Chapter 14
Pregnancy, Sex Hormones, and Exercise

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Introduction

The sequence of events from fertilization to the formation of an adult organism is known as developmental anatomy. This timeline begins with ***pregnancy***, which specifically relates to: (i) the fertilization of a secondary oocyte (i.e., an immature egg, female sex cell) by a spermatozoon (i.e., mature motile male sex cell), which usually occurs 12–24 h following ovulation and results in the formation of a single nucleus (i.e., a segmentation nucleus) with genetic material contributed by the spermatozoon (i.e., the male pronucleus) and ovum (i.e., the mature egg, the female pronucleus); (ii) implantation of the blastocyst (i.e., the cleaved fertilized ovum) into the endometrium (i.e., the lining of the womb), which typically occurs six days after fertilization; (iii) embryonic (i.e., the first two months of development) and fetal (i.e., the remaining seven months of development) growth; and (iv) parturition (i.e., labor and delivery, childbirth). Even though pregnancy is initiated by fertilization, its tenure starts on the first day of the menstrual cycle in which fertilization occurs (i.e., on the first day of the woman's last period). Pregnancy typically lasts 40 weeks and is divided into three trimesters each lasting 12–14 weeks. ***Gestation***, on the other hand, refers to the time a zygote (i.e., the fertilized ovum), embryo, or fetus is carried in the female reproductive tract and is usually 266 days, which is counted from the estimated date of fertilization. Throughout pregnancy, a multitude of synchronous anatomical and physiological changes occur (Fig. 14.1); the physiological changes include alterations mediated by (i) estrogen and progesterone (i.e., sex hormones) and (ii) sex hormones plus other mechanisms (e.g., weight gain, growing fetus). This chapter

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A. C. Hackney (ed.), *Sex Hormones, Exercise and Women*,
https://doi.org/10.1007/978-3-031-21881-1_14

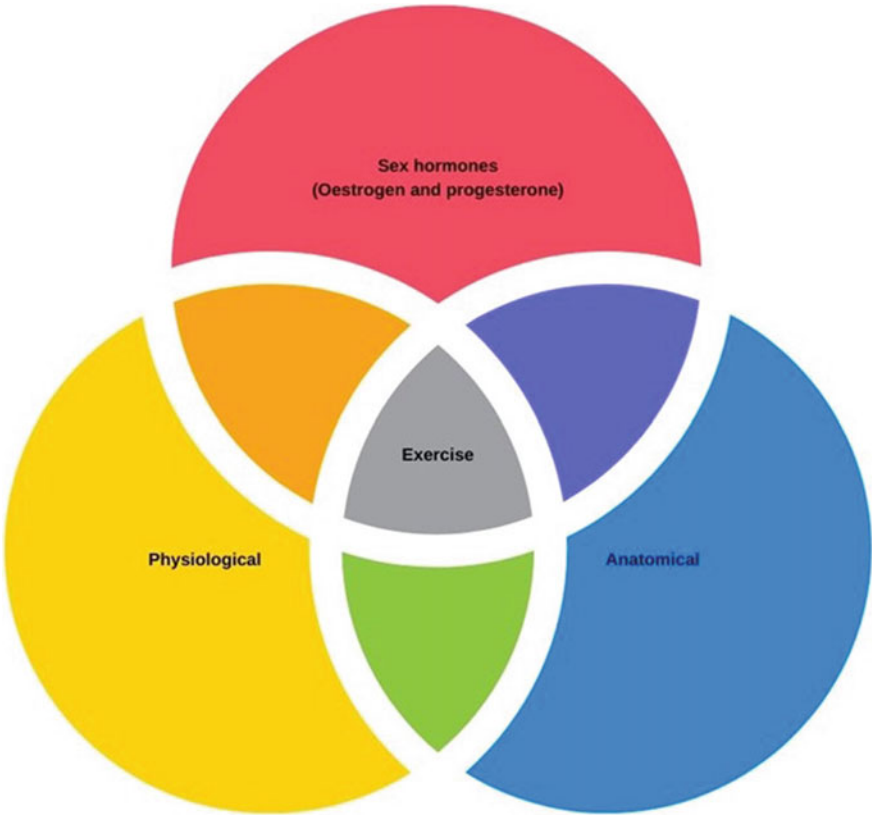

Fig. 14.1 Venn diagram showing the interaction among the hormonal (specifically sex hormones), physiological, and anatomical changes that occur during pregnancy and their potential to either individually and/or collectively influence exercise performance and capacity

aims to contextualize these changes and specifically focus on the impact of supra-physiological concentrations of estrogens and progesterone caused by pregnancy on exercise performance and capacity.

Changes and Outcomes Associated with Pregnancy

In the following sections, the pregnancy-related changes in sex hormone concentrations are discussed in detail (i.e., in keeping with the focus of this chapter), while the anatomical and wider physiological changes (i.e., all physiological changes excluding the changes in sex hormone concentrations) are just summarized with signposting provided to further specialized texts (Table 14.1).

Table 14.1 Abridged anatomical and wider physiological changes (i.e., all physiological changes excluding the changes in sex hormone concentrations) and outcomes, which occur during pregnancy, and their functional and practical implications on exercise performance and capacity

	Anatomical	Physiological					
		Integumentary	Cardiovascular	Respiratory	Digestion	Metabolism	Thermoregulatory
Panel a							
Changes	Weight gain and internal organ displacement	Deviations in pigmentation, nevi, vasculature, mucosa, connective tissue, glands, nails, and breasts	Increased cardiac load at rest and during exercise and altered mean arterial pressure	Disturbances in ventilatory function	Decreased digestive motility	Altered glucose metabolism	Altered response to evaporative and dry heat loss
Caused by	Growing fetus	Estrogen and progesterone and non-specific and specific pregnancy-related dermatoses	Mechanisms responsible for mediating the changes in systemic hemodynamics have yet to be completely elucidated—likely mediated by nitric oxide, relaxin, and estrogen	Hormonal (estrogen and progesterone) and mechanical effects (enlarged uterus)	Hormonal (estrogen and progesterone), mechanical (growing fetus), and hemodilution effects	Exact causes are unknown—likely affected by hormones (estrogen and progesterone), weight gain, a growing fetus, race, pre-gravid weight, pre-diabetes	Cardiovascular shifts that occur during pregnancy and changes in progesterone
Panel b							
Outcomes	Bladder irritation	Hyperpigmentation	Increased resting heart rate	Increased sensitivity to carbon dioxide	Constipation	Gestational diabetes mellitus	Enhanced thermoregulatory capacity

(continued)

Table 14.1 (continued)

	Anatomical	Physiological					
		Integumentary	Cardiovascular	Respiratory	Digestion	Metabolism	Thermoregulatory
	Reduced bladder volume	Edema	Increased stroke volume	Increased tidal volume	Gallstones—bloating, indigestion, heartburn, and gas	Increased maternal insulin resistance	Decreased body temperature thresholds for sweating
	Increased pelvic floor distensibility	Varicose veins	Hypotension	Decreased arterial carbon dioxide tension		Elevated maternal blood glucose levels	
	Incontinence, nocturia, and micturition	Spider angiomas, pyogenic granuloma, acne, and skin tags	Hypertension—leading to pre-eclampsia in severe cases	Increased arterial oxygen tension			
	Pelvic organ prolapse	Vulvar varicosity	Heart rate does not significantly correlate with ratings of perceived exertion	Respiratory discomfort (dyspnea)			
	Bladder and bowel dysfunction	Striae gravidarum					
	Gastrointestinal issues (e.g., gastroesophageal reflux, gallstones, diarrhea, and constipation)	Breast enlargement, tenderness, increased prominence of veins, striae, areolar enlargement, erectile nipples, and/or nipple sensitivity					

(continued)

Table 14.1 (continued)

	Anatomical	Physiological					
		Integumentary	Cardiovascular	Respiratory	Digestion	Metabolism	Thermoregulatory
	Decreased respiratory reserve volume	Hirsutism/hypertrichosis					
	Decreased functional residual capacity						
	Shift in center of gravity						
	Increased postural sway						
Panel c							
Functional implications (i.e., impact on exercise performance and/or capacity)	Altered gait and movement patterns (e.g., agility) Increased risk of falls	Edema—inability to stand for long periods of time	Hypotension can cause nausea, dizziness, unsteadiness fatigue, weakness, blurred vision, and cognitive impairment which can directly affect exercise performance and capacity	Dyspnea—can be compounded by some forms of exercise	Gastrointestinal distress—reduced exercise capacity	Women with gestational diabetes mellitus might experience hypoglycemia during and following exercise	Greatest blood flow (i.e., greater dry heat loss) and sweat losses (i.e., evaporation) at rest and during exercise occur in late pregnancy

(continued)

Table 14.1 (continued)

	Anatomical	Physiological					
		Integumentary	Cardiovascular	Respiratory	Digestion	Metabolism	Thermoregulatory
	Weight gain may significantly increase the forces across joints during weight-bearing exercise	Breasts/nipples—pain and/or sensitivity during exercise	Those with hypotension need to be watchful of post-exercise hypotension	Decreased oxygen availability for the performance of aerobic exercise		Women with gestational diabetes mellitus tend to gain more weight during pregnancy (see anatomical changes column for implications of weight gain)	
	Need to urinate more often	Some high-impact exercises could make varicose veins worse	A higher relative cardiac load during exercise				
	Leaking (urine), defecating, or vomiting during exercise		Distorted ratings of perceived exertion relative to exercising heart rate can lead to an increased risk of injury or adverse reaction				
	Feelings of vaginal heaviness and dragging during exercise						

(continued)

Table 14.1 (continued)

	Anatomical	Physiological					
		Integumentary	Cardiovascular	Respiratory	Digestion	Metabolism	Thermoregulatory
	Shortness of breath during exercise						
Panel d							
Practical considerations	Choice of activity (e.g., avoiding high-impact exercises), clothing (e.g., leak-proof underwear), and setting (e.g., access to toilets)	Choice of clothing (e.g., bra's) and footwear (e.g., comfortable shoes and socks)	Method of heart rate monitoring (i.e., direct recording rather than estimated or inferred is recommended)	Duration, intensity, and modality of activity (i.e., aerobic exercise is recommended for dyspnea)	Readiness to exercise due to side effects and symptoms	Continual self-monitoring of blood glucose levels—it may be better to exercise an hour after a meal and to take insulin medication well before exercise	Ability to distinguish between thermal sensation thermal discomfort and heat stress during exercise
	Inclination to exercise due to the extra burden and worry	Willingness to be seen in minimal or revealing clothing	Modality of exercise (e.g., aerobic exercise may be preferential)		Influence on the choice of activity and setting (e.g., access to toilets)	Motivation to exercise as gestational diabetes mellitus can cause mood dysregulations	Avoidance of hyperthermia in early pregnancy

(continued)

Table 14.1 (continued)

	Anatomical	Physiological					
		Integumentary	Cardiovascular	Respiratory	Digestion	Metabolism	Thermoregulatory
		Avoidance of activities with motionless standing	Supine positions should be avoided as much as possible during rest and exercise				Maintenance of euhydration, and therefore blood volume, is critical to heat balance
Panel e							
Supporting papers	Meekins and Siddiqui (2020)	Snarskaya et al. (2019)	Brislane et al. 2021	Milne et al. (1978)	Ferdinande et al. (2018)	Catalano et al. (1991)	Dervis et al. (2021)
	Cattani et al. (2021)	Motosko et al. (2017)	Magro-Malosso et al. (2017)	Kolarzyk et al. (2005)	Body and Christie (2016)	Stern et al. (2021)	Lindqvist et al. (2003)
	Body and Christie (2016)			Weissgerber et al. (1980)		Ali et al. (2020)	Clapp (1991)
	Nassikas et al. (2021)					Padayachee and Coombes (2015)	
	Forczek et al. (2018)						
	For an overview of the general changes that occur during pregnancy see Jackson et al. (2022), Tan and Tan (2013), and Artal and O'Toole (2003)						

Please note The details provided here are indicative, but not exhaustive

Sex Hormones

During the first trimester, the corpus luteum (i.e., an endocrine gland in the ovary formed when the follicle has discharged its secondary oocyte) secretes estrogens and progesterone in similar quantities to those produced during the luteal phase of the menstrual cycle. From the second trimester, the placenta adopts this secretory role. The placental unit, specifically the chorion (i.e., the fetal portion of the placenta), also secretes human chorionic gonadotropin (hCG), a glycoprotein hormone that imitates luteinizing hormone (LH), which further stimulates the production of estrogens and progesterone. Figure 14.2 shows the changes in 17-β-estradiol and progesterone before (panel a) and after (panel b) fertilization, during (panel c), and following pregnancy—with (panel d) and without (panel e) breastfeeding. The greatest increase in progesterone production occurs between weeks 18 and 30, during which time the placental unit produces approximately 250–500 mg of progesterone daily. During the last eight to ten weeks of gestation, little or no increase, and in some cases a slight decrease, in progesterone production occurs. At term, progesterone levels are 318–954 nmol L^{-1} (Tulchinsky et al. 1973), in comparison to 3–6 nmol L^{-1} and 32–111 nmol L^{-1} during the follicular and mid-luteal phases of the menstrual cycle (Abraham et al. 1972); a ninefold increase from the peak of the menstrual cycle to the peak of pregnancy. There is a rapid increase in 17-β-estradiol concentrations between weeks 20 and 35. At term, 17-β-estradiol levels are 22–110 nmol L^{-1} (Lindberg et al. 1974), in comparison to 0.4 and 1.5 nmol L^{-1}during the follicular and mid-luteal phases of the menstrual cycle (Abraham et al. 1972); a 73-fold increase from the peak of the menstrual cycle to the peak of pregnancy. In non-pregnant women, concentrations of estriol and estrone are less than 0.03 and 1 nmol L^{-1}; during pregnancy, these concentrations rise to approximately 35–104 and 7–110 nmol L^{-1} (Tulchinsky et al. 1973). In comparison to the other sex hormones, the rise in testosterone levels is weaker and more gradual; O'Leary et al. (1991) showed, between weeks 5 and 40 of pregnancy, a 1.7-fold increase in testosterone in comparison to a 33- and 11.9-fold increase in 17-β-estradiol and progesterone. As such, the concentrations of estrogens and progesterone during pregnancy are considered supraphysiological. During puerperium (i.e., the six weeks after childbirth when maternal physiologic changes related to pregnancy return to the non-pregnant state) and in the absence of lactation (i.e., the secretion and ejection of milk by the mammary glands), sex hormone levels return to normal values. Otherwise, following delivery, the withdrawal of progesterone coupled with the sharp rise in circulating prolactin levels—as a result of suckling—initiates and maintains milk production (i.e., lactation). Lactation can prevent ovarian cycles from occurring in the first few months following parturition, if suckling occurs eight to ten times per day, and is known as lactational amenorrhea. This phenomenon is, however, inconsistent; i.e., there is large between and within-individual variation in its occurrence and duration. It is worth noting that ovulation normally precedes the first postpartum menstrual bleed (i.e., ovulation is reinstated before menstruation returns).

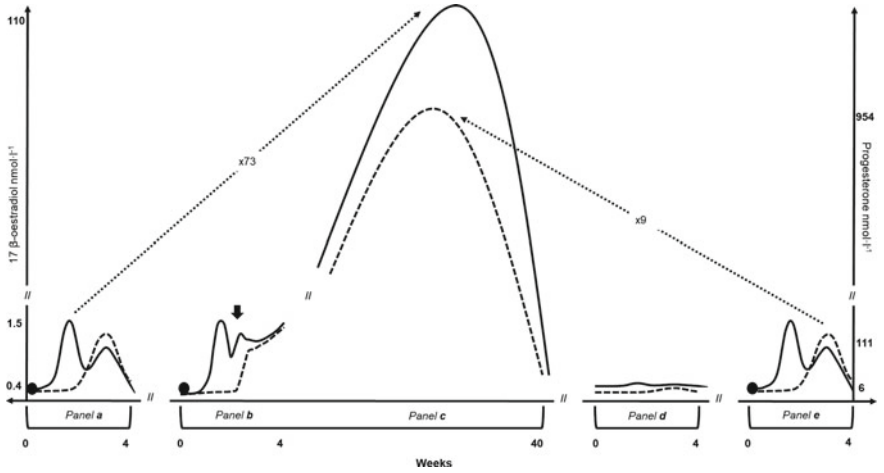

Fig. 14.2 Relative changes in 17-β-estradiol (solid line) and progesterone (dashed line) concentrations before, during, and after pregnancy. Panel **a** shows a eumenorrheic menstrual cycle without fertilization. Panel **b** shows a menstrual cycle with fertilization (as denoted by ↓). Panel **c** shows the hormonal changes during pregnancy. Panel **d** shows lactational amenorrhea (anovulatory cycles). Panel **e** shows the resumption of a eumenorrheic menstrual cycle following parturition. The circles denote menstruation. During pregnancy, the increase in 17-β-estradiol and progesterone can be as much as 73-fold and ninefold (Tal et al. 2001)

Anatomical and Physiological

During pregnancy, women gain approximately 11–16 kg, which consists of the following (approximate) changes: breasts 0.5 kg (equivalent to one to two bra cup sizes); uterus 1.6 kg (stretches 500–1000 times its pre-pregnancy size); placenta 0.7 kg; baby 3.5 kg; amniotic fluid 1–1.5 kg; and extra blood volume and fluid 4 kg. By the third trimester, the uterus occupies almost the entire pelvic cavity, resulting in the displacement of other organs (Fig. 14.3). These changes and subsequent outcomes are précised in Table 14.1 (panels a and b).

A myriad of physiological changes occurs during pregnancy. In short, pregnancy-related, physiological changes have been noted in the integumentary, cardiovascular, respiratory, digestive, metabolic, and thermoregulatory systems. These changes and downstream outcomes are summarized in Table 14.1 (panels a and b).

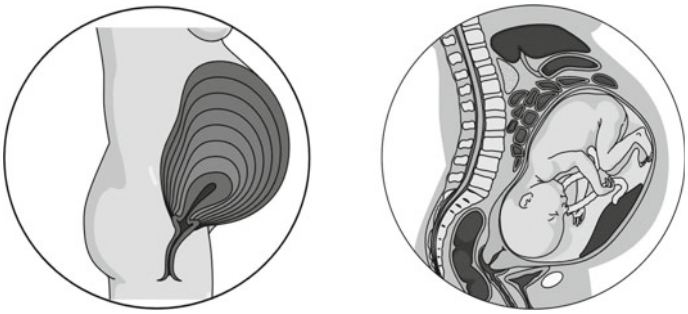

Fig. 14.3 Anatomical changes associated with pregnancy; note the enlargement of the uterus and the supplanting of the other internal organs

Functional Implications of Pregnancy on Exercise Performance and Capacity

In the absence of absolute and relative contraindications (Everson et al. 2014; Artal and O'Toole 2003), women are advised to maintain their current exercise and occupational habits (Mottola et al. 2018), as exercise has been shown to confer maternal and fetal benefits (Gregg and Ferguson 2017). Exercise performance and capacity could, however, be influenced by the changes that occur during pregnancy. In the following sections, the impact of supraphysiological concentrations of estrogen and progesterone on exercise performance and capacity is discussed in detail (i.e., in line with the focus of this chapter), while the impact of the anatomical and wider physiological changes (i.e., all physiological changes excluding the changes in sex hormone concentrations) is merely summarized in order to provide the backdrop information to the sex hormone-associated changes.

Sex Hormones

The following sections describe the relationship between sex hormones and athletic performance or components of athletic performance. These outcomes were explored as sex hormones have been implicated, either observed or theoretically, in the mediation, either solely or predominately, of these effects. This section complements the information provided in Table 14.1 related to the wider physiological data (i.e., all physiological changes excluding the changes in sex hormone concentrations).

Muscle

The potential influence of sex hormones on muscular function is often debated but seldom appropriately investigated (i.e., there is a lack of high-quality data on this topic). As such, the existence, direction, and magnitude of any effects remain to be scientifically (i.e., rather than anecdotally) established. Although muscle function has been assessed across menstrual and oral contraceptive pill cycles (McNulty et al. 2020; Elliott-Sale et al. 2020) and in response to menopause—with or without hormone replacement therapy—(Xu et al. 2020), it has rarely been assessed during pregnancy. Moreover, of the studies performed in this area, several have been cross-sectional study designs and few have included sex hormone analysis. For example, using a cross-sectional approach without the quantification of sex hormone status, Mbada et al. (2015) showed that pregnant women had significantly lower handgrip strength than non-pregnant women, while Abdullahi et al. (2021) did not observe any significant changes in handgrip strength between trimesters. Of those employing longitudinal study designs, Atay and Basalan (2015), Żelaźniewicz and Pawłowski (2018), and Bey et al. (2019), none measured sex hormone concentrations. Atay and Basalan (2015) measured handgrip strength at 20 and 32 weeks of pregnancy and observed a significant loss in strength as the pregnancy progressed. Similarly, Żelaźniewicz and Pawłowski (2018) showed that handgrip strength decreased from the first to the third trimester of pregnancy. Conversely, Bey et al. (2019) did not show any change in the lower limb (i.e., knee extensor) muscle strength between early and late pregnancy (16 ± 4 and 29 ± 4 weeks of pregnancy). Using a cross-sectional design—comparing pregnant (week 12, 20, and 36 of pregnancy) and postpartum women (six weeks following childbirth)—Elliott et al. (2005) showed that concentrations of total and bioavailable 17-β-estradiol, progesterone, and total testosterone were highest at week 36 of pregnancy (i.e., during the third trimester) and lowest at six weeks postpartum (Fig. 14.4). Bioavailable testosterone was also highest at 36 weeks of pregnancy but was still significantly elevated at six weeks postpartum. Concentrations of total estradiol were significantly different between all stages (all $p < 0.001$); concentrations of progesterone were significantly different between all stages (all $p < 0.001$) except between the first and second trimesters; concentrations of total testosterone concentration were significantly different between weeks 12 and 36, and 36 and 6 (all $p < 0.001$); concentrations of bioavailable estradiol were significantly different between all stages (all $p < 0.05$) except between weeks 20 and 36; and the concentration of bioavailable testosterone was significantly greater six weeks following childbirth than during week 20 ($p < 0.05$). Maximum voluntary isometric force per unit cross-sectional area of the first dorsal interosseus muscle was significantly higher at weeks 12 and 20 of pregnancy compared to six weeks postpartum; i.e., women in this study were stronger during the first and second trimesters than following parturition. The changes in strength did not, however, significantly and positively correlate to changes in sex hormone concentrations. As such, the authors proposed that the changes they observed in strength, during the first two trimesters of pregnancy, were not caused by supraphysiological concentrations of 17-β-estradiol and progesterone, although they suggested that further investigation

of this occurrence is warranted. Clearly, the impact of sex hormones on muscle function during pregnancy needs to be further investigated, ideally using longitudinal research designs (e.g., pre-pregnancy, throughout pregnancy, and postpartum—with and without lactation) with hormonal analysis. In addition, the quality of studies in this area could be further advanced by reporting gestational stage, parity, gravidity, and whether it is a singleton or twin/triplet/etc. pregnancy (Elliott-Sale et al. 2021), which will increase the accuracy and validity of the population sampled and reduce the variability in hormone status between participants.

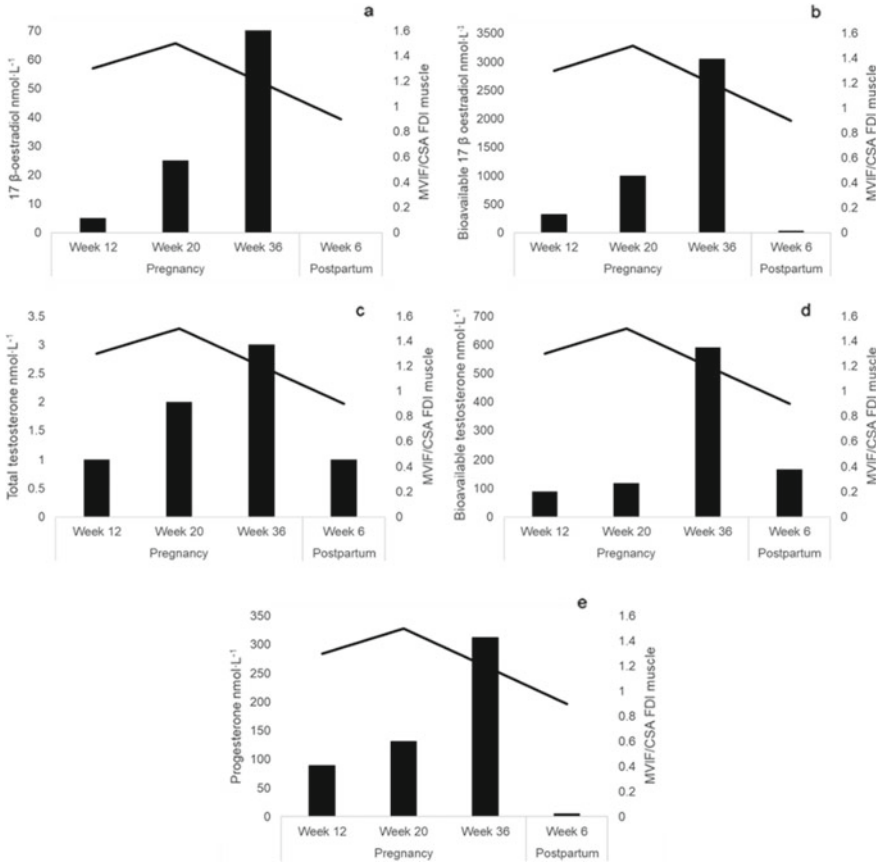

Fig. 14.4 Concentrations (bars) of total (panel **a**) and bioavailable (panel **b**) 17-β-estradiol, total (panel **c**) and bioavailable (panel **d**) testosterone and progesterone (panel **e**), and maximum voluntary isometric force per unit cross-sectional area (MVIF/CSA) of the first dorsal interosseus (FDI) muscle (lines) during (week 12, 24, and 36) and following (6 weeks) pregnancy

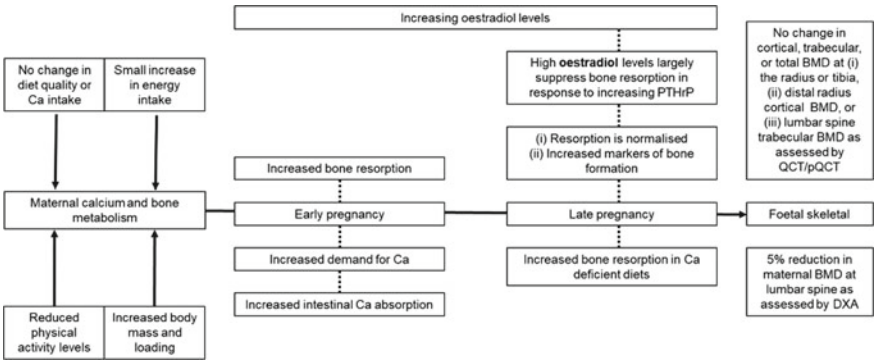

Fig. 14.5 Sequence of events involved in the regulation of maternal and fetal bone metabolism. Adapted from Winter et al. (2020). Abbreviations: *PTHrP* parathyroid hormone-related protein, *Ca* calcium, *BMD* bone mineral density, *DXA* dual X-ray absorptiometry, *QCT* quantitative computed tomography, *pQCT* peripheral quantitative computed tomography

Bone

During pregnancy, a complex cascade of events, involving many variables including 17-β-estradiol, is responsible for the changes in maternal bone health; these actions are summarized in Fig. 14.5. The effect of pregnancy on bone has been well documented (Kalkwarf and Specker 2002; Liu et al. 2019; Salari and Abdollahi 2014). The prevalence of pregnancy and lactation-induced osteoporosis is unknown but is believed to be rare. Its cause is currently unknown, although it is likely to be triggered by a combination of hormonal (oxytocin) influences and contributions from the sympathetic nervous system, genetics, and bone marrow fat (Winter et al. 2020). On the other hand, Song et al. (2017) showed, using a meta-analytical approach, that parity has a positive effect on bone in healthy, community-dwelling women, especially at the total hip site. In addition, fractures sustained during pregnancy appear to heal more quickly than during the non-pregnant state, and this effect is likely underpinned, at least in part, by estrogen (Beil et al. 2010), which is at supraphysiological levels during pregnancy.

Athletic Performance and Capacity

In the absence of complications, pregnancy-related confinement is no longer a viable (e.g., loss of earnings/sponsorship) or attractive (e.g., loss of training time and competition opportunity) option for competitive sportswomen. This sociocultural shift has prompted discussion on the effects of pregnancy on training and competition practices; however, the majority of previous research has examined the effects of exercise on pregnancy (i.e., maternal and fetal implications; see Wowdzia et al. 2021 for a review of this literature) as opposed to the effects of pregnancy on exercise (i.e.,

from a sports training or competition perspective). To date, no study has taken corresponding blood samples, to assess the hormonal milieu, alongside their outcome measures, meaning that the direct relationship between sex hormones and athletic performance and capacity during pregnancy is currently unknown. In the absence of this research, the general effects of pregnancy on exercise-related practices in elite and top-level sportswomen are summarized below. Of note, Hale and Milne (1996) commented that exercise in the elite athlete is only an accentuation of that found in the recreational athlete.

Using retrospective self-reported surveys, Beilock et al. (2001) showed that the number of athletes (89%) who trained during the first trimester fell by 24–65% in the third trimester, without a significant impact on their postpartum training programs. Similarly, Tenforde et al. (2015) reported that 70% of runners ran at some point during their pregnancy, but only 31% ran during their third trimester. They also showed that, on average, pregnant women reduced their training intensity to almost half of their non-pregnant running effort. Franklin et al. (2017) reported that 85% of pregnant recreational and elite rowers exercised during past pregnancies; 51.3, 42.4, and 15.7% of pregnant rowers met or exceeded the national guidelines during the first, second, and third trimesters. Sundgot-Borgen et al. (2019) also observed a downward trajectory in training volume from the first to the third trimester in 34 Norwegian elite athletes.

Conversely, when training was either monitored/tracked or prescribed, participation in moderate to high-level activity was (i) maintained and (ii) beneficial during pregnancy. Using a longitudinal observational approach, Clapp and Capeless (1991) showed a small but significant increase in maximum oxygen uptake in recreational athletes who maintained a moderate to high level of exercise training during and after pregnancy. Comparably, Kardel (2005) showed that top-level sportswomen maintained their high fitness levels during pregnancy when prescribed, appropriately strenuous, training.

Several case studies have investigated the lived experiences of elite athletes. Davies et al. (1999) conducted a case study on an elite marathon runner who preconceptionally ran 155 km week^{-1} at an intensity equivalent to 140–180 b min^{-1}. During pregnancy, the athlete completed 107 ± 19 km week^{-1} at an intensity equivalent to 130–140 b min^{-1}. Running velocity at a steady-state heart rate of 140, 150, and 160 b min^{-1} decreased by 20, 15, and 13% during pregnancy from week 1–32 antepartum. These data suggest it is possible for an elite endurance athlete to maintain a high exercise capacity during pregnancy. Soli and Sandbakk (2018) conducted a case study investigating the training characteristics and physiological capacity, of the world's most successful cross-country skier during pregnancy. Training volume was maintained at approximately 80–85% of pre-gravid load during the first and second trimesters but was reduced to approximately 50% in the third trimester. Training intensity was modified during pregnancy: (i) High-intensity training ceased at week 5 of pregnancy, and low and moderate-intensity training was used instead; and (ii) strength training was progressively modified. Oxygen uptake at the lactate threshold changed from 60.8 ml kg^{-1} min^{-1} before pregnancy to 57.0 ml kg^{-1} min^{-1} in the first trimester and 54.2 ml kg^{-1} min^{-1} in the second trimester. These findings suggest

that exercise (training) capacity can be maintained at a high level during the first two trimesters of pregnancy; however, substantial modifications are needed in the third trimester.

Psychosomatic

Although outside of the scope of this chapter, the psychological implications of pregnancy on exercise performance and capacity also need to be taken into account. Sex hormones have been shown to affect mood (i.e., resulting in mood disorders such as depression), especially during pregnancy and the postpartum period (Zsido et al. 2017) and, as such, the psychosomatic interactions between sex hormones, pregnancy, and exercise also need to be considered.

Anatomical and Physiological

The anatomical and physiological changes, which occur during pregnancy, largely result in discomfort (physical and emotional) for the exercising woman (Table 14.1; panel c). Moreover, these changes are associated with an increased risk of injury and illness. The prevalence and severity of these effects are subject to large within and between individual variability, but have been shown to impact the quality of life and exercise performance and capacity of physically active pregnant women (Barone Gibbs et al. 2021; Di Fabio et al. 2015; Everson and Wen 2011; Lagadec et al. 2018; Owe et al. 2009). Indeed, data from Whitaker et al. (2022) suggest that quality of life and participation in moderate to vigorous-intensity physical activity is affected by the stage of pregnancy, with better quality of life and participation in exercise seen in the second trimester, which is associated with fewer and less severe pregnancy-related symptoms.

Practical Implications of Pregnancy on Exercise Performance and Capacity

Sex Hormones

Given the paucity of information related to the effects of sex hormones on exercise performance and capacity, it is pre-mature to propose many practical considerations directly stemming from the changes in sex hormones experienced during pregnancy. Further high-quality research is needed and must include the determination of estrogens and progesterone from blood samples. Often, there is a reluctance to study the effects of pregnancy on exercise performance due to ethical concerns about the safety

of the mother and baby; however, the majority of recent evidence suggests that it is safe for both pre-gravid sedentary and well-trained women to exercise during pregnancy (Bø et al. 2016). At present, the only recommendations, based on the current state of the art, are related to the changes in bone health: (i) The choice (e.g., avoiding high-impact exercises, bending and twisting) and setting (e.g., slippery surfaces) of the activity need to be considered; and (ii) the disposition to exercise due to anxiety (i.e., fear of sustaining a fracture) needs to be taken into account.

Anatomical and Physiological

There are numerous practical considerations associated with the profound structural and functional changes that occur during pregnancy, as shown in Table 14.1 (panel d). Pregnant women, practitioners, and researchers need to be mindful of these factors in order to undertake/prescribe safe (i.e., to mother and fetus) and appropriate physical activity during pregnancy. In general, the mode, duration, intensity, frequency, and progression of the activity need to be assessed and potentially modified during pregnancy, perhaps even on a trimester-by-trimester basis. In addition, consideration needs to be given to the setting (e.g., location, environment), staffing (e.g., supervised versus unsupervised), equipment requirements (e.g., clothing, footwear), and motivation (e.g., weight management, elite sport, underlying medical conditions) associated with the proposed exercise program.

General Principles of Exercise Prescription During Pregnancy

In addition to the practical considerations listed above (i.e., as a result of the pregnancy-related changes in sex hormones, anatomy, and physiologic function), there are a number of general exercise principles that should be adhered to:

1. Medical screening (continual monitoring throughout pregnancy)—to establish relative and absolute contraindications to exercise (Artal and O'Toole 2003).
2. Consideration of pregnant women with comorbidities (e.g., obesity, chronic hypertension)—to further tailor the mode, duration, intensity, frequency, and progression of activity (Artal and O'Toole 2003).
3. Consideration of pre-gravid fitness—(i) 30 min or more of moderate-intensity physical activity on most, and preferably all, days of the week is suitable for all women with uncomplicated pregnancies regardless of pre-pregnancy activity level (Pate et al. 1995); (ii) competitive athletes experience the same pregnancy-related changes, implications, and limitations as recreational athletes, as such they require closer and more frequent obstetric supervision than routine care in order to maintain their more strenuous training programs (ACOG 2020).

4. Adequate fueling—to maximize maternal and fetal outcomes and limit excessive gestational weight gain (Elliott-Sale et al. 2019).
5. Consideration of post-exercise recovery—recovery time and hydration and nutrition strategies need to be considered (Soultanakis et al. 1996; Mottola et al. 2013).
6. Sports with a high likelihood of contact (e.g., ice hockey), falling (e.g., horse-riding, skating), and decompression sickness (e.g., scuba diving) should be avoided (ACOG 2002).
7. Consideration of altitude—(i) pregnant lowlanders can exercise (i.e., moderate intensity) safely between 1800 and 2500 m if acclimatized; (ii) pregnant highlanders can most likely exercise (i.e., moderate intensity) safely above 1800 m; and (iii) elite athletes need medical clearance to undertake strenuous (i.e., vigorous intensity) training at altitude (Artal et al. 1995; McManis 2021).

The Fourth Trimester

The first three months following childbirth are colloquially referred to as the ***fourth trimester***. For the majority of women with uncomplicated pregnancies and deliveries, most pregnancy-related anatomic and physiologic changes resolve spontaneously during the fourth trimester. As a result of gestational weight gain and the detraining that undoubtedly occurs during pregnancy, women should return gradually to exercise (Artal and O'Toole 2003). For some, pregnancy has been proposed as an ergogenic aid to athletic performance, due to the anecdotal accounts of improved exercise performance following childbirth; however, Pivarnik et al. (2017) concluded that there is insufficient data to confirm this tenet at present. Conversely, for other women, the legacy of pregnancy can be damaging, with sustained, long-term negative consequences, which need to be addressed (Jackson et al. 2022).

Conclusions

Pregnancy causes a multitude of morphological and physiological changes, which include supraphysiological concentrations of estrogens and progesterone. These changes result in numerous adaptations and implications for exercise capacity and performance. Given the diversity (Table 14.1) and interrelatedness (Fig. 14.1) of the changes associated with pregnancy, a multidisciplinary approach to exercise testing and prescription is recommended, with a specific focus needed on the direct and indirect effects of sex hormones on exercise performance and capacity, although the constellation of changes that occur during pregnancy makes it almost impossible to elucidate the discrete effects of sex hormones on exercise capacity and performance during pregnancy (Fig. 14.6).

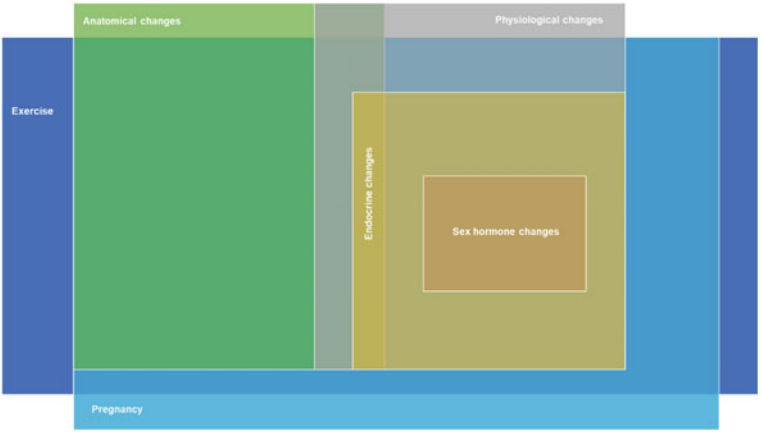

Fig. 14.6 Euler diagram showing the overlapping complexity of how the changes during pregnancy affect exercise performance and capacity

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