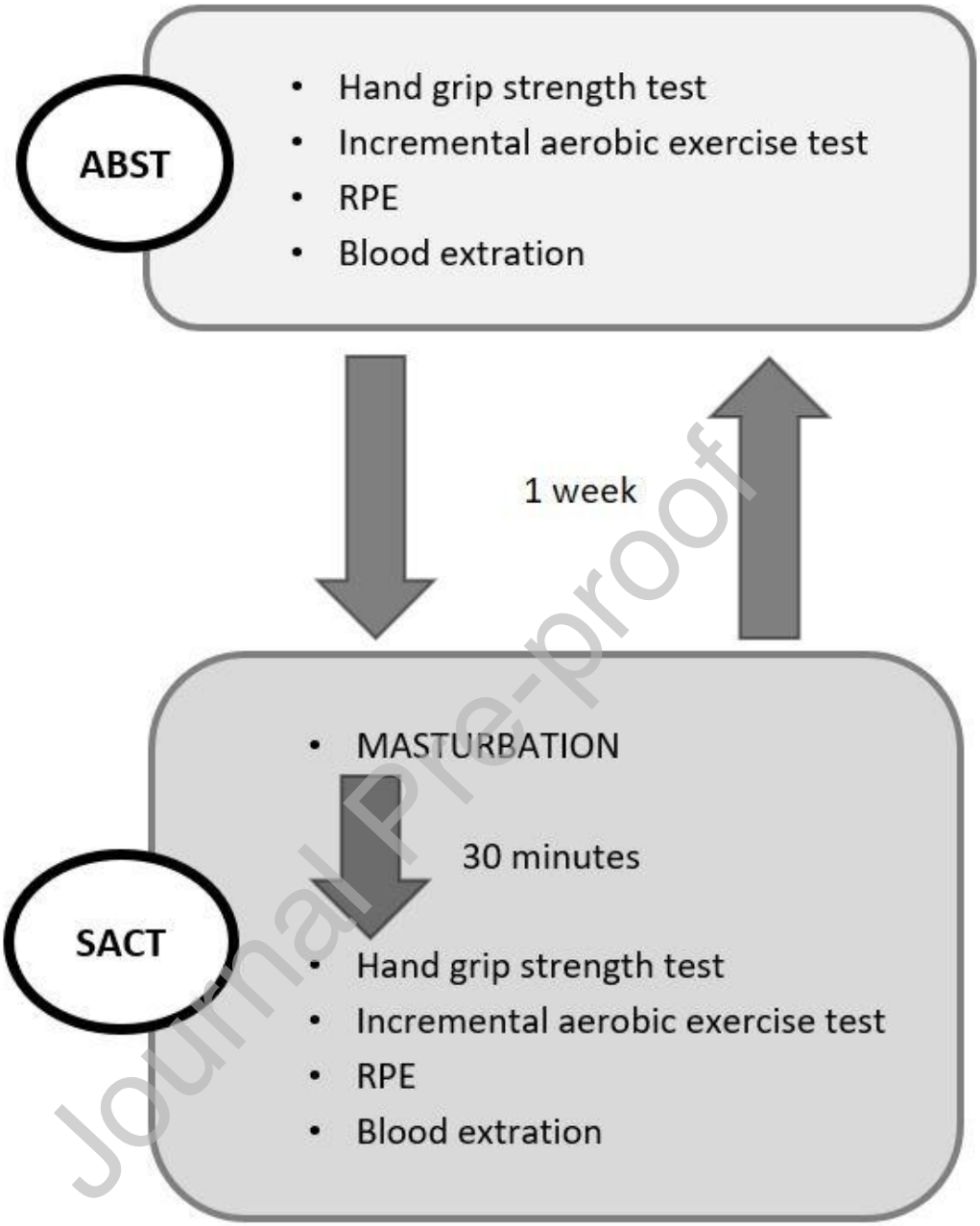

Figure 2. Experimental crossover design. Participants completed two experimental conditions in randomized counterbalanced order: ABST (abstinence condition) — no sexual activity for ≥48 h before testing; and SACT (sexual activity condition) — masturbation-induced orgasm performed 30 min before exercise testing. Each condition included, in sequence: (1) handgrip strength test, (2) incremental aerobic exercise test, (3) rating of perceived exertion (RPE) assessment, and (4) blood extraction for biochemical analyses. A 1-week washout period separated both conditions to avoid carryover effects. ABST: abstinence condition; SACT: sexual activity condition; RPE: rating of perceived exertion.

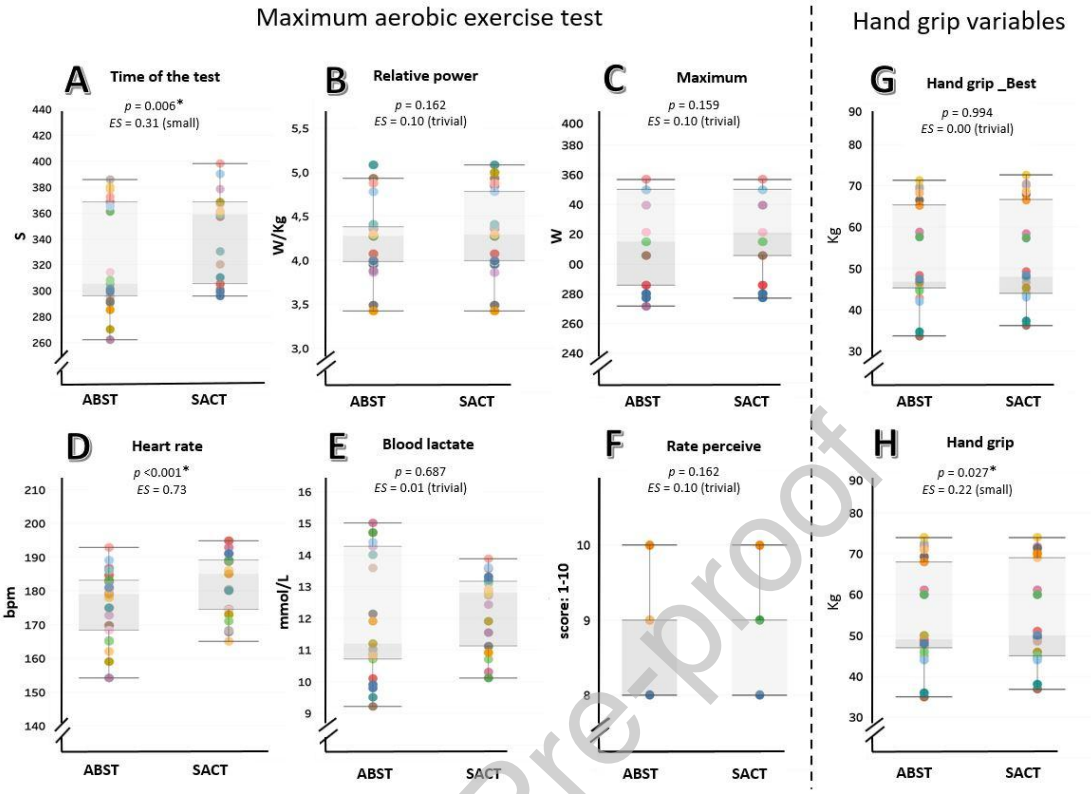

Figure 3. Differences between ABST and SACT conditions in physical performance variables. Panels A–F show maximum aerobic exercise test variables: (A) Time of the test, (B) Relative power, (C) Maximum power, (D) Heart rate, (E) Blood lactate, and (F) Rate of perceived exertion (RPE). Panels G–H show handgrip strength variables: (G) Hand Grip_Best and (H) Hand Grip_Mean.

Data are presented as individual values (colored dots) and group mean ± SD. *p*-values correspond to the Condition effect from the repeated-measures ANOVA (Condition × Time); significant pairwise differences were identified using Bonferroni-adjusted post hoc tests. ES: Cohen's $d_{(z)}$ standardized effect size. *$p < 0.05$ was considered significant. ABST: abstinence condition; SACT: sexual activity condition.

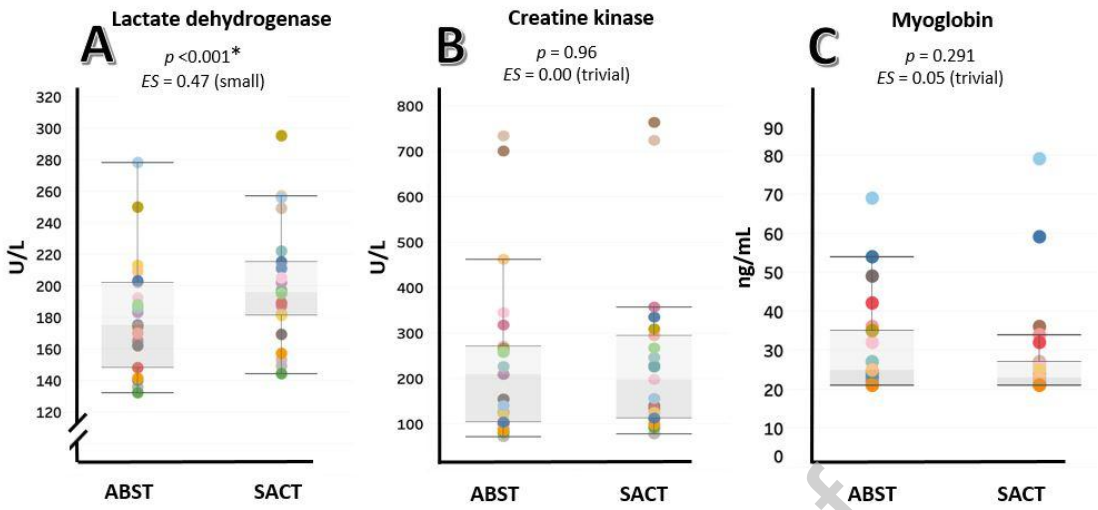

Figure 4. Differences between ABST and SACT conditions in muscle damage biomarkers. (A) Lactate dehydrogenase (LDH), (B) Creatine kinase (CK), and (C) Myoglobin (Mb). Data are shown as individual values (colored dots) and group mean ± SD. *p*-values correspond to the Condition effect from the repeated-measures ANOVA (Condition × Time); significant pairwise contrasts were identified using Bonferroni-adjusted post hoc tests. ES: Cohen's dz standardized effect size. *$p < 0.05$ was considered significant.* ABST: abstinence condition; SACT: sexual activity condition.

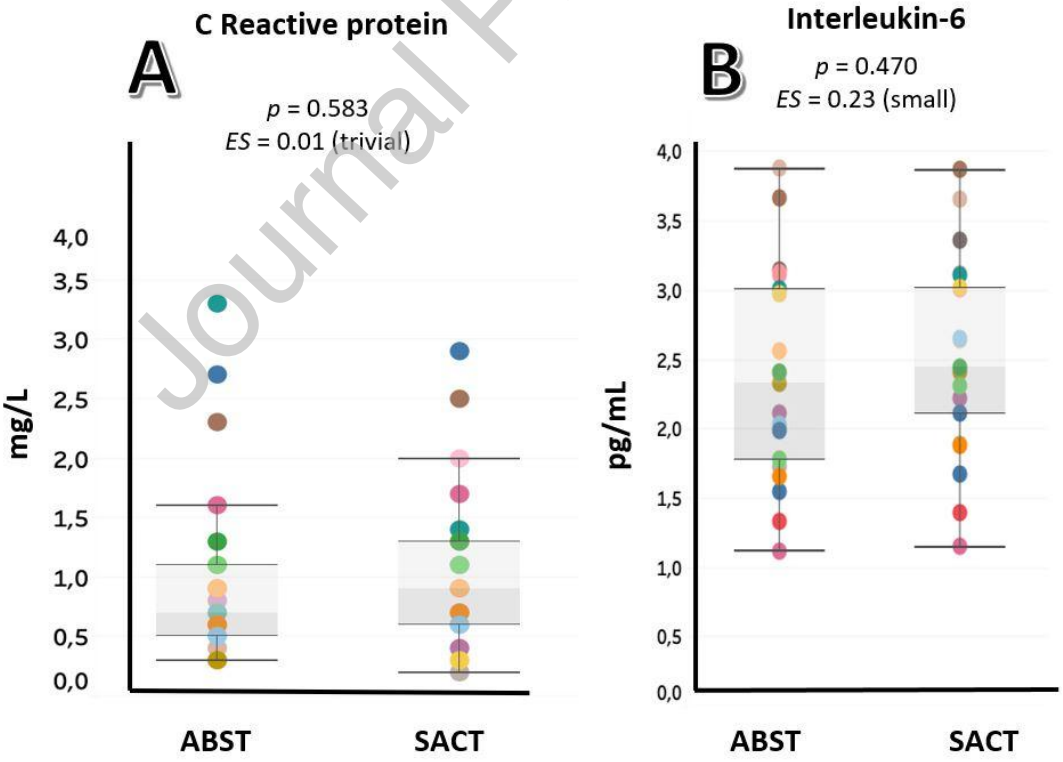

Figure 5. Differences between ABST and SACT on inflammatory response (panels A and B). *p: Differences between groups using Student's t-tests; ES: Cohen's d standardized effect size. *: Significant differences between ABST s SACT p < 0.05.* ABST: abstinence condition; SACT: sexual activity condition.

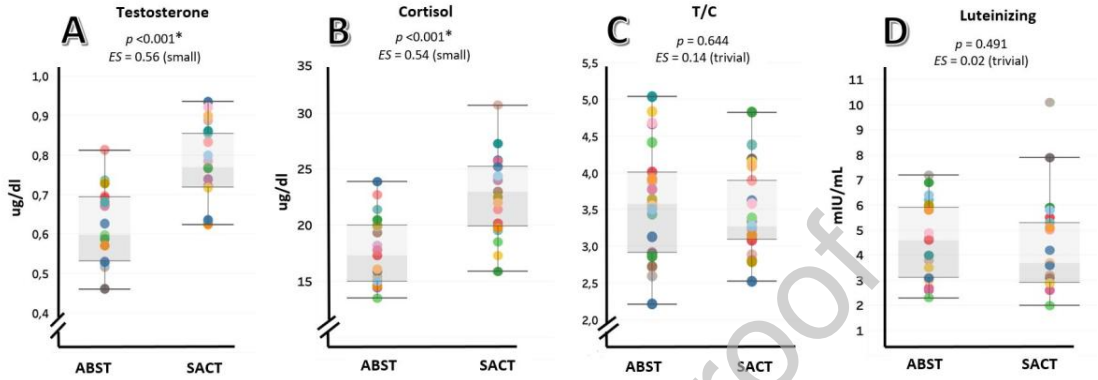

Figure 6. Differences between ABST and SACT on hormone variables (panels A-D). *p: Differences between groups using Student's t-tests; ES: Cohen's d standardized effect size. *: Significant differences between ABST s SACT p < 0.05.* ABST: abstinence condition; SACT: sexual activity condition; T/C: testosterone cortisol ratio.