Chapter 5

Menstrual Cycle and Physical Effort

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Abstract

In addition to affecting the sexual organs in women, ovarian hormones have a wide impact on processes related to metabolism, water and electrolyte balance, thermoregulation, and redox balance. Differences in the estradiol and progesterone concentrations during the follicular and luteal phases, as well as the increase in the concentration of these hormones under the influence of physical exercise, may cause a different course of exercise response in women depending on the phase of menstrual cycle. Estrogens affect the metabolism of women by reducing the rate of gluconeogenesis and glycogenolysis and, at the same time, by increasing the share of lipids in covering energy requirements. Progesterone affects respiratory system parameters causing, among others, an increase in pulmonary ventilation. The resultant antagonistic action of progesterone and estradiol is the effect on thermoregulatory mechanisms. Increased estradiol concentration at the low progesterone concentration level causes water and electrolyte retention. In turn, an increased level of progesterone leads to loss of water and sodium, causing a decrease in the volume of plasma during the postovulatory phase of the menstrual cycle. The processes described above are related to metabolic changes affecting the ability to perform physical efforts.

Keywords: menstrual cycle, aerobic efforts, anaerobic efforts, acid-base balance, redox balance

1. Introduction

Knowledge about the physiology of physical efforts is mostly based on research results in which only men participated. However, the increasing participation of women in many sports disciplines encourages observation of the physiological reactions and effects regarding intense physical efforts on the body of women associated with the process of sports training [1, 2].

A decrease in the level of sex hormones during rest, as a result of heavy and long-term training, can lead to disorders in the menstrual cycle of a woman [2, 3]. These disorders most have the characteristics of rare menstrual periods (oligomenorrhea) or secondary amenorrhea (amenorrhoea secundaria). In women with regular menstruation, burdened by sports training, anovulatory cycles or shortened luteal phases often occur. It is also possible to exclude the impact of physical exercise related to training on the later menarche age [2, 4]. The factors conducive to hormonal abnormalities are large decreases in body mass and the amount of adipose tissue, resulting not only from significant training loads but also frequent disorders in the way of eating [5]. Long-lasting estrogen deficiency leads to a decrease in bone density, causing deterioration of its structure, which may contribute to osteoporosis in the future [6]. Abnormalities in the way of eating, menstrual disorders, and disturbances in bone metabolism observed in women practicing various sports were called the "triad syndrome" [2, 7, 8]. Therefore, one should look for an answer to the question on how to program women's training in order to achieve high sports results without negative consequences for their health [1].

Assessing physiological responses induced by physical exercise in women, cyclic changes in the level of sex hormones that occur during the reproductive period in every normal menstrual cycle cannot be overlooked. The menstrual cycle is the result of complex interaction of the hypothalamus, pituitary gland, and ovarian hormones, which, apart from acting on the sexual organs, exhibit a broad, nonspecific effect on various processes related to metabolism, water, and electrolyte balance or thermoregulation in women [2, 9–13]. As shown in studies on animals, differences related to estradiol levels are a factor influencing the diversified use of energy substrates [14, 15]. Distinct differences in estradiol and progesterone levels between the follicular and luteal phases, as well as an increase in the concentration of these hormones under the influence of physical exercise, may cause a different course of exercise response in women depending on the phase of the menstrual cycle [2, 10, 16].

Research suggests that estrogens affect the metabolism of women by reducing the rate of gluconeogenesis and glycogenolysis and, at the same time, increase the share of lipids in covering energy demand [17–22]. The increased use of lipids as a source of energy occurs due to the increase in the amount of free fatty acids, which results from the increased synthesis of triglycerides and in the rate of lipolysis [23]. The effect of estradiol on metabolic processes in the liver, muscles, and adipose tissue can be achieved by changing the activity of key enzymes, changing membrane permeability or indirectly through changes in hormone levels: insulin, glucagon, cortisol, hGH, or catecholamines [24, 25].

Progesterone affects the parameters of the respiratory system, causing, inter alia, an increase in pulmonary ventilation per minute (V_E) [26]. In the postovulatory phase, a higher resting level of oxygen uptake (VO_2) is also observed [27].

The resultant antagonistic effect of progesterone and estradiol is the effect on thermoregulatory mechanisms, which leads to an increase in the body's core temperature during the luteal phase of the menstrual cycle [12, 28–30].

In the woman's body, we also observe changes in the total water content associated with the menstrual cycle phase. Observations of some authors indicate that elevated estradiol

concentration at low concentration of progesterone causes the retention of water and electrolytes in the body [31]. In turn, the increased level of progesterone, by blocking the action of aldosterone in the kidneys, leads to loss of water and sodium [32], causing a decrease in plasma volume during the postovulatory phase of the menstrual cycle [33].

All processes described above are directly related to metabolic changes affecting the ability to perform physical efforts. Differences in the use of substrates and in the intensity of individual energy changes during muscle work of different intensities, resulting from hormonal changes in the course of the menstrual cycle, may affect, e.g., the activity of the adrenergic system, the amount of lactic acid formed in muscle tissue and its level in the blood, and changes in the level of acid-base balance parameters or indicators of oxidative stress [2, 10, 16, 34]. Therefore, it can be expected that cyclical fluctuations in the level of endogenous ovarian hormones in women will affect the extent and size of exercise responses. However, the test results are not unequivocal.

2. Methods

Original and review scientific publications regarding the level of cardiopulmonary reactions, thermoregulation processes, and oxidative stress as a result of aerobic and anaerobic efforts during various phases of the menstrual cycle were reviewed. Conclusions were formulated on the basis of tests in which hormonal evaluation of the menstrual cycle was conducted.

3. Exercise responses during the menstrual cycle

3.1. Aerobic exercise

It is generally accepted that the maximal oxygen uptake index (VO_{2max}) is the indicator of aerobic efficiency, the level of which is directly determined during gradual physical exercise, performed up to the individual maximum intensity ("until exhaustion"). Such effort leads to the maximum involvement of the oxidative phosphorylation process while activating the processes of anaerobic adenosine triphosphate (ATP) resynthesis (phosphocreatine, anaerobic glycolysis). VE, cardiac output, and blood oxygen capacity are important factors determining the level of VO_{2max} [35].

3.1.1. Cardiorespiratory effect

One of the first studies comparing the physiological and biochemical stress response of women at the time of the menstrual cycle, during which laboratory testing days were determined based on sex hormone levels, was carried out by Jurkowski et al. [36]. In these studies, immediately after two 20-minute physical efforts at an intensity of 40 and 70% VO_{2max}, respectively, the woman performed a physical effort at an intensity of 90% VO_{2max} "until exhaustion." The time of extreme exercise was almost twice as long in the luteal phase. In

the luteal phase, higher values of the maximum minute pulmonary ventilation (VE_{max}) were also noted. The maximum heart rate (HR_{max}) and VO_{2max} did not differ between the menstrual cycle phases [36].

However, these data are not fully confirmed in the results of research conducted by other authors. The time of continuing the effort of gradually increasing work intensity did not differ significantly between the follicular and luteal phases of the menstrual cycle [37]. Similar in both phases of the menstrual cycle, Nicklas et al. [17] found that the time of continuing work "until exhaustion " was for the intensity of 70% VO_{2max}, and McCracken et al. [38] noted it for the intensity of 90% VO_{2max}.

The results of some studies indicate a significantly higher level of resting oxygen uptake during the postovulatory phase [27, 39]. However, most authors agree that the menstrual cycle phases do not significantly affect VO_2, neither at rest nor during submaximal and maximal intensity efforts [37, 40–45].

The conclusions from laboratory tests differ regarding the assessment of the influence of the menstrual cycle phases on the level of pulmonary ventilation. Schoene et al. [46] and Das [47] reported higher resting V_E values in the luteal phase of the menstrual cycle. During stress tests, Jurkowski et al. [36], as well as Hessemer and Bruck [39], obtained higher values in the postovulatory phase. However, differences between phases in the exercise level of this parameter are not statistically significant in many studies [37, 43, 45, 48].

Significant inter-phase differences concerning the maximal heart rate were noted by Pivarnik et al. [41]. In this research, HR_{max} was higher in the luteal phase by 10 beats per minute. Higher resting and exercise heart rate in the luteal phase were also noted by other researchers [39, 46, 49]. Some authors, however, did not find significant differences in the values of HR_{max} [37, 40, 43, 45, 48].

De Souza et al. [48], comparing the physiological responses of women with normal menstrual cycles and women with secondary menstrual irregularities, obtained similar results for both groups. In eumenorrheic women, both during the 40-minute effort at 80% intensity VO_{2max} and during the graded test "until exhaustion," in the luteal phase, slightly higher levels of oxygen uptake, pulmonary ventilation, and heart rate were found. However, the postexercise concentration of lactate in the blood was lower in this phase. Nevertheless, the differences between phases were not statistically significant [48].

3.1.2. Metabolic effect

It is undisputed that a gradual increase in the intensity of effort leads to an increase in anaerobic energy recovery processes. This causes an increase in the concentration of lactic acid in the blood, causing changes in the level of acid-base balance parameters [50–52]. Therefore, it is of great importance in the assessment of endurance capacities to determine the location of metabolic thresholds, using blood lactate measurement or based on the dynamics of changes in ventilation indicators. The first metabolic threshold is defined by the first ventilatory threshold, the aerobic or anaerobic threshold (AT), and means the addition of anaerobic metabolism. The second metabolic threshold is referred to as the second ventilatory threshold,

respiratory compensation point (RCP), or the threshold of uncompensated metabolic acidosis [50, 52–55]. The severity of anaerobic transformation after exceeding this threshold causes hyperventilation and leads to the development of fatigue induced by incomplete metabolic acidosis [52, 56].

The results of many studies indicate the lack of clear influence of the menstrual cycle phases on resting and postexercise levels of lactate and acid-base balance parameters in the blood [17, 37, 43, 48, 57, 58].

One of the studies [45], during which women performed a graded test, indicated lower blood lactate and lower respiratory exchange ratio (RER) values for submaximal loads in the luteal phase. Resting levels and maximum values of blood lactate concentration did not significantly differ in both phases of the menstrual cycle [45]. Significantly, higher levels of lactate in the follicular phase were found by Lavoie et al. [59] during a 90-minute effort at intensity of approximately 63% VO_{2max}. Similar results were obtained by Jurkowski et al. [36] during an effort of 90% intensity VO_{2max} and McCracken et al. [38] during a graded test. The authors found a statistically significant, higher level of resting blood lactate concentration in the follicular phase of the menstrual cycle. Also, increases in lactate concentration and decreases in buffering bases, after efforts with higher loads, were significantly greater in the follicular phase than the luteal phase [36]. Differences between phases in the postexercise lactate level were maintained for 30 minutes of restitution [38].

The purpose of the Devries et al.'s study [60] was, among others, to determine the effect of menstrual cycle phase upon glucose turnover and muscle glycogen utilization during moderate-intensity endurance exercise. In these studies, healthy, recreationally active young women underwent a primed constant infusion of glucose with muscle biopsies taken before and after a 90-minute cycling exercise at intensity of 65% VO_{2max}. In the studies, it was demonstrated that women in the luteal phase have lesser reliance on carbohydrate sources to fuel endurance exercise compared with follicular phase. It was evidenced by a lower glucose rate of appearance and disappearance as well as metabolic clearance rate and lower glycogen utilization during and at the end of exercise [60].

Higher oxidation of lipids and lower oxidation of carbohydrates in the luteal phase during submaximal efforts at an intensity higher than 50% VO_{2max} have also been demonstrated in other studies [61–63]. It was also noticed that interphase differences in the use of energy substrates are related to the effort intensity. During a 30-minute treadmill run, healthy, well-menstruating women performed three 10-minute efforts at the following intensities: 30, 60, and 75% VO_{2max}, successively. Higher lipid oxidation in the luteal phase was found during low- and moderate-intensity exercise, while there were no interphase differences during exercise at an intensity of 75% VO_{2max} [20]. Similar results were obtained that in cycling efforts, higher lipid oxidation in the luteal phase was found at the 30% intensity VO_{2max} and 50% VO_{2max}, but not at 70% VO_{2max} [64]. On the other hand, they are not confirmed by this study, in which, on the basis of RER, there were no differences between phases in the proportions of energy substrate consumption (lipids/carbohydrates) during efforts of a wide intensity range, i.e., 45% VO_{2max} [65], 50% VO_{2max} [66], 65% VO_{2max} [65], 70% VO_{2max} [67], as well as 80% VO_{2max} [48].

Later research [68], in which young female rowers (female athletes and women practicing recreationally and taking/not taking oral contraceptive pills) performed a graded test on a rowing ergometer during the follicular and luteal phases of the menstrual cycle, showed that there are no interphase differences in the level of power output, ventilator equivalents of O_2 (V_E/VO_2), HR, and blood lactate concentration at maximal and aerobic-anaerobic transition intensities in all three groups. However, higher values were observed for ventilatory equivalents of CO_2 (V_E/VCO_2) at both intensities in the luteal phase compared with the follicular phase in the group of women taking contraceptive pills [68]. There were no significant interphase differences in the oxidation of carbohydrates and fats during resting (before the exercise) or during the 1-hour rowing exercise at 70% VO_{2max}. Energy expenditure, oxygen uptake, HR, and lactate concentration were similar in the follicular and luteal phases during this exercise [69]. In both phases of the menstrual cycle, the female rowers obtained similar values of VO_{2max} and VO_2 at the threshold of anaerobic transitions [69]. These results are consistent with those previously presented by Smekal et al. [70], who showed that there are no significant intergroup differences in the level of power output, VO_2, RER, HR, and blood lactate concentration at rest, at maximal load, and at different thresholds of aerobic and anaerobic metabolism (lactate thresholds, respiratory thresholds: AT and RCP), which were measured during a cycle test in eumenorrheic women. In this study, minute ventilation and V_E/VO_2 and V_E/VCO_2 indices were higher in the luteal phase at rest, exhaustion, and AT [70].

3.1.3. Oxygen capacity of the blood

Another important factor determining the level of VO_{2max} is the oxygen capacity of the blood depending on the content of hemoglobin in the blood and its affinity for oxygen and on the total volume of blood [35]. The high correlation coefficient between hemoglobin concentration and the VO_{2max} value indicates a significant role of this protein in aerobic capacity [71]. A higher level of hemoglobin in the blood in the luteal phase was confirmed by Jurkowski et al. [36]. Increased oxygen availability in tissues during the luteal phase may also result in a higher core temperature during this phase, as well as an elevated level of 2,3-DPG, causing a decrease in the affinity of hemoglobin to oxygen [12, 72]. However, studies carried out by Dombovy et al. [40] showed a slight decrease in the hemoglobin level during the luteal phase. In this research, the differences between phases in the resting level of hemoglobin did not affect the value of VO_{2max}, which did not differ between phases.

Stephenson and Kolka [33] found that the resting values of hemoglobin and hematocrit (HCT) are slightly elevated in the luteal phase. Interphase differences in the level of these parameters increased during passive heating and remained unchanged during the exercise with an intensity of approximately 80% VO_{2max}. Elevated levels of HCT in the luteal phase were also noted by Stachenfeld et al. [11, 12].

3.1.4. Plasma volume

Based on the results of earlier studies [33], it may be assumed that higher resting HCT in the luteal phase results from the smaller plasma volume during it. Gaebelein and Senay [32] suggest that the reason for this phenomenon may be an increase in the luteal phase of vascular

wall permeability to plasma proteins. Other studies do not support these views [13]. The authors performed an experiment involving seven women, in whom administering a gonadoliberin inhibitor (GnRH) reduced the level of endogenous estradiol and progesterone. Then, successively at intervals of a few days, they added extrinsic preparations containing synthetic derivatives of estradiol and progesterone. In this way, they obtained a ratio of sex hormone concentrations corresponding to, according to the concept of Janse de Jonge [16], the early-follicular, late-follicular, and middle-luteal phases. They found that at elevated levels of both hormones, which correspond to the middle of the luteal phase, the volume of plasma is the largest and constitutes about 17% of the total volume of extracellular fluids. At the same time, in this situation we observe the lowest permeability of blood vessel walls for plasma proteins. Slightly smaller plasma volume and greater permeability of blood vessel walls were observed in a situation corresponding to the late-follicular phase. However, the differences were not statistically significant. Due to the much smaller volume of extracellular fluids, plasma was then 21%. In the situation when only the GnRH inhibitor was administered, the plasma volume was the lowest, and the permeability of blood vessel walls was the largest for albumin. At the same time, the total volume of extracellular fluids was the highest, with a plasma volume of approximately 16%. Differences in the volume of extracellular fluids may be reflected in small changes in the body mass of women during the menstrual cycle [37].

Stephenson and Kolka [33] found that a 9-minute effort at an intensity of about 80% VO_{2max} caused a significantly larger loss of plasma volume during the follicular phase. The percentage changes in plasma volume reached −15.8 and −13.3% in the follicular and luteal phases, respectively [33]. Other authors did not find significant interphase differences in plasma volume changes immediately after exercise and during the restitution period [12, 37, 38, 48].

3.2. Anaerobic exercise

In the majority of studies assessing the influence of sex hormones on women's exercise responses, efforts were made of constant submaximal intensity or gradually increasing until reaching maximal oxygen uptake, i.e., "until exhaustion." However, there is little information on the interphase variation in response to typical anaerobic efforts [73–78].

The concept of anaerobic capacity of an organism encompasses a set of factors determining the performance of short-term work, during which large force and maximal generated power are developed. These include skeletal muscle mass, the supply of muscle energy substrates [ATP, phosphocreatine (PCr), glycogen], and enzyme activity of anaerobic processes. The large buffer capacity that allows tolerance of homeostatic disorders and rapid restitution in the pH range is also extremely important [79].

Taking the metabolic effects of estradiol and progesterone into account, it can be assumed that the changing ratio of these hormones during the menstrual cycle effects different physiological responses of women under the influence of anaerobic exercise [80]. The presence of estrogen receptors in the human skeletal muscle [81] and the correlation between strength and high concentrations of 17β-estradiol and progesterone have been found [82]. Furthermore, buffering capacity during the 10-s rowing sprint was greater at a higher concentration of 17β-estradiol [83]. Research suggests that ovarian hormones may influence the rate of PCr

resynthesis after eutrophic luteal efforts in eumenorrheic women [84]. Other studies also indicate a faster rate of PCr regeneration after anaerobic efforts in the luteal phase due to the greater work performed in this phase of the menstrual cycle during a series of ten 6-s sprints [85]. This indicates the potential for generating more anaerobic power during the luteal phase of the menstrual cycle (high concentration of 17β-estradiol and progesterone) or just before ovulation (high concentration of 17β-estradiol) compared to the follicular phase. In the literature, however, there is little information on the effect of different levels of hormones on the size of the developed strength or the level of anaerobic power indicators during the menstrual cycle, and their results are not consistent.

While some studies show better results during the luteal phase for single and multiple sprints [85, 86], in others, there were no interphase differences in single or repeated anaerobic cycling tests [87, 88]. Bale and Nelson [89], examining 20 women training swimming, found that they achieved the best results for a distance of 50 m in the follicular phase. Also, Parish and Jakeman [90] found that in comparison with the ovulation period and the luteal phase, the highest maximal and average anaerobic power values were obtained by women in the follicular phase.

However, it should be emphasized that the choice of experiment day in some studies was not hormonally confirmed [86–90]. In many experiments on this subject, the research dates were chosen only by calendar or thermal method. Anovulatory cycles may occur without disturbing the length of the menstrual cycle, while in the case of using the calendar method, this may lead to an erroneous indication of the day of laboratory examination. In turn, a reliable diagnosis of the course of the sexual cycle, based on the measurement of basic body temperature, is possible only after the measurements have been performed in at least three consecutive cycles. The erroneous conclusions concerning the division of the cycle into follicular and luteal phases with the use of the thermal method may additionally be caused by factors such as incorrect method of core temperature measurement, night sleep less than 6 hours, disease states, or the use of hypnotics. Inference based on the results obtained by the authors using the calendar method or thermal method is therefore limited [16].

3.2.1. Research based on hormonal verification of menstrual cycle

The results of research based on hormonal verification of the division of the menstrual cycle into phases indicate lack of significant impact of sex hormones on the values of the developed strength [91] and generated anaerobic power [73]. Nonetheless, other studies [76] show that muscle strength returns to the baseline level faster after strenuous stretch-shortening cycle exercise during the ovulatory phase, when the estrogen level is high, compared with the follicular phase. However, the differences in exercise-induced muscle damage markers (CK, soreness, and low-frequency fatigue) between the two menstrual cycle phases were small.

In the research by Wiecek et al. [75], determining the size of the maximal anaerobic power, a 20-second cycle sprint was used (Wingate test version) [92]. The energy medium of this type of effort regards mainly anaerobic processes consisting in the resynthesis of ATP at the expense of PCr and muscle glycogen. According to the research review, participation of these two processes in ATP synthesis starts from the first seconds of effort, with the participation of the phosphagen energy source in favor of anaerobic glycolysis in subsequent seconds [79]. To

obtain fully reliable results, Wiecek et al. [75] performed initial assessment of the correctness and regularity of the menstrual cycle on the basis of the registration of basic body temperature. The correctness of experiment day selection was always verified by hormonal assays. In addition, the studies were repeated in two subsequent menstrual cycles. Each woman performed an anaerobic effort twice in the middle of the follicular phase (days 6–9 of the cycle) and two times in the middle of the luteal phase (5–8 days after ovulation). The first day of menstruation was adopted as the first day of the menstrual cycle. The studies concluded that there are no significant differences in the level of indicators determining anaerobic capacity of women in the follicular and luteal phases of the menstrual cycle. No interphase differences were found in the maximal level or average anaerobic power. The time of obtaining and maintaining maximal power and the rate of decrease in anaerobic power were not different either. The effect of anaerobic metabolism during a 20-second effort at supramaximal intensity is a significant increase in blood lactate concentration, which entails changes in the level of acid-base balance parameters. In both phases of the menstrual cycle, the anaerobic effort caused similar disturbances in the acid-base balance [75].

Also, in the research by Tsampoukos et al. [77], the days for exercise were carefully selected. Eumenorrheic women performed a test comprised of two, 30-s sprints separated by a 2-min break. The test was conducted during the follicular phase, just before ovulation and in the luteal phase. The mutual ovarian hormone system was characterized by adequately low levels of progesterone and 17β-estradiol, low progesterone levels and high levels of 17β-estradiol, and high levels of progesterone and 17β-estradiol. It was found that there are no interphase differences in the maximal level or average anaerobic power. Menstrual cycle hormones also did not affect postexercise changes in metabolic parameters (blood lactate and pH, plasma ammonia) or the rate of regeneration between sprints [77].

Furthermore, during the 6-s cycling sprint, there were no interphase differences in the amount of generated anaerobic power and changes in blood lactate or in the sympatho-adrenergic response tested by the measurement of adrenaline and noradrenaline in the blood [93]. The lack of influence of sex hormones on different exercise responses in the follicular and luteal phases was also demonstrated by studies in which no interphase differences were found in maximal accumulated oxygen deficit and sprint performance in repeated sprint cycling, i.e., three times at 120% VO_{2max} with 20-minute resting periods between consecutive sprints [78]. Also, the 40-yd running time preceded by a 15-minute warm-up (jogging, skipping by moving the legs in various directions, and sprinting alternating with jogging), performed at an ambient temperature of about 32.5°C, did not differ between the early-follicular and middle-luteal phases [94]. Regardless of the menstrual cycle phase, the warm-up triggered an increase in the core temperature of about 1°C, which resulted in a better result during the run [94].

Similar results were also obtained in earlier studies [74]. Domagala et al. [74] obtained results indicating a tendency for smaller increases in lactate concentration and changes in acid-base balance parameters in the luteal phase of the menstrual cycle. In the luteal phase, they also noted a slightly higher rate of lactate concentration restitution after exercise with a supramaximal load; however, the interfacial differences were not statistically significant [74].

However, in the research by Redman and Weatherby [83], in which rowers performed a test of anaerobic power (10-s all-out effort) and capacity (1000-m row), it was found that the peak

power output was higher and the 1000-m rowing ergometer time was faster when the concentration of progesterone and estradiol was low (quasi-follicular phase), in contrast to when the levels of both hormones were high (quasi-luteal phase). The concentration of sex hormones was regulated by oral contraceptive pills [83]. Julian et al. [95], by examining female soccer players who performed the Yo-Yo intermittent endurance test, multiple jumps, and 3 × 30 m sprints in the early-follicular phase and in the middle of the luteal phase, showed a reduction in maximal endurance performance during the middle-luteal phase. This effect was not observed for jumping or sprint performance.

3.3. Thermoregulation

An important factor conditioning the possibility of performing physical exercise is the efficient functioning of thermoregulation mechanisms. During rest, in thermoneutral conditions, the heat balance of the body is stabilized by the exchange of heat produced in metabolic processes. Heat is exchanged with the environment through conduction and convection, radiation, and evaporation. By these means, approximately 20, 60, and 20%, respectively, of heat is eliminated from the body [96].

During physical exercise, as a result of the intensification of metabolic processes, the thermal balance of the body and the stimulation of thermoregulation systems are disturbed. We observe an increase in the core temperature of the body, depending on the relative load expressed as % VO_{2max}, which during efforts at constant intensity is stabilized at an elevated level. The main role in the elimination of excess endogenous heat during physical exercise is played by evaporation of perspiration from the body's surface, constituting about 80% of heat loss [96]. The effectiveness of exercise-based thermoregulation depends on the rate of sweat secretion and external conditions affecting the efficiency of its evaporation, as well as the correct functioning of the circulatory system, on which the heat transfer from the muscles to the surface of the skin depends [96]. After core temperature exceeds the so-called vasodilation threshold, blood flow through the cutaneous vessels steadily increases along with the increase of exercise intensity to about 60–70% VO_{2max}, after which it gradually decreases due to the increase in muscular flow [97]. Thermoregulatory reactions also depend on hydration status of the body, the concentration of sodium and calcium ions in body fluids, the degree of acclimation to the conditions under which physical exercise is performed, and the level of physical fitness [96].

The increase in resting body temperature during the luteal phase of the menstrual cycle by about 0.3–0.5°C, as compared to the level during the follicular phase, is the result of the antagonistic effect of progesterone and estradiol on the thermoregulatory system in the hypothalamus [12]. Observations of many authors indicate the relationship between thermoregulatory responses in women and the course of the menstrual cycle [29, 39, 41, 98–100].

3.3.1. Core temperature and sweating

The average temperature measured in the esophagus (T_{es}), in thermoneutral conditions, during a 30-minute exercise at an intensity of 40 and 70% VO_{2max}, was higher in the luteal phase [101]. The increase in T_{es} obtained in these studies was the same in both phases of the

menstrual cycle [101]. In other studies [102], the temperature measured in the auditory canal (T_{ty}), at rest and during exercise tests (60-minute 50% VO_{2max} and during the graded test), was significantly higher in the luteal phase of the menstrual cycle, and T_{ty} increases were slightly higher in follicular phase.

The increases in core temperature, similar in both phases, during submaximal efforts performed in thermoneutral conditions and in conditions of elevated temperature, were also observed in the studies presented by other authors [12, 94, 100, 103]. These results are only partially consistent with the results of this study, during which a higher level of rectal temperature (T_{re}) was observed in the luteal phase of the menstrual cycle while performing a 60-minute effort at constant intensity of about 60% VO_{2max} (ambient temperature 22°C) [41]. However, T_{re} increments were comparable in both phases of the menstrual cycle only during the initial minutes of exercise, after which the differences between phases increased due to stabilization of T_{re} in the follicular phase at the level of 38.3°C and its continuous increase to 38.9°C (despite the constant load) in the luteal phase [41].

During a 15-minute exercise at a constant intensity of 70% VO_{2max}, performed in an ambient temperature of 18°C, significantly higher T_{re} increments were observed in the follicular phase of the menstrual cycle [39]. In conditions of passive overheating or physical exertion, activation of sweat secretion and dilation of cutaneous blood vessels were observed at higher temperatures as well as a higher intensity of perspiration secretion in the luteal phase [12, 28, 29, 39, 98–102]. There was also later occurrence of perspiration production during exercise tests in the follicular phase [28].

Despite the different course of T_{re} changes in the follicular and luteal phases, the sweat rate was similar in both phases of the menstrual cycle [41]. The authors suggest that this may be related to lower sensitivity of sweat secretion in the luteal phase (sweating rate—increase of core temperature dependency) [41]. However, these results are not confirmed by other laboratory tests, which indicate a slightly higher value in the luteal phase [28, 39, 98] or a similar sensitivity of the perspiration mechanism in both phases [12, 99, 100, 103]. The results obtained during submaximal physical exercise in thermoneutral conditions showed that the increase in rectal temperature in women was lower during the luteal than follicular phase, while the dynamics of sweating were higher in the luteal phase [104].

Tests during which women performed two different stress tests (graded test "to refusal" and 60-minute with 50% intensity of VO_{2max}) show that the temperature threshold for starting the perspiration release reaction is higher in the luteal phase and does not depend on the type of exercise [102]. In contrast, the sensitivity of the perspiration production reaction is independent of the menstrual cycle and is higher during the graded test [102]. According to other authors, the effectiveness of perspiration is greater in the luteal phase of the menstrual cycle [104, 105].

Lower core temperature during exercise in the follicular phase may be due to interphase differences in the value of dermal flow and skin temperature. Some studies [39, 98] show that in thermoneutral conditions, at rest and during submaximal efforts, blood flow through the forearm reaches significantly higher values in the luteal phase. The temperature level at which the cutaneous blood vessels dilate, determined on the basis of temperature measurements, T_{es},

T_{ty}, and T_{re}, shifted toward higher values in the luteal phase by about 0.5°C [39]. However, in other studies, resting blood flow in the forearm did not differ during the menstrual cycle [29]. The exercise-based (30 minutes 80% VO_{2max}) increase in cutaneous flow coincided with the increase in core temperature (T_{es}) to the level of 37.0°C in the follicular phase and 37.4°C in the luteal phase and stabilized at a significantly higher level during the latter phase [29].

3.3.2. Skin temperature

According to some authors, the average skin temperature (T_{sk}) is significantly higher in the luteal phase at rest and during submaximal efforts in thermoneutral conditions [100] at the time of heat exposure [99, 103] and during passive overheating [99]. The lack of differences between phases in the T_{sk} values measured at rest as well as during the exercise tests (30 minutes 40% VO_{2max} and 30 minutes 70% VO_{2max}) performed in thermoneutral conditions was also demonstrated [101]. Cutaneous flow tended to be higher in the luteal phase, but the resulting interphase differences were not statistically significant [101]. Other studies also showed similar resting values and a comparable course of T_{sk} exercise changes in both phases of the menstrual cycle [12, 41, 106]. Cutaneous flow assumed lower values in the luteal phase [106].

3.4. Oxidative stress

The body maintains homeostasis in the scope of redox reactions (prooxidation and antioxidative balance), which affects the proper course of biochemical intracellular processes and intercellular signaling. The condition of redox homeostasis is to maintain balance between the level of reactive oxygen and nitrogen species (RONS) and antioxidant defense. Antioxidative defense is provided by nonenzymatic low-molecular and macromolecular antioxidants and antioxidant enzymes, which together determine total antioxidant capacity. Physical activity gives health benefits by improving, among others, cardiovascular and respiratory system functioning, and metabolic processes as well as by increasing antioxidant capacity [107–109].

3.4.1. Exercise-based sources of oxygen and nitrogen

Research has shown that physical exercise influences prooxidation and antioxidative balance [110–114]. The mechanism of RONS formation depends on the duration, intensity of effort, and type of muscle work. An increase in ATP consumption during aerobic efforts results in an increase in the rate of oxidative phosphorylation and, consequently, increased electron leakage in the internal mitochondrial membrane. As a result, amounts larger than at rest of the superoxide anion ($O_2^{\bullet -}$), hydrogen peroxide (H_2O_2), and hydroxyl radical ($^{\bullet}OH$), which belong to the reactive oxygen species, are formed [109]. The superoxide anion radical is also formed by the reaction of NADPH oxidase in the sarcoplasmic reticulum and transverse tubules (T-tubules) of the sarcolemma, as well as in the reaction of xanthine oxidase, the activity of which increases after anaerobic efforts [108]. The formation of RONS is promoted by the increase in lactate dehydrogenase activity, lowering the pH value, increasing the concentration of catecholamines, as well as increasing intramuscular temperature [108, 109]. Contractile activity also leads to increased nitric oxide (NO) synthesis by induced nitric oxide synthase

(iNOS). The consequence of increased NO synthesis, at a high level of $O_2^{\bullet-}$, is the formation of peroxynitrite ($OONO^-$). Peroxynitrite is a reactive form of nitrogen. Excessive RONS production during exercise can also be the result of myocyte micro-injury associated with activation of the leukocytic system [107, 115].

3.4.2. Markers of oxidative stress

Oxidative stress is determined by changes in the level of many, various markers (**Table 1**). Different oxidation rates and different antioxidants are evaluated in the research, the changes of which are not always unidirectional [107].

3.4.3. The significance of reactive oxygen and nitrogen species

The formation of RONS in low concentrations (intracellular signaling) is necessary for the regulation and integration of biochemical processes. They activate primary signaling pathways depending on redox status. The main transcription factor, sensitive to redox status, is the nuclear factor erythroid 2-related factor (Nrf2). Nrf2 activation affects the strengthening of antioxidative defense and cytoprotection. It has been shown that as a result of regular exercise, upregulation of gene expression for peroxisome proliferator-activated receptor-γ coactivator-1α (PGC-1α) occurs, which upregulates Nrf2 in order to regulate the mitochondrial biogenesis. The upstream signals that regulate PGC-1α expression as mitogen-activated protein kinase (MAPK) and nuclear factor κB (NF-κB) are also redox-sensitive. RONS, through

Markers of oxidative stress	**Antioxidants**
4-Hydroxynonenal (4-HNE)	Endogenous enzymatic
F2-isoprostane	• Superoxide dismutase (SOD)
Isoprostanes (8-iso-PGF2α)	• Catalase (CAT)
Malondialdehyde (MDA)	• Glutathione peroxidase (GPx)
Oxidized low-density lipoprotein (ox-LDL)	• Glutathione reductase (GR)
Thiobarbituric acid reactive substance (TBARS)	Endogenous nonenzymatic
8-Hydroxy-2′-deoxyguanosine (8-OHdG)	• Albumin
3-Nitrotyrosine (3-NTR)	• Uric acid
Protein carbonyl content (PCC)	• Reduced glutathione (GSH)
Advanced oxidation protein products (AOPP)	Exogenous nonenzymatic
Oxidized glutathione (GSSG)	• β-Carotene, vitamin E, vitamin C
Total oxidative status (TOS)	Total antioxidative capacity (TAC)
Oxidative stress index (OSI = TOS/TAC)	

Table 1. Examples of indicators of oxidative stress and antioxidants.

MAPK, activate the NF-κB signaling pathway and, thus, affect the expression of antioxidant enzyme genes such as superoxide dismutase (SOD), catalase (CAT), or glutathione peroxidase (GPx) or glutathione reductase (GR). Therefore, they are responsible for maintaining an appropriate level of endogenous antioxidant defense [107–109, 115].

At high concentrations (oxidative damage level), RONS exhibits inhibitory and damaging effects [107]. Excessive production of RONS with low antioxidant capacity may be the reason for shifting the prooxidation and antioxidant balance toward oxidation and oxidative stress. The consequence of oxidative stress is increased lipid peroxidation, oxidation of thiol groups of proteins, damage to DNA and carbohydrates. Oxidative damage to macromolecules is the cause of disturbance in enzyme activity and permeability of biological membranes [107].

3.4.4. Antioxidant effect of estradiol activity

In women, the antioxidative role is attributed to estrogen. Animal experiments have shown that the amount of H_2O_2 formed in the mitochondria of females is lower than in the mitochondria of male rats. The positive effect of estradiol on oxidative stress has also been demonstrated in women. It was found that estradiol reduces the level of reactive oxygen species, and in postmenopausal women, it intensifies oxidative stress, which is counteracted by hormone replacement therapy. Estradiol, acting indirectly through the estrogen receptor, increases antioxidant capacity, affecting activation of the NF-κB signaling pathway, which consequently results in an increase in enzymatic antioxidant capacity [116–119].

3.4.5. Redox balance in the menstrual cycle

An interesting research model was carried out by Massafra et al. [120]. Young women (aged 20–27) participated in the study, declaring a regular menstrual cycle, the length of which ranged from 28 to 30 days. The first day of menstruation was accepted as the first day of the menstrual cycle. Changes in ovarian hormones were monitored based on the daily determinations of 17β-estradiol and progesterone. A preovulatory peak of 17β-estradiol concentration was determined for each woman, which was the "0" point. In relation to the "0" point, three tests were determined in the follicular phase (early, from bleeding to −10; middle, from days −8 to −4; late, from days −2 to 0) and in the luteal phase (early, from days 2 to 4; middle, from days 6 to 10; late, from days 12 to 14). Significant changes in the menstrual cycle were found in erythrocyte GPx activity, with higher values in the period from late follicular to early luteal phases compared to early-follicular phase. This coincided with the elevated level of 17β-estradiol, and the correlation coefficient was 0.8. In this study, there was no effect of 17β-estradiol, progesterone, and LH or FSH on the activity of CAT and SOD in erythrocytes, which were similar throughout the menstrual cycle [120].

In turn, in another study [121] involving 259 regularly menstruating women aged 18–44, it was found that the level of antioxidants in blood serum is dependent on ovarian hormones. Similar appointments for assays were determined as in previous studies [120]. Among others, antioxidant fat-soluble vitamins and carotenoid micronutrients (α-tocopherol, γ-tocopherol, β-carotene, retinol, lutein, lycopene) and ascorbic acid were determined. The concentration of F2-isoprostane was determined as an indicator of lipid oxidation. In most women, the assays

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were conducted during two menstrual cycles. Among others, it was found that the concentrations of fat-soluble vitamins and ascorbic acid are lower during menstruation. The concentration of fat-soluble vitamins positively correlated with the concentration of 17β-estradiol. The concentration of ascorbic acid also correlated positively with the concentration of 17β-estradiol and progesterone, while it was lower when the concentration of LH was higher. Women with higher ascorbic acid concentrations had lower F2-isoprostane concentrations. In this study, the ratio of α- to γ-tocopherol was associated with an increased risk of anovulatory cycles [121].

While regularly maturing young women (cycle length 26–31 days) were divided into two groups, i.e., ovulating and non-ovulating, it was found that plasma TBARS (lipid oxidation index), whole blood GSH concentration, and CAT, GPX, and GR activity in erythrocytes are similar in both groups in the first (7–9 days) and in the second (22–25 days) mid-menstrual cycle [122]. However, the activity of SOD in erythrocytes, in both measurements, was significantly higher in the non-ovulating group. In ovulating women, there was a significant negative correlation between the concentration of 17β-estradiol in the blood plasma and the activity of SOD in erythrocytes. Research has shown that the lack of ovulation in menstruating women does not affect increased lipid peroxidation. In contrast to previous studies [120], there was no effect of GSH-dependent erythrocyte antioxidant defense, while it was found that lower plasma estradiol resulted in attenuated erythrocyte SOD inhibition and elevated enzyme activity [122].

There are also studies in which it was concluded that women are subjected to oxidative stress for most of the menstrual cycle. In these studies, oxidative stress was assessed on the basis of the reactive oxygen metabolites-derived compound test (d-ROMs), the results of which correspond to the hydroxyperoxide level. The measurement in the blood was performed every 3 days, starting from the first day of menstruation up to the last day of the menstrual cycle (the day before the next menstruation). The level of dROMs was significantly elevated between days 9 and 24 of the menstrual cycle, when there was a peak of 17β-estradiol at low progesterone concentration, as well as when the levels of both hormones were elevated. However, there was no correlation between 17β-estradiol concentration and the level of dROMs; thus, it can be assumed that other factors influenced the increased lipid oxidation [123].

Other studies do not confirm the relationship between oxidative stress and the course of the menstrual cycle. There were no differences in the level of 8-OHdG in the urine (oxidative DNA damage index) between follicular phase, ovulation, and the luteal phase of the menstrual cycle [124]. There were also no differences between TBARS [34, 125] and MDA [125, 126], i.e., lipid oxidation indices, as well as H_2O_2 and nitrite/nitrate levels [127], nor in total GSH, GSH, and GSSG concentrations [125]. The effect of hormones during the menstrual cycle on the total activity of SOD and the activity of extracellular superoxide dismutase (EC-SOD) [34] or the total antioxidant capacity were not demonstrated [126]. Also, in older postmenopausal women who did not use hormone replacement therapy, there were no higher levels of dROMs or lower antioxidant capacity compared to premenopausal women of similar age (46–55 years). On the basis of dROM values in both groups of women, the middle oxidative stress level was found, and a slight deficit in antioxidant defense was detected [127]. However, the level of lipid oxidation in older women was higher compared to the young (25–35 years), properly menstruating women who did not use contraceptive pills [127].

It was shown that oral contraception (monophasic pills containing 0.02 mg ethinyl estradiol and 3 mg drospirenone) affects the prooxidative and antioxidant status of young women [128]. Compared to women who do not use oral contraception, they cause a decrease in GSH and glutathione S-transferase (GST), GR, and GPx in the blood while increasing CAT activity and lowering GSSG concentration, resulting in the GSH/GSSG oxidative stress index to not change. These studies show that external modification of the concentration of sex hormones causes catalase to play a main antioxidative role, which confirms the positive correlation between CAT activity and MDA concentration. Increased CAT activity may be the result of accumulation of H_2O_2 and other radicals. Detoxification of reactive oxygen species by the GSH system is weakened in this situation [128].

3.4.6. Exercise-induced changes in redox balance

Studies show that high levels of 17β-estradiol in non-training young women with normal biphasic menstrual cycles favor easier elimination of free radicals formed during exercise [34]. Despite the lack of interphase differences in resting TBARS level and SOD activity, after a 30-minute cycling effort with 60% intensity VO_{2max}, it was found that the TBARS level decreased in the follicular phase when the 17β-estradiol concentration was higher than during menstruation and the luteal phase. In the luteal phase, however, the activity of SOD in the blood decreased after the effort. Although in none of the phases (menstruation, follicular phase, luteal phase), neither at rest nor after exercise, was there any correlation between the concentration of 17β-estradiol and oxidative stress markers, it was nonetheless found that along with the higher concentration of this hormone, the decrease in SOD activity was lower [34].

In another study [125], in which young women also performed a 30-minute moderate-intensity exercise (about 75–80% VO_{2max}) in the follicular and luteal phases of the menstrual cycle, a significant postexercise increase in GSSG concentration was noted during the luteal phase (by 28%), while the concentration of total GSH decreased significantly after exercise only in the follicular phase (by 8%). The concentration of GSH after exercise, regardless of the phase of the menstrual cycle, significantly decreased by about 16–17%. These results show that at higher concentrations of 17β-estradiol (late follicular phase), exercise causes slightly less disturbances of redox homeostasis due to more efficient scavenging free radicals with the participation of glutathione [125].

Amenorrheic and eumenorrheic athletes underwent a 90-minute effort at an intensity of 60% VO_{2max} [129]. The level of 17β-estradiol was significantly lower in amenorrheic women both before and during exercises (30, 60, and 90 minutes) and at 15-minute recovery. In these studies, there was a greater effect of the effort on the oxidative stress markers in amenorrheic women. In this group, at rest and during exercise, GPx activity was higher. Before the effort, GR activity in both groups of women was comparable, but as a result of the effort, it also significantly increased in amenorrheic women. Plasma lipid peroxidation concentration and CAT activity were similar in both groups and did not change in response to physical effort. Contrary to other studies [120], there was a negative correlation between GPx activity and 17β-estradiol concentration, but, simultaneously, GPx activity depended positively on cortisol concentration, which was elevated in the group of amenorrheic women [129].

These results are contrary to those of other researchers [130] who found a positive relationship between GPx activity and estradiol levels both before and after physical exercises. Non-training, young women, during menstruation and the preovulatory phase, performed three isokinetic efforts to exhaustion, consisting of performing maximum alternating concentric and eccentric work of the knee extensor muscles of the dominant lower limb, preceded by a 15-minute submaximal bicycle effort at an intensity of 50% VO_{2max}. The concentration of MDA in the blood plasma did not change after exercise, but the activity of SOD and GPx in erythrocytes decreased significantly. The effort-induced changes were lower when the estradiol concentration was higher, what is more, the α-tocopherol supplementation (antioxidant vitamin) did not affect this [130].

Another study [131] showed that a low carbohydrate diet for 3 days (5% carbohydrate, 52% fat, 43% protein), preceded by exertion of glycogen depletion, supports the antioxidant defense system in healthy eumenorrhoeic women, both at rest and during graded exercises performed "until exhaustion," compared to women using a balanced diet at this time (59% carbohydrate, 27% fat, 14% protein). It seems reasonable to assume that the higher daily intake of heme iron, selenium, and α-tocopherol provided with a low carbohydrate diet contributed to the increase of antioxidative capacity by increasing the activity of CAT and increasing the concentration of selenium and α-tocopherol in the plasma, which gave better protection of the cell membranes against peroxidative damage caused by physical effort. This is reflected in the limited release of creatine kinase into the blood, which is an indicator of sarcolemma damage. Physical effort was repeated twice in the follicular phase (6–8 days), when the concentration of 17β-estradiol was low, and twice during the luteal phase (4–6 days after ovulation), when both the concentrations of 17β-estradiol and progesterone in the blood were high. In this study, the phase of the menstrual cycle had only a small effect on antioxidant defenses of blood [131].

3.4.7. Sex differences in exercise-induced oxidative stress

It can be assumed that due to differences in estradiol concentration, disturbances in prooxidative-oxidative balance of the blood after exercise are higher in men than women. In the research carried out by Wiecek et al. [111] among young individuals, it was found that the changes in the prooxidative-antioxidative balance of the blood induced by maximum intensity exercise (graded test) differ in women and in men. In men, there was a significant shift in prooxidant-antioxidant balance toward oxidation without increasing the total plasma antioxidant capacity (TAC). In women, the postexercise changes in total plasma oxidation status (TOS) were low due to the increase in TAC. Exercise-induced changes in TOS concentration depended on VO_{2max} and simultaneously, on the increase in lactate concentration [111]. The lack of such relationships in women may indicate the influence of other factors on postexercise oxidative stress in this group of subjects, such as damage to muscle fibers. Continuing research with the participation of young individuals, it was found that the result of physical effort at maximum intensity is significant, independent of VO_{2max} and VO_2 as well as the work intensity (% VO_{2max}) at the level of the second ventilatory threshold, increase in concentration of ox-LDL and 3-nitrotyrosin in the blood serum, testifying to similar lipid and protein damage in women and men. The significant increase in TAC after exercise at

maximum intensity was the result of micro-damage of muscle fibers that occurred in women [114]. However, when young people performed an anaerobic effort, it was found that with similar disturbances of acid-base balance, changes in TAC, TOS, and oxidative stress index (TOS/TAC) in the blood were the same in both women and men. The changes in concentration of low-molecular nonenzymatic antioxidants induced by this effort were also the same in both sexes. The level of the tested markers was indicative of oxidative stress persisting for at least 24 hours after the end of the work [110]. There was no evidence that anaerobic exercise caused muscle damage [113]. During the first hour after completing anaerobic exercise, there were no changes in the activity of xanthine oxidase (XO) in the blood plasma of men or women. The significant increase in the activity of this enzyme was found 24 hours after the completion of anaerobic exercise. The increase in XO activity in the blood after anaerobic exercise was greater in women than men. At the same time, the postexercise increase in XO activity was negatively correlated with the amount of total work performed during anaerobic exercise and with mean and peak anaerobic power, which were significantly lower in women [113]. In turn, the 45-minute submaximal (50% VO_{2max}) effort ending with a 15-minute eccentric exercise [downhill run (−4.5°)] caused oxidative damage to lipids only in women [112]. An increase in ox-LDL concentration indicated redox balance disturbances. In men, regardless of the type of muscle work (eccentric, concentric), submaximal running efforts did not cause oxidative stress. The probable cause of these gender differences was the higher antioxidant capacity of men's blood dependent on greater physical performance [112]. In the above studies, however, the phase of the menstrual cycle was not taken into account; women performed the exercise either during the follicular phase or in a randomly selected phase. Nonetheless, the results indicate that changes in redox balance among women depend on the intensity of the effort and on the type of muscle work involved.

4. Conclusion

Despite many years of interest in this subject, the current research does not allow to draw unambiguous conclusions about the impact of the changing level of sex hormones during individual phases of the menstrual cycle on the exercise capacity of women. There are indications that in the luteal phase, the capacity to perform efforts based mainly on aerobic energy transformations does increase. For example, in one of recent studies [132], an increase in cardiac and respiratory efficiency in the luteal phase of the menstrual cycle for normal-weight females was found, where as in overweight and obese individuals, there was an overall decrease in fitness capacity along with an increase in body mass index (BMI). However, the differences between groups noted by one researcher in the measured effects of the applied test are not confirmed by observations of other authors [133]. Often, however, differences in the results obtained by women in the pre- and postovulatory phase are small. According to a review of studies on women's exercise capacity, in most experiments, efforts were applied with constant submaximal intensity or gradually increasing until the oxygen uptake was achieved. However, there is little information on intergroup variation in response to typical anaerobic efforts. These studies, as one of the most recent [134], indicate a lack of hormone influence in the menstrual cycle on anaerobic efficiency indices. But these tests also provide divergent results.

The question arises as to why there are still no studies that explicitly determine the exercise capacity of women depending on the phase of the menstrual cycle [135].

Difficulties in undertaking research on exercise-related reactions in women lie, inter alia, in obtaining volunteers for research, especially those non-training ones if the experiment requires the implementation of very high intensity efforts. No physical examinations involve large groups of women. Usually, the research group consists of several to a dozen people. The problem is also obtaining the right motivation of the studied women to fully carry out their potential capabilities during laboratory exercise tests. The reason for the discrepancy of some results may be the determination of test date based on measurements of basic body temperature without hormonal determinations. On the other hand, the source of often conflicting results, given by different authors using sex hormone markers, may be the terminology used to determine the location of the performed laboratory test in the course of the menstrual cycle. Some authors use the division of menstrual cycle into follicular and luteal phases, without specifying the days on which the tests were performed and/or concentrations of estradiol and progesterone determined. Considering the changing ratio of these hormones during the menstrual cycle, the following division seems to be more appropriate: early-follicular (low estradiol and progesterone), late-follicular (elevated estradiol and low progesterone), and middle-luteal (high estradiol and progesterone levels) [16] or to reference the day of testing to the day of ovulation [120]. Difficulties in comparing the results obtained by different authors also result from significant differences in the age and level of physical activity of the surveyed women, or from the variety of applied stress tests and performed assays, which mainly concern markers of oxidative stress. Different methodological approaches and inconsistent presentation of data are a limitation when comparing the results of women obtained by them depending on the changing hormone concentrations of the menstrual cycle. So far, it has not been specified whether it is necessary to take the phase of the menstrual cycle into account in sports diagnostics, e.g., during stress tests that check the physical performance of women training different sports. Also, comparisons between exercise reactions of men and women may be imprecise due to methodological differences. A comprehensive and multi-aspect study of the exercise should be carried out, involving training and non-training women of all ages, in the pre- and postmenopausal period, as well as those using and not using hormonal contraceptives or hormone replacement therapy, which would precisely characterize whether there are differences in biochemical-physiological adaptation to the efforts of varying intensity and type of work and the course of changes in the recovery period, related to the level of hormones in the menstrual cycle.

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Conflict of interest

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