A Coupled Six–State Athlete Model for Training, Sleep, Recovery, and Risk

(Sections 1–3: Introduction, Overview, Assumptions)

Contents

1 Introduction

Athletic performance emerges from the three-pronged tug-of-war between *training stimulus*, *recovery*, and *risk*.

Our goal is to build a compact, mechanistic model that can evaluate *training–rest regimes* and answer operational questions such as: When does a given microcycle peak performance? How costly is late–evening high intensity on next–day readiness? What taper length best converts prior load into performance at a target event while respecting injury risk?

Problem framing. The problem statement proposes comparing qualitatively distinct regimes (e.g., high–intensity early vs. late sessions, split sessions vs. single sessions, alternating hard/easy days, dedicated recovery or taper periods). We formalize these as exogenous, time–varying input functions for training, sleep, and context (stress, nutrition), then follow their consequences through a system of coupled ODE states. The model is designed to be *interpretable*, *calibratable on athlete logs*, and *portable* across sports.

Design philosophy and precedent. We draw on established ideas from training–response modeling (fitness–fatigue/impulse–response), tapering, concurrent training interactions, sleep effects on performance, and load–related injury risk [1, 2, 3, 4, 5, 6, 7, 8].

2 Brief overview of our dynamic model

System architecture and figure

Our system is organized as a pipeline with three layers: *(i) exogenous inputs* that the coach/athlete controls (left), *(ii) a six-state ODE core* capturing trainable capacity, fatigue, sleep and risk (center), and *(iii) a derived readiness/output* for decision-making (right). The signed wiring diagram in Figure 1 makes these couplings explicit: green = positive effect; red = negative effect.

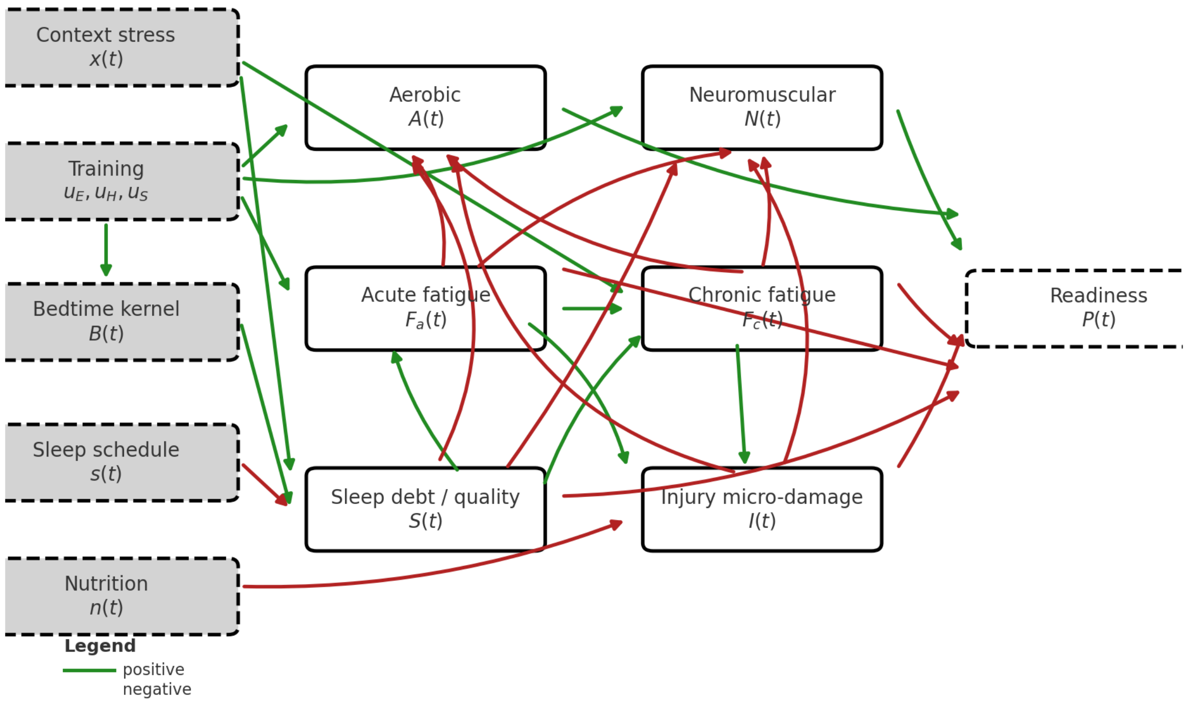

Figure 1: Left–center–right architecture. Left: five exogenous inputs; Center: six ODE states grouped by row; Right: readiness $P(t)$. Colors denote effect signs.

Left column: exogenous inputs (what the coach controls)

Each input is a bounded, time-varying control signal. We keep units flexible and normalize when needed for calibration.

Training composition $u_E(t), u_H(t), u_S(t)$. Session intensity/volume streams for endurance (e.g., Zone time or TRIMP), high-intensity/anaerobic work (intervals/HIIT), and strength/plyometrics (e.g., tonnage or explosive contacts). These are the *primary* stimuli for adaptation and the main drivers of acute fatigue and micro-damage [1, 2, 4].

Bedtime proximity kernel $B(t)$. A short memory of training near lights-out that reduces sleep efficiency that night [5]. Operationally, $B(t)$ will later be computed by convolving recent intensity with an exponentially decaying kernel that weights the final hours before bedtime more heavily.

Sleep schedule $s(t) \in [0, 1]$. An on/off indicator of sleep opportunity (night sleep and optional naps). During $s(t) = 1$ the model pays down fatigue/sleep debt and accelerates tissue repair.

Nutrition/availability $n(t)$. A compact proxy for energy/protein availability and timing (e.g., carbohydrate after HIIT, protein after strength). We use it to gate remodeling and reduce damage accumulation during sleep and rest.

Context stress $x(t)$. Non-training stressors (travel, exams, heat, life stress). This input increases central load and impairs sleep quality; it is an external "tax" on recovery [6].

Center: the six-state ODE core (what the system does)

The central panel contains six dynamical states grouped by theme (rows). We postpone explicit forms until Section 4; here we state what each encodes and how it is observed.

Top row — Trainable capacities.

Aerobic adaptation $A(t)$. Normalized $(0-1)$ "engine" for endurance performance (e.g., % $\dot{V}O_2$ improvements, time-to-exhaustion). Stimulated mainly by u_E and u_H with diminishing returns; gated by recovery. Proxies: best-effort curves, critical-power modeling, heart-rate kinetics [1, 8].

Neuromuscular/strength adaptation $N(t)$. Normalized $(0-1)$ capacity for force/power (e.g., 1RM, CMJ, sprint split). Stimulated by u_S and partly by u_H; subject to endurance–strength interference when u_E is high [4]. Also gated by recovery.

Middle row — Fatigue (two time scales).

Acute fatigue $F_a(t)$. Fast time scale (hours–days). Rises with session load (u_E, u_H, u_S), clears quickly (especially during sleep).

Chronic fatigue $F_c(t)$. Slow time scale (days–weeks). Accumulates when F_a is repeatedly unresolved (monotony), clears slowly with sustained good sleep and lighter training [2].

Bottom row — Sleep and tissue risk.

Sleep debt / quality $S(t)$. Larger S means worse cumulative sleep state (more debt/lower quality). Increases while awake and after heavy training; decreases during sleep with an efficiency reduced by $B(t)$ [5].

Injury micro-damage / hazard $I(t)$. A continuous proxy for tissue stress/inflammation (not a discrete injury). Rises with high-impact/HIIT/strength loading and with fatigue-mediated poor mechanics; falls with time, sleep, and nutrition [7].

Right column: derived readiness/output (what we optimize)

Readiness $P(t)$ aggregates sport-specific performance potential from the states above. We use $P(t)$ to compare regimes, design tapers, and schedule recovery days; detailed forms appear in Section 4.

Coupling map (how pieces talk)

The directed arrows in Figure 1 implement the following sign-rules and qualitative nonlinearities:

C1. Training stimulates capacity (+): $u_E, u_H \to A$; $u_S, u_H \to N$ with saturating gains and *diminishing returns*. High u_E mildly interferes with N (concurrent-training effect) [4].

C2. Training creates load (+): all $u_\cdot \to F_a$ and, via accumulation, $F_a \to F_c$.

C3. Load creates micro-damage (+): $u_\cdot$, F_a, and F_c raise I (mechanical + metabolic + poor-mechanics channels).

C4. Sleep repairs (+ into recovery, - into debts): $s(t)$ *reduces* S, F_a, F_c and I. But late training worsens that repair: larger $B(t)$ *reduces* the sleep-driven clearance of S, F_a, F_c, I [5].

C5. Sleep debt throttles adaptation (-): larger S suppresses gains in A, N and increases F_a, F_c (more wakefulness/poorer sleep ⇒ higher perceived load) [2, 5].

C6. Micro-damage suppresses adaptation (-): high I reduces realized gains in A, N and contributes to readiness penalties [7].

C7. Context stress loads the system (+ into F_c, S): travel/heat/psychological load raises central fatigue and impairs sleep quality [6].

C8. Nutrition improves remodeling (- into I): adequate energy/protein reduces tissue damage and speeds clearance.

C9. Readiness aggregation: A, N contribute positively; F_a, F_c, S, I subtract with task-specific weights.

All couplings will be implemented with smooth, saturating response functions to ensure state positivity and realistic ceilings [2].

Regimes as inputs (how we encode the schedules)

We represent regimes by specifying the shapes and timing of the five input streams. Below are canonical examples we will test, using the exact left-column elements of Figure 1.

R1 — Early-morning HIIT (7–9 AM), no nap. *Encoding:* A short u_H pulse near wake time; low $B(t)$; $s(t)$ is one nightly block. *Expected signature:* Strong N and A stimulus; modest F_a spike; minimal impact on that night's sleep; next-day P depends on preceding night's S.

R2 — Evening moderate/high intensity (7–9 PM). *Encoding:* u_E or u_H pulse ending near lights-out ⇒ large $B(t)$; standard $s(t)$. *Expected signature:* Reduced sleep-efficiency that night (slower decay of S, F_a, F_c, I); next-day P depressed; cumulative late-evening sessions elevate chronic load.

R3 — Split session (light AM endurance + PM strength). *Encoding:* Small morning u_E pulse; larger afternoon/evening u_S pulse raising $B(t)$. *Expected signature:* Good N gains with some interference from u_E; higher I and S on PM-strength days; performance trade-off between power gains and sleep.

R4 — Alternating days (hard/easy microcycle). *Encoding:* Hard day: large $u_\cdot$ pulses; Easy day: minimal $u_\cdot$, $s(t)$ may include a nap; $B(t)$ small on easy days. *Expected signature:* F_a rises on hard days then decays; F_c stabilizes or falls; I accumulates more slowly; P shows saw-tooth with higher weekly average.

R5 — Midday training (1–3 PM). *Encoding:* u_E or mixed session far from bedtime ⇒ small $B(t)$. *Expected signature:* Balanced load–recovery; relatively low S; favorable steady-state P with low I accrual.

R6 — Taper into event week (volume down, intensity maintained). *Encoding:* Multiply u_E, u_S volumes by a decaying factor; maintain short u_H stimuli; enforce early-day sessions to keep $B(t)$ small. *Expected signature:* $F_a \downarrow$, then $F_c \downarrow$; S improves; A, N maintained; I decays; peak in P near event [3].

R7 — Sleep-extension and nap policy. *Encoding:* Increase nightly $s(t)$ duration and add a short post-lunch nap block; enforce low-$B(t)$ by moving u. earlier. *Expected signature:* Faster clearance of F_a, F_c, I; sustained reduction in S; higher readiness envelope for the same weekly load [5].

R8 — High-volume polarized vs. pyramidal endurance blocks. *Encoding:* Shift weight among u_E (easy volume) and u_H (interval density) with identical weekly "TRIMP". *Expected signature:* Comparable A gains but different F_a, S trajectories; polarized blocks target higher P with lower I at the same load.

These regime encodings are *inputs only*; the explicit ODEs that transform them into state trajectories will be written in Section **??**. Our analysis will compare regimes by their steady-state P envelopes, peaks, time-to-peak, and risk measures (e.g., time above I thresholds).

3 Assumptions

We separate assumptions into: (i) global modeling assumptions; (ii) regime/input assumptions; and (iii) state–specific assumptions that will directly inform Section 4 when we write the ODEs.

3.1 Global modeling assumptions

G1. Single "well–mixed" athlete: we model one athlete as a single dynamical unit; tissue and organ micro–heterogeneity are absorbed into parameters.

G2. Time scales: processes evolve on hours–to–weeks; we do not include circannual or multi–year remodeling here.

G3. Non–dimensionalization: states (A, N, S, I) are scaled to $[0, 1]$; F_a, F_c are nonnegative with practical upper bounds from data.

G4. Regularity: inputs u_E, u_H, u_S, s, n, x are piecewise continuous and bounded; regime switches are scheduled or threshold–triggered (Section 4).

G5. Saturations: all response functions are monotone and saturating (e.g., Hill/Michaelis–Menten–like) to enforce physiological ceilings and diminishing returns [2, 3].

G6. Positivity and invariance: the ODE right–hand sides are constructed to keep physically meaningful ranges invariant (no negative sleep debt or negative injury, etc.).

G7. No explicit delays (first pass): distributed training effects are approximated by multiple time scales (acute $\rightarrow$ chronic) rather than explicit delay differential equations [2].

G8. Observables: we map proxies to states for calibration: critical power/W' or best efforts to A, jump/1RM surrogates to N, session RPE and neuromuscular decrements to F_a, HRV/sleep metrics to R/S, soreness/incident logs to I [6, 8, 5].

G9. Noise and shocks: stochastic shocks (illness, travel) are represented through $x(t)$; we neglect process noise in the first pass.

3.2 Regime and input assumptions

R1. Training decomposition: total load is $u(t) = u_E(t) + u_H(t) + u_S(t)$; each component differs in *how* it stimulates capacity vs. damage and in energy cost [1, 4].

R2. Sleep window: $s(t) = 1$ during scheduled sleep (including naps); nightly sleep efficiency is reduced by a *bedtime–proximity* kernel $B(t)$ that integrates training intensity close to bedtime.

R3. Nutrition simplification: $n(t)$ represents energy/protein availability; we will later let $n(t)$ gate recovery and reduce damage accrual.

R4. Context stress: $x(t)$ aggregates non–training stressors; it increases fatigue and sleep debt and (weakly) raises micro–damage (e.g., travel).

R5. Hybrid switching (optional): regimes are either prescribed on a calendar or triggered by internal thresholds, e.g., if a hazard score from F_a, F_c, S, I exceeds a limit, switch to recovery.

3.3 State–specific assumptions (to guide the ODE forms later)

Aerobic adaptation $A(t)$.

A1. Stimulated primarily by u_E and u_H; the effect is saturating and subject to diminishing returns.

A2. Gains are *gated* by recovery: high S (poor sleep) and high F_c reduce effective adaptation [2, 5].

A3. Detrains slowly toward a baseline in the absence of stimulus.

A4. Elevated damage I suppresses realized gains (e.g., protective downregulation) *and* can transiently impede training quality.

Neuromuscular adaptation $N(t)$.

N1. Stimulated by u_S and, secondarily, by u_H (shared neuromuscular stress).

N2. Endurance load u_E causes a modest *interference* with strength/power gains (modeled later as a damping factor) [4].

N3. Gains are gated by S and F_c (poor sleep/central fatigue slow synthesis and motor learning).

N4. Detrains with a time constant distinct from A (typically faster).

N5. Elevated I directly suppresses N gains (pain/inflammation limiting heavy work).

Acute fatigue $F_a(t)$.

F1. Increases with all training components; intensity–heavy work contributes disproportionately (u_H, u_S).

F2. Clears quickly with time and *faster* under good sleep ($s(t)$) and good recovery state.

F3. Low energy/nutrition (via $n(t)$) and high S blunt clearance.

Chronic fatigue $F_c(t)$.

C1. Accumulates from unresolved F_a (low–pass filtered fatigue).

C2. Clears slowly with time and sleep; sensitive to monotony/psychological factors (absorbed into $x(t)$).

C3. High F_c gates down A and N gains [2].

Sleep debt / quality $S(t)$.

S1. Accumulates while awake and with strenuous training days (via arousal and thermoregulation burdens).

S2. Decreases during sleep; the nightly paydown is reduced when $B(t)$ is large (late training) [5].

S3. Higher S raises F_a and F_c (worse sleep $\Rightarrow$ more fatigue) and gates down capacity gains.

Injury micro–damage $I(t)$.

I1. Increases with mechanical/metabolic stress, particularly u_S and high–intensity efforts u_H.

I2. Accrual is amplified by high F_a or F_c (poor mechanics, compromised tissue resilience).

I3. Clears with time, sleep, and adequate nutrition $n(t)$ (remodeling).

I4. A (soft) hazard from I contributes to regime switching/guard conditions in Section 4 and to performance penalties [7].

Derived outputs (for later use). We will define sport–specific *readiness* signals P_{end} and P_{str} as functions of A, N, F_a, F_c, S, I, to be used for evaluation and optimization in later sections.

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