The Menstrual Cycle and Sport Performance

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ABSTRACT

Since the first modern Olympic Games there has been increased female participation in high-level sport competitions and this growth has been exponential in the last 60 years. Among the issues linked to women in sport is the role of the menstrual cycle in performance. While a stable endocrinological condition is observed in males, the large variations between the follicular phase and luteal phase in women's cycles make it difficult to identify a comparable stable condition, which has practical implications for training and competition. Moreover, there are a number of menstrual disorders that can affect and/or are linked to intensive physical training. In this article, the authors review the existing literature on all key aspects of the menstrual cycle and sport performance. Their aim is to support coaches and others working to improve the performance of female athletes by providing short overviews of the following areas: a) the menstrual cycle, b) physiologic and metabolic variations in the menstrual cycle, c) adaptation of training to menstrual phases, d) menstrual disorders in athletes and the Female Athlete Triad, e) Dysmenorrhoea in athletes, f) oral contraceptives and performance.

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Introduction

At the first modern Olympic Games in Athens in 1896, no women took part because it was thought "impractical, uninteresting, unaesthetic and incorrect". Since then, however, there has been greater cultural acceptance of women in sport and increased female participation in high-level competitions. This growth has been exponential in the last 60 years. It could be said that the trend started at the second edition of the Games, in Paris in 1900, when women's events were introduced in tennis, golf, sailing, equestrian and croquet, although only the tennis and golf were exclusively female competitions. Just over a

century later, the 2012 Games in London were called the “Women’s Games” and hailed by the International Olympic Committee as "an historic step towards gender equality", as they were the first where women competed in all sports on the programme and 45% of participants were female (up from 10.5% in 1952).

The 1928 Games in Amsterdam were the first where women’s athletics events were included, despite the fact that organised track & field competitions for women had been staged at least since the last decade of the 19th century and the *Fédératiion Sportive Féminine Internationale* had begun maintaining a list of women’s world records in 1921. Nowadays, the international championship programmes currently offered for men and women are more or less equal (only men have a 50km walk and women have the Heptathlon instead of the Decathlon) and the prize money at the IAAF World Championships in Athletics is the same for both men’s and women’s events.

With the increased participation and the development of training methods, the gender difference in human performance has decreased and in some athletics disciplines a narrowing between elite male and female records has been observed. However, the gap in performances, particularly in anaerobic sprint events, started to increase again after 1988 together with improvements in doping control[1], which suggests that the performance evolution was probably also supported by other external factors.

Among the issues linked to women in sport is the role of the menstrual cycle in performance. While a stable endocrinological condition is observed in males, the large variations between the follicular phase (FP) and luteal phase (LP) in women’s cycles make it difficult to identify a comparable stable condition, which has practical implications for training and competition. For this reason, a number of scientific studies have been conducted, normally during in the FP when the ovarian estrogens and progesterone are at their lowest level.[2] In fact, female athletes compete continuously and independent of their physiologic condition or menstrual phase and for this reason changes of different human body systems should be examined in comparable periods of menstrual cycle.

The purpose of this article is to review the available studies and the rest of the existing literature for current knowledge on all key aspects of the menstrual cycle and sport performance. Our aim is to provide support for coaches and others working to improve the performance of female athletes through brief overviews of the following areas:

- the menstrual cycle;
- physiologic and metabolic variations in the menstrual cycle;
- adaptation of training to menstrual phases;
- menstrual disorders in athletes and the Female Athlete Triad;
- dysmenorrhoea in athletes;
- oral contraceptives and performance.

The Menstrual Cycle

In a typical menstrual cycle of 28 days, the first five days of menses are characterised by shedding of the uterine lining, which is then followed by new proliferation of mucosa in preparation for an egg. The first phase usually lasts 14 days and is called the follicular phase (FP). After ovulation, normally in day 15, the luteal phase (LP) starts, lasting until the day before menses, normally about day 28.

A complex system modulates the steroid hormones, predominantly estrogen and progesterone, which in turn are regulated in a feedback system by luteinizing hormone (LH) and follicular stimulating hormone (FSH) secreted by the pituitary gland. The cycle continues from the beginning of the first menses in young women until menopause. The pituitary gland is modulated by the hypothalamus, through gonadotropin-releasing hormone (GnRH), and influenced by a number of factors, including stress and exercise, body metabolic status, psychological factors, etc. (Figure 1).

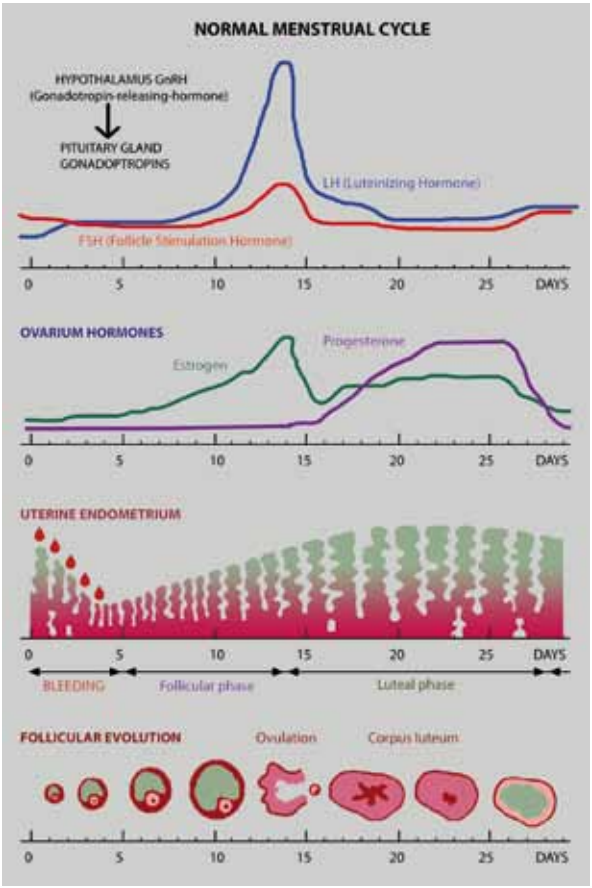

Figure 1: The hormonal mechanism in the menstrual cycle

In epidemiological studies, large differences are reported regarding the effects of the phases of the menstrual cycle on sport performance: no effects on competitive results are described by many interviewed women athletes (some of whom have won medals in major events) while others complain of detrimental effects on body feeling and performance.

These negative effects, including abdomen pain, cramps, water retention and mood changes, are connected particularly with the premenstrual week in the luteal phase (LP) and/or the first days of the cycle and with the bleeding itself, which is linked with increased fatigue and higher incidence of physical injuries.[7] In fact many women athletes and non-athletes alike, are familiar with some symptoms of the late LP, particularly headaches, fatigue, bloating, backache and cramping, breast tenderness, abdominal constriction and the feeling of fatigue with increased exertion: the so-called pre-menstrual syndrome (PMS). Conversely, the immediate post-menstrual days are thought by most of athletes to be the best period for sports performance.

The literature also reports increases in musculoskeletal and joint injuries during the luteal phase, probably related to increased relaxin levels and increased flexibility and elasticity of connective tissue, particularly in articular joints. Relaxin secretion seems correlated to ovulation, and its level rises about six days after luteinizing

hormone peak. Notably high levels have been detected in women using oral contraceptives, which probably induce relaxin secretion.[48,49]

Investigation of the effects of the different phases of the menstrual cycle on performance is made difficult by the frequency of non-ovulatory cycles (up to 12%) or luteal phase deficient cycles (up to 43%) and the unstable length of the follicular phase, even in athletes with apparent normal menstrual bleeding. It can be said that "not all women who menstruate regularly, ovulate regularly".[3] For these reasons, a simple count of days from the onset of menses will never give a sure indication of ovulation day.

Moreover, there are issues with most alternative methods for predicting ovulation. For example, there is the method of monitoring basal body temperature (BBT), which increases 0.3°C after ovulation and might be expression of onset of the luteal phase. However, not all women have an increase of BBT, and not all increases of BBT reflect an increase of the progesterone level. Then there is the imprecision of urinary luteinizing hormone (LH) peak concentration determined by home kits, which reflects blood LH peak, which in turn predicts ovulation within the next 14-26 hours. Measuring other hormones from urine or saliva is not practical (low reliability of the levels in saliva and practical difficulties in determining the levels of estrogen and progesterone or their metabolites in urine collected in 24 hours).[4]

Therefore, the current most scientifically valid method for determining the time of ovulation is through hormonal measure in the blood serum. Based on serum levels, three phases can be identified and used for comparing the effects of different periods of menstrual cycle on the body systems:

- early follicular phase: low estrogen and low progesterone;
- late follicular phase: high estrogen and low progesterone;
- mid luteal phase: high estrogen and high progesterone.

Keeping in mind possible circadian variations, it is important to note that:

1. Progesterone levels are higher in the morning.

2. Exercise, as opposed to rest, increases both estrogen and progesterone.

3. The estrogen/progesterone "ratio" is as important as the "absolute" levels, for the different effects that even similar levels of estrogen might have in the presence of different progesterone levels.

Physiologic and Metabolic Variations in the Menstrual Cycle

Some studies have shown that normally menstruating women appear stronger in the mid luteal phase, which is attributed to the higher progesterone levels in that period compared to the late follicular phase, with its higher estrogen level, when the lowest strength levels have been recorded.[5] However, with a critical examination of the literature and taking into account the possibility of non-ovulatory or luteal phase deficient cycles, no clear evidence can be found that the fluctuations in female steroid hormones in the menstrual cycle affect muscle strength or fatigability.[6, 7, 8, 9]

With regard to maximal oxygen consumption (VO_2max), different results have been found based on the interaction of menstrual cycle with food intake, fuel storage and mobilisation, hydration state, heart rate and cardiac output and general nutritional status. Generally no large changes are observed, even if, concerning the lactate metabolism, a possible improvement in exercise time to exhaustion during the mid luteal phase (LP) (with an uncertain increase in VO_2max) has been decribed.[10,11] It seems that an enhanced lipid metabolism might be present during the LP, with spared glycogen and lower lactate response to exercise, in contrast to the higher lactate levels observed in the mid follicular phase (FP). These effects seem to depend mainly on estrogen, as a longer exhaustion time

was observed in the mid LP when the estrogen level was increased and the estrogen to progesterone ratio was higher than in the early FP.[12, 13, 14]

There is a tendency of women to refer to an increase of body weight during the luteal phase (LP) and the first days of menstruation, and similar reports can be found in some older studies conducted without attention to the verification of hormonal status. A short peak in bodyweight is also reported just a couple of days after ovulation. This bloated feeling could be a consequence of a different distribution of fluids in the body or the vascular system more than a real change in water retention or excretion. However, some studies have shown that plasma volume increases within two days of estimated ovulation and in the mid to late LP, while the lowest plasma volume has been observed in the early LP.

Haemoglobin (Hb) and haematocrit (HCT) should have an opposite behaviour to plasma volume, with an increase in the early luteal phase (LP) (15th to 19th days of the cycle), and a mild reduction in the late LP. Of course, minimal plasma volume and Hb and HCT changes might be observed during the menstrual bleeding phase, which in 80% of females represents a blood loss in the range of 10-90 mL (mean value 43 ml), with more than 80 mL in 11% of females.[4]

Plasma electrolyte (sodium, potassium and chloride) concentrations seem higher during the follicular phase and lower during the luteal phase. Bicarbonate levels seem lower on the menstruation days and during ovulation, which might concur with increased fatigue during the menstrual phase.[2]

Of course, fluid and food intake, together with hydration state of the body and exercise level, might influence the above-mentioned changes and help to explain the wide and contrasting results observed in different studies.[8, 9]

Resting heart rate seems slightly higher in the mid luteal phase (LP). This is linked to increased plasma volume and probably to an increase, even if low, of the body temperature, with secondary effect on the sino-atrial node. Ventilation is also shown to increase in the LP, probably a central effect of progesterone on the hypothalamus or it could be due to the influence of body temperature. In any case, increases in either heart rate or ventilation are not evident during exercise.[15]

Conflicting findings are reported on the change of rectal and skin temperature at rest or during exercise (unchanged or increased during the LP). However, increased basal body temperature (BBT), heart rate and ventilation in the mid LP are partially balanced by an increased thermoregulatory set point (from the effect of progesterone on central sensitive neurons of preoptic area).[4] Unfortunately, the increased BBT, heart rate and ventilation at rest, during the mid to late LP, seem to give a subjective feeling of higher exertion or strain, which is linked to a decrease in prolonged exercise performance. These individual feelings might negatively impact training and/or competition performance, particularly in hot and humid environmental conditions. While training sessions might be freely adapted to the weather conditions, in the case of fixed competition date, an adjustment of menstrual cycle phase could be helpful.[7,15]

Reduced reaction time, neuromuscular coordination and manual dexterity have been observed during the pre-menstruation and menstrual phases. [16]

The level of androgens in athletes is a particularly interesting field.[17] The ovaries and the adrenal glands, stimulated by luteinizing hormone, produce testosterone in low amounts (in men the higher testosterone amount, 95%, is secreted by Leydig cells in testes) and androstenedione, while dehydroepiandrosterone (DHEA) is produced only by adrenal glands, stimulated by adrenocorticotrophic hormone (ACTH). The major portion of these androgens is chemically linked to sex hormone binding globulin (SHBG), while only a small amount (1-3%) is free. Androgens can be converted, in peripheral tissues to more potent androgens, namely testosterone

and 5-alfa dihydrotestosterone (DHT), or when stimulated by follicular stimulating hormone, they can be aromatised, in fat tissue, to estrogen (estrone and 17-beta estradiol).

Androgen levels may be influenced by the menstrual cycle, physical exercise, age, diet and contraceptives. In addition, physiologically, acute exercise induces increased levels of androgens in women (as well as in males) through the following pathways:

- decreased liver metabolism (by reduction of liver blood flow during intense exercise);
- increased synthesis in the adrenal glands (stimulated by higher secretion of corticotrophin releasing hormone (CRH) from hypothalamus and adreno corticotrophic hormone (ACTH) from the pituitary gland);
- increased release of prolactine (PRL) from the pituitary gland (stimulated by both exercise and body temperature change), which, conversely, has a negative feedback on gonadotropin releasing hormone (GNRH) production from the hypothalamus;
- reduced conversion to estrogens (due to the reduced amount of fat cells).

Both possible decreases and unaltered levels of circulating androgens were found in different studies on the effects of chronic endurance exercise training.

Adaptation of Training to Menstrual Phases

All athletes are individuals and may respond differently to similar training plans. Continuous adaptation and modifications in real time of training sessions, according to the recorded field and body answers, may increase performance and reduce the risk of injuries. Coaches and the rest of a female athlete's entourage should be conscious of the state of the individual athlete and that within a training group of females "one rule does not fit for all". Female athletes should individually monitor their menstrual cycles and be conscious of their physical and emotional states; exercise should be correlated with the needs of athletic performance and with subjective status, which will permit to them to know the best training days and the days with performance impairment. Training should be scheduled accordingly, by correctly planning strenuous sessions and necessary rest opportunities.[18]

The following points may be useful:

- volume (duration of training, number of repetitions), intensity (strength, speed and overload), and technically specific difficulties should be modulated, according to the periods of possible increased injury risk;
- hydration and nutrition should be focused to optimise recovery from training stress and replacement of fuel stores;
- considering the tendency towards testosterone peaks around ovulation, it may be beneficial to plan for more intense strength training sessions during this period of the menstrual cycle.

Menstrual Disorders in Athletes and the Female Athlete Triad

Although the typical duration of the menstrual cycle is 28 days as mentioned above, this in fact applies to no more than 60% of women. The cycle length in any individual is influenced by a number of factors including age, weight, environment, diet, ethnicity, stress and training. It is therefore important to list the most common abnormalities and disorders connected with duration or absence of cycles:

Eumenorrhoea: menstrual cycles of 26-32 days, within a 1% variation;

Oligomenorrhoea: menstrual cycles > 35 days;

Luteal Phase defects: lower levels of oestradiol, luteinizing hormone and progesterone, with normal, even if delayed, ovulation (the shortened and lowered progesterone secretion results in infertility);

Anovulation: estrogens and progesterone levels too low for ovulation, but enough for menstrual bleeding;

Amenorrhoea: persistent absence of menses for three months or more, irregular luteinizing hormone pulsatility with suppression of follicular development and ovulation, low estrogens and progesterone provoking the absence of endometrial proliferation;

Primary Amenorrhoea: never menstruated, even if with puberty changes (menarche thresholds considered at 15 years age);

Secondary Amenorrhoea: onset after menarche.

The incidence of menstrual disorders between five and 30 years of gynaecological age (the difference between chronologic age and menarcheal age), is higher in sportswomen (20-22% present Oligomenorrhoea), than in normal sedentary women (less than 10%), and is especially prevalent in younger athletes (up to 40% in the first years of gynaecological age), which it could rise to 70-80% when non-ovulatory and luteal phase deficient cycles are included.[19]

The higher incidence of menstrual disorders in athletes could be partially explained by the selective mechanisms in sport. For example, girls with delayed menarche might excel in certain sports (gymnastics), delayed closure of bone growth plates could permit a self-selection of higher height phenotypes for other sports (basketball, volleyball), low weight is an advantage in endurance activities (such as the marathon) and some diseases such as Polycystic Ovarian Syndrome (PCOS) might give advantage in strength events because of higher androgen levels. Girls and women with these conditions may tend to stay in a sport and develop their skills while others move on because of a relative lack of success.

On the other hand, many female athletes are pushed to decrease their body weight in order to improve performance. Lower body weight, high exercise loads (especially endurance training) and younger age are the common predisposing factors for Oligomenorrhoea and Amenorrhoea. In athletes, these conditions are sometimes the result of sudden (instead of progressive) increases in training loads, probably linked to individual predisposition or different diet and recovery strategies, even if at other times athletes with similar exercise regimens show different behaviour. [20,21]

Different mechanisms are proposed to explain and characterise menstrual disorders in athletes, starting with the body's "adaptation" to increased workload to what might become a serious "pathologic state":

- Some studies show that a 17% minimum of fat mass is needed for menarche, and 22% for regular menstrual cycles (in fat the androgens are partially transformed to estrogens), but such a strict relationship between body weight and height is not fully confirmed by all studies.

- Stress with associated altered gonadotropin-releasing hormone (GnRH) pulsatile secretion might play a key role, by causing a lower or altered or absent pulsatility of luteinizing hormone (LH), even if sometimes the LH pulse seems normal.

- Dietary availability associated with GnRH and with other factors also plays a role. In fact, it is suggested that in cases of acute increase of training volume, even in the presence of altered GnRH pulsatility, a complete and adequate dietary intake might prevent menstrual disorders.[19]

The exact pathophysiologic mechanism of Functional Hypothalamic Amenorrhoea (FHA) is very complex and unclear. Numerous neuropeptides are involved (neurotransmitters and neurosteroids), together with other substances such as allopregnenolone, neuropeptide Y, corticotropin-releasing hormone (CRH), leptin, ghrelin and beta-endorphin. Some studies connect significant changes in the release of these mentioned substances to patients with FHA.[22]

All these components together have the effect of decreased luteinizing hormone and

follicular stimulating hormone stimulation, with resulting lower levels of estrogens and progesterone. Low levels of estrogens, insulin, triiodothyronine (T3) and leptin linked with the elevated levels of growth hormone (GH), ghrelin, CRH and cortisol observed in amenorrhoic athletes (most of them as a consequence of stress or of energetic unbalance and of inadequate carbohydrates/fat or food availability), are concomitant factors able to produce other serious consequences in the female body.

There is a complex interrelationship between menstrual dysfunction, eating disorders and low bone mineral density (BMD) - the so-called "Female Athlete Triad" - evolving to serious, even if not at the same time, medical conditions of Anovulation and Amenorrhoea, stress fractures and Osteoporosis, and Psychopathologic Anorexia, which has been well known for many years.[23, 24, 25]

Athletes, coaches and parents should be aware of the clinical conditions of the Triad in order to educate and prevent problems, or to correctly react if the athlete presents some of these conditions. The annual health exam could be an excellent opportunity to address the topic but in cases where treatment is needed a multidisciplinary team must intervene. Increased energy availability in response to training workload increases seems crucial for the prevention of the Triad but a reduction of exercise intensity is necessary for recovery. Hormonal replacement therapy (HRT), by oral contraceptive pills (estrogens), alone is not able to reverse a pathologic condition, if restoration of body weight and an increase of body mass index (BMI) are missing. [26, 27, 28]

Not all women displaying Amenorrhoea have a hormonal pattern consistent with exercise induced menstrual changes, particularly when Hyperandrogenism with Hirsutism are observed. Some authors suggest that Oligomenorrhoea when not linked to eating disorders might be the presentation of various phenotypes of Polycystic Ovarian Syndrome (PCOS). The presence of an elevated luteinizing hormone to follicular stimulating hormone ratio, elevated androgens and low sex hormone binding globulin (SHBG) in cases of Oligomenorrhoea with

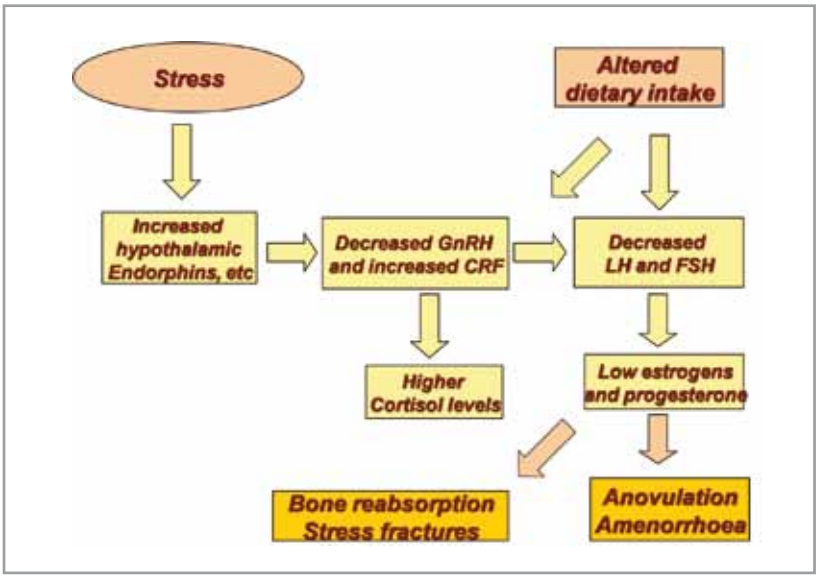

Figure 2: The "Female athlete triad" diagram

tendential Adiposity, might be expression of Hyperandrogenism linked to PCOS, instead of the Female Athlete Triad. [29, 30, 31]

Dysmenorrhoea in Athletes

According to the literature definitions, Dysmenorrhoea is a chronic and cyclic pelvic pain, sometimes spasmodic and generally associated with menstruation even in the absence of identifiable pathology; it is typically known as "menstrual cramps" or "period pain". It occurs before or during menstruation, with a peak in the first two days, is a pain in the lower abdomen or lower back that is sometimes associated with diarrhoea, nausea, vomiting, flushing, backache, headaches, concentration difficulties, insomnia, fatigue and in rare cases Syncope. Primary Dysmenorrhoea starts most frequently two to three years after menarche, is very frequent in adolescent women, and lowers in intensity after many years (partial adaptation). Secondary Dysmenorrhoea, on the other hand, appears after a number of painless years, and is generally associated with pelvic diseases or abnormalities. [32, 33]

Dysmenorrhoea seems to be due to excessive production of uterine endometrial prostaglandins (PGs) and vasopressin. The excessive uterine contractility, together with relative Ischemia, produces the typical pain, while the mentioned PGs induce systemic symptoms. Very intensive short duration exercises sometimes produce intense and acute pelvic pain independent of menstrual cycle. Non-steroidal anti-inflammatory drugs (NSAIDs) relieve the pain of Primary Dysmenorrhoea by prostaglandin (PG) reduction (by cyclo-oxygenase inhibition).[34] Oral contraceptive pills (OCPs) are frequently used for the same purpose, as they can reduce PG levels and the myometrial activity, but their real effectiveness is not confirmed. [35, 36]

Although there is no scientific consensus, endurance exercise is thought to reduce the prevalence of Primary Dysmenorrhoea, preventing or decreasing symptoms in at least 20% of women through a sort of adaptation training. The associated increased production of beta-endorphins (working as natural analgesic, and also reducing the effects of stress) and the partial suppression of PG release might elevate the pain threshold.[33] It is also supposed that endurance exercise, by chronically shunting blood from the viscera to the working muscles, could be able to reduce the basic abdominal-pelvic congestion during menstrual cycle (Figure 3).

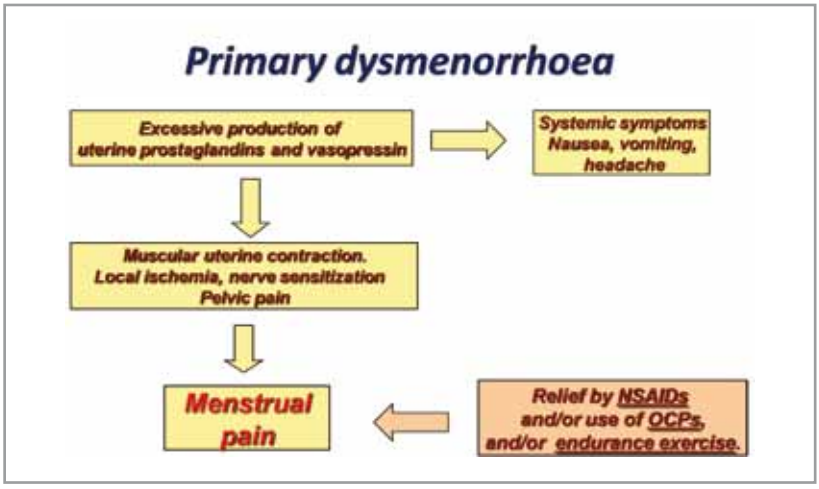

Figure 3: Aetiology and symptoms of Primary Dysmenorrhoea

Oral Contraceptives and Performance

In addition to controlling the pituitary secretion of gonadotropins and the natural production of estrogens and progesterone (inhibiting ovulation), oral contraceptive pills (OCPs) increase cervical mucus production (reducing sperm mobility) and reduce the endometrial lining (minimising the chance of implantation). Of course, the exogenous amounts produce a higher level of estrogens (3-5 times) and progestogen (1-2 times).[37] The incidence of OCPs use in female athletes is similar to the normal population, about 10-20%.

There is a tendency to use OCPs that contain both estrogens (usually ethinylestradiol) and one of the different progestogens (levonorgestrel, norethisterone, desogestrel, norgestrel, etc.). However, due to undesirable effects of estrogens (mainly nausea and tenderness), sometimes OCPs with only progestogen (or mini pills) are used. The amounts of progestogens contained in OCPs differ, based on their different potency (capacity to produce desired effects) and androgenocity (ability to produce masculine characteristics), which make OCPs differently able to counteract negative estrogenic effects and, at the same time, have different consequent impacts on physical performance. Three types of OCPs are available on the market, with formulations or dosages of synthetic hormones that are qualitatively different in various brands:

Monophasic: these have stable amounts of estrogen and progestogen in each pill and are taken for 21 days (+ seven days suspension or placebo).

Biphasic: these have a stable basic amount of estrogen in each pill, but the progestogen is dosed in two steps in two different periods of 21 days of the cycle (+ seven days suspension or placebo)-

Triphasic: these have different dosages of estrogen and progestogen for each of the 21 days.

Sometimes OCPs contain hormones for 24 (instead of 21) days, followed by four (instead of 7) days of placebo.

A frequent secondary effect reported by women using OCPs is water retention. In a normal luteal phase, the increase of progesterone initially produces water and electrolyte loss, with a secondary increase of aldosterone. When the progesterone decreases, the excess aldosterone leads to water and electrolyte retention. OCPs with high potency progestogens produce greater fluid retention and a feeling of been bloated.[38] Monophasic OCPs seem to produce fluid retention and increased bodyweight in athletes (up to a couple of kilos depending on the different potency and androgenicity of the different progestogens), which might be detrimental for some endurance disciplines. Triphasic pills seem to have lesser and delayed effects on body weight and body fat percentage, again depending on the different androgenicity of the progestogen used. In any case, the behaviour is different when comparing normal women and athletes.[39, 40]

Another secondary effect of OCPs can be an increase of body core temperature (BCT) and of thermoregulatory threshold, which could be a central effect of progestogens and might produce negative effects in endurance competitions, particularly in hot weather conditions.[41]

Apparently, women using monophasic (more than triphasic) OCPs have decreased glycogen utilisation, increased availability of lipids and higher levels of Free Fatty Acids (FFA) by estrogens, with positive effects in endurance, while sparing glycogen and increasing lipid oxidation. [42,43]

Aerobic capacity seems to be negatively influenced by triphasic OCPs (with a decrease up to 11-15% of VO_2max), maybe by direct and unclear hormone mediated cellular mechanisms in the absence of any change in haemoglobin levels, maximal heart rate, ventilation etc.[44] For this reason, endurance athletes should avoid triphasic OCPs, and, when

necessary, use the new low dose monophasic pills, in which in addition to other advantages, the possibility of fluid retention is reduced due to the low hormonal dosage.

On the other hand, in spite of possible increase of plasma volume, stroke volume and cardiac output induced by the estrogens in OCPs, no changes in cardiovascular responses were observed using low doses monophasic, or triphasic pills.[40]

The possible effect of estrogens on glycogen storage and on FFA utilisation is not useful in anaerobic performance. Even if the increased buffering capacity induced by estrogen might be helpful, no clear effect has been documented in not-endurance athletes using OCPs.[45]

It was supposed that muscle strength could be enhanced through the use of OCPs containing progestogens with high androgenicity. However, the most androgenic compounds also have some antiandrogenic properties. Moreover, there is no evidence of particular effects from the use of the recent low dosage formulations or the current triphasic pills.[46,47]

Considering that OCPs are not banned by WADA rules, they can be useful to female athletes for the following purposes:

- contraceptive effect;
- decreased menstrual pain;
- menstrual cycle control according to competition schedules, minimising the athletes concerns about menstruation during important competitions (The menstrual cycle can be manipulated by extending the duration of active pills, which can permit to delay menstruation successfully for at least 7 to 10 days. Alternatively, the OCPs can be stopped 10 days in advance, to anticipate the menstruation before the competition.).

On the other hand, some common effects cannot be forgotten, including:

- bodyweight increase;
- nausea and bloating;
- feeling of performance worsening;
- possible amenorrhoea after suspension;
- aterogenic effects (Low Density Lipoproteins (LDL) increase by progesterone; High Density Lipoproteins (HDL), Very Low Density Lipoproteins (VLDL) and triglycerides increase by estrogens), even if counteracted, in active women or athletes, by physical exercise.

Additional physiologic effects to be considered are:

- lower menstrual bleeding;
- increased mineral retention, with its preventive effect on bone demineralisation and decreased risk of stress fractures;
- improved neuromuscular coordination;
- enhanced flexibility and elasticity of connective tissues (useful in gymnastic, but dangerous in other sports). [48,49]

A large inter-individual variation has been observed concerning both positive and/or negative effects of OCPs on the performances of athletes competing in different events. Body weight increase, for example, has different consequences in a marathon runner or in a sprinter, even with the lower physiologic impact of current pharmacologic formulations, compared with the older versions.

To conclude we can say that each woman is different and each life period for an individual is different. Therefore the impact of OCPs and other external factors on hormonal status will vary greatly. Every female athlete should consult her treating medical doctor to make an analysis of her personal situation and then design with an individualised treatment approach. This process should include an evaluation of the level and amount of training as well as the different possible treatments. Any treatment should be tested or started well in advance of the competition period.

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