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The Impact of Menstrual Cycle Phase and Hormonal Contraception on the Risk of Anterior Cruciate Ligament (ACL) Injury and on Physical Performance and Motor Abilities in Women — A Narrative Review

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Abstract

Background: Female athletes sustain anterior cruciate ligament (ACL) injuries two to eight times more often than males in comparable sports, and cyclical fluctuations of ovarian hormones are among the most frequently invoked female-specific explanations. The same hormones are also assumed to shape endurance, strength and motor abilities across the cycle.

Aim: To synthesise and critically appraise evidence from the last decades on how menstrual cycle phase and hormonal contraceptive (HC) use influence ACL injury risk, knee joint laxity and neuromuscular control, and on how they influence aerobic capacity, strength, power and motor abilities in women.

Methods: A narrative review of peer-reviewed original studies, systematic reviews and meta-analyses identified in PubMed, Scopus, Web of Science and Google Scholar was performed.

Results: ACL injury risk is highest in the pre-ovulatory and ovulatory phases, when peak oestrogen coincides with increased anterior knee laxity and general joint laxity, whereas the luteal phase appears least associated with injury. Evidence on laxity is nevertheless inconsistent, and increased passive laxity does not translate uniformly into altered biomechanics. Oral contraceptives may modestly lower ACL injury risk in case-control data, but rigorous cohort designs suggest this reflects bias rather than a true protective effect. For performance, group-level effects of cycle phase on strength, power and endurance are trivial and inconsistent, although perceived performance and symptom burden fluctuate markedly. HC use exerts, at most, trivial group-level effects on performance.

Conclusions: Cycle phase and HC modify passive stability and perceived function more than objective performance. Prevention and training should rely on individualised menstrual

tracking, neuromuscular training and multidimensional screening rather than uniform phase-based protocols or routine contraceptive prescription.

Keywords: menstrual cycle; hormonal contraception; anterior cruciate ligament; knee laxity; athletic performance; female athletes; oestrogen; neuromuscular control

1. Introduction

Women's participation in competitive and recreational sport has expanded rapidly over the past decades, and with it the clinical and scientific attention devoted to female-specific physiology [1,2]. Two questions dominate this field. The first is why female athletes rupture the anterior cruciate ligament (ACL) far more frequently than males, and whether the cyclical hormonal environment of the menstrual cycle contributes to this disparity [2,3]. The second is whether the same hormonal fluctuations meaningfully alter endurance, strength, power and other motor abilities, such that training and competition might be periodised around the cycle [4,5]. Both questions carry direct consequences for injury prevention, athlete health and the design of evidence-based training, yet the literature remains fragmented and, in places, openly contradictory.

ACL injury is one of the most common and most disabling musculoskeletal injuries in athletes, occurring particularly in sports involving sudden deceleration, cutting, pivoting and jumping such as football, basketball, handball and skiing [1]. Its consequences extend well beyond the acute phase: ACL rupture is frequently accompanied by meniscal and chondral damage, is associated with prolonged absence from sport, incomplete return to the pre-injury level, and a substantially elevated long-term risk of post-traumatic osteoarthritis [1]. The etiology is multifactorial, arising from the interaction of anatomical, neuromuscular, biomechanical, hormonal and genetic influences [1,2]. Among these, sex-related differences are one of the most consistent findings, with female athletes showing a two- to eight-fold, and by some estimates up to ten-fold, higher risk than males participating in the same sports at the same level [2,3]. Because the majority of ruptures occur through non-contact mechanisms during sport-specific movements, attention has increasingly shifted toward modifiable contributors such as landing mechanics, dynamic knee valgus and neuromuscular control, but the persistent sex disparity has kept the potential role of ovarian hormones firmly on the research agenda [1,2].

The menstrual cycle plays a fundamental role in female physiology, influencing metabolism, muscle recovery, thermoregulation, cardiovascular function, neuromuscular control and psychological well-being through fluctuating concentrations of oestrogen and progesterone [4]. Because oestrogen, progesterone and relaxin receptors are expressed in the ACL, in synovial fibroblasts, in skeletal muscle, tendon and fascia, the ligament and the neuromuscular system are direct hormonal targets, providing a biological rationale for cyclical variation in both injury susceptibility and performance [3,6]. Hormonal contraceptives, used by a large and in some populations majority proportion of female athletes, suppress endogenous fluctuations and stabilise ovarian hormones at low levels, offering a natural experiment for testing whether removing hormonal peaks alters injury risk or performance [6,7].

The scientific and social context sharpens the urgency of these questions. Female athletes have historically been under-represented in sport and exercise science, in part because the cyclical variation of their hormones has been regarded as an inconvenient source of experimental noise to be avoided rather than a physiological phenomenon to be studied; the practical consequence is that much of what is assumed about training and injury prevention derives from male-based research of uncertain applicability to women [3,4]. At the same time, the topic has acquired a high public profile, with menstrual-cycle "phase-based training" widely promoted in the media and on social platforms, frequently outpacing the evidence [3,4]. A dispassionate, medically

grounded synthesis that distinguishes what is genuinely supported from what is merely plausible or popular is therefore timely, and that is the purpose of the present work.

Despite an expanding evidence base, unified prevention guidelines and clear performance recommendations are lacking, largely because findings regarding the exact impact of hormonal phases remain inconsistent across studies, and because methodological quality — in particular the verification of cycle phase — is frequently inadequate [3,8]. The aim of the present narrative review is therefore to synthesise and critically appraise the medical evidence on two intertwined themes: (i) the influence of menstrual cycle phase and hormonal contraception on ACL injury risk, knee joint laxity and neuromuscular injury-risk surrogates; and (ii) their influence on aerobic capacity, strength, power and motor abilities. Throughout, particular emphasis is placed on comparing conflicting findings, on the methodological factors that generate them, and on the practical translation of a genuinely complex and multidimensional body of evidence.

2. Methodology

This work is a narrative review synthesising peer-reviewed literature on the menstrual cycle, hormonal contraception, ACL injury and female athletic performance. Consistent with the approach adopted by the primary and secondary sources examined here, relevant articles were identified through the electronic databases PubMed, Scopus, Web of Science and Google Scholar, using combinations of keywords and Medical Subject Headings related to "menstrual cycle", "hormonal fluctuations", "female hormones", "oestrogen", "progesterone", "relaxin", "hormonal contraceptives", "oral contraceptives", combined with "anterior cruciate ligament", "ACL injury", "knee laxity", "joint laxity", "injury risk", and with "athletic performance", "exercise performance", "endurance", "aerobic capacity", "strength", "muscle power", "neuromuscular function", "perceived exertion" and "female athletes" [1,4].

Priority was given to original research articles, systematic reviews and meta-analyses, supplemented by foundational studies of continuing relevance. Because narrative reviews do not apply the formal risk-of-bias appraisal characteristic of systematic reviews, findings were interpreted qualitatively, with explicit attention to methodological rigour — notably the method of menstrual cycle phase verification, sample size, participant training status and the presence of conflicting evidence [1,8]. As this is a review of previously published work involving no direct participation of human subjects, ethical approval was not required; nevertheless, all referenced studies were understood to have adhered to the ethical guidelines documented in the original publications [1]. Recognised limitations of this methodological approach include variability in cycle-tracking methods across studies, individual differences in hormonal responses not captured by group-level analysis, and the predominance of research on recreationally trained rather than elite populations [1].

3. Physiological Background: The Menstrual Cycle, Ovarian Hormones and Hormonal Contraception

3.1. The menstrual cycle and its hormonal profile

The menstrual cycle is a periodic, hormonally regulated process governed by feedback within the hypothalamic–pituitary–ovarian axis, lasting on average 28 days but ranging normally between approximately 21 and 35 days [4,9]. It is conventionally divided into a follicular phase, which begins on the first day of menstruation and lasts until ovulation, and a luteal phase, which extends from ovulation to the onset of the next menses; ovulation itself is a brief mid-cycle event [4]. Some authors further subdivide the cycle into four phases — menstrual, follicular, ovulatory and luteal — or into as many as six sub-phases distinguished by characteristic hormone concentrations [4,10].

The two ovarian steroids of principal interest are oestradiol (the most potent endogenous oestrogen) and progesterone [6]. Both are at their lowest during the early follicular phase, at the onset of menstruation [3]. Oestradiol then rises through the follicular phase to reach a first, marked peak in the late follicular phase, immediately before ovulation, followed by the surge of luteinising hormone (LH) that triggers ovulation; a second, lower oestradiol rise occurs in the luteal phase [3]. Progesterone begins to increase in the late follicular phase just before ovulation, but its highest concentrations are reached in the mid-luteal phase; a decline in both progesterone and oestradiol at the end of the luteal phase precipitates menstruation and restarts the cycle [3]. This means that the three most hormonally distinct testing windows are the early follicular phase (low oestradiol and progesterone), the late follicular phase (high oestradiol, low progesterone) and the mid-luteal phase (elevated oestradiol and progesterone) [11]. A third hormone, relaxin, a peptide of ovarian and placental origin that contributes to laxity of the pubic symphysis in pregnancy, is also detectable in non-pregnant women during the follicular and luteal phases and has been implicated in ligamentous laxity [3].

3.2. Hormone receptors in musculoskeletal tissue

The biological plausibility of hormonal effects on injury and performance rests on the widespread distribution of sex-hormone receptors. Oestrogen and progesterone receptors have been identified in the human ACL, in synovial fibroblasts and chondrocytes, in fascia, in tendon and in skeletal muscle, indicating that these tissues are direct hormonal targets rather than passive structures [3,6]. Laboratory work demonstrates that exposure of the ACL to oestradiol produces a dose-dependent reduction in fibroblast proliferation and type I collagen synthesis, an effect attenuated by the addition of progestins, whereas progesterone is associated with increased fibroblast proliferation and collagen synthesis [3]. Oestrogen also decreases the activity of lysyl oxidase, the enzyme responsible for collagen cross-linking; engineered ligament tissue exposed to oestrogen shows no change in collagen content but a significant fall in lysyl oxidase and a corresponding decline in mechanical stiffness [3]. Relaxin, in turn, promotes matrix remodelling through activation of matrix metalloproteinases that degrade collagen [3]. Through these mechanisms, the hormonal environment can, in principle, modulate both the passive mechanical properties of the ligament and, via receptors in muscle and the central nervous system, the active neuromuscular control that protects the joint [3,6].

3.3. Hormonal contraception

Hormonal contraceptives (HCs) comprise combined oral contraceptive pills (OCPs) containing synthetic oestrogen (usually ethinylestradiol) and a progestin, progestin-only oral pills, intrauterine systems, implants, injections and vaginal rings [6,9]. Combined OCPs act by supplying exogenous oestrogen and progestin that suppress gonadotropin-releasing hormone and, consequently, the secretion of follicle-stimulating hormone (FSH) and LH, inhibiting follicular development and ovulation and holding endogenous oestradiol and progesterone at low, relatively stable concentrations [9]. A conventional monophasic regimen consists of 21 days of active-pill consumption followed by 7 days of withdrawal, during which withdrawal bleeding occurs [12]. The resulting hormonal milieu — chronically suppressed endogenous ovarian hormones with superimposed daily exogenous steroids — differs fundamentally from that of a naturally menstruating (eumenorrheic) woman, which is precisely why HC users are frequently studied both as a group of interest and as a "control" for the cyclical hormone fluctuations of the natural cycle [6,7]. HCs are used not only for contraception but also to treat dysmenorrhoea, to regulate irregular cycles and to manipulate or postpone menstrual bleeding around competition, and their prevalence among athletes has been reported at 40–68% depending on population [7,13]. Because different formulations deliver different doses and

generations of progestin, their physiological and potential performance effects cannot be assumed to be uniform [14,13].

3.4. Systemic physiological effects of ovarian hormones relevant to performance

Beyond their action on ligament and muscle tissue, oestradiol and progesterone exert wide-ranging systemic effects that provide the physiological rationale for cyclical variation in performance. Oestradiol has broadly anabolic and favourable actions: it possesses anti-inflammatory and antioxidant properties that may reduce exercise-induced muscle damage and oxidative stress, enhances neuromuscular efficiency through improved motor-unit recruitment and muscle contractility, promotes vasodilation and mitochondrial efficiency to support oxygen delivery and fat oxidation, and improves the intrinsic contractile quality of skeletal muscle by facilitating actin–myosin interaction rather than by increasing muscle size [4,5]. It also lowers core body temperature and enhances the sweat response, which may aid heat tolerance in endurance sport during the follicular phase [4]. Progesterone, dominant in the luteal phase, tends to act in the opposite direction: it raises the thermoregulatory set-point and core temperature by 0.3–0.5 °C, increases resting heart rate and ventilation through central chemoreceptor sensitisation, is associated with fluid retention and slowed digestion, and has been linked to protein catabolism and to sedative effects on the central nervous system that may reduce central drive [4,5].

These opposing profiles generate the intuitive but only partially supported expectation that the high-oestrogen follicular and ovulatory phases should favour strength, power and neuromuscular efficiency, while the high-progesterone luteal phase should impose cardiovascular and thermoregulatory strain and greater perceived effort but may confer fatigue resistance for prolonged low-intensity work [4,5]. As later sections show, the objective performance data only weakly bear out these predictions, and the discrepancy between mechanistically plausible expectation and empirically observed triviality is one of the defining features of this literature. The systemic reach of the ovarian hormones also explains why the cycle may influence cognition, mood, sleep and thermoregulation — domains in which effects appear more consistent than in maximal strength or aerobic power, precisely because they depend on central and autonomic actions of the hormones rather than on the contractile apparatus alone [4,9].

4. ACL Injury in the Female Athlete: Epidemiology and the Sex Disparity

4.1. Magnitude of the sex difference and its multifactorial basis

ACL injury is consistently more frequent in female than in male athletes, with reported ratios ranging from two- to eight-fold and, in some sport-specific analyses, up to ten-fold [2,3]. The annual incidence of ACL injury in professional athletes has been reported at 0.21–3.67%, compared with roughly 0.03% in the general population, and the absolute number of injuries continues to rise as female sport participation grows [2]. This female predominance is best understood as multifactorial, reflecting the combined contribution of anatomical, developmental, neuromuscular, biomechanical and hormonal factors rather than any single cause [2,3].

Anatomically, female athletes more often present with a narrower intercondylar notch — associated with a smaller ACL of inferior structural properties — a steeper posterior tibial slope, a wider quadriceps angle (Q-angle) secondary to a broader pelvis, and greater generalised joint laxity, all of which alter the transmission of forces across the knee and increase strain on the ligament during cutting, landing and deceleration [2,15]. A larger Q-angle, particularly above approximately 19°, generalised joint laxity and knee hyperextension have each been linked to elevated ACL injury risk [2]. Uhorchak and colleagues, cited in the classic keynote summary

by Beynnon and Shultz, prospectively demonstrated that a narrower femoral notch combined with greater generalised joint laxity conferred a nearly eight-fold increased risk in men, while in women a narrower notch, a higher body mass index and general joint laxity predicted injury with high specificity and sensitivity; knee laxity values more than one standard deviation above the mean were associated with a 2.7-fold higher risk in women, with no equivalent relationship in men, suggesting that at least some anatomical risk factors are sex-specific [15].

4.2. Biomechanical and neuromuscular contributors

The predominance of non-contact injury — approximately 70–80% of all ACL ruptures — has directed attention toward modifiable biomechanical and neuromuscular deficits [1]. High-risk movements involve multiplanar loading combining dynamic knee valgus (medial collapse), limited knee and hip flexion, internal tibial rotation and strong quadriceps activation without sufficient hamstring co-contraction [1]. Female athletes tend to land with a stiffer, more extended posture, greater knee abduction and higher ground reaction forces, and to preferentially activate the quadriceps, which increases anterior tibial translation and strain on the ACL [2]. The hamstrings act as agonists to the ACL by resisting anterior tibial translation; any delay in their activation relative to quadriceps contraction — and the latency between foot contact and hamstring activation can approach or exceed the interval to ACL disruption — reduces the dynamic protection of the ligament [2]. Bencke and colleagues emphasise in their narrative review that neuromuscular control during such risk movements, and specifically the muscle activation patterns of the medial hamstrings and other stabilisers, has received far less attention than external loading factors, yet is a decisive and at least partly modifiable determinant of injury risk in young female athletes [16].

The prospective biomechanical evidence for these mechanisms is substantial. Hewett and colleagues, cited by Mancino and colleagues, prospectively studied knee kinematics during a vertical drop jump in 205 female athletes and found that increased knee abduction, higher ground reaction forces and shorter stance times distinguished those who subsequently sustained ACL injury, while Krosshaug and colleagues reported that medial knee displacement during the contact phase was associated with up to a 40% increase in injury risk in elite handball and football players [2]. Additional biomechanical risk factors identified across this literature include dynamic knee valgus, weak hip-abductor musculature, delayed activation of the vastus medialis, increased femoral anteversion and tibial torsion, poor trunk and core control with lateral trunk displacement, and a wider pelvis producing a greater Q-angle [2]. Kamitani and colleagues showed that fatigue in elite female footballers was associated with an inability to sustain a stable landing posture and with decreased hip flexion and ankle dorsiflexion, whereas repetition and training improved postural stability and impact absorption in single-leg landing, reinforcing the modifiability of these deficits [2]. Together these findings locate a large share of ACL risk in neuromuscular and postural control that can be trained, which is central to the prevention argument developed later.

Maturation adds a further dimension. In a systematic review of sixteen studies encompassing 2,323 female athletes, Ramachandran and colleagues found strong evidence that post-pubertal girls display higher peak knee abduction angles, higher external knee abduction moments and higher internal rotation moments, together with lower relative peak vertical ground reaction force, during jump-landing tasks than pre-pubertal girls — a constellation consistent with the well-documented rise in ACL injury rates after puberty, and coinciding developmentally with the establishment of cyclical hormonal function [17]. Fatigue likewise degrades protective mechanics: in a systematic review and meta-analysis of sex differences, Liu and colleagues reported that fatigue alters lower-limb landing biomechanics, with women showing patterns — reduced knee and hip flexion, greater dynamic valgus and slower responses — that increase

ACL loading and may interact with hormonal and other intrinsic factors [18]. These findings frame the hormonal question appropriately: cyclical ovarian hormones operate not in isolation but against a background of anatomical predisposition, faulty movement patterns and fatigue.

4.3. Consequences, reconstruction and return to sport

The clinical importance of the sex disparity is amplified by the consequences of ACL rupture, which are more severe for women in several respects. For athletes wishing to return to pivoting or high-demand sport, ACL reconstruction remains the standard treatment, with the goals of restoring mechanical stability, reducing the risk of further intra-articular damage and enabling progression through rehabilitation toward safe return [1]. Yet reconstruction alone does not guarantee a good outcome: rehabilitation quality, adherence and the criteria used for return to sport are decisive, and a criteria-based rather than purely time-based return is increasingly recommended, since up to 20–30% of younger and highly active athletes sustain a second ACL injury in either knee, and only approximately 60–70% return to their pre-injury level [1]. Women fare worse than men on these outcomes: female athletes have been reported to have lower return-to-sport rates (for example 65% versus 75% in one large series), an approximately 2.3-fold higher risk of graft re-rupture with hamstring autografts, smaller-diameter hamstring grafts that may predispose to failure, and a fourfold higher risk of a second ACL injury than men [2].

These downstream disparities are relevant to the hormonal question in two ways. First, they raise the stakes of primary prevention, making even a modest, potentially modifiable hormonal or neuromuscular contribution worth understanding. Second, they interact with the cyclical physiology discussed later: women who have undergone reconstruction retain elevated relaxin and greater dynamic knee valgus, and the reconstructed graft responds to the cycle differently from native tissue, so that the hormonal environment may bear not only on first injury but on re-injury and on the long-term trajectory toward post-traumatic osteoarthritis [1,2]. Contemporary reconstruction practice has evolved toward anatomical techniques, selective use of lateral extra-articular tenodesis to reduce rotational instability and graft failure in high-risk athletes, and structured, criterion-based rehabilitation integrating psychological readiness, all of which aim to narrow these sex-based outcome gaps [2,19]. None of these surgical or rehabilitative advances, however, addresses the upstream question of why the female ACL is injured so much more often in the first place — the question to which the remainder of this review is devoted.

5. Menstrual Cycle Phase and ACL Injury Risk

5.1. Evidence for elevated risk in the pre-ovulatory and ovulatory phases

A recurring conclusion across the literature is that the likelihood of ACL injury is not constant across the cycle, being greater during the pre-ovulatory (late follicular) and ovulatory phases than during the post-ovulatory (luteal) phase [15]. Beynnon and Shultz summarised a consensus emerging from early epidemiological work: Wojtys and colleagues, using self-reported menstrual history and later urinary metabolites of oestrogen, progesterone and LH, demonstrated a greater incidence of non-contact ACL injury during the pre-ovulatory phase, with injuries concentrated on days 9–14 of a 28-day cycle and fewer in the post-ovulatory phase [15]. Arendt and colleagues and Slauterbeck and colleagues similarly reported a disproportionate concentration of injuries in the pre-ovulatory phase, and a case–control study of recreational alpine skiers using serum progesterone and oestradiol found skiers in the pre-ovulatory phase far more likely to tear their ACL than those in the post-ovulatory phase, with an odds ratio of 3.22 [15]. The striking numerical concordance between studies — approximately 72–74% of non-contracepting injured women being in the pre-ovulatory phase

— reinforced the impression of a genuine cyclical pattern, although Myklebust and colleagues, studying European handball players, instead found increased risk in the week before or just after the onset of menstruation, an early signal of the heterogeneity that persists to this day [15].

The individual studies underpinning this pattern illustrate both its consistency and its imprecision. In Beynnon and colleagues' case–control study of recreational alpine skiers, characterised by urine and serum assays, 74% of injured women were in the pre-ovulatory phase and 26% in the post-ovulatory phase, mirroring Wojtys and colleagues' report that, among women not using oral contraception, 72.5% and 27.5% of injuries occurred in the pre-ovulatory and post-ovulatory phases respectively [6,15]. When the cycle was divided into three phases, Adachi and colleagues found 72% of teenage non-contact ACL injuries in the ovulatory phase (against 11% follicular and 17% luteal), and Wojtys and colleagues likewise located the majority of injuries in the ovulatory window, whereas Arendt and colleagues, using athletic-trainer reports, instead found the highest proportion (44.8%) in the follicular phase, 17.2% in the ovulatory and 37.9% in the luteal phase [6]. This scatter — ovulatory in some studies, follicular in others, but rarely luteal — is precisely why the pooled conclusion emphasises the luteal phase as the least dangerous rather than pinpointing a single peak-risk day [6].

The systematic review and meta-analysis by Herzberg and colleagues, which pooled 21 studies and 68,758 participants, provided the most comprehensive appraisal of this question [6]. Of five studies of women not using hormonal contraception, four indicated that the luteal phase was the phase least associated with ACL injury; when the cycle was divided into three phases, two studies located the highest incidence in the ovulatory phase and one in the follicular phase [6]. Taken together, these studies suggested that the lowest-risk window is the luteal phase [6]. The authors advanced two candidate mechanisms: an unopposed rise in oestradiol during the pre-ovulatory phase — high oestradiol with relatively low progesterone — acting directly on the ACL, and an indirect effect whereby hormones modulate the sequence and magnitude of muscle contraction and thus the dynamic stiffness of the knee [6,15]. Notably, in the alpine-skier data, serum progesterone tended to be elevated by a mean of 70% in uninjured controls relative to injured skiers, hinting that progesterone may be relatively protective, consistent with its collagen-promoting and oestrogen-antagonising actions in vitro [15]. This luteal-phase "safety" is echoed in the review by Woźniakowska and colleagues, which concluded that the late follicular and ovulatory phases represent the period of highest ACL injury risk because passive joint stability and active motor control deteriorate simultaneously [3]. Narrative and comprehensive reviews on performance also acknowledge that increased joint laxity and collagen turnover around ovulation heighten susceptibility to injury in high-impact sports [4,5].

5.2. Conflicting evidence from prospective team-sport surveillance

Against this relatively coherent picture from case–control and case-series studies stands a body of prospective injury-surveillance data in team sport that is markedly less consistent, and in several instances points to the luteal phase or the early follicular phase rather than the ovulatory phase. Fort-Vanmeerhaeghe and colleagues prospectively tracked 59 elite adolescent team-sport athletes (basketball, volleyball, handball) across a season and found that, although no significant association emerged when injuries were distributed across four phases, a dichotomous follicular-versus-luteal comparison was significant: 78.4% of injuries, and specifically 88.4% of joint and ligament injuries and 71.4% of muscle and tendon injuries, occurred during the luteal phase, which was also characterised by poorer sleep and greater fatigue [20]. This directly contradicts the ovulatory-risk model and instead aligns with reports by Barlow and colleagues in elite footballers, cited within several of the reviewed sources, of elevated luteal-phase injury risk [21,20].

Ferrer and colleagues conducted one of the largest and methodologically most careful surveillance studies, following 33 elite female footballers over four seasons and 852 menstrual cycles, during which 80 injuries were recorded [21]. Crucially, they argued that without hormonal confirmation only the bleeding (early follicular) phase can be reliably identified, and therefore compared bleeding with non-bleeding days [21]. Injury incidence did not differ significantly between bleeding (5.36 per 1,000 h) and non-bleeding phases (6.48 per 1,000 h), but the injury burden — days lost per 1,000 h — was more than three times higher during bleeding (684 versus 206), driven in part by two of four cruciate-ligament injuries occurring during menstruation [21]. The authors interpreted this as evidence that menstruation may not increase the frequency of injury but may be associated with more severe injuries, and they explicitly cautioned that the low circulating oestrogen of the early follicular phase, together with non-hormonal factors such as iron loss, fatigue, symptom severity, nutrition and recovery, could all contribute [21]. Reports collated in the surveillance literature describe yet another pattern, with muscle and tendon injuries nearly twice as frequent in the late follicular phase [20]. The scoping review by MacMillan and colleagues, which screened 10,696 articles and included 96, captured the root of this disorder: definitions of injury, eumenorrhoea and menstrual irregularity differ vastly between studies, most research is confined to North America and Europe, and methodological and terminological heterogeneity fundamentally hinders comparative synthesis [22].

A further reason for caution is that injury is not one entity. The reviewed surveillance studies mix acute ligamentous injuries with muscle, tendon and overuse injuries, which may follow different hormonal logics. Ferrer and colleagues, for example, found that ligament injuries carried by far the greatest burden (a median of 29 days lost and 187 days per 1,000 h), that muscle injuries were the most frequent but least disabling, and that two of the four cruciate injuries in their cohort occurred during menstruation [21]. Fort-Vanmeerhaeghe and colleagues separately reported that joint and ligament injuries and muscle and tendon injuries were both concentrated in the luteal phase, whereas the surveillance data collated within their and other reports place muscle and tendon injuries preferentially in the late follicular phase [20]. When the specific outcome of interest is the ACL, the hormonally verified case–control literature retains its coherence around the pre-ovulatory and ovulatory phases; it is chiefly for the broader category of "lower-limb injury" that the luteal phase emerges, and largely, it appears, through the fatigue, sleep disruption and symptom burden that peak premenstrually rather than through a direct ligamentous effect [21,20,22].

Table 1 summarises the principal studies bearing on the relationship between menstrual cycle phase, hormonal contraception and ACL injury or knee laxity, together with their designs, phase-verification methods and main findings, to make the pattern of agreement and disagreement explicit.

Table 1. Principal studies on menstrual cycle phase, hormonal contraception, ACL injury and knee laxity.

Study [ref.]	Design	Population	Phase verification	Main finding
Beynnon & Shultz [15]	Keynote review of case–control/case-series	Female athletes, alpine skiers	Self-report, urine/serum hormones	Higher ACL injury risk pre-ovulatory vs post-ovulatory (OR 3.22 in skiers)
Herzberg et al. [6]	Systematic review + meta-analysis (21 studies, n = 68,758)	Women ± hormonal contraception	Varied (mostly self-report/hormones)	Luteal phase least associated with injury; ovulatory laxity +0.36 mm vs follicular; OCPs ~20% lower risk; evidence very low quality

Samuelson et al. [28]	Systematic review (7 studies)	Females of any age	Self-report/registry	Two high-quality case–control studies suggest OCP protection; injury-distribution data inconsistent
Herzog et al. [29]	Active-comparator new-user cohort vs replicated case–control	~3 million US women	Prescription/claims records	No difference OCP vs IUD (adjHR 0.95); case–control design reproduced spurious 10% protection
Shagawa et al. [23]	Repeated-measures experiment	15 university students	Basal temperature + ovulation kit + salivary E2	Anterior laxity/stiffness unchanged; genu recurvatum and general joint laxity higher at ovulation
Shafiei et al. [24]	Descriptive	40 female athletes	Serum hormones + LH kit	No difference in ACL laxity across three phases; no hormone–laxity relationship (manual testing)
Shagawa et al. [25]	Case series	9 women post-ACL reconstruction	Basal temperature + ovulation kit + salivary E2	Reconstructed-side laxity lower at ovulation than early follicular; side-to-side difference unchanged
Dos'Santos et al. [26]	Systematic review	Eumenorrheic/naturally menstruating women	Varied	Evidence limited/heterogeneous; no firm phase-dependent change in neuromuscular/biomechanical surrogates
Kirschbaum et al. [27]	Pilot experiment	10 elite team athletes	Three-step method	Cycle × urinary-incontinence interaction for single-leg sway; incontinence linked to higher hip adduction:abduction ratio
Kirschbaum et al. [31]	Cross-sectional	301 elite team athletes	Questionnaire	Menstrual dysfunction associated with lower ACL injury rate; regular examinations linked to more detected cartilage injury
Ferrer et al. [21]	4-season prospective cohort (852 cycles)	33 elite footballers	Calendar/bleeding vs non-bleeding	Injury incidence similar; burden >3× higher during bleeding (684 vs 206 days/1,000 h)
Fort-Vanmeerhaeghe et al. [20]	Prospective cohort (one season)	59 adolescent team athletes	Calendar app (4 phases)	78% of injuries in luteal phase; poorer sleep and greater fatigue luteally

5.3. Interpreting the discordance

Two considerations reconcile much of this apparent contradiction. First, the older case–control and case-series studies that support ovulatory-phase risk often relied on retrospective self-reported cycle timing at the moment of injury, whereas the prospective surveillance studies

track injuries in real time but frequently classify phase from calendar counting alone, without hormonal confirmation [21,22]. Both approaches are vulnerable to phase misclassification, but in different directions. Second, the outcome measured differs: some studies count injury incidence, others injury burden or severity, and Ferrer and colleagues showed that these can diverge, with menstruation associated with severity but not frequency [21]. The most defensible current synthesis is therefore that the pre-ovulatory and ovulatory phases are most consistently identified as the highest-risk window in hormonally verified studies of predominantly non-contracepting women [6,15], that the luteal and early follicular phases feature prominently in team-sport surveillance possibly through non-hormonal mediators such as fatigue, sleep, symptoms and iron status [21,20], and that the overall effect of cycle phase on injury, while biologically grounded, is neither large nor uniform enough to support rigid phase-based restriction of training or competition [22].

6. Sex Hormones and Knee Joint Laxity

6.1. The oestrogen–laxity hypothesis and its meta-analytic support

The mechanistic bridge most often proposed between cycle phase and ACL injury is anterior knee laxity. The classic noncontact injury mechanism combines change of direction, internal rotation and valgus, producing anterior tibial translation and a "pivot shift"; any increase in ligamentous laxity would allow greater tibiofemoral motion and, plausibly, greater injury risk [6]. Herzberg and colleagues performed the first quantitative meta-analysis of this relationship, drawing on six of twelve studies that provided suitable data, and found that knee laxity was significantly increased in the ovulatory phase compared with the follicular phase, with a mean difference of 0.36 mm (95% CI 0.08–0.65) [6]. Laxity did not differ significantly between luteal and ovulatory or between luteal and follicular phases [6]. Four of five studies with quantitative oestradiol and laxity data reported the highest ligament laxity when oestradiol concentration was highest, consistent with a contributory role for oestrogen [6]. The authors nonetheless graded the overall strength of evidence as very low, and explicitly noted that it remains unclear whether such small, millimetre-scale differences are clinically meaningful [6].

6.2. Inconsistency and the role of measurement

Direct experimental studies illustrate why the laxity hypothesis, although attractive, remains unsettled. Shagawa and colleagues measured oestradiol, anterior knee laxity, stiffness, genu recurvatum and general joint laxity in the late follicular and ovulatory phases of fifteen university students with regular cycles [23]. Oestradiol was significantly higher in the ovulatory phase, yet anterior knee laxity and stiffness did not differ between phases; instead, genu recurvatum and general joint laxity were significantly greater in the ovulatory phase [23]. The authors interpreted this to mean that rising oestradiol may affect knee hyperextension and generalised laxity more than the specifically anterior laxity usually measured, and pointedly demonstrated that a study measuring only anterior knee laxity would have wrongly concluded that ovulation does not compromise joint stability [23]. This methodological lesson — that the parameter chosen determines the conclusion — recurs throughout the field.

Shafiei and colleagues reached the opposite conclusion using different methods, finding no significant difference in ACL laxity across menstruation, ovulation and the mid-luteal phase in 40 female athletes, and no significant relationship between female-hormone concentrations and laxity [24]. They attributed the discrepancy with earlier positive studies to their reliance on the Lachman and anterior drawer tests performed by a knee surgeon, rather than instrumented arthrometry such as the KT-1000/2000 or radiographic measurement, arguing that manual clinical examination may be too insensitive to detect subtle, hormonally induced, millimetre-scale changes in tissue stiffness [24]. Several other studies cited within the reviewed corpus —

by Belanger, Hertel, Eiling and others — similarly found no cyclical variation in laxity, whereas Park, Shultz, Heitz, Deie and Lee reported significant increases around ovulation or in the early luteal phase [6,24]. The pattern of quantitative data assembled by Herzberg and colleagues is instructive: of six studies suitable for meta-analysis, the pooled ovulatory-versus-follicular difference of 0.36 mm was statistically significant, yet the absolute laxity values and their timing varied widely between laboratories, and four of five studies that reported both oestradiol and laxity found peak laxity when oestradiol peaked — but one (Hoffman and colleagues) found the highest laxity at day 22 in the late luteal phase, and one reported laxity values nearly double those of the others, underscoring the poor between-study comparability of arthrometric measurement [6]. Shultz and colleagues additionally showed that laxity may lag oestradiol concentration by three to four days, so that the timing of the laxity response and the hormonal peak need not coincide, which alone can explain why cross-sectional studies sampling on fixed calendar days reach conflicting conclusions [6]. Some authors have therefore proposed that only a subset of women — laxity "responders" — exhibit meaningful cyclical laxity change, while "non-responders" do not, so that a group-level mean can be simultaneously statistically significant and clinically irrelevant for most individuals [25].

The functional significance of these millimetre-scale changes is itself uncertain. Shagawa and colleagues noted that a change in genu recurvatum from 9° to 10° may matter, because ACL injury is more common in athletes with more than 10° of hyperextension, yet the clinical meaning of the roughly 1° difference they observed between phases had not been established [23]. Even where hormonally driven laxity is real, the classic case series by Wojtys and colleagues and by others reviewed by Herzberg found no consistent relationship between the phase of peak laxity and the phase of peak injury, weakening the assumption that laxity is the operative mechanism linking oestrogen to injury [6]. Relaxin adds further complexity: Dragoo and colleagues, as reported by Herzberg and by the ACL-focused reviews, found that athletes with serum relaxin above 6.0 pg/mL were four times more likely to sustain an ACL tear, and relaxin has been associated with decreased patellar tendon stiffness and with ACL injury; relaxin peaks in the luteal phase rather than at ovulation, adding a second, temporally distinct hormonal candidate whose risk window does not coincide with that of oestradiol [3,6].

6.3. Laxity after ACL reconstruction and the response paradox

An instructive complication comes from the reconstructed knee. Shagawa and colleagues, in a case series of eighteen knees in nine women at least one year after hamstring-autograft ACL reconstruction, found that anterior knee laxity on the reconstructed side was lower in the ovulatory than in the early follicular phase, whereas the contralateral (native) side showed no significant difference between phases [25]. The reconstructed ligament thus behaved differently — even oppositely — from the healthy contralateral knee across the cycle, and the side-to-side difference in laxity did not vary significantly with phase, implying that the menstrual cycle need not be controlled when the clinically standard side-to-side laxity measure is used [25]. This dissociation suggests that the reconstructed graft, which undergoes ligamentisation and remodelling for many months and possibly years after surgery, may respond to oestradiol differently from native tissue [25,19]. It also underscores an under-appreciated phenomenon reported elsewhere in the laxity literature: individual women appear to be "responders" or "non-responders" to cyclical hormonal influences on laxity, so that group means may obscure clinically important individual variation [25]. Post-reconstruction, women also retain elevated serum relaxin relative to uninjured peers and greater dynamic knee valgus, contributing to a persistent biomechanical mismatch between the operated and healthy limb that may help explain the high rate of second injury and early osteoarthritis in this population [3].

7. Neuromuscular and Biomechanical Injury-Risk Surrogates Across the Cycle

Because passive laxity alone incompletely explains injury, attention has turned to whether cycle phase alters the active neuromuscular and biomechanical surrogates of ACL injury risk — landing kinematics, joint moments, muscle activation and postural control. The systematic review by Dos'Santos and colleagues addressed exactly this, examining the effect of cycle phase on ACL neuromuscular and biomechanical injury-risk surrogates in eumenorrheic and naturally menstruating women [26]. Their synthesis emphasised that the evidence is limited, heterogeneous and hampered by inconsistent phase-verification methods, and that firm conclusions about phase-dependent alterations in high-risk movement patterns cannot yet be drawn — a measured position that contrasts with the more confident laxity-based narratives and reinforces the distinction between passive and active stability [26].

A small number of studies have examined muscle activation directly. Within the meta-analysis by Herzberg and colleagues, two studies used electromyography to assess quadriceps and hamstring activity across the cycle: one found no difference, while Khowailed and colleagues reported increased quadriceps activity in the follicular phase, most pronounced in the lateral quadriceps, and increased hamstring activity — with the medial hamstring most active before impact — in the ovulatory compared with the follicular phase, proposing that these differences produce knee malalignment before impact and may contribute to the increased ACL injuries observed in the ovulatory period [6]. This is a rare mechanistic bridge from hormone concentration to a neuromuscular pattern with plausible injurious consequences, but it rests on very small samples and has not been consistently replicated [6]. Bencke and colleagues, reviewing muscle activation during ACL-injury-risk movements more broadly, argued that the pre-activation and reflex activation patterns of the medial hamstrings are decisive for dynamic knee stability during cutting and landing, that these patterns are trainable, and that they have been comparatively neglected relative to external loading and passive-tissue factors — a neglect that is even more pronounced when it comes to how they might vary with the cycle [16]. The honest conclusion is that whether, and how much, cyclical hormones alter the active muscular protection of the knee remains among the least resolved questions in the field [6,16,26].

The pilot study by Kirschbaum and colleagues illustrates both the promise and the difficulty of this line of enquiry [27]. Studying ten elite female team-sport athletes across three hormonally verified cycle phases, they examined hip strength and static and dynamic postural control, additionally stratifying by urinary incontinence as a proxy for pelvic-floor and core function [27]. A significant interaction between cycle phase and urinary incontinence emerged for single-leg sway area with eyes closed, and incontinence was associated with a higher hip adduction-to-abduction strength ratio [27]. Such findings hint that cyclical hormonal effects on neuromuscular injury-risk surrogates may be real but small, may interact with other individual characteristics, and may be detectable only with rigorous phase verification and adequately sensitive measures [27]. Consistent with the broader message of this section, the qualitative and cognitive literature reviewed within several sources indicates that attentional, anticipatory and spatial cognition, as well as reaction time, fluctuate across the cycle — with reported deficits in the early follicular phase — which could plausibly influence the split-second decision-making that governs safe landing and cutting, though the evidence remains preliminary [4,5]. The overall conclusion of this domain mirrors that of the laxity literature: cyclical hormones may nudge neuromuscular control, but the effect is subtle, individually variable and not yet translatable into phase-specific injury-prevention prescriptions [26].

8. Hormonal Contraception and ACL Injury Risk

8.1. The protective-effect hypothesis

If cyclical oestrogen peaks increase laxity and injury risk, then suppressing those peaks with hormonal contraception should, in theory, be protective. Early and mid-sized studies lent some support to this idea. Herzberg and colleagues, reviewing seven studies of hormonal contraceptives and ACL injury, found that the two largest and highest-quality studies suggested that oral contraceptives may be protective, offering up to a roughly 20% reduction in injury risk [6]. The population-based case–control study from the Danish Knee Ligament Reconstruction Registry (4,497 cases, 8,858 controls) reported an 18% reduction in the risk of operatively treated ACL injury among OCP users (relative risk 0.82; 95% CI 0.75–0.90), and a comparable US commercial-insurance analysis by Gray and colleagues (12,819 cases) found a nearly identical adjusted odds ratio of 0.82 (95% CI 0.75–0.91), with the protective association concentrated in the youngest age group (15–19 years) [6].

Samuelson and colleagues, in a systematic review specifically devoted to OCPs and ACL injury, concurred that the two case–control studies of greatest methodological quality — those of Rahr-Wagner and of Gray — indicated that OCP use may reduce ACL injury risk, particularly the need for reconstruction in adolescents, while the remaining, lower-quality studies found no significant effect [28]. They stressed, however, that these findings are generalisable only to women who undergo reconstruction rather than to all ACL injuries, that important lifestyle confounders (athletic status, body mass index, smoking, ethnicity, injury mechanism) could not be controlled in registry and insurance data, and that studies of injury distribution across the cycle in users versus non-users were inconsistent and underpowered [28]. Mancino and colleagues similarly reported that OCP use was associated with a lower adjusted risk of ACL reconstruction in Danish registry data and, in one large study, with up to a 63% reduction in injury, while cautioning that most such evidence is retrospective, prone to confounding, and insufficient to justify prescribing OCPs as a protective measure [2].

8.2. The refutation from a rigorous cohort design

The most important corrective to the protective-effect narrative comes from Herzog and colleagues, who directly compared study designs in a single large database [29]. Using an active-comparator, new-user cohort design — comparing women newly initiating low-dose OCPs with women undergoing intrauterine device insertion, a group with a similar contraceptive indication but different treatment — they analysed 2,370,286 OCP initiators and 621,798 IUD users [29]. In this design, which synchronises the start of follow-up, avoids the inclusion of prevalent users and minimises confounding by indication, there was no difference in the risk of ACL injury between OCP initiators and IUD users (adjusted hazard ratio 0.95; 95% CI 0.89–1.01) [29]. When the same authors replicated the traditional "ever versus never" case–control design within the identical population, they reproduced the familiar slight protective association over five years (odds ratio 0.90; 95% CI 0.85–0.94) [29]. The clear implication is that the apparent protective effect of OCPs seen in earlier case–control studies is an artefact of uncontrolled confounding and selection — for example, women who choose contraception may differ systematically in activity level and health-seeking behaviour — rather than a genuine pharmacological effect [29]. Woźniakowska and colleagues reached the same conclusion, adding that OCPs address only passive laxity while leaving neuromuscular deficits unresolved, and that spinal excitability (H-reflex) and early-follicular strength do not differ between users and non-menstruating controls, so the body fails to neurologically compensate for any hormonally reduced stiffness [3].

8.3. The limits and paradoxes of contraceptive "protection"

Even setting aside the confounding problem, contraception is not a clean solution. Woźniakowska and colleagues drew attention to a potential detrimental effect: by chronically suppressing oestrogen and rendering the ACL stiffer, OCPs may impair the ligament's shock-absorbing capacity, and OCP users have been reported to show greater ligament damage and higher post-exercise muscle pain after heavy exercise [3]. The systematic review by Martínez-Fortuny and colleagues, and the scoping review by MacMillan and colleagues, both concluded that the influence of contraception on injury is an important but insufficiently studied question, and that relaxin, oestrogen and progesterone concentrations — and their pharmacological regulation — deserve dedicated investigation as possible protective or harmful factors [30,22]. Kirschbaum and colleagues, in a cross-sectional study of 301 elite female team athletes, produced a genuinely counter-intuitive result: menstrual dysfunction was associated with lower, not higher, ACL injury rates, an effect the authors attributed to possible reverse causality (reduced training load and improved energy availability after reconstruction restoring regular menstruation), to the acute traumatic nature of ACL injury contrasting with the overuse injuries typical of relative energy deficiency, and to behavioural factors such as ovulatory testosterone peaks and risk-taking in naturally menstruating athletes [31]. Taken together, the contemporary evidence does not support routine prescription of hormonal contraception as an ACL-injury-prevention strategy; any such decision requires a careful, individualised cost–benefit analysis that acknowledges systemic side effects extending well beyond the knee [3,29].

8.4. Menstrual dysfunction, relative energy deficiency and injury risk

The hormonal question cannot be confined to the phases of a regular cycle, because a substantial minority of athletes do not menstruate regularly at all. In elite team-sport athletes, menstrual dysfunction — encompassing oligomenorrhoea, primary and secondary amenorrhoea, and polymenorrhoea — is common: among non-contracepting athletes in the study by Kirschbaum and colleagues, 19% had a menstrual dysfunction, while in the large survey by Ekenros and colleagues 10% of non-users reported current secondary amenorrhoea and 6% oligomenorrhoea [31,13]. Menstrual dysfunction is a cardinal feature of relative energy deficiency in sport (RED-S), driven by low energy availability, and is associated through impaired physiological and psychological function with a broader spectrum of health and performance consequences, including a heightened risk of time-loss injuries and stress fractures [31,22]. Prolonged menstrual cycles and menstrual irregularity have been tied to greater injury frequency, and athletes with menstrual dysfunction have been reported to be more prone to time-loss injuries in the wider literature synthesised by the scoping review of MacMillan and colleagues [22].

Against this background, the counter-intuitive finding of Kirschbaum and colleagues — that menstrual dysfunction was associated with lower, not higher, ACL injury rates — is doubly instructive [31]. It cautions that RED-S-related injury risk is concentrated in overuse and bone-stress injuries that develop gradually under chronic energy deficiency, whereas the ACL typically fails in an acute, traumatic event, so that the two need not move together [31]. It also highlights the problem of temporal alignment in cross-sectional designs: injuries were recorded over the preceding year while menstrual status was assessed at a single later time point, raising the possibility of reverse causation whereby reduced training load and improved energy availability after an ACL reconstruction restored a regular cycle [31]. Iron status provides a further, frequently overlooked link between menstruation and injury: menstruating women lose approximately 1 mg of iron per day during bleeding, and iron deficiency has been associated with reduced training capacity and endurance and with impaired recovery, offering one plausible non-hormonal route by which the bleeding phase might raise injury burden, as observed by Ferrer and colleagues [21]. Menstrual dysfunction should therefore be regarded

not as a protective state but as a marker of underlying energy deficiency that warrants assessment in its own right, and its complex, sometimes paradoxical relationship with acute ligamentous injury is a further argument against reductive hormonal models of ACL risk [31,22].

9. Menstrual Cycle Phase and Physical Performance

9.1. Endurance and aerobic performance

The influence of cycle phase on endurance is genuinely contested. The systematic review and meta-analysis by McNulty and colleagues, cited across the corpus as the reference synthesis, concluded that exercise performance might be trivially reduced during the early follicular phase, when oestrogen and progesterone are lowest, relative to all other phases [4,8]. Consistent with this, endurance-trained women have reported poorer sleep quality and slower recovery after high-intensity training in the early follicular phase, suggesting that recovery dynamics rather than aerobic capacity per se may fluctuate [4]. Yet physiological measures such as maximal oxygen uptake and time to exhaustion show inconsistent patterns, and several comprehensive reviews report minimal differences in maximal oxygen uptake across phases [4,5].

Two carefully controlled experimental studies of endurance-trained women illustrate the modest and variable nature of these effects. Rael and colleagues examined the cardiorespiratory response to high-intensity interval running in the early follicular, late follicular and mid-luteal phases, using a rigorous three-step verification method [11]. Most variables — oxygen consumption, carbon dioxide production, respiratory exchange ratio, breathing frequency, energy expenditure and perceived exertion — were unaffected by phase, but ventilation and heart rate were significantly modified, with the authors concluding that sex-hormone fluctuations are generally insufficient to disrupt the tissue-level adjustments produced by high-intensity exercise, while cautioning that heart-rate-based training prescription should nonetheless account for phase [11]. The specific findings of these controlled studies are worth noting because they bound the magnitude of any true effect. In the study by Rael and colleagues, the ventilation and heart-rate differences that reached significance were small and, for ventilation, not always confirmed on pairwise comparison, and the authors attributed the residual heart-rate signal to the thermogenic action of luteal progesterone, which raises core temperature and prompts a redistribution of blood flow toward the skin that must be compensated by increased cardiac output [11]. Substrate metabolism, indexed by the respiratory exchange ratio and energy expenditure during the interval bouts, was unchanged across phases, leading the authors to conclude that training status, diet and substrate availability probably influence metabolism more than sex-hormone fluctuations in trained women [11]. Docter and colleagues, within the FENDURA project, evaluated running economy before and after typical low- and high-intensity training sessions across three verified phases and found that cycle phase did not confound the ability to maintain running economy, providing a further argument against phase-based exclusion of women from endurance research and against phase-specific endurance prescription [32]. They further found that whether economy is expressed as oxygen cost or energy cost slightly changed the result — the high-intensity session raised oxygen cost by 1.8% but left energy cost unchanged — a reminder that even the choice of outcome metric can determine whether a "phase effect" or a "session effect" is reported [32]. The comprehensive review by Rosińska-Lewandoska and colleagues offered a more phase-differentiated interpretation, arguing that higher follicular-phase oestrogen enhances carbohydrate utilisation and glycogen sparing to favour endurance, while elevated luteal progesterone promotes fat utilisation and fatigue resistance but may impair high-intensity performance dependent on rapid energy turnover, and that basal metabolic rate rises in the luteal

phase — a metabolic framing that yields plausible but as-yet-unproven periodisation hypotheses [5].

9.2. Strength and power

The strength and power literature has moved decisively toward a null or trivial group-level conclusion, but with important nuances. Blagrove and colleagues, in a meta-analysis of 21 studies reviewed within the umbrella synthesis, found only trivial effects of cycle phase on maximal voluntary contraction force, isokinetic peak torque and explosive strength, and concluded that strength-related performance is not meaningfully affected by phase [10]. McNulty and colleagues reached a similar conclusion, and Colenso-Semple and colleagues, in an umbrella review of systematic reviews and meta-analyses, found scant, low-quality and largely inconsistent evidence of any influence of cycle phase on strength, power or resistance-training adaptation [10]. Critically, when McNulty and colleagues weighted studies by quality, 90% of the moderate-to-high-quality studies found no difference in strength between phases, whereas most of the studies reporting significant differences were of low quality — a pattern that Colenso-Semple and colleagues attributed largely to inadequate cycle-phase verification and to the unfounded assumption of a standard 28-day cycle with mid-cycle ovulation [10].

Empirical studies of trained athletes support this null position while adding subtle qualifications. Dasa and colleagues studied maximal isometric grip strength, 20-m sprint, countermovement jump and leg-press power in 29 high-level team-sport athletes, verifying phase by serum hormones in the non-contracepting group, and found no statistically significant change across phases and no difference between naturally menstruating athletes and hormonal-contraceptive users, concluding that the cycle does not alter acute strength and power at the group level in high-level athletes [33]. Isenmann and colleagues, in a three-arm crossover of 24 female strength athletes with saliva-hormone and diary verification, found no significant effect of phase on a composite power-performance score, on jumping ability or on readiness; however, when athletes were stratified by performance level, higher-level athletes showed indications of reduced back-squat strength during menstruation, and well-being was significantly lower during menstruation than in the follicular or luteal phases [34]. This suggests that any genuine phase sensitivity of strength may be confined to a minority of highly trained athletes and to specific tasks, and may be mediated as much by well-being as by contractile function [34]. Reflecting the residual uncertainty, the meta-analysis by Niering and colleagues did report phase-dependent maxima — isometric strength peaking in the late follicular phase (weighted standardised mean difference 0.60), isokinetic strength during ovulation (0.39) and dynamic strength in the late follicular phase (0.14), with the early follicular phase least favourable for all strength types [35]. The contrast between Niering's phase-specific optima and the null conclusions of the umbrella-level syntheses exemplifies how differences in inclusion criteria, strength modality and phase verification generate divergent conclusions from an overlapping evidence base [10,35].

9.3. Energy metabolism and neuromuscular function

The proposed mechanisms underlying phase-related performance changes centre on energy metabolism and neuromuscular function. During the follicular phase, higher oestrogen is thought to enhance carbohydrate utilisation, insulin sensitivity and glycogen storage, favouring aerobic and high-intensity work, whereas the luteal phase, with elevated progesterone and reduced insulin sensitivity, shifts substrate use toward fat oxidation, potentially benefiting prolonged endurance but limiting glycogen availability for rapid, high-intensity efforts [5]. Neuromuscular fatigue has been reported to be more pronounced during the luteal phase in resistance-trained women, attributed to progesterone's effects on calcium handling in muscle and to a possible sedative effect on central drive, alongside slower recovery linked to increased

inflammation and reduced protein synthesis [5]. Cabre and colleagues, in a narrative review specifically of metabolic outcomes, strength and recovery, integrated these mechanisms while emphasising that the practical performance consequences are small and highly individual, and that nutritional and recovery strategies may be tailored to phase-specific metabolic tendencies without necessarily altering the training itself [7]. The systematic review by Elorduy-Terrado and colleagues, examining thirteen studies of eumenorrheic athletes, similarly reported that flexibility is enhanced around ovulation and that aerobic and anaerobic capacity may be better in the luteal phase, while maximal and explosive strength showed no consistent phase effect — again illustrating that motor abilities other than raw strength may be more phase-sensitive than strength itself [36].

9.4. Perceived performance, symptoms and psychological factors

Perhaps the most consistent cyclical phenomenon is not objective performance but its subjective perception. Ihalainen and colleagues, cited within the reviewed reviews, found that 56% of athletes reported a decline in perceived performance during menstruation due to bloating, cramps and mood swings, even when strength and endurance metrics showed trivial or no change, and that this perceptual effect was more pronounced in naturally menstruating athletes than in contraceptive users [4]. The large survey by Ekenros and colleagues, encompassing 1,086 athletes from 57 sports, quantified this burden directly [13]. Among non-contracepting athletes, 74% experienced dysmenorrhoea and 78% premenstrual symptoms; overall physical performance, muscle strength, aerobic fitness, mental sharpness, balance and sleep quality were perceived to be worst in the early follicular and late luteal phases and best around ovulation, and — crucially — the perceived negative impact in the early follicular phase was statistically linked to menstrual pain, while that in the late luteal phase was linked to premenstrual symptoms [13]. In other words, the perceived performance decrement is mediated by symptoms rather than being a direct hormonal effect on the muscle [13]. Despite this, only 18% of athletes considered the cycle when planning training or competition, and although 45% had at some point refrained from training and 14% from competition for menstrual reasons, most did not regard symptoms as a sufficient reason to alter training [13]. The qualitative study by Ryman Augustsson and colleagues enriched this picture: women described feeling strongest and most motivated in the late follicular phase around ovulation, a distinct "breaking point" of declining energy after ovulation, and the poorest, most effortful training during the late luteal phase, while emphasising marked individual variation and the importance of social support — leading the authors to reject general phase-based protocols in favour of individualised approaches [37].

9.5. Match performance and sport-specific outcomes in team sports

Laboratory tests of strength and power capture only part of what matters to athletes, and a smaller literature has therefore examined sport-specific and match-derived outcomes across the cycle, chiefly synthesised in the systematic review by Elorduy-Terrado and colleagues [36]. The picture here is as heterogeneous as elsewhere, but with some tendency toward better team-sport output in the luteal phase. In football, distances covered in high-intensity zones and the number of sprints have been reported to increase in the luteal phase relative to the follicular phase, and maximal aerobic capacity in field tests such as the Balke and Yo-Yo protocols has likewise favoured the luteal phase in several studies, whereas total distance and high-speed running were lower in the follicular phase in one cohort [36]. In basketball, by contrast, shooting accuracy and rebounding were higher in the follicular than the luteal phase, and in athletics anaerobic performance over repeated 400-m efforts was poorer in the menstrual and premenstrual phases [36]. Handball data using repeated-sprint protocols showed reduced peak power and greater muscle damage in the premenstrual phase, together with lower

neuromuscular efficiency of the vastus lateralis and rectus femoris, one of the few reports to link a specific phase to measurable exercise-induced muscle damage [36].

Two caveats temper these findings. First, match statistics are confounded by contextual variables — opponent strength, match result, tactical role — that the reviewed studies could seldom control, so apparent phase effects on shooting or distance covered may reflect the game rather than the cycle [36]. Second, several of the underpinning studies verified phase only by calendar or app-based tracking, importing the misclassification problem discussed later. The most that can be concluded is that discrete, task-specific components of team-sport performance may vary across the cycle in directions that differ between sports and even between studies of the same sport, that no single phase is uniformly "best", and that — consistent with the strength and endurance literature — the effects are neither large nor consistent enough to justify scheduling competition around cycle phase at a squad level [36,38].

10. Hormonal Contraception and Physical Performance

The performance effects of hormonal contraception broadly mirror those of the cycle: real at the level of perception and physiology, but trivial at the level of objective group performance. Elliott-Sale and colleagues conducted the definitive meta-analysis, pooling 42 studies and 590 participants within a Bayesian framework [14]. In between-group comparisons, OCP users performed, on average, slightly worse than naturally menstruating women, but the probability of even a small effect was low to moderate and the pooled effect most likely trivial; in within-group comparisons, performance did not differ between OCP consumption and withdrawal [14]. The authors concluded that OCP use might, on average, produce slightly inferior performance, but that any group-level effect is most likely trivial, that performance is consistent across the OCP cycle, and that the evidence does not warrant general guidance for or against OCP use — an individualised approach being more appropriate when performance is a priority [14].

Experimental studies reinforce this conclusion. Reif and colleagues measured isokinetic and isometric knee-extensor and flexor strength in 16 first-division female handball players during OCP consumption and withdrawal, verifying the hormonal contrast with serum FSH, LH, oestradiol and progesterone [12]. Despite significantly higher FSH and LH during withdrawal, no physiologically meaningful difference in any strength parameter emerged between phases, and the authors concluded that manipulating the cycle with OCPs to enhance thigh strength is unjustified [12]. Smith and colleagues examined a battery of physical and cognitive performance tests — countermovement and squat jumps, isometric mid-thigh pull, sprint, power pass and a Stroop test — across the cycle and across HC use in national-level rugby league athletes, again with hormonal verification [38]. Menstrual-cycle phase and HC use did not influence jump height, peak force, sprint time, throwing distance or the Stroop effect, and there were no differences between naturally menstruating and HC-using athletes; only small, arguably within-day-variability, differences in some kinetic and kinematic outputs were detected [38]. The authors argued explicitly that the evidence is insufficient to justify "phase-based" or "status-based" testing at a squad level, a conclusion of considerable practical importance for applied sport science [38]. That said, HC use is not physiologically neutral: Ekenros and colleagues found that 40% of users reported side effects — most commonly mood changes, breast tenderness and decreased libido — and that perceived physical performance was worst during the hormone-free (inactive-pill) interval, when withdrawal bleeding and dysmenorrhoea occur, rather than during active-pill consumption [13]. This locates the main perceived performance decrement of contraceptive users in the same symptomatic window as that of naturally menstruating women, again implicating symptoms rather than direct hormonal suppression of muscle function [13].

Two further nuances qualify the null conclusion. First, the meta-analysis of Elliott-Sale and colleagues detected relatively large between-study heterogeneity even as the pooled effect approached zero, indicating that individual women or individual formulations may respond in ways that group averages conceal, and the authors accordingly recommended an individualised rather than a general approach when performance is at stake [14]. Second, different contraceptive formulations were rarely separated: the reviewed studies aggregated combined and progestin-only pills, and different progestin generations and delivery routes, despite pharmacological reasons to expect them to differ, so that a genuine effect of a particular formulation could be masked within a heterogeneous "OCP user" category [14,13]. The prevalent practice of manipulating the pill cycle around competition — for instance skipping inactive pills to avoid withdrawal bleeding, reported by a large proportion of users — is therefore better justified by the avoidance of dysmenorrhoea and symptom-related performance decrement during the hormone-free interval than by any direct ergogenic effect of the active pills themselves [14,13]. In short, hormonal contraception neither reliably helps nor reliably harms objective performance at the group level, but it does shift the timing and, through symptom control, potentially the severity of the perceived performance fluctuations that athletes actually experience [14,13].

11. Motor Abilities: Flexibility, Balance, Agility and Cognition

Beyond strength, power and endurance, the cycle appears to influence several discrete motor abilities, although the evidence is thinner. Flexibility is the most consistently phase-sensitive: Elorduy-Terrado and colleagues reported a significant increase in hamstring and lumbar flexibility around ovulation compared with the early follicular phase, consistent with the general rise in joint laxity at that time, and the broader literature they reviewed corroborates cyclical variation in flexibility [36]. Balance and postural control also fluctuate; women with premenstrual symptoms show impaired postural control in the late luteal phase, and the pilot data of Kirschbaum and colleagues indicate cycle-by-continence interactions in single-leg sway [20,27]. Agility appears to change only marginally, with a small, non-significant improvement in change-of-direction speed reported in the follicular phase [36]. Cognitive function is an emerging domain of particular relevance to team sports: reaction time, cognitive processing speed and attentional, anticipatory and spatial cognition have been reported to fluctuate across the cycle, with deficits noted in the early follicular phase, which could affect rapid decision-making and tactical execution even when physical capacity is preserved [4,5]. Sleep and fatigue, closely tied to motor performance and injury risk, are reliably poorer in the luteal phases — Fort-Vanmeerhaeghe and colleagues documented significantly worse sleep and greater fatigue in the early-luteal and pre-menstrual phases of adolescent athletes, coinciding with the phase of highest injury incidence in their cohort [20]. Collectively, these findings suggest that the cycle's influence on the "softer" motor and cognitive abilities, and on the states (sleep, fatigue, mood) that support them, may be more consistent than its influence on maximal strength or aerobic power [20,36].

Table 2 summarises the principal studies bearing on the influence of menstrual cycle phase and hormonal contraception on physical performance and motor abilities, again with their designs, verification methods and main findings.

Table 2. Principal studies on menstrual cycle phase, hormonal contraception and physical performance or motor abilities.

Study [ref.]	Design	Population	Outcome	Main finding
Colenso-Semple et al. [10]	Umbrella review of reviews/meta-analyses	Eumenorrheic women	Strength, power, hypertrophy	No credible influence of cycle phase; higher-quality studies null

Meignié et al. [8]	Critical systematic review (7 studies)	Elite athletes	Performance	Inconsistent, mostly non-significant; no clear optimal phase
Niering et al. [35]	Systematic review + meta-analysis (22 studies)	Healthy women	Maximal strength	Early follicular unfavourable; isometric peaks late follicular, isokinetic at ovulation (small–medium effects)
Dasa et al. [33]	Prospective repeated measures	29 high-level team athletes	Strength, sprint, jump, power	No change across phases; no difference vs hormonal-contraceptive users
Isenmann et al. [34]	Randomised 3-arm crossover	24 strength athletes	Squat, jumps, well-being	No group effect; possible reduced squat during menses in highest-level athletes; lower well-being during menses
Ryman Augustsson & Findhé-Malenica [37]	Qualitative interviews	5 recreationally trained women	Perceived strength training	Strongest late follicular/ovulation; poorest late luteal; marked individual variation
Rael et al. [11]	Experimental (interval running)	21 endurance-trained women	Cardiorespiratory response	Most variables unchanged; ventilation and heart rate modified by phase
Docter et al. [32]	Experimental (LIT/HIT sessions)	15–16 endurance-trained women	Running economy	Cycle phase did not confound maintenance of running economy
Elorduy-Terrado et al. [36]	Systematic review (13 studies)	Eumenorrheic athletes	Multiple + match statistics	Flexibility better at ovulation; aerobic/anaerobic possibly better luteal; no consistent strength effect
Elliott-Sale et al. [14]	Systematic review + Bayesian meta-analysis (42 studies)	Women ± OCP	Exercise performance	OCP effect most likely trivial; performance stable across OCP cycle
Reif et al. [12]	Cross-sectional (OCP consumption vs withdrawal)	16 handball players	Knee-extensor/flexor strength	No meaningful difference between OCP phases
Smith et al. [38]	Observational, hormonally verified	National rugby league athletes	Physical + cognitive battery	No influence of phase or HC on jump, force, sprint, throw, Stroop
Ekenros et al. [13]	Survey	1,086 athletes, 57 sports	Perceived performance/symptoms	Perceived performance worst early follicular/late luteal; worst OCP window = inactive pills

12. Practical Implications: Tracking, Individualised Training and Injury Prevention

The translation of this evidence into practice must respect two overarching realities: the effects of cycle phase and HC on objective performance are small and inconsistent, whereas their effects on symptoms, perception, laxity and injury are real but individually variable. A one-size-fits-all, phase-based approach to training or competition is therefore not supported [4,38]. Instead, the reviewed sources converge on individualised menstrual tracking combined with adaptive training as the most defensible strategy [4,37]. A menstrual and training diary, ideally

incorporating symptom severity, sleep and perceived exertion, allows athletes and coaches to identify each individual's pattern — including whether she is a laxity "responder", how severe her dysmenorrhoea or premenstrual symptoms are, and in which phases she perceives her performance to decline — and to adjust load, recovery, nutrition and hydration accordingly [7,37]. Effective management of dysmenorrhoea deserves specific attention, since menstrual pain is a documented mediator of both perceived performance decline and refraining from training; beyond pharmacological analgesia and, where appropriate, hormonal suppression, non-pharmacological options such as kinesiology taping have shown statistically significant reductions in menstrual pain intensity and frequency and in the associated limitation of physical activity in female athletes [13,39].

Where the cycle appears most amenable to constructive adaptation is in nutrition and recovery rather than in the training load itself. Because the follicular phase is associated with enhanced carbohydrate utilisation, insulin sensitivity and glycogen storage, and the luteal phase with a shift toward fat oxidation, reduced insulin sensitivity and a modestly elevated basal metabolic rate, several reviews propose tailoring substrate provision to phase — favouring carbohydrate availability and glycogen replenishment in the follicular phase, when high-intensity and strength work may be prioritised, and accommodating greater fat utilisation and total energy needs in the luteal phase [5,7]. Recovery strategies may likewise be aligned with phase-specific tendencies: the slower recovery, greater inflammation and reduced protein synthesis reported in the luteal phase argue for adjusted training loads and extended recovery windows during that time, while restorative practices, active recovery, adequate protein intake and attention to sleep hygiene are particularly relevant when premenstrual fatigue and sleep disruption are at their peak [5,7]. Hydration and thermoregulatory considerations gain importance in the luteal phase, when elevated core temperature and fluid retention may impair endurance in the heat [5]. These strategies remain hypotheses in need of confirmation, and their effect sizes are likely small, but — unlike phase-based restriction of competition — they carry little downside and can be individualised through the same tracking that identifies each athlete's symptom and performance pattern [5,7].

For ACL injury prevention specifically, the evidence directs effort away from hormonal manipulation and toward neuromuscular training. Structured neuromuscular programmes targeting balance, coordination, landing mechanics, trunk control and lower-limb alignment reduce ACL injury incidence, by up to 90% in some analyses and by roughly 65% for non-contact injuries with individualised programmes, and decrease overall football injuries in women by about 20% [2]. The FIFA 11+ warm-up and similar protocols have been shown to reduce injuries in young female athletes, and the key elements identified — sessions longer than 20 minutes, at least twice weekly, with varied exercises and verbal feedback — provide a concrete template [2]. Because most ACL injuries are non-contact and arise from modifiable movement patterns and neuromuscular deficits that hormonal contraception does not address, such training addresses the larger and more actionable share of risk [2,3]. Screening should be multidimensional, integrating menstrual history and cycle regularity, specific anatomical predispositions such as genu recurvatum and generalised laxity, objective biomechanical assessment, and psychological and symptom factors, rather than relying on cycle phase alone [3]. Attention to modifiable extrinsic factors — appropriately fitted footwear, playing-surface and load management, and adequate recovery — and to relative energy deficiency and menstrual dysfunction, which carry their own musculoskeletal and health consequences, completes a coherent, female-specific prevention framework [2,22]. Mancino and colleagues highlight that sex disparities in access to structured, coached training, and the design of equipment around the male body, may themselves contribute to female ACL risk: football boots are engineered primarily around male foot anatomy and gait, an ill-fitting boot affects fatigue,

mobility and performance, and a large majority of female players report discomfort in their footwear, while shoe–surface traction is a recognised risk factor and pitch quality and congested fixture scheduling are cited by international players among the leading perceived causes of injury [2]. Such environmental and structural factors are, in principle, more readily modifiable than a woman's hormonal profile, and their neglect illustrates how narrowly the prevention discourse has sometimes focused on biology at the expense of context [2].

The psychological dimension of both injury and return to sport should not be omitted from a prevention and rehabilitation framework. High levels of personal and sport-related stress increase ACL injury risk through altered attention and coordination and increased muscular tension, and after injury, fear of re-injury and low knee-related confidence make female athletes markedly less likely to return to sport and more likely to underperform when they do [1,2]. Criteria-based return-to-sport decisions should therefore integrate strength symmetry, functional and movement-quality testing and psychological readiness rather than relying on time since surgery alone, and ongoing neuromuscular and injury-prevention training should continue after return to reduce the substantial risk of a second injury [1]. Embedding this within an individualised, cycle-aware framework — in which symptoms are treated, load is adapted to the athlete's own pattern, and neuromuscular competence is continually developed — offers a more coherent and evidence-consistent model of female-specific care than any protocol keyed rigidly to a nominal cycle phase [1,2,37].

13. Methodological Considerations and Limitations of the Evidence Base

No synthesis of this literature is credible without confronting its methodological fragility, which is the single most important reason for the field's contradictions. The classification and verification of menstrual cycle phase is the central problem. Many studies rely on calendar counting alone, which incorrectly assumes a 28-day cycle with ovulation on day 14, whereas the follicular phase can range from roughly 10 to 22 days and the luteal phase from 7 to 17 days, cycle length varies by more than seven days within the same woman, and regular menstruation does not guarantee ovulation, so that anovulatory or luteal-phase-deficient cycles can be misclassified [10,11]. Basal body temperature is convenient but unreliable, coinciding with the LH surge in only a minority of cycles, while urinary LH tests and serum hormone measurement are more accurate but costly and logistically demanding [10,11]. The recommended gold standard — the three-step method combining calendar counting, urinary ovulation testing and serum hormone confirmation — was employed in only a minority of the studies reviewed, notably those of Rael, Docter, Kirschbaum, Smith and the Feminae protocol [27,11,32,38,40]. Where it was applied rigorously, phase effects tended to shrink toward null, and Smith and colleagues found that a "true" late-follicular phase could be confirmed in only one of eleven ostensibly eligible athletes, a sobering illustration of how easily cycle research misclassifies its exposure [38].

Beyond phase verification, sample sizes are frequently small, statistical power is low, and study populations skew toward recreationally trained rather than elite women, limiting generalisability precisely to the athletes for whom the questions matter most [1,22]. Definitions of injury, eumenorrhoea and menstrual irregularity differ vastly, the geographic base is overwhelmingly North American and European, team-sport and non-elite athletes are under-studied, and hormonal contraceptive research aggregates heterogeneous formulations that may exert different effects [22,29]. Elite female athletes are systematically under-represented in sport and exercise science, partly because researchers regard hormonal fluctuations as difficult and expensive to control, a scientific disparity with real consequences: prevention and training programmes built predominantly on male-derived assumptions leave women systematically exposed [3,28]. The internationally coordinated Feminae multisite project by Elliott-Sale and

colleagues represents a direct response to these deficiencies, employing gold-standard phase verification, adequate power and a common protocol across seven sites in eumenorrheic and contraceptive-using athletes to test hypotheses about the effects of hormonal profiles on exercise physiology and performance, and offers a research-design blueprint that future studies should emulate [40].

14. Discussion

The evidence reviewed here supports a nuanced and, in places, deliberately deflationary synthesis. On injury, cyclical ovarian hormones do appear to modulate ACL risk, with the pre-ovulatory and ovulatory phases most consistently identified as the highest-risk window in hormonally verified studies and the luteal phase as the least associated with injury [6,15]. The mechanistic chain — peak oestradiol reducing collagen synthesis and lysyl-oxidase activity, increasing anterior knee and generalised joint laxity, and potentially altering neuromuscular control — is biologically coherent [3,6]. Yet the chain is weaker at every link than the headline association suggests. The meta-analytic increase in ovulatory-phase laxity is only 0.36 mm and of uncertain clinical significance; direct experimental studies disagree on whether anterior laxity changes at all, with the parameter measured (anterior laxity versus genu recurvatum versus generalised laxity) and the instrument used (arthrometry versus manual testing) frequently determining the conclusion; individual women differ as "responders" and "non-responders"; and prospective team-sport surveillance often locates injuries in the luteal or early follicular phase, plausibly through non-hormonal mediators such as fatigue, poor sleep, symptom burden and iron loss [6,21,20,24]. The reconstructed knee, which responds to the cycle oppositely to the native knee, further undermines any simple oestrogen–laxity–injury model [25].

The contraception evidence exemplifies how design determines conclusion. Case–control and registry studies repeatedly suggested a roughly 20% protective effect of OCPs, but the rigorous active-comparator new-user cohort of Herzog and colleagues found no such effect and demonstrated, within a single dataset, that the traditional design reproduces the spurious protection through confounding and selection [29]. The most defensible position is therefore that hormonal contraception does not reliably prevent ACL injury, that it addresses passive laxity while leaving neuromuscular and behavioural risk untouched, and that it carries potential detrimental effects and systemic side effects that preclude its use as a prevention tool [3,29]. The counter-intuitive association between menstrual dysfunction and lower ACL injury rates reported by Kirschbaum and colleagues, most plausibly reflecting reverse causation and behavioural mediators, is a further caution against simplistic hormonal reasoning [31].

On performance, the convergence is clearer and largely negative at the group level. Higher-quality, better-verified studies of strength, power and endurance repeatedly find trivial or no effect of cycle phase, and the meta-analysis of oral contraceptives likewise finds effects that are, at most, trivial and probably not practically relevant [10,14]. The apparent phase-specific optima reported in some meta-analyses, such as that of Niering, arise largely from the inclusion of lower-quality, poorly verified studies [10,35]. What does vary reliably is the subjective and symptomatic domain: perceived performance, well-being, sleep and fatigue worsen in the early follicular and late luteal phases, and this decrement is mediated by dysmenorrhoea and premenstrual symptoms rather than by a direct hormonal effect on contractile function [13]. This distinction — objective performance largely stable, perceived performance and symptoms cyclically variable — is the key to translating the evidence responsibly, because symptoms are treatable and perception is individual, whereas the underlying hormonal effect on muscle is small [34,13].

A recurring theme, cutting across both the injury and performance literatures, is the progressive displacement of purely hormonal explanations by mediating factors that are behavioural, symptomatic and contextual. On the performance side, the decrement that athletes reliably perceive in the early follicular and late luteal phases is statistically tied to dysmenorrhoea and premenstrual symptoms rather than to circulating oestradiol or progesterone, and the same symptom-linked window — the hormone-free interval — accounts for the perceived performance nadir of contraceptive users [13]. On the injury side, the phases most implicated by prospective team-sport surveillance coincide with the phases of poorest sleep, greatest fatigue, highest symptom burden and greatest iron loss, and menstrual dysfunction tracks energy availability rather than a specific hormone [21,20,31]. This does not render the hormones irrelevant — the in-vitro effects of oestradiol on collagen and of relaxin on matrix remodelling are real, and the ovulatory laxity signal, however small, is reproducible in meta-analysis — but it relocates much of the practically actionable variance away from the hormones themselves and toward states and symptoms that are measurable, individual and, crucially, treatable [6,13]. For the clinician and coach this is liberating rather than nihilistic: one cannot readily alter a woman's oestradiol peak without pharmacology of uncertain benefit and real cost, but one can treat her dysmenorrhoea, protect her sleep, correct iron deficiency, manage fatigue and train her neuromuscular control.

Several tensions in the literature deserve explicit acknowledgement. First, the injury and performance literatures reach opposite practical conclusions about the same hormonal peaks: the ovulatory oestrogen peak is cast as a risk factor for the ligament but, if anything, a neutral-to-favourable state for muscle strength, so that the "best" phase for performing may coincide with a vulnerable phase for the knee [6,35]. Second, the field's most confident claims come from its least rigorous designs, and its most rigorous designs — hormonally verified experiments and active-comparator cohorts — consistently deflate those claims [29,10,38]. Third, group-level nullity coexists with genuine individual variability, which means that dismissing cycle effects entirely is as unjustified as prescribing uniform phase-based protocols; the resolution is individualised tracking rather than either extreme [37,38]. Finally, the persistent under-representation of elite women and the methodological heterogeneity of the field mean that even these conclusions are provisional, and that adequately powered, phase-verified, multisite research such as the Feminae project is required before firmer guidance can be offered [22,40].

15. Future Directions

The priorities for future research follow directly from the deficiencies exposed above. Foremost is the universal adoption of rigorous cycle-phase verification. Studies should abandon calendar counting and basal body temperature as sole methods and adopt the three-step approach combining calendar tracking, urinary luteinising-hormone testing and serum measurement of oestradiol and progesterone, ideally sampling over more than one cycle to accommodate within-woman variability, and reporting anovulatory and luteal-phase-deficient cycles rather than silently misclassifying them [10,11,40]. The internationally coordinated Feminae project exemplifies the required standard, employing gold-standard verification, adequate statistical power and a common, single-blinded protocol across multiple sites in both eumenorrheic and contraceptive-using athletes, and its design should serve as a template [40].

Second, the field must broaden its populations. Elite female athletes — the group for whom marginal effects matter most — remain under-represented, as do team-sport athletes and athletes from Africa, Asia, Oceania and South America, and future work should deliberately recruit these groups and use standardised definitions of injury, eumenorrhoea and menstrual irregularity to permit comparative synthesis [22,38]. Third, research on hormonal contraception

should disaggregate formulations, since combined and progestin-only methods, and different progestin generations and delivery routes, cannot be assumed to act identically, and should prefer active-comparator new-user cohort designs capable of controlling the confounding that has distorted the case–control literature [29,14]. Fourth, mechanistic studies should move beyond anterior knee laxity to a multidimensional assessment integrating generalised laxity, genu recurvatum, neuromuscular and biomechanical injury-risk surrogates, and cognition, with attention to individual "responder" status, and should test whether relaxin, oestradiol and progesterone can be measured serially in field conditions [23,25,26]. Finally, prospective studies are needed to connect cyclical variation in laxity, neuromuscular control, symptoms, sleep and energy availability to actual injury occurrence and to performance, and to evaluate whether individualised, tracking-based interventions — as opposed to fixed phase-based protocols — improve outcomes in adequately powered trials [21,37,40]. Only research of this kind will allow the genuine but subtle influence of female reproductive hormones to be translated into truly evidence-based, female-specific guidance.

16. Conclusions

This review examined how menstrual cycle phase and hormonal contraception influence ACL injury risk, knee joint laxity and neuromuscular control on the one hand, and aerobic capacity, strength, power and motor abilities on the other. Several principal conclusions emerge.

First, ACL injury risk is most consistently elevated in the pre-ovulatory and ovulatory phases and lowest in the luteal phase, a pattern biologically linked to peak oestrogen and increased joint laxity; however, the effect is modest, the supporting laxity data are inconsistent and often clinically marginal, and prospective team-sport surveillance frequently implicates the luteal or early follicular phase through non-hormonal mediators such as fatigue, sleep and menstrual symptoms.

Second, hormonal contraception does not reliably reduce ACL injury risk. The roughly 20% protective effect seen in case–control and registry studies is best explained by confounding and selection bias, as demonstrated by a rigorous active-comparator cohort that found no effect; contraception addresses only passive laxity, leaves neuromuscular deficits unresolved, and carries potential harms, so it cannot be recommended as an injury-prevention strategy.

Third, at the group level the menstrual cycle and hormonal contraception exert trivial and inconsistent effects on objective strength, power and endurance, a conclusion strengthened by the higher-quality, hormonally verified studies. What varies reliably is perceived performance, well-being, sleep and fatigue, which worsen in the early follicular and late luteal phases and are mediated by dysmenorrhoea and premenstrual symptoms rather than by direct hormonal effects on muscle.

Fourth, motor abilities such as flexibility, balance and cognition, and the states of sleep and fatigue, appear somewhat more phase-sensitive than maximal strength or aerobic power, and warrant consideration in the design of training and recovery.

Fifth, and most importantly for practice, neither uniform phase-based training protocols nor routine contraceptive prescription is justified. The evidence supports individualised menstrual tracking, symptom-focused management including effective treatment of dysmenorrhoea, adaptive adjustment of load and recovery, and — for injury prevention — structured neuromuscular training and multidimensional screening that integrate menstrual history with anatomical, biomechanical and psychological factors.

Finally, the field is constrained by inadequate cycle-phase verification, small and predominantly recreational samples, heterogeneous definitions, and the systematic under-representation of elite women. Future research should adopt gold-standard three-step phase

verification, adequate statistical power and standardised, multisite designs, so that the genuine but subtle influence of female reproductive hormones on injury and performance can finally be characterised with confidence and translated into truly evidence-based, female-specific guidance.

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