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REVIEW ARTICLE

The Impact of Hormonal Contraceptives on Skeletal Muscle Hypertrophy

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Abstract

Female reproductive hormones such as progesterone and estrogen play an important role in the body as they orchestrate functions of numerous cells including skeletal muscle cells. Onset of menses marks the beginning of reproductive life, whereas menopause marks its cessation. We distinguish three phases of the 28-day menstrual cycle namely follicular, ovulatory and luteal phases. The follicular phase is characterised by marked increases in estrogen, which triggers ovulation. During this phase, estrogen peaks, whereas progesterone levels are low. Following ovulation, the luteal phase commences marked by high progesterone levels and reduced estrogen. Indeed, these periodic fluctuations in reproductive hormones may affect rates of muscle protein synthesis and hence hinder sought adaptations such as skeletal muscle hypertrophy in female athletes. With the introduction of hormonal contraceptives, female athletes were able to have ameliorate the negative effects of the menstrual cycle by reducing menstrual cramping and bleeding. Hormonal contraceptives are constituted of a single or multiple synthetic hormones namely estrogen and progestin. Nevertheless, the impact of hormonal contraceptives on skeletal muscle hypertrophy remains elusive. In this review, we aim to present the potential implications of hormonal contraceptives on skeletal muscle hypertrophy.

Keywords: hormonal contraceptives · muscle hypertrophy · menstrual cycle · resistance training · estrogen · progesterone

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Introduction

Female reproductive biology has received substantial scientific attention in the context of sport and exercise. Although the divergence in markers of exercise performance between males and females before puberty is only minor, onset of puberty is accompanied with a significant change in these markers, which are at least partly mediated by changes in sex hormones such as estrogen, progesterone and testosterone. These puberty-related hormonal changes are partly mediated by gonadotropin-releasing hormone (GRH), which is released by the hypothalamus in the hypothalamic-pituitary-gonadal (HPG) axis, and promotes release of luteinizing (LH) and follicle-stimulating hormones (FSH) (Herbison, 2016). Secretion of LH and FSH during puberty modulates testosterone, estrogen and progesterone production from the gonads. In females, the GRH-mediated cascade during puberty initiates the menstrual cycle (MC), which cyclically repeats approximately every 28 days from the first menstruation (menarche) until menopause.

The MC starts with vaginal bleeding (menstruation), which persists between day 0 to day 5, and marks the onset of the follicular phase (Critchley et al., 2020). The follicular, or proliferative, phase starts on the first day of the MC and continues until day 14, based on the duration of an average 28-day MC. However, the duration of the follicular phase, which determines the duration of the MC, is influenced by the time it takes for one dominant follicle to develop (Draper et al., 2018). During menstruation, the functional layer of the endometrium, which includes glandular and stromal components that have thickened in preparation for potential pregnancy, separates and flows out. Pain due to uterine contractions often occurs, while estrogen and progesterone levels are still low. Some evidence indicates that while stable hormone levels protect females from nociception, fluctuations in estrogen levels may cause pain (Athnaiel et al., 2023). At the end of menstruation, the pituitary gland starts releasing FSH (Orlowski & Sarao, 2024). This hormone stimulates the ovaries to produce follicles that contain immature eggs (Miro & Aspinall, 2005). The dominant hormone during this phase is estrogen, or estradiol, which is the main secreted isoform from puberty until menopause. While progesterone levels are still low, estrogen levels tend to be at higher. In the days that follow, just one follicle gains dominance. The uterine endometrium grows new tissue as a result of estrogen production, thickening and preparing the endometrium for the implantation of an embryo. In normal MC, ovulation takes place on day 14, which is halfway through the MC (Holesh et al., 2024). The spike in LH is influenced by a significant increase in estrogen (Nedresky & Singh, 2024), then the follicle bursts and releases the mature egg cell into the fallopian tube, which is viable for the next 12 to 24 hours (Oliver & Basit, 2024). If fertilization is unsuccessful, the egg cell leaves the body during the next menstruation. With ovulation, the level of estrogen starts to rapidly decline and secretion of progesterone becomes more dominant. After ovulation, this second phase of the MC is called the luteal, or secretory, phase. The dominant follicle turns into a structure called the corpus luteum (Devoto et al., 2009). If pregnancy does not occur, the corpus luteum disappears and the drop in progesterone affects the remodelling of the endometrium. Figure 1 shows the fluctuation of hormones during a normal MC.

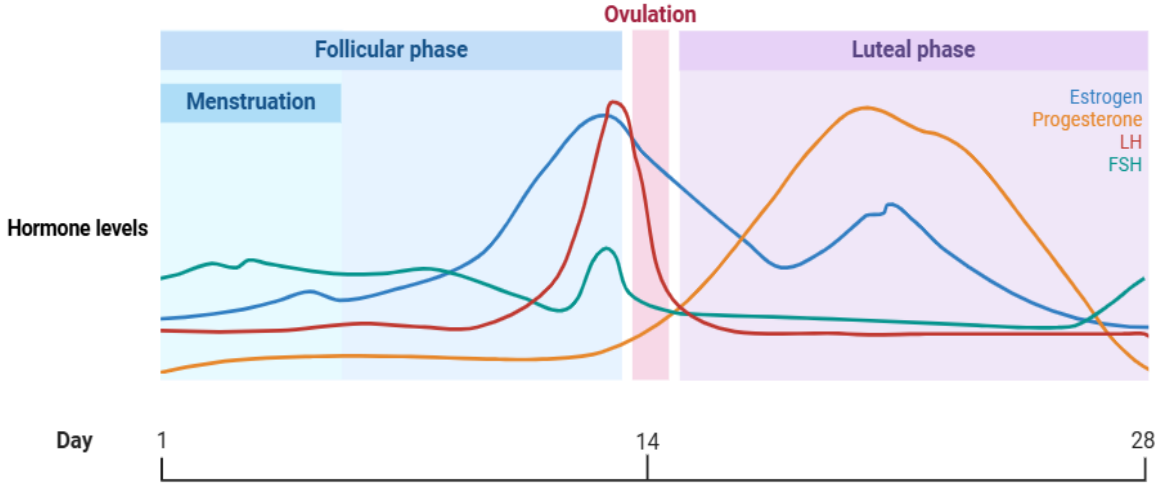

Figure 1. Hormonal fluctuation during a normal MC. Changing concentrations of female sex hormons(estrogen, progesterone, LH, FSH) that characterize phases of the menstrual cycle

Abnormal MC pertaining to frequency, regularity, duration, and volume of flow that do not occur during pregnancy are together referred to as abnormal uterine bleeding (AUB) (Davis & Sparzak, 2024). Up to 90% of females experience issues related to their MC during adolescence (Odongo et al., 2023). Often, women experience amenorrhea, which is characterised by the absence of menstruation over a prolonged period. Primary amenorrhea is the lack of menarche, whereas secondary amenorrhea is the absence of menstruation in a woman with previously regular and normal cycles for at least six months (Klein et al., 2019). Age, weight, physical activity, diet, coffee intake, smoking, workplace, medical conditions, and lifestyle choices have all been implicated in altered MC (Saei Ghare Naz et al., 2022).

The cycle consists of ovulation and two entirely distinct phases. Throughout the cycle, a woman's body functions differently and her hormone levels also fluctuate. Premenstrual syndrome (PMS) precedes bleeding and is characterized by anxiety, exhaustion, pain, insomnia, and social isolation (Mishra et al., 2024). The same authors discuss premenstrual dysphoric disorder (PMDP), a diagnosis made when a woman's ability to function is impaired by more severe symptoms. During the luteal phase, there is also a reduced motivation to engage in physical activity and a greater sensitivity to physical exertion (Green & Graham, 2022). During menstruation, the characteristics of PMS deteriorate, as a result of which women deviate from exercise and other work habits. Progesterone has an anxiolytic effect, promotes calmness and reduces anxiety (Reddy et al., 2005). It is believed that its decline during PMS affects the previously described changes. Estrogen, on the other hand, is responsible for regulating the level of serotonin, which is a potent modulator of appetite, physical activity and mood (Hwang et al., 2020). This hormone grows during the follicular phase, so a woman's mood is the best before ovulation.

The MC is also influenced by acute and chronic perturbations in energy balance. For instance, menstrual dysfunction is a common symptom of suppressed sex hormones caused by low energy availability (LEA), a state where energy intake is substantially lower than energy demands. LEA is a developing issue that can cause athletes to face a number of challenges (Suzuki & Suzuki, 2024). For the best health and performance, female athletes should strive for energy availability of 45 kcal·kg–1 fat-free mass·day–1 (Holtzman & Ackerman, 2021). Although there is no absolute universal threshold for energy availability, disruptions to multiple hormonal secretory patterns have been observed in as little as 4-5 days in untrained adult women who fall below an energy availability threshold of approximately 30 kcal kg−1 fat-free mass day−1 (Loucks & Thuma, 2003). LEA can be brought on by dietary restriction or increased exercise energy expenditure (i.e., failing to increase caloric intake to match increased exercise) (Wikström-Frisén, 2016). Levels of LH and FSH, as well as progesterone and estrogen, are often impacted by LEA, which may have both acute and chronic detrimental effects such as impaired judgement and coordination, as well as reduced bone mineral density and muscle protein synthesis (Ihalainen et al., 2024). Menstrual irregularities, low bone mineral density, and LEA are symptoms of a syndrome known as the female athlete triad that affects young women who are physically active (Maya & Misra, 2022). LEA and physical or mental stress can both result in the HPG axis being downregulated (K. J. Elliott-Sale et al., 2018). Also, HPG axis is inhibited by contraceptive pills, which suppresses follicular growth and prevents ovulation (de Castro Coelho & Barros, 2019).

Method

A structured literature search was conducted to identify relevant studies examining the impact of hormonal contraceptives on skeletal muscle hypertrophy. The following databases were searched: PubMed, ResearchGate and ScienceDirect. Search terms included combinations of “hormonal contraceptives” OR “oral contraceptive pills” OR “OCP” AND “muscle hypertrophy” OR “muscle strength” OR “resistance training” AND “females” OR “women” OR “female athletes”. The literature search was conducted between April 1st, 2024, and January 1st, 2025. Included studies were original research articles or systematic reviews that investigated healthy women, including both athletes and non-athletes. The studies had to report on outcomes related to resistance training adaptations and/or muscle hypertrophy, and only articles published in English were considered eligible. Studies were excluded if they involved animal subjects, were case reports or expert opinions, or examined hormonal contraceptive methods other than oral contraceptive pills, unless they provided comparative data that included oral contraceptive use. The selection process involved screening titles and abstracts for relevance, followed by full-text review of potentially eligible articles.

Results

Oral contraceptives (OC) are a non-invasive hormone-based strategy used for prevention of conception, for the treatment of acne, irregular menstruation and other menstrual-related disorders (Evans & Sutton, 2015). Today, 87% of US women confirm the use of contraception pills throughout their lives (Teal & Edelman, 2021). Generally, progesterone and estrogen are combined in two types of combination OCs: monophasic and multiphasic (Kitson, 2022). According to the same author, each OC contains a different amount and ratio of synthetic hormones. In contrast to monophasic pills, which continuously contain the same dose of progestin and estrogen, multiphasic pills contain varying amounts of synthetic hormones throughout the cycle. On the other side, there are two types of OCs: progesterone-only and combination estrogen-progesterone (Cooper et al., 2024). It is advisable to start taking OCs on any day between the first and the fifth day of the MC, continue for 21 days, and then discontinue (Shah & Patil, 2018). This will be followed by withdrawal from vaginal bleeding. Thereafter, the first day of withdrawal from bleeding is considered day 1. The fluctuation of dominant hormones whilst using OCP is substantially different (Figure 2).

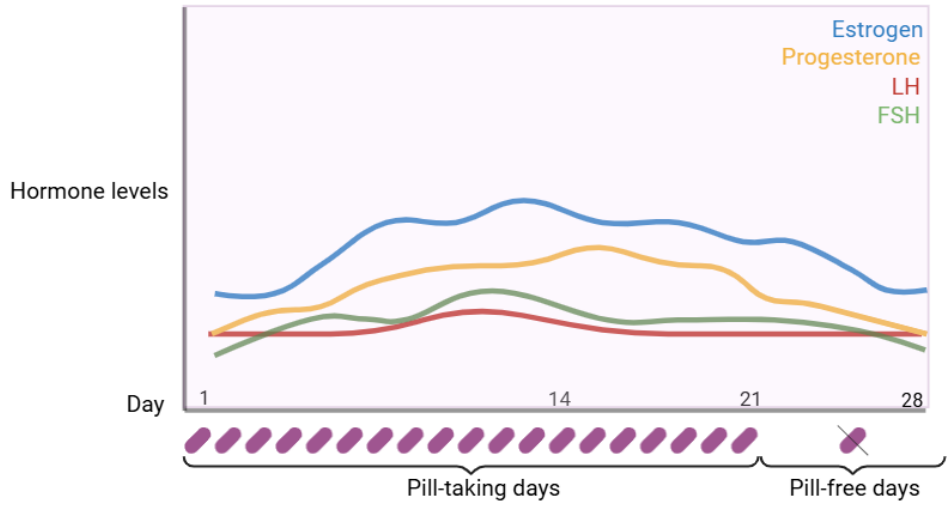

Figure 2. Hormonal fluctuation while taking an OCP containing both estrogen and progesterone. Different hormone concentrations compared to the menstrual cycle of females who do not use OCP

OCP are common among competitive and recreationally active young women because they control the entire MC, which is seen as a barrier to physical exercise (Schaumberg et al., 2018). In female elite athletes, OC are used by almost half of them (Reif et al., 2021). There is evidence that the OCP can directly and indirectly affect markers of sport and exercise performance (K. Elliott-Sale et al., 2020).

OCP not only offer diverse applications, such as managing menstrual-related disorders and facilitating athletic performance, but their primary purpose remains the prevention of pregnancy (Cooper et al., 2024). The primary hormone that prevents pregnancy is progesterone. The hypothalamus uses progesterone negative feedback to lower GRH pulse frequency. FSH and LH output would consequently decline as a result of this. Estradiol levels don't rise if the follicle is not developing because the follicle produces estradiol. Ovulation is inhibited when there is no follicle development and no increase in LH to release the follicle (Sondheimer, 2008).

Discussion

Skeletal Muscle Hypertrophy and Oral Contraceptives

One of the processes that is important for athletes is hypertrophy of the skeletal muscle. When a positive net protein balance is reached and synthesis of muscle proteins exceeds their breakdown over time, muscular hypertrophy occurs (Damas et al., 2018). The smallest contractile unit, a sarcomere is primarily made up of thick (myosin) and thin (actin) filaments. Actin-myosin cross-bridges are made up of many myosin heads that join to actin in the thin filaments to form the thick filaments (Ahmed et al., 2022). Sarcomeres can be added in parallel or series to induce hypertrophy (Schoenfeld, 2010). Overload stimuli that affect skeletal muscle disrupt the structure of myofibers and the surrounding extracellular matrix. Actin and myosin, two myofibrillar contractile proteins, grow in size and

quantity as a result, as does the total number of sarcomeres in parallel. Consequently, this increases the diameter of individual fibers, increasing the cross-sectional area of the muscle.

The mTOR (mammalian target of rapamycin) signalling pathway is a crucial regulator of protein synthesis and ribosome biogenesis. It also plays a major role in hypertrophic-hyperplasic growth and cellular metabolism (Yang et al., 2008). mTOR functions as a catalytic component of two different complexes in mammals, called mTOR complex 1 (mTORC1) and complex 2 (mTORC2) (Roberts et al., 2023). Activation of the mTORC1 stimulates protein synthesis by increasing mRNA translation and increasing the activity of translation factors. This can lead to an increase in muscle mass and improvement of recovery from muscle injuries (Yoon, 2017). As a strong signal in the mTOR signaling pathway, leucine promotes the activation of the mTORC1 complex (Dodd & Tee, 2012). The activation of mTORC1 in response to overload stimuli has been attributed to a number of signals over time. These signals include growth factor signalling (namely insulin-like growth factor 1 or IGF1), membrane-associated proteins involved with mechanotransduction and proteins involved with amino acid sensing that converge to activate mTORC1 (Roberts et al., 2023).

Mechanical tension, muscle damage, and metabolic stress are the three primary factors that cause the hypertrophic response during resistance training (Kizilay et al., 2024). Using heavy loads during the eccentric phase of a movement has been linked to mechanical tension and severe exercise-induced muscle injury, both of which have been linked to a hypertrophic response (Krzysztofik et al., 2019). Metabolite accumulation in muscle cells, including lactate, phosphate inorganic (Pi), and hydrogen (H+) ions have been reported and signalling metabolic factors that mediated exercise-induced metabolic stress (de Freitas et al., 2017). The amount of metabolic stress during acute exercise is determined by changes in intensity, volume, and rest intervals.

Predicting the hypertrophic response to resistance exercise appears to depend on the amount of androgen receptors in skeletal muscle (Morton et al., 2018). It is unclear exactly how progesterone and estrogen affect muscular strength and function. Estrogen may have a role in promoting hypertrophy by reducing the damage that exercise causes to muscles and increasing the activity of anabolic signaling pathways that are important for muscular anabolism (Gharahdaghi et al., 2021). These hormones affect a variety of physiological processes, and how they behave during exercise may have an impact on performance. Their level varies throughout the MC, as was previously mentioned. Estrogen concentrations during the luteal phase reduce the demand for muscle glycogen during exercise. Estrogen promotes endurance performance during exercise by increasing the availability of free fatty acids and oxidative capacity (Jensen et al., 1994). Furthermore, the precise mechanism by which progesterone accelerates the breakdown of muscle proteins remains unclear, though it could be linked to increased satellite cell activation (Smith et al., 2014). OCP may impact steroid hormone receptors in peripheral tissues like skeletal muscle and tendon, which may have an impact on sports performance (Dalgaard et al., 2019). Reduced androgen levels are caused by amenorrhea and OC intake (Bermon et al., 2014).

Sex differences in exercise physiology

There are differences between sexes within physiological systems implicated in exercise performance. At the outset, skeletal muscles should be mentioned. Men tend to have a higher capacity for glycolysis (Esbjörnsson et al., 1993), whereas females have a higher capacity for whole-muscle oxidative processes (Russ et al., 2005). Also, by using high-resolution respirometry following muscle biopsy, it was determined that trained males and females had different mitochondrial oxidative functions. Specifically, females showed about one-third higher intrinsic respiratory rates in their mitochondria than males (Cardinale et al., 2018). As for the latter, in females, the proportionate area of type I fibres is greater (Nuzzo, 2024). In comparison to type II fibres, type I fibres also exhibit slower Ca2+ kinetics, poorer power generation, and slower shortening and relaxation velocities (Schiaffino & Reggiani, 2011). It is important to note that females have more capillaries per skeletal muscle unit than males (Roepstorff et al., 2006).

Substrate use during physical exercise can also differ between males and females. Females oxidize more lipids during submaximal exercise, as indicated by a lower respiratory exchange ratio (Cano et al., 2022). Notably, estradiol increases fat oxidation, thus having a glycogen sparing effect during exercise (Hackney, 1999). On the other hand, progesterone could counteract the effects of estradiol (Beidleman et al., 2002). The impact of MC and OC phases on substrate oxidation during rest and moderate-intensity continuous exercise was examined. It was observed that substrate oxidation remained consistent across the stages of the MC. However, there was lower confidence in these results, and a similar lack of variation was noted in substrate oxidation among OC users (D'Souza et al., 2023).

Other sex differences can also affect the divergence in exercise-related physiology. As with skeletal muscle, females generally have smaller airways, altered lung shape, and smaller lungs than males (Molgat-Seon et al., 2018; Schwartz et al., 1988). These anatomical variations have an impact on elements like respiratory efficiency and the work of breathing, which is the product of volume and pressure for each breath. As for the nervous system, it should not be forgotten that muscle contraction is initiated by the central nervous system (CNS). Voluntary activation differs during specific stages of the MC. It appears that voluntary activation is the highest when progesterone levels are lower and estrogen concentrations are higher. On the other hand, voluntary activation is the lowest when progesterone concentrations are high (Ansdell et al., 2019).

Periodization for the female athlete

Assuming that the MC affects women's athletic performance, research has been conducted that focused on periodization of training. Compared to regular training, menstrual-adjusted training showed higher strength adaptations (Reis et al., 1995). The training units were scheduled every other day during the follicular phase and roughly once a week, with almost continuous testing intervals in between during the luteal phase. This was the basis for the MC induced training concept. During the periodization process, it is important to note that the luteal phase of the MC experiences a higher rise in protein catabolism than the follicular phase. (Lamont et al., 1987). In addition, growth hormone in women is associated with increased estrogen levels (R. R. Kraemer et al., 1995). However, muscular growth is not significantly impacted by the elevated estrogen levels that occur in the last 1 - 2 days of the late follicular phase (Sakamaki-Sunaga et al., 2016). During the follicular phase, maximum voluntary contraction increases, and during ovulation, muscular strength is reduced (S. K. Phillips et al., 1996).

Mixed results are found regarding the MC's impact on athletic performance outcomes. According to the female athletes themselves, the period immediately after bleeding is optimal for perceived fitness and performance (Solli et al., 2020). It has been shown that during the luteal phase, track and field athletes' 100- and 200-meter running performances were much quicker; however, in the 2,000-meter race, there was no difference between menstrual phases (Guo et al., 2005). In one research, there was no discernible variation in strength throughout the MC, although there was a decrease in counter-movement jump performance and Wingate average power at the end and beginning of the cycle (Dam et al., 2022). In contrast, some studies have shown that menstrual phases do not affect adaptations during strength training (Colenso-Semple et al., 2023; de Jonge et al., 2001). There are several possible explanations for the diversity observed in the effects of MC. These variables include variations in the selected performance challenge and the athletes' level of skill. The most significant is that several studies have not accurately determined the participant's real MC phase. Researchers must use gold-standard techniques in order to completely explain how ovarian hormones affect important facets of exercise physiology (Van Every et al., 2024). Additionally, there is a great deal of intraindividual variation in how menstruation symptoms affect training and performance. It's possible that there will never be a standard template that researchers and practitioners can use to guide training and performance because each woman's MC is unique and can change over the course of a lifetime (K. J. Elliott-Sale et al., 2021).

The impact of oral contraception on skeletal muscle hypertrophy

Muscle and other musculoskeletal tissues contain estrogen receptors (Barros & Gustafsson, 2011). Women who menstruate also experience a higher incidence of ACL ruptures than men, which is consistent with estrogen's involvement in controlling musculoskeletal function (Shultz et al., 2011). As for the latter, during menopause, there is a higher chance of musculoskeletal injuries (Enns & Tiidus, 2010). Estrogen influences metabolism in skeletal muscle in a number of ways. Animal research shows that low levels of estrogen cause abnormalities such as insulin sensitivity, membrane microviscosity, in levels of antioxidant proteins, and mitochondrial activity (Baltgalvis et al., 2010; Torres et al., 2018). However, there is greater interest in the way that estrogen influences skeletal muscle mass and strength. Estrogen may play a role in increasing sensitivity to anabolic stimuli because it has been connected to myogenic gene expression after resistance training (Dieli-Conwright et al., 2009). Given that the use of OCs modifies endogenous hormone production, the impact of estrogen on myofibrillar protein synthesis in response to anabolic stimuli is a matter of debate. In terms of lean mass, females receiving hormonal replacement therapy do not differ significantly from their untreated counterparts, even in light of these suggested effects (Javed et al., 2019). It is crucial to remember that lean mass does not necessarily equal muscle mass; rather, it is the total of all mass that is

free of fat and bone. In human skeletal muscle, progestogen-specific receptors are also expressed (Ekenros et al., 2017). However, progesterone may counteract the effects of estrogen by competing with estrogen for receptors and inhibiting its action, according to certain theories (Colenso-Semple et al., 2023). Recent research indicates that progesterone levels in the blood may enhance protein catabolism (Oosthuyse & Bosch, 2010). As mentioned earlier, one of the types of OCs contains only synthetic progesterone. When women who took OCs with combined hormones are compared with women who took pills with only progesterone, females utilizing progestogen-only OC showed greater grip strength and more muscle mass than OC combination users (Suuronen et al., 2019).

The effect of the tablets is still unclear. Primarily the type of tablet leads to certain differences. Antiandrogenic and androgenic contraceptive pills have been studied in relation to their effects on strength training (Ruzić et al., 2003). After a 16-week period of pill use, antiandrogens were found to reduce the benefits of strength training in women. However, there was no control group present in this study (which does not take OCP), so the interpretation of the results is questionable. The effect on myofibrillar protein synthesis also depends on the type of tablets (Hansen et al., 2011). When one version of OC (35 µg ethinyl estradiol and 0.25 mg norgestimate/day) was used, the researchers observed reduced levels of myofibrillar protein synthesis. However, when another formulation (30 µg ethinyl estradiol, and gestoden 0.0075 mg/day) was used, there was no effect on myofibrillar protein synthesis. These conflicting results may be explained by differences in the androgenic vs. antiandrogenic properties of the progestins used. Norgestimate has mild androgenic properties (A. Phillips et al., 1992), whereas gestodene is considered more potent and has different metabolic effects (Shoupe, 1994). Furthermore, variations in estrogen dose and individual hormonal responses may also contribute to divergent outcomes in protein synthesis and training adaptations.

Dalgaard et al. (2019) conducted research in which they tracked adaptations to strength training in OCP users and non-OC users. The trend toward a higher increase in muscle mass and a noticeably greater rise in type I muscle fibre area was associated with the use of OCPs. They proposed that OC use keeps estrogen levels constant, in contrast to endogenous estrogen levels, which change throughout the MC. This causes continuous stimulation of intramuscular estrogen receptors throughout the first 21 days of the OC cycle. This study implies that women on OC may have higher levels of estrogen, which favourably impact satellite cells and enhance anabolic stimulation. It has also been proven that the use of OCP increases the number of satellite cells per type II fibre and total fibre (Oxfeldt et al., 2020). Bernardes & Radomski (1998) compared the effect of OCP on growth hormone. The same 7 women went through the phase of taking and not taking pills. Growth hormone responses were shown to be significantly higher during the OC use period compared to the OC non-use phase. When estrogen levels peak during the MC, exercise induces greater growth hormone responses than during other phases of the cycle (Hornum et al., 1997). With the use of 4 different methods, it has been proven that OC use increased growth hormone response to acute heavy resistance exercise (W. J. Kraemer et al., 2008).

On the other hand, certain studies have shown that the use of contraceptive pills does not lead to significant results (Myllyaho et al., 2021; Romance et al., 2019; Sung et al., 2022). It has been reported that regardless of a woman's use of birth control pills, strength training causes hypertrophy parameters to increase. Furthermore, it has been noted that there are no positive effects of using this type of contraception, nor are there any negative effects on indices of body composition or strength. Also, without sufficient evidence, researchers frequently assume a symmetrical, repeating MC, which increases outcome heterogeneity (Van Every et al., 2024). Instead, researchers should aim for methodological consistency. Due to the inconsistency of findings and the diversity of study designs and OCP formulations, a summary of selected studies is presented in Table 1.

Table 1. Summary of Selected Studies Examining the Effects of Oral Contraceptives on Muscle Hypertrophy

Study	Sample	OCP Type	Intervention	Main Findings
Bernardes & Radomski (1998)	7 women	Combined OCP	Continuous and intermittent exercise protocols	Grester growth response with OCP use
Ruzić et al. (2003)	50 women	"Antiandrogen" group (n=29) used contraceptive pills containing antiandrogens and estrogens, "Estrogen-progestogen", group (n=31) used standard estrogens-progestogen pill	16 weeks of strength training	Antiandrogens appeared to reduce strength training benefits in women, but the lack of a non-OCP control group limits the interpretation of these findings
Hansen et al. (2011)	11 OC users and 12 non-OC users	Third-generation combined OCs	One-legged kicking exercise; Protein synthesis measured post-exercise	The use of oral contraceptives appears to suppress myofibrillar protein synthesis, and the degree of this inhibition may vary based on the specific formulation of the contraceptive
Dalgaard et al. (2019)	14 regular OC users and 14 non-OC users	Third-generation OCs	10 weeks of resistance training	Greater increase in Type I fiber area in OCP users
Romance et al. (2019)	12 OC users and 11 non-OC users	Second-generation monophasic combined OCs and third-generation triphasic OCs	8-weeks of non-linear resistance-training program	No between-group differences in strength or hypertrophy outcomes
Suuronen et al. (2019)	400 women	Combined OCP vs progesterone-only OCs	Cross-sectional	Progesterone-only users had greater grip strength and more muscle mass than OC combination users
Oxfeldt et al. (2020)	20 OC users and 18 non-OC users	Second-generation monophasic combined OCs	10-weeks of supervised progressive resistance training program	Increased satellite cell number in OCP users
Myllyaho et al. (2021)	9 OC users and 9 non-OC users	Monophasic combined OCs and progesterone-only OCs	10-weeks of high-intensity combined strength and endurance training	No between-group differences in strength or hypertrophy outcomes
Sung et al. (2022)	34 OC users and 40 non-OC users	Second-generation monophasic combined OCs	12 weeks of submaximal strength training	Skeletal muscle adaptations to exercise were comparable between women using oral contraceptives and those who were not, with both groups demonstrating similar responsiveness to strength training

Conclusion

This review has synthesized the current understanding of how OCPs may influence skeletal muscle hypertrophy in response to resistance training. The central theme emerging from the existing literature is one of considerable ambiguity and conflicting evidence. While resistance exercise remains the undisputed primary driver of muscle growth, the potential for OCPs to modulate this adaptive process by altering the endogenous hormonal milieu is a critical area of investigation for both athletic and general female populations.

The disparate finding, ranging from suggestions of attenuated gains with anti-androgenic progestins to potentially enhanced anabolic signaling and satellite cell activation, can be largely attributed to significant methodological heterogeneity across studies. A primary confounding factor is the wide variety of OCP formulations used in research, which differ in the type, dose, and androgenicity of their synthetic progestins and estrogens. As highlighted, different progestins can exert varied effects, yet many studies fail to distinguish between them, grouping all OCP users into a single category. This oversimplification likely masks formulation-specific effects on muscle protein synthesis, catabolism, and overall hypertrophic potential.

Furthermore, a critical limitation in the current body of work is the inconsistent and often inadequate verification of menstrual cycle phases in non-OCP-using control groups. The natural fluctuations of endogenous estrogen and progesterone have well-documented physiological effects, including on protein metabolism and anabolic signaling. Without robust hormonal verification, comparisons between OCP users and naturally cycling women are fundamentally flawed, making it difficult to isolate the true effect of the OCPs themselves.

In conclusion, the question of whether OCPs help, hinder, or have no significant effect on resistance training-induced skeletal muscle hypertrophy remains unanswered. The current body of evidence shows contrasting results, which limits any definitive clinical or practical recommendations. The observed discrepancies are not necessarily indicative of a lack of effect, but rather a reflection of the complex interplay between different OCP formulations and individual physiological responses, compounded by a lack of methodological standardization.

Practical implications and recommendations

Monitoring the hormonal profile of female athletes using OCPs may support better training personalization and performance management. For women aiming to optimize hypertrophy outcomes, considering the type of OCP and its androgenic or antiandrogenic properties may be crucial. The possible impact of various hormonal contraceptive methods (implants, injections, intrauterine devices), which produce various hormonal profiles, on adaptations to resistance training, should also be investigated in the future. Future studies might also examine whetherif a person's training state could mitigate the effects of using OCs on the adaptations that come with exercise training. To improve clarity and consistency in future findings, researchers should aim to standardize the classification of menstrual cycle phases and OCP use stages, precisely match participants by hormonal profile and training status, and report detailed training variables such as volume, intensity, and frequency.

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