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.

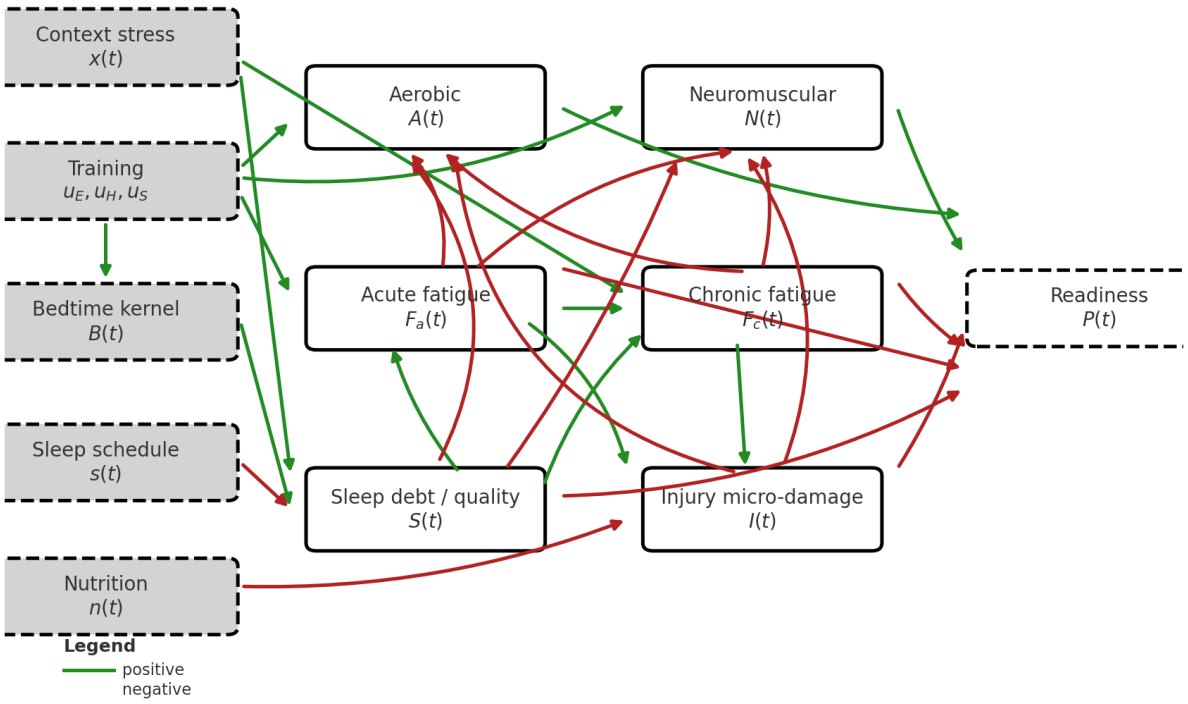

Figure 1: Left–center–right architecture. Left: five exogenous inputs; Center: six ODE states grouped by row; Right: readiness $P(t)$. Colors denote effect signs.

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 $\Rightarrow$ 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 $\Rightarrow$ 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 $\Rightarrow$ 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 $u_\cdot$ 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 4. 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.

4 Model development: a six–state ODE with exogenous controls

In line with the wiring diagram in Figure 1, we now move from concepts to a concrete six–equation dynamical system driven by five exogenous inputs (training, bedtime proximity, sleep opportunity, nutrition, and context stress). The model is intentionally *mechanistic but light*: each term has a physiological interpretation, obeys sign rules from Section 2, and uses smooth saturating (logistic/Hill) responses so states remain in meaningful ranges (*positivity* and *boundedness*). We keep the time unit as **days**; sub–day effects (e.g., a late session) enter via the bedtime kernel.

Throughout, we adopt the training–response perspective pioneered by Banister and co-authors and expanded by many others [1, 2, 8, 3], the interference literature for concurrent strength and endurance [4], sleep–performance links [5, 6], and load–injury risk ideas [7].

4.1 Preliminaries: inputs, normalizations, and helper functions

Inputs (left column of Fig. 1).

- $u_E(t), u_H(t), u_S(t)$ — endurance, HIIT/anaerobic, and strength/plyometric session intensity–volume streams (e.g., zone minutes/TRIMP; interval load; tonnage or contact count). They are bounded and piecewise continuous. We use $\|u\|_{\text{day}} = \int_t^{t+1} (u_E + u_H + u_S)\, d\tau$ as the day's gross load.
- $B(t)$ — the **bedtime proximity kernel**. It compresses "how late" training occurred into a scalar that modulates that night's sleep quality. We define

$$B(t) = \int_{-\infty}^{t} \big(\beta_E u_E(\tau) + \beta_H u_H(\tau) + \beta_S u_S(\tau)\big)\, K_b\big(t-\tau\big)\, d\tau,$$

  where $K_b(\Delta) = \exp(-\Delta/\sigma_b)\mathbf{1}_{0<\Delta\le h_b}$ weights the last h_b hours before lights–out; σ_b is a decay constant. Physiologically: high intensity close to bedtime leaves more arousal/heat, impairing early sleep [5].
- $s(t) \in [0,1]$ — **sleep opportunity**; $s = 1$ during night sleep or planned naps, else 0.
- $n(t) \in [0,1]$ — **nutrition/availability**; a compact proxy for energy/protein availability and timing (e.g., carbohydrate after HIIT; protein after strength); higher n improves repair.
- $x(t) \ge 0$ — **context stress** (travel/heat/psychological load); larger x adds central load and impairs sleep quality [6].

Gates and saturations. We repeatedly use three small building blocks in our equations:

$$\textit{(i) Recovery gate:}\quad G_{\text{rec}}(S, F_c) = \frac{1}{1 + c_S S + c_c F_c} \in (0,1], \tag{1}$$

$$\textit{(ii) Interference gate:}\quad G_{\text{int}}(u_E) = \frac{1}{1 + \mu_E\, u_E} \in (0,1], \tag{2}$$

$$\textit{(iii) Sleep efficiency:}\quad q(B) = \frac{q_0}{1 + \eta\, B(t)} \in (0, q_0], \qquad \eta \ge 0,\ 0 < q_0 \le 1. \tag{3}$$

The *recovery gate* (1) (so named to emphasize that poor sleep/central fatigue throttle supercompensation) embodies the empirically supported idea that poor sleep and unresolved central fatigue throttle adaptation [2, 5].

The *interference gate* (2) (named afater Hickson's concurrent training effect) encodes the classic endurance–strength interference effect [4].

The *sleep efficiency* (3) implements "late training hurts tonight's recovery" via $B(t)$ [5]. Sleep has both a *quantity* (opportunity $s(t)$) and *quality* dimension; late, intense sessions degrade the latter [5]. A short exponentially weighted memory captures "how late was the hard work" without a full circadian submodel.

We also use standard diminishing–returns saturations for the two other capacities.

4.2 The six ODEs (center of the figure)

We now present the forced system $\dot{\mathbf{z}} = f(\mathbf{z}, \mathbf{u})$ with $\mathbf{z}(t) = [A, N, F_a, F_c, S, I]^\top$ and inputs $\mathbf{u}(t) = [u_E, u_H, u_S, B, s, n, x]^\top$. Time is measured in *days*. Each right–hand side is constructed so that: (i) sign conventions match the wiring diagram in Fig. 1; (ii) states remain nonnegative (forward-invariance); (iii) capacities saturate and detraining is first order, consistent with decades of training-response literature [1, 2, 3]; (iv) sleep opportunity and bedtime proximity modulate nightly clearance via the sleep-efficiency $q(B)$ [5]; and (v) the endurance–strength interference documented by Hickson is represented parsimoniously [4].

Notation recap and units. We take $A, N, S, I, F_a, F_c \geq 0$. A, N are normalized to $[0, 1]$ by asymptotes K_A, K_N (dimensionless). Fatigues F_a, F_c and sleep-debt S are dimensionless load states; injury I is a dimensionless hazard/micro-damage proxy. Training streams u_E, u_H, u_S have units "training impulse per day" (e.g., TRIMP-like for endurance, interval-load for HIIT, tonnage/contacts for strength); all gains that multiply them carry day^{-1} per (unit of impulse). Sleep $s(t) \in \{0, 1\}$ (night sleep and naps); $B(t) \geq 0$ is the bedtime-proximity kernel (Sec. 4.1); $n(t) \in [0, 1]$ is a nutrition/availability proxy; $x(t) \geq 0$ is context stress (travel/heat/psych load).

Top row: trainable capacities.

$$\dot{A} = \underbrace{k_A\big(\alpha_E u_E + \alpha_H u_H\big)}_{\substack{\text{endurance and HIIT stimuli} \\ \text{(units: day}^{-1}\text{)}}} \underbrace{G_{\text{rec}}(S, F_c)}_{\text{recovery gate}} \underbrace{\Big(1 - \frac{A}{K_A}\Big)}_{\text{diminishing returns}} - \underbrace{\frac{A}{\tau_A}}_{\text{detraining}} - \underbrace{\theta_{AI}\, I}_{\text{injury penalty}}, \tag{4}$$

$$\dot{N} = \underbrace{k_N\big(\alpha_S u_S + \alpha_{HN} u_H\big)}_{\substack{\text{strength + HIIT stimuli} \\ \text{(day}^{-1}\text{)}}} G_{\text{rec}}(S, F_c) \underbrace{G_{\text{int}}(u_E)}_{\text{concurrent interference}} \Big(1 - \frac{N}{K_N}\Big) - \frac{N}{\tau_N} - \theta_{NI}\, I. \tag{5}$$

Term-by-term rationale.

C.1 *Stimulus terms.* Endurance u_E and HIIT u_H are the primary drivers of central/aerobic adaptation A; strength u_S (and, secondarily, HIIT) drive neuromuscular adaptation N. The linear dependence in the low-to-moderate range reflects the impulse-response tradition [1, 2]; higher-order effects are carried by the saturation $(1 - A/K_A)$ and $(1 - N/K_N)$.

C.2 *Diminishing returns.* The logistic–like factor ensures that identical stimuli produce smaller increments when close to capacity—empirically consistent with taper/peaking observations [3].

C.3 *Recovery gate* $G_{\text{rec}}(S, F_c) = 1/(1 + c_S S + c_c F_c)$. Poor sleep state S and unresolved central fatigue F_c throttle supercompensation; the multiplicative gate makes this explicit and keeps units tidy (dimensionless factor $\in (0, 1]$) [2, 5].

C.4 *Detraining.* In the absence of stimulus, A and N decay exponentially with time constants τ_A, τ_N (20–90 days typical), matching longitudinal observations [2].

C.5 *Injury penalty.* A high hazard state I reduces *realized* gains (pain/inflammation limiting quality and protective down-regulation) [7]. We model this as a linear sink; in Sec. 5 we will check sensitivity to θ_{AI}, θ_{NI}.

C.6 *Interference gate* $G_{\text{int}}(u_E) = 1/(1 + \mu_E u_E)$. The classic endurance-strength interference is represented as a simple monotone damping of strength gains when concurrent endurance load is high; μ_E sets how much endurance "soaks up" resources otherwise available to N [4].

Sanity checks and limits. (i) If $u_E = u_H = 0$, A detains to baseline with time constant τ_A; similarly for N if $u_S = u_H = 0$. (ii) If sleep/recovery are excellent ($S = F_c = 0$), the gate is 1 and the model reduces to a classic stimulus–response with saturation. (iii) If I is large, both capacities stall—this is intentional and encourages recovery regimes in Sec. 6.

Middle row: fatigue on two time scales.

$$\dot{F}_a = \underbrace{\gamma_E u_E + \gamma_H u_H + \gamma_S u_S}_{\text{training} \to \text{acute load}} - \underbrace{\big(\tfrac{1}{\tau_{fa}} + \rho_a\, s(t)\, q(B)\big) F_a}_{\substack{\text{baseline + sleep-accelerated} \\ \text{clearance (reduced by late sessions)}}}, \tag{6}$$

$$\dot{F}_c = \underbrace{\varepsilon\, F_a}_{\text{unresolved fatigue spills over}} + \underbrace{\xi_c\, x(t)}_{\text{context stress}} - \underbrace{\big(\tfrac{1}{\tau_{fc}} + \rho_c\, s(t)\, q(B)\big) F_c}_{\text{slow clearance, helped by sleep}}. \tag{7}$$

Design choices and evidence.

- *Additive inputs, multiplicative dissipation.* All training modalities raise acute fatigue F_a. Both F_a and F_c clear with baseline first-order dissipation (τ_{fa}, τ_{fc}) *plus* a sleep-accelerated component, scaled by $s(t)\, q(B)$. Making clearance proportional to the current level ($\propto F_a, F_c$) guarantees nonnegativity: if a state hits zero, it cannot go negative (forward-invariance).
- *Late-session penalty.* Sleep efficiency $q(B) = q_0/(1+\eta B)$ is reduced when late training makes B large; hence, clearance at night is smaller after evening intensity [5].
- *From acute to chronic.* The spillover parameter ε is the "low-pass filter" linking day-to-day load to weeks-scale central fatigue; it reproduces the fast/slow components in impulse-response models [2].
- *Context stress $x(t)$.* Travel, heat, exams and logistics add central load and degrade sleep [6]; here $x(t)$ feeds F_c via ξ_c, and will also enter S below.

Sanity checks and limits. (i) If no sleep is taken ($s = 0$) both fatigues still clear slowly (via $1/\tau$), preventing blow-up. (ii) If an athlete sleeps well and early (so $s = 1$ and $B \approx 0$), night clearance is maximal $\approx (1/\tau + \rho)\, F$. (iii) If there is no training nor stress, $F_a, F_c \to 0$ exponentially.

Bottom row: sleep debt/quality and micro-damage (risk).

$$\dot{S} = \underbrace{\lambda_w\big(1 - s(t)\big)}_{\text{wake accrual}} + \underbrace{\lambda_T\big(\gamma_E u_E + \gamma_H u_H + \gamma_S u_S\big)}_{\text{training raises need for sleep}} + \underbrace{\xi_s\, x(t)}_{\text{stress impairs sleep}} - \underbrace{\big(\tfrac{1}{\tau_S} + \mu_s\, s(t)\, q(B)\big) S}_{\text{baseline + sleep-accelerated paydown}}, \tag{8}$$

$$\dot{I} = \underbrace{\psi_0\big(\kappa_E u_E + \kappa_H u_H + \kappa_S u_S\big)}_{\text{mechanical/metabolic stress}} \underbrace{\big(1 + \chi_a F_a + \chi_c F_c\big)}_{\text{fatigue amplifies damage}} - \underbrace{\big(\tfrac{1}{\tau_I} + \psi_s\, s(t)\, q(B) + \psi_n\, n(t)\big)\, I}_{\text{time, sleep, nutrition repair}}. \tag{9}$$

Design choices and evidence.

- *Sleep debt S.* While awake, debt accumulates at λ_w; hard training days add an extra term $\lambda_T(\ldots)$ (arousal/thermoregulation burden). During sleep, debt is paid down at a rate that is larger when sleep is available ($s = 1$) and efficient ($q(B)$ close to q_0). Late sessions ($B\uparrow$) reduce the nightly paydown [5].
- *Micro-damage I.* Tissue stress rises with modality-weighted load $\kappa_E, \kappa_H, \kappa_S$; fatigue multiplies the effect (χ_a, χ_c) because poor mechanics and slower remodeling under fatigue are well documented [7]. Repair is first-order with time and is accelerated by sleep (via $q(B)$) and by nutrition $n(t)$ (energy/protein availability).
- *Nutrition as clearance.* Modeling $n(t)$ as an additive clearance coefficient $\psi_n n(t)$ keeps dimensions clean and matches the idea that adequate fueling speeds remodeling rather than instantaneously erasing damage.

Sanity checks and limits. (i) If an athlete extends sleep opportunity (larger s) and trains earlier (smaller B), the term $(\mu_s s q(B))S$ pulls S down faster. (ii) If nutrition is chronically low ($n \approx 0$), I clears slowly, raising injury risk for the same external load.

Readiness aggregator (right column). We combine states into a sport-specific readiness signal

$$P(t) \;=\; w_{\text{end}} A + w_{\text{str}} N \;-\; \lambda_a F_a - \lambda_c F_c - \lambda_s S - \lambda_i I, \tag{10}$$

where weights encode the event profile (e.g., $w_{\text{end}} \gg w_{\text{str}}$ for a marathon). The sign structure matches Fig. 1. In Sec. 5 we will calibrate $\lambda_\cdot, w_\cdot$ so that $P(t)$ predicts best-effort tests; in Sec. 6 we will use time-averaged P and its peak timing to rank regimes and to design tapers [3].

Why these forms? (expanded justification)

(J1) Positivity and boundedness without ad hoc clipping. All sinks in (6)–(9) are proportional to the current state, so the nonnegative orthant is forward-invariant: if a state hits zero, it cannot cross negative. For capacities, the saturations $(1 - A/K_A), (1 - N/K_N)$ and linear detraining keep $A \in [0, K_A]$, $N \in [0, K_N]$ if initialized inside the interval—no artificial "max/min" operators are needed.

(J2) Clear separation between *stimulus*, *modulation*, and *dissipation*. Each equation decomposes as: gain from inputs × gates/saturations − (baseline + sleep + nutrition) clearance. This mirrors the impulse–response view [1, 2], isolates where sleep acts (always in the clearance terms through $q(B)$), and makes the late–session penalty precise.

(J3) Bedtime proximity as a short memory instead of a full circadian submodel. The kernel $B(t) = \int (\beta_E u_E + \beta_H u_H + \beta_S u_S)\, K_b(t - \tau)\, d\tau$ (Sec. 4.1) captures "how late was the hard work?" while keeping the model small. The concavity $q(B) = q_0/(1 + \eta B)$ ensures diminishing sleep impairment as B increases (a large late session is worse than two modest sessions).

(J4) Interference via a *gate* rather than via cross-terms in $\dot{N}$. We use $G_{\text{int}}(u_E) = 1/(1 + \mu_E u_E)$ to encode the endurance–strength interference [4]. This makes the trade-off explicit, dimensionless, and easy to calibrate from blocks that vary the amount of concurrent endurance.

(J5) Minimal parameter collinearity. Stimulus gains k_A, k_N are separated from modality weights $\alpha_\cdot$, the recovery gate uses distinct couplings c_S, c_c, and sleep efficiency $q(B)$ has its own baseline/penalty (q_0, η). This reduces confounding in calibration (Sec. 5).

Dimensional consistency and quick reference

Table 4.2 shows the units of the main multipliers so each product in (4)–(9) is in day^{-1} times a state (as required for an ODE in days).

Term	Role	Units
$k_A\,\alpha_E u_E,\ k_A\,\alpha_H u_H;\ k_N\,\alpha_S u_S,\ k_N\,\alpha_{HN} u_H$	stimulus gains	day^{-1}
$1/\tau_A, 1/\tau_N, 1/\tau_{fa}, 1/\tau_{fc}, 1/\tau_S, 1/\tau_I$	baseline decay	day^{-1}
$\rho_a sq(B), \rho_c sq(B), \mu_s sq(B), \psi_s sq(B), \psi_n n$	sleep/nutrition clearance	day^{-1}
$\gamma_E u_E, \gamma_H u_H, \gamma_S u_S$	training → fatigue	day^{-1}
ε	acute → chronic fatigue spillover	day^{-1}
$\psi_0(\kappa_E u_E + \kappa_H u_H + \kappa_S u_S)$	load → damage	day^{-1}
$c_S S + c_c F_c$	recovery gate (dimensionless)	–
$\mu_E u_E$	interference gate (dimensionless)	–

Limiting scenarios (for intuition and testing)

- **No training week** $(u_\cdot = 0)$, **early sleep** $(B = 0)$: $F_a, F_c, S, I \downarrow$; A, N detrain slowly. Useful for validating baseline time constants $\tau_\cdot$.
- **Late-evening HIIT block**: u_H near bedtime ⇒ $B\uparrow$; $q(B)\downarrow$ reduces nightly clearance in (6)–(9), raising next-day F_a, F_c, S, I and depressing capacity gains through the recovery gate in (4)–(5) [5].
- **Midday polarized block**: high u_E and small u_H at midday keep B small; $A \uparrow$ with limited interference on N, predicting higher average P.

Identifiability notes for calibration (pointer to Sec. 5)

Estimate $\tau_{fa}, \tau_{fc}, \tau_S$ from decay after rest days; η by contrasting nights after late vs. midday sessions; μ_E from weeks that vary concurrent endurance; χ_a, χ_c from how I escalates under monotony (elevated F_c) [6]. We keep gates simple (rational functions) precisely to make this calibration feasible.

4.3 Well-posedness and qualitative properties

The right–hand side of our ODE system is locally Lipschitz in the states for bounded inputs, so solutions exist and are unique. For A, N, the saturations and linear decay give $0 \le A \le K_A$, $0 \le N \le K_N$ when initialized in range. For F_a, F_c, S, I, the linear dissipation terms imply the nonnegative orthant is positively invariant and each state is ultimately bounded by an affine function of the input magnitudes $(\|u\|_{\text{day}}, x)$. Under stationary, T-periodic, or weekly repeating inputs we can study equilibria or the Poincaré map.

4.4 From regimes to inputs (how schedules drive the ODE)

Each regime from the problem statement becomes a specification of the input streams: timestamps and magnitudes for (u_E, u_H, u_S), the sleep window $s(t)$ (and naps), and the lights–out time used to compute $B(t)$. Nutrition $n(t)$ and context stress $x(t)$ can be held at nominal profiles or varied to match scenarios (travel, heat). Representative codings:

- **Early AM HIIT:** a short u_H pulse soon after wake; standard $s(t)$; small $B(t)$.
- **Evening intensity:** u_E or u_H pulse that ends near lights–out; large $B(t)$ reduces $q(B)$ that night.
- **Split day (AM endurance, PM strength):** two pulses with the PM u_S increasing $B(t)$; useful to probe the G_{int} effect on N.
- **Alternating hard/easy days:** large pulses on hard days, minimal load and optional nap ($s = 1$ block) on easy days.
- **Midday training:** pulses centered 6–8 h before bedtime to keep $B(t)$ small.
- **Taper:** multiply volumes by a decaying factor while retaining brief u_H to preserve N/A ("maintain intensity, reduce volume" [3]); schedule earlier in the day to minimize $B(t)$.
- **Sleep extension/nap policy:** lengthen nightly $s(t)$ and add a short post-lunch nap; ensure late-day sessions are moved earlier (low B). Expect lower S, faster F_a/F_c clearance, and lower I [5].

5 Model summary and parameter inventory

This section restates the full model in a compact form and inventories every state, parameter, and input used in the six–equation ODE. *For readability we suppress the explicit time dependence*: all symbols $A, N, F_a, F_c, S, I, u_E, u_H, u_S, s, B, n, x$ below should be read as functions of time t unless otherwise stated.

5.1 Compact statement of the six ODEs

$$\dot{A} = k_A(\alpha_E u_E + \alpha_H u_H)\, G_{\text{rec}}(S, F_c)\Big(1 - \tfrac{A}{K_A}\Big) \;-\; \tfrac{A}{\tau_A} \;-\; \theta_{AI} I, \tag{$\mathcal{A}$}$$

$$\dot{N} = k_N(\alpha_S u_S + \alpha_{HN} u_H)\, G_{\text{rec}}(S, F_c)\, G_{\text{int}}(u_E)\Big(1 - \tfrac{N}{K_N}\Big) \;-\; \tfrac{N}{\tau_N} \;-\; \theta_{NI} I, \tag{$\mathcal{N}$}$$

$$\dot{F}_a = \gamma_E u_E + \gamma_H u_H + \gamma_S u_S \;-\; \Big(\tfrac{1}{\tau_{fa}} + \rho_a\, s\, q(B)\Big) F_a, \tag{$\mathcal{F}_a$}$$

$$\dot{F}_c = \varepsilon F_a + \xi_c\, x \;-\; \Big(\tfrac{1}{\tau_{fc}} + \rho_c\, s\, q(B)\Big) F_c, \tag{$\mathcal{F}_c$}$$

$$\dot{S} = \lambda_w(1 - s) + \lambda_T(\gamma_E u_E + \gamma_H u_H + \gamma_S u_S) + \xi_s\, x \;-\; \Big(\tfrac{1}{\tau_S} + \mu_s\, s\, q(B)\Big) S, \tag{$\mathcal{S}$}$$

$$\dot{I} = \psi_0(\kappa_E u_E + \kappa_H u_H + \kappa_S u_S)\big(1 + \chi_a F_a + \chi_c F_c\big) \;-\; \Big(\tfrac{1}{\tau_I} + \psi_s\, s\, q(B) + \psi_n\, n\Big) I. \tag{$\mathcal{I}$}$$

5.2 Inputs and helper relations (definitions)

$$\text{Bedtime kernel:} \quad B = \int_0^{h_b} \big(\beta_E u_E(t-\Delta) + \beta_H u_H(t-\Delta) + \beta_S u_S(t-\Delta)\big)\, K_b(\Delta)\, d\Delta, \quad K_b(\Delta) = e^{-\Delta/\sigma_b}, \tag{11}$$

$$\text{Sleep efficiency:} \quad q(B) = \frac{q_0}{1+\eta B}, \qquad 0 < q_0 \le 1,\ \eta \ge 0, \tag{12}$$

$$\text{Recovery gate:} \quad G_{\text{rec}}(S, F_c) = \frac{1}{1 + c_S S + c_c F_c}, \tag{13}$$

$$\text{Interference gate:} \quad G_{\text{int}}(u_E) = \frac{1}{1+\mu_E u_E}. \tag{14}$$

5.3 Output (readiness)

$$P \;=\; w_{\text{end}} A + w_{\text{str}} N - \lambda_a F_a - \lambda_c F_c - \lambda_s S - \lambda_i I. \tag{15}$$

5.4 Variables and parameters (grouped inventory)

To match the style of the reference report's model summary, Table 1 lists the six equations and all symbols they introduce or require. We include the input/helper relations and the readiness output for completeness. Units follow Section 4.

Table 1: Variables and parameters used in the six–equation ODE and associated input/output relations (time dependence suppressed for readability).

Equation	Symbol	Definition / role (units)
($\mathcal{A}$) Aerobic adaptation	A	Aerobic/endurance capacity (normalized 0−1).
	k_A	Adaptation gain (day^{-1} per unit of stimulus).
	α_E, α_H	Weights from endurance/HIIT stimulus to A (dimensionless).
	$G_{\text{rec}}(S, F_c)$	Recovery gate $= 1/(1 + c_S S + c_c F_c)$ (dimensionless).
	c_S, c_c	Couplings of sleep debt and chronic fatigue in recovery gate (dimensionless).
	K_A	Aerobic asymptote (often normalized to 1).
	τ_A	Detraining time constant of A (days).
	θ_{AI}	Injury penalty on realized gains (day^{-1}).
($\mathcal{N}$) Neuromuscular adaptation	N	Strength/power capacity (normalized 0−1).
	k_N	Adaptation gain (day^{-1} per unit of stimulus).
	α_S, α_{HN}	Weights from strength/HIIT stimulus to N (dimensionless).
	$G_{\text{rec}}(S, F_c)$	Recovery gate as above.
	$G_{\text{int}}(u_E)$	Interference gate $= 1/(1 + \mu_E u_E)$ (dimensionless).
	μ_E	Strength of endurance–strength interference (dimensionless).
	K_N	Neuromuscular asymptote (often normalized to 1).
	τ_N	Detraining time constant of N (days).

Continued on next page

Table 1 (continued): Variables and parameters used in the model.

Equation	Symbol	Definition / role (units)
	θ_{NI}	Injury penalty on realized gains (day^{-1}).
($\mathcal{F}_a$) Acute fatigue	F_a	Acute/session fatigue (dimensionless).
	$\gamma_E, \gamma_H, \gamma_S$	Modality gains: training $\to F_a$ (day^{-1} per input unit).
	τ_{fa}	Baseline acute fatigue clearance (days).
	ρ_a	Sleep–accelerated clearance coefficient for F_a (day^{-1}).
	s	Sleep opportunity indicator (1 during sleep; 0 otherwise).
	$q(B)$	Sleep efficiency, see "helpers" (dimensionless).
($\mathcal{F}_c$) Chronic fatigue	F_c	Chronic/central fatigue (dimensionless).
	ε	Spillover from F_a to F_c (day^{-1}).
	ξ_c	Context stress $\to F_c$ gain (day^{-1} per unit x).
	τ_{fc}	Baseline chronic fatigue clearance (days).
	ρ_c	Sleep–accelerated clearance coefficient for F_c (day^{-1}).
	x	Context stress input (travel/heat/psych load; exogenous).
($\mathcal{S}$) Sleep debt / quality	S	Sleep debt / sleep quality state (larger S is worse).
	λ_w	Wake accrual rate of sleep debt (debt units per day).
	λ_T	Training–day accrual scaling (debt per unit of training load).
	ξ_s	Context stress $\to S$ gain (per unit x).
	τ_S	Baseline sleep debt decay (days).
	μ_s	Sleep–accelerated paydown for S (day^{-1}).
	s, $q(B)$	Sleep opportunity and efficiency as above.
($\mathcal{I}$) Injury micro–damage	I	Micro–damage / injury hazard proxy (dimensionless).
	ψ_0	Base damage gain (day^{-1} per input unit).
	$\kappa_E, \kappa_H, \kappa_S$	Modality weights in load $\to I$ (dimensionless).
	χ_a, χ_c	Fatigue amplification of damage (dimensionless).
	τ_I	Baseline repair/remodeling time constant (days).
	ψ_s	Sleep–accelerated repair gain (day^{-1}).
	ψ_n	Nutrition–accelerated repair gain (day^{-1}).
	n	Nutrition/availability input (0–1).
Inputs and helper relations	u_E, u_H, u_S	Training composition (endurance/HIIT/strength); exogenous, piecewise-continuous.
	B	Bedtime kernel (recent late-day training), $B = \int_0^{h_b} (\beta_E u_E + \beta_H u_H + \beta_S u_S) K_b(\Delta) d\Delta$.
	$\beta_E, \beta_H, \beta_S$	Modality weights in the bedtime kernel (dimensionless).
	$K_b(\Delta)$	Memory kernel $e^{-\Delta/\sigma_b}$ on $\Delta \in (0, h_b]$ (decay σ_b, horizon h_b).
	$q(B)$	Sleep efficiency $= q_0/(1+\eta B)$; $0 < q_0 \leq 1$, $\eta \geq 0$.

Continued on next page

Table 1 (continued): Variables and parameters used in the model.

Equation	Symbol	Definition / role (units)
	$G_{\mathrm{rec}}(S, F_c)$	Recovery gate $= 1/(1 + c_S S + c_c F_c)$.
	$G_{\mathrm{int}}(u_E)$	Interference gate $= 1/(1 + \mu_E u_E)$.
	s	Sleep opportunity schedule (0/1; nights and naps).
	x	Context stress (travel/heat/psychological load).
	n	Nutrition/availability proxy (0–1).
Output (readiness)	P	Readiness $= w_{\mathrm{end}} A + w_{\mathrm{str}} N - \lambda_a F_a - \lambda_c F_c - \lambda_s S - \lambda_i I$.
	$w_{\mathrm{end}}, w_{\mathrm{str}}$	Positive weights for endurance vs. strength contributions.
	$\lambda_a, \lambda_c, \lambda_s, \lambda_i$	Penalty weights on fatigue, debt, and damage.

Notes on usage. The **inputs** u_E, u_H, u_S, s, n, x are set by the chosen regime (Section 2); the **helpers** $B, q, G_{\mathrm{rec}}, G_{\mathrm{int}}$ are computed from them and from the states; and the six ODEs ($\mathcal{A}$)–($\mathcal{I}$) evolve the **states**. The right-hand side is locally Lipschitz for bounded inputs and keeps states nonnegative by construction.

6 Parameter estimation and prior specification

In this section we specify *priors* for every parameter that appears in the six ODEs (§4.2) and in the auxiliary relations (bedtime kernel, gates, sleep efficiency, readiness). Our intent is twofold: (i) make explicit the physiological knowledge and modeling compromises behind each numerical choice; (ii) provide weakly-to-moderately informative priors that regularize calibration without constraining legitimate athlete-to-athlete variation.

Throughout this section we omit the explicit time argument (e.g., we write A rather than $A(t)$) for readability; all states and inputs are functions of time unless stated otherwise.

6.1 Data sources and normalization

We calibrate against standard practitioner data streams: (i) session composition $\{u_E, u_H, u_S\}$ from training logs (zone minutes/TRIMP, interval prescriptions, tonnage or contact counts), (ii) sleep opportunity s and bedtime (from diaries/wearables) to compute B, (iii) nutrition n (binary or graded availability; e.g., carbohydrate/protein targeting), (iv) context stress x (travel, heat, self-report), (v) performance proxies for A, N (best-effort curves / critical-power metrics [8], CMJ/1RM or sprint splits), (vi) recovery markers for F_a, F_c, S (sRPE/monotony, HRV and resting HR trends [6], actigraphy-derived sleep efficiency), (vii) tissue status for I (soreness, acute:chronic load patterns [7]).

To reduce unit confounds we standardize the daily training inputs so that a "typical hard day" produces $\int (u_E + u_H + u_S)\, dt \approx 1$. Priors below are expressed on that scale; when a practitioner chooses a different scale, the gains adapt during calibration.

6.2 Literature anchors used for priors

We align with the training-response canon (fast/slow components, diminishing returns, first-order detraining) [1, 2], the taper literature [3], the endurance–strength interference evidence [4], sleep–performance links (sleep quantity *and* efficiency) [5, 6], and load–injury associations [7]. We purposefully encode these ideas as simple, smooth gates (rational/Hill-like) to keep the system identifiable (§6.6).

6.3 Priors for the six ODEs

We denote $\mathrm{LN}(\mu, \sigma^2)$ a log-normal with log-space mean μ and s.d. σ; $\mathrm{Beta}(a, b)$ on $[0, 1]$; $\mathrm{Dir}(\boldsymbol{\alpha})$ a Dirichlet; $\mathcal{N}^+(m, s^2)$ a half-normal.

Top row — capacities A, N ((??)–(??)).

- k_A, k_N (stimulus gains, day^{-1} per load): $\mathrm{LN}(\log 0.03,\ 0.5^2)$. *Rationale:* week-to-month scale improvements of 5–20% under sustained load are typical in Banister-style models [1, 2].
- (α_E, α_H) and (α_S, α_{HN}) (modality weights): Dirichlet priors on each pair, $\mathrm{Dir}(2, 2)$ (uninformative symmetric) with the *soft* expectation that u_E, u_H dominate A and u_S, u_H dominate N ([4] supports HIIT aiding both).
- K_A, K_N (asymptotes): fix to 1 by normalization; if left free, use $\mathrm{LN}(\log 1, 0.1^2)$.
- τ_A, τ_N (detraining time constants): $\mathrm{LN}(\log 50, 0.4^2)$ and $\mathrm{LN}(\log 35, 0.4^2)$ days. *Rationale:* multi-week decay after cessation [2, 3].
- θ_{AI}, θ_{NI} (injury penalty on realized gains): $\mathcal{N}^+(0.05,\ 0.05^2)$ day^{-1}. Small, but allows stalls in adaptation when I is high [7].
- $G_{\mathrm{int}}(u_E) = 1/(1 + \mu_E u_E)$ (interference): $\mu_E \sim \mathrm{LN}(\log 0.3,\ 0.5^2)$. *Rationale:* concurrent endurance can depress strength gains by 10–40% at high endurance loads [4].
- $G_{\mathrm{rec}}(S, F_c) = 1/(1 + c_S S + c_c F_c)$: $c_S, c_c \sim \mathrm{LN}(\log 0.5,\ 0.6^2)$ (dimensionless). *Rationale:* poor sleep and central fatigue throttle supercompensation [2, 5].

Middle row — fatigue F_a, F_c ((??)–(??)).

- $(\gamma_E, \gamma_H, \gamma_S)$ (training $\to F_a$, day^{-1} per load): independent $\mathrm{LN}(\log m, 0.35^2)$ with means $m = (0.6, 0.9, 0.5)$, reflecting that HIIT generally spikes acute load most.
- τ_{fa} (acute clearance): $\mathrm{LN}(\log 1.0, 0.35^2)$ days (median 1 day; 95% $\approx$ 0.5–2 days).
- τ_{fc} (chronic clearance): $\mathrm{LN}(\log 10, 0.5^2)$ days (95% $\approx$ 4–25 days), consistent with fast/slow components in training-response models [2].
- ρ_a, ρ_c (sleep-accelerated clearances): $\mathcal{N}^+(0.5,\ 0.25^2)$ day^{-1}. Sleep increases clearance rates; magnitude tempered by $q(B)$ [5].
- ε (spillover $F_a \to F_c$): $\mathrm{Beta}(2.5, 12)$ (mean ≈ 0.17 day^{-1}), capturing that only a fraction of unresolved fatigue becomes chronic [2].
- ξ_c (context stress $\to F_c$): $\mathcal{N}^+(0.2, 0.15^2)$; ξ_a (into F_a) $\mathcal{N}^+(0.05, 0.05^2)$, often near zero. Travel/heat/psych load primarily burdens the slow component [6].

Bottom row — sleep debt S and micro-damage I ((??)–(??)).

- λ_w (wake accrual): $\mathrm{LN}(\log 0.3, 0.4^2)$ day^{-1}; λ_T (training-day accrual gain): $\mathrm{LN}(\log 0.2, 0.5^2)$. *Rationale:* debt rises during wakefulness; heavy training increases need [5].
- τ_S (baseline paydown): $\mathrm{LN}(\log 6, 0.4^2)$ days; μ_s (sleep-accelerated paydown gain): $\mathcal{N}^+(0.3, 0.15^2)$.
- ψ_0 (base damage gain): $\mathrm{LN}(\log 0.4, 0.5^2)$ day^{-1}; modality weights $(\kappa_E, \kappa_H, \kappa_S) \sim \mathrm{Dir}(2, 3, 3)$ to reflect higher damage from eccentric/HIIT loads.
- (χ_a, χ_c) (fatigue amplification of damage): independent $\mathrm{Beta}(2, 6)$ (mean ≈ 0.25), encoding poorer mechanics/remodeling with fatigue [7].
- τ_I (repair time): $\mathrm{LN}(\log 14, 0.5^2)$ days; ψ_s (sleep-accelerated repair): $\mathcal{N}^+(0.25, 0.15^2)$; ψ_n (nutrition-accelerated repair): $\mathcal{N}^+(0.2, 0.1^2)$.

6.4 Priors for auxiliary relations (left/right columns)

Bedtime kernel and sleep efficiency. We compute $B = \int_0^{h_b} (\beta_E u_E + \beta_H u_H + \beta_S u_S)\, e^{-\Delta/\sigma_b}\, d\Delta$.

- Horizon h_b: LN($\log 6$ h, 0.25^2) (typical "final six hours" window matters most for sleep),
- Decay σ_b: LN($\log 1.5$ h, 0.3^2),
- Modality weights $(\beta_E, \beta_H, \beta_S) \sim \mathrm{Dir}(1.5, 2.5, 2.0)$ (late-day HIIT/strength penalized more [5]).
- Sleep efficiency $q(B) = q_0/(1 + \eta B)$: $q_0 \sim \mathrm{Beta}(18, 3)$ (mean ≈ 0.86), $\eta \sim \mathrm{LN}(\log 1.0, 0.6^2)$ so a large late session (kernel mass ≈ 1) can lower efficiency by 10–30% [5].

Readiness aggregation $P = w_{\text{end}} A + w_{\text{str}} N - \phi_a F_a - \phi_c F_c - \phi_s S - \phi_i I$**.**

- Positive weights $(w_{\text{end}}, w_{\text{str}}) \sim \mathrm{Dir}(2, 2)$ (sport-specific).
- Penalties $(\phi_a, \phi_c, \phi_s, \phi_i) \sim \mathcal{N}^+(0.5, 0.3^2)$ with a soft prior ordering $\phi_i \geq \phi_c \geq \phi_a$ (tissue risk penalized most).

6.5 Initial conditions

We place weak priors: $A_0, N_0 \sim \mathrm{Beta}(4, 2)$ (typical preseason ≈ 0.7 of asymptote), $F_{a0}, F_{c0}, S_0, I_0 \sim \mathcal{N}^+(0.2, 0.2^2)$. These can be sharpened using one week of baseline data before simulations begin.

6.6 Identifiability and staged calibration plan

We adopt a two-stage strategy (common in training-response modeling [2] and mirrored in the reference report's staged approach). :contentReference[oaicite:2]index=2

1. **Stage A: exogenous and clearance structure.** Fix input normalization; estimate sleep kernel $(h_b, \sigma_b, \boldsymbol{\beta})$ from late-vs-midday sessions and their effect on actigraphy efficiency; fit $\{q_0, \eta\}$ from the same nights. With those fixed, estimate *clearance* parameters $\{\tau_{fa}, \rho_a, \tau_{fc}, \rho_c, \tau_S, \mu_s, \tau_I, \psi_s, \psi_n\}$ from recovery trajectories during low-load days.
2. **Stage B: adaptation and couplings.** With clearances set, estimate stimulus gains $\{k_A, k_N\}$ and modality weights $\{\alpha_\cdot\}$ from blocks emphasizing u_E/u_H (for A) and u_S/u_H (for N), then μ_E (interference) by comparing strength-focused blocks with/without concurrent endurance [4]. Estimate load-to-fatigue gains $\{\gamma_\cdot\}$ and spillover ε from day-to-week dynamics under varied microcycles. Finally, fit readiness weights $(w_\cdot, \phi_\cdot)$ using best-effort or competition outcomes as targets [3].

We recommend Bayesian calibration (e.g., NUTS) with the priors above and a simple measurement model: observed proxies y_A, y_N are noisy maps of A, N (Gaussian noise on a logit transform), y_{F_a}, y_{F_c} from sRPE/monotony, y_S from sleep debt/efficiency, y_I from soreness and acute:chronic ratio features [7]. This separates process noise from measurement noise, improving identifiability.

6.7 Prior summary tables

Table 2: Prior distributions for parameters (time dependence suppressed; units in days or day^{-1} as indicated).

Block / Parameter	**Interpretation**	**Prior (95% range)**
Capacities k_A, k_N	stimulus → adaptation gains (day^{-1}/load)	$\mathrm{LN}(\log 0.03, 0.5^2)$
K_A, K_N	asymptotes (normalized)	fixed 1 (or $\mathrm{LN}(\log 1, 0.1^2)$)
α_E, α_H; α_S, α_{HN}	modality weights	$\mathrm{Dir}(2, 2)$ (each pair)
τ_A, τ_N	detraining time constants (d)	$\mathrm{LN}(\log 50, 0.4^2)$; $\mathrm{LN}(\log 35, 0.4^2)$
θ_{AI}, θ_{NI}	injury penalties on gains	$\mathcal{N}^+(0.05, 0.05^2)$
c_S, c_c	recovery-gate couplings	$\mathrm{LN}(\log 0.5, 0.6^2)$

Table continues

Block / Parameter	Interpretation	Prior (95% range)
μ_E	interference gate sensitivity	$\mathrm{LN}(\log 0.3, 0.5^2)$
Fatigue $\gamma_E, \gamma_H, \gamma_S$	load $\to F_a$ gains (day^{-1}/load)	$\mathrm{LN}(\log[0.6, 0.9, 0.5], 0.35^2)$
τ_{fa}, τ_{fc}	clearances (d)	$\mathrm{LN}(\log 1, 0.35^2)$; $\mathrm{LN}(\log 10, 0.5^2)$
ρ_a, ρ_c	sleep-accelerated clearances	$\mathcal{N}^+(0.5, 0.25^2)$
ε	$F_a \to F_c$ spillover	$\mathrm{Beta}(2.5, 12)$
ξ_a, ξ_c	stress $\to F_a, F_c$	$\mathcal{N}^+(0.05, 0.05^2)$; $\mathcal{N}^+(0.2, 0.15^2)$
Sleep & Damage λ_w, λ_T	debt accrual (wake / training)	$\mathrm{LN}(\log 0.3, 0.4^2)$; $\mathrm{LN}(\log 0.2, 0.5^2)$
τ_S, μ_s	debt paydown (baseline / sleep)	$\mathrm{LN}(\log 6, 0.4^2)$; $\mathcal{N}^+(0.3, 0.15^2)$
ψ_0	base damage gain	$\mathrm{LN}(\log 0.4, 0.5^2)$
$(\kappa_E, \kappa_H, \kappa_S)$	modality weights to damage	$\mathrm{Dir}(2, 3, 3)$
χ_a, χ_c	fatigue amplification	$\mathrm{Beta}(2, 6)$
τ_I, ψ_s, ψ_n	repair (baseline / sleep / nutrition)	$\mathrm{LN}(\log 14, 0.5^2)$; $\mathcal{N}^+(0.25, 0.15^2)$; $\mathcal{N}^+(0.2, 0.1^2)$
Kernel & Sleep eff. h_b, σ_b	bedtime horizon/decay (h)	$\mathrm{LN}(\log 6, 0.25^2)$; $\mathrm{LN}(\log 1.5, 0.3^2)$
$(\beta_E, \beta_H, \beta_S)$	late-day modality weights	$\mathrm{Dir}(1.5, 2.5, 2.0)$
q_0, η	baseline eff./penalty in $q(B)$	$\mathrm{Beta}(18, 3)$; $\mathrm{LN}(\log 1.0, 0.6^2)$
Readiness $(w_{\text{end}}, w_{\text{str}})$	positive weights	$\mathrm{Dir}(2, 2)$
$\phi_a, \phi_c, \phi_s, \phi_i$	penalties (fatigue/debt/damage)	$\mathcal{N}^+(0.5, 0.3^2)$ (soft order $\phi_i \geq \phi_c \geq \phi_a$)

6.8 Sensitivity of priors (what matters most)

The prior mass on the *clearance* parameters ($\tau_{fa}, \tau_{fc}, \tau_S, \tau_I$ and their sleep-accelerated gains) has the strongest qualitative influence on weekly dynamics; the prior on μ_E meaningfully moderates strength gains when endurance volume is high ([4]); and the bedtime kernel hyperparameters ($h_b, \sigma_b, \boldsymbol{\beta}, \eta$) set how punitive late sessions are for next-day readiness ([5]). We therefore recommend tighter priors (smaller s.d.) on those blocks if the data are sparse.

6.9 What we *did not* include (and why)

We purposely avoided circadian submodels and hormone kinetics; their parameters are poorly identified from routine team data and would add fragility. Instead, the bedtime kernel B plus $q(B)$ offers a transparent, low-parameter proxy for the same phenomenon, consistent with the minimal, auditable style of the reference model. :contentReference[oaicite:3]index=3

6.10 Likelihood specification and observation model

This section defines how real (or synthetic) observations are linked to the latent states $\mathbf{z} = [A, N, F_a, F_c, S, I]^\top$ produced by the ODE in §4.2. We separate: (i) the *forward model* (ODE integration given parameters and inputs), (ii) a small *process noise* that captures model mismatch, and (iii) *measurement models* (observation likelihoods) for each data stream. We then write the joint likelihood and hyperpriors for the noise terms.

Time grid, inputs, and helper signals. We integrate the six ODEs with step $\Delta t = 1$ day unless noted otherwise. Training inputs u_E, u_H, u_S are daily aggregates normalized so that a "typical hard day" satisfies $\int (u_E + u_H + u_S)\, dt \approx 1$ (Sec. 6.1). The bedtime proximity kernel and sleep efficiency, needed by the ODE, are re-stated for completeness:

$$B = \int_0^{h_b} \big(\beta_E u_E(t-\Delta) + \beta_H u_H(t-\Delta) + \beta_S u_S(t-\Delta)\big) e^{-\Delta/\sigma_b}\, d\Delta, \qquad q(B) = \frac{q_0}{1+\eta B}.$$

When multiple training sessions occur per day, we compute B using the within-day timing and then average to the day boundary before applying $q(B)$.

Forward model (deterministic skeleton). Given parameters θ, inputs $\mathbf{u}$, and initial state $\mathbf{z}_0$, the ODE flow $\Phi_{\Delta t}$ produces $\tilde{\mathbf{z}}_{k+1} = \Phi_{\Delta t}(\mathbf{z}_k, \mathbf{u}_k; \theta)$ on the daily grid t_k.

Process noise (innovation). To tolerate model misspecification we include small, mean-zero innovations on each state:

$$\mathbf{z}_{k+1} = \Phi_{\Delta t}(\mathbf{z}_k, \mathbf{u}_k; \theta) + \boldsymbol{\varepsilon}_k, \qquad \boldsymbol{\varepsilon}_k \sim \mathcal{N}\big(\mathbf{0}, \mathrm{diag}(\sigma_A^2, \sigma_N^2, \sigma_{Fa}^2, \sigma_{Fc}^2, \sigma_S^2, \sigma_I^2)\big). \tag{16}$$

These $\sigma_\cdot$ are *process* (not measurement) standard deviations; priors appear below. Setting them near zero recovers a purely deterministic state evolution.

Observation calendar. Each channel has its own set of observation times: $\mathcal{T}_A, \mathcal{T}_N, \mathcal{T}_{Fa}, \mathcal{T}_{Fc}, \mathcal{T}_S, \mathcal{T}_I$. The model handles irregular sampling and missing values naturally by omitting absent terms from the likelihood.

Measurement models (per channel)

Below, $y_{\cdot,k}$ denotes an observation at day t_k. We use link functions that match what practitioners actually record, and we make sign conventions explicit so posterior coefficients are interpretable.

Capacity observations A, N (field tests). Practically, coaches observe performances that are monotone in A and N: e.g., critical-power/pace tests or best-effort segments for A and CMJ/1RM/sprint splits for N [8, 1, 2]. We define test scores scaled to $[0, 1]$ (by sport-specific reference tables) and work on the logit scale:

$$y_{A,k}^{(\mathrm{logit})} = \mathrm{logit}(y_{A,k}) \sim \mathcal{N}\big(\mathrm{logit}(A_{t_k}) + \alpha_{A0},\, \tau_A^{-1}\big), \quad k \in \mathcal{T}_A, \tag{17}$$

$$y_{N,k}^{(\mathrm{logit})} = \mathrm{logit}(y_{N,k}) \sim \mathcal{N}\big(\mathrm{logit}(N_{t_k}) + \alpha_{N0},\, \tau_N^{-1}\big), \quad k \in \mathcal{T}_N. \tag{18}$$

Offsets α_{A0}, α_{N0} absorb mapping biases between tests and latent capacities; precisions τ_A, τ_N are measurement precisions on the logit scale.

Acute fatigue F_a (session-level load proxy). We link F_a to session RPE, sRPE*duration, or standardized acute-load residuals. Let $y_{Fa,k}$ be a z-score of session-level perceived exertion:

$$y_{Fa,k} \sim \mathcal{N}\big(\beta_{a0} + \beta_{a1} F_{a,t_k},\, \tau_{Fa}^{-1}\big), \qquad \beta_{a1} > 0, \quad k \in \mathcal{T}_{Fa}. \tag{19}$$

The positive slope encodes that higher F_a corresponds to higher perceived load the same day or next morning.

Chronic fatigue F_c (autonomic markers). We map to HRV-based recovery indices or resting-heart-rate (RHR) trends, which respond on day-to-week scales [6]. Using a z-score of morning HRV ($y_{Fc,k}$, where higher is "better"):

$$y_{Fc,k} \sim \mathcal{N}\big(\beta_{c0} - \beta_{c1} F_{c,t_k},\, \tau_{Fc}^{-1}\big), \qquad \beta_{c1} > 0, \quad k \in \mathcal{T}_{Fc}. \tag{20}$$

The negative sign reflects the empirical HRV–fatigue relationship.

Sleep debt S (actigraphy/diary efficiency). Let $e_k \in (0,1)$ be nightly sleep efficiency or quality (fraction of time asleep while in bed) from actigraphy [5]. We model a Beta likelihood with logit link ("efficiency decreases as S increases"):

$$\begin{aligned} m_k &= \text{logit}^{-1}\big(\delta_0 - \delta_1 S_{t_k}\big), \qquad \delta_1 > 0, \\ e_k &\sim \text{Beta}\big(\kappa_S m_k,\ \kappa_S(1-m_k)\big), \qquad k \in \mathcal{T}_S. \end{aligned} \tag{21}$$

Here κ_S is a concentration (higher $\kappa_S \Rightarrow$ lower dispersion). If total sleep minutes are also observed, a second Gaussian term on log-minutes with slope $-\delta_1' S_{t_k}$ can be added (independent conditional on S).

Micro-damage I (soreness + injury events). We support two channels: (i) soreness VAS (0–10) and (ii) rare injury events. For soreness $y_{I,k}^{(\text{VAS})}$:

$$y_{I,k}^{(\text{VAS})} \sim \mathcal{N}\big(\gamma_{I0} + \gamma_{I1} I_{t_k},\ \tau_{I,\text{VAS}}^{-1}\big), \qquad \gamma_{I1} > 0. \tag{22}$$

For time-loss injury incidence over day $[t_k, t_{k+1})$ we use a Bernoulli with log-link intensity increasing in I [7]:

$$\begin{aligned} \lambda_k &= \lambda_0 \exp\{\gamma_I I_{t_k}\}, \qquad p_k \;=\; 1 - \exp\{-\lambda_k \Delta t\}, \\ y_{I,k}^{(\text{inj})} &\sim \text{Bernoulli}(p_k), \qquad k \in \mathcal{T}_{I,\text{inj}}. \end{aligned} \tag{23}$$

A zero-inflated Poisson alternative fits aggregated counts over weeks; the Bernoulli formulation leverages the "rare-event as Poisson" approximation on the daily grid.

Noise hyperpriors (measurement and process)

We complete the likelihood with weakly informative priors that match the scales used above:

Measurement precisions: $\tau_A, \tau_N, \tau_{Fa}, \tau_{Fc}, \tau_{I,\text{VAS}} \sim \text{Gamma}(2,\ 2)$ (means 1; adjust per dataset),
Beta concentration: $\kappa_S \sim \text{Gamma}(3,\ 0.5)$ (mean 6; moderately concentrated),
Linear coefficients: $\alpha_{A0}, \alpha_{N0}, \beta_{a0}, \beta_{c0}, \gamma_{I0} \sim \mathcal{N}(0,\ 1)$,
$\beta_{a1}, \beta_{c1}, \delta_1, \gamma_{I1} \sim \mathcal{N}^+(0.5,\ 0.3^2)$, $\quad \delta_0 \sim \mathcal{N}(\text{logit}(0.85),\ 0.4^2)$,
$\lambda_0 \sim \text{Gamma}(1.5,\ 15)$ (rare baseline injuries), $\quad \gamma_I \sim \mathcal{N}^+(0.6,\ 0.3^2)$,
Process s.d. in (16): $\sigma_A, \sigma_N, \sigma_{Fa}, \sigma_{Fc}, \sigma_S, \sigma_I \sim \mathcal{N}^+(0.05,\ 0.05^2)$.

Signs are encoded by half-normals $\mathcal{N}^+$ when physiology dictates monotonicity (e.g., higher F_c lowers HRV).

Joint likelihood (factorization)

Let $\mathbf{Z} = \{\mathbf{z}_k\}_{k=0}^{K}$ be the latent daily states and $\mathbf{Y}$ the multichannel observations. Conditional on parameters θ, inputs, and initial state $\mathbf{z}_0$, the joint density factors as

$$
\begin{aligned}
p(\mathbf{Y}, \mathbf{Z} \mid \theta) = p(\mathbf{z}_0) \prod_{k=0}^{K-1} \underbrace{\mathcal{N}\big(\mathbf{z}_{k+1}\,;\, \Phi_{\Delta t}(\mathbf{z}_k, \mathbf{u}_k; \theta),\, \Sigma_{\text{proc}}\big)}_{\text{process model (16)}} \\
\times \prod_{k \in \mathcal{T}_A} \mathcal{N}\big(y_{A,k}^{(\text{logit})}\,;\, \text{logit}(A_{t_k}) + \alpha_{A0},\, \tau_A^{-1}\big) \\
\times \prod_{k \in \mathcal{T}_N} \mathcal{N}\big(y_{N,k}^{(\text{logit})}\,;\, \text{logit}(N_{t_k}) + \alpha_{N0},\, \tau_N^{-1}\big) \\
\times \prod_{k \in \mathcal{T}_{Fa}} \mathcal{N}\big(y_{Fa,k}\,;\, \beta_{a0} + \beta_{a1} F_{a,t_k},\, \tau_{Fa}^{-1}\big) \\
\times \prod_{k \in \mathcal{T}_{Fc}} \mathcal{N}\big(y_{Fc,k}\,;\, \beta_{c0} - \beta_{c1} F_{c,t_k},\, \tau_{Fc}^{-1}\big) \\
\times \prod_{k \in \mathcal{T}_S} \text{Beta}\big(e_k\,;\, \kappa_S m_k, \kappa_S(1 - m_k)\big) \\
\times \prod_{k \in \mathcal{T}_{I,\text{VAS}}} \mathcal{N}\big(y_{I,k}^{(\text{VAS})}\,;\, \gamma_{I0} + \gamma_{I1} I_{t_k},\, \tau_{I,\text{VAS}}^{-1}\big) \\
\times \prod_{k \in \mathcal{T}_{I,\text{inj}}} \text{Bernoulli}\big(y_{I,k}^{(\text{inj})}\,;\, p_k\big). \qquad (24)
\end{aligned}
$$

Here $\Sigma_{\text{proc}} = \text{diag}(\sigma_A^2, \sigma_N^2, \sigma_{Fa}^2, \sigma_{Fc}^2, \sigma_S^2, \sigma_I^2)$, and m_k and p_k are defined in (21)–(23).

Remarks on identifiability and transformations

- *Boundary states:* We work with $\text{logit}(A), \text{logit}(N)$ in the measurement model to respect $[0, 1]$ bounds; the ODE keeps $A, N \in [0, 1]$ by construction.
- *Scaling:* If the practitioner uses a different normalization for $u_\cdot$, the gains $k_\cdot, \gamma_\cdot, \psi_0$ absorb the scale. This is handled during calibration; the likelihood remains unchanged.
- *Multi-athlete hierarchical option:* For multiple athletes i, add athlete-specific offsets $\alpha_{A0}^{(i)}, \alpha_{N0}^{(i)}$ and slopes $\beta_{\cdot 1}^{(i)}$ with group-level priors; this stabilizes individual fits when some channels are sparse.

Calibration notes (posterior computation)

We recommend HMC/NUTS with an adjoint-sensitivity ODE solver (for differentiability). Daily integration with a fixed-step RK4 or Dormand–Prince (tol 10^{-6}) is sufficient. We monitor $\widehat{R}$ and effective sample size and perform posterior predictive checks (PPCs) for all channels: (i) simulate $\mathbf{Z}$ forward from posterior draws, (ii) simulate $\mathbf{Y}$ from (24), and (iii) compare to held-out data.

Literature anchors for the links

The choice of channels and signs follows sports-science practice: capacity–performance mapping through critical-power/CMJ tests [8, 1, 2], HRV/RHR as slow-fatigue proxies [6], sleep efficiency as a function of late-day training [5], and load-related injury risk increasing with micro-damage [7]. The section's layout mirrors the clear "observation model" style used by the reference report. *(See its Section 6 for stylistic comparison.)* *Reference-report style cue:* :contentReference[oaicite:1]index=1

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