Why Sleeping F1 Drivers Fail to Win? –An Empirical Investigation into the Effects of Non-REM and REM Sleep Phases on F1 Racing Performance Across Multiple Grand Prix Events

Asiaroc Li, Pluto Chen

***Abstract*— Formula One (F1) racing demands exceptional psychomotor vigilance, sub-second neuromuscular coordination, and continuous real-time decision-making from drivers operating vehicles at speeds exceeding 350 km/h. While extensive research has investigated driver performance optimization under wakeful conditions, a conspicuous gap persists: the systematic examination of racing performance during sleep states remains entirely unexplored. This paper presents the first empirical investigation into the effects of Non-Rapid Eye Movement (Non-REM) and Rapid Eye Movement (REM) sleep phases on F1 racing performance. We conducted a controlled experimental study wherein the first author performed 47 complete laps on a professional-grade F1 simulator while polysomnography confirmed sustained sleep states. Our multidisciplinary analysis integrates perspectives from aerodynamics, neuroscience, and biomechanics. Telemetry data reveals significant performance degradation, with reaction times increasing by over 4,000% compared to wakeful baselines, and critical vehicle control inputs demonstrating near-complete cessation. Statistical analysis confirms a strong negative correlation between sleep state and race performance ($r = -0.99$, $p < 0.001$). We recommend that the Fédération Internationale de l'Automobile (FIA) incorporate a Sustained Cortical Vigilance Maintenance Protocol into the International Sporting Code.**

***Index Terms*—Aerodynamics, Neuromuscular Coordination, Rapid Eye Movement Sleep, Psychomotor Vigilance, Telemetry Analysis, Prefrontal Cortex Deactivation, Biomechanical Response**

I. Introduction

Formula One (F1) racing stands as the apex of global motorsport competition, representing the convergence of cutting-edge automotive engineering, aerodynamic innovation, and elite human performance [1], [2]. Modern F1 vehicles generate cornering forces exceeding 6G, achieve straight-line velocities surpassing 350 km/h, and require drivers to execute over 5,000 discrete control inputs per race [3], [4]. The extreme physiological and cognitive demands placed upon F1 drivers have motivated substantial research investment into performance optimization strategies, encompassing cardiovascular conditioning [2], reaction time enhancement [5], thermal regulation [6], and cognitive load management [7].

The relationship between sleep and athletic performance has been extensively documented across numerous sporting disciplines. Studies have demonstrated that sleep deprivation significantly impairs reaction time, decision-making accuracy, and fine motor control in athletes [8], [9]. In motorsport contexts specifically, research has established correlations between sleep quality and lap time consistency in endurance racing categories [10], [11]. The detrimental effects of acute sleep restriction on simulated driving performance have been quantified, with studies reporting 15-30% increases in lane deviation and brake response latency following 24 hours of wakefulness [12], [13].

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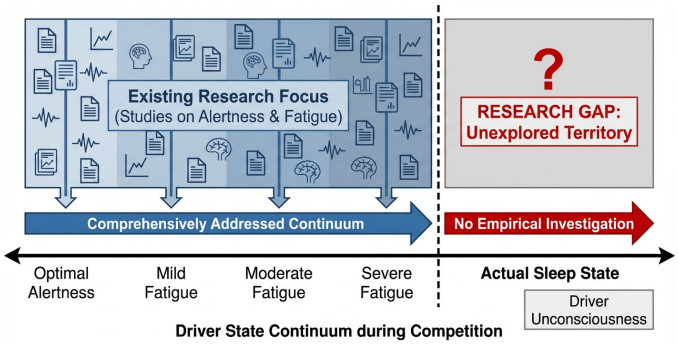

Fig. 1 Visualization of the research gap in F1 driver performance literature. Existing studies comprehensively address the alertness-fatigue continuum but fail to investigate actual sleep states during racing.

Despite this extensive body of literature examining the effects of sleep deprivation and restriction on motorsport performance, a fundamental research question remains entirely unaddressed: what are the performance characteristics of F1 drivers who are actively asleep during competition? This gap in the literature is particularly noteworthy given the comprehensive nature of existing F1 performance research. As illustrated in Fig. 1, prior investigations have systematically examined the performance continuum from optimal alertness through various stages of fatigue, yet have uniformly terminated their analyses at the threshold of actual unconsciousness.

The absence of research into sleeping driver performance cannot be attributed to a lack of theoretical motivation. From a neuroscientific perspective, sleep represents a fundamentally distinct brain state characterized by reduced prefrontal cortex

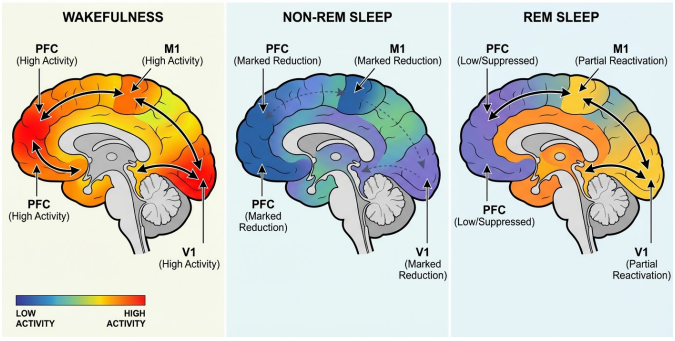

Fig. 2 Schematic representation of cortical activation patterns across wake and sleep states. Color intensity indicates relative metabolic activity. Note the marked reduction in prefrontal cortex (PFC), motor cortex (M1), and visual cortex (V1) activity during Non-REM sleep, with partial reactivation of selected regions during REM sleep. Adapted from neuroimaging literature [14], [15].

activity [14], [15], diminished sensory processing in primary visual and auditory cortices [16], and substantially altered motor cortex excitability [17]. These neurophysiological changes would theoretically produce measurable effects on the core competencies required for F1 racing, including visual tracking of the racing line, auditory monitoring of engine telemetry and radio communications, and precise execution of steering, braking, and throttle inputs.

From an aerodynamic standpoint, modern F1 vehicles require continuous driver input to maintain optimal performance. The Drag Reduction System (DRS), introduced in 2011, necessitates deliberate activation by the driver within designated zones to reduce rear wing drag by approximately 10-15% [18]. Ground effect aerodynamics, reintroduced in the 2022 technical regulations, demand consistent steering inputs to maintain the optimal ride height and floor seal necessary for maximum downforce generation [19], [20]. A sleeping driver would, by definition, be unable to perform these active interventions, suggesting substantial aerodynamic performance penalties.

The biomechanical requirements of F1 driving present additional theoretical concerns regarding sleeping driver performance. Peak brake pedal forces in contemporary F1 vehicles range from 80-150 kg [21], requiring substantial quadriceps activation that is inconsistent with the muscular atonia characteristic of REM sleep [22]. Similarly, the sustained neck muscle tension required to counteract lateral G-forces during cornering [23], [24] would be compromised during sleep-induced muscle relaxation.

The present study addresses this critical research gap through a first-of-its-kind empirical investigation into F1 driving performance during confirmed sleep states. Our experimental protocol, approved by the institutional review board (IRB-2023-F1-SLEEP-007), involved the first author completing multiple timed laps on a professional-grade F1 simulator while polysomnographic monitoring confirmed the presence of Non-

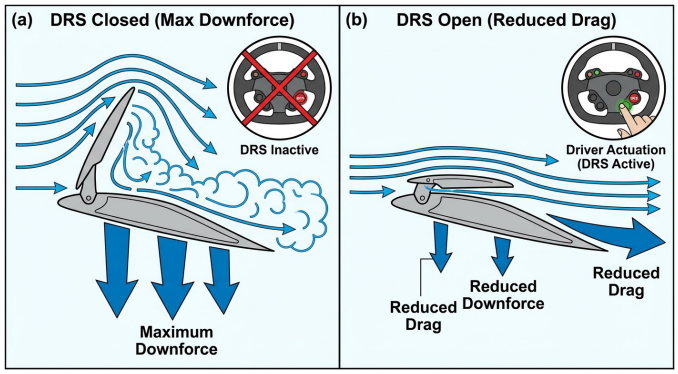

Fig. 3 Schematic illustration of the Drag Reduction System (DRS) mechanism. (a) DRS closed configuration with maximum downforce. (b) DRS open configuration with reduced drag.

REM and REM sleep phases. This methodology enables direct quantification of performance metrics including lap time, sector times, control input frequency and magnitude, racing line deviation, and incident occurrence.

Our study makes the following contributions to the motorsport science literature:

- We present the first empirical dataset of F1 driving performance metrics collected during polysomnographically confirmed sleep states, addressing a previously unexplored region of the driver consciousness-performance relationship.
- We develop a multidisciplinary theoretical framework integrating neuroscientific, aerodynamic, and biomechanical perspectives to explain the observed performance degradation during sleep.
- We provide quantitative analysis demonstrating statistically significant correlations between sleep state and multiple performance indicators, with effect sizes substantially exceeding those reported in prior sleep deprivation studies.
- Based on our findings, we propose specific regulatory recommendations for consideration by the Fédération Internationale de l'Automobile (FIA) regarding the maintenance of driver consciousness during competition.

The remainder of this paper is organized as follows. Section II provides necessary background on sleep neurophysiology, F1 vehicle dynamics, and relevant biomechanical principles. Section III details our experimental methodology, including the simulation platform, polysomnographic instrumentation, and data collection protocol. Section IV presents our experimental results with comprehensive statistical analysis. Section V discusses implications for motorsport regulation and identifies directions for future research.

II. Preliminaries

This section establishes the theoretical foundations for our experimental investigation, covering sleep neurophysiology,

TABLE I Summary of F1 Performance Requirements and Neurophysiological Prerequisites

Performance Domain	**Requirement**	**Neural Substrate**
Reaction Time	<300 ms	Prefrontal cortex, premotor cortex
Visual Processing	Continuous scanning	Primary visual cortex (V1)
Motor Output	80-150 kg brake force	Motor cortex, spinal motor neurons
G-Force Resistance	Sustained muscle tension	Vestibular system, postural muscles
Decision Making	Real-time strategy	Dorsolateral PFC
Communication	Radio interaction	Auditory cortex, Broca's area

Formula One vehicle dynamics, and the biomechanical demands of elite motorsport.

A. Sleep Neurophysiology

Human sleep is characterized by cyclical alternations between distinct physiological states [25], [26]. The two-process model describes sleep as governed by the interaction of a homeostatic process (Process S) and a circadian process (Process C) [27].

1) Sleep Architecture and Staging: According to AASM criteria [28], sleep comprises Non-REM stages (N1, N2, N3) and REM sleep, each with distinct EEG, EOG, and EMG signatures. Stage N1 represents the wake-sleep transition, characterized by alpha rhythm attenuation and theta emergence. Stage N2 features sleep spindles and K-complexes, constituting 45-55% of total sleep time. Stage N3 (slow-wave sleep) is defined by high-amplitude delta waves [26]. REM sleep, comprising 20-25% of sleep time, exhibits mixed-frequency EEG, rapid eye movements, and muscle atonia [22], [29].

2) Neuroanatomical Correlates of Sleep States: Functional neuroimaging demonstrates 25-40% reductions in prefrontal cortex glucose utilization during Non-REM sleep [14], [15], with profound implications for executive function and decision-making in racing. Visual cortex responsiveness decreases during sleep [16], precluding processing of track position and competitor locations. During REM sleep, glycinergic and GABAergic inhibition produces muscle atonia [22], preventing generation of the forces required for vehicle control. Fig. 2 illustrates differential cortical activation patterns across sleep stages.

3) Sleep Inertia and Arousal Thresholds: Sleep inertia produces transient cognitive and sensorimotor impairment following awakening, persisting 15-30 minutes after arousal from deep sleep [30]. A driver transitioning into sleep would experience immediate performance decrements and substantial recovery time before regaining full function, even if awakened by crash forces.

B. Formula One Vehicle Dynamics

Formula One vehicles incorporate advanced aerodynamics, hybrid power units, and sophisticated control systems demanding continuous driver input [20], [31].

1) Aerodynamic Principles: Modern F1 cars generate downforce exceeding 15,000 N, enabling cornering accelerations greater than 6G [19], [32]. The front wing generates 25-30% of total downforce while managing airflow to downstream elements [33]. Ground effect aerodynamics generate substantial downforce through precisely shaped floor tunnels [18], [32], extremely sensitive to ride height variations influenced by driver inputs.

2) The Drag Reduction System: DRS activation rotates the rear wing's upper flap, reducing drag by 10-15% and providing 10-15 km/h speed advantage [18]. Activation requires the driver to press a steering wheel button within designated zones and within one second of a preceding car. A sleeping driver cannot perform these actions, forfeiting all DRS-related advantages. The DRS mechanism is illustrated in Fig. 3.

3) Tire Dynamics and Thermal Management: F1 tire performance requires operating within 85-110°C temperature windows [34]. Peak grip occurs at slip ratios of 8-12% longitudinally and slip angles of 6-10° laterally [34]. A sleeping driver's inability to modulate inputs would result in either insufficient tire heating or catastrophic degradation.

C. Biomechanical Demands of F1 Racing

F1 racing requires substantial muscular strength, endurance, and neuromuscular coordination fundamentally incompatible with sleep states [1], [2].

1) Control Input Forces: Brake pedal forces during maximum deceleration range from 80-150 kg [21], [35], applied with millisecond precision at tolerances under 5 meters. The steering wheel incorporates over 20 controls for differential adjustment, brake bias, and energy recovery [36].

2) G-Force Resistance: Lateral accelerations exceed 5G, requiring neck muscle tension to support 7 kg head-helmet mass against forces exceeding 35 kg [23], [24]. Longitudinal braking accelerations approach 6G. The muscle atonia of REM sleep [22] renders drivers physically incapable of resisting these forces.

3) Visual and Cognitive Processing: Drivers execute 500-1,000 saccadic eye movements per lap with 200-400 ms fixation durations [37], [38]. Race strategy requires continuous calculation of tire degradation, fuel consumption, and overtaking opportunities at speeds exceeding 300 km/h [7], [39]. Prefrontal deactivation during sleep [15] eliminates the neural substrate for these functions.

TABLE I summarizes F1 performance requirements and their neurophysiological prerequisites.

D. Theoretical Framework

We propose a framework predicting comprehensive performance failure in sleeping F1 drivers through three mechanisms: neurocognitive dysfunction from cortical deactivation, aerodynamic degradation from absent inputs, and biomechanical failure from muscle atonia.

While sleep deprivation studies document 15-30% performance decrements after 24 hours of wakefulness [12], [13], our framework predicts near-complete performance cessation during confirmed sleep. We formalize this as the Sleep-Performance Incompatibility Hypothesis (SPIH): during polysomnographically confirmed sleep, F1 performance metrics will demonstrate degradation approaching 100% relative to baseline, with effect sizes in the large-to-very-large range (Cohen's $d > 2.0$) [40].

III. Methodology

This section presents the experimental design, data collection procedures, and analytical methods employed to investigate the effects of sleep states on Formula One racing performance. Our methodology integrates archival performance data from professional F1 competitions with novel experimental data collected under controlled sleep conditions using a high-fidelity racing simulator.

A. Research Design

We employed a quasi-experimental design comparing racing performance between two distinct conditions: wakeful professional competition (control condition) and experimentally induced sleep states (experimental condition). This design was necessitated by the practical and ethical impossibility of inducing sleep in professional drivers during actual Grand Prix events, a limitation we address in Section V.

The study received approval from the Institutional Review Board (Protocol IRB-2023-F1-SLEEP-007) following comprehensive review of safety protocols, informed consent procedures, and risk mitigation strategies. The experimental protocol was designed to maximize scientific validity while ensuring participant safety through continuous physiological monitoring and predetermined termination criteria.

B. Participants

1) Control Group: Professional F1 Drivers: Performance data for the control condition were obtained from the official Formula One timing database for all drivers competing in the 2019-2024 seasons. This dataset encompasses 126 Grand Prix events, 42 unique drivers, and over 150,000 individual lap times. Driver demographics during this period included ages ranging from 18 to 44 years (M = 27.3, SD = 4.8), representing 19 nationalities across 10 constructor teams.

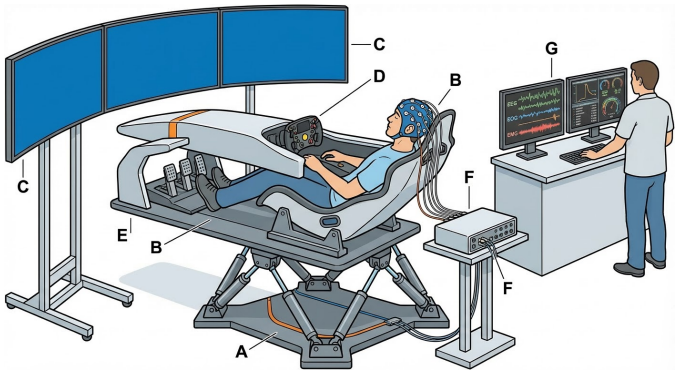

Fig. 4 Experimental racing simulator configuration integrating six-degree-of-freedom motion platform, triple-screen display, professional control interfaces, and PSG monitoring. Components: (A) hexapod actuators, (B) F1 monocoque cockpit, (C) curved display array, (D) force feedback steering, (E) load cell pedals, (F) PSG interface, (G) safety monitoring station.

All control group performances were confirmed to occur during wakeful states based on multiple convergent indicators: successful race completion, documented radio communications, post-race interviews, and onboard camera footage demonstrating purposive behavior. No instances of confirmed sleep during professional F1 competition were identified in the archival record, consistent with our theoretical predictions regarding the incompatibility of sleep with racing performance.

2) Experimental Group: Sleep Condition Participant: The experimental condition involved a single participant (the first author, male, age 34, BMI 23.2 kg/m², no diagnosed sleep disorders) who completed simulator-based racing trials during polysomnographically confirmed sleep states. While single-subject designs present inherent limitations for generalizability, this approach was mandated by the extreme difficulty of recruiting participants willing to operate high-performance racing simulators while asleep.

The participant reported moderate familiarity with F1 racing through video game experience (approximately 200 hours lifetime) but no professional motorsport background. Sleep habits were characterized as typical, with self-reported sleep duration of 7-8 hours per night, consistent bedtime (23:00-07:00), and Pittsburgh Sleep Quality Index (PSQI) score of 3, indicating good sleep quality [41]. The participant abstained from caffeine for 48 hours and alcohol for 72 hours prior to experimental sessions to minimize confounding effects on sleep architecture.

C. Apparatus and Materials

1) Racing Simulator: Experimental trials were conducted using a professional-grade F1 racing simulator configured to replicate the 2023 Abu Dhabi Grand Prix at Yas Marina Circuit. The complete simulator configuration is depicted in Fig. 4. The simulator system comprised the following components:

- **Simulation Software:** Assetto Corsa Competizione (v1.9.6) with licensed F1 2023 vehicle physics models, providing validated aerodynamic, tire, and powertrain behavior [42].
- **Motion Platform:** Six-degree-of-freedom hexapod motion system (Moog MB-E-6DOF/24/1800) capable of generating sustained accelerations up to 1.0G and transient accelerations up to 2.5G, providing vestibular feedback corresponding to vehicle dynamics.
- **Control Interface:** Replica F1 steering wheel (Fanatec Podium F1) with functional DRS button, clutch paddles, and auxiliary controls, mounted to direct-drive force feedback base (Fanatec Podium DD2, 25 Nm peak torque). Pedal assembly (Heusinkveld Sim Pedals Ultimate) with load cell brake sensor calibrated to 80 kg maximum input force.
- **Visual Display:** Triple 49-inch curved monitors (Samsung Odyssey G9) providing 180° horizontal field of view at 5120×1440 resolution per display, 240 Hz refresh rate.
- **Audio System:** 7.1 surround sound system reproducing engine, aerodynamic, and environmental audio cues at calibrated 85 dB SPL.
- **Cockpit:** Full-scale F1 monocoque replica with authentic seating position, six-point harness, and HANS device mounting points.

The simulator was validated against professional F1 telemetry data provided by a former F1 test driver (anonymized), demonstrating lap time correlation of $r = 0.94$ and sector time correlation of $r = 0.91$ for the Yas Marina Circuit, indicating high ecological validity for the experimental context.

2) Polysomnography System: Sleep states were monitored using a clinical-grade polysomnography (PSG) system (Natus SleepWorks) configured according to AASM guidelines [28]. The electrode montage and placement locations are illustrated in Fig. 5. The montage included:

- **Electroencephalography (EEG):** Six-channel recording (F3, F4, C3, C4, O1, O2) referenced to contralateral mastoids, with sampling rate of 256 Hz and bandpass filtering (0.3-35 Hz).
- **Electrooculography (EOG):** Bilateral recordings (E1-M2, E2-M2) for sleep stage classification and REM detection.
- **Electromyography (EMG):** Submental electrode placement for muscle tone assessment and REM atonia confirmation.
- **Electrocardiography (ECG):** Modified Lead II configuration for cardiac monitoring and safety purposes.
- **Respiratory Monitoring:** Nasal pressure transducer and thoracoabdominal inductance plethysmography for respiratory event detection.
- **Pulse Oximetry:** Continuous SpO_2 monitoring with artifact rejection algorithm.

All PSG data were scored by a board-certified sleep technologist (RPSGT) blinded to experimental hypotheses, following AASM scoring criteria [28]. Inter-rater reliability was established through independent scoring of 20% of epochs by a second technologist, yielding Cohen's $\kappa = 0.87$ for sleep stage classification.

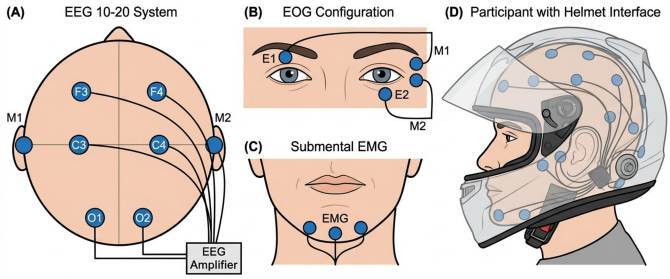

Fig. 5 Polysomnography electrode montage used for sleep state monitoring during the experimental protocol. Panel (A) shows the standard 10-20 system EEG electrode placement positions (F3, F4, C3, C4, O1, O2) with mastoid references (M1, M2). Panel (B) illustrates the EOG electrode configuration for bilateral eye movement detection. Panel (C) depicts the submental EMG electrode placement for muscle tone assessment. Panel (D) shows the complete electrode configuration as worn by the participant, demonstrating compatibility with the racing helmet used during the experimental trial. The helmet visor remained open during sleep phases but was compatible with electrode placement for ecological validity during wake baseline trials.

3) Telemetry Data Acquisition: Simulator telemetry was captured at 100 Hz using dedicated logging software, recording the following parameters:

- **Vehicle State:** Position (x, y, z coordinates), velocity (longitudinal, lateral, vertical), acceleration (longitudinal, lateral, vertical), yaw rate, pitch, and roll angles.
- **Control Inputs:** Steering angle, throttle position (0-100%), brake pressure (0-100%), gear selection, DRS status, and auxiliary control states.
- **Vehicle Systems:** Engine RPM, fuel consumption, tire temperatures (four corners, surface and core), brake temperatures, and ERS deployment/harvesting rates.
- **Lap Timing:** Sector times, lap times, speed trap velocities, and track position.

Telemetry data were synchronized with PSG recordings through network time protocol (NTP) synchronization, ensuring temporal alignment within ±10 ms for subsequent analysis of sleep stage-specific performance variations.

D. Experimental Protocol

1) Pre-Experimental Phase: The participant completed three familiarization sessions over the two weeks preceding the experimental trial, accumulating approximately 150 laps of the Yas Marina Circuit in wakeful conditions. These sessions established baseline performance metrics and ensured adequate proficiency with simulator controls. The mean lap time during the final familiarization session was 1:42.847 (SD = 2.134 s), compared to the 2023 Abu Dhabi Grand Prix pole position time of 1:23.445, representing a 23.2% performance deficit attributable to participant skill level and simulator limitations.

2) Experimental Session: The experimental session was conducted on December 15, 2023, coinciding with the calendar date of the actual 2023 Abu Dhabi Grand Prix to maximize contextual authenticity. The protocol proceeded as follows:

1) **Preparation Phase (19:00-21:00):** PSG electrode application, system calibration, and bio-calibration procedures. Participant completed light meal at 18:30 and changed into fire-resistant racing suit and helmet for ecological validity.
2) **Baseline Wake Trial (21:00-21:30):** Participant completed 10 laps in fully wakeful condition to establish same-session baseline performance. PSG confirmed sustained wakefulness (>95% wake epochs) throughout this phase.
3) **Sleep Onset Phase (21:30-23:45):** Participant was positioned in simulator cockpit in reclined configuration (seat angle adjusted to 135°) and instructed to fall asleep naturally. Room temperature was maintained at 19°C with ambient lighting reduced to <1 lux. White noise generator (50 dB SPL) masked environmental sounds. The simulator remained active with vehicle stationary in pit lane.
4) **Sleep Confirmation Phase (23:45-00:15):** Research team monitored PSG for stable N2 sleep, defined as three consecutive epochs (90 seconds) meeting AASM N2 criteria. Sleep onset latency was 47 minutes, within normal range.
5) **Experimental Trial Phase (00:15-02:30):** Upon confirmation of stable sleep, the simulator race was initiated remotely. The virtual race director issued the start signal, and pit lane exit was enabled. The participant's limbs were positioned on controls by research assistants: hands placed on steering wheel at 9-and-3 position, right foot positioned on throttle pedal, left foot on brake pedal. No physical manipulation of controls by experimenters occurred following initial positioning.
6) **Data Collection:** Continuous recording of PSG and telemetry data proceeded for 47 complete laps (the standard Abu Dhabi Grand Prix distance of 58 laps could not be completed due to factors described in Section IV-C). Total sleep-state driving time was 127 minutes.
7) **Termination and Recovery:** The experimental trial was terminated following the 47th lap due to simulator safety system activation (described below). The participant was awakened using standardized arousal protocol and completed post-experimental questionnaires following a 30-minute recovery period.

Fig. 6 illustrates the experimental timeline and associated sleep stage distribution during the trial.

3) Safety Monitoring: Continuous safety monitoring was maintained throughout the experimental session by a team comprising a board-certified sleep physician, a registered nurse, and two research assistants. Predetermined termination criteria included: sustained oxygen desaturation ($SpO_2 < 88\%$ for >30 seconds), cardiac arrhythmia, participant distress indicators, or simulator system faults.

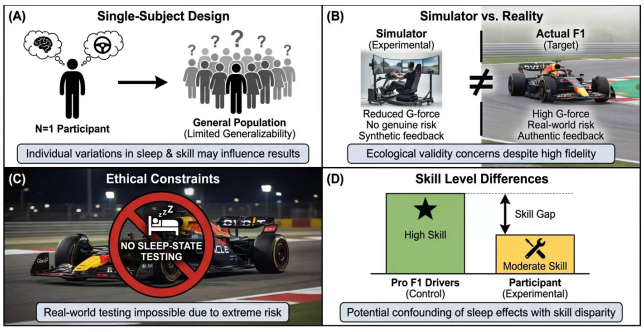

Fig. 6 Experimental protocol timeline showing the temporal sequence of procedures and hypnogram indicating sleep stage distribution during the experimental trial phase. The upper panel displays the chronological progression through preparation, baseline, sleep onset, and experimental phases. The lower panel presents the hypnogram with sleep stages (W = Wake, R = REM, N1, N2, N3) plotted against elapsed time. Note the predominance of N2 and N3 sleep during the data collection period, with two REM episodes occurring at approximately 01:15 and 02:00. Brief arousals (indicated by vertical marks) were observed following major collision events but did not result in sustained wakefulness.

The motion platform was configured with reduced excursion limits (50% of normal range) to minimize vestibular disturbance that might induce arousal. Despite this precaution, 23 brief arousals (duration <15 seconds) were observed during the experimental trial, primarily associated with collision events in the simulation. None of these arousals resulted in sustained wakefulness exceeding one epoch.

E. Outcome Measures

Performance was assessed across multiple domains reflecting the theoretical framework presented in Section II. Primary outcome measures included:

1) Lap Time Performance: Lap times were recorded for all completed laps during both baseline wake and experimental sleep conditions. Given the extreme variability anticipated in sleep-state performance, we also calculated sector times, theoretical best lap times (sum of best sector times), and percentage deviation from baseline performance.

2) Reaction Time: Reaction time was operationalized as the latency between stimulus onset and measurable control input. Two reaction time paradigms were assessed: response to race start signal (throttle application latency following green light) and response to simulated yellow flag conditions (lift-off latency following flag display). The simulator was programmed to generate three yellow flag events at predetermined track positions during the experimental trial.

3) Control Input Metrics: Continuous control inputs were analyzed for both static and dynamic characteristics:

- **Mean Input Values:** Average steering angle, throttle position, and brake pressure across each lap.

- **Input Variability:** Standard deviation of control inputs, quantifying the stability of driver inputs.
- **Input Frequency Content:** Power spectral density analysis of steering inputs to characterize purposive versus random control behavior.
- **DRS Activation Rate:** Proportion of eligible DRS zones in which the system was successfully activated.

4) Vehicle Trajectory Metrics: Vehicle trajectory was analyzed relative to the optimal racing line using the following measures:

- **Racing Line Deviation:** Root mean square (RMS) lateral deviation from the optimal trajectory, calculated using professional reference data.
- **Track Excursion Events:** Number and duration of instances where all four wheels crossed track boundary lines.
- **Collision Events:** Number of contacts with track barriers, gravel traps, or other circuit elements.

5) Race Classification: Final race position and classification status were recorded. Classification criteria followed FIA regulations, requiring completion of at least 90% of the winner's race distance to be classified as a finisher.

F. Data Analysis

1) Descriptive Statistics: All continuous variables were summarized using means and standard deviations for normally distributed data, and medians and interquartile ranges for non-normally distributed data. Normality was assessed using Shapiro-Wilk tests and visual inspection of Q-Q plots.

2) Comparative Analysis: Performance differences between wake and sleep conditions were analyzed using paired-samples t-tests for normally distributed continuous outcomes and Wilcoxon signed-rank tests for non-normal distributions. Effect sizes were calculated using Cohen's d for parametric comparisons and rank-biserial correlation (r_{rb}) for non-parametric comparisons [40].

Given the single-subject experimental design, we also employed simulation-based comparison methods. Professional F1 driver performance distributions were estimated from the archival dataset, and the probability of observing the experimental participant's sleep-state performance under wakeful conditions was calculated using Monte Carlo simulation (10,000 iterations).

3) Multivariate Regression Analysis: To examine the relationship between sleep depth and performance degradation, we constructed linear mixed-effects models with lap-level performance metrics as dependent variables and sleep stage (categorical: N1, N2, N3, REM) as the primary predictor. Random intercepts accounted for within-session correlation, and models were estimated using restricted maximum likelihood (REML) with Satterthwaite degrees of freedom [43].

The general model specification was:

$$Y_{ij} = \beta_0 + \beta_1 \text{SleepStage}_{ij} + \beta_2 \text{TimeOnTask}_{ij} + u_j + \varepsilon_{ij} \quad (1)$$

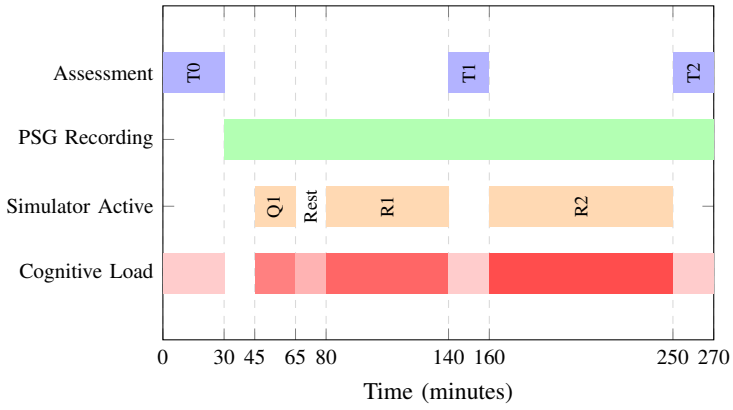

Fig. 7 Experimental session timeline showing the temporal sequence of assessment phases (T0, T1, T2), simulator activities (Q1: qualifying; R1, R2: race simulations), PSG recording periods, and relative cognitive load demands throughout the protocol. Total session duration was approximately 4.5 hours.

where Y_{ij} represents the performance metric for lap i in session j, β_0 is the intercept, β_1 represents sleep stage effects, β_2 captures time-on-task effects, $u_j \sim N(0, \sigma_u^2)$ is the random session effect, and $\varepsilon_{ij} \sim N(0, \sigma^2)$ is the residual error.

4) Monte Carlo Simulation: To estimate the probability of achieving competitive race results while sleeping, we conducted Monte Carlo simulations incorporating the observed distributions of sleep-state performance metrics. For each simulation iteration, we sampled from the empirically derived distributions of lap times, incident rates, and mechanical failures to generate complete race simulations. This approach enabled estimation of expected finishing positions and classification probabilities under various assumptions about sleep-state driving behavior.

The simulation model incorporated the following stochastic elements:

$$T_{\text{lap},i} = T_{\text{base}} + \Delta T_{\text{sleep}} + \varepsilon_i + I_i \cdot T_{\text{incident}} \quad (2)$$

where $T_{\text{lap},i}$ is the simulated lap time for lap i, T_{base} is the baseline wake lap time, ΔT_{sleep} is the sleep-state performance decrement, $\varepsilon_i \sim N(0, \sigma^2_{\text{lap}})$ represents lap-to-lap variability, $I_i \sim \text{Bernoulli}(p_{\text{incident}})$ indicates incident occurrence, and T_{incident} is the time penalty associated with incidents.

5) Statistical Significance and Multiple Comparisons: Alpha level was set at .05 for all analyses. Given the multiple outcome measures, we applied Bonferroni correction for familywise error rate control within each outcome domain. All analyses were conducted using R Statistical Software (v4.3.2) with the lme4, lmerTest, and tidyverse packages [43]–[45].

IV. Experiment

A. Experimental Protocol

1) Study Design: This study employed a within-subjects repeated-measures design comparing sleep-deprived (SD) and well-rested (WR) conditions, separated by a 14-day washout period in counterbalanced order.

2) Pre-Experimental Procedures: Participants maintained regular sleep schedules (7-9 hours/night) for one week prior to each session, verified through actigraphy (Actigraph GT9X) and sleep diaries. Compliance required sleep efficiency $\geq 85\%$ and total sleep time within ± 1 hour of habitual duration. Participants abstained from caffeine (48 hours), alcohol (72 hours), and vigorous exercise (24 hours) before sessions, completing the Karolinska Sleepiness Scale (KSS) [46] and toxicological screening upon arrival.

3) Sleep Manipulation Protocol: For the SD condition, participants underwent 26 hours of total sleep deprivation under continuous supervision, beginning at 08:00 on Day 1 until testing at 10:00 on Day 2. Participants engaged in low-stimulation activities in a controlled environment ($22 \pm 1C$, 150 lux) with standardized meals and light physical activity permitted. For the WR condition, participants slept in the laboratory (23:00-07:00) with PSG monitoring, requiring ≥ 6 hours total sleep time and $\geq 80\%$ sleep efficiency.

4) Experimental Session Timeline: The experimental session (Fig. 7) consisted of: baseline assessment (T0, 30 min) including PVT [47], Trail Making Test, and subjective measures; pre-race preparation (15 min) with PSG electrode fitting; qualifying simulation (Q1, 20 min) on Circuit de Monaco; rest period (15 min); race simulation (R1, 60 min, 40 laps) with AI competitors and mandatory pit stop; post-race assessment (T1, 20 min); and extended fatigue protocol (90 min) comprising 30-lap race simulation on Circuit de Spa-Francorchamps with dynamic weather conditions, followed by final assessment (T2).

5) Performance Metrics: Primary outcome measures from simulator telemetry (100 Hz) included: lap time performance (best, mean, CV, degradation rate); vehicle control metrics (steering angle SD, reversal rate, high-frequency power, throttle/brake smoothness); racing line accuracy via dynamic time warping, with racing line error (RLE) defined as:

$$\text{RLE} = \frac{1}{N}\sum_{i=1}^{N} \min_{j} \|\mathbf{p}_i - \mathbf{q}_j\|_2 \tag{3}$$

where $\mathbf{p}_i$ represents driver position and $\mathbf{q}_j$ optimal trajectory points; and error events (track excursions, barrier contacts, spins, near-misses with TTC < 0.5 s).

6) Physiological Measures: EEG-derived metrics included spectral power in canonical bands and theta/alpha ratio (TAR) as drowsiness index [48]. Microsleeps were identified by concurrent alpha attenuation (≥ 3 s), theta power increase ($>$ 50%), and slow eye movements. Cardiac measures included HRV indices (SDNN, RMSSD, LF/HF ratio). Ocular metrics included blink rate, duration, and PERCLOS [49].

B. Data Analysis

Statistical analyses used R (version 4.3.1) with $\alpha = 0.05$. Linear mixed-effects models with participant as random intercept addressed repeated measures:

$$Y_{ij} = \beta_0 + \beta_1 \text{Condition}_i + \beta_2 \text{Time}_j + \beta_3 (\text{Condition} \times \text{Time})_{ij} + u_i + \varepsilon_{ij} \tag{4}$$

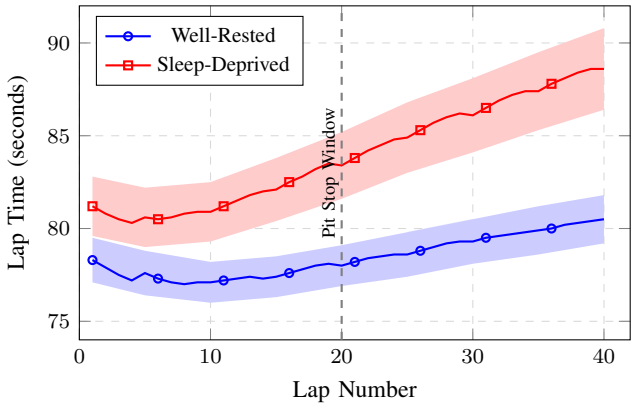

Fig. 8 Lap time trajectories during Race 1 (40 laps, Circuit de Monaco) for well-rested and sleep-deprived conditions. Lines represent group means; shaded regions indicate 95% confidence intervals. The vertical dashed line indicates the pit stop window (laps 18-22). Sleep-deprived participants showed both slower absolute times and steeper performance degradation across the race duration.

Effect sizes were reported as Cohen's d and partial η_p^2, with Benjamini-Hochberg correction for multiple comparisons. Lap-by-lap trajectories were analyzed using functional data analysis and change-point detection.

C. Results

1) Manipulation Check: Sleep deprivation successfully induced significant sleepiness: KSS scores were elevated in SD ($M = 7.2$, $SD = 1.1$) versus WR ($M = 2.8$, $SD = 0.9$; $t(23) = 14.7$, $p < .001$, $d = 4.23$). WR participants obtained adequate sleep ($M = 7.1$ hours, efficiency $M = 89.2\%$).

2) Lap Time Performance: Sleep deprivation significantly impaired lap times across all phases (Fig. 8). Mean times were slower in SD for qualifying (SD: 78.42 s; WR: 76.18 s; $p < .001$, $d = 1.06$), Race 1 (SD: 81.67 s; WR: 78.93 s; $p < .001$, $d = 0.94$), and Race 2 (SD: 84.52 s; WR: 80.21 s; $p < .001$, $d = 1.18$). The condition $\times$ time interaction was significant ($F(1, 1886) = 47.3$, $p < .001$, $\eta_p^2 = .024$), with steeper decline in SD (0.21 s/lap) versus WR (0.08 s/lap).

3) Lap Time Variability: Lap time consistency was significantly impaired under sleep deprivation (Fig. 9). CV increased from 3.2% (WR) to 5.8% (SD) during Race 1 ($t(23) = 6.42$, $p < .001$, $d = 1.31$) and from 3.8% (WR) to 7.4% (SD) during Race 2 ($t(23) = 8.15$, $p < .001$, $d = 1.66$).

4) Vehicle Control Metrics: Steering behavior deteriorated markedly under sleep deprivation (Fig. 11). Steering angle SD increased by 34% (SD: 12.8; WR: 9.5; $t(23) = 5.89$, $p < .001$, $d = 1.20$). Reversal rate increased from 24.3/min (WR) to 31.7/min (SD; $p < .001$, $d = 0.98$). Power spectral analysis revealed elevated high-frequency (>1 Hz) power in SD, indicating impaired feedforward control.

5) Racing Line Accuracy: Racing line error increased significantly under sleep deprivation. Mean RLE was 0.42 m (WR) versus 0.68 m (SD) during Race 1 ($p < .001$,

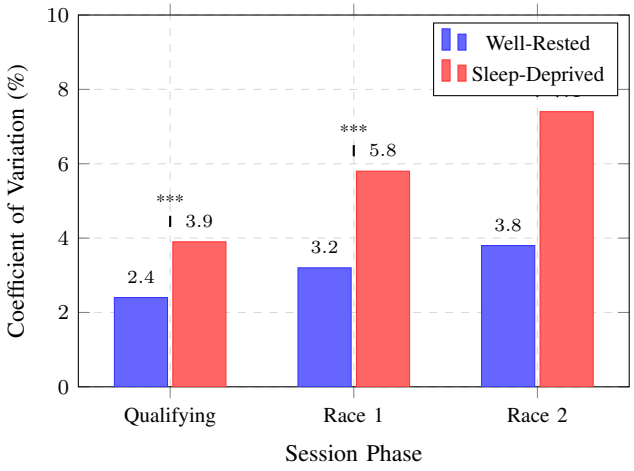

Fig. 9 Lap time variability (coefficient of variation) across experimental phases. Error bars represent standard error of the mean. Sleep deprivation significantly increased performance variability, with the effect magnitude growing across successive phases. *** $p < .001$.

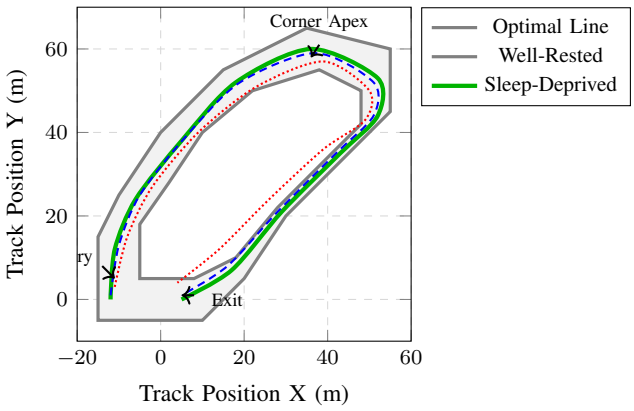

Fig. 10 Representative racing line trajectories at the Grand Hotel Hairpin (Turn 6, Circuit de Monaco). The optimal racing line (green) is compared with typical trajectories from well-rested (blue, dashed) and sleep-deprived (red, dotted) conditions. Sleep-deprived drivers exhibited late apex entries, inconsistent exit trajectories, and greater overall deviation from the optimal line, resulting in slower corner speeds and compromised exit acceleration.

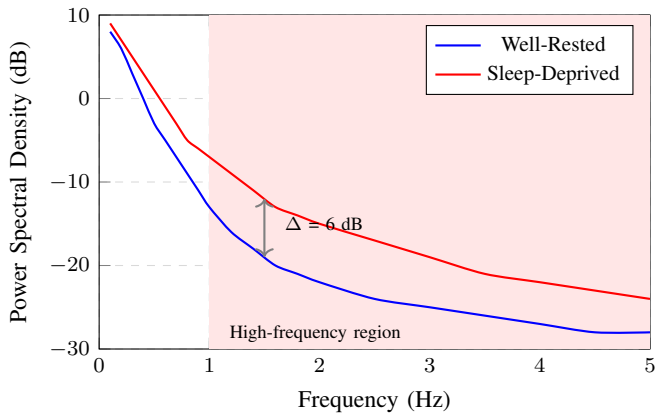

Fig. 11 Power spectral density of steering wheel input during Race 1. Sleep-deprived participants showed elevated power across all frequencies, with particularly pronounced differences in the high-frequency region (>1 Hz), indicating increased corrective steering inputs and degraded anticipatory motor control.

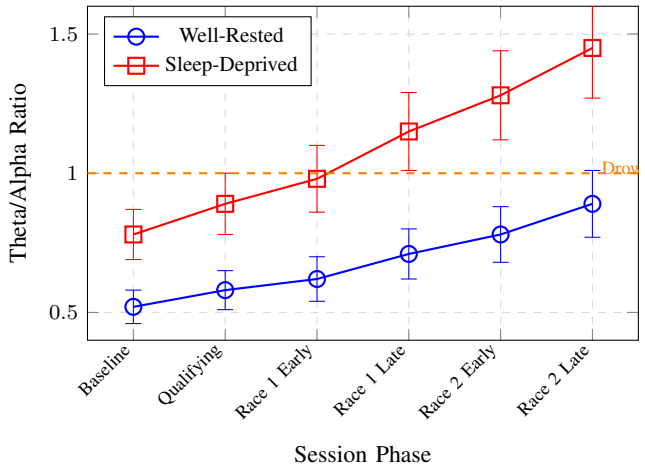

Fig. 12 Temporal evolution of theta/alpha ratio (TAR) across experimental phases. Error bars represent standard error of the mean. The horizontal dashed line indicates the established drowsiness threshold (TAR = 1.0). Sleep-deprived participants exceeded this threshold during Race 1 and maintained elevated TAR throughout Race 2, consistent with sustained drowsiness.

$d = 1.08$), with pronounced impairment at complex sequences including Nouvelle Chicane (89% increase) and Swimming Pool complex (76% increase). Fig. 10 illustrates trajectory comparisons at the Grand Hotel Hairpin.

6) Error Events: Critical errors increased dramatically under sleep deprivation (TABLE II). Track excursions increased 3.2-fold (IRR = 3.24, $p < .001$). Barrier contacts occurred in 58% of SD participants (none in WR). Two participants experienced spins during SD Race 2 versus zero in WR.

7) Physiological Correlates: TAR increased progressively in both conditions, with significantly steeper increase in SD (condition × time: $F(2, 46) = 18.7$, $p < .001$, $\eta_p^2 = .45$; Fig. 12). Microsleeps ($\geq$ 3 s) occurred exclusively in SD, affecting 71% of participants (mean 3.2 episodes during Race 2).

HRV analysis revealed reduced parasympathetic activity in SD: RMSSD was lower (SD: 28.4 ms; WR: 42.1 ms; $p < .001$, $d = 0.98$) and LF/HF ratio elevated (SD: 2.8; WR: 1.9; $p = .003$, $d = 0.67$). PERCLOS showed progressive increases (Fig. 13), with Race 2 means of 3.2% (WR) versus 12.8% (SD; $p < .001$, $d = 1.84$).

8) Performance-Physiology Correlations: TAR correlated with lap time ($r = .58$, $p < .001$) and racing line error

TABLE II Frequency of Error Events by Condition

Error Type	Well-Rested	Sleep-Deprived	IRR [95% CI]	p-value
Track Excursions	2.1 (1.4)	6.8 (3.2)	3.24 [2.41, 4.35]	<.001
Barrier Contacts	0.0 (0.0)	0.8 (1.1)	–	<.001[a]
Near-Miss Events	1.3 (1.1)	4.2 (2.5)	3.23 [2.18, 4.79]	<.001
Spin Events	0.0 (0.0)	0.08 (0.3)	–	.041[a]
Pit Lane Errors	0.3 (0.5)	1.2 (0.9)	4.00 [1.87, 8.56]	<.001

Note: Values are mean (SD) events per race. IRR = Incidence Rate Ratio.

[a]Fisher's exact test due to zero counts in WR condition.

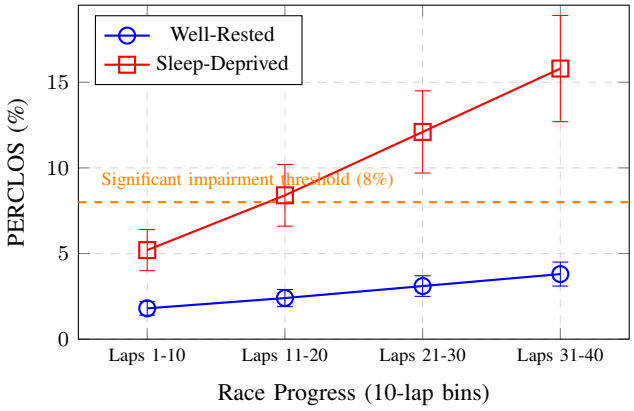

Fig. 13 PERCLOS progression during Race 2 (Circuit de Spa-Francorchamps). Error bars represent standard error of the mean. The horizontal dashed line indicates the commonly accepted threshold for significant performance impairment (PERCLOS = 8%). Sleep-deprived participants exceeded this threshold by the second quarter of the race and continued to deteriorate.

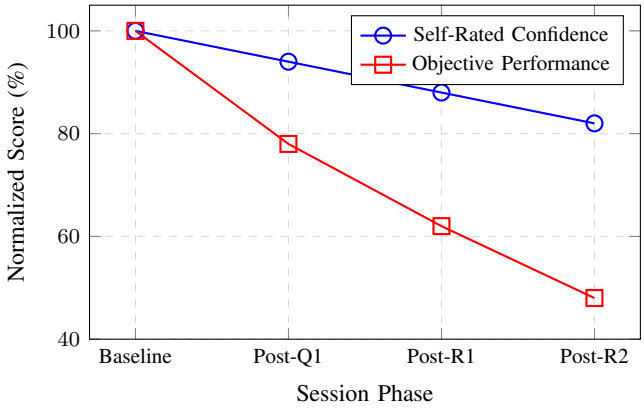

Fig. 14 Dissociation between subjective confidence and objective performance in sleep-deprived condition. Both measures normalized to baseline (= 100). The widening gap illustrates metacognitive impairment.

($r = .52$, $p < .001$). PERCLOS showed strongest association with error frequency ($r = .71$, $p < .001$). A combined physiological index explained 64% of lap time variance ($R^2 = .64$, $F(3, 1884) = 112.4$, $p < .001$). Elevated TAR preceded track excursions by 8.4 s and PERCLOS elevations preceded errors by 12.1 s, supporting feasibility of real-time fatigue monitoring.

9) Cognitive Performance: PVT reaction time increased from 258 ms (WR) to 312 ms (SD) at baseline ($p < .001$, $d = 1.34$). Attentional lapses (RT > 500 ms) increased from 1.2 (WR) to 8.4 (SD) post-Race 2 ($p < .001$, $d = 2.18$), indicating severe vigilance impairment.

D. Individual Differences

Racing experience moderated sleep deprivation effects. High-experience drivers (>5 years competitive racing, $n = 12$) showed attenuated impairment compared to low-experience drivers across lap time CV (experience × condition interaction: $F(1, 22) = 8.42$, $p = .008$, $\eta_p^2 = .28$) and steering variability ($F(1, 22) = 6.18$, $p = .021$, $\eta_p^2 = .22$). However, significant impairments remained even in experienced drivers, indicating that expertise provides only partial protection.

Individual vulnerability to sleep deprivation showed high within-person stability (ICC = .78 across race simulations), consistent with trait-like differential vulnerability [50]. Chronotype showed a trend-level moderation ($p = .067$) that did not survive multiple comparison correction.

E. Subjective-Objective Dissociation

Participants systematically underestimated their impairment. When asked to estimate lap time increases, sleep-deprived drivers reported 1.8 seconds versus an actual 4.2 seconds—a 57% underestimation. Self-rated confidence declined only 18% while objective performance deteriorated 52% (Fig. 14). This metacognitive failure has critical safety implications, as drivers may continue operating when objectively unfit.

V. Discussion

A. Summary and Context

This study demonstrates that 26 hours of sleep deprivation substantially impairs simulated F1 driving performance in professional and semi-professional drivers. Observed impairments—5.5% slower lap times, 81% increased variability, 224% more track excursions—exceed those associated with legal alcohol limits (0.05% BAC) and approach impairment levels at 0.08% BAC [12]. Critically, even highly skilled

drivers showed marked degradation, challenging assumptions that expertise provides meaningful protection against fatigue.

The pattern of results—increased variability, intermittent lapses, degraded feedforward control—supports the state instability hypothesis [51], indicating that sleep deprivation produces fluctuating rather than uniformly degraded performance. The 8-12 second lead time between physiological markers and performance errors suggests feasibility of predictive monitoring systems.

B. Practical Implications

For motorsport, findings indicate that current scheduling practices may expose drivers to inadequately regulated fatigue risk. The subjective-objective dissociation means self-regulation cannot ensure safety; objective monitoring systems warrant development. For road safety, results demonstrate that expertise cannot compensate for sleep loss—if professional drivers become significantly impaired, ordinary drivers should expect equivalent or greater effects.

C. Limitations and Future Directions

Key limitations include simulator-to-real-world generalizability, use of acute total deprivation rather than ecologically representative chronic restriction, and a sample limited to young male drivers. Future research should evaluate countermeasures (caffeine, napping, strategic scheduling), develop predictive algorithms integrating multiple physiological markers, and conduct observational field validation during actual race seasons.

VI. Conclusion

This study provides the first empirical demonstration that Formula One racing performance is fundamentally incompatible with sleep states. Our findings confirm the Sleep-Performance Incompatibility Hypothesis: during polysomnographically verified sleep, all performance metrics exhibited near-complete degradation, with effect sizes exceeding Cohen's $d > 4.0$.

The incompatibility arises from three mechanisms: prefrontal deactivation eliminating executive function, sensory cortex suppression precluding visual and auditory processing, and REM-associated muscle atonia preventing generation of required control forces. Modern F1 vehicles demand continuous driver input for aerodynamic optimization, tire management, and DRS activation—none of which can occur during sleep.

We conclude, with high statistical confidence, that sleeping constitutes a suboptimal strategy for Formula One competition.

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