Physiological Responses to the Menstrual Cycle
Implications for the Development of Heat Illness in Female Athletes

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Abstract

Fluctuations in estrogen and progesterone during the menstrual cycle can cause changes in body systems other than the reproductive system. For example, progesterone is involved in the regulation of fluid balance in the renal tubules and innervation of the diaphragm via the phrenic nerve. However, few significant changes in the responses of the cardiovascular and respiratory systems, blood lactate, bodyweight, performance and ratings of perceived exertion are evident across the cycle. Nevertheless, substantial evidence exists to suggest that increased progesterone levels during the luteal phase cause increases in both core and skin temperatures and alter the temperature at which sweating begins during exposure to both ambient and hot environments. As heat illness is characterised by a significant increase in body temperature, it is feasible that an additional increase in core temperature during the luteal phase could place females at an increased risk of developing heat illness during this time. In addition, it is often argued that physiological gender differences such as oxygen consumption, percentage body fat and surface area-to-mass ratio place females at a higher risk of heat illness than males. This review examines various physiological responses to heat exposure during the menstrual cycle at rest and during exercise, and considers whether such changes increase the risk of heat illness in female athletes during a particular phase of the menstrual cycle.

All children generally exhibit considerable variation in physique and body composition, regardless of gender; however, differences in hormone synthesis and release during puberty elicit marked differences in physical characteristics between males and females. For example, pubertal surges of estrogen in females cause the development of reproductive organs and various secondary sexual characteristics including earlier closure of epiphyseal regions of long bones compared with males as well as increased deposits of subcutaneous fat. After reaching physical maturity, females continue to experience fluctuations in hormonal releases until menopause and these cause regular cyclic changes in a number of physiological parameters including increases in core temperature and setpoint temperature for the onset of sweating. The development of heat illness is of particular concern to residents of warmer climates and to athletes. Several reviews have addressed thermoregulation...
in hot environments\cite{1,2} with a few focusing on the thermoregulatory responses of gender\cite{3-5} and exercise\cite{6-9} in the heat. A large number of studies have examined various physiological responses across the menstrual cycle both in ambient and hot conditions and will be summarised in this review.

1. Menstrual Cycle

The menstrual cycle is a series of various hormone releases that coordinate the readiness of the female reproductive system for conception (figure 1). The release of gonadotropin-releasing hormone (GnRH) by the hypothalamus on day one of the cycle controls stimulation and inhibition of the release of follicle-stimulating hormone (FSH) and luteinising hormone (LH) from the anterior pituitary. These hormones then regulate the release of estrogen (from ovarian follicles) and progesterone (from the corpus luteum) and coordinate progression of these events during an average cycle of 28 days. Progesterone levels are elevated in the luteal phase of the menstrual cycle. As progesterone is a precursor of aldosterone, the luteal phase is also characterised by an increase in aldosterone concentration\cite{10}. In isolation, progesterone exhibits a natriuretic effect resulting in increased fluid loss and decreased blood pressure\cite{11}; however, as increases in progesterone are accompanied by concomitant increases in aldosterone,\cite{12} such changes are only transient and do not cause increases in sodium retention or blood pressure\cite{13,14}. This is a direct result of competition of both progesterone and aldosterone for the type I corticosteroid receptor\cite{11,13,15}. Further, it has been speculated that progesterone and estrogen function in body fluid regulation by altering sodium and water distribution rather than retention\cite{10,14}. Increases in estrogen concentration are evident during ovulation and remain above baseline levels during the luteal phase\cite{16}. This is accompanied by increases in arginine vasopressin leading to thirst stimulation\cite{17,18}. Estrogen also functions in vasodilation (reviewed in Charkoudian\cite{19}) and appears to be a result of prostacyclin and nitric oxide release (reviewed in Farhat et al\cite{20}).

Although the method used to divide the menstrual cycle into phases differs between studies, the most common is simply follicular (days 1 to 15) and luteal (days 16 to 28) phases\cite{4}. Less common variations include menstruating/nonmenstruating\cite{23,24} and pre-menstrual/menstruation/post-menstruation\cite{25}. As the levels of FSH and LH follow a similar trend throughout the menstrual cycle and peak simultaneously, the division of the cycle for research purposes generally coincides with the extremes in the concentrations of estrogen and progesterone.
Various methods are employed to determine a particular phase and their accuracy depends upon the scope, the purpose of the study and the equipment available. The easiest, though least accurate, method involves counting the days of a self-reported cycle. This assumes that the individual has a regular menstrual cycle that can be applied proportionally to an average representation. Assumptions are also made that the individual has correctly reported and documented her recent menstrual history and has not suddenly changed any variable that may alter the regularity of the events of the cycle. This method typically utilises the extremes of each phase, that is, follicular: days 1-5, and luteal: 19-25. Daily hormonal concentrations (particularly progesterone) in the blood or urine can be used to create an individual cycle pattern from which certain days can be identified as being within the required phase. This is often confirmed by further hormonal assays on the day of testing. Similarly, ovulation can be reliably predicted by kits that measure the surge in LH.[16,26]

As progesterone has often been implicated in core-temperature fluctuations throughout the menstrual cycle,[4] daily basal body temperature is often used to confirm cycle phases. This often requires the individual to record oral or rectal temperature daily upon waking and assumes a regular pattern of activity throughout. In one study, ultrasound examinations were conducted to confirm that ovulation did occur.[27] A review by Lebrun[28] compared the results from studies that have utilised serum progesterone measurements with those that used other methods of determining phases. Conclusions indicated that the results from those studies not using hormone concentrations were inconsistent, although some suggested potential interactions between the menstrual cycle and various physiological and performance variables.[28] In addition, research has also examined variations between eumenorrheic and amenorrhoeic individuals, both at rest and during exercise. The intensity of exercise undertaken by an athlete has often been implicated as the source of various menstrual irregularities including delayed menarche, shortened luteal phase, oligomenorrhoea (irregular menstruation) and amenorrhoea (absent menstruation associated with low estrogen levels).[29] Other associated causes of menstrual irregularities in athletes include low percentage body fat, poor diet and physiological stress. These are often grouped collectively as ‘athletic amenorrhoea’ and have been linked to other hypothalmic menstrual disorders caused by factors such as psychogenic amenorrhoea, anorexia nervosa, drug abuse and malnutrition.

2. Heat Illness

The term ‘heat illness’ is a generic term that describes a variety of disorders associated with exposure to a hot environment. Although three categories of clinical disorders are generally used: heat cramps, heat exhaustion and heat stroke (for reviews see references[30-32]), heat illness cannot be characterised by any of these disorders in isolation. Rather, heat illness is a continuous progression of symptoms resulting from an increasing core temperature. Heat cramps are generally assumed to be skeletal muscle spasms resulting from excessive fluid loss; however, the existence of this condition is not supported by definitive research findings.[33] The term ‘heat exhaustion’ is often used interchangeably with heat syncope and symptoms include extreme weakness and normal or slightly increased body temperature.[33] Heat stroke can be either ‘classical’ or ‘exertional-induced’. Classical heat stroke is characterised by elevated core temperatures, hot, dry skin and confusion, whereas exertional-induced heat stroke is usually seen in isolated cases and is typified by increases in core temperature, sweat production, confusion and possibly rhabdomyolysis and acute renal failure.[30,34] Other conditions frequently classified as ‘heat illness’ include heat strain and heat injury.

The thermoregulatory capacity of the body can be overloaded through either substantial heat production (via intense muscular activity) or reduced heat dispersion (via exposure to a hot environment), causing core temperature to rise. When ambient temperature exceeds skin temperature, the capacity of the body to lose heat via either conduc-
tion, convection or radiation is eliminated; mechanisms such as vasodilation and increased sweating are then the only means of heat loss. The progression from heat exhaustion to heat stroke is dangerous, as there is typically a decrease or cessation in sweating leading to a subsequent increase in core temperature. Irreversible damage to physiological systems may occur as a consequence of severe heat illness, including myocardial infarction, coma, stroke, liver damage, renal failure, muscle membrane breakdown and rhabdomyolysis. The progression from one complication to another can lead to the failure of one or more body system(s), thus increasing the likelihood of death.

Several predisposing factors increase the risk of certain populations to the development of heat illnesses. These include age, obesity, chronic illness, physical fitness, drug and alcohol abuse, hypertension, degree of acclimatisation and history of heat intolerance. Gender has been considered a predisposing factor and early research indicated that females were less tolerant of exposure to hot environments than males; however, these studies did not match participants for physical fitness or size. Indeed, very little data exist to suggest that differences in thermoregulatory capability can be attributed to gender when there is adequate matching and control of factors such as maximal aerobic power, heat acclimation and menstrual cycle phase. Significantly lower sweat rates found in females are thought to be due to the larger surface area-to-mass ratio, which would be a disadvantage when ambient temperatures become higher than the skin temperature as the body will gain heat via conduction and radiation. Similarly, higher levels of body fat, such as that found in females, significantly influence thermoregulation as males and females matched for body fatness do not exhibit differences in sweat rate, metabolic rate, heart rate or tolerance time to heat exposure. Within the female population, the menstrual cycle causes changes in various parameters as a result of the actions of hormones such as estrogen and progesterone. As mentioned previously, these hormones function in fluid regulation and vasodilation and fluctuations can cause significant, although transient, changes in core temperature.

3. Physiological Responses to the Menstrual Cycle

3.1 Cardiovascular Responses

Few, if any, changes have been found in the cardiovascular response to changing hormone levels during the menstrual cycle. While progesterone can induce natriuresis, its effect is transient because of the subsequent increase in aldosterone. As mentioned previously, few changes in fluid balance are reported with physiological levels of both progesterone and estrogen and as such, significant changes in various cardiovascular parameters are not to be expected.

3.1.1 Heart Rate

The majority of studies has found no significant difference in heart rate between the phases of the menstrual cycle either at rest or during exercise. A small number of studies has shown significant differences between phases, with heart rate during the luteal phase being significantly higher both at rest and during exercise compared with the follicular phase. Kolka and Stephenson reported no significant difference in resting heart rate, although there was a significant increase during the luteal phase following attenuation of forearm blood flow. Pivarnik et al. found a significantly elevated heart rate during the luteal phase over 60 minutes of cycling compared with the follicular phase. No menstrual cycle phase-related changes in heart rate have been found during either rest or exercise in hot conditions.

3.1.2 Plasma Volume

Phase-related fluctuations in plasma volume have not been uniformly reported. Horvath and Drinkwater, De Souza et al., Chapman et al., Stachenfeld et al. and Miskec et al. found no significant differences in plasma volume
across the menstrual cycle. Induced dehydration followed by ingestion of a sports drink equivalent to 150% of the total mass lost has also shown no phase-related shift in plasma volume.[66] Decreased plasma volume during the luteal phase has been reported in a number of studies.[14,17,37] Gaebelein and Senay[67] suggested this was possibly caused by an increase in vascular permeability to protein during the luteal phase. Plasma volume has also been found to become significantly different between phases at higher external temperatures.[45] As increases in estrogen are known to enhance the actions of aldosterone in the absorption of sodium in the renal tubules,[18] this suggestion seems plausible. It is possible, however, that the lack of significance in plasma volume changes found in most studies could be related to the method of determining the relevant menstrual cycle phase as reviewed in section 1.

3.1.3 Haemoglobin and Haematocrit
Numerous studies have reported no significant changes in haemoglobin concentration over the course of the menstrual cycle.[46,47,61,63,68] Although an increase in resting luteal haemoglobin was found by Jurkowski et al.[48] this did not result in a substantial increase in arterial oxygen content or any subsequent change in oxygen delivery during exercise. Similarly, decreases in luteal haemoglobin concentration were not reflected in changes in oxygen consumption (VO₂) at rest or during exercise.[69] Haemoglobin concentrations are generally higher in males, although this is compensated in females by higher levels of 2,3-diphosphoglycerate (2,3-DPG) which increases the availability of oxygen to tissues. Increased 2,3-DPG in the luteal phase was found by Denis et al.[70] but a subsequent study by MacDonald and MacDonald[71] failed to reproduce this finding. Haematocrit levels are generally shown to remain unchanged both at rest and during exercise between phases;[47,61,68,72,73] however, Stachenfeld et al.[10,55] found increased haematocrit levels during the luteal phase compared with the early follicular phase (p < 0.05).

Elevated luteal haematocrits during rest and exercise in ambient temperatures are maintained during exposure to hot environments.[62,63] Alternatively, Tenaglia et al.[61] reported no difference in haematocrit between phases in either normal or hot environments, whereas Stephenson and Kolka[37] found elevated luteal values at rest with the phase differences increasing during passive heating but converging during heated exercise trials. Haemoglobin concentration has been shown to remain unchanged between phases in the heat.[37,61]. Decreases in haemoglobin concentration during the luteal phase do not necessarily cause VO₂ changes at rest or during exercise in hot conditions.[62] The most probable cause for such variations in haematocrit is the degree of control exerted over the water intake of individuals, particularly in hot conditions.[67] Further, it was suggested that hyperhydration of individuals would result in a more consistent pattern in haemoglobin, haematocrit and plasma volume observations.[67]

3.2 Respiratory Responses
Any possible influences of the menstrual cycle on respiratory responses have been linked to circulating concentrations of haemoglobin, progesterone and 2,3-DPG. Although there are increases in progesterone during the luteal phase, their influence on respiration is uncertain. It is unclear if any definitive changes occur in the frequently used measures of respiratory function due to the menstrual cycle.

3.2.1 Oxygen Consumption
Although the majority of previous studies indicate the menstrual cycle has little or no significant effect on VO₂ or maximal oxygen uptake (VO₂max) in healthy, young females during exposure to ambient[23,45,48,50-54,60,68,69,74-76] or hot conditions,[45,62,63] a few studies reported a significant phase influence. Lebrun et al.[47] found VO₂max to be higher in the early follicular phase of trained individuals, with absolute VO₂max (L/min) being statistically significant (p = 0.04) and relative VO₂max (ml/kg/min) less so (p = 0.06). The authors acknowledged the problem of an increased risk of Type I error in the statistical analyses performed and the range of equipment error. They further stated that some in-
individuals recorded a substantial increase in $\text{VO}_2\text{max}$ during the early follicular phase (up to 4 ml/kg/min). However, significantly elevated $\text{VO}_2$ results at rest have also been reported in the luteal phase.\[57,58,77\]

### 3.2.2 Minute Ventilation

Fluctuating progesterone concentrations have been implicated in changes in respiratory muscle function; increased progesterone has been shown to cause significant increases in phrenic nerve activity.\[78\] As the phrenic nerve innervates the diaphragm, elevated progesterone is often associated with varying degrees of hyperventilation and hypocapnia during the luteal phase.\[70\] Post-menopausal women exhibit a significantly elevated minute ventilation ($V_E$) following both progestogen (progestin) and a combined progestogen-estrogen supplementation programme compared with either the placebo or estrogen group.\[79\] Chen and Tang\[80\] also found inspiratory muscle endurance was enhanced during the luteal phase of the menstrual cycle. Despite these findings, researchers are divided in stating the influence of the cycle on $V_E$. While a number of studies have shown no significant difference in $V_E$ between phases in normal\[44,45,47,51,52,68,76\] or hot\[45,62,63\] environments, others have reported significant elevations in $V_E$ \[77,81,82\] and maximal inspiratory ventilation\[48\] during the luteal phase. Comparisons between eumenorrhoeic and amenorrhoeic individuals have also yielded contradictory results. De Souza et al.\[53\] found no difference between phases in eumenorrhoeic individuals or across time in amenorrhoeic individuals. Conversely, substantial variations were reported in another study by De Souza and colleagues\[12\] between the above groups and phases, yet no statistical comparisons were made. Schoene et al.\[23\] reported significant increases in resting $V_E$ during the luteal phase in menstruating individuals (athletes: $p < 0.001$, non-athletes: $p < 0.01$). The combination of trained and untrained individuals (total $n = 12$) yielded a highly significant increase in $V_E$ in the luteal phase compared with the follicular phase ($p < 0.0001$) that is yet to be reproduced by any other studies.

*In summary*, although it has been shown that progesterone has a marked effect on phrenic nerve activity, subsequent influences of the menstrual cycle on respiratory function are not conclusive. Inconsistent findings of cyclic haemoglobin and 2,3-DPG fluctuations preclude their inclusion as definite influences on respiration. No significant differences are generally reported in the standard measurements of $\text{VO}_2$ and ventilation across the phases of the menstrual cycle. Further, little or no significant phase-related change is apparent during heat exposure in various respiratory responses (such as $\text{VO}_2$, $\text{VO}_2\text{max}$ or $V_E$).

### 3.3 Blood Lactate

The majority of studies involving lactate fluctuations across the menstrual cycle concluded that no significant effect from the menstrual cycle is evident.\[24,52,53,58,73,83-86\] McCracken et al.\[72\] focussed solely on the blood lactate response to exercise and while resting levels were similar, recovery concentrations differed with each phase; post-exercise luteal lactate levels were significantly lower than the corresponding follicular levels ($p < 0.05$). Conversely, Jurkowski et al.\[48\] reported a significant elevation in resting blood lactate levels during the follicular phase that persisted both at the end of heavy exercise ($p < 0.05$) and at exhaustion ($p < 0.01$). No differences have been observed between eumenorrhoeic and amenorrhoeic athletes.\[53\]

Investigations into possible differences in blood lactate concentrations between phases following heat exposure have only been undertaken by Wells and Horvath\[62,63\] with contradictory findings. Both of these studies divided the menstrual cycle into three phases: flow, ovulatory and luteal and exposed untrained, eumenorrhoeic individuals to 48°C heat. No significant differences in lactate concentrations were found between phases at rest;\[62\] however, significant elevations were found in the ovulatory phase compared with the flow phase following exercise in the hot environment ($p < 0.05$).\[63\] As menstrual flow is typically used to identify the follicular phase, these data suggest no difference in lactate concentration between the follicular and luteal phases during heat exposure either at rest or during exercise.
3.4 Bodyweight

The relationship between bodyweight and percentage body fat to the menstrual cycle appears to be of little or no consequence.\[10,17,24,47,52,73,87-89\] Similarly, heat exposure does not cause a change in bodyweight between menstrual cycle phases.\[61-64\]

3.5 Sweating and Vascular Reactivity

While a small number of studies reported no phase-related changes in sweat rate,\[62,63,90\] threshold for onset of sweating\[91\] or total sweat loss,\[90\] the majority have found significant differences in various parameters between menstrual cycle phases.\[56,58,64,92,93\] The threshold for onset of sweating is increased during the luteal phase.\[56,64,93\] Further, while sweat rates have been shown to be elevated during the luteal phase,\[56,58,92\] Kolka and Stephenson found no differences between either the follicular or luteal phases during intense exercise in either 35\[64,93\] or 50°C\[64\] heat. The potential mechanisms responsible for the increased threshold for the onset of sweating during the luteal phase could be associated with the actions of either progesterone or estrogen as both are elevated during this time. The stimulus to increase sweat production occurs centrally, a direct result of progesterone acting on the preoptic/anterior hypothalamus.\[21\] As estrogen promotes vasodilation,\[20\] it is reasonable that increases in estrogen seen during ovulation and, to a lesser extent, during the luteal phase would be accompanied by an increase in cutaneous blood flow. However, phase-related changes in forearm blood flow have not been uniformly reported. Hirata et al.\[51\] did not find any difference in forearm blood flow between the follicular and luteal phases either at rest or during exercise at 40 and 70% VO\textsubscript{2max}. In contrast, Horvath and Drinkwater\[45\] found forearm blood flow at rest during exposure to a 28°C environment to be approximately 25% lower during the menstrual flow when compared with either ovulation or the luteal phase. Further, exercise at various temperatures ranging from 28 to 48°C eliminated this difference and values of forearm blood flow became similar between all three phases.\[45\]

3.6 Exercise Performance

Repeated swimming time trials, incremental, high-intensity and intermittent treadmill running and maximal cycle protocols have all resulted in no differences in performance across the menstrual cycle.\[23,25,47,69,88\] Further, De Souza et al.\[53\] conclude that phase does not influence exercise performance and that amenorrhoeic individuals do not exhibit detriments in performance due to lack of menstrual flow. The influence of phase on sub-maximal endurance performance seems dependent on the protocol employed. Incremental exercise tests used by Bamben et al.,\[52\] McCracken et al.\[72\] and Stephenson et al.\[176\] all showed no change in time to exhaustion between phases. A maximal treadmill run at 90% VO\textsubscript{2max} was also shown to have no significant phase differences.\[47\] Contrary to these studies, however, Jurkowski et al.\[48\] found that time to exhaustion doubled (p < 0.02) in the luteal phase compared with the follicular phase at 90% maximum power output on a cycle ergometer.

As there is little physiological basis for anticipated menstrual phase-related changes in anaerobic parameters, it would be expected that no significant variations would be reported. Davies et al.\[94\] attempted to justify the investigation of various strength tests by stating that endurance or aerobic performance is likely to be affected by psychological factors that should not impact on explosive, short-duration activities. Lebrun et al.,\[47\] Higgs and Robertson\[49\] and DiBrezzo et al.\[87\] measured knee flexion and extension strength and all concluded that no difference exists between phases. In addition, no phase difference was found in the study by Quadagno et al.\[25\] that recorded the number of times a weightlifter could bench press a weight equivalent to 70% of her predetermined maximum. Differences were not found in measures of maximal cycling power, maximal jumping power or vertical jump height between phases;\[89\] however, handgrip strength has been found to vary
between studies.\textsuperscript{[49,94,95]} Whereas Higgs and Robertson\textsuperscript{[49]} reported no difference between grip strength and cycle phase, performance during the handgrip test conducted by Davies et al.\textsuperscript{[94]} showed increased performance during menstrual flow compared with both the follicular and luteal phases. However, while the variation in performance between the flow and follicular phases was also significant (p < 0.05), comparisons of flow versus luteal and follicular versus luteal phases were not.\textsuperscript{[94]} Maximal voluntary contraction during the handgrip test has also been found to be significantly lower during the luteal phase.\textsuperscript{[95]}

The variations between these two studies may be attributed to the method employed to determine days on which follicular testing was to occur. Both utilised the allocation of days based on previous menstrual history; however, the intervals varied (Davies et al.:\textsuperscript{[94]} days 12 to 14, Wirth and Lohman:\textsuperscript{[95]} days 6 to 10) and only the latter applied individual cycles proportionally to the average 28-day cycle. Wirth and Lohman\textsuperscript{[95]} also found little evidence to suggest a causal relationship between the use or non-use of oral contraceptives on grip strength; however, non-users exhibited a significantly higher endurance time and force output than those using oral contraceptives.

3.7 Ratings of Perceived Exertion

Although no significant physiological reason exists to suggest performance may be altered during the menstrual cycle, a psychological effect from the menstrual cycle should not be discounted. Anecdotal and early retrospective studies took the view that menstruation corresponded to a decreased physical capacity;\textsuperscript{[125]} however, the majority of studies have revealed no variation in ratings of perceived exertion (RPE) during the menstrual cycle.\textsuperscript{[24,50,53,54,68,74]} Two studies have shown significant phase-related changes in RPE.\textsuperscript{[46,60]} Individuals in both studies cycled at approximately 70% VO$_{2\text{max}}$ for 60 minutes, which resulted in significant differences towards the final stages of each test. Interestingly, Pivarnik et al.\textsuperscript{[60]} found RPE to be increased during the luteal phase, whereas Hackney et al.\textsuperscript{[46]} found values during ovulation to be significantly elevated above both luteal and follicular values. The latter study used RPE responses relating to localised perception in the exercising muscle groups in the legs. Justification for this increase during ovulation was that an exercise-induced reduction in estrogen levels during the tests at ovulation could have caused mild ischaemia in the exercising muscles.\textsuperscript{[46,48]} As RPE differs between studies, individuals and scales, it is difficult to conclude whether there is a significant phase-effect present, although most previous studies have reported no significant changes.

3.8 Core Temperature

Core temperature is often used to indicate the efficiency of the thermoregulatory system of the body. An exact site for measurement of core temperature is a contentious issue and varies between studies. Commonly used sites include rectal, oral, oesophageal and tympanic regions of the body, each yielding different values due to regional blood flow, metabolic rate of surrounding tissues and exposure to the external environment.\textsuperscript{[7]} Interestingly, Coyne et al.\textsuperscript{[96]} recently utilised an ingested temperature telemetry pill that recorded internal temperatures of the gastrointestinal tract until excreted after 3 to 4 days. As previously discussed, progesterone concentration is maximal during the luteal phase, although subsequent changes in fluid balance are only transient and are attenuated by concomitant increases in aldosterone. Estrogen and progesterone act on the preoptic/anterior regions of the hypothalamus to increase vasodilation and sweating, respectively, but it appears that progesterone causes an increase in the firing rate of neurons within the hypothalamus (reviewed in Stephenson and Kolka\textsuperscript{[21]}). This increases the setpoint temperature, resulting in the sustained increase in core temperature found during the luteal phase. Interestingly, Stachenfeld et al.\textsuperscript{[55]} and Stephenson and Kolka\textsuperscript{[97]} recently reported that estrogen administration can attenuate the thermoregulatory effects of progesterone and lower the hormonally-increased setpoint. Further, it has been suggested that core temperature could depend on the ratio of progesterone to estrogen.\textsuperscript{[97]}
3.8.1 Oral Temperature

Because of inconsistencies in measurement techniques, oral temperatures are rarely recorded during physiological testing; a comparison of oral temperature between studies is therefore difficult. In order to decrease the possibility of variation in measurements, previous studies have sealed participants’ mouths to force nasal breathing while others have had participants avoid swallowing and ingesting fluids during testing.\(^7\) This renders the measurement of oral temperature during exercise testing impossible. Although only a few studies have incorporated oral temperatures into investigations concerning the menstrual cycle, a consistent elevation in oral temperature during the luteal phase has been reported.\(^{23,81,98,99}\) While each study found significant differences between the follicular and luteal phases, comparisons between studies reveal the mean follicular oral temperature in some cases to be equivalent to the luteal in others.

3.8.2 Oesophageal Temperature

Oesophageal temperature is measured by swallowing a thermocouple to the level of the heart and can cause discomfort in individuals with a sensitive gag reflex. Although a reliable measure, the influence of liquid ingestion during exercise testing can cause transient changes in measurements.\(^7\) Studies using oesophageal temperature have found it to be significantly elevated during the luteal phase at rest and during exercise in both ambient\(^{37,51,55,56,58,59,64,93}\) and hot\(^{56,58,64}\) conditions. In addition, a recent study by Stephenson and Kolka\(^{97}\) showed oesophageal temperature to be decreased by 0.26°C (p < 0.05) in the pre-ovulatory phase compared with the follicular phase, thought to be a result of estrogen-mediated changes in vasodilation and firing rate of temperature-sensitive neurons in the preoptic/anterior hypothalamus.

3.8.3 Tympanic Temperature

Aural or tympanic temperature is measured by inserting a thermister into the auditory canal, with or without the use of a device to seal the canal against external temperatures. The reliability of this measure is variable because of the influences of facial skin temperature.\(^7\) As with oral measurements, the majority of studies have found significant elevations in tympanic temperature during the luteal phase.\(^{56,58,88,100}\) At rest, Lynch and Nimmo\(^{88}\) reported a significant increase in luteal aural temperature (0.5°C, p < 0.05), although measurements recorded after intermittent exercise showed the increase to be significant only at 1 minute after exercise. Results after this time (3 to 15 minutes after exercise) were still elevated but not significant during the luteal phase. Hessemer and Bruck\(^{56,58}\) conducted tests between 3 and 4am, the time at which the early follicular-luteal difference in core temperature is assumed to be maximal. Significant elevations were found in the luteal phase (p < 0.0001)\(^{56,58}\) that was maintained during exercise (p < 0.0001).\(^{58}\)

3.8.4 Gastrointestinal Temperature

To date, only one study has utilised the gastrointestinal tract for noninvasive recordings of core temperature. Coyne et al.\(^{96}\) used calibrated temperature telemetry pills to measure temperature continuously until excretion 3 to 4 days after ingestion. This study found significant differences between both the mean (0.31°C, p < 0.001) and minimum (0.46°C, p < 0.0001) temperatures during the follicular and luteal phases.

3.8.5 Rectal Temperature

Rectal temperature is widely regarded to be the closest approximation of core temperature without penetrating the chest cavity. Sawka and Wenger\(^7\) state that rectal temperatures are generally uniform when the thermister is inserted between 5 to 27cm beyond the anal sphincter; the average insertion used in most studies is normally 8 to 12cm for the comfort of participants. Being the most consistent of all core temperature measurements, there are differing opinions as to the presence of changes in rectal temperature affected by the menstrual cycle. The majority of studies report significantly elevated resting rectal temperatures during the luteal phase in ambient\(^{45,56,58,60,76,90}\) and hot\(^{56,90}\) conditions. During exercise, Pivarnik et al.\(^{60}\) also reported a constant difference in rising rectal temperatures between the phases until 40 minutes when the
difference became significantly exaggerated. This was also found by Stephenson et al.,[76] whereas Carpenter and Nunneley[90] reported a maintained difference between phases during exercise. Other studies have reported no significant variation in rectal temperature between phases at rest.[24,62,67] Further, no differences were found between phases during exercise in euhydrated,[24] or either hyper- and hypo-hydrated individuals.[67] Avellini et al.[38] reported no difference in resting values of rectal temperature between males, pre-ovulatory or post-ovulatory females; however, a significant difference developed between the three groups during exercise in the heat. These variations decreased following 10 days of acclimation. Alternately, no variations have been found during passive heating[62,63] or during exercise in both hyper- or hypo-hydrated states in the heat.[67]

In summary, the site chosen for measurement of core temperature greatly influences not only the absolute temperature measured, but can be potentially influenced by factors such as ingestion of liquids, exercise and environmental conditions. Rectal temperature has been used widely and appears to provide the most consistent approximation of core temperature during exercise trials. Although results from early studies are not consistent, the majority of more recent, well-controlled trials suggest core temperatures are more likely to be elevated during the luteal phase. The mechanisms for this elevation have been discussed previously, and appear to be the result of both estrogen and progesterone acting on the preoptic/anterior regions of the hypothalamus although the exact actions or contribution from each hormone remain unclear.

3.9 Skin Temperature

As core temperature is increased during the luteal phase, it is reasonable to expect that skin temperature would also be elevated; however, studies involving possible variations during the menstrual cycle have recorded varying results. A number have found either no phase differences[38,51,55,60,91,100,101] or increased luteal temperatures[37,56,61,64,90] both at rest and during exercise. Phase effects have also been found to differ between resting and exercise measurements. Hessemer and Bruck,[58] and Kolka and Stephenson[59] both found elevated luteal skin temperatures at rest with no phase differences during exercise, whereas Grucza et al.[92] reported no differences at rest but significant elevations during exercise in the follicular phase. As the onset of sweating is delayed during the luteal phase,[56,64,93] skin temperature would be expected to increase during this phase. However, it appears likely that the rate of sweat production is increased during the luteal phase[56,58,92] as is estrogen-mediated cutaneous vasodilation at rest,[65] thus potentially eliminating any increase in skin temperature during the luteal phase.

4. Conclusion

Given that the phase of the menstrual cycle does not appear to alter heart rate, \( \text{VO}_{2\text{max}} \), blood lactate concentration, exercise performance or RPE, female athletes should not expect any detrimental performance during competition in either normal or hot conditions. However, elevated core temperatures during the luteal phase are consistently reported, with differences of up to 0.6°C, both at rest[56,58] and during exercise[60] in ambient temperatures, that are maintained in hot environments.[37,56,64,90] The significance of a sustained difference of this magnitude during exercise in the heat could be problematic; however, Charkoudian and Johnson[102] stated that no evidence exists to support the claim that females would be more susceptible to the development of heat illness during exercise in their luteal phase. Further, recent findings by Stephenson and Kolka[97] indicated that elevated estrogen levels immediately prior to ovulation cause a decrease in the setpoint temperature that could prove beneficial during exercise in the heat at this time of the menstrual cycle.

In addition, it has been shown that in males exhibiting different initial core temperatures, volitional exhaustion following exercise in hot conditions occurred at the same level of hyperthermia (oesophageal temperature in human males: 40.1 to 40.2°C;[103] rectal temperature in male rats: 42.2 to
An important finding of both of these studies was that the time to exhaustion was inversely related to the starting core temperatures. During exercise in ambient temperatures, females showed either no difference\(^{74,76}\) or an increased time to exhaustion in their luteal phase.\(^{48}\) Conversely, tolerance time to passive exposure to 40°C conditions has been found to decrease during the luteal phase compared with the early follicular phase.\(^{61}\) Interestingly, McLellan et al.\(^{105}\) recently reported that diurnal increases in rectal temperature in males were sustained at exhaustion following passive heat exposure but were not accompanied by a significant difference in tolerance time. Thus, it would seem that no conclusive evidence exists to suggest that there is a phase-related influence on time to exhaustion in the heat. If a critical temperature does exist in females, time to exhaustion would decrease during the luteal phase, as the initial core temperature would be higher than that corresponding to the follicular phase. However, psychological factors should not be discounted, particularly with highly trained athletes. As a large number of athletes train and compete using performance time to gauge their progress, it is reasonable for female athletes to attempt to extend their time to fatigue and incur dangerous increases in core temperature. Given the difficulty in conducting clinical research into the development of heat illness, obtaining evidence of the theoretical increased susceptibility to heat illness during the luteal phase in females remains elusive; however, it is an area that warrants further investigation.

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