











SYSTEMATIC REVIEW

Acute and chronic effects of different training protocols on testosterone levels in men: a systematic review

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Abstract

Background: This systematic review examines the physiological basis of age-related testosterone decline and the acute and chronic effects of different exercise protocols on testosterone levels. This study aimed to evaluate the influence of different training modalities on testosterone responses and explore the potential relevance of these findings to age-related hormonal changes, including andropause. **Methods:** A systematic literature search was performed in PubMed, Web of Science, Scopus, and Google Scholar from July to October 2025. Studies were considered for inclusion if they investigated the acute and chronic effects of various exercise protocols on testosterone levels and andropause-related outcomes in humans. Twenty studies met the eligibility criteria after the screening process and were included in the qualitative synthesis. **Results:** This bidirectional effect of exercise on testosterone might play a role in maintaining anabolic–catabolic balance and may be relevant to the physiological processes associated with andropause in older men. Regular, age-appropriate, and capacity-appropriate exercise programs might support testosterone regulation; however, their direct effects on andropause-related symptoms are unclear. While these findings provide important insights into exercise-induced hormonal regulation, most of the included studies were conducted in younger, healthy, or athletic populations. Therefore, the observed responses should primarily be interpreted as mechanistic or physiological evidence rather than direct clinical evidence applicable to men experiencing andropause. **Conclusions:** In conclusion, exercise-based lifestyle interventions could represent a promising supportive strategy for maintaining testosterone homeostasis and mitigating the features associated with age-related decline in testosterone levels. However, since most of the included studies were conducted in younger, athletic, or otherwise non-clinical populations, the current evidence should be interpreted primarily as mechanistic and supportive rather than as direct clinical evidence for men with established andropause. **The PROSPERO Registration:** <https://www.crd.york.ac.uk/PROSPERO/view/CRD420261345492>, CRD420261345492.

Keywords

Andropause; Exercise; Hypogonadism; Male health; Testosterone; Training intensity

1. Introduction

The beneficial effects of exercise on human health are well-established [1–3]. Regular exercise enhances metabolic control, cardiovascular health, musculoskeletal health, and psychological wellbeing [4–9]. Evidence is mounting that exercise induces physiological adaptations of the endocrine system that are required for maintenance of homeostasis and response to environmental stressors [10]. Despite these well-known benefits, physical inactivity is highly prevalent worldwide and associated with accelerated functional decline and increased risk of age-related disorders. Many studies have shown that

long-term and regular exercise extends life span and delays the appearance of cardiovascular and metabolic diseases [8, 11–14].

Andropause, or late-onset hypogonadism (LOH), is one of the most common examples of changes in men's hormones that are associated with aging. Andropause is characterized by a gradual decline in testosterone levels, usually starting after age 40 years of age [15–19]. This decline is gradual rather than abrupt, unlike menopause, and does not always present with clinically specific symptoms. The decrease in testosterone has been associated with decreased muscle mass, decreased libido, fatigue, mood disturbances, and diminished physical

performance [18–23]. Although criteria for diagnosis are still debated, age-related testosterone decline is increasingly recognized as a contributor to functional deterioration in older men [19].

With age, the risk of developing chronic diseases such as diabetes, sarcopenia, cardiovascular disease, osteoporosis, and neurodegenerative diseases increases [11, 24]. Important contributors to this decline are considered to be testosterone deficiency and physical inactivity. Understanding how different exercise modalities influence testosterone levels is important in the context of aging, given the interaction between physical activity and endocrine regulation. While prior research has explored the effects of exercise and testosterone, a comprehensive review systematically comparing the acute and chronic effects of different training protocols, such as high-intensity interval training (HIIT), resistance, aerobic, and sprint interval training, on andropause risk is missing. The purpose of our study was therefore to analyse the acute and chronic effects of different training protocols on testosterone levels and whether the results are relevant to the age-related decline in testosterone levels. In this setting, we investigated the physiological and mechanistic aspects of exercise, without making direct clinical inferences regarding the effects of andropause. Furthermore, this study provides an evidence-based approach for exercise prescription during andropause and directs future studies toward understanding the molecular mechanisms underlying exercise-induced hormonal adaptations. However, it is important to note that much of the evidence in this area comes from younger or athletic populations and is not directly applicable to specific populations with andropause.

2. Methods

The current systematic review aimed to examine the acute and chronic effects of different training protocols on testosterone levels in association with the risk of andropause. Database searching identified a total of 85 records. Following screening of titles and abstracts, 65 records were excluded for irrelevance or failure to meet the eligibility criteria. The remaining 20 full-text articles were assessed for eligibility; all met the inclusion criteria and were incorporated into the qualitative synthesis. The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flowchart of the study is presented in Fig. 1 (See the PRISMA checklist in the **Supplementary material**).

All screening and eligibility assessments were performed independently by two reviewers using predefined inclusion and exclusion criteria. Discrepancies were discussed in consensus meetings after each screening stage, and unresolved disagreements were resolved by a third reviewer. Although a formal inter-rater reliability coefficient was not calculated, a structured dual-review process and consensus-based resolution were implemented to enhance methodological reproducibility. The methodological approach was developed in accordance with the framework of the Joanna Briggs Institute (JBI) [25].

2.1 Literature review strategy

PubMed, Web of Science, Google Scholar, and Scopus databases were searched between July 2025 and October 2025. The search process was conducted systematically using predefined keywords and their relevant combinations to ensure comprehensive coverage of the literature. Searches were limited to studies published in English and were all conducted in humans (Table 1, Ref. [4, 9, 10, 13, 26–41]).

Detailed search strategies and additional supporting information are provided in the **Supplementary material**.

While some studies included both male and female participants, this review focuses on andropause in men; therefore, only data from men were used.

The following keywords and combinations were used in the search process:

“exercise and testosterone”, “resistance training and testosterone”, “endurance training and testosterone”, “high-intensity interval training and testosterone”, “training protocols and testosterone”, “acute testosterone effect”, “chronic testosterone effect”, “andropause”, “hypogonadism”, “andropause and exercise”, and “hypogonadism and exercise”. An example of the search syntax applied in PubMed is: “testosterone” AND (“exercise” OR “resistance training” OR “endurance training” OR “HIIT”) AND (“andropause” OR “hypogonadism”). The same keywords, Boolean operators, and search combinations were applied consistently across all databases (PubMed, Web of Science, Scopus, and Google Scholar) during the literature search process. Searches were performed in the title, abstract, and keyword fields when possible. The last search was repeated on 31 October 2025, before final synthesis to ensure that the database search was up to date. Duplicate detection was performed by manually cross-checking author names, publication years, and article titles across databases.

2.2 Risk of bias and study quality assessment

The methodological quality of the included studies was assessed using an author-adapted risk-of-bias framework informed by the JBI checklist for quasi-experimental studies and tailored to the methodological characteristics of exercise intervention research [25]. Five key methodological domains were evaluated: (1) group comparability, (2) validity of testosterone measurement, (3) control of major confounding factors, (4) completeness of outcome reporting, and (5) clarity of intervention procedures. Each domain was scored as 1 (yes) or 0 (no or uncertain). Based on the total score, studies that scored 4–5 were classified as low-risk, 3 as medium-risk, and 0–2 as high-risk (Table 2, Ref. [4, 9, 10, 13, 25–41]). Because the included studies comprised acute crossover experiments, chronic training interventions, pre–post quasi-experimental trials, and mixed repeated-measures designs, the direct application of the fully revised JBI quasi-experimental checklist was considered methodologically suboptimal. Therefore, an author-adapted framework was developed by mapping the most exercise-relevant JBI domains onto five harmonized methodological dimensions. Specifically, group comparability reflects baseline equivalence or within-subject

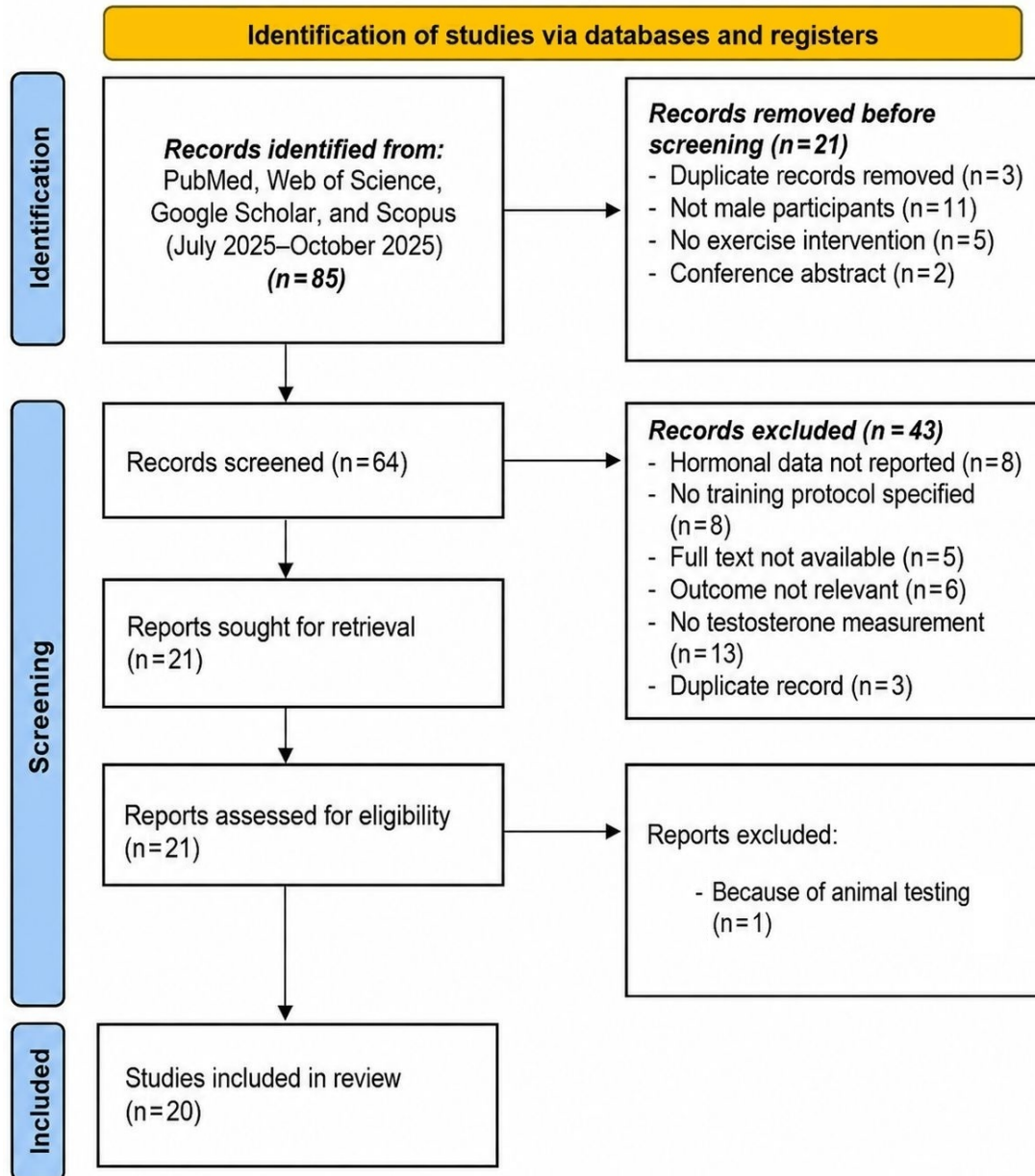


FIGURE 1. PRISMA research flow diagram.

temporal consistency; testosterone measurement validity corresponds to the JBI outcome measurement domain; confounder control integrates key endocrine modifiers, such as circadian timing, fasting, sleep, and prior exercise; outcome completeness reflects follow-up and reporting integrity; and intervention clarity corresponds to the reproducibility of the intervention protocol. This simplification was intentionally designed to improve comparability across heterogeneous exercise designs rather than to replace formal, study-specific appraisal tools. Therefore, the high proportion of studies categorized as low-risk should be interpreted as indicating generally adequate reporting of core exercise-endocrine methods rather than uniformly high methodological rigor across all epidemiological domains.

In this study, we included two studies despite their moderate risk scores. First, Kuusi *et al.* [36] (1984) was one of the first studies relevant to our research topic, and excluding such an early and highly cited study could lead to systematic bias;

therefore, we included the study. Second, Moravej *et al.* [38] (2023) was included because it provides data on a rare adolescent sample and reported findings consistent with other low-risk studies.

2.3 Study selection criteria

Database searches were completed, and duplicated records were removed. Then, titles and abstracts were checked for relevance. Full-text articles were reviewed to see if they met the set inclusion and exclusion criteria. Studies were included if they involved human male participants, examined exercise or training interventions, and reported results related to testosterone levels. Studies using animal models, case reports, or those without hormonal outcome measures were excluded.

TABLE 1. Main characteristics of the studies included in the systematic review.

Study reference	Title	N	Age (mean \pm SD)	Fitness level	Exercise modality (effect type)	Training protocol	Timing of sample collection (sample type)	Testosterone response
Ahmadi <i>et al.</i> [4], 2018 (Indexed in ESCI)	Testosterone and cortisol response to acute intermittent and continuous aerobic exercise in sedentary men	11	22.3 (1.9)	Non-trained	Acute intermittent exercise, running (acute)	1-week, two sessions per week, 40 min per session	Pre-post exercise (serum)	IE (+), CE (//)
Zar <i>et al.</i> [9], 2021 (Indexed in Scopus)	Effects of morning and afternoon high-intensity interval training (HIIT) on testosterone, cortisol and testosterone/cortisol ratio response in active men	11	19.8 (1.0)	Active	HIIT, running (acute)	Two trials (7-day interval), each consisting of 5 \times 40 m sprints with 30 s rest	Pre-post (morning and afternoon)/serum	Morning (//), afternoon (+)
Purnomo <i>et al.</i> [10], 2023 (Indexed in ESCI)	Acute and adaptation effect of high-intensity interval training on testosterone, cortisol and performance among collegiate running athletes	20	20.2 (0.7)	Athletes	HIIT, running (acute and chronic)	6-week, three sessions per week (sets lasting approximately 1 hour)	Pre-post exercise (serum and saliva)	After each set (acute, +), after 6 weeks (//)
Velasco-Orjuela <i>et al.</i> [13], 2018 (Indexed in SCIE)	Acute effects of high-intensity interval, resistance or combined exercise protocols on testosterone-cortisol responses in inactive overweight individuals	51	23.6 (3.5)	Physically inactive, overweight adults	HIIT with resistance training (RT) (acute)	HIIT (4 \times 4 min high-intensity intervals with 4 min active recovery), resistance training (strength training, 12–15 repetitions per set with 60 s rest), HIIT with resistance training (combined protocol)	Pre- and 1-min post-exercise/serum	HIIT (//), resistance training (//), HIIT with resistance training (//)
Williams <i>et al.</i> , 2018 [26] (Indexed in SCIE)	The effect of lower limb occlusion on recovery following sprint exercise in academy rugby players	24	21.8 (3.0)	Athletes	Repeated sprint training, running (acute)	4–5 days a week, 1–2 sessions per day during preseason	Pre-post exercise (saliva)	+
Vieira <i>et al.</i> [27], 2018 (Indexed in SCIE)	Concurrent training protocol for men with androgen deficiency in the aging male: a randomized clinical trial	58	Between 40–59 years old	Healthy Subjects	Aerobic and resistance training (chronic)	3 times/week for 6 months	Baseline, 1st month, 3rd month, post-intervention/serum	+
Zurek <i>et al.</i> [28], 2022 (Indexed in SCIE)	Effects of Dominance and Sprint Interval Exercise on Testosterone and Cortisol Levels in Strength-, Endurance-, and Non-Training Men	96	21.3 (1.8)	Active	Sprint interval training, running (acute)	5 \times 10 s all-out bouts with a 50 s active recovery	Unstimulated (at rest): 10 min before and 12 min after exercise/saliva	//

TABLE 1. Continued.

Study reference	Title	N	Age (mean ± SD)	Fitness level	Exercise modality (effect type)	Training protocol	Timing of sample collection (sample type)	Testosterone response
Adebero <i>et al.</i> [29], 2019 (Indexed in SCIE)	Salivary and serum concentrations of cortisol and testosterone at rest and in response to intense exercise in boys versus men	30	14.3 (1.9)/21.7 (3.1)	Athletes	HIIT, swimming (acute)	Swim-bench maximal strength task and an all-out 200 m swim, high-intensity interval swimming (5 × 100 m, 5 × 50 m, and 5 × 25 m)	Pre-exercise +15 min post-exercise (saliva and serum)	Serum (–), saliva (//)
Ambroży <i>et al.</i> [30], 2021 (Indexed in SCIE)	The effect of high-intensity interval training periods on morning serum testosterone and cortisol levels and physical fitness in men aged 35–40 years	30	37.7 (1.9)	Active	HIIT, strength and endurance training (chronic)	8-week, three sessions per week, 60 min per session	Pre–post exercise (serum)	+
Chasland <i>et al.</i> [31], 2021 (Indexed in SCIE)	Testosterone and exercise: effects on fitness, body composition, and strength in middle-to-older-aged men with low-normal serum testosterone levels	80	Between 50–70 years old	Non-active	A circuit of eight resistance (Cybex) and eight cycling (Monark) stations performed for 45 s each, with 15 s transition intervals (chronic)	12-week randomized, placebo-controlled trial	Pre–post exercise (serum)	+
Cobo <i>et al.</i> [32], 2017 (Indexed in SCIE)	Hypogonadism associated with muscle atrophy, physical inactivity, and ESA hyporesponsiveness in men undergoing hemodialysis	57	65 (49–80)	Hypogonadal patients	Physical activity, walking with a Geonaute-onstep-400® pedometer (acute)	6 consecutive days (two HD days, two HD-free midweek days, and two HD-free weekend days)	Pre–post exercise (plasma)	+
Cofré-Bolados <i>et al.</i> [33], 2019 (Indexed in SCIE)	Testosterone and cortisol responses to HIIT and continuous aerobic exercise in active young men	13	20.2 (2.1)	Active	HIIT, running (acute)	Once per week, 20 min per session	Pre-exercise, post-exercise, and 12-hour post-exercise (plasma)	Pre–post exercise (+), 12-hour post training (–)
Crewther <i>et al.</i> [34], 2017 (Indexed in SCIE)	Is salivary cortisol moderating the relationship between salivary testosterone and hand-grip strength in healthy men?	65/15	22.6 (4.9)	Active	Repeated sprint training, cycling (acute)	1-week, three sessions per week (sets lasting approximately 1 hour)	–5 min pre-exercise and +15 min post-test (saliva)	+
Hawkins <i>et al.</i> [35], 2008 (Indexed in SCIE)	Effect of exercise on serum sex hormones in men: a 12-month randomized clinical trial	102	56.2 (6.7)	Non-active	Moderate intensity aerobic exercise, running and cycling (chronic)	Aerobic physical exercise intervention (60 min per day for 6 days per week)	Baseline, 3, and 12 months (serum)	//

TABLE 1. Continued.

Study reference	Title	N	Age (mean \pm SD)	Fitness level	Exercise modality (effect type)	Training protocol	Timing of sample collection (sample type)	Testosterone response
Kuusi <i>et al.</i> [36], 1984 (Indexed in SCIE)	Acute effects of marathon running on levels of serum lipoproteins and androgenic hormones in healthy males	20	33.9 (2.7)	Athletes	Marathon running (acute)	42.2 km marathon run	Pre–post exercise (serum)	–
Mangine <i>et al.</i> [37], 2018 (Indexed in ESCI)	Testosterone and cortisol responses to five high-intensity functional training competition workouts in recreationally active adults	5 male, 5 female	Male: 34.4 (3.8), Female: 35.5 (7)	Active	High-intensity functional training, mixed training (chronic)	5 weeks, utilizes aerobic, gymnastic, and weightlifting exercises	Pre-exercise, +30 min, and +60 min post-exercise (saliva)	Male (+), female (+)
Moravej <i>et al.</i> [38], 2023 (Indexed in DOAJ)	The effect of high-intensity strength and endurance training on cortisol, testosterone, and physical fitness of 15–20-year-old male taekwondo athletes in Rasht	20	17.7 (2.3)	Athletes	High-intensity strength and endurance training, mixed training (chronic)	8 weeks, endurance training (<i>e.g.</i> , running and jumping rope), resistance training (<i>e.g.</i> , chest press, squats, front and back lats, front lunges, front arm, back arm, and back thigh)	Pre–post exercise (serum)	+
Schwanbeck <i>et al.</i> [39], 2020 (Indexed in SCIE)	Effects of Training with Free Weights Versus Machines on Muscle Mass, Strength, Free Testosterone, and Free Cortisol Levels	15 male, 16 female	Male: 22 (3), female: 23 (4)	Healthy Subjects	Free weights or machines for 8 weeks (chronic)	With each muscle group trained 2–3 times per week, 3–4 sets of 4–10 repetitions	Pre–post exercise (saliva)	Male (+), female (//)
Sellami <i>et al.</i> [40], 2018 (Indexed in SCIE)	The effect of acute and chronic exercise on steroid hormone fluctuations in young and middle-aged men	32	21 (1)/41 (3)	Trained	Repeated sprint training, cycling (acute and chronic)	13 weeks, combined sprint and resistance training	Pre-exercise, post-exercise, and 10 min post-exercise (serum)	Young (+), middle-aged (+)
Smith <i>et al.</i> [41], 2013 (Indexed in SCIE)	Dihydrotestosterone is elevated following sprint exercise in healthy young men	14	28.3 (4.1)	Active	Repeated sprint training, cycling (acute)	40 min cycling (10 \times 30 s)	Pre-exercise, +5 min, and +60 min post-exercise (serum)	+

- SD: Standard Deviation; HIIT: high-intensity interval training; RT: Resistance Training; IE: Intermittent Exercise; CE: Continuous Exercise; HD: Hemodialysis; SCIE: Science Citation Index Expanded; ESCI: Emerging Sources Citation Index; DOAJ: Directory of Open Access Journals; ESA: Erythropoietin-Stimulating Agents.

- Serum/Plasma Testosterone Studies ($n = 13$), Salivary Testosterone Studies ($n = 5$), Combined Sampling Studies ($n = 2$). Symbols: +: increase; -: decrease; //: no change.

- Serum and plasma measurements reflect total testosterone concentrations, whereas salivary testosterone represents free testosterone levels. Studies were categorized according to sampling matrix and temporal nature of exercise exposure (acute vs. chronic) to improve methodological clarity.

TABLE 2. Risk of bias assessment of included studies.

Study reference	Group comparability ¹	Testosterone measurement validity ²	Confounder control ³	Outcome completeness ⁴	Intervention clarity ⁵	Score	Overall risk
Ahmadi <i>et al.</i> [4], 2018	1	1	1	1	1	5	Low
Zar <i>et al.</i> [9], 2021	1	1	1	1	1	5	Low
Purnomo <i>et al.</i> [10], 2023	0	1	1	1	1	4	Low
Velasco-Orjuela <i>et al.</i> [13], 2018	1	1	1	1	1	5	Low
Williams <i>et al.</i> [26], 2018	1	1	1	1	1	5	Low
Vieira <i>et al.</i> [27], 2018	1	1	1	1	1	5	Low
Zurek <i>et al.</i> [28], 2022	1	1	1	1	1	5	Low
Adebero <i>et al.</i> [29], 2019	1	1	1	1	1	5	Low
Ambroży <i>et al.</i> [30], 2021	1	1	0	1	1	4	Low
Chasland <i>et al.</i> [31], 2021	1	1	1	1	1	5	Low
Cobo <i>et al.</i> [32], 2017	0	1	1	1	1	4	Low
Cofré-Bolados <i>et al.</i> [33], 2019	1	1	0	1	1	4	Low
Crewther <i>et al.</i> [34], 2017	1	1	0	1	1	4	Low
Hawkins <i>et al.</i> [35], 2008	1	1	1	1	1	5	Low
Kuusi <i>et al.</i> [36], 1984	0	1	0	1	1	3	Moderate
Mangine <i>et al.</i> [37], 2018	1	1	0	1	1	4	Low
Moravej <i>et al.</i> [38], 2023	0	1	0	1	1	3	Moderate
Schwanbeck <i>et al.</i> [39], 2020	1	1	1	1	1	5	Low
Sellami <i>et al.</i> [40], 2018	1	1	1	1	1	5	Low
Smith <i>et al.</i> [41], 2013	1	1	1	1	1	5	Low

¹Group comparability refers to the appropriateness of group structure, including control conditions, baseline similarity, or pre–post design consistency.

²Measurement validity refers to the use of clearly described serum, plasma, or salivary testosterone assessment methods with standardized timing.

³Confounder control includes consideration of major factors affecting testosterone response such as age, circadian timing, fasting status, sleep, prior exercise, and training status.

⁴Outcome completeness refers to complete reporting of testosterone values and statistical outcomes.

⁵Intervention clarity refers to the clear description of exercise modality, duration, intensity, and frequency.

Each domain was scored as 1 (yes) or 0 (no/unclear). Total scores of 4–5 were classified as low risk, 3 as moderate risk, and 0–2 as high risk of bias.

Adapted from the JBI revised critical appraisal framework for quasi-experimental studies [25].

Importantly, the predominance of low-risk ratings may partly reflect the fact that most studies were tightly controlled laboratory-based exercise experiments with standardized intervention delivery and clearly timed hormonal sampling, which inherently score favorably in domains related to intervention clarity and outcome measurement. As summarized in Table 2, most of the included studies achieved scores of 4 or 5, indicating an overall low risk of bias in the studies. Studies with a score of 3 were classified as moderate risk, most commonly due to limited control of confounding variables in acute exercise settings or the absence of formal group randomization. No study was categorized as having a high risk of bias.

2.3.1 Inclusion criteria

- We included studies involving male participants in adolescent, young adult, middle-aged, and older age groups. Since the research aimed to assess testosterone responses to exercise throughout the lifecycle of men and investigate their relationship with age-related testosterone decline, studies involving adolescent male athletes that provide mechanistic information on acute endocrine responses were also included.

- Experimental designs examining acute and/or chronic training interventions.

- Research evaluating circulating testosterone levels (*i.e.*, serum, plasma, or saliva) measured using validated biochemical methods.

- Original articles published in peer-reviewed academic journals.

2.3.2 Exclusion criteria

- Observational studies and case reports.

- Publications with inadequate testosterone measurement reporting.

- Studies with insufficient methodological information or that addressed irrelevant topics.

- Short reports and abstracts.

2.4 Data evaluation process

All selected articles were examined for content, methodological quality, and outcome themes. The findings were evaluated using a thematic analysis approach and synthesized under the headings of physiological basis of testosterone, effects of exercise on the endocrine system, hormonal responses according to exercise types, and clinical implications during andropause. Data were independently collected by two reviewers using a pre-designed extraction template that documented study characteristics, participant demographics, intervention protocols, duration, testosterone assessment methods, and primary outcomes. The obtained data were qualitatively integrated, and findings from the literature were interpreted comparatively. This study did not rely on primary data collection and included only an analysis of existing literature.

3. Results

For the purposes of this study, the relevant literature is systematically summarized and synthesized below.

3.1 Physiological foundations of andropause

Andropause is an important biological transitional period that individuals encounter in their lifespan, and it is often surrounded by misunderstandings and social stigmas. This process, which marks the end of the reproductive period, is considered a universal biological transition in human life [33, 42].

The aging process is characterized by numerous physiological changes, including a decrease in anabolic hormones, an increase in catabolic activity, loss of muscle strength, decreased functional performance, and cognitive decline. Exercise stands out as an effective strategy for reducing age-related hormonal imbalances [22, 43, 44]. Physical exercise plays a crucial role

in maintaining and restoring endocrine system functions that decline with aging. Exercise not only supports the overall activity of the endocrine system but also enhances the local effects of sex steroid hormones in tissues. These local effects can be more decisive than circulating hormone levels in maintaining skeletal muscle function [45].

In men, sex steroid hormones are primarily secreted by the testes and adrenal glands. Andropause is characterized by a gradual decrease in total and bioavailable testosterone levels due to a decrease in the number of Leydig cells and a loss of functional capacity. Age-related decreases in episodic and stimulated release of gonadotropins are also observed. In healthy men aged 25–75 years, a decrease of approximately 30–35% in total circulating testosterone levels is observed, while free testosterone concentration decreases by approximately 50% during the same period due to an increase in sex hormone-binding globulin (SHBG) levels. In parallel, aging causes a decrease in both testosterone levels and estradiol concentrations [19, 22, 45]. The rate-limiting step in testosterone synthesis is regulated by the Steroidogenic Acute Regulatory (StAR) protein, which facilitates the transport of cholesterol across the mitochondrial membrane to initiate steroidogenesis. Impaired expression or function of StAR can lead to reduced testosterone production [46].

Men experience psychological problems at different times of their lives. Often these problems are aggravated during andropause. For example, sexual function is optimal between 15 and 30 years of age when testosterone levels are at their maximum. However, these levels decrease significantly with age. Testosterone levels in young men are usually above 1000 ng/dL, but this value declines to an average of approximately 200 ng/dL in men aged 80 years. This can contribute to a reduction in sexual desire of about 80% during andropause [43].

Andropause management is a holistic approach including lifestyle changes and medical treatments when necessary. Aging along with a sedentary lifestyle, are two major factors that can have a profound effect on men's health, and they can reinforce each other. Sedentary behaviour is both a cause and a consequence of declining testosterone levels. The best way to break this vicious cycle is regular and conscious physical activity. Exercise is a safe and effective anti-aging strategy based on evidence, and it plays an important role in improving quality of life and healthy lifespan [11, 18].

One of the major challenges in the assessment of testosterone deficiency in young men is the lack of consensus on what is a normal testosterone level. The American Urological Association recommends using a lower limit of 300 ng/dL to define low testosterone. However, this threshold has limited validity for young men, as it is mainly based on studies performed in men older than 45 years. Testosterone levels also naturally decline with age in men. There are differences in the literature and among professional organizations regarding this threshold value: the American Society of Clinical Endocrinologists (200 ng/dL), the Endocrine Society (264 ng/dL), the British Society for Sexual Medicine (345 ng/dL), the European Urological Society (345 ng/dL), the International Society for Sexual Medicine (350 ng/dL), and the International Society for the Study of Aging Men (350 ng/dL) all report different

threshold values [47].

Testosterone plays a fundamental role in the regulation of metabolism, reproductive functions, and mental state. Marked decreases in blood testosterone levels (<7.0 nM) define the condition of hypogonadism, which leads to both somatic and psychological health problems [30, 47, 48]. Pathologies related to the testes, pituitary gland, hypothalamus, genetic factors, or the aging process are among the main causes of male hypogonadism [43].

Hypogonadism is a potentially reversible syndrome characterized by low testosterone levels without an organic pathology in the hypothalamus–pituitary–gonadal axis. Hypogonadism most commonly appears as LOH in middle-aged and older men. However, factors such as obesity, type 2 diabetes, opioid use, and anabolic steroid abuse can also contribute to this condition [19, 21, 49].

LOH describes symptomatic testosterone deficiency that develops with aging and occurs without an organic disorder in the hypothalamus–pituitary–testis axis. LOH incorporates elements of primary and secondary hypogonadism due to a decrease in the number of Leydig cells and their response to luteinizing hormone (LH). While a compensatory increase in LH levels keeps testosterone within the normal range in some individuals (*i.e.*, compensated hypogonadism), primary hypogonadism can develop if Leydig cell reserves are depleted [20].

Aging, obesity, a sedentary lifestyle, and chronic diseases also negatively affect the hypothalamus–pituitary–gonadal axis, accelerating testosterone decline. Therefore, cases that can be corrected with lifestyle changes and management of comorbid conditions are defined as functional hypogonadism [20]. The first-line treatment for the management of hypogonadism is lifestyle modification. However, if these interventions do not adequately increase testosterone levels within a certain time, hormone replacement therapy (HRT) and other pharmacological options can be considered. HRT is considered an effective treatment method for restoring testosterone levels in appropriate indications [11, 20, 49, 50].

3.2 The role of testosterone and changes with age

Aging leads to a decline in fundamental physical skills, such as strength, power, balance, and flexibility, along with negative changes in body structure [50–52]. This process is closely related to disruptions in the dynamic regulation of the hypothalamus–pituitary–testis axis [50]. However, it has been reported that regular aerobic exercise can reduce or even partially reverse these endocrine changes [8, 45, 53, 54]. Changes in physical functions and the maintenance of hormonal balance are among the most important determinants of health status and functional capacity in older individuals [54, 55].

As human life progresses, major physical and physiological transformations occur in the organism. Cellular-level changes in organs responsible for the synthesis of metabolic regulators and target tissues lead to a decrease in the maximum strength, mass, and endurance of skeletal muscles. This process can be monitored using various biomarkers that cause biological age to differ from chronological age. Among these markers,

testosterone, a steroid hormone belonging to the androgen group, stands out as one of the most crucial indicators of biological aging [30, 40, 43, 56].

Testosterone is a key hormone produced by Leydig cells in the testes and plays a crucial role in skeletal muscle development, nervous system function, protein synthesis, and general anabolic processes [4, 9, 28, 33]. While the testes are the primary production site in men, the adrenal glands also contribute to testosterone production to a lesser extent in both sexes [43]. In men, testosterone levels gradually decrease from age 30–40 years, and this decrease becomes more pronounced in later life during andropause (or male menopause) [11, 30, 57, 58]. The decrease in testosterone levels with age leads to substantial changes in biological, cognitive, and psychological function [59]. Loss of libido, mood disorders, low energy levels, decreased muscle mass, and a deterioration in overall quality of life are frequently observed during this period. Testosterone levels decrease every year, and the serum testosterone levels are below the normal range in about 20% of men at the age of 60 years and in more than half of men older than 80 years [16, 18, 47, 60, 61]. This reduction is closely related to aging and lifestyle and environmental factors such as obesity, lack of physical activity, diabetes, stress, anxiety, burnout, smoking, alcohol use, and inflammatory processes. For example, testosterone levels in older men with untreated diabetes have been reported to be approximately 15% lower than those of men without diabetes [11, 52, 59, 62].

Blood levels of testosterone are regulated by a complex feedback loop between the hypothalamus, anterior pituitary gland, and the testes. The hypothalamus releases gonadotropin-releasing hormone (GnRH), which stimulates LH release. In turn, LH stimulates the Leydig cells in the testes to produce testosterone. At the same time, follicle-stimulating hormone, secreted from the anterior pituitary, has a direct effect on spermatogenesis [18, 22, 43, 48]. The number of Leydig cells decreases, as does their sensitivity to LH, with aging. In addition, the hypothalamus secretes less GnRH, which disrupts the normal daily rhythm of testosterone secretion. Furthermore, levels of SHBG increase with age, and this reduces free circulating testosterone, limiting its bioavailability in tissues [11, 22, 43, 52].

Testosterone is an important hormone for sexual function as well as for overall metabolic balance. Testosterone promotes muscle tissue development by supporting protein synthesis; stimulates erythropoiesis in the bone marrow; and regulates bone formation, lipid and carbohydrate metabolism, liver function, and prostate gland enlargement. Therefore, while low testosterone levels might not affect all target organs equally, they have multifaceted consequences at the systemic level [10, 38, 43].

Testosterone deficiency triggers multifaceted health problems by causing abnormalities in body composition [11, 63–65]. Low testosterone levels are associated with a wide range of symptoms in cardiovascular (*e.g.*, hypertension and ischemia), metabolic (*e.g.*, insulin resistance and diabetes), psychological (*e.g.*, depression, fatigue, difficulty concentrating, and anger), physical (*e.g.*, muscle and bone loss), and sexual (*e.g.*, decreased libido and erectile

dysfunction) areas. Increased visceral fat accumulation and decreased muscle mass (*i.e.*, sarcopenia) are particularly prominent features of reduced testosterone. Increased visceral fat increases the risk of metabolic syndrome and cardiovascular disease, while sarcopenia leads to loss of physical performance and limits mobility. Furthermore, testosterone deficiency reduces bone mineral density, increasing the risk of osteoporotic fractures. All these effects increase the individual's fragility and significantly raise morbidity and mortality rates [8, 20, 23, 63, 66].

Participation in physical activity generally decreases with age; however, regular exercise has pronounced positive effects on physiological systems in older individuals. Exercise can improve overall health and quality of life by slowing down age-related hormonal and metabolic decline. Exercise also contributes to the prevention of chronic diseases and disabilities and plays an important role in maintaining the functional integrity of the endocrine system [8, 45, 67]. In this context, strategies to reduce the negative effects of aging include lifestyle changes, medical treatments, and biotechnological approaches. However, among these, the safest, most accessible, and most effective method to reduce the effects of aging involves lifestyle modifications based on physical exercise [11, 67, 68].

3.3 Acute and chronic effects of exercise types on testosterone release

Aging causes a major decrease in physical function and hormonal balance, consequently leading to a decline in an individual's strength, endurance, balance, and overall performance, whereas exercise can reverse these negative effects [17, 55, 58, 69]. Studies show that intense exercise increases circulating hormone concentrations in both young individuals and adult athletes. In particular, HIIT sessions temporarily alter the homeostasis of the endocrine system by increasing both catabolic (*e.g.*, cortisol, adrenaline, and glucagon) and anabolic (*e.g.*, testosterone, growth hormone, and Insulin-like Growth Factor 1 (IGF-1)) hormone levels. These hormones play key roles in energy metabolism, protein synthesis, and muscle tissue remodeling. Testosterone is one of the main hormones regulating post-exercise protein synthesis and muscle tissue recovery [29].

Physical exercise initiates numerous physiological adaptations in the endocrine system that maintain the organism's internal balance and allow adaptation to environmental demands. At the heart of these regulations is testosterone [7, 29, 70, 71]. Exercise has been shown to have a direct and significant effect on testosterone levels in men [53, 54, 69]. Indeed, a 6-week exercise program significantly increased total testosterone levels in older men who previously led a sedentary lifestyle [17]. Therefore, monitoring testosterone levels is of great importance for maintaining performance and preventing overtraining syndrome in athletes [71].

Normal testosterone levels are essential for both physiological adaptation and safe exercise participation in men. Moderate but regular physical activity has positive effects on erectile function, libido, and overall sexual satisfaction [62, 72]. However, exercise can have both beneficial and negative effects on

male reproductive health. The reproductive system is highly sensitive to the effects of exercise-related stress; therefore, testosterone release can increase or decrease depending on the type, duration, and intensity of exercise [62]. As a powerful stimulant for the endocrine system, exercise directly determines an individual's physiological and hormonal responses. Therefore, knowing the hormonal response to a single exercise session is important for determining appropriate training strategies [4, 28, 33, 38, 44].

Exercise is one of the most powerful physiological stimuli for reducing the negative effects of aging. Regular physical activity is considered one of the most effective non-pharmacological methods of increasing testosterone production [44, 73]. The magnitude of the testosterone response varies depending on parameters such as frequency, duration, intensity, and type of exercise [9–11, 31, 74]. Physical inactivity is a major risk factor that accelerates age-related testosterone decline. Low muscle mass, increased fat tissue, decreased blood flow, increased stress, and sleep disturbances lead to the suppression of testosterone synthesis. Visceral fat accumulation increases the release of inflammatory cytokines, which interfere with processes involved in testosterone synthesis and disrupt hypothalamus–pituitary–gonadal axis functioning, further reducing testosterone production [11, 31, 53, 54].

The hormonal response to exercise is related not only to the intensity and type of exercise but also to the training history of individuals. Hormonal adaptations are more balanced in trained individuals, while more pronounced fluctuations are observed in untrained individuals [71]. Therefore, the effect of a particular exercise model can vary depending on the individual's fitness level.

When exercise is not performed regularly, muscle mass decreases, which in turn leads to a decrease in testosterone synthesis. Inactivity facilitates testosterone decline by increasing the risk of metabolic syndrome, insulin resistance, and obesity. Furthermore, increased stress levels, along with increased cortisol secretion, directly suppress testosterone production [11, 13, 39]. Thus, a vicious cycle is created between inactivity and low testosterone levels. Although inactivity is a cause of reduced testosterone, its deficiency aggravates the process, which is associated with fatigue, depression, muscle loss, cognitive impairment, lack of energy, and cardiovascular disorders [11, 59]. The intensity of these experiences may vary between individuals. Many of these biological changes also the basis for psychosocial changes [59].

Different types of exercise elicit different physiological responses. Acute endurance exercises (*e.g.*, running, cycling, and swimming) lead to a short-term increase in testosterone levels, while prolonged, high-volume endurance exercise generally results in a decrease in testosterone. In contrast, resistance exercises and strength training involving large muscle groups cause both acute and chronic increases in testosterone levels. However, increased cortisol secretion in cases of overexertion and inadequate recovery can suppress testosterone synthesis [11, 38, 54].

The magnitude of the hormonal response depends on factors such as muscle group size, number of sets and repetitions, and exercise intensity. High-volume resistance training in-

volving large muscle groups leads to significant increases in testosterone levels, while prolonged endurance exercise lowers testosterone levels due to energy deficits and increased cortisol levels [11, 54]. In this context, HIIT has been found to be effective in increasing testosterone levels, reducing cortisol levels, and improving anabolic–catabolic balance, especially in men aged 35–40 years. Regular HIIT training has been associated with improved muscle strength, general wellbeing, and quality of life due to enhanced testosterone levels [30, 38, 43]. In addition, HIIT protocols have been demonstrated to induce significant changes in biochemical and anthropometric parameters [75].

In conclusion, exercise has a strong and positive influence on cardiovascular health and hormonal balance. Regular physical activity is associated with an increase in serum testosterone levels, which may counteract the adverse effects of age-related endocrine changes, especially in older adults [67]. Serum testosterone levels are significantly higher in well-trained older men, but testosterone levels are much lower in sedentary people. Therefore, regular exercise should be considered a fundamental lifestyle strategy for preventing age-related testosterone decline and mitigating the effects of andropause [40, 43, 76].

4. Discussion

The findings were normalized according to three major methodological and demographic factors: age group, training status, and testosterone sampling matrix, to account for variability across studies. The studies were divided into adolescents (14–20 years), young adults (21–40 years), and middle-aged to older adults (41–80 years), as well as into sedentary or insufficiently active, recreationally active, and athletic populations. Differences in biological sampling methods were also considered by distinguishing between serum and plasma (reflecting total testosterone) and saliva (reflecting free testosterone) samples. This stratification indicates that the acute increase in testosterone following high-intensity exercise was more likely to occur in young and middle-aged physically active men, and that responses were more variable in older sedentary men with chronically low baseline levels. Salivary free testosterone responses also seemed less consistent than serum total testosterone measures, especially in the older cohorts.

Nevertheless, this should be interpreted with caution, as most studies were scored as low risk of bias by the adapted scoring system. In exercise-based designs, the binary scoring approach may overestimate study quality, as the control of confounders is often partial. Thus, the large share of studies classified as low risk is likely to reflect the limitations of the adapted tool rather than consistently high methodological quality.

However, while this stratification allows for better interpretability, the included studies were highly heterogeneous and did not allow for consistent direct comparisons between subgroups. The wide age range (14 to 80 years), the prevalence of young and physically active populations, and the heterogeneity of training status limit the strength of conclusions from subgroups. Moreover, variability in findings may be due to differences in testosterone sampling methods, but the small

number of studies using salivary measures and inconsistencies in sampling protocols do not allow for definitive comparisons across matrices. Thus, the observed heterogeneity appears to be a complex interplay between methodological differences and participant characteristics rather than the effects of individual subgroups.

In addition to the stratified framework previously presented, the heterogeneity between the 20 studies included in this systematic review should be taken into account when considering the results. The participants included in the reviewed studies ranged in age from 14.3 to 80 years, covering a wide spectrum from children and adolescents [29, 38] to older adults [31, 32, 35]. Age is one of the primary determinants of testosterone regulation due to physiological processes (*e.g.*, puberty, andropause, *etc.*), which represents an important limitation when interpreting the overall findings. In addition, several factors may influence both the magnitude and temporal pattern of testosterone responses to exercise. These include fitness level (*e.g.*, sedentary individuals to trained athletes), health status (*e.g.*, healthy participants to patients undergoing hemodialysis), exercise modality (*e.g.*, HIIT, resistance training, running, cycling, functional training, and sprint interval exercise), and intervention duration (*e.g.*, acute exercise sessions to 13-week chronic training protocols). A further important methodological factor is the sample matrix (*e.g.*, saliva, serum, or plasma). Saliva reflects free testosterone, and serum and plasma reflect the total testosterone concentrations. This difference between serum/plasma and saliva studies restricts direct comparison of results. All these factors contribute to the variability reported between studies and highlight the need for standardized protocols stratified by age groups, fitness level, and health status in future research.

In addition to these stratified and methodological considerations, the type and intensity of exercise are important determinants of the testosterone response. Of particular importance, an increasing number of studies indicate that different exercise modes induce different endocrine responses, emphasizing the significance of training design in modulating hormonal responses.

Current evidence suggests that exercise-based lifestyle interventions may induce beneficial physiological and hormonal adaptations. Nevertheless, because the majority of the included studies were conducted in young, healthy, or physically active individuals, these findings should not be directly interpreted as evidence for reducing the clinical consequences of andropause in older adult populations. In addition, the available findings remain inconsistent across studies. While some studies have reported increases in testosterone concentrations after exercise, others have reported neutral results or even chronic decreases. Such differences are likely attributable to sample size, participant characteristics, training status, and methodological procedures, as well as differences in hormonal sampling and analysis protocols. Therefore, the hormonal responses evoked by exercise should be considered within their experimental and contextual factors.

A major challenge is understanding and managing age-related testosterone decline. In addition to medical treatments that directly target testosterone production, lifestyle strategies such as regular, well-balanced nutrition, physical activity,

and sufficient sleep are increasingly emphasised. Moderate-intensity exercise is one of the most frequently mentioned effective ways to increase the synthesis of testosterone among non-pharmacological strategies [11]. Moreover, regular physical activity is beneficial for maintaining male health and for preventing and reducing symptoms related to andropause [58, 60, 64].

Hormonal responses appear to be determined by the nature and intensity of exercise. Data suggest that interval protocols may elicit greater endocrine responses than continuous aerobic exercise. For example, Ahmadi *et al.* [4] (2018) showed that acute interval exercise significantly increased testosterone concentrations, but continuous aerobic exercise did not change the testosterone level significantly in healthy sedentary individuals [4]. Such findings support the hypothesis that greater levels of metabolic stress, neuromuscular activation, and muscle contraction force during interval training can stimulate a greater neuroendocrine response. Thus, HIIT has received considerable attention. Several studies have demonstrated favorable hormonal responses to structured HIIT programs. Zar *et al.* [9] (2021) studied the effect of exercise timing and found a significant increase in testosterone levels after HIIT only in the afternoon, not in the morning.

Similar results were obtained by Ambroży *et al.* [30] (2021), who reported an increase in testosterone levels by 36.7% after an 8-week training program. In conclusion, these studies suggest that HIIT may be a useful strategy to counteract age-related hormonal decline and improve general physical fitness. However, these positive findings are not consistent across all studies. Velasco-Orjuela *et al.* [13] (2018) investigated the effects of HIIT and resistance training on serum testosterone concentrations in overweight physically inactive adults and did not find significant acute effects. Himel and Keefe (2025) also proposed that high-intensity of exercise combined with psychological or physiological stress may interfere with the hypothalamic–pituitary–gonadal axis and cause functional hypothalamic hypogonadism [77]. These conflicting results underscore the importance of participant characteristics (age, body composition, training history, and baseline hormonal levels) and protocol characteristics (duration, intensity, recovery intervals, and total workload). The results may be due to other factors, such as the timing of hormone measurements and circadian rhythm.

Also, the timing of hormonal responses seems to be important in addition to the intensity. Some evidence suggests that post-high-intensity exercise elevations in testosterone are transient rather than sustained. Cofré-Bolados *et al.* [33] (2019) demonstrated a significant increase in plasma testosterone immediately after an HIIT protocol, which returned to baseline within 12 h. Similarly, Dote-Montero *et al.* [73] (2021) speculated that testosterone may increase after a session, return to baseline within 15–30 min, and in some cases decrease below baseline within 60–180 min. These results suggest that short-term endocrine responses can be robust but transient and not necessarily representative of longer-term adaptations.

There is additional context from systematic evidence in older populations. In a review, Zouhal *et al.* [44] (2022) reported that HIIT, endurance, and resistance exercise, or combined protocols, generally increased testosterone concen-

trations in healthy individuals over 40 years of age, while low-intensity exercise had limited effects. This finding aligns with the results reported by Zurek *et al.* [28] (2022), who did not observe any change in salivary testosterone concentrations in young, active men after sprint interval training. Such differences can be explained by variation in training protocols, subject characteristics, and biological sampling methods.

One major caveat is that most of the studies reporting acute increases in testosterone after HIIT or sprint interval training have involved young (18–30 years) physically active men. Whether these acute responses translate into sustained improvements in older men with age-related reductions in testosterone remains to be determined. Thus, our findings should be considered mechanistic evidence of exercise-induced endocrine responsiveness and not direct evidence for a treatment for andropause.

Hormone measurement is fraught with methodological problems that add to the difficulties of interpretation. Serum and saliva testosterone levels have been measured, but they are different physiological fractions. Salivary testosterone levels represent free (unbound) hormone and are highly sensitive to sampling conditions, hydration status, storage temperature, and timing. This has led to several studies reporting a poor correlation between salivary and serum measurements [56]. Comparative protocols following strenuous exercise have demonstrated a decrease in serum testosterone [29], but no change in salivary testosterone. Blood-based measures are therefore often considered more accurate indicators of hormonal change, particularly in older individuals [27].

In summary, these findings suggest that exercise-induced testosterone responses are influenced by complex interactions among exercise type, intensity, timing, measurement technique, and subject characteristics. The variability observed highlights the need for standardized methodologies and well-controlled long-term studies to elucidate the true magnitude and clinical significance of these hormonal adaptations.

Endocrine adaptations seem to be more stable with combined interventions and in the long term than with short-term or single-modality protocols. For example, in men aged 40–59 years with symptoms of androgen deficiency, a 6-month program of aerobic and resistance exercise performed 3 days per week led to significant increases in serum testosterone levels and marked improvements in clinical symptoms [27]. These results are in agreement with the previous observations reported by Smith *et al.* [41] (2013). Likewise, a 13-week combined sprint and resistance protocol in soldiers increased basal serum testosterone levels in young and middle-aged men [40].

In contrast, long-term exercise modes do not elicit similar endocrine effects. Hawkins *et al.* [35] (2008) investigated the effects of moderate-intensity aerobic training for 12 months in middle-aged men and observed no significant changes in total or free testosterone levels despite improvements in cardiopulmonary fitness. Interestingly, the same study reported an increase in dihydrotestosterone, a more potent androgen, indicating that aerobic exercise can affect androgen metabolism without directly increasing testosterone synthesis. This finding underscores that exercise-induced physiological adaptations can occur through several endocrine pathways and not solely

through testosterone.

Mangine *et al.* [37] (2018) observed significant increases in salivary testosterone concentrations following a 5-week high-intensity functional training program with 5 recreationally active males (mean age 34.4 ± 3.8 years) and 5 recreationally active females (mean age 35.5 ± 7 years). Similar findings were reported in another study in which HIIT increased salivary testosterone release rates in 20 young athletes (age 20.2 ± 0.7 years) [10]. Similarly, an 8-week HIIT intervention in 20 young male athletes (age 17.7 ± 2.3 years) resulted in higher serum testosterone levels at the end of the training period [38].

Despite these positive observations, some methodological limitations related to salivary assessment should be considered. Because saliva reflects only the free fraction of testosterone, sampling conditions, timing of sample collection, storage temperature, and individual hydration status could all substantially affect results. Several studies have shown weak correlations between salivary and serum testosterone values [56]. In addition, side-by-side comparisons of biological matrices have yielded inconsistent findings. For example, saliva and venous blood samples taken from 30 adolescent swimmers, after a protocol of intense swimming followed by high-intensity interval swimming (5×100 m, 5×50 m, and 5×25 m), showed that serum testosterone was decreased after exercise, whereas salivary testosterone did not [29]. Such differences emphasize the importance of the biological matrix in the interpretation of hormonal responses, with blood-based measures generally considered the most reliable indicator of endocrine changes, especially in the older adults [27].

Studies have been conducted comparing resistance modalities and provide additional support for the interaction between mode of exercise and hormonal response. Schwanbeck *et al.* [39] (2020) found that after 8 weeks of training, men had significantly greater increases in salivary testosterone when performing free-weight resistance training compared to machine-based exercises. Because free-weight movements demand higher levels of balance, stabilization, and central nervous system activation, they can provoke stronger anabolic and neuroendocrine stimulation. These findings reinforce the notion that both exercise intensity and mechanical and neuromuscular characteristics influence hormonal adaptations.

Moreover, the relationship between exercise dose and testosterone seems to be non-linear. Evidence suggests that very strenuous or prolonged activities may actually suppress rather than stimulate. Significant reductions in serum testosterone concentrations were reported after a marathon run (42.2 km) in healthy male subjects by Kuusi *et al.* [36] (1984). This finding differs from studies reporting increases following resistance or high-intensity protocols [27, 35] and is consistent with the concept of a dose-response relationship, where extreme energy depletion and physiological stress transiently suppress the hypothalamic–pituitary–gonadal axis. Therefore, increasing exercise volume does not necessarily lead to better hormonal outcomes, and the optimal balance between intensity and duration appears to be critical.

The role of exercise is particularly relevant in those with already low testosterone concentrations, with broader clinical implications. Low levels of hormones have been associated with decreased muscle mass, strength, and habitual physical

activity, which may lead to a cycle of functional decline [32]. Structured training programs have the ability to break this cycle, improving metabolic health, body composition, and endocrine balance all at the same time. Indeed, exercise interventions have been reported to be superior to HRT in terms of physical performance and functional outcomes, as well as in increasing endogenous testosterone levels [31]. Moreover, the additive benefits of pharmacological approaches with exercise have not been consistently documented, emphasizing the independent therapeutic value of structured physical activity.

Outside of clinical populations, research investigating the association between testosterone and performance outcomes indicates that change in hormone levels should not be considered in isolation. For example, Crewther *et al.* [34] (2017) found that testosterone levels increased by an average of 6.1% following a sprint training program in healthy, active men. This suggests endocrine responses are present with performance adaptations, but are not the only driving response. Testosterone is just one component of a complex physiological web that is influenced by neuromuscular, metabolic, and psychological factors.

In summary, the findings suggest that the exercise-induced testosterone responses are not mediated by a single or uniform mechanism. Differences in age, training history, body composition, duration, intensity, recovery intervals, and sampling procedures strongly contribute to the variability between studies. Long-term exhaustive endurance training can inhibit testosterone generation. A more stable avenue to testosterone production is through resistance training and high-intensity interval training. This is more likely to produce short-term gains and more consistent adaptations with continued practice. Heterogeneity in reported outcomes is also due to measurement techniques (*e.g.*, serum vs. saliva sampling).

In general, literature suggests that exercise is a safer, more sustainable, and holistic strategy for the prevention and management of andropause than pharmacological treatment. Importantly, exercise has not only hormonal modulation effects, but also cardiometabolic health, muscle mass, psychological well-being, and quality of life benefits. The primary foundation for addressing age-related testosterone decline should therefore be structured and individualized exercise programs, rather than pharmacological options.

In this systematic review, testosterone was assessed in different matrices: Serum ($n = 10$), plasma ($n = 3$), saliva ($n = 5$), or both ($n = 2$). This distinction is methodologically important because serum and plasma measurements reflect total testosterone concentrations, including protein-bound (mainly to SHBG and albumin) and free fractions, whereas saliva represents the free (biologically active) fraction of testosterone. These two measures are not directly comparable, as free testosterone is only 2–3% of total testosterone and may have different dynamics in response to exercise. Salivary levels can be influenced by the salivary flow rate and the collection method. Serum and plasma levels can be influenced by binding proteins such as SHBG. Total testosterone concentration measured in serum or plasma remains the preferred biomarker for diagnosis of hypogonadism and monitoring of testosterone replacement therapy in clinical practice. Salivary testing is non-invasive but poorly standardized clinically and has a weak-to-moderate

correlation with serum values in older adults. Therefore, in the context of andropause, measurements of serum total testosterone are more clinically useful and dependable than measurements of SHBG.

Most of the included studies were conducted on young or athletic populations, with only three studies specifically targeting middle-aged and older men. Therefore, generalizing these findings to men in andropause should be approached with caution. Accordingly, findings from adolescent cohorts should be interpreted primarily as mechanistic evidence of acute endocrine responsiveness rather than as direct evidence of LOH or andropause-related interventions.

Notably, most of the included studies were conducted on young, healthy, and physically active individuals. Therefore, extrapolation of these findings to aging or clinical populations, particularly men with andropause, should be approached with caution. The current evidence does not sufficiently represent individuals with clinically low testosterone levels.

Two reviewers independently screened and extracted data and resolved any disagreements by consensus, but formal inter-rater reliability statistics (*e.g.*, Cohen's kappa) were not calculated, which is a methodological limitation affecting the reproducibility of this study.

Importantly, most of the included studies were conducted on young, healthy, or athletic populations rather than men diagnosed with andropause or LOH. As such, the evidence presented in this review should be considered mechanistic evidence of exercise effects on testosterone dynamics rather than direct clinical evidence for the treatment or management of andropause. Future research should investigate the clinical relevance of these findings, especially in aging and clinical populations.

5. Conclusions

The findings of the present systematic review suggest that testosterone responses are influenced by different exercise modalities, intensities, and durations. High-intensity and resistance-based training protocols are frequently associated with acute increases in testosterone levels; however, this response is not consistently observed across all studies. Moreover, these physiological changes do not necessarily correspond to clinically meaningful benefits, particularly in populations other than those investigated. In contrast, prolonged endurance exercise is more commonly linked to attenuated or unchanged testosterone responses. **Supplementary material** includes additional supporting tables and methodological details.

It should also be emphasized that most of the included studies were conducted in young, healthy, or athletic individuals. Therefore, the current findings should primarily be interpreted as physiological and mechanistic evidence, with limited direct clinical relevance for men with andropause.

In addition, regular exercise may contribute to the maintenance of overall hormonal balance and general health. But there is no evidence that exercise is a primary treatment for andropause. Instead, exercise should be viewed as an adjunct, as the direct clinical benefit of exercise is unclear.

Future studies using well-defined populations with

andropause, long-term interventions, and standardized measurement protocols are needed to clarify the clinical relevance of exercise in age-related decline in testosterone levels.

Based on our findings, we recommend the following in accordance with the existing evidence and its limitations for future research and clinical applications:

- Future studies should explore the effects of exercise on andropause in larger sample sizes and with standardized serum-based measurements in long-term studies.

- Longitudinal studies, well-designed, homogeneous groups of participants, should monitor the effects of regular exercise on muscle strength, bone density, cardiovascular capacity, and psychological well-being during andropause.

- Exercise should be considered as part of an overall strategy for andropause management, especially in sedentary and at-risk populations, by healthcare professionals. It is important to note, however, that the evidence so far is preliminary, with small sample sizes and short intervention durations. Furthermore, detailed investigations over a longer period of time are required before firm clinical guidelines can be developed.

ABBREVIATIONS

HIIT, High-Intensity Interval Training; HRT, Hormone Replacement Therapy; LOH, Late-Onset Hypogonadism; SD, Standard Deviation; RT, Resistance Training; IE, Intermittent Exercise; CE, Continuous Exercise; HD, Hemodialysis; SHBG, Sex Hormone-Binding Globulin; GnRH, Gonadotropin-Releasing Hormone; LH, Luteinizing Hormone; JBI, Joanna Briggs Institute; StAR, Steroidogenic Acute Regulatory; PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses; IGF-1, Insulin-like Growth Factor 1; SCIE, Science Citation Index Expanded; ESCI, Emerging Sources Citation Index; DOAJ, Directory of Open Access Journals; ESA, Erythropoietin-Stimulating Agents.

AVAILABILITY OF DATA AND MATERIALS

Not applicable.

AUTHOR CONTRIBUTIONS

MÖ and MA—conceptualization. İK, MÖ, MŞÖ and MA—literature analysis. MŞÖ, OÇ, EB and MÖ—writing—original draft preparation. AE, MT and MA—writing—review and editing. MÖ—supervision. MS and İP—data curation. All authors have read and agreed to the published version of the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found, in the online version, at <https://oss.jomh.org/files/article/2062816906652336128/attachment/Supplementary%20material.pdf>.

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