Chapter 11
The Effect of the Menstrual Cycle on Exercise and Sports Performance

Kirsty M. Hicks, Kelly McNulty, and Paul Ansdell

Abbreviations

FSH	Follicle-stimulating hormone
LH	Luteinizing hormone
MVC	Maximal voluntary contraction
RER	Respiratory exchange ratio
$\dot{V}E$	Ventilatory rate
$\dot{V}O_2$	Volume of oxygen consumption
$\dot{V}CO_2$	Volume of carbon dioxide produced
$\dot{V}O_{2peak}$	Peak oxygen consumption

Introduction

Women's participation in sport and exercise is increasing, with full gender equivalence expected to be achieved for the first time in the history of the Olympic Games in Paris in 2024. Therefore, for women athletes and those working with them, it is important that any potential modulations, either positive or negative, in performance, training, and adaptation can be understood and optimized. Research-informed exercise prescription aims to achieve optimal performance for an individual or group of athletes, based on available evidence. However, despite the surge in women's sport and exercise participation and subsequently, financial investment, media coverage, and sponsorship deals, women remain underrepresented in the sport and exercise science literature (Cowley et al. 2021). Consequently, it is not uncommon for the

K. M. Hicks (✉) · K. McNulty · P. Ansdell
Department of Sport, Exercise and Rehabilitation, Northumbria University, Newcastle Upon Tyne, UK
e-mail: kirsty.hicks@northumbria.ac.uk

A. C. Hackney (ed.), *Sex Hormones, Exercise and Women*,
https://doi.org/10.1007/978-3-031-21881-1_11

understanding and prescription of exercise to be based on data from men, which is concerning given the known differences between men and women and their physiological responses to exercise (Ansdell et al. 2020). Indeed, the most notable biological sex difference is the menstrual cycle; during which, there are significant fluctuations in endogenous sex hormones; namely estrogen and progesterone. As this chapter outlines, understanding the effects of variable concentrations of estrogen and progesterone is important, as they potentially exert an influence on the underpinning components of exercise performance (i.e., cardiovascular function, respiratory function, substrate metabolism, and neuro-muscular function). Performance can be quantified in various ways; however, for the purpose of this chapter, exercise performance was defined as force/torque production, power output, time to exhaustion, and time to completion.

In light of the above, the specific aims of this chapter are as follows:

(1) to describe the fluctuations in sex hormones that underpin the menstrual cycle;
(2) to discuss physiological mechanisms that might affect exercise performance across the menstrual cycle;
(3) to summarize the literature regarding the impact of the menstrual cycle on exercise performance;
(4) to provide practical implications and future recommendations for women athletes, and those working with them.

The Menstrual Cycle

A menstrual cycle lasts between 21 and 35 days, with an average cycle length commonly being reported as 28 days. The primary purpose of the menstrual cycle is to prepare a woman's body for reproduction, which is broadly achieved through naturally occurring changes in circulating endogenous hormones and structural changes to the endometrial lining. The menstrual cycle is regulated by hormones released by the hypothalamus (gonadotropin-releasing hormone, GnRH), pituitary gland (luteinizing hormone [LH] and follicle-stimulating hormone [FSH]), and the ovaries (estrogen and progesterone). Often, the concentrations and/or ratio of these hormones are used to separate the menstrual cycle into specific phases. Due to complexities within applied practice and research, for simplicity the menstrual cycle is often divided into two phases: (1) the follicular phase (from day 1 of menses to ovulation) and (2) the luteal phase (post-ovulation to the start of the following menses). However, this two-phase division is an oversimplification, as it does not capture all time points whereby circulating concentrations of estrogen and progesterone, and the ratios between the two, vary substantially. To achieve this, the menstrual cycle can more accurately be separated into four phases (Elliott-Sale et al. 2021): (1) the early-follicular phase, the onset of bleeding to day five, during which estrogen and progesterone are low; (2) the late-follicular phase, < 26 h pre-ovulation, whereby estrogen peaks but progesterone remains low (< 6.4 nmol·L^{-1}); (3) ovulation, 24–36 h after a positive ovulation test, when estrogen is high and progesterone is still low (< 6.4 nmol·L^{-1}); and

(4) the mid-luteal phase, occurring seven to nine days after a positive ovulation test, during which both estrogen and progesterone are high. Importantly, these time points allow for three key hormone ratios (low:low, high:low, and high:high) of estrogen and progesterone to be investigated. To achieve a consensus on the taxonomy used to describe these time points, and the criteria for each menstrual cycle phase, the interested reader is directed to the work of Elliott-Sale et al. (2021).

It is important to note that the menstrual cycle can be subject to internal (i.e., anovulatory, oligomenorrhea, amenorrhea) and external (i.e., hormonal contraceptive use) modulations, adding further complexity to the support provided to, and research conducted on, women athletes. Internal permutations could be indicative of menstrual dysfunction and should be discussed with appropriate medical professionals (Davison et al. (2022). External manipulation, however, is common, with almost half of the women athletic population studied using hormonal contraceptives (Martin et al. 2018). Hormonal contraceptives downregulate fluctuations in endogenous sex hormones resulting in a significantly different hormonal profile compared to that of the naturally occurring menstrual cycle. Therefore, given the varying hormonal statuses of women athletes, it is pertinent to understand how both internal and external modulations of the menstrual cycle affect exercise performance. While Elliott-Sale and Hicks (2018) discuss external modulations of the menstrual cycle and exercise performance, the current chapter focuses on the internal modulations of endogenous hormones and exercise performance.

The Menstrual Cycle and Physiological Function

Sex hormone receptors mediate the biological and physiological processes of estrogen and progesterone. In addition to the reproductive system, estrogen (ERα and ERβ) and progesterone (hPR-A and hPR-B) receptors are expressed within a multitude of tissues and organs, including musculoskeletal tissues such as bone, ligaments, and tendons, as well as the cardiovascular, respiratory, and central nervous systems. The expression of sex hormone receptors is of importance as the functions of the aforementioned physiological systems are fundamental to exercise performance. Constantini et al. (2005) compiled a network of components of exercise performance, where the effect of the menstrual cycle might exert its influence; this interaction commonly provides the rationale for subsequent implications on strength, aerobic and anaerobic performance. However, in addition to physiology, exercise performance is a multifaceted process (e.g., motivation, competition, etc.) with many external and internal nuances; thus, it is not surprising that the research investigating the effects of the menstrual cycle on exercise performance remains equivocal. This section discusses the proposed influence of the menstrual cycle on key physiological systems involved in exercise performance.

Mechanisms Potentially Altering Strength Performance Across the Menstrual Cycle

Force generation, commonly referred to as strength, is not only a determinant of sporting performance, but is altered by exercise in the form of fatigue, muscle damage, and recovery. The ability to produce force is a sequence of chemical and protein interactions from the propagation of nervous system impulses along the motor pathway to the contractile proteins within the skeletal muscle (Dulhunty 2006). As detailed below, estrogen and progesterone exert their influences at several segments of the motor pathway, making it difficult to isolate the individual mechanisms underpinning potential influences on strength.

Beginning within the motor cortex, estrogen and progesterone are both neurosteroids with contrasting impacts; estrogen increases cortical excitability (Smith et al. 1999), whereaBembens progesterone enhances cortical inhibition (Smith et al. 1999). More recently, Ansdell et al. (2019) demonstrated the link between hormone-induced changes in excitability and voluntary activation of the knee extensors. Specifically, around ovulation (day 14 of the menstrual cycle) the neuro-excitatory effects of high-estrogen concentrations were observed alongside an increased voluntary activation of the knee extensors. Furthermore, during the mid-luteal phase, cortical inhibition was increased, alongside a concomitant decrease in voluntary activation, highlighting the neuro-inhibitory effect of progesterone. Thus, the hormonal influence on central nervous system function might influence muscle function. At the other end of the motor pathway, previous work has proposed that changes in motor unit firing rates across the menstrual cycle might be a potential mechanism which underpins changes in maximal strength (Tenan et al. 2016). Tenan et al. (2013) reported greater motor unit discharge rates at recruitment (~10%) in the latter half of the menstrual cycle, which taken together with the findings of Ansdell et al. (2019) could suggest that the changes in pre-synaptic properties (i.e., cortical excitability) modulate post-synaptic output (i.e., motor unit discharge rate) within the motor pathway.

In addition to influences within the central nervous system, estrogen has been reported to improve strength due to its effects on the binding of calcium and troponin C and contractile proteins, such as myosin. Myosin consists of two heavy chains and two pairs of light chains. These pairs are known as the essential light chain and the regulatory light chain and are both pertinent to muscle contractions; the former is considered to act as a structural stabilizer, whereas the latter acts as a lever arm which results in the movement of myosin and force production (Rayment et al. 1993). Estrogen has also been reported to modulate phosphorylation of the regulatory light chain (Lai et al. 2016); this has functional consequences, as phosphorylation of the regulatory light chain erects the essential light chain from the thick filament, positioning the myosin heads for optimal actin-myosin interaction (Levine et al. 1996). The influence of estrogen on contractile proteins during active muscle contractions is suggested to promote a stronger binding of the myosin head to actin, thus enhancing force production (Lowe et al. 2010). These suggestions are supported by a reduction in the fraction of strong-binding myosin during active

contractions occurring to the same extent as maximal isometric tetanic force loss in ovariectomized mice (Moran et al. 2006). Whereas estrogen replacement reversed decrements in maximal isometric tetanic force and increased the fraction of strong-binding myosin during active contractions (Moran et al. 2007). Additionally, in six pairs of postmenopausal monozygotic female human twins, active stiffness, a marker of the number of strongly attached cross-bridges, in type IIa muscle fibers was higher in the female twin receiving hormone replacement therapy compared to the female twin not using hormone replacement therapy (Qaisar et al. 2013). Therefore, it is suggested that estrogen might enhance strength performance by improving the intrinsic quality of contractile function (Lowe et al. 2010) but any potential effects might be fiber-type dependent (Qaisar et al. 2013). Although the body of evidence is predominantly within animal studies or in vitro settings, estrogen's influence on the function of myosin could be speculated to underpin the potential influence of estrogen on muscle strength (Collins et al. 2019). Although the role of progesterone has been recognized, possibly by acting synergistically with estrogen (Greeves 2000), the mechanistic effect of progesterone on muscle function remains relatively understudied and further research is required (Kim et al. 2016). Nonetheless, it is evident that estrogen, however, might influence components of muscle strength but whether this translates to changes in measures of strength performance is unclear.

Measures of Strength Performance Across the Menstrual Cycle

Within the body of literature, there is a substantial divide between research that supports (e.g., (Rodrigues et al. 2019; Sarwar et al. 1996; Tenan et al. 2016) and refutes (e.g., (Ansdell et al. 2019; Elliott et al. 2003; Janse De Jonge et al. 2001)) an effect of the menstrual cycle on measures of strength performance in humans. Sarwar et al. (1996) reported both quadricep and handgrip strength (maximal voluntary contraction; MVC) to be higher "mid-cycle" (unconfirmed ovulatory phase), compared to the early- and mid-follicular and mid- and late luteal phases of the menstrual cycle. In agreement, Bambaeichi et al. (2004) reported greater knee extensor MVC at confirmed ovulation compared to the early- and late-follicular and mid- and late luteal phases of the menstrual cycle. Although both Sarwar et al. (1996) and Bambaeichi et al. (2004) speculated that elevated estrogen levels at ovulation were conducive to strength performance, neither study subsequently verified sex hormone concentrations. A study that did address this was Janse De Jonge et al. (2001), who utilized calendar-based counting, and changes in basal body temperature, as well as serum hormone analysis and progesterone thresholds (>16 nmol.l^{-1}) to identify and verify the early-follicular, late-follicular, and mid-luteal phases of the cycle. Confident that testing occurred at distinctly different ratios of estrogen to progesterone, Janse De Jonge et al. (2001) concluded that both voluntary and electrically evoked quadriceps strength and handgrip strength did not alter across

the menstrual cycle. Likewise, using the advocated "three-step" methodological recommendation to confirm phases (Janse De Jonge et al. 2019) for menstrual cycle research, Fridén et al. (2003) and Ekenros et al. (2013) reported no differences in handgrip strength between the early-follicular, ovulatory, and mid-luteal phases of the menstrual cycle. Interestingly, however, Ekenros et al. (2013) reported knee extensor MVC to be greater in the mid-luteal phase of the menstrual cycle, a finding that contradicts earlier research such as Sarwar et al. (1996). The inconclusive effects of the menstrual cycle on strength performance could reflect high heterogeneity in study design and quality (McNulty et al. 2020). Furthermore, studies use a variety of strength measures (e.g., dynamic and isometric contractions in the upper and lower limbs), participants of differing training status (e.g., recreational to elite), and menstrual phases, making it difficult to draw direct comparisons between this literature.

Mechanisms Potentially Altering Endurance Performance Across the Menstrual Cycle

Endurance exercise requires the integration of several physiological systems and processes such as substrate metabolism, musculoskeletal function, and the cardiovascular and respiratory systems. Endogenous hormones, such as estrogen and progesterone, have been suggested to exert their influence on nearly all of these physiological mechanisms. To that end, estrogen is a vasodilator, which increases blood flow to the heart and muscles through vasodilation of the coronary, brachial arteries, and the peripheral microvascular beds (Adkisson et al. 2010; Traupe et al. 2007). Adkisson et al. (2010) partly attributed improved central hemodynamic and reactivity of peripheral vascularity during the late-follicular phase of the menstrual cycle due to an estrogen-mediated increase in nitric oxide (NO) bioavailability. Although vasodilation of the feeder arteries to the working muscle could promote greater oxygen delivery to that working muscle, the ability to capitalize on this response is still partly dependent on oxygen extraction (Wagner 2000).

Earlier in the oxygen transport pathway, hormone-mediated effects are evident. For example, Smith et al. (2015) reported an increase in lung diffusion capacity during the mid-follicular compared to the early-follicular phase of the menstrual cycle. The increase in diffusion capacity is attributed to the concurrent increase in capillary blood volume, which was speculated to be a result of estrogen increasing water retention during the luteal phase of the menstrual cycle (Smith et al. 2015). However, this study included tri-phasic oral contraceptive users within their cohort and reported no significant differences in the hormone concentrations between phases; therefore, it is difficult to attribute Smith et al. (2015) findings to fluctuations in endogenous hormone changes alone. At rest, ventilatory rate ($\dot{V}E$) has been demonstrated to be greater in the luteal phase (MacNutt et al. 2012; Schoene et al. 1981), while during exercise, several studies have shown a greater ventilatory equivalent ($\dot{V}E/\dot{V}O_2$ and

$\dot{V}E/\dot{V}CO_2$, (Dombovy et al. 1987; Schoene et al. 1981)) in the luteal phase. As discussed by Janse De Jonge (2003), these changes are likely driven by the effects of progesterone on central respiratory drive, or indirectly through the increase in body temperature. However, it is important to note that conflicting literature exists demonstrating no effect of the menstrual cycle on minute ventilation, particularly at high intensities of exercise (Beidleman et al. 1999; Bemben et al. 1995; De Souza et al. 1990). Similarly, conflicting literature exists regarding the respiratory exchange ratio (RER, i.e., $\dot{V}CO_2/\dot{V}O_2$), which is influenced by substrate utilization (Dombovy et al. 1987; Hackney et al. 1994; Kanaley et al. 1992).

Substrate metabolism is a large contributor to endurance performance, and the ability to optimize fat metabolism is deemed preferable during moderate- and heavy-intensity exercise (i.e., especially of a prolonged duration). During endurance exercise, research has demonstrated that estrogen initiates a shift from carbohydrate utilization to fat oxidation (D'Eon et al. 2002; Hackney et al. 1991, 2022); however, these findings are not always consistent (Frandsen et al. 2020; Horton et al. 2002). The genomic and non-genomic mechanisms of estrogen on substrate utilization are complex; therefore, the interested reader is encouraged to read Oosthuyse and Bosch (2012) for more information. Although fat oxidation has been reported to be highest during ovulation (Hackney et al. 1991), the anti-estrogenic effect of progesterone must be taken into account when considering substrate utilization during the menstrual cycle (D'Eon et al. 2002; Hackney et al. 2022). For example, during submaximal exercise, D'Eon et al. (2002) demonstrated that the estrogenic reduction in carbohydrate oxidation and increase in fat oxidation was reversed when high levels of exogenous estrogen and progesterone were administered. Furthermore, Hackney et al. (2022) reported an "in vivo" shift toward fat metabolism from the follicular to luteal phase of the menstrual cycle during exercise. The magnitude of this shift was greater in those women with a smaller increase in progesterone relative to estrogen in the luteal phase (Hackney et al. 2022). As such, it is plausible that the inconsistencies in research to date investigating the effect of the menstrual cycle on substrate metabolism could be attributed to focusing on the absolute hormone concentrations rather than the ratio across the menstrual cycle (Hackney et al. 2022). Therefore, although the estrogenic effect on substrate metabolism might favor endurance performance, research must consider the estrogen-to-progesterone ratio to fully elucidate the potential mechanisms and impact on endurance performance. Although estrogen and progesterone might influence several of the physiological systems involved in endurance performance, the literature regarding such exercise performance is less clear.

Endurance Performance Across the Menstrual Cycle

Typically, endurance performance can be inferred using time to exhaustion tests or time to completion (i.e., time or distance trials). Time to exhaustion tests requires the athlete to maintain a constant power output or speed to the limit of tolerance,

with no controlled endpoint. Whereas time trial performance tests require athletes to complete a set distance or workload within the shortest time possible.

Research investigating the effect of the menstrual cycle on endurance performance is inconclusive, with studies supporting (e.g., (Bandyopadhyay and Dalui 2012; Campbell et al. 2001); Nicklas et al. 1989) or refuting (e.g., (Campbell et al. 2001; Janse De Jonge et al. 2012; Mattu et al. 2020; Redman et al. 2003)) an effect. Nicklas et al. (1989) had women cycle to exhaustion (70% VO_{2max}) at their mid-follicular and mid-luteal phases of the MC (hormonal confirmed). The mean luteal performance was nearly 13 min greater in duration ($p \leq 0.06$). Bandyopadhyay and Dalui (2012) investigated running time to exhaustion at 70% of heart rate maximum during the early-follicular, late-follicular, and mid-luteal phases of the menstrual cycle. Time to exhaustion was reduced during the early-follicular phase; however, no menstrual cycle phase verification method was used in this study. On the contrary, Mattu et al. (2020) reported no difference in cycling time to exhaustion, at 85% peak power output, between the mid-follicular and mid-luteal phases. These discrepancies could be attributed to the differences in exercise duration, ~ 31 min (Bandyopadhyay and Dalui 2012) and ~ 2.5 min (Mattu et al. 2020). With these trial durations implying that exercise was occurring in different intensity domains, the limiting factors to performance most likely differed. For instance, the short exercise duration reported by Mattu et al. (2020) was likely limited by substrate-level phosphorylation and the accumulation of fatiguing metabolites, rather than shifts in oxidative fuel sources. In another study, Campbell et al. (2001) reported that following 2 h of cycling at 70% of peak oxygen consumption ($\dot{V}O_{2peak}$), within a fasted state, maximal 4 kJ/kg body weight time trial performance (~ 24 to 28 min) was better within the follicular compared to the luteal phase of the menstrual cycle. Interestingly, when participants repeated the time trial exercise with carbohydrate supplementation, any phase difference noted was removed. Campbell et al. (2001) proposed that the potential effects of the menstrual cycle on endurance performance can be reduced with adequate ingestion of carbohydrates, which has partially been supported by Hulton et al. (2021) who concluded that carbohydrate consumption during endurance exercise (90 min at 60% $\dot{V}O_{2peak}$) curtails any metabolic variations incurred by the menstrual cycle. As it stands, the conflict between studies investigating the effects of the menstrual cycle on endurance performance could be partly explained by the different exercise durations, fueling practices, and specific phases of the menstrual cycle investigated. To fully elucidate the effect of fluctuating sex hormones on endurance performance, more studies are required to perform longer exhaustive exercise durations (e.g., triathlons, ultra-endurance events, marathons (Carmichael et al. 2021)), be more transparent with fueling protocols, and include the late-follicular/ovulation phase within their investigations.

Overall Effect of the Menstrual Cycle on Exercise Performance

Despite the recent surge in research attempting to establish the effect of the menstrual cycle on performance, a consensus by the scientific community has not been reached. Recently, several high-quality narrative (Carmichael et al. 2021) and systematic reviews (Blagrove et al. 2020, McNulty et al. 2020) have been conducted to identify, evaluate, and summarize the available empirical evidence. Indeed, McNulty et al. (2020) performed a systematic review and meta-analysis on 78 studies investigating the effects of the menstrual cycle on strength and endurance performance. The conclusions were that on average, exercise performance might be reduced by a trivial amount during the early-follicular phase compared with all other phases of the menstrual cycle. Exclusively investigating strength-related measures, Blagrove et al. (2020) agreed that the fluctuations in endogenous sex hormones across the menstrual cycle might have a small to trivial effect on strength performance. Furthermore, these reviews have unanimously agreed that the current quality of evidence in this area is low; e.g., McNulty et al. (2020) findings were based on 68% of their studies having a quality rating of "low" to "very low". Additionally, there was a large between-study variance (McNulty et al. 2020), which might reflect the notoriously low participant numbers and varied protocols often used within this research area (Blagrove et al. 2020). Together, these limitations should be heavily considered when interpreting a small or trivial effects noted. Therefore, future studies should address these methodological shortcomings to improve the quality and consistency of the evidence used by and given to women athletes and those working with them.

While it is evident that estrogen and progesterone can exert their influences on physiological systems which might underpin both strength and endurance performance, it is important to note that exercise performance is not exclusively determined by the integration of physiological function. In addition to factors such as motivation and environment, performance could be altered by the likes of cycle-related symptoms, athlete perceptions (e.g., the perceived effects of the menstrual cycle on performance and training), as well as lived experiences (e.g., menses/menstrual stigma) and changes in behavior (e.g., training habits, gym attire) associated with the menstrual cycle phases (Carmichael et al. 2021; Kolić et al. 2022, 2021). For instance, in a recent narrative review, Carmichael et al. (2021) reported that ~ 71 and ~ 65% of athletes felt their performance in training and competition, respectively, varied across the menstrual cycle. Although causation cannot be concluded, perceived variations in performance were attributed to menstrual cycle symptomologies, such as feelings of fatigue, lethargy, and menstrual pain (Carmichael et al. 2021). Therefore, to fully understand the impact of the menstrual cycle on exercise and performance, the impact of all facets of the menstrual cycle must be considered using a holistic approach (e.g., physiological, psychology, behavior, etc.), rather than the potential effects of endogenous hormones in isolation.

Practical Recommendations

Practically, the current evidence and the large intra- and inter-individual variations and experiences of the menstrual cycle do not warrant general guidance on exercise performance across the menstrual cycle. As such, scientific support for women athletes, regarding the mensural cycle, should be individualized to each female and their unique menstrual cycle characteristics and history. For example, this can be achieved through regular menstrual cycle screening, and as a minimum, consistent menstrual cycle tracking (e.g., menses and cycle length) for at least three months. Furthermore, consistent monitoring of the type, prevalence, frequency, and severity of symptoms (e.g., blood flow, discharge, breast soreness, fatigue, etc.) experienced during the menstrual cycle can help understand, manage, or exploit the impact of negative and positive symptoms, respectively. Additional insight can be provided by including perceptual and/or objective noteworthy changes in exercise performance experienced by athletes across their menstrual cycle. For more in-depth recommendations on how to monitor and interpret menstrual health within an exercise setting, please see other chapters in this volume (those developed by Bruinvels and Pedlar, and Mikkonen et al.) or see reference Davison et al. (2022). Overall, it is imperative that high-quality research on women is used to inform practice, generate critical discussion, and close the sports science gender gap, but the application should be shaped, examined, and adjusted (if required) to the individual athlete and their own individual experiences of the menstrual cycle to optimize long-term performance outcomes and goals.

References

Adkisson EJ, Casey DP, Beck DT, Gurovich AN, Martin JS, Braith RW. Central, peripheral and resistance arterial reactivity: fluctuates during the phases of the menstrual cycle. Exp Biol Med. 2010;235(1):111–8.

Ansdell P, Brownstein CG, Škarabot J, Hicks KM, Simoes DC, Thomas K, Goodall S. Menstrual cycle-associated modulations in neuromuscular function and fatigability of the knee extensors in eumenorrheic women. J Appl Physiol. 2019;126(6):1701–12.

Ansdell P, Thomas K, Hicks KM, Hunter SK, Howatson G, Goodall S. Physiological sex differences affect the integrative response to exercise: acute and chronic implications. Exp Physiol. 2020;105(12):2007–21.

Bambaeichi E, Reilly T, Cable N, Giacomoni M. The isolated and combined effects of menstrual cycle phase and time-of-day on muscle strength of eumenorrheic females. Chronobiol Int. 2004;21(4–5):645–60.

Bandyopadhyay A, Dalui R. Endurance capacity and cardiorespiratory responses in sedentary females during different phases of menstrual cycle. Kathmandu Univ Med J. 2012;10(4):25–9.

Beidleman BA, Rock PB, Muza SR, Fulco CS, Forte VA Jr, Cymerman A. Exercise V̇ e and physical performance at altitude are not affected by menstrual cycle phase. J Appl Physiol. 1999;86(5):1519–26.

Bemben D, Salm P, Salm A. Ventilatory and blood lactate responses to maximal treadmill exercise during the menstrual cycle. J Sports Med Phys Fitness. 1995;35(4):257–62.

Blagrove RC, Bruinvels G, Pedlar CR. Variations in strength-related measures during the menstrual cycle in eumenorrheic women: A systematic review and meta-analysis. J Sci Med Sport. 2020;23(12):1220–1227.

Campbell S, Angus D, Febbraio M. Glucose kinetics and exercise performance during phases of the menstrual cycle: effect of glucose ingestion. Amer J Physiol-Endocrinol Metabolism. 2001;281(4):E817–25.

Carmichael MA, Thomson RL, Moran LJ, Wycherley TP. The impact of menstrual cycle phase on athlete's performance: a narrative review. Int J Environ Res Public Health. 2021;18(4):1667.

Collins BC, Laakkonen EK, Lowe DA. Aging of the musculoskeletal system: How the loss of estrogen impacts muscle strength. Bone. 2019;123:137–44.

Constantini NW, Dubnov G, Lebrun CM. The menstrual cycle and sport performance. Clin Sports Med. 2005;24(2):e51–82.

Cowley ES, Olenick AA, McNulty KL, Ross EZ. "Invisible sportswomen": the sex data gap in sport and exercise science research. Women in Sport and Phys Activity J. 2021;29(2):146–51.

D'Eon TM, Sharoff C, Chipkin SR, Grow D, Ruby BC, Braun B. Regulation of exercise carbohydrate metabolism by estrogen and progesterone in women. Amer J Physiol-Endocrinol Metabolism. 2002;283(5):E1046–55.

Davison R, Smith PM, Hopker J, Price MJ, Hettinga F, Tew G, Bottoms L. Sport and exercise physiology testing guidelines: volume I-sport testing: the british association of sport and exercise sciences guide. Routledge; 2022.

De Souza MJ, Maguire MS, Rubin KR, Maresh CM. Effects of menstrual phase and amenorrhea on exercise performance in runners. Med Sci Sports Exerc. 1990;22(5):575–80.

Dombovy ML, Bonekat HW, Williams TJ, Staats BA. Exercise performance and ventilatory response in the menstrual cycle. Med Sci Sports Exerc. 1987;19(2):111–7.

Dulhunty A. Excitation–contraction coupling from the 1950s into the new millennium. Clin Exp Pharmacol Physiol. 2006;33(9):763–72.

Ekenros L, Hirschberg AL, Heijne A, Fridén C. Oral contraceptives do not affect muscle strength and hop performance in active women. Clin J Sport Med. 2013;23(3):202–7.

Elliott K, Cable N, Reilly T, Diver M. Effect of menstrual cycle phase on the concentration of bioavailable 17-β oestradiol and testosterone and muscle strength. Clin Sci. 2003;105(6):663–9.

Elliott-Sale KJ, Hicks KM. Hormonal-based contraception and the exercising female. The exercising female. Routledge;2018. p. 30–43.

Elliott-Sale KJ, Minahan CL, de Jonge XA, Ackerman KE, Sipilä S, Constantini NW, Hackney AC. Methodological considerations for studies in sport and exercise science with women as participants: a working guide for standards of practice for research on women. Sports Med. 2021;51(5):843–61.

Frandsen J, Pistoljevic N, Quesada JP, Amaro-Gahete FJ, Ritz C, Larsen S, Helge JW. Menstrual cycle phase does not affect whole body peak fat oxidation rate during a graded exercise test. J Appl Physiol. 2020;128(3):681–7.

Fridén C, Hirschberg AL, Saartok T. Muscle strength and endurance do not significantly vary across 3 phases of the menstrual cycle in moderately active premenopausal women. Clin J Sport Med. 2003;13(4):238–41.

Greeves J. Circamensal rhythmicity and muscle function: the role of reproductive hormones in the regulation of strength. Biol Rhythm Res. 2000;31(1):15–28.

Hackney AC, Curley C, Nicklas B. Physiological responses to submaximal exercise at the mid-follicular, ovulatory and mid-luteal phases of the menstrual cycle. Scand J Med Sci Sports. 1991;1(2):94–8.

Hackney AC, Koltun KJ, Williett HN. Menstrual cycle hormonal changes: estradiol-β-17 and progesterone interactions on exercise fat oxidation. Endocrine. 2022;76(1):240–2.

Hackney AC, McCracken-Compton MA, Ainsworth B. Substrate responses to submaximal exercise in the midfollicular and midluteal phases of the menstrual cycle. Int J Sport Nutr Exerc Metab. 1994;4(3):299–308.

Horton TJ, Miller EK, Glueck D, Tench K. No effect of menstrual cycle phase on glucose kinetics and fuel oxidation during moderate-intensity exercise. Amer J Physiol-Endocrinol Metabolism. 2002;282(4):E752–62.

Hulton A, Malone J, Campbell I, MacLaren D. The effect of the menstrual cycle and hyperglycaemia on hormonal and metabolic responses during exercise. Eur J Appl Physiol. 2021;121(11):2993–3003.

Janse De Jonge X. The effect of the menstrual cycle on exercise metabolism. Sports Med. 2003;33(11):833–51.

Janse De Jonge X, Boot C, Thom J, Ruell P, Thompson M. The influence of menstrual cycle phase on skeletal muscle contractile characteristics in humans. J Physiol. 2001;530(Pt 1):161.

Janse De Jonge X, Thompson B, Han A. Methodological recommendations for menstrual cycle research in sports and exercise. Med Sci Sports Exerc. 2019;51(12):2610–7. https://doi.org/10.1249/mss.0000000000002073.

Janse De Jonge XAK, Thompson MW, Chuter VH, Silk LN, Thom JM. Exercise performance over the menstrual cycle in temperate and hot, humid conditions. Med Sci Sports Exerc. 2012;44(11):2190–8. https://doi.org/10.1249/mss.0b013e3182656f13.

Kanaley J, Boileau R, Bahr J, Misner J, Nelson R. Substrate oxidation and GH responses to exercise are independent of menstrual phase and status. Med Sci Sports Exerc. 1992;24(8):873–80.

Kim YJ, Tamadon A, Park HT, Kim H, Ku S-Y. The role of sex steroid hormones in the pathophysiology and treatment of sarcopenia. Osteoporosis and Sarcopenia. 2016;2(3):140–55.

Kolić P, Thomas L, Morse CI, Hicks KM. Presentation of self, impression management and the period: a qualitative investigation of physically active women's experiences in sport and exercise. J Appl Sport Psychol. 2022;1–20.

Kolić PV, Sims DT, Hicks K, Thomas L, Morse CI. Physical activity and the menstrual cycle: a mixed-methods study of women's experiences. Women in Sport and Phys Activity J. 2021;29(1):47–58.

Lai S, Collins BC, Colson BA, Kararigas G, Lowe DA. Estradiol modulates myosin regulatory light chain phosphorylation and contractility in skeletal muscle of female mice. Am J Physiol Endocrinol Metab. 2016;310(9):E724-733. https://doi.org/10.1152/ajpendo.00439.2015.

Levine R, Kensler RW, Yang Z, Stull JT, Sweeney HL. Myosin light chain phosphorylation affects the structure of rabbit skeletal muscle thick filaments. Biophys J. 1996;71(2):898–907.

Lowe DA, Baltgalvis KA, Greising SM. Mechanisms behind estrogen's beneficial effect on muscle strength in females. Exerc Sport Sci Rev. 2010;38(2):61.

MacNutt MJ, De Souza MJ, Tomczak SE, Homer JL, Sheel AW. Resting and exercise ventilatory chemosensitivity across the menstrual cycle. J Appl Physiol. 2012;112(5):737–47.

Martin D, Sale C, Cooper SB, Elliott-Sale KJ. Period prevalence and perceived side effects of hormonal contraceptive use and the menstrual cycle in elite athletes. Int J Sports Physiol Perform. 2018;13(7):926–32.

Mattu AT, Iannetta D, MacInnis MJ, Doyle-Baker PK, Murias JM. Menstrual and oral contraceptive cycle phases do not affect submaximal and maximal exercise responses. Scand J Med Sci Sports. 2020;30(3):472–84.

McNulty KL, Elliott-Sale KJ, Dolan E, Swinton PA, Ansdell P, Goodall S, Hicks KM. The effects of menstrual cycle phase on exercise performance in eumenorrheic women: a systematic review and meta-analysis. Sports Med. 2020;50(10):1813–27.

Moran AL, Nelson SA, Landisch RM, Warren GL, Lowe DA. Estradiol replacement reverses ovariectomy-induced muscle contractile and myosin dysfunction in mature female mice. J Appl Physiol. 2007;102(4):1387–93.

Moran AL, Warren GL, Lowe DA. Removal of ovarian hormones from mature mice detrimentally affects muscle contractile function and myosin structural distribution. J Appl Physiol. 2006;100(2):548–59.

Oosthuyse T, Bosch AN. Oestrogen's regulation of fat metabolism during exercise and gender specific effects. Curr Opin Pharmacol. 2012;12(3):363–71.

Nicklas BJ, Hackney AC, Sharp RL. The menstrual cycle and exercise: performance, muscle glycogen, and substrate responses. Int J Sports Med. 1989;10(4):264–9.

Qaisar R, Renaud G, Hedstrom Y, Pöllänen E, Ronkainen P, Kaprio J, Bergquist J. Hormone replacement therapy improves contractile function and myonuclear organization of single muscle fibres from postmenopausal monozygotic female twin pairs. J Physiol. 2013;591(9):2333–44.

Rayment I, Holden HM, Whittaker M, Yohn CB, Lorenz M, Holmes KC, Milligan RA. Structure of the actin-myosin complex and its implications for muscle contraction. Science. 1993;261(5117):58–65.

Redman LM, Scroop GC, Norman RJ. Impact of menstrual cycle phase on the exercise status of young, sedentary women. Eur J Appl Physiol. 2003;90(5):505–13.

Rodrigues P, de Azevedo Correia M, Wharton L. Effect of menstrual cycle on muscle strength. J Exercise Physiol Online. 2019;22(5):89–96.

Sarwar R, Niclos BB, Rutherford O. Changes in muscle strength, relaxation rate and fatiguability during the human menstrual cycle. J Physiol. 1996;493(1):267–72.

Schoene RB, Robertson HT, Pierson DJ, Peterson AP. Respiratory drives and exercise in menstrual cycles of athletic and nonathletic women. J Appl Physiol. 1981;50(6):1300–5.

Smith JR, Brown KR, Murphy JD, Harms CA. Does menstrual cycle phase affect lung diffusion capacity during exercise? Respir Physiol Neurobiol. 2015;205:99–104.

Smith M, Keel J, Greenberg B, Adams L, Schmidt P, Rubinow D, Wassermann EM. Menstrual cycle effects on cortical excitability. Neurology. 1999;53(9):2069–2069.

Tenan MS, Hackney AC, Griffin L. Maximal force and tremor changes across the menstrual cycle. Eur J Appl Physiol. 2016;116(1):153–60.

Tenan MS, Peng Y-L, Hackney AC, Griffin L. Menstrual cycle mediates vastus medialis and vastus medialis oblique muscle activity. Med Sci Sports Exerc. 2013;45(11):2151–7.

Traupe T, Stettler CD, Li H, Haas E, Bhattacharya I, Minotti R, Barton M. Distinct roles of estrogen receptors α and β mediating acute vasodilation of epicardial coronary arteries. Hypertension. 2007;49(6):1364–70.

Wagner PD. New ideas on limitations to VO2max. Exerc Sport Sci Rev. 2000;28(1):10–4.