The Menstrual Cycle and Human Performance: An Overview

Thomas Reilly
Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Liverpool, UK

ABSTRACT
Physiological events during the normal menstrual cycle are determined by feedback loops within the hypothalamic-pituitary-ovarian axis. Hormonal changes within the menstrual cycle have potential impact on human performance. Relevant stages to consider are pre-menses and menses, the follicular and luteal phases separated by an abrupt elevation in lutenizing hormone and characterised by a sharp rise in body temperature coinciding with ovulation. Strenuous athletic training may affect the normal menstrual cycle. Such disruptions include delayed menarche in ballet dancers and gymnasts, shortened luteal phase and secondary amenorrhea associated with high training loads and competitive stress. Amenorrhea is also noted in flight attendants, linked with an inhibiting effect of disrupted circadian rhythm on lutenizing hormone. The so-called ‘athlete triad’ considers secondary amenorrhea, abnormal eating behaviour and osteoporosis (attributed to chronic hypoestrogenia). The normal cycle may also be disrupted when circadian rhythms are disturbed, for example in rapid time-zone transitions. Fluctuations in the steroid hormones have been associated with changes in muscle strength. There is evidence also of elevations in heart rate: changes may be partly specific to time of day. Effects on muscle strength may be determined at selected stages of the menstrual cycle, using whole-body performance, local muscle groups or isolated individual muscles. Whilst oestrogen has been implicated in the ergogenic effect of steroid hormones, there is accumulating evidence that a role for progesterone cannot be discounted. The isolation of the ovarian hormones separately is feasible with studies of IVF patients or groups on hormone replacement therapy.

KEYWORDS: Circamensal rhythms, exercise, premenstrual tension, osteoporosis, training.

INTRODUCTION
Circamensal rhythms are well illustrated in the human female menstrual cycle. The rhythm incorporates cyclical changes in both ovaries and uterus and the production of ova as part of the reproductive process. The normal menstrual cycle
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has a length of 28 days on average but varies between individuals and between cycles from 23 to 38 days. The cycle is manifest throughout the reproductive years commencing with the first cycle or menarche at 11–13 years and terminating in menopause at about 50 years.

Exercise and the menstrual cycle may interact in various ways to the extent that few experimental studies of female athletes are considered without controlling for menstrual cycle phase. The first question is whether the physiological changes that occur during the menstrual cycle affect responses to exercise and in particular maximal exercise performance. The converse question is whether strenuous physical training influences characteristics of the normal menstrual cycle and in combination with diet has potential consequences for women’s health. A further consideration is whether oral contraceptive manipulation of reproductive hormones influences the capacity for exercise. Finally, there are possible interactions between circadian and circamensal rhythms, especially when the former are disturbed.

These considerations are addressed in this overview of circamensal rhythms in athletes. As background, the physiology of the menstrual cycle is first summarily outlined.

The physiology of the menstrual cycle
The menstrual cycle is regulated by a complex incorporating the hypothalamus (producing gonadotrophin–releasing–factor or GnRH) the anterior pituitary gland with its output of follicle stimulating hormone (FSH) and luteinizing hormone (LH), the ovaries, follicles, and the corpus luteum (producing oestrogens, progesterone and inhibin). The feedback loops to the pituitary and hypothalamus (Fig. 1) illustrate why the system is referred to as the hypothalamic-pituitary-ovarian axis (Reilly et al., 1997).

The menstrual cycle starts with menses (menstruation), a phase which lasts 4–5 days during which about 40 ml of blood is discharged along with the surface part of the endometrial wall. Blood losses may vary from 25 to 65 ml, but can in some instances exceed 200 ml. Consistent heavy losses of blood with menstruation can lead to anaemia and a decrease in oxygen carrying capacity in the circulatory system, which would adversely affect endurance athletes. Once menses ceases, renewal of the endometrial lining is promoted by oestrogens (mainly oestradiol), whilst FSH stimulates the maturation of the ovum into a graafian follicle. This part of the cycle constitutes the follicular phase. The maturing follicle ovulates about mid-cycle (Day 14), ovulation being triggered by a sharp rise in LH and signified by an elevation of about 0.5°C in body temperature. For conception to occur, the liberated ovum has to be fertilized within 24 h. The cycle now enters the luteal phase characterized by the collapse of the ruptured follicle from which the ovum has burst to form the corpus luteum which produces increased
amounts of progesterone. If implantation of the fertilised egg has not occurred, the corpus luteum usually regresses by day 21. As the main function of progesterone is to prepare the uterine wall for implantation of the fertilized ovum, progesterone falls to a low level pre-menses. Consequently the endometrium regresses and about two-thirds of its lining is shed in menstruation as the next cycle starts.

The hormonal changes affecting the uterus are closely integrated with ovarian functions within the overall regulation of the menstrual cycle (Fig. 2). The proliferative phase of the endometrium following menses is stimulated by the steroid hormones oestradiol and progesterone. Oestradiol output enhances development of the endometrial surface and the spiral arteries. At ovulation progesterone causes the endometrium to develop mucous-secreting glands and endometrial cells to accumulate glycogen in anticipation of receiving a fertilized ovum. As the ovum reaches the uterus, the follicular cells now form the corpus luteum which, as explained above, secretes large amounts of oestrogen (oestradiol) and progesterone. Negative feedback of the levels of these hormones acts to suppress the release of GnRH, FSH and LH. Once the fertilised ovum is implanted in the uterus, progesterone protects the integrity of the endometrium and inhibits uterine contractions during pregnancy.

Events in the ovarian phases are harmonized with the thickening and subsequent shedding of the endometrial lining in a complete menstrual cycle. During
the follicular phase of the ovarian cycle that follows menstrual bleeding, both FSH and LH levels remain fairly constant until a peak in oestradiol secretion occurs on the day before ovulation. Oestradiol acts by means of positive feedback to induce a rise in LH and GnRH which then stimulates output of both FSH and LH. The outcome is a pronounced surge in LH and a lesser rise in FSH.

At onset of the luteal phase post-ovulation, progesterone levels increase further, linked to the secretory phase of the endometrial cycle. In the absence of

Fig. 2. The ovarian and endometrial phases during the normal circamensal menstrual cycle (from Reilly et al., 1997). Progesterone is indicated by P, oestradiol by E2.
implantation, oestrogen and progesterone drop towards their lowest level as the corpus luteum regresses and GnRH, FSH and LH are no longer subject to negative feedback. The loss of endometrial lining is a result of the large drop in progesterone secretion which causes blood vessel spasms, ischaemia and death of the surface cells of the endometrium. As menstruation begins, FSH promotes follicle development and the cycle gets underway again.

Another change that is evident pre-menses is an increase in body weight. The extra weight is due to storage of water and is associated with altered potassium: sodium ratios. The fluid retention is due to a rise in aldosterone secretion (normally blocked by the elevated progesterone level) which stimulates the renin-angiotensin system and causes an increase in anti-diuretic hormone. The extra weight could be a disadvantage in sports events such as jumping or hurdle races where body mass has to be lifted repeatedly against gravity.

The quality of sports performance is influenced by psychological entities such as attitude, motivation and a readiness for strenuous effort. In normal eumenorhoeic women in their twenties, mood factors have been found to change consistently with menstrual cycle phase (O’Reilly & Reilly, 1990). Positive moods were pronounced in the follicular and post-ovulatory (early luteal) phase whereas more negative moods were evident preceding and during menses (Table 1). Although moods are essentially labile, these variations should nevertheless be taken into account by mentors and coaches of female athletes engaged in physical training programmes.

**Menstrual cycle phase and exercise**

The predisposition for strenuous physical performance can be influenced by menstrual cycle phase. An appreciable proportion of women experience dysmenorrhea or painful menses and are unlikely to be able to concentrate on maximal exercise in such circumstances. Regular exercise training appears to ameliorate the problem of abdominal cramps, probably due to lowering the levels of prosta-

<table>
<thead>
<tr>
<th></th>
<th>Menses</th>
<th>Follicular</th>
<th>Ovulatory</th>
<th>Luteal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tension-anxiety</td>
<td>13.2 ± 3.4</td>
<td>8.2 ± 1.9</td>
<td>6.9 ± 2.5</td>
<td>14.4 ± 3.5</td>
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<tr>
<td>Depression</td>
<td>16.1 ± 6.7</td>
<td>8.1 ± 4.2</td>
<td>8.3 ± 5.1</td>
<td>17.4 ± 6.2</td>
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<td>Anger-hostility</td>
<td>3.9 ± 10.0</td>
<td>4.4 ± 2.3</td>
<td>4.7 ± 3.4</td>
<td>7.4 ± 3.0</td>
</tr>
<tr>
<td>Vigour-activity</td>
<td>9.0 ± 2.4</td>
<td>16.2 ± 1.9</td>
<td>22.7 ± 1.3</td>
<td>12.2 ± 2.3</td>
</tr>
<tr>
<td>Fatigue-inertia</td>
<td>11.2 ± 2.8</td>
<td>5.5 ± 0.7</td>
<td>1.5 ± 0.7</td>
<td>10.4 ± 1.8</td>
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<tr>
<td>Confusion</td>
<td>9.1 ± 1.4</td>
<td>4.8 ± 1.0</td>
<td>2.8 ± 1.4</td>
<td>8.2 ± 2.0</td>
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The syndrome of premenstrual tension (PMT) is linked with increased prostaglandin production and relieved by administration of prostaglandin inhibitors. In its extreme form PMT is characterised by irritability, aggression, confusion and sometimes abnormal behaviour. In less extreme forms subjects may feel anxious, tired and unable to relax. A diurnal variation in bouts of irritation has been linked with fluctuations in blood glucose, irritation peaking in late morning when breakfast is missed (Dalton, 1978). Alternative mechanisms linked with PMT have implicated a range of neurotransmitters including monoamine oxidase and serotonin (5HT), the latter leading to the use of 5HT-reuptake inhibitors in treatment.

There is evidence that women are more vulnerable to errors during pre-menses and this has been reflected in the incidence of injuries incurred by Swedish soccer players (Moller-Nielsen & Hammar, 1989). Oral contraceptives are used to regulate the cycle by some sportswomen and the Swedish researchers found fewer traumatic injuries in those players using contraceptive pills to reduce pre-menstrual and menstrual symptoms of discomfort.

Exercise performance is not necessarily impaired during any stage of the menstrual cycle. Indeed, Olympic gold medals have been won and world records set at all stages of the menstrual cycle. In contemporary society female participation in professional sport is widely accepted as is the principle that menses is no bar to training or competing. Nevertheless the experience of women athletes is variable and it is relevant to consider how the alterations during menstrual cycle phases might affect physiological responses to exercise, and hence exercise performance. An associated issue is the extent that strenuous training effects changes in the normal menstrual cycle, thus offsetting potential impairments and enabling the female athlete to adapt to the training level.

**Physiological responses to exercise**

Physiological responses to continuous exercise may be influenced by the variations in endocrine hormones that occur according to menstrual cycle phase. Progesterone, for example, as well as possessing a thermogenic effect can also have consequences for minute ventilation ($\dot{V}_E$). Elevations in $\dot{V}_E$ response to exercise during the luteal phase have been reported (O’Reilly & Reilly, 1990), attributed to the corresponding surge in progesterone and associated with a higher rating of subjective exertion. The increased $\dot{V}_E$ would effect a greater output of $CO_2$ but does not seem to impair maximal oxygen consumption ($\dot{VO}_2_{max}$). The substrate for oxidative metabolism can influence performance in prolonged sustained exercise. In endurance athletic events lasting 90 min or more, the level of performance may be determined by prestart stores of glycogen in liver and muscle. Mechanisms that increase these depots or spare existing stores by increasing fat oxidation can enhance overall performance. The elevated levels of progesterone and
oestrogen during the luteal phase of the menstrual cycle might benefit submaximal exercise of long duration by diminishing the utilization of glycogen. This view is corroborated by the finding of increased free fatty acids during exercise in the luteal phase (De Mendoza et al., 1979) and lowered levels of blood lactate (Jurowski-Hall et al., 1983). Eumenorrheic athletes have a lower respiratory exchange ratio (RER) in mid-luteal compared to mid-follicular phases of the menstrual cycle during exercise at 35% and 60% of VO2 max (Hackney et al., 1994). The mechanism for altering fuel utilisation is likely to be a hormone-sensitive lipase which promotes lipolysis and is activated by the hormonal changes in the luteal phase. The time-span over which performance might be enhanced is likely to be only a matter of 3–4 days, before progesterone falls pre-menses. This possible enhancement may be counteracted in a competitive context at other phases of the menstrual cycle when catecholamine secretion, which leads to similar effects, is increased in the course of competitive stress.

Low oestrogen levels may have an adverse influence on human strength. The effect has been demonstrated in ovariectomized mice whose force production was impaired after surgery. The ergogenic effect of oestrogen has also been demonstrated in post-menopausal women when the adductor pollicis muscle (which draws the thumb in over the palm of the hand) was isolated for measurement of isometric force under experimental conditions (Phillips et al., 1993). The active stretch force - the tension within the muscle in response to its being stretched - is not impaired and the weakness can be offset by hormone-replacement therapy. This loss of strength in the muscles of ageing females may accentuate the loss in bone strength due to demineralization. A cyclical variation of muscle strength with changes in oestrogen levels during the normal menstrual cycle is difficult to show. Indeed, Greeves et al. (1997) have claimed that progesterone rather than oestrogen may play the dominant role in influencing muscle performance. It should also be emphasised that gross muscular strength is influenced by a host of factors other than circulating hormone levels.

The menstrual cycle phase can also be relevant in ergonomics contexts when physiological responses to physical work are being interpreted. In 16 eumenorrheic subjects studied by Birch and Reilly (1997), the heart rate response to a standard 10-min lifting task was elevated by 10 beats min⁻¹ in the post-ovulation phase of the cycle. Although the impact of the menstrual cycle on lifting performance was minimal, the recommendation was that the changes in heart rate should be taken into account in any international determination of standards for manual handling of physical loads. The conclusion was that in studies employing heart rate as a variable for examining fatigue, risk of injury during repetitive lifting or predicting maximal oxygen consumption or maximal acceptable load, allowance must be made for the menstrual cycle phase.
Effects of exercise on the menstrual cycle

Delayed menarche has been linked with the more advanced competitive levels in girls’ running, but also with low body mass and low percentage body fat. Menarche is markedly delayed in girls who start systematic training at an early age and whose training regimens entail very high overall energy expenditure. These females include ballet dancers and gymnasts, average age of menarche in these groups being 15.4 (Warren, 1980) and 15.0 (Marker, 1981) years, respectively. The delay in menarche, potentially caused by strenuous training, has no subsequent adverse effect on female reproductive processes. Females engaged in strenuous athletic training programmes are known to experience disruption of the normal menstrual cycle. One irregularity is a shortened luteal phase (Bonen et al., 1981). Secondary amenorrhea or absence of menses for a prolonged period, is also regularly reported. So-called ‘athletic amenorrhea’ is linked with low levels of body fat, low body weight, energy imbalance and high training loads, while psychological stress is also implicated.

Secondary amenorrhea is associated with low values of body fat, though the mechanism responsible for a link between them has not been clearly established. Endurance training lowers body fat which in turn leads to a reduced peripheral production of oestrogens through aromatization of androgens, catalysed by aromatase in fat cells. The peripheral production of oestrogens is thought to be important in stimulating the hypothalamic-pituitary-ovarian axis. Hard exercise or intentional weight reduction will lower pituitary FSH secretion, prevent follicular development and ovarian oestrogen secretion, and decrease progesterone secretion.

Exercise-induced amenorrhea occurs in 20% of female athletes, compared to a prevalence of 5% in the general population. In runners, the prevalence increases linearly with training volume to nearly 50% in all athletes covering 130 km per week overall (Drinkwater, 1986). This linear increase is not found in swimmers and cyclists as these athletes do not have to support their body weight during exercise and their bones are not subject to the same repetitive loads as in the runners. Stress may also be implicated in the occurrence of amenorrhea. In a study of British athletes, a sample of international, club, and recreational distance-runners was divided into those who were amenorrheic, oligomenorrheic (irregular) or had regular (normal) menstrual cycles (Reilly & Rothwell, 1988). The amenorrheic athletes were younger and lighter, had less body fat, experienced more life stress, had a higher training mileage, and trained at a faster pace than the other groups (Table 2). A high frequency of competition was the most powerful discriminator of the amenorrheic individuals from the other groups. This finding supports the possibility that increased outputs of catecholamine, cortisol, and endorphins interfere with the normal menstrual cycle by down-regulating the hypothalamic-pituitary-ovarian axis. Training-induced amenorrhea does not nec-
THE MENSTRUAL CYCLE AND HUMAN PERFORMANCE

The menstrual cycle and human performance: Ovulation can occur spontaneously and fertility can be restored after a long absence of menses. Exercise-related menstrual disturbances are quickly reversed with a reduction of high training loads (by about 10%), and a marginal increase in body fat (up to 2 kg in weight).

The so-called ‘athletic triad’ is a contemporary concern to sports medicine practitioners. The triad refers to the links between amenorrhea, eating disorders and bone mineral loss. Demineralization in bone is a result of prolonged low levels of circulating oestrogen which upset the balance between bone resorption and bone remodelling. The result is a reduction in bone density akin to the rapid bone loss which occurs post-menopause. Although a moderate level of exercise stimulates bone growth and reverses bone loss in older women, decreased bone density and occurrence of stress fractures reflect overtraining, particularly in younger distance runners. Reducing the training load and decreasing the frequency of racing helps to restore the normal menstrual cycle, as mentioned earlier. However, the interactions between training parameters and liability to osteoporosis have yet to be fully explored. Sparling et al. (1998), for example, reported that whole-body bone mineral density for the 1996 United States Olympic women’s field hockey team was 13% higher than normative standards. This contrasts with the many reports of premature bone loss and high rates of stress fractures in female endurance runners and gymnasts.

Oral contraceptives

The reproductive process can be prevented by the oral administration of pills, known as oral contraceptives. These work by blocking the normal hormonal feedback mechanisms and inhibiting ovulation. Oral contraception may also be used to treat menstrual discomfort and to stabilize the menstrual cycle. Athletes may use oral contraceptives to ensure that important competitions do not coincide with menses.

| TABLE 2. Factors related to amenorrhea in a sample of British athletes (Reilly & Rothwell, 1988). |
|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|
| Amenorrheic | Oligomenorrheic | Normal |
| Age (years) | 25.4 (5.7) a | 31.8 (7.3) | 32.7 (6.6) |
| Body mass (kg) | 50.0 (5.4) a | 55.3 (3.9) | 57.0 (6.9) |
| Body fat (%) | 16.1 (4.3) b | 27.5 (2.8) | 25.6 (2.9) |
| Life stress/year | 693 (393) a | 286 (234) | 494 (282) |
| Years running | 6.0 (4.6) | 5.0 (6.4) | 5.3 (4.8) |
| Miles/week | 55 (25) | 44 (27) | 34 (20) |
| Pace/mile (min) | 7.0 (1.0) | 7.6 (1.4) | 7.9 (1.1) |
| Races/year | 29.1 (14.0) a | 9.1 (7.2) | 16.3 (14.0) |

*p < 0.05; *p < 0.01 compared with normal.
The administration of oestrogen or progesterone in appropriate amounts in the follicular phase can prevent the preovulatory surge of LH secretion that triggers ovulation. Contraceptive pills contain combinations of oestrogen in small amounts and progestins (substances that mimic the actions of progesterone), since excess of either type of hormone can cause heavy bleeding. Medication is started early in the cycle and is continued beyond the time that ovulation would normally have taken place. Administration can then be stopped to allow menstrual flow to occur as usual and a new cycle to commence. The use of contraceptive pills demonstrates that the menstrual cycle is produced by a series of feedback loops.

The relationship between exogenous oestrogen consumption and cardiovascular disease, with particular reference to the exercising female, has been reviewed elsewhere (Birch, 1997). There is disagreement about whether or not oral contraceptives affect the performance of exercise. The adverse effects of body-water retention may have been due to the particular combination of hormones used in early generations of contraceptive pills. If use of the contraceptive pill prevents adverse menstrual effects, the result is likely to be beneficial. Their main influence is probably that performance is more consistent than normal, since they stabilize the fluctuations in peptide and steroid hormone concentrations linked with the menstrual cycle.

Reilly and Whitley (1994) investigated the effects of menstrual cycle phase and oral contraceptive use on endurance exercise at 70% \( \dot{V}O_2 \text{max} \) to exhaustion. The groups, consisting of eumenorrheic cross-country runners and a corresponding group of runners taking monophasic oral contraceptive preparations with constant doses of steroid hormones for 21 days, were matched for age, height, body mass, body fat, \( \dot{V}O_2 \text{max} \), running experience and training volume. Exercise was sustained for longer during the luteal phase in the non-users of contraceptives whereas performance was stable in the runners using oral contraceptives. The enhanced performance in the luteal phase was linked to a favourable alteration in lipid metabolism. In a subsequent study, the same researchers (Reilly & Whitley, 1995) employed a similar research design but incorporated an all-out anaerobic test (treadmill run at 12.8 km h\(^{-1}\) and 20% slope). Results revealed that the enhancement of sustained aerobic performance in the luteal phase in the eumenorrheic athletes did not extend to anaerobic endurance efforts. The users of oral contraceptives had similar results during the contraceptive use and non-contraceptive phases of their dosage.

Circadian and circamensal rhythms
There are many questions relating to potential effects of menstrual cycle changes on human circadian rhythms. The existence of circadian rhythms in exercise performance have been explored largely in male subjects. It has not been established how the elevation in body temperature observed at ovulation affects the
thermoregulatory responses to exercise at different points of the day, or the capability to tolerate thermal stress.

Disruption of the circadian rhythm as a result of long-haul flights may affect female travellers, as evidenced by the higher than normal incidence of amenorrhea in Finnish flight attendants. Disturbances of the menstrual cycle in women travellers have been linked to disruptions in melatonin secretion (Harma et al., 1994) which in turn affect luteinizing hormone and progesterone. The extent to which the menstrual disturbances accompanying multiple time-zone transitions alter the performances or exercise capabilities of female athletes over and above the effects of jet-lag on their male counterparts is not known.

There is a progesterone-mediated effect on melatonin which affects the circadian rhythm in body temperature (Cagnacci et al., 1996). The effect is evident during the luteal phase in a delay of the nadir in body temperature and a reduction in amplitude. How these changes affect the adjustment of circadian rhythms of female long-haul travellers remains to be established.

**CONCLUSIONS**

The tremendous improvements in the sports performances of elite women athletes have illustrated how interactions of exercise with the normal menstrual cycle can be overcome. Some activities, notably gymnastics and ballet, are associated with a delayed menarche. Secondary amenorrhea is linked with impairment of the hypothalamic-pituitary-gonadal axis in highly competitive endurance athletes. A further related problem is reduced bone mineral density when amenorrhea persists for a prolonged period and is linked to the resulting low levels of oestrogen. Disturbed eating behaviour completes the ‘athletic triad’. Performance is stabilised by use of oral contraceptives, although the contents of the pills require regular updating of research. The study of circadian rhythms in exercise performance of female subjects has lagged behind the chronobiological research on male athletes. Disturbances of melatonin secretion in women travellers are linked with an increased incidence of amenorrhea. The influence of travel stress on female athletes is likely to be a fruitful area of research in the future.

**REFERENCES**


