Sport Sciences for Health (2026) 22:261
https://doi.org/10.1007/s11332-026-01824-4

REVIEW

The influence of female sex hormones on knee laxity, neuromuscular function, and performance: a systematic review

Holly L. Whiter[1] · **Christopher J. Holland**[1] · **Darren J. Cooper**[1]

Received: 5 December 2025 / Revised: 10 June 2026 / Accepted: 12 June 2026

Abstract

Background Female-specific research in sport science is gaining momentum, addressing a historical gap where evidence has predominantly stemmed from male participants. Understanding how female sex hormones influence performance and injury risk is essential for developing training and rehabilitation strategies to the menstrual cycle.

Objective This systematic review aimed to evaluate the effects of female sex hormones on knee joint laxity, neuromuscular function, and performance, identifying patterns, methodological limitations, and implications for athletic practice.

Methods Following PRISMA 2020 guidelines, systematic searches of PubMed, MedLine, and CINAHL databases were conducted using defined MESH terms. Studies published between January 2010 and June 2025 were screened against strict inclusion criteria, focusing on eumenorrheic women aged 18–45 years old (26.91 years old ± 4.18). Quality appraisal and risk of bias were performed using the PEDro scale, with a cut-off score of ≥ 6/10.

Results Evidence suggests that female sex hormones may influence aerobic performance and recovery, with exogenous testosterone supplementation showing notable enhancement. Aerobic training may benefit from alignment with mid-cycle phases, while anaerobic and strength training appear less affected. Neuromuscular function remains largely stable across menstrual phases, though some studies indicate improved rehabilitation outcomes during the ovulatory phase. No consistent evidence was found linking menstrual hormones to changes in knee joint laxity.

Conclusion Emerging evidence supports hormone-sensitive modulation of aerobic performance and recovery, findings across neuromuscular function and joint laxity remain inconclusive and underpowered. Practical applications are emerging, particularly in endurance training and cycle-informed rehabilitation. Further high-quality, longitudinal research is needed to validate insights and inform evidence-based practice.

Keywords Menstrual cycle · Sex hormones · Injuries · Knee · Lower extremity · Female

Introduction

Despite the growing number of women participating in sports, the female population remains significantly underrepresented in sports and exercise research, particularly randomised control trials [1]. In this review, the term "female" as used in its scientific meaning refers to individuals assigned female at birth. Only 35–37% of study participants are female across articles [2, 3], posing a serious challenge for evidence-based practice in female sport, performance, and rehabilitation, making it difficult for coaches, clinicians, and practitioners to make informed decisions tailored to female athletes. This deficit in the research is concerning, particularly given the overall injury statistics observed in female sports, where recent data indicates an injury incidence rate of 4.4 injuries per 1000 h of exposure with an average of 10 days lost per injury [4]. Females are more likely to sustain an overuse injury, such as a sprain or strain, than males (19.7 vs 13.6 per 100,000 athletic exposures) across all sports, particularly in sports such as basketball and soccer (82%, 37% higher risk) [5]. Furthermore, females experienced at least one time loss injury and were at a significantly higher risk of knee injuries compared to males [3]. In sport, the noun "performance" can be defined as an individual's competence or achievement in such a performance that may involve winning, achieving personal bests or achieving an increase in a measurable outcome [6]. These

✉ Holly L. Whiter
whih1_25@uni.worc.ac.uk

[1] School of Sport and Exercise Science, University of Worcester, Henwick Grove, Worcester WR2 6AJ, UK

Published online: 15 July 2026

are the typical benchmarks of outstanding achievements; however, performance is subjective (through athlete perception or judged sports such as gymnastics) and therefore is often difficult to truly define [7].

Males and females exhibit physiological differences due to both genetic and hormonal factors [8]. While certain performance-enhancing polymorphisms (PEPs) occur at comparable frequencies in both sexes [9], fundamental differences in sex chromosomes (XX in females vs XY in males) lead to sex-specific development and endocrine profiles. Females' two X chromosomes facilitate the development of ovaries and the cyclical production of female sex hormones (oestrogen, progesterone, luteinising hormone [LH], follicle-stimulating hormone [FSH], and smaller amounts of testosterone) [10, 11].

At puberty, the hypothalamus begins pulsatile release of gonadotropin-releasing hormone (GnRH), which prompts the pituitary to secrete FSH and LH; these gonadotropins stimulate the ovaries to produce oestrogen and progesterone in a cyclical pattern [11]. This initiates ovulation, menarche, and the onset of the menstrual cycle, characterised by fluctuating levels of female sex hormones [10] (Fig. 1). The cycle begins with menstruation (day 1), when falling progesterone and oestrogen levels (due to lack of fertilisation) trigger shedding of the uterine lining (menses, the start of the follicular phase) [12]. In the follicular phase (approximately days 1–14), rising FSH stimulates ovarian follicular growth. A dominant follicle emerges by ~ day 7 and produces oestradiol (17-β E2) and inhibin B to suppress further FSH release [13]. Increasing oestrogen also stimulates endometrial thickening (the uterine proliferative phase) and produces thin, elastic cervical mucus to facilitate sperm passage [14, 15]. Around day ~ 14, an LH surge triggers ovulation (oocyte release) [16]. At this time, female total and free testosterone levels peak, remaining elevated into the early luteal phase [17]. Following ovulation, the luteal phase is dominated by progesterone as the ruptured follicle forms the corpus luteum, which secretes predominantly progesterone and some oestrogen while estradiol levels fall [18]. If no fertilisation occurs, the corpus luteum regresses and hormone levels decline, causing the next menstrual period [12]. If pregnancy occurs, the embryo's human chorionic gonadotropin (hCG) sustains the corpus luteum and its hormone production [12]. High luteal-phase progesterone matures the endometrium (secretory phase, with thickened uterine lining and viscid cervical mucus) [16].

Menstrual cycle length is typically 26–35 days (mean ≈28 days, SD ± 3.4) among eumenorrheic (regular-cycling) females [18, 19]. About 95% of cycles last 22–36 days, and ~ 42.5% of women experience an intra-cycle length variability greater than 7 days [21]. Menstrual cycles may be considered "irregular" if their length varies by more than ~ 8–10 days, depending on age group [19, 20]. Irregular cycles can stem from hormonal imbalances, changes in lifestyle or environment, or underlying medical conditions [22]. Importantly, the cyclical fluctuations in female hormones can induce physiological, psychological, and perceptual changes that affect athletic performance [23, 24]. For example, rising oestrogen in the follicular (pre-ovulatory) phase is associated with increased collagen synthesis and improved

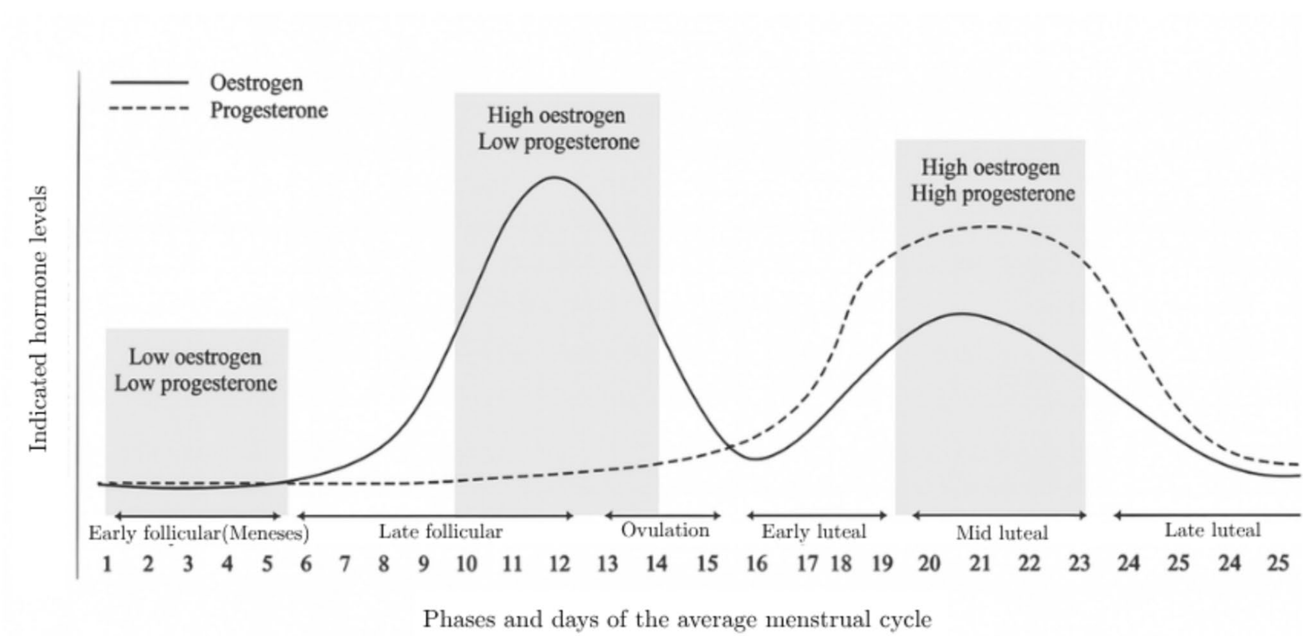

Fig. 1 The pattern of hormonal fluctuations over a standard 28 day menstrual cycle. Adapted from McNaulty [12]

skin hydration [23–25]. In contrast, the progesterone-dominant luteal phase is linked to diminished muscle strength compared to the oestrogen-rich follicular phase [8, 26]. Furthermore, one study found that female athletes with naturally high free testosterone (upper tertile) performed ~ 2–4% better in 400 m, 800 m, and pole vault events, suggesting a competitive performance advantage from greater endogenous testosterone [27]. These observations support the concept that fluctuating female sex hormones may influence performance and possibly alter injury risk across different phases of the menstrual cycle [27].

Females consistently exhibit a greater anterior knee laxity than males [28]. Hormonal changes throughout the menstrual cycle have been hypothesised to increase anterior cruciate ligament (ACL) injuries in females [29]. Research has shown an increase in knee joint laxity during the follicular phase due to an increase in oestrogen, and again in the early luteal phase when progesterone peaks [29, 30]. Contrasting research suggests significantly higher genu recurvatum and joint laxity in the ovulatory phase than the follicular phase based on the rise in concentration of oestradiol [31]. Even small increases in anterior knee laxity (≈1.3 mm) can markedly elevate joint loading during dynamic tasks, including a 30% greater adduction impulse during cutting and 20–45% higher external rotation loads during jumping and stopping [32]. Complementary evidence shows that greater laxity is also linked to poorer balance performance during ovulation [33], suggesting that increased joint laxity may alter movement mechanics in ways that impair performance and elevate injury risk.

Stable hormone levels are associated with consistent neuromuscular function, whereas hormonal fluctuations across the menstrual cycle led to notable changes in the functionality of the central nervous system (CNS) [34]. Oestrogen can be seen to increase neural excitability and voluntary activation, while progesterone increases inhibition and reduces activation [34, 35]. As these hormones rise and fall throughout the menstrual cycle, corresponding changes in neuromuscular activity and muscle function can occur. Early research [36] observed significant differences in the maximal voluntary contraction in the mid-cycle/ovulatory phase compared to other phases of the menstrual cycle ($p \leq 0.05$). Weidauer et al. [26] supports this, by stating that as oestrogen rises and progesterone is low in the follicular phase, the isokinetic peak torque (60°/sec) at the knee was lower (83 ± 14 nM) compared the ovulation phase (86 ± 15 nM, $p \leq 0.05$). Collectively, these findings indicate that hormonal fluctuations across the menstrual cycle can influence neuromuscular performance and motor control.

The current research investigating the influence of the menstrual cycle on performance presents mixed and often conflicting findings. Research suggests that fluctuations in oestrogen, progesterone, and testosterone may influence strength, endurance, coordination, and fatigue [26, 29, 33–35]. Other reports minimal or no significant phase-related effects [37].

This review is timely given the growing focus on female-specific research in sport science and the need to address the historical reliance on male-derived evidence. Although several systematic reviews have attempted to synthesise existing findings, conclusions remain inconsistent. A brief examination of the literature suggests that some associations do exist, and this discrepancy highlights how relying on isolated studies can lead to a skewed understanding of female physiology. To address this, the review aims to critically evaluate how fluctuations in female sex hormones influence knee laxity, neuromuscular function, and performance with a specific focus on identifying whether these hormonal effects meaningfully alter injury risk or require adjustments to training and rehabilitation practise. This includes analysing methodological strengths and limitations across studies and determining the extent to which current evidence can inform applied decision making.

Given that much of the existing research is derived from athletic populations, limiting its applicability to the wider female population, the review also considers the applicability of findings beyond sport. The insights gained aim to guide athletes, coaches, and medical teams in tailoring training and injury-prevention strategies to the menstrual cycle.

Methods

Study design

A systematic review was conducted following the recommendations of Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 criteria [38]. This review was registered in PROSPERO 2025 (CRD420251045911).

Data source

The searches were performed by two independent investigators, HW and CH, in the PubMed, MedLine and CINAHL databases.

Search strategy

A search equation using Medical Subject Headings (MeSH) terms was defined to obtain a precise search: "menstrual cycle" OR "sex hormones" AND "injuries" AND "knee" OR "lower limb" OR "lower extremity" AND "female". A publication date filter was added from January 2010 to June 2025 along with the following filters were applied: free full text, randomised control trial, English, Humans, and Adult 19–44 years.

Inclusion and exclusion criteria

After duplicates were removed, studies were excluded if they did not match the following inclusion: (i) article written in English; (ii) population: females aged 18–45 years; (iii) a defined menstrual cycle (eumenorrheic); (iii) at least a year post-menarche; (iv) studies examining knee laxity, neuromuscular function, or performance; (v) randomised control trials and primary studies only; (vi) peer reviewed and full published journals; and (vii) publication date between January 2010 and June 2025. Exclusion criteria were as follows: (i) post-menopausal females; (ii) pregnant or breastfeeding females; (iii) studies not written in English; (iv) participants outside the 18–45 age range, (v) non-primary research; (vi) non-peer-reviewed sources; and (vii) studies published outside the January 2010 and June 2025 window.

Review process and quality of studies

The review process was conducted in accordance with the PRISMA guidelines. The initial search was completed by HW using the defined inclusion and exclusion criteria and Boolean operators. The resultant article list was screened independently by HW and CH and the selected articles were cross-referenced to establish the studies to be included in the Physiotherapy Evidence Database (PEDro) analysis. A PRISMA flow diagram was used to document the identification, screening, eligibility, and inclusion of studies (Fig. 2). Data extraction, quality appraisal, and risk of bias were assessed using the PEDro 11-point scale independently by HW to ensure objectivity and consistency across articles. On the PEDro scale, item 1 (eligibility criteria) although analysed it is not included in the calculation of the score [39]. The PEDro cut-off score was set at > 6/10 based upon recommendations from Cashin and McAuley [39] to ensure the standard of the methodological criteria for internal validity and statistical reporting [40].

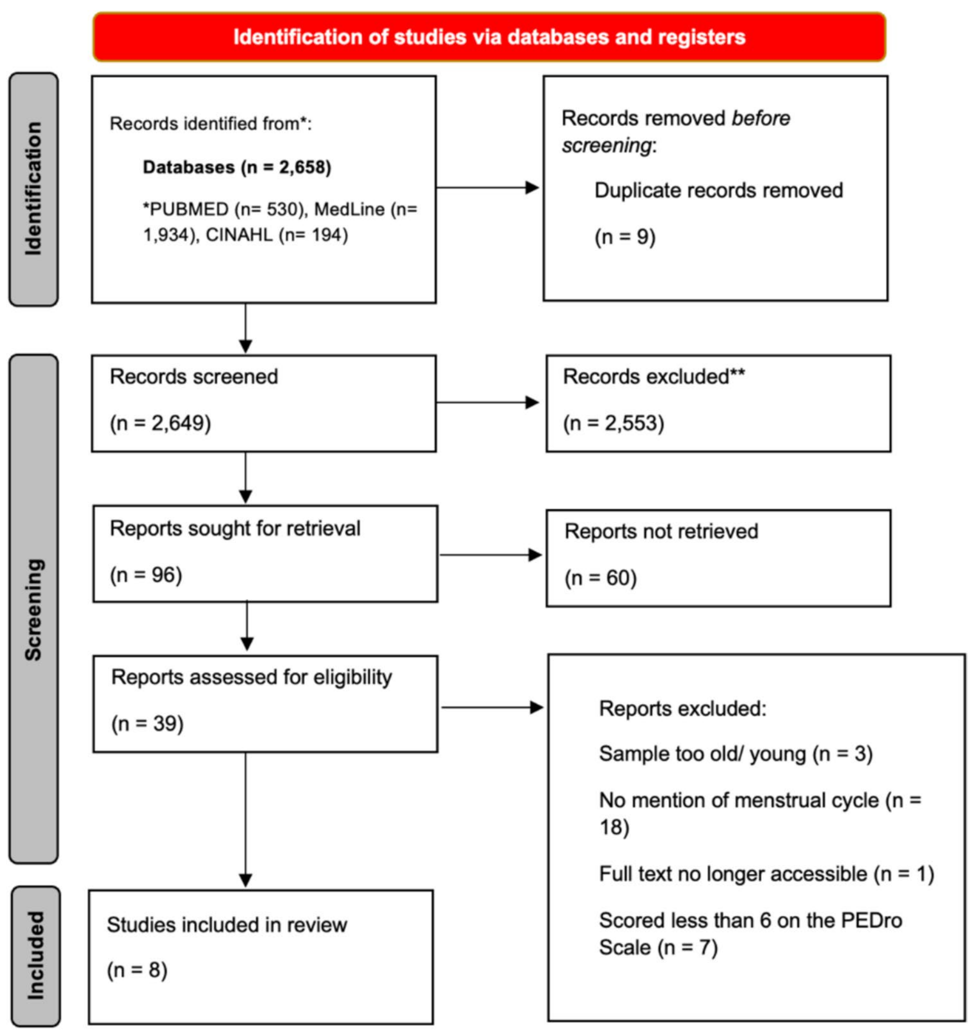

Fig. 2 PRISMA workflow diagram for this systematic review

Data extraction and Synthesis

The following parameters were extracted from the collected studies: (a) author and year of publication, (b) study design, (c) participant details, (d) protocols, (e) results, and (f) conclusion. Data were extracted from the included studies and collated into a table for further analysis.

Results

Search results

Eight articles were selected in this systematic review according to the inclusion and exclusion criteria (Table 1). The sample sizes ranged from 12 to 391 women aged 18 to 35 years old (26.91 years old ± 4.18). The studies included in this review had scores based on the PEDro guidelines for appraising studies between 6/10 and 9/10 (Table 1). Two studies scored 6/10 [41, 42], five studies scored 7/10 [43–47], and one study scored 9/10 [48].

Menstrual cycle phase and verification

Across the eight studies selected, researchers used a combination of methods to determine menstrual cycle phase. Six of the studies relied on self-tracking, in the form of menstrual diaries [43–47]. Five studies verified cycle phase using blood serum analysis, measuring oestradiol (E2), progesterone (P4), and/or testosterone [41, 42, 45, 47, 48]. Although Shahid and Noor [43] also used blood serum, their focus was on how integrated exercise affected testosterone rather than on identifying cycle phase. Pfeifer et al. [47] and O'Loughlin et al. [46] used ovulation kits, and O'Loughlin et al. [46] also tested basal body temperature. Finally, saliva sampling was used by Dias et al. [42] to measure oestradiol (E2) and progesterone (P4) levels to verify menstrual cycle phases.

Effect of exercise on hormonal levels

Three studies monitored hormonal fluctuations across the menstrual cycle in relation to exercise performance. Two focused specifically on natural and supplemented [43, 48], whereas another assessed a broader panel of sex hormones (testosterone, progesterone, oestrone sulphate, SHBG) [41]. In the natural testosterone studies, exercise caused a transient rise in testosterone 15 min post-exercise, especially during mid-cycle phase, followed by a drop below baseline 24 h later, again strongest in mid-cycle ($p \le 0.05$ for group, time, and phase interactions) [43]. When testosterone was provided as a supplement, increasing levels from 4.8 times to 4.3 nmol/L produced measurable performance benefits with a 6.2% improvement in time to exhaustion and higher maximal aerobic speed, equivalent to running 7.7 s faster over 1500 m and 10.8 s over 2000 m [48]. In contrast, Smith et al. [41] report no significant change in testosterone following exercise. Instead, exercise led to a small increase in oestradiol (exercisers 2.3 ± 2.8 pg/mL, control 1.4 ± 3.0 pg/mL, ($p = 0.83$) and a modest decrease in progesterone in the exercise group ($p \le 0.05$), though this reduction did not differ significantly from the control group.

Knee joint laxity

Only Pfeifer et al. [47] referred to joint laxity at all, noting in their discussion that oestrogen phases may lead to increased joint laxity and instability. However, the study's findings mainly indicated an increased vulnerability of muscle fibres during the late follicular phase rather than changes in joint structures.

Neuromuscular function

From the included articles, six discussed the impact hormones can have on strength in females; resistance training was consistently associated with strength gains over time, regardless of hormonal intervention. O'Loughlin et al. [46] reported a significant time effect for knee extension strength in the non-injured leg ($p \le 0.01$) across both control and menstrual cycle phase-based rehabilitation (MCPBR) groups, indicating that strength improved with training over time. Although the MCPBR group showed higher knee extension 1RM in the injured leg (38.8 kg) compared to controls (30.40 kg), the difference narrowly missed statistical significance ($p = 0.06$). Improvements in strength, specifically knee extension, were primarily driven by the training itself, not by the menstrual cycle-related hormonal variations.

Dias [42] saw significant increases in muscle cross-sectional area (CSA) during the later phases of the menstrual cycle (O2, L2, M3), with moderate effect sizes ($d = 0.65$–0.76), suggesting that resistance training may be more effective during the luteal or mid-luteal phases. In contrast, Colenso–Semple et al. [45] found no significant differences in muscle protein breakdown (MPB) across phases, although a slight, non-significant increase was noted in the luteal phase ($p = 0.24$). Similarly, Shahid and Noor [44] reported only small changes in quadriceps strength across menstrual phases in the control group following a BRACTS exercise approach. For example, right quadriceps strength in the follicular phase post-intervention was 68.31 ± 11.07 kg ($d = 0.47$), with minimal variation across mid-cycle and luteal phases, suggesting that natural hormonal fluctuations alone do not drive meaningful strength adaptations. Taken together suggest that natural fluctuations in ovarian hormones exert only a limited influence on key

Table 1 Summary of articles used in the systematic review

Author (year)	Study type	Participant details	Study aim	Intervention and/or results	PEDro score
Smith et al. (2011)	Randomised, controlled, parallel-arm clinical trial	391 women, 18–30 years (mean age 25.3 years, SD ± 3.45), with a sedentary lifestyle and a self-reported menstrual cycle length of 24–25 days, no report of injury status	Examine whether moderate-to-vigorous aerobic exercise alters sex hormone and SHBG levels	Women randomised to the exercise intervention trained aerobically 5 × a week for 30 min on a treadmill, stair stepper, or elliptical machine, at a specified intensity (65–70%, then increased by 5% every 4 weeks until 80–85%) based on age-predicted maximal HR for 4 menstrual cycles. No significant changes in oestradiol, oestrone sulphate, testosterone, or SHBG between exercise and control groups. Aerobic fitness increases with exercise	6/10
Shahid and Noor (2025)	Single-blinded, two-armed, parallel-group randomised controlled trial	40 women, 20–40 years, with consistent cycles spanning 24–35 days, must be able to maintain a sitting balance without requiring upper body support and a minimum score of 25 on the trunk control test, not using oral contraceptives or participated in any exercise regimen over the preceding 6 months	To determine if integrated exercise approach on total testosterone levels	A control group was advised to walk for 30 min 3 times a week on alternate days for 16 weeks, while experimental group was subjected to an integrated exercise protocol of 50 min on alternative days, totalling 3 weekly sessions for 16 weeks. Testosterone levels increased significantly within 15 min post-exercise in the experimental group across all cycle phases and testosterone decreased below baseline 24 h post-exercise. Peak levels of testosterone were observed during the mid-cycle phase, and the control group showed no significant hormonal fluctuations	7/10

Table 1 (continued)

Author (year)	Study type	Participant details	Study aim	Intervention and/or results	PEDro score
Hirschberg et al. (2020)	Double-blind randomised, placebo-controlled parallel study	48 healthy women, 18–35 years (mean age 28.4 years, SD ± 3.75), at a moderate to high self-reported level of recreational activity, not oligomenorrhea or amenorrhea, not taking contraception, no report of injury status	Examine weather moderately increased testosterone concentration (10 mg testosterone via a cream) on physical performance	Following a series of performance tests, namely, jump tests, knee extension torque, running test on treadmill and the Wingate test. Moderately increasing testosterone in females causally enhances aerobic endurance (time to exhaustion) and increases lean mass, without affecting anaerobic power or muscle strength. Changes in serum testosterone also correlated with changes in total lean mass. There were significant positive correlations between changes in total lean mass and lower limb lean mass and Wingate average power	9/10
Shahid and Noor (2025)	Single-blinded, two-armed, parallel randomised controlled trial	40 women, 20–40 years (mean age 29.85 years, SD ± 6.11), included women had a menstrual cycle of 24–35 days, women must have been able to maintain a sitting balance without requiring upper limb support or had a minimum score of 25 on the trunk control test with participants refrained from any exercise regimen over the preceding six months. However, excluding if women were taking oral contraceptives	Discover the impact of BRACTS exercises on muscular strength	The control group engaged in a 30 min brisk walk 3 × a week on alternate days for 16 weeks and adhere to a standard dietary plan, while experimental group went through 50 min of BRACTS exercises on alternative days 3 × per week over 16 weeks. Experimental group showed significant improvements across all phases, particularly hand grip (left/right) – peak Cohen's d during follicular phase and quadriceps (left/right) and gastrocnemius–soleus – peak gains in mid-cycle phase	7/10

Table 1 (continued)

Author (year)	Study type	Participant details	Study aim	Intervention and/or results	PEDro score
Colenso-Semple et al. (2024)	Randomised, controlled, within-subject crossover study	12 healthy women, 18–30 years, women had a regular menstrual bleed as determined by a tracking app and not used any hormonal contraceptives for 6 months prior to the study. Participants were excluded if they suffered from any disorders (including orthopaedic and menstrual disorders)	Menstrual cycle phase does not influence muscle protein synthesis or whole-body myofibrillar proteolysis in response to resistance exercise	Unilateral knee extension exercise for 10 reps max each leg, one leg serves as the exercise leg and the other a control, alternating between phases. Menstrual cycle phase does not influence acute muscle protein synthesis or whole-body proteolysis in response to resistance exercise. This suggests exercise-induced muscle adaptation mechanisms are not hindered or enhanced by natural hormonal fluctuations in eumenorrheic women	7/10
O'Loughlin, Reid, Sims and Larsen (2024)	Single-blind, two-arm randomised controlled trial	43 females, 18–35 years (mean age 31.6 years, SD ± 8), only females using copper or progestin-only IUD were included, females had to be under 6 weeks post-ACL reconstruction and those which undertook allograft or revision surgery were excluded	Determine if a menstrual cycle-based rehabilitation programme is suitable for patients following ACL reconstruction	All females attended twice – weekly, supervised, fully funded, individual 30 min gym-based physiotherapy sessions for 12 weeks, commencing at 6 weeks post-ACLR. Adding menstrual cycle-based phase timing (follicular-focused resistance training) did not significantly enhance knee strength or patient-reported outcomes compared to usual rehabilitation. A follow-up of outcomes at 4, 5, 6 months and 1 year was advised within the *Menstrual Cycle Phase-Based ACLR Rehabilitation Guide for Physiotherapists* located in Appendix A	7/10
Dias et al. (2024)	Randomised, controlled, single-blind, within-subject, repeated-measures trial	20 females 18–35 years (mean age 26 years, SD ± 4.5, with a self-reported menstrual cycle lasting 21–35 days. Females were excluded if on hormonal contraception, on any medication which could influence body composition, engaging in regular physical activity or injury preventing physical activity	Determine if there are changes in muscle cross-sectional area during two menstrual cycles may not be exclusively attributed to resistance training	Following resistance training protocol, which consisted of leg press and leg extension, resistance training increased muscle cross-sectional area. Muscle size also fluctuated across menstrual cycle phases, even in the non-trained leg. These variations suggest that muscle hypertrophy during short-term studies might partially reflect hormonal and fluid shifts	6/10

Table 1 (continued)

Author (year)	Study type	Participant details	Study aim	Intervention and/or results	PEDro score
Pfeifer et al. (2024)	Randomised, controlled, single-blind experimental study	28 healthy females, 18–25 years, (mean age 20.8 years, SD ± 2.15), not used contraceptives in previous 6 months, in regular physical activity (<2 h/wk, no strength training), with no previous leg injuries or surgery, regular menstrual cycle	Discover if there are menstrual cycle phase differences in myofiber damage and macrophage infiltration following electrical stimulation-induced muscle injury	Maximal voluntary isometric contraction was performed on a Biodex Dynamometer, while electrical stimulation was tested on the quadriceps. Other assessments included soreness, blood draw and CK analysis, oestradiol analysis, biopsy analysis of the vastus lateralis, and immunohistochemistry of this muscle biopsy High oestrogen may increase susceptibility to muscle injury. Low oestrogen supports greater macrophage infiltration, potentially facilitating more robust inflammatory and regenerative responses	7/10

determinants of muscle adaptation, such as protein turnover and strength development [44, 45]. Although small, non-significant trends may occur across phases, these do not appear sufficient to drive meaningful differences in training outcomes. This indicates that consistent resistance training remains the dominant stimulus for strength and hypertrophy, with hormonal phase exerting only a modest modulatory effect rather than a primary determinant of adaptation.

Both found significant improvements in aerobic endurance, measured by time to exhaustion, following increases in testosterone (21.1 s and 21.2 s, respectively) [33, 48]. Hirschberg et al. [48] also reported a significant gain in lean mass (+ 398 g, $p \leq 0.05$); however, neither study demonstrated improvements in maximal strength, as measured by knee extension torque ($p = 0.267$). Collectively, these findings suggest that elevations in testosterone may enhance aerobic capacity and body composition, specifically lean tissue accretion, rather than maximal strength in females. The observed improvements in time to exhaustion and modest gains in lean muscle mass suggest that testosterone may support metabolic efficacy and endurance capacity, yet its influence on strength output appears limited. This reinforces the notion that hormonal variations alone are insufficient to drive substantial strength adaptation; therefore, more research is needed into the direct effect female hormones can have on strength throughout the phases of the menstrual cycle.

Performance and rehabilitation

O'Loughlin [46] compared rehabilitation outcomes following ACL reconstruction following usual care and a menstrual cycle-based protocol (MCPBR). While between-group differences in strength were not statistically significant, the MCPBR group showed higher limb symmetry (81.2% vs 73.5%) and greater knee extension strength in both limbs. Strength improved over time in both groups ($p \leq 0.05$), suggesting a general training effect. These findings suggest that although overall training effects account for most rehabilitation gains, incorporating hormone-informed timing could enhance functional outcomes and interlimb balance, both of which are critical for safe return to sport. Thus, menstrual cycle-based rehabilitation represents a promising, individualised strategy that warrants further investigation in larger, more controlled trials.

Discussion

Hormone testing

Across the reviewed literature, a wide range of methods were employed to determine menstrual cycle phase and measure hormone concentrations, each with distinct advantages and limitations. Serum-based hormone analysis emerged as the most robust and reliable approach, particularly when combined with objective ovulation-detection methods, such as urinary LH kits [41, 44, 45]. These methods enabled precise phase classification and detailed hormonal profiling, particularly for oestradiol, progesterone, and testosterone. The use of liquid chromatography–tandem mass spectrometry (LC–MS/MS) is considered the gold standard for testosterone measurement, which provides high analytical specificity and sensitivity, while accompanying electrochemiluminescence assays for follicular stimulating hormone (FSH), luteinising hormone (LH), and sex hormone binding globulin (SHBG) further strengthened methodological rigour [48]. In contrast, Dias et al. [42] tested using salivary hormone assays, a non-invasive and practical alternative, especially for field-based or large-scale studies, but were limited by lower hormone concentrations and susceptibility to variability from saliva flow rate, pH, and collection techniques. Despite reporting acceptable intra-assay precision (CV: 8.8% for E2; 4.9% for P4), salivary methods remain less reliable than serum assays. Serum-based assays, particularly when paired with ovulation confirmation and LC–MS/MS, provide the most accurate representation of hormonal fluctuations, thereby enhancing confidence in phase-specific analyses. In contrast, the salivary method, while more practical for applied or large-scale settings, can introduce greater variability.

Urinary ovulation kits and basal body temperature tracking provided accessible, low-cost phase estimation tools, yet their accuracy varied with user compliance and interpretation [42, 46, 47]. Self-tracking methods such as menstruation diaries, symptom logs, and app-based calendars [41, 44, 45] were frequently used to guide or complement sampling but carried risks of recall bias and phase misclassification when not biologically validated [41, 44, 45].

The most methodologically rigorous studies integrated multiple approaches combining self-reports with ovulation kits and confirmatory serum testing to enhance phase accuracy and hormonal insight. However, it is important to acknowledge the misclassification risks which cannot be controlled, for example, the LH surge, oestradiol peak, or rapid luteal progesterone rise, as outcomes are time sensitive to these short hormonal windows which could pose a threat to the validity of research findings. The variability in methods across studies, highlighted in Sects. "Knee joint laxity" and "Neuromuscular function", emphasises the need for validated, multimodal assessment approaches in future research to more accurately examine hormone performance relationships in females.

Knee joint laxity

Notably, only one study by Pfeifer et al. [47] explicitly mentioned the commonly held assumption that elevated oestrogen levels primarily increase injury risk through heightened tissue laxity within the discussion. While acknowledging that high oestrogen phases could lead to increased joint laxity and instability, Pfeifer et al. [47] reported that their finding suggest that the late follicular phase may confer a vulnerability to the muscle myofibers themselves. Thus, highlighting a significant gap in current research, despite the widespread speculation about hormone-mediated laxity.

Neuromuscular function

The influence of female sex hormones on neuromuscular function and muscular strength appears to be modest and context dependent, with consistent evidence across studies indicating that hormonal fluctuations alone do not significantly enhance strength but may modulate training responsiveness. In intervention studies involving exogenous testosterone administration, significant improvements in aerobic performance (e.g. time to exhaustion) were observed, but no meaningful increases in strength, as measured by isokinetic knee extension torque ($p \geq 0.26$ in both cases) [43, 48]. These findings suggest that although testosterone, a key female sex hormone, appears to support lean muscle gains (with testosterone group increasing lean mass by 923 g compared to only 135 g in the placebo group, $p = 0.040$) and improve endurance performance (evidenced by a 15.2 W increase in Wingate average power in the testosterone group), it does not directly enhance maximal strength output during short duration efforts such as maximal muscle strength [48]. These findings suggest that female sex hormones play a supportive rather than primary role in muscular strength. While hormones, such as testosterone, may enhance lean muscle mass and endurance, they do not directly increase maximal strength. However, studies examining natural hormonal fluctuations across the menstrual cycle report that muscle protein synthesis (MPS) and muscle strength are not significantly altered by menstrual phase [45, 47]. However, there were no differences in MPS or MPB between the follicular and luteal phases, indicating that strength-relevant metabolic processes are not hormone-phase dependent [45]. When investigating the integrated exercise approach of bending, roll-ups, arm swings with loads, crunches, tandem walks, and squats (BRACTS), it suggests that strength varies across phases of the menstrual cycle, with quadriceps strength increased in exercise intervention groups, although more research is needed into this method to provide validity. However, the interaction between hormones and resistance training tells a more nuanced story [44]. The MCPBR found that menstrual cycle-based resistance training produced higher (though not statistically significant) knee extension 1RM and LSI compared to usual care [46]. Dias et al. [42] further demonstrated that muscle CSA increased significantly during later cycle phases (ovulation, luteal and menses) in the resistance training group, but not earlier phases or in controls. However, the studies fail to explain how later phases of the menstrual cycle might enhance the responsiveness of oestrogens role in reducing inflammation and muscle repair and progesterone's effect on ligament laxity and why psychological readiness is enhanced. Thus, there are conflicting views in the research as to why hormonal environments during the mid-luteal and ovulatory phases may enhance the muscle's responsiveness to training.

Taken together, these findings highlight a possible distinction between hormonal influence on baseline strength and hormonal modulation of training adaptations. For instance, an athlete might maintain similar maximal strength throughout the menstrual cycle but experience greater exercise-induced hypertrophy when resistance sessions are more concentrated in the mid-luteal or ovulatory phase.

Female sex hormones do not appear to significantly alter strength in the absence of training, but their timing may influence the effectiveness of resistance training, especially during phases with favourable hormonal profiles. Therefore, while strength gains are primarily driven by exercise, aligning training with specific menstrual phases could optimise adaptation in some females.

Performance

The relationship between the menstrual cycle hormones and neuromuscular performance remains complex and subtle. Oestrogen is understood to have neuro-excitatory properties that could enhance central nervous system activation and muscle firing efficiency [47]. However, evidence on consistent neuromuscular performance enhancement across the menstrual cycle remains mixed.

Acute hormonal responses to training, such as transient increases in testosterone, may positively increase neuromuscular drive. Exercise-induced surges in total testosterone occurred across all phases of the menstrual cycle, with the highest absolute levels during mid-cycle. These acute hormonal shifts have been associated with enhanced motor output and neuromuscular responsiveness in other contexts, though the effect in eumenorrheic females appears transient and phase dependent [43]. However, randomised trials examining phase-based training interventions failed to show a statistically significant neuromuscular strength advantage from follicular phase-targeted rehabilitation [46]. Likewise, Colenso-Semple et al. [45] found that resistance exercise stimulated muscle protein synthesis equally in both follicular and luteal phases, suggesting that the neuromuscular adaptations to training were not constrained by menstrual cycle phase.

While hormonal fluctuations can exert subtle, phase-specific effects on neuromuscular performance, the core capacity for motor output and adaptation to resistance training remains robust across the menstrual cycle. Oestrogen and testosterone may transiently enhance neural activation, but there is no consistent evidence suggesting major constraints or advantages tied to any one phase.

The influence testosterone has on females' performance as distinct patterns emerge depending on the nature of hormonal modulation, whether exogenous or endogenous, and the type of performance outcome measured. Testosterone supplementation study provides the strongest evidence for a causal relationship between increased serum testosterone and aerobic performance enhancement, reporting a 4.8-fold rise in testosterone in the experimental group (to 4.3 nmol/L) that led to a 6.2% improvement in time to exhaustion and gains in maximal aerobic speed (MAS) [48]. These results suggest central or peripheral adaptations, such as enhanced cardiac output or oxidative capacity, may mediate testosterone's ergogenic effects. In contrast, endogenous hormonal fluctuations and found significant testosterone variation across menstrual phases, with peak levels 15 min post-exercise during the mid-cycle phase (from 36.48 ng/dl $\pm$ 2.80 at pre exercise to 40.80 ng/dl $\pm$ 7.12 post-intervention) compared to the follicular phase (25.80 ng/dl $\pm$ 2.57 at pre exercise to 33.04 ng/dl $\pm$ 8.67 post-intervention) and the luteal phase (31.10 ng/dl $\pm$ 3.44 at pre exercise to 34.97 ng/dl $\pm$ 5.60 post-intervention), supporting the effect testosterone can have on performance in females [44]. These acute responses, absent in control participants, suggest a transient hormonal window that may be advantageous for recovery or performance adaptation, though no direct link to measurable performance outcomes was established. Meanwhile, Smith et al. [41] conducted an intervention study which highlights that aerobic fitness improvements (increase of 0.90 METs) and favourable changes in body composition occurred in the absence of significant changes in key sex hormones, apart from a reduction in oestrone sulphate and a minor, statistically comparable decrease in progesterone. This indicates that exercise stimulus, rather than hormonal fluctuations, is the primary driver of cardiovascular and metabolic adaptation. The minor reductions observed in oestrone sulphate and progesterone likely reflect secondary physiological adjustments rather than causal mechanisms; however, further research is needed to support the extent of this.

Notably, all three studies converge on the finding that anaerobic performance shows limited responsiveness to both testosterone supplementation and natural hormonal variation, as recognised by the research itself. A conclusion based on a short intervention duration, making it difficult to capture the long-term hormonal adaptations. Therefore, while exogenous testosterone can directly enhance aerobic capacity, endogenous hormonal rhythms may play a more subtle, modulatory role, and anaerobic performance remains largely unaffected by either mechanism [44, 48]. These contrasting findings highlight the need for future research to determine whether other hormones, such as oestrogen or progesterone, as well as hormone-driven versus training-driven performance adaptations, are associated with these outcomes, and to ensure that both hormonal context and performance domain are considered when interpreting results in female athletes.

Rehabilitation considerations

The study by O'Loughlin et al. [46] provides preliminary evidence that aligning rehabilitation with phases of the menstrual cycle, through menstrual cycle-based progressive resistance training (MCPBR), may enhance strength recovery following ACL reconstruction. Participants in the MCPBR group demonstrated greater improvements in knee extension 1RM and limb symmetry index (LSI) compared to usual care. However, the lack of statistically significant between-group differences and the presence of a general time effect ($p \leq 0.05$) indicate that strength gains were primarily driven by consistent training over time, rather than the timing of training alone.

While MCPBR is not yet supported as a standard protocol, these findings suggest that menstrual cycle phase could be a meaningful variable in optimising rehabilitation outcomes for female athletes. Professionals should consider individual hormonal profiles or cycle timing when designing rehabilitation or performance programmes.

Limitations of the evidence base

Across eight studies, the most consistent limitation was a small sample size with seven studies having a sample of 50 participants or less, this restricts the statistical power and generalisability of the research [42–48]. Another universal issue was short intervention or observation windows, ranging from acute tracer or exercise protocols [44, 45] to slightly longer interventions of only 10–16 weeks [41, 48]. The short duration of the studies limits the ability to observe long-term hormonal or performance adaptations, highlighting the need for future research to move beyond the current field-wide reliance on short-term designs. Similarly, studies show inconsistent results and sometimes limited hormonal profiling. While some studies use robust hormone assays [42, 47, 48], others relied on salivary testing [42] or some only measured a single hormone, testosterone [43]. Moreover, phase verification was inconsistent, ranging from multimodal strategies [45, 47] to self-reported cycle tracking [43, 44]. This introduces risks of phase misclassification, particularly when trying to capture finite hormonal surges, for example. Finally, the findings are restricted to young, healthy, eumenorrheic females and therefore lack generalisability. Several studies further reduced generalisability by narrowing inclusion criteria to very specific groups, for example, sedentary females in Smith et al. [41] or ACL reconstructed patients in O'Loughlin et al. [46]. Overall, the evidence is constrained by small, underpowered samples, short-term interventions, inconsistent hormonal profiling and limited generalisability, thus reducing confidence in the findings and limiting comparability across studies.

Practical implications

Despite methodological limitations, several practical implications emerge for athletes, coaches, and clinicians. Evidence suggests anaerobic performance and maximal strength are largely unaffected by short-term hormonal fluctuations, allowing consistent training across the menstrual cycle [44, 45, 47, 48]. In contrast, aerobic capacity and recovery may be influenced by hormones, particularly testosterone and oestrogen, indicating endurance training could benefit from alignment with mid-cycle phases when these hormones peak [41, 44, 48].

From a rehabilitation perspective, menstrual cycle-informed strategies may improve adaptation post-injury, especially ACL reconstruction, where knee laxity and neuromuscular control are critical [46]. While evidence remains preliminary, these findings highlight the need to consider aerobic demands, injury risk, and individual symptom patterns when designing progressive resistance and neuromuscular programmes for female athletes.

Future research directions

This review identifies key gaps in the literature and outlines a framework to advance research on the effects of female sex hormones on knee laxity, strength, neuromuscular performance, and aerobic/anaerobic capacity. Future studies should prioritise methodological refinement and deeper exploration of performance outcomes to improve understanding of strength adaptations and inform rehabilitation strategies.

Hormone testing remains essential. Standardising sampling protocols is critical to enhance cross-study comparability. Menstrual cycle phase should be validated using multimodal approaches that combine serum analysis, ovulation kits, and self-tracking techniques. Longer-term interventions should integrate hormone monitoring with physiological markers to determine whether aerobic and anaerobic performance is truly hormone sensitive.

Longitudinal studies tracking muscle hypertrophy and strength across multiple menstrual phases are needed to distinguish acute hormonal effects from cumulative training adaptations, as the current research does not clearly establish hormones' direct impact on baseline strength. Furthermore, mechanistic insights into oestrogen's role in muscle repair and inflammation versus progesterone's effect on connective tissue laxity could support rehabilitation strategies aligned with menstrual phases.

Synthesis in relation to the aim of the review

This review aimed to evaluate the effects of female sex hormones on knee laxity, neuromuscular function, and performance by examining patterns in the literature, methodological strengths and weaknesses, and implications for training, rehabilitation, and competition. Evidence suggests hormonal influence is inconsistent. Aerobic capacity appears most sensitive, with exogenous testosterone significantly enhancing performance [43, 48]. This indicates aerobic training may benefit from mid-cycle alignment, while anaerobic and strength training can occur across all phases [45, 46].

Findings on neuromuscular function and muscle adaptation are mixed: Colenso-Semple et al. [45] report no phase-based differences in muscle protein synthesis, whereas Dias et al. [42] and O'Loughlin et al. [46] suggest improved rehabilitation outcomes during mid-cycle. Overall, baseline neuromuscular function seems stable, but hormones may influence training responsiveness and recovery. Menstrual cycle-based rehabilitation strategies could support ACL recovery, though further evidence is needed.

Conclusion

The current evidence supports the impact female sex hormones can have on performance, as aerobic function and recovery appear to be more hormone-sensitive than anaerobic performance or maximal strength, and training responsiveness may be phase modulated. There was no consistent evidence to conclude that menstrual hormones meaningfully affect joint laxity. Overall, the scientific evidence is still inconclusive, underpowered, and inconsistent due to the nature of the method. Emerging clinical implications include tailoring endurance training, monitoring injury risk, and exploring cycle-informed rehabilitation. Robust, long-term studies are required to confirm these effects and guide evidence-based practice.

Acknowledgements The corresponding author would like to express sincere gratitude to the supervisors, Dr Christopher Holland and Dr Darren Cooper, for their invaluable guidance and support throughout the development of this systematic review. The corresponding author is also grateful to The University of Worcester for providing access to the databases and resources necessary to conduct this review.

Author contributions The main manuscript was written by HW with the supervisors, CH and DC overseeing the research.

Funding This review was not funded.

Data availability No datasets were generated or analysed during the current study.

Declarations

Conflict of interest The authors declare no competing interests.

References

1. Daitch V, Turjeman A, Poran I, Tau N, Ayalon-Dangur I, Nashashibi J et al (2022) Underrepresentation of women in randomized controlled trials: a systematic review and meta-analysis. Trials 23(1):1038
2. Gribble KD, Callaway L, Raven M, Smith JP, Gamble J, Dahlen HG (2025) Sex, gender identity and women's health research and equality: an urgent need for clarity of language and accurate data collection. Women Birth 38(1):101854
3. Costello JT, Bieuzen F, Bleakley CM (2014) Where are all the female participants in sports and exercise medicine research? EurJ Sport Sci 14(8):847–851
4. Beech J, Jones B, Hughes T, Emmonds S (2024) Injury profile in youth female athletes: a systematic review and meta-analysis. Sports Med 54(5):1207–1230
5. Bunstine JL, Yang J, Kistamgari S, Collins CL, Smith GA (2024) Differences in overuse injuries in gender-comparable sports: a nationally representative sample of high school athletes. J Athl Train 59(9):962–968
6. Hornblower S, Spawforth A, Eidinow E (eds) (2012) The Oxford classical dictionary. Oxford University Press
7. Raysmith BP, Jacobsson J, Drew MK, Timpka T (2019) What is performance? A scoping review of performance outcomes as study endpoints in athletics. Sports 7(3):66
8. Lebrun CM, McKenzie DC, Prior JC, Taunton JE (1995) Effects of menstrual cycle phase on athletic performance. Med Sci Sports Exerc 27(3):437–444
9. Raj RD, Fontalis A, Grandhi TS, Kim WJ, Gabr A, Haddad FS (2023) The impact of the menstrual cycle on orthopaedic sports injuries in female athletes. Bone Joint J 105(7):723–728
10. Ostrander EA, Huson HJ, Ostrander GK (2009) Genetics of athletic performance. Annu Rev Genomics Hum Genet 10(1):407–429
11. Guarneri AM, Kamboj MK (2019) Physiology of pubertal development in females. Pediatr Med. https://doi.org/10.21037/pm.2019.07.03

12. McNulty KL, Elliott-Sale KJ, Dolan E, Swinton PA, Ansdell P, Goodall S et al (2020) The effects of menstrual cycle phase on exercise performance in eumenorrheic women: a systematic review and meta-analysis. Sports Med 50(10):1813–1827
13. Sherman BM, Korenman SG (1975) Hormonal characteristics of the human menstrual cycle throughout reproductive life. J Clin Invest 55(4):699–706
14. Pache TD, Wladimiroff JW, de Jong FH, Hop WC, Fauser BC (1990) Growth patterns of nondominant ovarian follicles during the normal menstrual cycle. Fertil Steril 54(4):638–642
15. Herbison AE (2020) A simple model of estrous cycle negative and positive feedback regulation of GnRH secretion. Front Neuroendocrinol 57:100837
16. Thiyagarajan DK, Basit H, Jeanmonod R (2024) Physiology menstrual cycle. In: StatPearls. Treasure Island (FL), StatPearls Publishing.
17. Atukorala KR, Silva W, Amarasiri L, Fernando DM (2022) Changes in serum testosterone during the menstrual cycle—an integrative systematic review of published literature. Gynecol Reprod Endocrinol Metab 3:9–20
18. Reed BG, Carr BR (2015) The normal menstrual cycle and the control of ovulation.
19. Mihm M, Gangooly S, Muttukrishna S (2011) The normal menstrual cycle in women. Anim Reprod Sci 124(3–4):229–236
20. Munro MG, Critchley HO, Fraser IS, Haththotuwa R, Kriplani A, Bahamondes L et al (2018) The two FIGO systems for normal and abnormal uterine bleeding symptoms and classification of causes of abnormal uterine bleeding in the reproductive years: 2018 revisions. Int J Gynaecol Obstet 143(3):393–408
21. Fehring RJ, Schneider M, Raviele K (2006) Variability in the phases of the menstrual cycle. J Obstet Gynecol Neonatal Nurs 35(3):376–384
22. Mittiku YM, Mekonen H, Wogie G, Tizazu MA, Wake GE (2022) Menstrual irregularity and its associated factors among college students in Ethiopia, 2021. Front Glob Womens Health 3:917643
23. Farage MA, Neill S, MacLean AB (2009) Physiological changes associated with the menstrual cycle: a review. Obstet GynecolSurv 64(1):58–72
24. Farage MA, Osborn TW, MacLean AB (2008) Cognitive, sensory, and emotional changes associated with the menstrual cycle: a review. Arch Gynecol Obstet 278(4):299–307
25. Itriyeva K (2022) The normal menstrual cycle. Curr Probl Pediatr Adolesc Health Care 52(5):101183
26. Weidauer L, Zwart MB, Clapper J, Albert J, Vukovich M, Specker B (2020) Neuromuscular performance changes throughout the menstrual cycle in physically active females. J Musculoskelet Neuronal Interact 20(3):314
27. Bermon S, Garnier PY (2017) Serum androgen levels and their relation to performance in track and field: mass spectrometry results from 2127 observations in male and female elite athletes. Br J Sports Med 51(17):1309–1314
28. Pollard CD, Braun B, Hamill J (2006) Influence of gender, estrogen and exercise on anterior knee laxity. Clin Biomech 21(10):1060–1066
29. Heitz NA, Eisenman PA, Beck CL, Walker JA (1999) Hormonal changes throughout the menstrual cycle and increased anterior cruciate ligament laxity in females. J Athl Train 34(2):144
30. Shultz SJ, Sander TC, Kirk SE, Perrin DH (2005) Sex differences in knee joint laxity change across the female menstrual cycle. J Sports Med Phys Fitness 45(4):594
31. Shagawa M, Maruyama S, Sekine C, Yokota H, Hirabayashi R, Hirata A et al (2021) Comparison of anterior knee laxity, stiffness, genu recurvatum, and general joint laxity between late follicular and ovulatory phases. BMC MusculoskeletDisord 22(1):886
32. Park SK, Stefanyshyn DJ, Loitz-Ramage B, Hart DA, Ronsky JL (2009) Changing hormone levels during the menstrual cycle affect knee laxity and stiffness in healthy female subjects. Am J Sports Med 37(3):588–598
33. Namie T, Yoshiaki K, Takakuni S (2022) Influence of menstrual cycle on the relationship between joint laxity and dynamic balance ability [dissertation]. Tokyo Ariake Univ Med Health Sci, Tokyo
34. Ansdell P, Brownstein CG, Škarabot J, Hicks KM, Simoes DC, Thomas K et al (2019) Menstrual cycle-associated modulations in neuromuscular function and fatigability of the knee extensors in eumenorrheic women. J Appl Physiol 126(6):1701–1712
35. Piasecki J, Guo Y, Jones EJ, Phillips BE, Stashuk DW, Atherton PJ et al (2023) Menstrual cycle associated alteration of vastus lateralis motor unit function. Sports Med Open 9(1):97
36. Sarwar R, Niclos BB, Rutherford OM (1996) Changes in muscle strength, relaxation rate and fatiguability during the human menstrual cycle. J Physiol 493(1):267–272
37. Constantini NW, Dubnov G, Lebrun CM (2005) The menstrual cycle and sport performance. Clin Sports Med 24(2):e51-62
38. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, The PRISMA et al (2020) statement: an updated guideline for reporting systematic reviews. BMJ 2021:372
39. Cashin AG, McAuley JH (2020) Clinimetrics: physiotherapy evidence database (PEDro) scale. J Physiother 66(1):59
40. Matos AP, Pegorari MS (2020) How to classify clinical trials using the PEDro scale? J Lasers Med Sci 11(1):1
41. Smith AJ, Phipps WR, Arikawa AY, O'Dougherty M, Kaufman B, Thomas W et al (2011) Effects of aerobic exercise on premenopausal sex hormone levels: results of the WISER study. Cancer Epidemiol Biomarkers Prev 20(6):1098–1106
42. Dias NF, Bergamasco JG, Scarpelli MC, Silva DG, Chaves TS, Bittencourt D et al (2024) Changes in muscle cross-sectional area during two menstrual cycles may not be exclusively attributed to resistance training. Appl Physiol Nutr Metab 49(12):1729–1739
43. Shahid W, Noor R (2025) Effects of integrated exercise approach on total testosterone levels in eumenorrheic women: a randomized controlled trial. Sci Rep 15(1):15692
44. Shahid W, Noor R (2025) Impact of BRACTS exercises on muscular strength in eumenorrheic women. Sci Rep 15(1):4430
45. Colenso-Semple LM, McKendry J, Lim C, Atherton PJ, Wilkinson DJ, Smith K et al (2025) Menstrual cycle phase does not influence muscle protein synthesis or whole-body myofibrillar proteolysis in response to resistance exercise. J Physiol 603(5):1109–1121
46. O'Loughlin E, Reid D, Sims S, Larsen P (2024) The effect of menstrual cycle phase-based rehabilitation for females following ACL reconstruction: a randomized controlled trial. N Z J Physiother 52(3):219–35
47. Pfeifer B, King B, Ahmadi M, Kaluhiokalani JP, Shimizu KS, Hunter WN et al (2024) Menstrual cycle phase differences in myofiber damage and macrophage infiltration following electrical stimulation-induced muscle injury. Am J PhysiolEndocrinol Metab 327(5):E616–E625
48. Hirschberg AL, Knutsson JE, Helge T, Godhe M, Ekblom M, Bermon S et al (2020) Effects of moderately increased testosterone concentration on physical performance in young women: a double-blind, randomised, placebo-controlled study. Br J Sports Med 54(10):599–604