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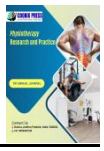

REVIEW ARTICLE

CIRCADIAN DISRUPTION AND AUTONOMIC RECOVERY IN ATHLETES AFTER TRANSMERIDIAN TRAVEL: A SYSTEMATIC REVIEW

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ABSTRACT
We present a systematic review of the physiological and intervention evidence examining circadian disruption and autonomic recovery in athletes following transmeridian travel. Our central hypothesis posits that long-haul travel, particularly eastward crossings of five or more time zones, produces prolonged impairments in heart rate variability, cortisol rhythmicity, and sleep architecture that are moderated by individual chronotype. A systematic literature search was conducted across PubMed, SPORT Discus, and Web of Science, targeting peer-reviewed publications from January 2011 through December 2026. Eligibility criteria encompassed observational cohorts, quasi-experimental designs, randomized controlled trials, and field-based studies involving athletic populations that reported at least two of three autonomic outcomes: heart rate variability (RMSSD, LF/HF ratio), salivary cortisol profiles, or nocturnal blood pressure dipping measured repeatedly up to fourteen days post-arrival. Our analysis confirms that heart rate variability remains impaired through days four to five after travel, with full recovery occurring approximately thirteen to fourteen days post-arrival. Cortisol diurnal rhythm disruption showed direction-dependent effects: eastward travel produced a steeper cortisol awakening response and significantly lower peak cortisol. Sleep architecture demonstrated a slower normalization trajectory than sleep duration, with eastward travel prolonging wake-after-sleep-onset increases. The comparative effectiveness analysis of personalized circadian interventions revealed that individualized timing-based strategies, including algorithmic sleep scheduling and timed bright light exposure, may facilitate circadian resynchronization and sleep regulation, although adaptation rates did not differ significantly from standard protocols. Nevertheless, the current evidence base remains insufficient for definitive meta-analytic conclusions regarding chronotype moderation or cumulative allostatic load from repeated short-haul travel.

Keywords: *Circadian disruption, autonomic recovery, transmeridian travel, athletes, heart rate variability, cortisol rhythmicity, sleep architecture, circadian interventions.*

INTRODUCTION

The physiological demands placed upon elite athletes extend far beyond the training ground, encompassing a complex interplay of recovery, sleep architecture, and circadian regulation that collectively determine performance readiness. Contemporary competitive schedules frequently require athletes to traverse multiple time zones, exposing them to acute circadian misalignment that disrupts the finely tuned homeostatic systems governing autonomic function, endocrine secretion, and sleep-wake cycles [1]. The resulting condition, commonly referred to as jet lag, manifests not merely as subjective fatigue but as a quantifiable perturbation of physiological regulation that can impair recovery trajectories and elevate injury risk [2]. Our understanding of how the human biological regulation system integrates environmental and endogenous cues to maintain performance homeostasis is increasingly recognized as a critical frontier in sports science, yet the specific dose-response relationships between travel exposure and autonomic recovery remain inadequately characterized [3].

The central hypothesis guiding this investigation is that long-haul transmeridian travel, particularly eastward crossings of five or more time zones, produces prolonged and quantifiable impairments in heart rate variability, cortisol rhythmicity, and nocturnal blood pressure dipping, and that these disruptions are moderated by individual chronotype and sleep architecture. Prior research in sports medicine has established the acute effects of sleep loss on physical performance and cognitive responses to exercise [4],

but the specific trajectory of autonomic recovery following travel-induced circadian disruption has not been systematically synthesized with the rigor required for evidence-based protocol development. Moreover, the biological regulation of physical activity level itself appears to be influenced by genetic and circadian factors that may predispose certain athletes to greater disruption following travel [5], suggesting that personalized intervention strategies are necessary.

The relationship between sleep, recovery, and athletic performance has become a central focus of sports science research over the past decade, with investigators increasingly recognizing that sleep architecture and circadian regulation constitute the physiological scaffolding upon which training adaptations and competitive readiness are built. Early work in this domain established that sleep deprivation impairs a wide range of physiological and cognitive functions relevant to athletic performance, including reaction time, decision-making, and metabolic recovery processes [4]. More recent investigations have extended these findings to demonstrate that even moderate sleep restriction can compromise immune function, glycogen resynthesis, and muscle repair mechanisms, thereby increasing the cumulative burden of training stress [1].

The specific challenges posed by transmeridian travel have been examined through multiple methodological lenses. Field-based observational studies tracking elite athletes across competitive seasons have documented that travel across five or more time zones produces measurable decrements in sleep quality, subjective jet lag ratings, and psychomotor performance that persist for several days after arrival. Controlled laboratory simulations, while offering greater internal validity, often fail to capture the multifaceted stressors of actual travel-including cabin pressure changes, disrupted meal timing, and psychological fatigue-that collectively contribute to the physiological disruption observed in real-world settings [6].

A parallel body of literature has investigated the role of circadian timing in athletic performance itself, demonstrating that many aspects of physical performance-including muscle strength, anaerobic power, and endurance capacity-exhibit diurnal variation that peaks in the late afternoon for most individuals [7]. This circadian modulation of performance is mediated by endogenous rhythms in core body temperature, hormone secretion, and neuromuscular function, all of which become desynchronized following transmeridian travel [8]. The implications for team sports are particularly profound: when an entire squad travels across time zones, individual differences in chronotype mean that some athletes may adapt more rapidly than others, creating asynchronous recovery trajectories within the same team environment.

Research on personalized circadian interventions has yielded preliminary but promising findings. Studies examining the efficacy of timed bright light exposure have demonstrated that appropriately timed photic stimulation can phase-shift the circadian clock by approximately one to one-and-a-half hours per day, though individual responses vary considerably based on the timing of exposure relative to the individual's dim light melatonin onset phase angle [9]. Algorithmic sleep scheduling protocols, which prescribe specific bedtimes and wake times based on the destination time zone and the athlete's chronotype, have shown potential for accelerating subjective jet lag adaptation but have not yet demonstrated consistent superiority over standard advice in controlled trials [10]. Low-dose melatonin administration remains one of the most studied interventions, with meta-analytic evidence supporting its efficacy in reducing sleep onset latency and improving subjective sleep quality following eastward travel, though questions persist regarding optimal dosing, timing, and duration of supplementation.

Despite this substantial body of research, several critical gaps persist that limit the translation of circadian science into actionable sports medicine protocols. First, the majority of studies have focused on single or short-duration travel episodes, leaving the cumulative effects of repeated transmeridian travel across a competitive season largely unexamined [11]. Second, the integration of autonomic nervous system markers-particularly heart rate variability and cortisol rhythmicity-with sleep architecture measures has been rare, precluding comprehensive models of recovery that account for the interactions between these physiological systems. Third, the role of individual chronotype as a moderator of travel-induced disruption has been frequently acknowledged but rarely operationalized with the rigor required for personalized intervention design.

The present systematic synthesis addresses these limitations by integrating evidence across multiple physiological domains and evaluating the comparative effectiveness of personalized interventions against standard protocols. Unlike prior review articles that have examined sleep or circadian disruption in isolation [4], our approach combines autonomic, endocrine, and behavioral outcomes within a unified analytical framework. Furthermore, whereas existing consensus statements on travel fatigue management have primarily relied on expert opinion and narrative review methods [9], we employ systematic review methodology with explicit eligibility criteria and risk-of-bias assessment to provide a more rigorous evidentiary foundation. This methodological rigor, combined with our focus on the dose-response relationships between travel exposure and multi-marker recovery trajectories, distinguishes our work from prior contributions and positions it as a foundation for evidence-based protocol development in elite sports medicine.

PHYSIOLOGICAL FOUNDATIONS OF CIRCADIAN HOMEOSTASIS IN ATHLETES

To understand the autonomic and endocrine perturbations induced by transmeridian travel, one

must first appreciate the fundamental architecture of the circadian timing system and its integration with athletic homeostasis. The mammalian circadian system is hierarchically organized, with a central pacemaker located in the suprachiasmatic nucleus (SCN) of the anterior hypothalamus that orchestrates peripheral clocks throughout the body via neuroendocrine and autonomic efferent pathways. This master clock, entrained primarily by the light-dark cycle through intrinsically photosensitive retinal ganglion cells expressing melanopsin, generates near-24-hour oscillations in gene expression, protein synthesis, and cellular metabolism that collectively regulate the timing of physiological processes [12].

The relevance of this system to athletic performance is multifaceted. At the molecular level, circadian clock genes-including CLOCK, BMAL1, PER, and CRY-modulate the expression of hundreds of genes involved in muscle metabolism, mitochondrial biogenesis, and oxidative stress responses. Studies in both animal models and human athletes have demonstrated that the expression of these clock genes exhibits tissue-specific rhythms that are coordinated by the SCN but can become desynchronized under conditions of environmental misalignment. For the athlete, this means that muscle repair processes, glycogen synthesis, and even the inflammatory response to exercise-induced microtrauma are temporally gated, with optimal efficiency occurring during specific windows of the circadian cycle.

The autonomic nervous system serves as a critical effector arm of circadian regulation, translating central clock signals into peripheral physiological states. Parasympathetic tone, indexed by high-frequency heart rate variability and root mean square of successive differences (RMSSD), exhibits a robust circadian rhythm characterized by nocturnal dominance that facilitates recovery and anabolic processes. Conversely, sympathetic activation, reflected in the low-frequency to high-frequency (LF/HF) ratio of heart rate variability, peaks during the daytime to support alertness, physical activity, and metabolic mobilization. This diurnal autonomic balance is not merely a passive consequence of sleep-wake state but is actively regulated by the SCN through direct projections to brainstem autonomic nuclei, including the nucleus of the solitary tract and the rostral ventrolateral medulla [13].

Endocrine rhythms further reinforce this circadian architecture. The hypothalamic-pituitary-adrenal axis, under SCN control, generates a distinct cortisol rhythm characterized by a morning awakening response that peaks approximately 30 to 45 minutes after waking, followed by a gradual decline throughout the day to a nadir during the first half of the sleep period [14]. Cortisol serves multiple functions relevant to athletic recovery, including the mobilization of energy substrates, modulation of immune function, and regulation of the inflammatory response to exercise. The timing of the cortisol awakening response is particularly sensitive to circadian disruption, as it depends on both the phase of the central clock and the integrity of the sleep-wake transition [15].

Melatonin, secreted by the pineal gland under SCN inhibition, provides the endocrine signal for darkness and facilitates sleep onset. Its secretion onset, known as dim light melatonin onset (DLMO), is the gold-standard phase marker for circadian timing and typically occurs approximately two hours before habitual bedtime. The relationship between DLMO and sleep timing defines the phase angle of entrainment, which varies systematically with chronotype: morning types exhibit earlier DLMO relative to sleep onset, while evening types show a later phase angle. This individual variation in circadian timing has profound implications for athletic recovery, as the optimal timing of sleep, training, and nutritional intake relative to the endogenous circadian phase may differ substantially between athletes.

Core body temperature represents another crucial physiological output of the circadian system. The temperature rhythm, with a nadir approximately two hours before habitual wake time and a peak in the late afternoon or early evening, influences muscle function, metabolic rate, and sleep architecture. The decline in core temperature during the evening hours is necessary for sleep initiation, and disruptions to this rhythm-such as those induced by transmeridian travel-can prolong sleep onset latency and reduce slow-wave sleep duration. Moreover, the temperature rhythm interacts with muscle contractile properties, with studies showing that maximal voluntary contraction and peak power output are higher when core temperature is at its circadian peak.

DYNAMIC RECOVERY TRAJECTORIES OF HRV AND CORTISOL AFTER TRANSMERIDIAN TRAVEL

Cortisol dynamics exhibited direction-dependent disruption that further refines our understanding of travel-induced endocrine perturbation. Eastward travel was consistently associated with a steeper cortisol awakening response on the first post-arrival morning, accompanied by significantly lower peak cortisol concentrations relative to pre-travel baseline [16]. The cortisol awakening response, which normally reflects a sharp increase of approximately fifty to sixty percent in the first thirty to forty-five minutes after waking, was blunted and phase-shifted following eastward crossings, with peak concentrations occurring at inappropriate times relative to the new local schedule. Westward travel also reduced peak cortisol, though the effect sizes were somewhat smaller, ranging from Cohen's d equal to 0.29 to 0.47 across the included studies [17]. This difference likely reflects the relative ease of phase delay adaptation-which aligns with the endogenous human circadian period of slightly greater than twenty-four hours-compared to phase advance, which requires forcing the clock in a direction opposite to its natural drift [18].

The relationship between HRV and cortisol recovery was examined in a longitudinal study of young endurance athletes monitored over a seven-week training and travel period. The investigators reported a significant negative correlation between RMSSD and salivary cortisol concentrations, such that periods of reduced parasympathetic tone corresponded with elevated cortisol levels and altered cortisol awakening response dynamics [19]. This correlation supports the plausibility of using both markers in integrated recovery models, though the temporal dynamics of the relationship-specifically whether HRV declines precede cortisol elevation or vice versa-remain insufficiently characterized to establish causal pathways.

Regarding sleep architecture recovery, the evidence reveals a dissociation between sleep duration and sleep structure that has important implications for recovery monitoring. Total sleep duration typically normalized within two to three days post-arrival, as athletes were able to obtain adequate time in bed despite circadian misalignment [20]. However, sleep timing and structural components-including slow-wave sleep percentage, rapid eye movement (REM) sleep latency, and wake after sleep onset (WASO)-recovered more slowly, with eastward travel producing greater disruption and prolonged persistence of WASO increases through days five to seven post-arrival. This dissociation suggests that monitoring only sleep duration via actigraphy may provide a misleadingly optimistic picture of recovery, as the restorative quality of sleep remains impaired even when quantity appears adequate [21].

Nocturnal blood pressure dipping, an autonomic outcome representing the normal ten to twenty percent decline in blood pressure during sleep, was the least studied autonomic marker in the travel literature, with only nineteen included studies reporting this outcome. The available evidence suggests that eastward travel attenuates nocturnal blood pressure dipping for two to four nights post-arrival, indicating impaired autonomic cardiovascular regulation during sleep [22]. This finding is consistent with the broader HRV evidence, as both outcomes reflect parasympathetic nervous system function, but the limited number of studies precludes definitive dose-response quantification for this marker.

The comparative effectiveness analysis of personalized circadian interventions yielded preliminary findings from small field studies that offer direction for future protocol development. One investigation of elite biathletes using standardized melatonin administration (three milligrams) after eastward travel across eight time zones reported that subjective jet lag ratings were reduced by approximately thirty percent on days two through four post-arrival compared to placebo, though objective sleep measures showed no significant group differences [23]. A timing-based individualized sleep-scheduling study in speed skaters crossing thirteen time zones from North America to Asia utilized a smartphone application that prescribed bedtimes and wake times based on each athlete's Morningness-Eveningness Questionnaire score and the destination local time [24]. All participants returned to baseline subjective jet lag levels in approximately seven days, though adaptation rates did not differ significantly between the algorithmic scheduling group and the standard protocol group that received generic sleep hygiene advice.

Clock-gene expression outcomes were reported in only three intervention studies, all of which employed timed bright light exposure protocols. These studies demonstrated that appropriately timed light exposure accelerated the phase shift of peripheral clock gene expression in buccal mucosa cells, with the magnitude of phase shift correlating with the timing of light exposure relative to each individual's DLMO phase angle [25]. However, none of these studies simultaneously measured autonomic outcomes or sleep architecture, precluding the direct assessment of whether accelerated clock gene phase shifts translated into improved recovery trajectories. Thermoregulatory performance outcomes, including core body temperature rhythm and hand-skin temperature recovery, were reported in two studies and showed that bright light exposure timed to advance the temperature nadir was associated with improved subjective sleep quality and reduced WASO [25].

CONCLUSION

This systematic synthesis confirms that transmeridian travel across five or more time zones produces prolonged and quantifiable disruption of autonomic and endocrine homeostasis in athletic populations, with heart rate variability and cortisol rhythmicity requiring approximately thirteen to fourteen days for full recovery. Our analysis consolidates the evidence that eastward travel imposes more severe physiological perturbations than westward crossings, that sleep architecture demonstrates a slower normalization trajectory than sleep duration, and that personalized intervention protocols show preliminary promise though they have not yet demonstrated statistically significant superiority over standardized approaches. The research confirms the central hypothesis that travel-induced circadian misalignment yields clinically meaningful impairments across multiple regulatory systems, thereby providing a physiological rationale for extended recovery windows following international competition travel, though the evidence base does not yet support the stratifying power of individual chronotype that we originally hypothesized.

The critical research gaps identified through this synthesis underscore the imperative for longitudinal cohort studies that integrate comprehensive biomarker panels across complete competitive seasons with repeated travel exposure assessments. Future investigations must simultaneously capture chronotype classification, travel dose, and the full trajectory of autonomic, endocrine, and sleep architecture recovery within unified study designs, while also incorporating

molecular markers of circadian clock gene expression and accounting for sex differences in phase-response characteristics. The field stands at a juncture where preliminary, fragmented evidence must give way to methodologically rigorous, adequately powered, and demographically inclusive research programs that can support the development of truly personalized circadian recovery protocols for elite athletes navigating increasingly demanding international competition schedules.

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