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SLEEP, CHRONOTYPE, AND JET LAG IN ATHLETIC PERFORMANCE: A NARRATIVE REVIEW

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ABSTRACT

Background: The circadian timing system governs near-24-hour rhythms in body temperature, hormone secretion, and neuromuscular function that shape athletes' capacity to train, recover, and compete. Sleep, chronotype, and transmeridian travel are three interrelated circadian domains with direct medical relevance for performance.

Aim: To critically appraise current evidence on how sleep quantity and quality, chronotype, and jet lag influence physical and cognitive performance in athletes, and to review countermeasures.

Material and methods: Narrative review of experimental studies, systematic reviews, and meta-analyses addressing sleep, circadian rhythms, chronotype, and travel in athletic populations.

Results: Athletes habitually sleep under 8 hours per night with reduced efficiency, and 28–65% screen as poor sleepers. Acute sleep loss degrades physical performance by approximately

7.6%, with afternoon and evening tasks most affected. Chronotype shifts the diurnal performance curve: morning types perform best early and evening types later, while late chronotypes may vary by up to 26% across the day. Time since entrained awakening predicts peak performance more reliably than clock time. Athletes report higher perceived exertion when training against their circadian preference. Social jetlag and eastward transmeridian travel impair strength, power, and aerobic capacity and raise injury risk. Sleep extension, strategic napping, light management, and melatonin partially offset these deficits.

Conclusions: Sleep and circadian alignment are modifiable determinants of performance and health. Chronotype-informed scheduling and structured sleep and travel protocols should be integrated into athlete care, though heterogeneous methods limit firm conclusions.

Keywords: sleep; chronotype; circadian rhythm; jet lag; athletic performance; recovery; rate of perceived exertion

1. Introduction

Human physiology is organised in time. Nearly every tissue and physiological process oscillates over a period of approximately 24 hours, and these near-daily rhythms are coordinated by an endogenous circadian timing system whose central pacemaker resides in the suprachiasmatic nucleus (SCN) of the anterior hypothalamus, functioning as a master clock in concert with peripheral oscillators distributed throughout the body, including skeletal muscle and the cardiovascular system [1]. A complex network of neuronal, hormonal, and autonomic signals coordinates these clocks, generating outputs that regulate physiological, psychological, and behavioural processes across the day [1]. The clocks are synchronised to the external environment by zeitgebers—exogenous cues that maintain the regularity of circadian rhythms—of which the light–dark cycle is the most powerful, exciting or inhibiting the SCN through the retinohypothalamic pathway; additional zeitgebers include the sleep–wake transition, physical activity, social cues, and nutrition [1]. From these foundations arise systematic daily fluctuations in core body temperature, blood pressure, heart rate, and the secretion of hormones such as cortisol and melatonin, all of which influence the physical capabilities that determine athletic outcomes [2].

For athletes, the practical significance of this temporal architecture is threefold. First, sleep—the most conspicuous behavioural expression of circadian and homeostatic regulation—is indispensable for tissue regeneration, endocrine balance, exercise adaptation, and cognitive function, and its disruption consistently degrades performance and health [2]. Second, individuals differ in the phase of their circadian rhythms relative to the external day, a trait termed chronotype, which shapes the time of day at which an athlete's performance peaks and the effort they perceive when exercising at any given hour [1]. Third, rapid travel across multiple time zones desynchronises the internal clock from local time, producing jet lag: a transient but consequential disturbance of physiology and performance that is inherent to the professional athletic lifestyle [1]. These three domains—sleep, chronotype, and jet lag—are not independent phenomena but different manifestations of the same underlying circadian biology, and their interaction is of growing interest to sports medicine and the wider high-performance community [3].

The relationship between sleep and exercise is bidirectional [4]. On one hand, adequate sleep duration and quality form the foundation of recovery, supporting muscle repair, hormonal regulation, and the cognitive processes that underlie decision-making and motor coordination; on the other, physical activity, training intensity, competition timing, and travel themselves influence sleep quantity, quality, and architecture [4]. Despite the well-documented importance of sleep, a substantial body of evidence indicates that athletes frequently fail to obtain sufficient sleep, averaging fewer than eight hours per night across age groups and disciplines, with reduced sleep efficiency relative to non-athlete adults [5,6]. Compounding chronic sleep insufficiency, the frequent transmeridian travel demanded by international competition introduces circadian misalignment, travel fatigue, and jet lag, each of which can impair physical and mental performance and elevate injury risk [7].

Interest in the circadian dimensions of sport has expanded considerably in the past two decades, reflecting the recognition that, at the highest levels of competition, success is often decided by margins so small that any legitimate physiological advantage is worth pursuing [3]. Yet the literature remains fragmented across sub-disciplines—sleep science, chronobiology, exercise physiology, and travel medicine—and is frequently characterised by small samples, heterogeneous measurement tools, and conflicting findings [1]. The purpose of this narrative review is therefore to integrate and critically appraise the current evidence on how sleep, chronotype, and jet lag influence athletic performance, with an emphasis on the underlying medical and physiological mechanisms. The review first describes the circadian and sleep physiology that underpins the field; it then examines the epidemiology and consequences of sleep disturbance in athletes; it analyses the influence of chronotype on the diurnal variation of performance, perceived exertion, and anaerobic capacity; it addresses social jetlag and transmeridian travel; and finally it evaluates the countermeasures available to clinicians, coaches, and athletes. Throughout, particular attention is paid to comparing discordant findings and to identifying the methodological limitations that continue to constrain firm conclusions.

2. The Circadian System and the Temporal Organisation of Athletic Function

Chronobiology, the study of the rhythmic nature of living organisms, classifies biological rhythms into three categories according to their period: ultradian rhythms of less than 20 hours, circadian rhythms of approximately 24 hours, and infradian rhythms of more than 28 hours, the last further subdivided into circaseptan (seven days), circatrigintan (thirty days), and circannual (yearly) cycles [1]. Of these, the circadian rhythm is most relevant to the daily challenges of training and competition because it operates over the same timescale as the athlete's day [1]. The SCN acts as the central clock, receiving photic information and entraining a hierarchy of peripheral clocks so that the whole organism maintains internal temporal order [1]. Because the light–dark cycle is the dominant synchroniser, circadian phase can be influenced by the timing, intensity, duration, and spectral composition of light exposure, a fact that becomes clinically important when athletes travel or train under artificial illumination [1].

The behavioural and physiological outputs of this system converge on a small number of variables that are decisive for exercise capacity. Core body temperature (CBT) follows a robust circadian rhythm, reaching its nadir in the early hours of the morning (around 03:00) and its acrophase in the late afternoon and early evening (approximately 16:00–18:00) [8]. This rhythm has direct mechanical consequences: a higher CBT facilitates actin–myosin cross-bridging in skeletal muscle, enhances muscle compliance, increases the use of carbohydrate as a substrate, and accelerates nerve conduction and enzymatic activity, so that muscular strength and short-term power output tend to peak in the late afternoon coinciding with the temperature acrophase [8]. It is for this reason that much of the classical literature reports optimal performance in the late afternoon and early evening and impaired performance near the temperature nadir [8]. Hormonal rhythms reinforce this pattern. Cortisol, an indicator of psychophysiological stress associated with catabolism, peaks in the early morning under normal conditions, whereas the anabolic milieu that favours tissue repair is more prominent later; melatonin, secreted by the pineal gland, rises in the evening to promote sleep onset [2]. Peaks in cortisol, melatonin, and temperature occur earlier in individuals with a morning orientation than in those with an evening preference, illustrating the tight coupling between chronotype and the phase of these endogenous rhythms [1].

Empirical studies confirm that the influence of time of day extends across both aerobic and anaerobic exercise, although the direction of the effect is not always toward evening superiority, which is itself a source of the field's complexity. On the aerobic side, endurance exercise capacity at a fixed percentage of peak oxygen uptake has been reported to be greater in the morning than in the evening—including when exercise is performed in the heat—and reductions in heart rate have followed a period of aerobic training; yet other work has documented better performance capacity after several weeks of high-intensity training undertaken in the evening, and lower blood pressure during evening exercise associated with increased skin blood flow, so that the aerobic time-of-day literature is genuinely mixed [1]. The anaerobic literature is similarly heterogeneous: morning resistance training has produced greater anaerobic gains than evening training in some investigations, whereas evening exercise has yielded greater peak muscle power, higher Wingate power output, and greater grip and isokinetic strength in others, and short-duration maximal performance under neutral climatic conditions has been reported to peak between 16:00 and 20:00 [1]. Time-of-day effects on repeated-sprint ability have not always been detected, and where an evening advantage in muscle power and fatigue resistance is observed it appears to reflect a rise in the contractile properties of muscle driven by higher body temperature rather than a change in neural drive, since electromyographic activity often does not differ between morning and evening [1]. This pattern—an evening advantage for some qualities, a morning advantage for others, and no effect for still others—compounded by variation across sport, measurement method, and gender, is precisely what motivated the shift toward chronotype and time since awakening as more explanatory frameworks than clock time alone [1].

This temporal framework has been repeatedly confirmed across measures of muscular strength, anaerobic power, and short-term exercise, which independent of muscle group or contraction speed tend to peak in the late afternoon and early evening in the general population [8].

However, the assumption of a single, universal optimal time of day is an oversimplification. The amplitude and phase of the performance rhythm differ between individuals as a function of their chronotype, and the interaction between the endogenous circadian pacemaker and the homeostatic sleep drive means that performance at any given clock time also depends on how long the athlete has been awake [8]. Recognising this individual variability is the conceptual bridge between the physiology of the circadian system and the applied questions that dominate the remainder of this review.

3. Sleep Physiology and Its Role in Athletic Recovery

Sleep is not a passive state of rest but a highly active and structured biological process organised into cycles of approximately 90 minutes, of which a healthy athlete completes four to six per night [2]. Each cycle progresses through non-rapid eye movement (NREM) sleep, subdivided into stages N1, N2, and N3, and rapid eye movement (REM) sleep, each defined by characteristic electroencephalographic patterns [2]. Stage N1 marks the transition from wakefulness; stage N2, identified by sleep spindles and K-complexes, is strongly implicated in the consolidation of procedural and motor memory, such that adequate time in this stage is important for the long-term retention of sporting techniques and movement patterns [2]. Stage N3, or slow-wave sleep, is dominated by high-amplitude delta activity and constitutes the principal window for physical restoration, whereas REM sleep supports emotional memory processing, the generation of new strategies, and the integration of information into broader tactical patterns [2].

The physiological importance of these stages for athletes lies in their regulation of the endocrine and metabolic processes that govern recovery. During slow-wave sleep there is a pronounced surge in the secretion of growth hormone, testosterone, and insulin-like growth factor 1, hormones central to tissue repair, protein synthesis, and muscle growth, and there is robust evidence linking slow-wave sleep to elevated growth hormone release [2]. Conversely, insufficient sleep disrupts this anabolic environment: even a single night without sleep can reduce testosterone concentrations by nearly a quarter, while chronic sleep deprivation promotes catabolic dominance through dysregulation of cortisol, which in excess accelerates protein breakdown and impairs recovery [2]. Sleep restriction also curtails the resynthesis of muscle glycogen; experimental work has shown that reduced sleep limits glycogen replenishment, directly lowering endurance capacity and the ability to sustain high-intensity exercise [9]. In addition, sleep supports immune regulation and anti-inflammatory activity, whereas its deficiency elevates pro-inflammatory cytokines such as interleukin-6 and C-reactive protein, which hinder tissue repair and delay the restoration of peak performance [2].

Beyond these systemic effects, sleep loss exerts consequences at the molecular and cellular level that are only beginning to be characterised in athletes. Acute sleep deprivation combined with intense anaerobic exercise has been shown to shift gene expression toward pro-apoptotic pathways, increasing pro-apoptotic markers while reducing anti-apoptotic proteins and the core circadian regulator BMAL1 in peripheral blood mononuclear cells, potentially compromising cellular survival and rhythmicity [2]. Sleep deprivation has additionally been associated with increased accumulation of beta-amyloid in brain regions such as the hippocampus and thalamus after even a single night, which in athletes may contribute to slower reaction times, impaired decision-making, and reduced coordination [2]. Because these regenerative and protective processes are concentrated in sleep, and because the growth hormone essential for harmonious development and optimal adult performance is secreted in association with sleep, sleep is appropriately conceptualised as a "regeneration window" whose insufficiency can counteract the benefits of even the most carefully designed training programme [2].

The regulation of sleep and wakefulness rests on a neurochemical balance that is itself of interest to exercise physiology. Sleep is promoted by the inhibitory neurotransmitter gamma-aminobutyric acid, released by sleep-active neurons in the anterior hypothalamus to suppress wake-promoting regions, and by adenosine, which accumulates progressively during wakefulness to build sleep pressure and thereby links prior time awake to subsequent sleep need; wakefulness is sustained by acetylcholine, dopamine, norepinephrine, serotonin, histamine, and the orexin peptides [2]. Because regular training can modulate these dopaminergic, noradrenergic, and orexinergic pathways, physical activity and sleep are neurochemically as well as behaviourally coupled [2]. At the molecular level, circadian rhythmicity is generated by a core set of clock genes—including BMAL1 and the PER and CRY families—that interact in transcriptional–translational feedback loops to set the period of cellular oscillations, and adequate, well-synchronised sleep supports the expression of genes governing tissue repair and bone remodelling, whereas sleep fragmentation and circadian disruption impair these gene-mediated regenerative mechanisms [2]. Sleep architecture also changes with age: adolescents require approximately nine to ten hours of sleep and are prone to a delayed circadian phase and excessive daytime sleepiness, while slow-wave sleep declines and nocturnal awakenings increase into adulthood, so that younger athletes may require longer sleep to support neurodevelopment, motor learning, and adaptation to rising training loads [2]. These mechanistic considerations reinforce the clinical view of sleep as an active, regulated process whose disruption has consequences extending from the molecular to the whole-organism level.

The systemic consequences of sleep loss further underscore its medical significance. Short-term sleep deprivation imposes measurable cardiovascular strain: twenty-four hours of wakefulness has been shown to elevate resting blood pressure and to amplify the pressor response to submaximal isometric exercise, effects attributed to impaired baroreflex sensitivity and heightened sympathetic activation, and even a brief recovery nap during the deprivation period proved insufficient to normalise these haemodynamic changes [2]. Sleep restriction also dysregulates appetite through its effects on the orexigenic and anorexigenic hormones: two consecutive nights of restricted sleep in healthy young men increased the hunger hormone ghrelin by roughly a quarter and decreased the satiety hormone leptin by nearly a fifth, while shifting food preference toward calorie-dense, carbohydrate-rich items—a pattern that in athletes can undermine the nutritional strategies on which recovery and body composition depend [2]. Considered alongside the endocrine, immune, and molecular effects already described, these observations position chronic sleep insufficiency as a systemic physiological stressor rather than a mere behavioural inconvenience, carrying potential long-term cardiometabolic as well as immediate performance implications [2].

Given the physical and cognitive demands placed on athletes, it has been proposed that they may require more sleep than non-active individuals—on the order of nine to ten hours—to recover and adapt adequately between exercise sessions, compared with the seven to nine hours recommended for the general adult population [6]. Whether a single quantitative recommendation is appropriate has, however, been questioned, and an individualised approach incorporating an assessment of perceived sleep need has been advocated, since sleep requirement varies between individuals and some athletes report poor sleep while objective measures indicate sufficiency [4]. This tension between population-level guidance and individual variability recurs throughout the field and is directly relevant to the chronotype-based reasoning developed later in this review.

It should also be emphasised that the relationship between exercise and sleep is bidirectional, so that the timing and nature of training themselves shape subsequent sleep [4]. Contrary to the traditional caution against exercising close to bedtime, evening endurance and resistance exercise have been shown to alter sleep architecture only modestly and without demonstrable harm to sleep in healthy individuals, with predictive modelling suggesting that evening exercise compromises sleep on only a minority of nights [4]. Conversely, reduced physical activity—whether through injury, illness, enforced confinement, or retirement from sport—is associated with poorer sleep quality and increased insomnia, whereas maintaining training intensity is linked to better sleep, so that appropriately dosed and timed exercise is itself a sleep-promoting behaviour [4]. This reciprocity reinforces the view that sleep and physical activity should be managed together rather than in isolation, and that recovery modalities and strategic rest can be integrated with the training programme to optimise both limbs of the relationship [4,6].

4. Prevalence and Determinants of Sleep Disturbance in Athletes

A paradox lies at the heart of athletic sleep: although sleep is repeatedly ranked by athletes themselves as the single most important recovery strategy, a large proportion of them sleep poorly. The most comprehensive objective synthesis to date, a systematic review of eighty-one studies encompassing 1,830 athletes monitored over 18,958 nights by polysomnography or actigraphy, found that athletes slept on average only 7.2 ± 1.1 hours per night with a sleep efficiency of 86.3 ± 6.8%, and that in every dataset mean total sleep time fell below eight hours [5]. Reduced sleep efficiency was attributable chiefly to prolonged wakefulness after sleep onset rather than to difficulty initiating sleep, and it was particularly marked in young athletes, whose sleep efficiency averaged only 80.3 ± 8.8% [5]. Sleep quality and architecture were further shown to fluctuate across the training year, with sleep duration and efficiency reduced during heavy training periods relative to pre-season and in-season phases [5]. These objective data corroborate the widely cited observation that average sleep duration in elite athletes measured by actigraphy is approximately 6.5 hours, that roughly two-fifths self-report inadequate sleep, and that poor sleep quality, daytime fatigue, and excessive daytime sleepiness are common regardless of sport [6].

The interpretation of these prevalence figures depends on the assessment tools employed, and a brief consideration of methodology is therefore warranted. Polysomnography, which records the electroencephalogram together with eye movements, muscle activity, and cardiorespiratory variables, is the gold-standard measure of sleep architecture and disorders, but it is resource-intensive, intrusive, and impractical for routine longitudinal monitoring of athletes in their home or travel environments [2,6]. Actigraphy, using a wrist-worn accelerometer to infer sleep and wake from movement, offers a non-invasive, field-based alternative validated against polysomnography with roughly 85–90% agreement, although it tends to underestimate total sleep time and to misclassify quiet wakefulness as sleep [6]. Subjective instruments remain the most accessible tools: the Pittsburgh Sleep Quality Index quantifies overall sleep quality, with a global score above five indicating poor sleep, though a more conservative cut-off of eight has been proposed for athletes given their potentially greater sleep needs; the Epworth Sleepiness Scale captures daytime sleepiness; consensus sleep diaries record night-to-night continuity; and athlete-specific instruments such as the Athlete Sleep Screening Questionnaire and the Athlete Sleep Behaviour Questionnaire identify clinically significant problems and maladaptive practices [2,10]. Chronotype is assessed with the Morningness–Eveningness Questionnaire, the Munich ChronoType Questionnaire, and the athlete-oriented Athlete Morningness–Eveningness Scale and reduced MEQ, while purpose-built chronometric tests and longitudinal sleep records have been developed to characterise circadian disruption in athletes specifically [1,8,11]. Because athletes tend to overestimate self-reported sleep relative to objective measures, and because heterogeneous methods yield divergent findings, the choice of assessment approach materially shapes conclusions and complicates cross-study comparison [5].

Subjective assessments converge on the same conclusion while adding population-specific detail. Among 817 elite Japanese athletes assessed with the Pittsburgh Sleep Quality Index (PSQI), 28% exceeded the clinical cut-off for poor sleep quality, and poor sleep was independently associated with shorter time in bed, skipping breakfast, using electronic devices before bedtime, depressive mood, and ruminating about problems in bed—five modifiable factors that define natural targets for sleep education [12]. A survey of 338 elite and sub-elite athletes reported an even higher prevalence, with 64% classified as poor sleepers by a PSQI global score of five or more, and with poor sleep attributed to difficulty falling asleep within thirty minutes, nocturnal and early awakenings, and nocturia [11]. A cross-sectional study of 82 young elite university athletes similarly documented that sleep duration and quality deteriorated at the start of the academic year, coinciding with the highest acute-to-chronic training workloads and the accumulation of academic and social stressors, and that nights of eight or more hours were associated with improved mood, energy, perceived training quality, and a reduced risk of injury and illness [13]. Comparable findings of poor sleep quality have been reported in collegiate athletes, in whom disturbed sleep was widespread and further exacerbated during the COVID-19 pandemic period [14], and in elite adolescent athletes, in whom sleep difficulty correlated with impaired recovery [15].

The systematic review literature has attempted to determine whether elite sport itself degrades sleep. A review of thirty-seven studies concluded that athletes exhibit a high overall prevalence of insomnia symptoms characterised by longer sleep latencies, greater sleep fragmentation, non-restorative sleep, and excessive daytime fatigue, and identified three broad risk factors for sleep disturbance: training, travel, and competition [16]. Two mechanisms were implicated in the mediation of sport-related insomnia symptoms—pre-sleep cognitive arousal and sleep restriction—with sleep quality appearing most vulnerable prior to major competitive events, during periods of high-intensity training, and following long-haul travel [16]. These sport-specific determinants operate alongside the individual and environmental factors familiar from general sleep medicine, including irregular sleep–wake schedules, late-night light exposure, caffeine, and psychological stress [11]. Post-competition obligations such as media commitments, medical care, and travel delay bedtimes, while later competition timing increases arousal and elevates CBT at night, making sleep onset more difficult [6]. Pre-competition anxiety and excitement raise pre-sleep hyperarousal and can precipitate acute insomnia, and heightened adrenaline and cortisol after stressful competition, particularly after losses, further delay sleep onset [6].

The night before competition deserves specific mention as a recurrent flashpoint for acute sleep disturbance. Pre-competition anxiety, arousal, and the disruption of routine frequently precipitate difficulty falling asleep, and disturbed sleep before important events is so common as to be almost normative in elite sport, even though it may still impair next-day performance [6]. This vulnerability is consistent with the systematic-review conclusion that sleep quality is most fragile immediately before major competition, during intensified training, and after long-haul travel, and that it is mediated principally by pre-sleep cognitive arousal rather than by a lack of sleep opportunity [16]. Because competition-eve insomnia is driven by arousal, the relaxation, mindfulness, and cognitive strategies discussed later are especially pertinent to it, and unrealistic expectations about achieving perfect sleep before competition should be actively countered so as not to compound the underlying anxiety [6,16].

An important and consistent thread is the elevated vulnerability of young and student-athletes, in whom the biological tendency toward eveningness during adolescence collides with early training schedules and academic demands to produce chronic sleep restriction [5,17]. Because sleep in this developmental window supports neurodevelopment, motor learning, and adaptation to increasing training loads, and because insufficient sleep in young athletes is linked to higher injury rates, the young athlete emerges repeatedly as a priority population for intervention [2,5]. The recognition that a majority of athletes across levels obtain inadequate sleep provides the essential backdrop for the next question: what are the measurable consequences of that sleep loss for performance and health?

5. Consequences of Sleep Loss for Physical and Cognitive Performance

The evidence that inadequate sleep impairs athletic performance is now quantitatively robust. A systematic review and meta-analysis of 227 outcome measures from sixty-nine publications found that sleep loss produced an overall negative effect on the percentage change in exercise performance, with a mean decrement of approximately 7.6%, and that the effect was significant across all exercise categories examined, including anaerobic power, speed and power endurance, high-intensity interval exercise, strength, endurance, strength-endurance, and skill [17]. Crucially, this meta-analysis clarified two moderators of central practical importance. First, the pattern of sleep loss mattered: consistent negative effects were observed only with total sleep deprivation and with late sleep restriction (waking earlier than normal), whereas early restriction (delayed sleep onset) was less uniformly detrimental. Second, and most relevant to the circadian theme of this review, the time of day at which the exercise task was performed determined the magnitude of impairment—tasks performed in the afternoon and evening were consistently degraded by sleep loss, while morning tasks were largely unaffected, and a significant positive relationship linked time awake before the task to the size of the performance decrement, with performance falling by roughly 0.4% for every additional hour of prior wakefulness [17]. This time-of-day dependence is itself a circadian phenomenon and foreshadows the interaction between sleep and chronotype developed later.

A separate meta-analysis of forty-five studies extended these conclusions across populations and outcomes, reporting that sleep deprivation significantly impaired aerobic endurance (standardised mean difference −0.66), explosive power (−0.63), maximum force (−0.35), speed (−0.52), and skill control (−0.87) in athletes, and significantly increased the rating of perceived exertion (+0.39), with aerobic endurance also reduced in healthy non-athletes (−1.02) [18]. The comparatively large effect on skill control is notable, since technical execution and decision-making are decisive in most sports and may be more vulnerable to sleep loss than gross strength. Subgroup analysis again implicated the timing of sleep loss, with early sleep deprivation significantly reducing explosive power, maximum force, speed, and perceived exertion, reinforcing the meta-analytic conclusion that the pattern and timing of sleep loss, not merely its occurrence, determine its performance cost [18]. These pooled findings are reinforced by mechanistic experimental work. In a controlled crossover study, a single night of partial sleep deprivation following prolonged exercise did not significantly change maximal isometric strength or oxygen kinetics during submaximal running the next morning, but it significantly shortened time to exhaustion during high-intensity running and lowered the respiratory exchange ratio, indicating augmented fat utilisation consistent with reduced muscle glycogen availability [9]. The dissociation observed in that study—preserved maximal strength but impaired endurance—illustrates that sleep loss does not affect all performance qualities uniformly, a nuance that pooled effect sizes can obscure and that is consistent with the broader observation that high-intensity intermittent exercise, skill-based tasks, explosive power, and speed are among the qualities most sensitive to sleep loss [2].

The cognitive and neurological consequences of sleep loss are equally consequential for sports that demand rapid, accurate decisions. Sleep deprivation impairs attentiveness and reaction time and degrades an athlete's capacity to choose quickly and accurately between a safe and a risky option during a game or event, and because sleep supports learning and the generation of novel solutions, it is also linked to the creative problem-solving on which elite athletes rely at decisive moments [6]. Sleep and injury interact bidirectionally in the specific context of concussion: insomnia and excessive daytime sleepiness have been identified as risk factors for concussion that in some analyses outperform established risk factors such as participation in high-risk sports or a prior concussion history, and a high proportion of individuals—on the order of half—experience sleep disturbance following a concussion, with persistent post-concussion sleep problems serving as a reliable predictor of prolonged recovery [6]. At the level of recovery physiology, disrupted sleep reduces growth-hormone release, alters cortisol secretion, and elevates pro-inflammatory cytokines such as interleukin-6 and C-reactive protein, while sleep-deprived individuals tend to consume less healthy food, together impairing glycogen repletion and muscle protein synthesis and predisposing to overtraining [6]. These cognitive, neurological, and recovery-related effects broaden the clinical footprint of sleep loss well beyond the acute performance decrements captured by meta-analysis [6].

The consequences of sleep loss extend well beyond acute performance to encompass injury risk, recovery, and immune and mental health, giving the phenomenon a distinctly medical character. A systematic review of thirty-one studies concluded that insufficient sleep is a critical and modifiable risk factor for sports injury, with a dose–response relationship in which injury risk rises progressively as sleep duration falls below eight hours per night, and with sleep deprivation in the fourteen days preceding an injury showing the strongest association, suggesting a cumulative effect of accrued sleep debt [19]. The proposed mechanisms—impaired cognitive function and reaction time, weakened neuromuscular control and proprioception, increased systemic inflammation, impaired tissue repair through disrupted growth hormone secretion, hormonal dysregulation, and autonomic imbalance—map directly onto the physiological roles of sleep described earlier and provide a coherent biological rationale for the epidemiological association [19]. Poor sleep quality has additionally been shown to independently increase injury likelihood beyond duration alone [19]. In parallel, sleep is a determinant of immune competence: poor sleep quality disrupts the normal circadian coordination of pro- and anti-inflammatory processes, elevating markers such as interleukin-6 and tumour necrosis factor-alpha and reducing the functionality of immune cells, thereby increasing susceptibility to infection and impairing recovery, effects that are further compounded by the stress and depressive symptoms that frequently accompany disturbed sleep [20]. Because a lack of adequate sleep is associated with reduced reaction times, altered cognitive function, and mood disturbance, and because heavily trained athletes with signs of sleep disturbance report more upper respiratory tract infections, the cumulative picture is one in which sleep insufficiency simultaneously undermines performance, recovery, immunity, and safety [13].

6. Chronotype: Definition, Assessment, and Distribution in Athletes

Whereas the preceding sections concern the amount and quality of sleep, chronotype concerns its timing and, more broadly, the phase of the individual's circadian rhythms relative to the external day. Biologically, individuals prefer to rest and to be active at particular times, a tendency toward morning or evening orientation termed chronotype and conventionally assessed by self-report questionnaires [1]. The morning chronotype describes a preference for completing activities early and retiring early, the evening chronotype a preference for afternoon and later activity with delayed sleep, and the intermediate or "neither" type, which is the most common among adults, lies between these poles [1]. The most widely used instrument is the Horne–Östberg Morningness–Eveningness Questionnaire (MEQ), which classifies respondents into morning, evening, and neither types; related instruments include the Munich ChronoType Questionnaire (MCTQ), which derives the corrected mid-sleep point on free days, and athlete-specific tools such as the Athlete Morningness–Eveningness Scale and the reduced MEQ [1,8,11]. Importantly, chronotype is not merely a behavioural preference but is anchored in physiology: diurnal preference has been correlated with the endogenous circadian rhythms of core body temperature and melatonin, such that peaks in cortisol, melatonin, and temperature occur earlier in morning-oriented than in evening-oriented individuals, and chronotype further encompasses variation in synchronisation to jet lag, personality, and cognitive function [1].

A recurrent and clinically interesting observation is that athletes, as a group, exhibit a distribution of chronotypes skewed toward morningness relative to the general population. In a controlled comparison of sixty-three elite winter-sport athletes with eighty-three non-athlete good sleepers, the athletes showed a greater prevalence of morningness despite there being no group difference in self-reported bedtime or wake time, and greater eveningness within the athlete group was significantly correlated with poorer sleep quality [21]. Prior surveys have reported that morning types constitute a substantial fraction of elite endurance athletes—37% of over five hundred cyclists, runners, and triathletes were morning types who preferred to train in the morning—and comparable morning-skewed distributions have been documented in bobsleigh, Paralympic athletics, and multi-sport cohorts [21]. Two explanations have been proposed for this skew, and they are not mutually exclusive: athletes may self-select into sports whose training and competition times align with their innate chronotype, thereby increasing their likelihood of excelling, or the external demands of elite sport—particularly early-morning training—may generate a form of social jetlag that masks or shifts the expressed chronotype [21]. The prevalence of late chronotypes among elite athletes has been estimated at only around 10%, markedly lower than the roughly 40% estimated in the general population, an observation interpreted as evidence that late chronotypes may be less likely to reach national or international level and that chronotype could be a factor shaping the path to a successful athletic career [8].

Distribution data within specific team sports reinforce this picture while revealing sport-specific nuance. Among fifty-eight young soccer players, the sample comprised 19% morning types, 50% neither types, and 31% evening types, a distribution consistent with the known adolescent drift toward eveningness that peaks around the age of twenty before a lifelong return toward morning preference [22]. In a squad of thirty-six elite female Australian footballers, only three players (10%) were evening types, with the majority classified as intermediate (55%) or morning (35%) types, mirroring data from male counterparts and other team codes such as hockey and soccer [23]. Among 665 Japanese student-athletes, the chronotype distribution was 15.9% eveningness, 72.9% intermediate, and 11.1% morningness [24]. Across these cohorts the intermediate type predominates, but the consistent under-representation of confirmed evening types among athletes—especially at elite level—supports the view that chronotype is a non-trivial correlate of athletic participation and success. Having established what chronotype is, how it is measured, and how it is distributed, the central applied question can now be addressed: does chronotype modify the time of day at which athletes perform best?

7. Chronotype and the Diurnal Variation of Athletic Performance

The classical model of diurnal variation holds that performance peaks in the late afternoon and early evening in synchrony with the core body temperature acrophase [8]. Chronotype introduces a systematic individual departure from this population average, and a substantial body of work—synthesised in a dedicated narrative review of chronotype and athletic performance—has examined whether accounting for chronotype reveals diurnal performance profiles that clock time alone conceals [1]. The evidence, drawn from rowing, swimming, running, basketball, and field hockey, is instructive precisely because it is not uniform.

The rowing literature provides some of the clearest support for a chronotype effect. In a study of sixteen collegiate rowers who completed a 2,000-metre rowing test and a standing broad jump during morning (05:00–07:00) and afternoon (16:30–18:00) schedules, participants rowed significantly more slowly in the evening than in the morning, with thirteen of the sixteen slowing by an average of 2.4 seconds (0.52%) from morning to evening [1]. When the sample was stratified by chronotype, morning types substantially reduced their rowing speed from morning to evening by 4.8 seconds (1.1%)—double the mean decrement—whereas evening and neither types showed no significant change across the day, and each individual's morning-to-evening change correlated strongly with their morningness score ($r = 0.880$ and $r = 0.863$ on two scales) [1]. Swimming research points in the same direction. Among twenty-six trained swimmers compared at 06:30 and 18:30, the group as a whole performed no differently between the two times (158.8 versus 158.5 seconds, $p = 0.611$), but stratification by chronotype and habitual training time exposed significant diurnal variation, with morning-type swimmers and those training in the morning performing better in the 06:30 trials ($p = 0.036$ and $p = 0.011$) [1]. A second swimming study of twenty-seven swimmers and divers performing 200-metre time trials at 07:00 and 19:00 found that evening types were on average 6% slower in the morning with higher α-amylase concentrations, whereas morning types required five to seven times greater exertion to achieve the same performance in the evening trial [1]. This last finding is important because it demonstrates that the cost of performing outside one's optimal phase may be paid in effort even when the outcome is matched.

Not all evidence supports a chronotype effect, and the discrepant findings are as informative as the positive ones. In a study of eleven professional male basketball players competing in evening games (after 18:00), in-game performance was not significantly affected by chronotype—no significant differences ($p = 0.21$ for most performance measures) emerged between morning and neither types—prompting the authors to suggest that practitioners may not need to consider chronotype when scheduling and assigning roles for evening competition [1]. This apparent contradiction is reconciled by noting that the basketball games were played exclusively in the evening, so the study could not detect a morning–evening interaction, and that team-sport in-game performance is a coarse, multifactorial outcome relative to a timed rowing or swimming trial [1]. Marathon data add a further dimension: a comparison of South African and Dutch runners found that MEQ scores were negatively correlated with best and current marathon times among the more morning-oriented South African runners, and that runners preferring morning starts might better tolerate early-morning marathon start times [1]. Together these studies suggest that chronotype effects are most readily detected in individual, precisely timed, and physiologically demanding tasks, and are most easily obscured in team sports, in single-time-point designs, and in coarse performance measures—an interpretive principle that helps explain the "conflicting evidence" repeatedly noted in the field [1].

Perhaps the most influential contribution has been the reconceptualisation of the problem in terms of circadian phenotype and time since entrained awakening rather than clock time. Using a novel chronometric test in competition-level field hockey players categorised as early (ECT, 28%), intermediate (ICT, 48%), or late (LCT, 24%) circadian types, investigators found that peak performance on a repeated shuttle-run test occurred at markedly different clock times across phenotypes: ECTs peaked at approximately 12.2 hours, ICTs at 15.8 hours, and LCTs at 19.7 hours [1]. The time-of-day fluctuation in performance was modest for early types (7.6%) and intermediate types (10.0%) but striking for late types (26.2%), and the single best predictor of peak performance was the duration since entrained awakening, which accounted for up to 26% of individual performance variation across the day [1]. The applied implication is profound: the time of day per se does not determine when an athlete performs best; rather, what matters is the interval since they awoke, together with their diurnal preference [1].

This time-since-awakening framework has been extended from athletes to physically active volunteers in a rigorously controlled study that measured multiple domains simultaneously. Fifty-six individuals categorised as early (n = 25) or late (n = 31) chronotypes by the MCTQ, circadian phase markers, and actigraphy completed measures of daytime sleepiness, psychomotor vigilance, executive function, and isometric grip strength at 14:00, 20:00, and 08:00 [8]. Late chronotypes were significantly impaired in the morning across every measure: at 08:00, early types outperformed late types by 8.4% in psychomotor vigilance, 5.9% in executive function, and 7.4% in maximal voluntary contraction, and reported markedly lower sleepiness [8]. The amplitude of diurnal variation differed by phenotype and task—grip strength varied by 3.8% across the day in early types but 10.1% in late types—and peak grip strength occurred at 14:00 in early types but 20:00 in late types [8]. When analysed by time since awakening, cognitive performance in early types peaked almost immediately after waking and physical performance some hours later, whereas late types did not reach their peak until at least twelve hours after waking, implying a much narrower window of opportunity for late chronotypes to perform optimally during a conventional day [8]. Complementary work on time-of-day and anaerobic performance in trained men, analysed in relation to chronotype, similarly reports diurnal variation in anaerobic output and blood lactate response, reinforcing that anaerobic as well as aerobic and cognitive qualities are subject to chronotype-dependent temporal modulation [25].

An additional subtlety from the healthy-volunteer work concerns the differential vulnerability of simple and complex cognitive tasks. Late chronotypes showed significant morning impairment on a simple psychomotor vigilance task but a damped diurnal amplitude on a more complex executive-function task, a dissociation interpreted as a compensatory increase in engagement and arousal during the more demanding task, and consistent with meta-analytic evidence that simple, monotonous tasks are more adversely affected by reduced alertness than complex, stimulating ones [8]. For the applied setting this implies that the cognitive cost of performing outside one's optimal circadian window may fall most heavily on the sustained-attention demands—vigilance, anticipation, and consistency—that underpin reliable performance in many sports, even where more engaging, complex skills appear partially protected [8].

The aerobic domain provides a final confirmatory example that also demonstrates the interaction of chronotype with sleep. In fifty-eight young soccer players tested by a six-minute run at 08:30 and 18:00, repeated-measures analysis revealed a significant effect of chronotype specifically on aerobic endurance: morning types performed better in the morning session and evening types in the evening session, while neither types did not differ between sessions, and no chronotype effect was found for agility or lower-limb strength [22]. The selective susceptibility of endurance—but not strength or agility—to chronotype in that study echoes earlier rowing data in which aerobic performance was affected while strength was not, and suggests that longer, more metabolically demanding efforts may better expose the underlying circadian differences than brief explosive tasks [22]. Taken together, the diurnal-variation literature supports a genuine but task-dependent and phenotype-dependent influence of chronotype on performance, most robustly demonstrated in individual endurance and repeated-effort tasks and best understood through the lens of time since entrained awakening rather than absolute clock time.

Beyond the individual athlete, chronotype operates at the level of the team. The circadian-phenotype composition of a squad has been reported to be a major predictor of its diurnal physical performance, giving rise to the concept of a "circadian advantage" whereby aligning a team's aggregate chronotype with competition timing may confer a collective edge [8]. Conversely, internal circadian desynchrony can undermine team output: in professional basketball, the circadian disadvantage associated with eastward travel has been linked to a reduction in winning probability of around 6%, together with lower shooting accuracy and rebounding, and in synchronised sports such as rowing, crews with mismatched internal clocks show poorer technical coordination and slower race times, particularly in early-morning competition when evening-type athletes are forced to perform during their biological night [26]. These team-level observations extend the applied relevance of chronotype from individual scheduling to squad selection and tactical planning [8,26].

8. Chronotype, Perceived Exertion, and Training Load

If chronotype shifts the phase of peak performance, it should also shift the phase of lowest perceived effort, and a coherent body of evidence indicates that it does. Chronotype correlates with ratings of perceived exertion (RPE) and fatigue such that morning types feel less exhausted, and perform equivalent work as less effortful, in the morning [1]. In qualified male cyclists classified as morning types, RPE during a submaximal cycle test was higher in the evening than in the morning even though there was no time-of-day effect on power output, speed, or cadence—that is, the same relative workload felt harder in the evening, an experience the authors linked to greater morning motivation and intensity [1]. Swimming data reveal a subtler pattern: after warm-up there was no difference in RPE between morning and neither types, and the intensity of the time trials abolished any post-trial RPE difference, yet a time-by-group interaction emerged for vigour and fatigue, with morning-type swimmers reporting lower fatigue before the 06:30 trial than before the 18:30 trial and higher vigour scores in the morning [1]. The dissociation between RPE, which was insensitive, and mood dimensions such as vigour and fatigue, which were sensitive, cautions against relying on any single subjective marker to detect chronotype effects [1].

The most direct experimental demonstration comes from high-intensity interval exercise performed at different times by morning and evening types. In a randomised crossover study, morning types showed higher RPE in the pre-condition before evening training, whereas evening types showed elevated RPE before morning training, and intragroup analysis indicated that evening types reported higher RPE than morning types both before and thirty minutes after the interval session; the evening session produced no significant changes [1]. The authors concluded that chronotype significantly influences fatigue, perceived exertion, and stamina, that evening types are especially likely to struggle with early-morning physical tasks, and that high-intensity interval exercise should be scheduled according to the athlete's chronotype [1]. Field data from team sport corroborate the laboratory findings: among elite female Australian footballers training in the evening, morning types reported significantly higher RPE (6.7 ± 1.1) than intermediate types (5.9 ± 1.2, $p = 0.01$) despite no difference in training duration or internal training load, consistent with earlier reports that morning-type cyclists and active young people experience higher perceived exertion when training in the afternoon or evening [23]. Notably, this same footballer study observed no difference in RPE between chronotypes after a shorter (roughly two-and-a-half-minute) swimming time trial in a related cohort, and it was suggested that the aerobic demand and longer duration of a football session, relative to a brief time trial, may be necessary to expose chronotype-related differences in perceived exertion [23]. This duration-dependence again parallels the performance literature and points to a consistent principle: the synchrony or mismatch between exercise timing and chronotype is expressed more clearly in longer, aerobically demanding efforts.

The practical corollary is that an athlete required to train or compete at a time opposing their circadian preference may experience the session as more effortful without any change in the externally prescribed load, a discrepancy with direct implications for training monitoring [23]. Because internal training load is commonly derived from session RPE, systematic chronotype-related inflation of perceived effort at non-optimal times could distort load calculations and, if unrecognised, lead to misinterpretation of an athlete's training tolerance [23]. Practitioners have therefore been advised to monitor individual trends in RPE and training load with explicit consideration of chronotype and training time [23].

9. Chronotype, Sleep Quality, and Anaerobic Performance

Chronotype influences performance not only through the direct circadian modulation described above but also indirectly, through its association with sleep quality. This indirect pathway has been examined most directly in a study of 340 elite athletes screened for chronotype with the Korean MEQ and assessed with the PSQI and the Wingate Anaerobic Test [27]. Sleep quality varied systematically with chronotype: PSQI global score, subjective sleep quality, sleep-onset latency, sleep disturbance, and daytime dysfunction all differed significantly across groups, with morning types reporting better sleep than evening types, and the significant difference in sleep-onset latency between morning and evening types was highlighted as a key discriminating variable [27]. Crucially, anaerobic performance mirrored this gradient: power drop, mean power, and peak power on the Wingate test were significantly higher in moderately morning types than in definitely evening types, and a significant negative correlation linked the PSQI score to mean and peak power, indicating that poorer sleep quality was associated with lower anaerobic output [27]. The authors concluded that poor sleep quality and a late chronotype together may reduce performance in elite athletes, that sleep quality is higher in early than in late chronotypes, and that athletic performance is better in early than in late chronotypes [27].

This finding is coherent with the broader sleep-quality-by-chronotype literature. Among 665 student-athletes, individuals with later chronotypes had a higher prevalence of poor subjective sleep quality and were more likely to skip breakfast and to use a smartphone after lights-out, while a lower proportion participated in frequent morning practice; strikingly, frequent morning practice was associated with poor subjective sleep quality only among evening-chronotype student-athletes and not among intermediate or morning types, implying that the collision between an early schedule and a late chronotype is specifically deleterious [24]. In elite athletes compared with non-athlete controls, chronotype was significantly correlated with sleep quality ($r = -0.35$, $p = 0.005$) such that greater eveningness was associated with poorer sleep, a correlation not observed in the non-athlete controls, which was interpreted as evidence of a misalignment between preferred and actual sleep times imposed by the demands of sport [21]. The convergence of these datasets supports a model in which eveningness predisposes athletes to shorter, later, and poorer-quality sleep—particularly when sport imposes early schedules—and in which the resulting sleep debt compounds the direct circadian disadvantage of performing outside one's optimal phase, plausibly explaining part of the performance decrement observed in late chronotypes [21,24,27]. This dual mechanism, direct circadian plus indirect sleep-mediated, provides the conceptual link to the phenomenon of chronic circadian misalignment considered next.

10. Social Jetlag and Chronic Circadian Misalignment

When an athlete's endogenous chronotype is repeatedly forced out of alignment with imposed social and training schedules, the result is a chronic form of circadian misalignment termed social jetlag, quantified as the absolute difference between the mid-point of sleep on scheduled days and the mid-point of sleep on free days [26,28]. Social jetlag is highly prevalent—estimated to affect a majority of the population and up to around 70% of some young-adult samples—and is particularly acute in the young-adult and adolescent demographic, in whom a developmental shift toward eveningness collides with early academic and training obligations, forcing physiological activity to begin during the biological night and producing cumulative sleep restriction [26]. Unlike transmeridian jet lag, social jetlag arises without any travel and reflects a persistent mismatch between biological and social time [26].

The performance consequences of social jetlag have been documented across cardiorespiratory, neuromuscular, and tactical domains. Higher social jetlag is inversely associated with cardiorespiratory fitness: adolescents and young adults with social jetlag of 1.5 hours or more demonstrate significantly lower maximal oxygen uptake, with each additional hour of social jetlag linked to a measurable decrement in fitness that is more pronounced in males than in females and that persists after adjustment for total sleep duration, implying that the timing of sleep, not merely its amount, drives the effect [26]. In the anaerobic domain, social jetlag blunts the normal diurnal rhythm of explosive power: sub-elite athletes with high social jetlag show lower vertical-jump height and lose the expected afternoon superiority in jump performance, and late chronotypes—who characteristically experience chronic social jetlag—achieve lower mean and peak power on the Wingate test and reduced maximal voluntary contraction during morning testing [26]. These neuromuscular deficits are accompanied by endocrine and metabolic disturbances, including a phase shift and blunting of the cortisol awakening response, suppression of nocturnal melatonin, and impaired glucose tolerance, which together provide a plausible physiological substrate for the observed performance impairments [26].

The metabolic dimension of social jetlag compounds these acute effects and carries implications for both training adaptation and long-term health. Chronic circadian misalignment has been associated with impaired glucose tolerance and reduced insulin sensitivity, which may compromise glycogen resynthesis and substrate utilisation during sustained exercise, and with adverse changes in body composition including increased fat mass and body-mass index [26]. At the cellular level, experimental data indicate that social jetlag attenuates the exercise-induced adaptations of mitochondrial biogenesis and oxidative metabolism in cardiac and skeletal muscle, so that training performed during circadian desynchrony may yield a diminished return on investment, with the same training stimulus producing smaller gains in cardiorespiratory fitness [26]. Together with the suppression of the nocturnal melatonin surge, which serves as an endogenous antioxidant, these metabolic disturbances suggest that social jetlag not only impairs performance on the day but may also blunt the physiological adaptations that underpin longer-term athletic development [26].

The most consequential evidence connects social jetlag to injury. In a prospective study of Reserve Officers' Training Corps cadets undertaking time-mandated early-morning physical training, chronotype and social jetlag were assessed alongside prospective injury surveillance and salivary interleukin-6 [28]. Evening chronotypes performed markedly worse on the Army Physical Fitness Test than intermediate and morning types (103.8 versus 221.9 versus 216.6, $p < 0.05$), indicating that maximal exercise performance was compromised when it was not synchronised with circadian preference, although chronotype itself did not significantly predict injury [28]. Social jetlag, by contrast, emerged as a novel behavioural correlate of musculoskeletal injury: cadets who sustained an injury during the training period reported significantly greater social jetlag than those who remained uninjured (2 hours 40 minutes ± 1 hour 3 minutes versus 1 hour 32 minutes ± 55 minutes, $p < 0.05$), and those with more than two hours of social jetlag scored significantly lower on the fitness test [28]. This was, to the authors' knowledge, the first study to link social jetlag to musculoskeletal injury, and it raised the possibility that social jetlag functions as an easily measured behavioural biomarker of injury risk during time-mandated training [28]. Exercise-induced interleukin-6 was so elevated in evening-type cadets that their samples exceeded the assay detection limit, hinting that misaligned morning exercise was more pro-inflammatory in these individuals, consistent with the inflammatory mechanism proposed for the sleep–injury relationship [28]. Extending these observations, the same near-doubling of social jetlag magnitude in injured relative to uninjured individuals has been highlighted as evidence of a dose-dependent relationship between sleep-timing variability and injury vulnerability, mechanistically attributed to reduced neuromuscular power and impaired postural and cognitive control when physical demands are imposed during the biological night [26]. Social jetlag therefore represents a modifiable circadian risk factor that unifies the chronotype and sleep themes of this review and that carries direct implications for the scheduling of training in young athletic and tactical populations.

11. Jet Lag and Transmeridian Travel

Whereas social jetlag arises from schedule–chronotype mismatch, transmeridian jet lag arises from the physical crossing of time zones, and it is an unavoidable feature of international competition. Desynchronisation of the circadian rhythm results from external changes such as transcontinental flights, and because the body maintains its temperature and other rhythms in accordance with the departure time zone, several days are required to adapt and resynchronise to the new local time [1]. Jet lag characteristically follows flights crossing three or more time zones and manifests at onset as fatigue, headache, nausea, decreased concentration, and insomnia, symptoms that gradually fade as the biological clock re-entrains [1]. A widely cited rule of thumb holds that roughly one day is required to restore normal rhythm for each time zone crossed, so that recovery from a six-time-zone transition can take up to around two weeks, and adaptation is generally faster after westward than eastward travel because lengthening the day (phase delay) is physiologically easier than shortening it (phase advance) [1]. Because physical performance is tied to the circadian temperature rhythm, the modification period during which the internal clock is not yet aligned with the destination is a period of impaired readiness, and elite athletes travelling across six to eight time zones have been reported to show weaker grip strength during training and for several days after arrival [1].

Individual factors modulate the speed of adaptation, with younger people generally re-entraining more rapidly than older individuals, and the process can be facilitated by immediately adopting day-to-day routines—sunlight exposure, physical activity, meals, waking, and sleeping—according to local time upon arrival, so as to begin reconstructing the biological clock in the new time zone [1]. Position statements and systematic reviews on managing travel fatigue and jet lag in athletes therefore emphasise advance planning, including preparing the arrival sufficiently early to permit adaptation and, where feasible, undertaking prior training in a time zone aligned with the destination; it has nonetheless been noted that no studies have specifically examined how the timing of sleep during travel affects sleep–wake cycles at the destination, leaving important practical questions unresolved [1].

The most comprehensive recent synthesis, a systematic narrative review of eighty-nine studies of long-haul travel and jet lag in athletes, confirms and quantifies the breadth of these effects [7]. Travel in that review spanned one to twelve time zones in both directions, and the common physiological changes reported included sleep disturbances (in thirty-six studies), body-temperature alterations (eighteen studies), blood-pressure changes, hormonal shifts (nine studies), altered heart-rate variability (four studies), and immune alterations (four studies), while the downstream effects comprised fatigue (twenty-five studies), sleep changes (twenty-one studies), decreased physical performance (eighteen studies), mood changes (fifteen studies), and cognitive impairment (nine studies) [7]. Physical performance outcomes spanned anaerobic power, strength, velocity, aerobic capacity, coordination, and reaction time, and the magnitude of impairment was influenced by travel direction, the number of time zones crossed, and individual susceptibility [7]. Importantly, the review distinguished the two directions:

eastward travel was generally associated with stronger circadian disruption and greater impairment of aerobic capacity, coordination, and technical performance, whereas westward travel more often induced greater fatigue and adversely affected team-sport outcomes [7]. This directional asymmetry is corroborated by analysis of two decades of Major League Baseball data, in which eastward travel exerted a significantly greater negative impact on winning percentage than westward travel, with home teams experiencing an approximate 3.5% decrease in wins, an effect attributed to the greater difficulty of the circadian phase advance required by eastward travel [2]. The physiological basis of this asymmetry lies in the direction of the required phase shift: eastward travel demands an advance of the circadian clock, a process inherently slower than the phase delay required by westward travel, so that adaptation is often estimated at approximately one day per time zone for eastward journeys but roughly half a day per time zone for westward journeys [7,29].

The mechanisms by which circadian misalignment degrades neuromuscular performance are increasingly well delineated. At a physiological level, misalignment depresses core body temperature, dysregulates the cortisol rhythm in a manner that affects arousal and motivation, and reduces sleep quality in ways that hamper motor-memory consolidation and recovery, with the downstream consequences of decreased central drive, impaired motor-unit recruitment, and slower reaction times [29]. Beyond these neuromuscular effects, transmeridian travel perturbs autonomic and cardiovascular regulation—altering heart-rate variability and shifting blood pressure, which tends to rise with westward and fall with eastward travel—and modifies immune and hormonal function, changes that collectively help explain the impairments in strength, power, aerobic capacity, coordination, and reaction time catalogued in the systematic literature [7]. Insomnia and sleep loss during travel additionally compromise mental performance, and the constellation of symptoms may persist for several days, in some reports until around the seventh day after arrival, before rhythms re-entrain [1,29]. The practical implication is that the days immediately following long-haul travel represent a window of reduced readiness during which training intensity and volume, and expectations of competitive performance, should be adjusted accordingly [7,29].

The clinical texture of jet lag in an elite athlete is illustrated by a high-resolution case report of a world-class female Paralympic shot-putter who travelled eastward across six time zones from Chile to France to prepare for a major Games [29]. Daily monitoring over the twenty-one days surrounding the flight revealed transient reductions in handgrip strength for the first three to four days after travel, followed by day-to-day fluctuations, with all observed changes exceeding the smallest worthwhile change threshold [29]. Subjective jet-lag and fatigue scores were elevated in the early post-travel days—peaking at 8 out of 10 in the morning—and progressively declined to near-baseline levels by approximately two weeks before competition, consistent with the one-day-per-time-zone adaptation expected for eastward travel [29]. Throwing performance correlated positively with non-dominant-hand handgrip strength ($\rho = 0.52$) and negatively with session rating of perceived exertion ($\rho = -0.56$), so that simple, field-expedient markers tracked performance readiness through the adaptation period, and gastrointestinal disturbances in stool frequency and consistency persisted longer than the perceptual and neuromuscular symptoms [29]. The persistence of some symptoms after others had resolved underscores that jet-lag recovery does not follow a single uniform timeline and is modulated by travel direction, the specific metric assessed, and the timing of testing relative to the athlete's endogenous rhythm [29].

A frequently overlooked but medically important dimension of long-haul travel is dehydration. The microclimate of an airliner cabin consists of dry, recirculated, cool air maintained at a pressure lower than at sea level, and prolonged exposure to this environment together with the enforced immobilisation of long flights can promote fluid shifts to the lower extremities and changes in blood viscosity that may accelerate dehydration and thereby compromise an athlete's readiness on arrival [30]. Although the evidence base drawn from actual travelling athletes remains limited and provides little direct quantification of the incidence, magnitude, or duration of travel-associated hypohydration, the combination of cabin conditions with the circadian and sleep disruptions of transmeridian travel plausibly compounds the overall performance decrement, and maintaining adequate hydration is a recurrent recommendation for managing jet-lag-related fatigue [1,30]. It has additionally been noted that no studies have specifically examined sleep hygiene in athletes during travel or the impact of sleep timing during travel on sleep–wake cycles at the destination, and that athletes practising sleep hygiene are more likely to recover from travel fatigue after simulated international travel, whereas impractical habits such as exposure to electronic devices can shift circadian phase in the wrong direction—so that the countermeasures reviewed in the following section are of particular relevance to the travelling athlete [1].

12. Countermeasures and Practical Interventions

The recognition that sleep insufficiency, chronotype mismatch, and jet lag are modifiable determinants of performance has prompted the development of a range of behavioural, environmental, and pharmacological countermeasures. Consistent with the wider consensus that non-pharmacological strategies should be prioritised, the interventions with the strongest evidence in athletes are sleep hygiene education, sleep extension, and strategic napping, complemented by light management, melatonin, nutrition, and chronotype-informed scheduling [10].

12.1. Sleep hygiene and education. Basic sleep hygiene—maintaining a regular sleep–wake cycle, creating a dark, quiet, and cool sleep environment, and limiting caffeine and electronic-device use before bed—remains the foundational intervention, and sleep-education sessions have been shown to improve sleep duration and reduce the variability of wake time in elite athletes [10]. A sleep-education programme for athletes should assess habitual sleep patterns, identify common sleep problems, emphasise the role of sleep in performance and recovery, and provide realistic expectations to avoid generating anxiety about night-to-night variation, and it is most effective when tailored to the demands of the specific sport [6]. Practical behavioural adjustments include delaying early-morning training by around thirty minutes to permit additional sleep, encouraging earlier bedtimes, limiting weekend "sleep-in" to within about an hour of the habitual wake time to preserve circadian regularity, and reducing cognitively and emotionally stimulating activity before bed; consistently applied over one to three months, such measures are likely to improve both sleep and performance [6]. Where insomnia is established, cognitive behavioural therapy for insomnia (CBT-I) is the gold-standard non-pharmacological treatment and has been applied to athletes with improvements in sleep quality, with trials indicating that CBT-I can increase sleep time by approximately thirty minutes both subjectively and objectively; its components—stimulus control, sleep restriction, cognitive restructuring, relaxation training, and sleep-hygiene education—address the pre-sleep cognitive arousal implicated in sport-related insomnia [10,31]. Relaxation and mindfulness-based techniques, which reduce pre-sleep hyperarousal and shorten sleep-onset latency, are useful adjuncts [31]. Structured chronotherapy—the timed use of bright light (typically 5,000–10,000 lux) and low-dose melatonin to shift the circadian clock, combined with practical sleep scheduling—has demonstrated benefit for circadian-rhythm sleep problems and is directly applicable to athletes contending with early schedules or travel, morning bright light advancing and appropriately timed evening measures delaying the clock [31].

12.2. Sleep extension. Because chronic sleep restriction is common in athletes, deliberately increasing the sleep opportunity is a logical and well-supported strategy. Extending nightly sleep improves sport-specific skill, reaction time, shooting accuracy, and aerobic performance, and short naps can partly compensate for nocturnal deficits [2]. The landmark demonstration in collegiate basketball players who extended sleep toward ten hours per night for several weeks—yielding faster sprint times and improved shooting accuracy, mood, and daytime alertness—established sleep extension as a practical ergogenic strategy, and even a single night of extension appears beneficial [2,32]. Mechanistically, the benefits of sleep extension are thought to arise from enhanced overnight recovery—greater growth-hormone-mediated tissue repair, more complete clearance of the adenosine that drives sleep pressure, and additional slow-wave and rapid-eye-movement sleep supporting physical and cognitive restoration—so that extending time in bed effectively enlarges the regenerative window rather than merely offsetting an existing deficit [2,32]. In a randomised crossover study, one night of sleep extension increased total sleep time by approximately fifty-five minutes and significantly improved morning (08:00) performance on a five-metre shuttle-run test (best distance 102.8 versus 93.3 metres), reduced the fatigue index, and enhanced squat-jump height, simple reaction time (252.8 versus 296.4 milliseconds), and digit-cancellation performance, with benefits most pronounced in the morning [32]. Notably, sleep extension partially attenuated the normal circadian decline in morning performance, reducing the amplitude of diurnal variation and thereby offering a countermeasure for athletes required to compete or perform cognitively demanding tasks early in the day—precisely the situation that disadvantages late chronotypes [32]. Correlations between the additional sleep obtained and the magnitude of performance gain support a dose–response relationship and reinforce the value of both sleep duration and sleep efficiency [32].

12.3. Strategic napping. Daytime napping is an effective means of supplementing insufficient nocturnal sleep and improving readiness. A controlled comparison of nap durations following a normal night's sleep found that both a forty-minute and a ninety-minute nap opportunity improved maximal voluntary isometric contraction, five-metre shuttle-run distance, and attention while reducing perceived sleepiness, perceived exertion, and muscle soreness, and that the ninety-minute nap was superior—yielding greater gains in strength, total shuttle distance, and attention—likely because its longer duration permits more slow-wave and rapid-eye-movement sleep and greater metabolic recovery [33]. The benefit of napping is, however, task-dependent, and this nuance is clinically important. In athletes who had slept only five hours, a thirty-minute nap significantly reduced subjective sleepiness and improved objectively measured vigilance (pupillographic sleepiness index and Karolinska Sleepiness Scale) but did not improve endurance performance measured by time to exhaustion or maximal oxygen uptake, underscoring that aerobic capacity is a multidimensional construct that napping after mild sleep restriction may not enhance, even as it clearly restores wakefulness and attention [34]. This dissociation is echoed in sleep-deprived combat-sport athletes, in whom a forty-five-minute nap produced superior cognitive recovery—improving decision accuracy by 14.1% and reaction time by 16.1%—but only modest physical gains, such as a 2.4% increase in vertical-jump height, leading to the recommendation that napping be used specifically to protect decision-making during competition involving sleep loss [35]. Post-lunch naps of twenty-five and forty-five minutes have similarly enhanced physical performance and cognitive focus in elite volleyball players, and the timing as well as the duration of the nap influences its efficacy in youth athletes, indicating that nap protocols should be individualised [13,36]. Collectively, the napping literature supports naps of roughly twenty to ninety minutes as a versatile tool, with longer naps favouring physical recovery and even short naps reliably restoring vigilance, while cautioning that endurance performance may be relatively resistant to acute napping [11,33]. The timing of the nap also matters: naps taken in the early post-lunch window, when vigilance naturally dips and the propensity to sleep is high, have proved most effective, and allowing sufficient time after waking for any sleep inertia to dissipate before performance is advised [33].

12.4. Light exposure and circadian management. Because light is the dominant zeitgeber, its strategic use is central to managing chronotype mismatch and jet lag. The amount of light integrated by the circadian clock strongly influences energy metabolism and cycling performance, and a dose–response relationship has been observed between the duration of light exposure before a cycling trial and the work subsequently accomplished, so that variations in the light–dark cycle can alter athletes' metabolic activity, circadian hormonal rhythmicity, and mental performance [1]. Morning bright-light exposure advances the circadian phase and evening light delays it, and reducing exposure to short-wavelength (blue) light in the pre-sleep window preserves the nocturnal melatonin surge; these principles are applied both to shift the phase of peak performance and to accelerate re-entrainment after travel [1,26]. For the travelling athlete, appropriately timed light exposure—morning light to advance the clock for eastward travel and evening light to delay it for westward travel—is a core recommendation, and light was among the interventions most frequently reported in the systematic travel literature alongside melatonin and caffeine [7,26].

12.5. Melatonin and pharmacological approaches. Exogenous melatonin, by virtue of its chronobiotic action, is the pharmacological agent most closely aligned with the circadian mechanisms discussed in this review and is principally used to shift the timing of sleep and to counter jet lag [10]. A Cochrane meta-analysis confirmed that melatonin is effective in preventing and reducing jet lag when travelling across multiple time zones, with short-term use being safe, although sport-specific evidence remains limited: a review of jet-lag management in athletes identified only two small controlled studies of melatonin in elite athletes, with mixed results and methodological limitations, so that its efficacy in athletic settings requires confirmation in larger trials [10]. A systematic review of randomised controlled trials specifically examining melatonin supplementation in highly trained athletes has begun to characterise its effects on sports performance and circulating biomarkers, but the evidence base is still developing [37]. Where pharmacological hypnotics are considered, they should be used with caution: sedative–hypnotics such as the "Z-drugs" and, less preferably, benzodiazepines have been used to advance the sleep phase around travel and competition, but benzodiazepines carry a risk of dependence and residual next-day impairment and are generally to be avoided in favour of behavioural strategies and, where appropriate, melatonin [6]. In the management of established insomnia, the pharmacological armamentarium—benzodiazepines, non-benzodiazepine "Z-drugs" such as zolpidem, dual orexin-receptor antagonists, melatonin-receptor agonists, sedating antidepressants, and first-generation antihistamines—differs in mechanism, duration of action, and dependence potential, and the newer orexin-receptor antagonists and melatonin-receptor agonists are notable for reducing sleep latency with comparatively favourable safety and low dependence risk [31]. Nonetheless, non-pharmacological approaches remain first-line, pharmacological agents should be reserved for selected cases and short durations, and any pharmacological use in athletes must additionally account for anti-doping considerations [10,31].

12.6. Nutrition, hydration, and recovery modalities. Nutritional timing can support circadian adaptation. Protein-rich foods that boost alertness are recommended at breakfast, whereas carbohydrate-rich foods, which promote tryptophan uptake and consequent drowsiness, are better consumed in the evening, and adequate hydration is important to counter the fatigue and dehydration associated with jet lag and long-haul flight [1,30]. Meal timing itself acts as a zeitgeber for peripheral clocks, and misalignment between feeding schedule and circadian rhythm can disturb substrate metabolism, endocrine responses, and sleep architecture, so that aligning meal timing with the destination schedule is a component of travel adaptation [2]. Certain post-exercise recovery modalities also improve subsequent sleep: whole-body cryostimulation has been shown to enhance sleep duration, onset, and efficiency, and evening red-light exposure has produced improvements in subjective and objective sleep and modest performance gains, providing additional tools for athletes with disturbed sleep [6].

12.7. Chronotype-informed scheduling and travel planning. The most direct application of the chronotype literature is the individualisation of training and competition timing. Systematic screening with validated instruments such as the MEQ or the Athlete Sleep Screening Questionnaire allows identification of evening-type athletes at highest risk of circadian misalignment, and for these individuals delaying high-intensity morning sessions by even one to two hours can reduce the magnitude of social jetlag and the associated neuromuscular and cognitive deficits [26]. Because morning-type footballers experience higher perceived exertion during evening training and because athletes who participate in sports matching their chronotype are more likely to excel, practitioners have been advised to avoid early-morning starts for intermediate and evening types and to monitor individual RPE and training-load trends with reference to chronotype and training time [23]. High-intensity interval exercise in particular should be scheduled according to the athlete's chronotype to minimise fatigue and mood disturbance [1]. For competition at a fixed, non-optimal time, targeted warm-up can partially compensate: specific, sport-relevant warm-up protocols that raise body temperature have been shown to improve evening performance on judo-specific fitness testing, illustrating how the temperature-dependent component of the performance rhythm can be manipulated acutely [1]. For transmeridian travel, planning in advance is essential: gradually shifting sleep timing by thirty to sixty minutes per day before departure with appropriately timed light exposure, adjusting sleep, light, meals, and training to the destination time zone on arrival, allowing a minimum of around twenty-four hours of recovery, and using naps of twenty to ninety minutes to support physical and cognitive performance together constitute a coherent pre- and post-travel protocol [1,2]. Given the individual variability in adaptation, individualised monitoring with simple, reliable field markers such as handgrip strength and session RPE has been recommended to guide training decisions during the travel-adaptation period [29].

13. Individual Moderators and Special Populations

The magnitude of sleep, chronotype, and jet-lag effects is not uniform across athletes but is moderated by age, sex, and disability, and attention to these moderators refines the clinical picture.

Age is a powerful moderator operating through the developmental trajectory of the circadian clock. Chronotype is strongly age-dependent: children tend toward morningness, adolescents drift toward eveningness and reach their peak of lateness around the age of twenty before a slow, lifelong return toward earlier preferences [22]. This adolescent eveningness collides with early school and training schedules to produce chronic sleep restriction and social jetlag precisely in a population whose sleep need is elevated—approximately nine to ten hours—to support neurodevelopment, motor learning, and adaptation to rising training loads [2]. Consistent with this, objective data show that young athletes have the lowest sleep efficiency of any age group, around 80%, and that sleep insufficiency in young athletes is linked to higher injury rates, marking adolescents and student-athletes as a priority group [5]. The start of the academic year, when training, academic, and social stressors converge, is an especially vulnerable period for the sleep of university athletes [13].

Sex differences, though less consistently studied, are discernible across the sleep and circadian domains. Objective sleep data indicate that female athletes may obtain slightly better sleep than males, with higher sleep efficiency (approximately 89% versus 85%) and longer total sleep time, and field data show females sleeping somewhat longer on certain days of the week [5,13]. In the circadian domain, the inverse association between social jetlag and cardiorespiratory fitness has been reported to reach significance in males but not females, suggesting a sex-dependent susceptibility possibly related to differences in hormonal regulation or physical-activity patterns [26]. However, sleep-architecture data specific to female athletes remain sparse—several datasets could not analyse sex at all—so conclusions about sex as a moderator must remain provisional and represent a clear priority for future research [5].

Athletes with disability constitute a further special population in whom circadian disruption may be amplified. In the detailed case of a world-class Paralympic shot-putter with a spinal cord injury, disability-specific factors including altered thermoregulation, modified body composition, autonomic dysregulation, and potential disruption of circadian entrainment were proposed to heighten susceptibility to jet lag and travel fatigue, and the athlete indeed showed transient neuromuscular and gastrointestinal disturbance after eastward travel [29]. Because such physiological particularities may modify both the magnitude and the time course of circadian disruption, individualised monitoring is especially important in Para-athletes, and the incorporation of chronotype assessment into the management of this population has been explicitly recommended [29]. Across all these subgroups, the recurring message is that the effects reviewed here are real at the group level but individually variable, so that assessment of chronotype, sleep, and travel response at the level of the individual athlete is the logical foundation for effective management.

14. Discussion

The evidence synthesised in this review supports a coherent central thesis: sleep, chronotype, and jet lag are not three separate topics but three expressions of a single circadian and homeostatic biology that, through its regulation of core body temperature, hormone secretion, neuromuscular function, and cognition, exerts a measurable and clinically relevant influence on athletic performance and health. Sleep insufficiency—habitual in athletes, who average fewer than eight hours of sleep with reduced efficiency and among whom a quarter to two-thirds screen as poor sleepers—degrades physical performance by an average of roughly 7.6% and impairs cognition, recovery, immunity, and injury resistance [5,11,12,17,19]. Chronotype systematically shifts the phase of both peak performance and lowest perceived exertion, with the interval since entrained awakening emerging as a more reliable predictor of the optimal performance window than absolute clock time, and with late chronotypes showing the greatest diurnal variability and the greatest morning impairment [1,8]. Social jetlag and transmeridian travel, the chronic and acute forms of circadian misalignment respectively, impair strength, power, aerobic capacity, coordination, and cognition and are associated with elevated injury risk, the effects of transmeridian travel being asymmetric with eastward journeys generally more disruptive [7,26,28]. That these domains share a common substrate is not merely conceptual: the same inflammatory, neuromuscular, and endocrine mechanisms recur across the sleep-loss, social-jetlag, and travel literatures, and the same countermeasures—light, melatonin, napping, sleep extension, and schedule adjustment—apply across all three [7,10,21,26]. The principal primary studies underpinning this synthesis, spanning the sleep, chronotype, social-jetlag, and travel domains, are summarised in Table 1.

A central interpretive theme is the task-dependence of these effects, which explains much of the apparent inconsistency in the literature. Chronotype and time-of-day effects are most reliably demonstrated in individual, precisely timed, and metabolically demanding tasks—rowing, swimming time trials, repeated shuttle runs, and six-minute runs—and are most easily obscured in team sports, single-time-point designs, and coarse in-game performance measures [1,22]. The null finding for chronotype in evening basketball competition is best read not as evidence against a chronotype effect but as a consequence of a design that could not detect a morning–evening interaction, whereas the strong effects in rowing and swimming reflect sensitive, individual, timed measures [1]. Similarly, the dissociation whereby aerobic and endurance qualities are affected while maximal strength is relatively spared—seen in the partial-sleep-deprivation, chronotype-soccer, and napping-endurance studies alike—suggests that longer, more metabolically demanding efforts better expose the underlying circadian and homeostatic differences than brief explosive tasks [9,22,34]. The ultra-endurance context adds a further layer of complexity: a systematic review found that in ultra-endurance events the relationship between sleep and performance may be non-linear and even paradoxical, with better physical performance sometimes associated with less sleep during competition and with pre-race and intra-race napping strategies benefiting performance, indicating that context and individual strategy modulate the sleep–performance relationship in ways that simple dose–response models cannot capture [38]. This heterogeneity of task, context, and phenotype is the single most important reason that firm, universal conclusions remain elusive.

Beyond physical performance, the medical significance of sleep and circadian health in athletes extends into mental health, forming a bidirectional loop of considerable clinical importance. Sleep disturbance and depression are reciprocally linked: insomnia is a consistent predictor of subsequent depression while depression degrades sleep, and shared mechanisms including rapid-eye-movement sleep instability, circadian misalignment, and emotional dysregulation underlie the association, with physical activity capable of improving both poles of the cycle [39]. This is directly relevant to athletes, in whom the pre-sleep cognitive arousal that mediates sport-related insomnia overlaps with the anxiety and stress of competition [16,39]. Among undergraduate sports students specifically, psychological problems including competition anxiety, career anxiety, and sleep disturbance are prevalent and interrelated, with sleep quality acting as a mediating variable through which training stress is translated into psychological outcomes [40]. The bidirectionality of the sleep–exercise relationship also opens a therapeutic avenue, since physical activity is an effective, low-cost intervention for anxiety and mood—mind–body modalities such as yoga being particularly effective—and structured physical and mind–body activity has been shown to improve sleep quality as a non-pharmacological strategy, albeit demonstrated most robustly outside elite-athlete populations [4,41,42]. These findings situate the management of sleep and circadian alignment within a broader model of athlete health in which physical performance, recovery, immunity, and mental well-being are interdependent [4].

Several methodological limitations temper the strength of the conclusions and were candidly acknowledged within the primary literature itself. Many studies employ small samples without reported power calculations, and measurement methods vary substantially between studies—subjective questionnaires versus objective actigraphy or polysomnography for sleep, and different chronotype instruments and performance tests—so that direct comparison and synthesis are difficult [1,5]. Because self-reported sleep tends to overestimate objectively measured sleep, and because some athletes report poor sleep while objective measures indicate sufficiency, the choice of measurement approach materially affects conclusions [4]. The diurnal times selected for performance testing differ between studies, which can itself determine whether a chronotype effect is detected, and the interaction of age, sex, sport type, and training intensity with sleep and circadian variables remains insufficiently characterised, with several datasets unable to analyse sex differences at all [1,5]. Sample compositions are frequently skewed toward particular sports, levels, and geographic regions, limiting generalisability, and the jet-lag and melatonin literatures in particular contain few high-quality controlled studies in genuine elite-athlete populations [10]. These constraints indicate that the present conclusions should be read as a synthesis of converging but imperfect evidence rather than as definitive proof, and they define a clear agenda for methodologically rigorous future work.

Notwithstanding these limitations, the practical direction of the evidence is consistent and actionable. The convergence of the chronotype, social-jetlag, and travel literatures on the concept of circadian alignment—and on the value of the interval since awakening as a predictor of readiness—suggests that individualised, chronotype-informed management of training and competition timing, structured sleep optimisation, and deliberate travel and light protocols should be integrated into routine athlete care rather than reserved for exceptional circumstances [8,26]. The recurring finding that morning and mid-to-late-adolescent athletes, evening chronotypes, and travelling athletes constitute higher-risk subgroups provides a rational basis for targeting limited resources [17,26,28].

The applied stakes of this evidence are best appreciated in the context of elite competition, where winning margins are frequently minute: differences of well under one percent can decide finals, and an improvement of a fraction of a second can separate a medal from no medal [8]. Against a backdrop in which aerobic performance can vary by as much as 26% across the day and even simple measures of physical performance by around 10%, the deliberate alignment of training and competition timing with an athlete's circadian phenotype represents a rational, legitimate, and non-pharmacological route to the marginal gains that elite sport relentlessly pursues [1,8]. That circadian rhythms have been proposed as a potential new frontier in athletic performance reflects precisely this recognition that a physiological dimension long treated as background variability may, when systematically managed, yield a genuine competitive edge [3]. At the same time, the modesty and task-specificity of many of the reported effects counsel against overstatement: circadian and sleep interventions should be positioned as one component of a comprehensive, individualised performance-and-health strategy rather than as a panacea [3].

15. Conclusions

On the basis of the evidence reviewed, the following principal conclusions are offered.

First, sleep is a modifiable determinant of athletic performance and health whose habitual insufficiency in athletes measurably impairs physical and cognitive performance, recovery, immunity, and injury resistance; acute sleep loss reduces performance by approximately 7.6% overall, with afternoon and evening tasks and endurance qualities most affected, and injury risk rising progressively as sleep falls below eight hours per night.

Second, chronotype systematically modulates the diurnal variation of performance and perceived exertion: morning types perform best and feel least exertion earlier in the day and evening types later, late chronotypes exhibit the largest diurnal swings and the greatest morning impairment, and the time elapsed since entrained awakening predicts the timing of peak performance more reliably than absolute clock time.

Third, the influence of chronotype is task-dependent, being most robustly demonstrated in individual, precisely timed, and metabolically demanding efforts and most easily obscured in team sports, coarse measures, and single-time-point designs—an interpretive principle that reconciles much of the apparently conflicting evidence.

Fourth, chronic circadian misalignment in the form of social jetlag and acute misalignment in the form of transmeridian jet lag impair strength, power, aerobic capacity, coordination, and cognition and are associated with elevated musculoskeletal injury risk, with eastward travel generally more disruptive than westward travel and with adaptation requiring approximately one day per time zone crossed.

Fifth, evidence-based countermeasures—sleep hygiene education, sleep extension, strategic napping, light management, appropriately timed melatonin, nutritional and hydration strategies, and chronotype-informed scheduling—are available and effective, though their benefits are partly task-specific, and non-pharmacological approaches should be prioritised.

Sixth, the field remains constrained by small samples, heterogeneous measurement, variable test timing, and under-representation of certain sports, sexes, and populations, so that chronotype-informed and travel-related recommendations, while directionally sound, require confirmation in larger and more rigorously designed studies.

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