

Sleep as Obligate Phenotypic Switching: An Ultimate Explanation for Temporal Niche Partitioning

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Abstract

Why does natural selection mandate an involuntary, internally generated state change rather than permit flexible behavioral inactivity? Existing theories explain what sleep accomplishes – synaptic homeostasis, metabolite clearance, energy conservation – but these are proximate mechanisms, not ultimate explanations: themselves products of selection, yet adaptations acquired by the sleeping state rather than reasons the state exists. Here I propose that sleep evolved as an obligate phenotypic switching mechanism enabling temporal niche partitioning within a single genome. Without enforced state transitions, natural selection would drive organisms toward a compromised generalist phenotype suboptimal for both diurnal and nocturnal environments. Niche breadth theory predicts that the predictable, high-frequency, mutually exclusive nature of day–night cycling favors obligate switching over temporal generalism. This framework situates restorative theories as derived proximate mechanisms serving the ancestral ultimate function, differs from adaptive inactivity in addressing the ultimate rather than the proximate question, and generates testable predictions confirmed by cavefish sleep loss, cross-kingdom rest–activity cycling, and the ecological scaling of sleep duration and architecture. The theory extends to all periodic biological rhythms driven by predictable environmental oscillation.

1. Sleep theories explain what sleep does, not why it exists

Despite decades of intensive research, no consensus exists on why sleep evolved. The question is not trivial. Sleep and sleep-like states extend across kingdoms: sleep is universal among animals, present in organisms ranging from mammals to nematodes to jellyfish, and analogous circadian dormancy and nyctinastic leaf movement have been described in plants since Darwin's *The Power of Movement in Plants* (1880) and are the subject of ongoing molecular investigation (Ueda et al., 2019). Its deprivation produces severe fitness consequences including death. Yet the evolutionary origin of sleep – the selection pressure that first favored an involuntary, internally generated state of reduced arousal and environmental responsiveness – remains unresolved.

The difficulty lies in a persistent conflation of two distinct levels of biological explanation. Mayr (1961) formalized the distinction between proximate causes, which describe how a biological process operates within an individual, and ultimate causes, which explain why natural selection favored that process over evolutionary time. Tinbergen (1963) elaborated this framework into four complementary questions: mechanism, ontogeny, adaptive value, and phylogeny. The vast majority of sleep research addresses mechanism and ontogeny – proximate questions – while treating adaptive value as self-evident or secondary.

Consider the dominant theories. The synaptic homeostasis hypothesis holds that sleep permits the downscaling of synaptic connections potentiated during wakefulness, restoring the brain's capacity for new learning (Cirelli & Tononi, 2008). The glymphatic clearance hypothesis proposes that sleep enables the removal of metabolic waste products, including amyloid-beta, through convective flow in the interstitial space (Xie et al., 2013). Energy restoration theories posit that sleep replenishes cellular energy stores, particularly brain glycogen, depleted during wakefulness (Benington & Heller, 1995). Each of these theories is supported by substantial experimental evidence, and each identifies a real function performed during sleep. However, each describes a proximate mechanism – what sleep accomplishes in the body – rather than an ultimate explanation for why natural selection mandated the state itself. The phylogenetic breadth of sleep-like states, extending across kingdoms to organisms that lack the neural substrates posited by these theories (Anafi et al., 2019), further underscores the inadequacy of proximate accounts as evolutionary explanations.

Understanding why sleep evolved requires an ultimate explanation that accounts for the origin of the obligate state change itself, not merely the derived

mechanisms that have evolved to serve it. The proximate–ultimate distinction provides the necessary framework for separating these questions (Figure 1).

2. Sleep is obligate phenotypic switching for temporal niche partitioning

I propose that sleep evolved as an obligate phenotypic switching mechanism enabling temporal niche partitioning within a single genome. An earlier, preliminary formulation of this theory was published in Freiberg (2020) and expanded in Freiberg (in press); the present work states it in rigorous evolutionary-biological terms, makes its predictions and empirical commitments explicit, and distinguishes it from adjacent proximate-level frameworks. The core of the argument is as follows.

Day and night present fundamentally different selective environments. They differ in light availability, thermal regime, predator, prey, and competitor communities, and the sensory and locomotor demands imposed on organisms. These differences are not minor perturbations; they define distinct ecological niches with distinct optimal phenotypes. Temporal niche partitioning – the division of species’ activity into distinct temporal phases – has been extensively documented as a major axis of ecological organization (Kronfeld-Schor & Dayan, 2003). Experimental work has confirmed that closely related species partition temporal niches to reduce interspecific competition, with measurable fitness consequences (Gutman & Dayan, 2005).

Optimization for one temporal niche necessarily entails trade-offs with performance in the other. A visual system optimized for photopic conditions operates poorly in scotopic environments; thermoregulatory strategies suited to daytime solar radiation are suboptimal for nocturnal radiative cooling; foraging strategies effective when prey are diurnally active fail against nocturnally active prey. A single, fixed phenotype cannot simultaneously optimize for both phases.

Sleep is the mechanism by which a single genome produces two temporally specialized phenotypic states. This constitutes a form of phenotypic switching analogous to discrete phenotypic alternatives documented in other biological systems – wing dimorphism in insects, reproductive morphs in fish, phase polyphenism in locusts (Roff, 1996; West-Eberhard, 2003). The critical feature is that the state change is obligate: internally generated, involuntary, and rhythmic. It is not a facultative behavioral decision that an organism can choose to forgo. This obligate character requires explanation, because a facultative system – in which organisms could choose when or whether to switch states – would appear

to offer greater flexibility. The costs and limits of phenotypic plasticity, however, predict that obligate switching should be favored over facultative plasticity when environmental variation is predictable and the costs of maintaining flexible responsiveness are high (DeWitt et al., 1998). A facultative system would be vulnerable to selection against the state change itself: individuals that reduced or eliminated the quiescent phase would gain immediate short-term benefits (more foraging time, more mating opportunities) at the cost of operating in a suboptimal niche without appropriate physiological preparation. Obligate switching prevents this defection by enforcing the state transition internally.

Box 1: Formal Statement of the Theory

- (1) Day and night present distinct selective environments requiring different phenotypic optima.
- (2) Optimization of physiological, sensory, and behavioral traits for one temporal niche reduces performance in the other.
- (3) A generalist phenotype attempting to function across both temporal niches is suboptimal for each.
- (4) Obligate phenotypic switching enables a single genome to produce temporally specialized phenotypic states, each more closely approaching the optimum for its respective niche.
- (5) Therefore, sleep is the obligate mechanism by which natural selection enforces temporal niche partitioning, and the proximate functions of sleep are derived mechanisms that have evolved in service of this ultimate function.

Sleep is thus the phenotypic switching mechanism by which natural selection enforces temporal niche partitioning, enabling a single genome to produce physiological states separately optimized for activity and quiescence periods defined by predictable environmental cycling.

3. Adaptive Avoidance differs from adaptive inactivity at the level of explanation

Siegel (2009) proposed that sleep is adaptive inactivity: organisms are inactive during periods when activity would be energetically inefficient or dangerous, and sleep enforces this inactivity. This proposal shares surface features with the present framework and must be carefully distinguished from it. The distinction is structural, not verbal – the two theories operate at different levels of biological explanation.

Adaptive inactivity is a proximate adaptive argument. It explains why individual organisms benefit from being inactive during particular periods: reduced energy expenditure, reduced predation risk, reduced likelihood of injury. These are immediate fitness benefits accruing to the individual within its lifetime. Siegel's ecological correlates of sleep duration across mammals (Siegel, 2005) – the observation that species in safer environments sleep more, while those facing greater predation pressure sleep less – are consistent with this proximate account. Comparative phylogenetic analysis across mammals points the same way: sleep duration and architecture track ecological variables — diet, trophic level, and the exposure of the sleeping site — more strongly than metabolic body-size scaling alone would predict (Capellini et al., 2008), which weakens purely energetic accounts of why sleep exists while remaining, like Siegel's correlates, evidence at the proximate level: it documents what sleep covaries with across species, not why the obligate switch exists. Restorative accounts of sleep share a common inverted ordering: they treat neural restoration as the ultimate reason for sleep and the sleeping state as a proximate vehicle co-opted to serve it. Siegel comes closest to the present argument — his adaptive-inactivity framing already recognizes that sleep's benefit is ecological rather than tissue-level — but stops short at the same inversion. Adaptive Avoidance reverses the ordering: the obligate switch is the ultimate function, and restoration is the proximate process that later colonized the off-state the switch created.

Adaptive Avoidance operates at the ultimate level. It does not ask why inactivity is beneficial during suboptimal periods (this is relatively obvious), but rather why natural selection mandated an involuntary physiological state change to produce that inactivity. If the sole function of sleep were to enforce inactivity during dangerous or unproductive periods, quiet wakefulness would suffice – and would in fact be preferable, because a wakeful but inactive animal retains the capacity to detect and respond to threats or unexpected opportunities. The survival costs of reduced arousal during sleep are substantial and well-documented. The fact that natural selection imposes a state of diminished environmental responsiveness, despite these costs, indicates that the state change itself serves an evolutionary function that transcends mere inactivity.

The distinction is illustrated by the great frigatebird (*Fregata minor*). Rattenborg et al. (2016) documented that frigatebirds sleep an average of only 42 minutes per day during multi-day flights over the ocean, compared with approximately 12 hours per day on land. Sleep is drastically reduced during flight but never eliminated, even when the ecological costs of inactivity are irrelevant (the bird must remain in flight regardless). Adaptive inactivity cannot explain why a flying

bird sleeps at all; there is no inactivity benefit to sleeping in mid-flight. The persistence of sleep under these conditions is, however, consistent with an obligate switching mechanism maintained by selection for temporal niche partitioning.

Adaptive inactivity and Adaptive Avoidance are complementary, not competing. The former accounts for the timing and immediate benefit of the quiescent phase; the latter explains why natural selection mandated an involuntary physiological state transition rather than permitting flexible behavioral rest. They address different questions in the Tinbergen (1963) framework and are both correct at their respective levels of analysis.

4. Predictable temporal cycling favors obligate switching over generalism

A critical objection to the framework presented here is that natural selection routinely produces ecological generalists – organisms that tolerate a range of habitats, diets, or thermal regimes without resorting to obligate phenotypic switching. Why should temporal niches be different? If a single genome can produce a phenotype that functions adequately across a range of spatial environments, why can it not produce one that functions adequately across both day and night?

The answer lies in three properties of the day–night cycle that collectively distinguish it from other forms of environmental variation and that, according to niche breadth theory, specifically favor obligate switching over generalism.

First, temporal niches are *mutually exclusive and sequential*. An organism cannot occupy day and night simultaneously, as it might sample multiple spatial microhabitats within a single foraging bout. At any given moment, the organism is in one temporal niche or the other. This mutual exclusivity means that a generalist strategy cannot hedge by sampling both environments concurrently; it must maintain a compromised phenotype that performs suboptimally in whichever phase it currently occupies.

Second, the day–night cycle is *perfectly predictable* over evolutionary time. Unlike spatial environmental variation, which may be stochastic and patchy, the alternation of day and night is determined by planetary rotation and has been constant since before life originated on Earth. Levins (1968) demonstrated that when an environment changes in a predictable, large-block pattern (what he termed *coarse-grained* variation: each state persists long enough that an

organism experiences it as the only state present, rather than as a statistical average of rapidly mixed states), selection favors phenotypic specialization over generalism. The same framework also predicts that where the fitness trade-off between two environments is sharply peaked rather than gradual – that is, where a generalist phenotype pays a *disproportionate* performance penalty for each increment of compromise away from a specialist optimum (a relationship Levins formalized in 1968 as a *concave* (non-convex) fitness set: the Pareto frontier between specialist optima bows inward toward the origin, such that intermediates fall below what a linear trade-off would permit; Figure 2) – a switching specialist achieves higher geometric mean fitness than a compromised generalist. The day–night trade-off is plausibly of this sharply peaked form: sensory systems, thermoregulatory mechanisms, and predator-avoidance strategies cannot be linearly interpolated between diurnal and nocturnal optima without incurring nonlinear performance penalties.

Third, the alternation is *high-frequency*. Switching occurs approximately every 12 hours, creating continuous and unrelenting selection pressure. Futuyma & Moreno (1988) showed that ecological specialization is favored when each environmental state persists long enough that organisms experience it as the dominant context, rather than as one of many rapidly interleaved conditions. For virtually all multicellular organisms, day and night meet this criterion: each phase lasts a substantial fraction of the organism’s daily time budget, which is the operationalization of Levins’ coarse-grained variation at the diel scale. This high-frequency cycling imposes persistent selection against maintaining a generalist physiology that is suboptimal during both phases.

Cathemeral species (active during both day and night) and crepuscular species (active primarily at dawn and dusk) represent the closest natural approximations to temporal generalism. Yet even these species sleep (Aulsebrook et al., 2016). Their existence does not refute the framework; rather, it demonstrates that even partial relaxation of temporal niche specialization does not eliminate the selection pressure for obligate state transitions. Cathemerality may reduce the *duration* of sleep, but it does not eliminate the switching mechanism. The relevant prediction is not that all organisms must be strictly diurnal or nocturnal, but that no organism subject to day–night cycling has eliminated obligate phenotypic switching entirely – a prediction that is universally confirmed.

The mutual exclusivity, perfect predictability, and high frequency of day–night cycling create selection conditions that niche breadth theory predicts will favor obligate phenotypic switching over temporal generalism, explaining why no

organism has evolved the capacity to eliminate sleep. This entire argument rests on one empirical premise, worth stating plainly because it is the theory's single falsifiable commitment: that the fitness trade-off between a day-optimized and a night-optimized phenotype is concave — that no intermediate generalist can discharge both roles without a disproportionate loss. Were the trade-off convex, a generalist would suffice and no obligate switch would be needed. It is the concavity of the day–night trade-off, and that alone, on which the necessity of sleep depends.

5. Cross-kingdom evidence supports an ultimate, not proximate, explanation

The strongest evidence that sleep requires an ultimate rather than proximate explanation comes from organisms whose sleep-like states cannot be accounted for by neural restorative theories.

The jellyfish *Cassiopea* exhibits a behavioral state meeting the standard criteria for sleep: reduced activity, increased arousal threshold, and homeostatic rebound following deprivation (Nath et al., 2017). *Cassiopea* lacks a centralized nervous system entirely, possessing only a diffuse nerve net. It has no synapses of the type that the synaptic homeostasis hypothesis proposes require downscaling, no interstitial spaces through which glymphatic convective flow could operate, and no brain whose energy stores require restoration. Yet it sleeps. The nematode *Caenorhabditis elegans*, with precisely 302 neurons connected by a fully mapped connectome, exhibits lethargus – a sleep-like state with reduced behavioral activity and increased arousal threshold during developmental transitions (Raizen et al., 2008). The cyanobacterium *Synechococcus elongatus*, a prokaryote with no nervous system at all, uses the KaiABC protein oscillator to temporally partition incompatible metabolic processes: photosynthesis, which requires light, and nitrogen fixation, which is inhibited by oxygen produced during photosynthesis. This is obligate temporal niche partitioning at the biochemical level – phenotypic switching within a single genome in response to a predictable environmental oscillation.

These cross-kingdom observations are predicted by Adaptive Avoidance. If the ultimate function of sleep is temporal niche partitioning via obligate phenotypic switching, then any organism subject to predictable environmental cycling should exhibit the switching mechanism, regardless of whether it possesses the neural substrates posited by proximate theories. Allada & Siegel (2008) documented the deep phylogenetic conservation of sleep-like states across animal phyla, consistent with a selection pressure that predates the evolution of complex

nervous systems. Restorative theories, by contrast, predict sleep-like states only in organisms possessing the specific substrates whose restoration they invoke.

The cavefish *Astyanax mexicanus* provides what may be the single most informative piece of evidence for the present framework. Three independently derived cave populations of this species have convergently evolved dramatic reductions in sleep duration – from approximately 8 hours in surface-dwelling conspecifics to approximately 2 hours in cave populations (Duboue et al., 2011). Several features of this natural experiment are critical.

First, the sleep reduction is convergent. Three geographically isolated cave populations have independently evolved reduced sleep, indicating that the phenotypic change is the product of natural selection rather than genetic drift or a single founding event. Second, the molecular mechanisms underlying sleep reduction differ across populations. Jaggard et al. (2018) demonstrated that increased hypocretin (orexin) signaling mediates sleep loss in at least one cave population, and that pharmacological blockade of hypocretin receptors partially restores sleep. Third, these mechanisms are not merely different but have been shown to be independent: the hypocretin-linked mechanosensory (lateral-line) pathway that drives sleep loss in Pachón was tested directly in the other cave populations and found not to operate there — ablating the lateral line increases sleep in Pachón but leaves sleep unchanged in Molino, Tinaja, and Chica (Jaggard et al., 2018) — while circadian-clock dysregulation has evolved repeatedly across populations through distinct molecular routes (Mack et al., 2021). This reciprocal exclusion, rather than the mere absence of overlapping gene lists, establishes that convergent selection, not shared ancestry, drives the phenotype (Figure 3). Quantitative-trait-locus mapping in surface–cave hybrids further shows that sleep loss is itself a discrete evolutionary target, genetically separable from the enhanced sensory and prey-seeking behavior with which it co-occurs (Yoshizawa et al., 2015).

The cave environment eliminates the day–night cycle. Caves are aphotic – there is no light variation, no diel thermal cycling, no temporal partitioning of predator and prey communities into diurnal and nocturnal phases. This is precisely the condition under which Adaptive Avoidance predicts relaxation of selection for the switching mechanism: when the environmental oscillation that necessitates temporal niche partitioning is removed, the obligate state transition loses its adaptive value, and organisms should evolve toward reduced or absent sleep.

Restorative theories generate no such prediction. Cave-dwelling fish possess brains that presumably require synaptic homeostasis and metabolite clearance

no less than surface-dwelling conspecifics. If sleep exists to serve these neural functions, there is no reason to expect that inhabiting a cave would reduce the need for them. Adaptive inactivity predicts reduced sleep only if caves present fewer dangers than surface environments, but the predation regime in caves is not obviously safer – cave-adapted predators are present, and cavefish have lost visual defenses. Adaptive Avoidance alone makes the specific, directional prediction that removing temporal environmental variation should relax selection for obligate phenotypic switching, producing evolutionary sleep reduction – which is precisely the observed outcome.

Two features of the cavefish case deserve emphasis. First, sleep is reduced but not eliminated. Residual sleep persists in all three cave populations despite millions of years of aphotic conditions devoid of zeitgebers, and the molecular circadian clock machinery is attenuated rather than lost: *per1* oscillations are retained with reduced amplitude and altered phase, and Mack et al. (2021) showed that cavefish populations have independently evolved clock dysregulation via different molecular mechanisms – the convergence is functional, not mechanistic. This incompleteness is itself informative: a lineage that lost the obligate switching mechanism outright would be unable to re-evolve it if later exposed to a new temporal niche, because the machinery is phylogenetically ancient and mechanistically complex. The retention of residual sleep – even under relaxed selection – is therefore consistent with a switching module that is costly to discard but tolerates attenuation, and predicts that transient cave populations returned to surface conditions should recover circadian sleep more readily than they would have to re-evolve it from scratch. Second, a weaker but directionally parallel pattern is visible in recently domesticated species. Domesticated cats and dogs, whose wild ancestors display strict crepuscular or nocturnal organization but whose provisioned descendants are released from natural predator-prey cycles, show polyphasic and increasingly owner-synchronized sleep patterns rather than elimination. In this case zeitgebers are preserved, but sleep architecture has been uncoupled from them. The comparison is tentative – domestication has operated for 10,000–40,000 years rather than millions – but the direction of change is the one the theory predicts when temporal environmental variation is attenuated.

Sleep-like states in organisms without centralized nervous systems, and the convergent loss of sleep in cave-dwelling fish deprived of day–night cycling, are predicted by a temporal niche partitioning framework but not by proximate restorative theories, providing strong evidence that the ultimate function of sleep is ecological rather than physiological.

6. The same logic extends to all predictable environmental oscillations

If sleep is obligate phenotypic switching driven by the predictable oscillation of day and night, the same evolutionary logic should apply to environmental oscillations at other timescales. This extension represents a novel unifying contribution of the present framework.

Seasonal dormancy – hibernation in mammals, brumation in reptiles, diapause in insects, estivation in organisms facing dry seasons – represents obligate phenotypic transitions enforced by the predictable alternation of summer and winter (or wet and dry seasons). The organism cannot simultaneously maintain physiological states optimized for both warm-season activity and cold-season survival. A compromised phenotype that attempted year-round activity without seasonal physiological adjustment would be suboptimal for both seasons. Natural selection therefore mandates an obligate state transition – a seasonal phenotypic switch – just as it mandates a daily one.

Migration is the second, ecologically complementary response to the same seasonal problem. Rather than switching phenotype in place, migratory organisms physically relocate to an environment where the current phenotype remains appropriate – birds following favorable photoperiods and food supplies, caribou tracking seasonal forage, marine species tracking thermal and productivity fronts. Migration and dormancy are thus alternative, theory-consistent solutions to a shared adaptive problem: seasonal environmental unsuitability. Notably, migration is unavailable at the diel timescale. An animal cannot outrun the Earth's rotation, because the environmental oscillation propagates globally at a speed that exceeds any sustainable animal locomotion. The constraint that rules out diel migration is therefore the same constraint that makes obligate diel phenotypic switching (sleep) the only available solution, and explains why sleep – unlike seasonal migration – admits no ecological alternative.

Deciduous abscission in plants follows the same logic. Leaves optimized for summer photosynthesis are liabilities in winter: they increase surface area for ice loading, create avenues for water loss when soil water is frozen, and cannot photosynthesize efficiently at low temperatures and short photoperiods. The organism switches phenotype – shedding leaves and entering dormancy – rather than maintaining a compromised structure. Evergreen species achieve the same end by different means, fundamentally altering needle physiology for winter conditions rather than maintaining summer-optimized photosynthetic tissue year-round (Kronfeld-Schor & Dayan, 2003).

Tidal organisms exhibit rest–activity cycles synchronized to tidal periodicity rather than solar periodicity, further supporting the claim that the relevant selective variable is predictable environmental oscillation, not the day–night cycle per se. The cyanobacterial KaiABC oscillator, discussed above, represents the same principle at the prokaryotic level: temporal separation of incompatible metabolic processes driven by light–dark cycling.

Table 1. Predictable environmental oscillations and the kinds of phenotypic change each recruits

Mode	Period	Reversible within one individual?	Principal timing cue (zeitgeber)	Morphological change	Physiological-state change	Behavioral change
Phylogenetic	Non-recurring (across lineages)	No — between generations, not within an individual	None (selection, drift)	Major	Major	Major
Ontogenetic	Non-recurring (once per lifetime)	No — one-way	Internal developmental program; some environmental triggers	Major	Major	Major
Circannual (seasonal)	~365 days	Yes	Photoperiod (day length)	Minor to moderate (pelage, gonads, fat stores)	Major (many examples — see note)	Major
Lunar / semilunar	~29.5 / ~14.8 days	Yes	Moonlight; tidal amplitude	None to minor	Moderate	Major
Circadian (daily)	~24 hours	Yes	Light–dark cycle	None	Major (the sleep–wake switch; daily torpor in daily heterotherms)	Major
Circatidal	~12.4 hours	Yes	Tide (pressure, agitation, salinity)	None	Moderate	Major

Note. The table is restricted to rhythms entrained by a predictable oscillation in the external environment; cycles whose period is set internally and tracks nothing outside the organism – the ultradian NREM–REM alternation, ovarian cycles, pulsatile hormone release, the cardiac and respiratory cycles – are excluded, because the argument advanced here concerns the fit between an organism and a cycling world. Rows 1–2 are irreversible; rows 3–6 are reversible alternative phenotypes expressed by a single genome. Circannual examples, by column – morphological: coloration (pelage and plumage), leaf abscission, antler cycles, gonadal regression and recrudescence, seasonal polyphenism; physiological state: cryoprotectant and osmolyte chemistry, estivation, hibernation, bud dormancy in perennial plants, seasonal morphs differing in lifespan, diapause and other life-cycle timing; behavioral: aggregation, altitudinal or depth shifts, migration. Estivation, hibernation and daily torpor share a mechanism, regulated metabolic depression, but differ in season, trigger, depth and bout length, and are largely expressed by different species (Geiser, 2004); daily torpor is placed in the circadian row because its bouts are shorter than 24 hours and recur daily, although in many species it is expressed only in one season.

These phenomena – daily sleep, seasonal dormancy, tidal activity rhythms, deciduous abscission, prokaryotic metabolic switching – are typically studied in separate literatures with separate theoretical frameworks. Adaptive Avoidance unifies them as instances of a single evolutionary principle: obligate phenotypic switching in response to predictable environmental oscillation (Figure 4; Table 1). No existing sleep theory offers this scope of unification. Restorative theories apply only to daily neural sleep in organisms with nervous systems. Adaptive inactivity applies to daily and possibly seasonal inactivity in animals, and in plants. Only a framework grounded in temporal niche partitioning and phenotypic switching accounts for the full range of obligate periodic state transitions observed across biological kingdoms and timescales.

7. The theory generates testable predictions that distinguish it from alternatives

A theory's scientific value depends on its capacity to generate specific, testable predictions that competing theories do not share. Adaptive Avoidance generates the following distinguishing predictions (Table 2).

Prediction 1: Reduced temporal environmental variation should produce reduced sleep. Organisms inhabiting environments with attenuated or absent day–night cycling – caves, the deep ocean, polar regions during continuous light or darkness – should exhibit reduced or absent circadian sleep relative to closely related species in environments with normal diel cycling. This prediction is confirmed by the convergent sleep reduction in three independent cave populations of *Astyanax mexicanus* (Duboue et al., 2011), in which sleep duration decreased from approximately 8 hours to approximately 2 hours following colonization of aphotic cave environments. The prediction is specific to Adaptive Avoidance: restorative theories predict no relationship between environmental light cycling and sleep need, and adaptive inactivity predicts reduced sleep only if the environment is safer, not if temporal variation is reduced.

Prediction 2: Sleep duration should correlate with temporal niche differentiation more strongly than with neural complexity. Among closely related species occupying similar spatial niches but differing in the degree of temporal niche specialization, sleep duration should vary with temporal ecology rather than brain size or neural complexity. Lesku et al. (2006) found that ecological variables – including predation risk and foraging requirements, which are temporally structured – predict mammalian sleep architecture better than phylogenetic position or brain mass. This is consistent with sleep serving a temporal niche function rather than a purely neural restorative one.

Prediction 3: Sleep is obligate and resists voluntary elimination. If sleep is an enforced phenotypic switching mechanism, it should resist suppression through behavioral choice. Organisms should be unable to eliminate sleep voluntarily; sleep deprivation should produce homeostatic rebound (increased sleep pressure following deprivation). This prediction is universally confirmed across every organism in which it has been tested, from mammals to *Drosophila* to *C. elegans*. While this prediction alone does not distinguish Adaptive Avoidance from restorative theories (which also predict homeostatic rebound), it is a necessary condition that the theory must meet and does.

Prediction 4: Sleep-like states should be present in organisms without nervous systems, provided they face predictable environmental oscillation. If the ultimate function of sleep is temporal niche partitioning rather than neural restoration, then obligate rest–activity cycling should be observed in organisms lacking the neural substrates posited by proximate theories. This prediction is confirmed by sleep-like states in *Cassiopea* (Nath et al., 2017) and *C. elegans* (Raizen et al., 2008), and by circadian metabolic switching in cyanobacteria. Restorative theories centered on synaptic homeostasis or glymphatic clearance cannot account for these observations.

Prediction 5: Experimental elimination of temporal environmental variation over evolutionary time should produce evolutionary relaxation of sleep. Populations maintained under constant environmental conditions (constant light, constant temperature, constant resource availability) for sufficient generations should exhibit evolved reductions in sleep duration or intensity relative to control populations maintained under cycling conditions. This prediction is untested and represents a strong, novel, and uniquely distinguishing experimental test. Neither restorative theories nor adaptive inactivity predict this outcome, because neither identifies temporal environmental variation as the selection pressure maintaining sleep.

Prediction 6: Sleep architecture should be ecologically sculpted, not neurally imposed. If restorative processes are derived proximate mechanisms evolved in service of temporal niche partitioning, then the architectural features of sleep – NREM/REM ratios, bout length, monophasic versus biphasic or multiphasic organization, total duration, unihemispheric versus bihemispheric patterns – should covary across species with ecological demands (predation pressure, foraging structure, thermoregulatory environment, migratory requirement, need to remain vigilant or aquatic) rather than with brain mass or phylogenetic position alone. This prediction is supported by observational and experimental

comparative evidence. Cetacean sleep is unihemispheric, an architecture that follows from the ecological constraint of obligate surfacing (Lyamin et al., 2008). Northern fur seals switch architecture with habitat within a single individual: roughly 80 minutes of REM sleep per day on land, almost none through periods of up to two weeks at sea, and little or no REM rebound on return (Lyamin et al., 2018) — architecture tracking ecology within a lifetime, and doing so without the homeostatic debt a restorative account of REM predicts. Male pectoral sandpipers compress sleep to under two hours per day through three weeks of intense sexual selection, and the males that sleep least sire the most offspring (Lesku et al., 2012). Avian unihemispheric sleep is under facultative behavioral control: mallards at the exposed ends of a row use roughly 150% more unihemispheric sleep than birds in the interior, and direct the open eye away from the group (Rattenborg et al., 1999). Vigilance of this kind explains why one hemisphere remains awake; it does not explain why the other must sleep, and it is the second question that is at issue here. Phylogenetic regression of mammalian sleep architecture on ecological covariates explains variance that brain size alone does not (Lesku et al., 2006; Capellini et al., 2008). The prediction distinguishes Adaptive Avoidance from restorative theories (which predict architecture should track neural requirements) and extends adaptive inactivity (which predicts only that *timing* follows ecology, not that *architecture* is ecologically sculpted).

Table 2. Comparative predictions of three classes of sleep theory

Prediction	Adaptive Avoidance	Adaptive Inactivity	Restorative
Reduced temporal variation reduces sleep	Yes	No prediction	No
Sleep correlates with temporal ecology > neural complexity	Yes	Partial	No
Sleep is obligate (homeostatic rebound)	Yes	Yes	Yes
Sleep-like states in organisms without CNS	Yes	Partial	No
Experimental	Yes	No prediction	No

Prediction	Adaptive Avoidance	Adaptive Inactivity	Restorative
removal of cycling relaxes sleep			
Sleep architecture ecologically sculpted, not neurally imposed	Yes	Partial	No

Adaptive Avoidance generates six specific predictions – four supported by independent evidence, one universally observed, and one novel and untested – several of which cannot be derived from restorative or adaptive inactivity theories, providing a basis for empirical differentiation among competing frameworks.

8. Proximate mechanisms serve an ultimate function

The framework presented here does not reject restorative theories of sleep. It situates them.

Synaptic homeostasis (Cirelli & Tononi, 2008), glymphatic clearance (Xie et al., 2013), and energy restoration (Benington & Heller, 1995) describe proximate mechanisms that operate during the sleep state. The experimental evidence supporting each of these functions is substantial and is not contested here. What is contested is the inference that these functions explain *why* sleep exists – that they constitute the ultimate evolutionary cause of the state itself.

Under the Adaptive Avoidance framework, the restorative processes observed in sleeping organisms are derived proximate mechanisms that have evolved to serve an ancestral ultimate function. They are not the evolutionary reason sleep exists; they are adaptations that have accrued because sleep exists. The distinction at issue is between the origin of a trait and its subsequent maintenance and elaboration: restorative functions may be fully adaptive and still not be the reason the state arose, being what selection has since installed in a window that was already open. The relationship parallels other well-established proximate/ultimate pairings in physiology and behavior:

Table 3. Proximate mechanisms and the ultimate functions they serve

Proximate mechanism	Ultimate function
Thirst	Fluid balance

Proximate mechanism	Ultimate function
Hunger	Nutrient intake
Pain	Injury avoidance
Fever	Pathogen clearance
Fatigue	Energy conservation
Restorative processes (synaptic homeostasis, glymphatic clearance, energy restoration, immune consolidation)	Temporal niche partitioning

In each row of Table 3, the proximate mechanism evolved *in service of* a pre-existing adaptive problem, not as a later function that supplanted the original. Thirst did not create the need for fluid balance; it evolved as a mechanism maintaining it. Hunger did not create the need for nutrient intake; it evolved as a mechanism ensuring it. The restorative processes of sleep did not create the need for sleep; they evolved to support and extend the fitness benefits of a state that already existed for temporal niche partitioning reasons. Proximate mechanisms serve ultimate functions; they do not replace them.

This framing also accommodates the diversity of sleep architecture observed across taxa. If restorative processes are derived proximate mechanisms shaped by selection to support the ancestral function, they should be expected to *tune to ecology*. The specific composition of sleep – NREM/REM ratios, bout structure, monophasic versus biphasic or multiphasic organization, total duration, unihemispheric patterns – should vary across species as ecological demands vary, precisely because the restorative machinery is ecologically sculpted. Sleep architecture is not a universal neural requirement imposed identically on all organisms; it is a species-specific elaboration of proximate mechanisms serving a universal ultimate function.

This distinction has practical implications. It explains why sleep-like states are phylogenetically universal while specific restorative functions are not: the ultimate function (temporal niche partitioning) applies to all organisms subject to predictable environmental cycling, whereas the proximate functions (synaptic homeostasis, glymphatic clearance) apply only to organisms possessing the relevant neural substrates. It explains why no single restorative theory has achieved consensus as *the* function of sleep: each describes one of several derived mechanisms serving the ultimate function, not the underlying evolutionary cause. And it explains why sleep persists in organisms – such as cave-dwelling

fish in constant darkness – that presumably still require neural restoration: because the question of whether an organism still benefits from restorative sleep is distinct from the question of whether the selection pressure that originally mandated the state transition remains operative.

Restorative functions of sleep are real, measurable, and adaptively elaborated, but they are derived proximate mechanisms serving an ancestral ultimate function whose origin lies in the temporal niche partitioning demands imposed by predictable environmental cycling. Identifying this ultimate function does not diminish the importance of proximate mechanisms; it contextualizes them within a coherent evolutionary framework.

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Data availability

No new data were generated or analysed in support of this research.

Conflict of interest

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Figure Legends

Figure 1. Levels of explanation applied to sleep theories. Sleep theories placed within the Mayr/Tinbergen 2×2 matrix. Columns partition questions by how they are asked: *static* (how does the process operate within the organism?) versus *dynamic* (why does it exist, and how did it come to exist?). Rows partition them by the level at which they are answered: *proximate* (within the individual organism) versus *ultimate* (across evolutionary time). Restorative theories (synaptic homeostasis, glymphatic clearance, energy restoration) address proximate mechanism. Adaptive inactivity addresses proximate adaptive value. Adaptive Avoidance addresses the ultimate phylogenetic question: why natural selection mandated the obligate state change itself.

Figure 2. Fitness-set analysis of temporal niche specialization, adapted from Levins (1968). Axes represent fitness in the diurnal and nocturnal environments. A linear trade-off between specialist optima defines a *convex* fitness set (dashed chord), within which any compromise generalist lies on the frontier. A *concave* fitness set (solid curve) bows inward: the Pareto frontier falls below the chord, and any single intermediate phenotype lies inside the penalty zone (shaded). Under coarse-grained, predictable environmental cycling, a switching specialist alternating between the corner optima achieves higher geometric-mean fitness than any compromised generalist on the concave frontier. The day–night trade-off is plausibly concave: sensory, thermoregulatory, and predator-avoidance phenotypes cannot be linearly interpolated without nonlinear performance penalties.

Figure 3. Cavefish evidence for Adaptive Avoidance. (A) Schematic relationships among surface-dwelling *Astyanax mexicanus* and three independently derived cave populations (Pachón, Tinaja, Molino); the cave populations derive from independent surface-dwelling ancestors, indicating convergent phenotypic

evolution rather than shared descent; branching order among the populations is contested and is drawn unresolved. (B) Mean sleep duration (hours per 24 h) for each population. Surface fish sleep approximately 8 h; all three cave populations sleep approximately 2 h. (C) Molecular routes to reduced sleep. A shared substrate is evident across cave populations: core clock machinery is retained but attenuated, with *per1* oscillations persisting at reduced amplitude and altered phase; independent molecular routes converge on the same functional outcome (Mack et al., 2021). Pachón fish show increased hypocretin (orexin) signaling, and HCRT receptor blockade partially restores sleep (Jaggard et al., 2018). The Pachón hypocretin/lateral-line route was tested directly and found not to operate in the other populations — lateral-line ablation alters sleep in Pachón but not in Molino, Tinaja, or Chica (Jaggard et al., 2018) — and clock dysregulation has evolved repeatedly through distinct routes (Mack et al., 2021), so Tinaja and Molino reach the same phenotypic endpoint via mechanisms non-homologous to Pachón and to each other. Three populations, one phenotypic outcome, three non-homologous molecular routes: convergence here is functional rather than mechanistic — the outcome predicted by a relaxed-selection framework in which what is being relaxed is the switching *requirement*, independent of the particular molecular hardware through which any given lineage happens to have built the switch.

Figure 4. Timescale unification of obligate phenotypic switching. Concentric rings, numbered from innermost to outermost, represent predictable environmental oscillations at distinct timescales: (①) metabolic (~24 h, intracellular; the KaiABC oscillator in cyanobacteria, which partitions photosynthesis from nitrogen fixation); (②) circadian/diel (~24 h, whole-organism; sleep in animals, nyctinastic leaf movement in plants); (③) tidal (~12.4 h; intertidal invertebrates, coastal fish foraging); (④) seasonal/annual (~1 yr; hibernation, insect diapause, deciduous abscission, bird migration). All instantiate the same evolutionary principle – obligate phenotypic switching within a single genome – enabling a lineage to occupy two otherwise-incompatible adaptive peaks across each environmental cycle.

Table 1. Predictable environmental oscillations and the kinds of phenotypic change each recruits. Rows are the recurring environmental oscillations to which the framework applies, ordered by period, with the two irreversible modes of biological change above them for contrast. Columns record whether the mode is reversible within one individual, what entrains it, and how much morphological, physiological-state and behavioral change it recruits (none / minor / moderate / major). The seasonal row recruits all

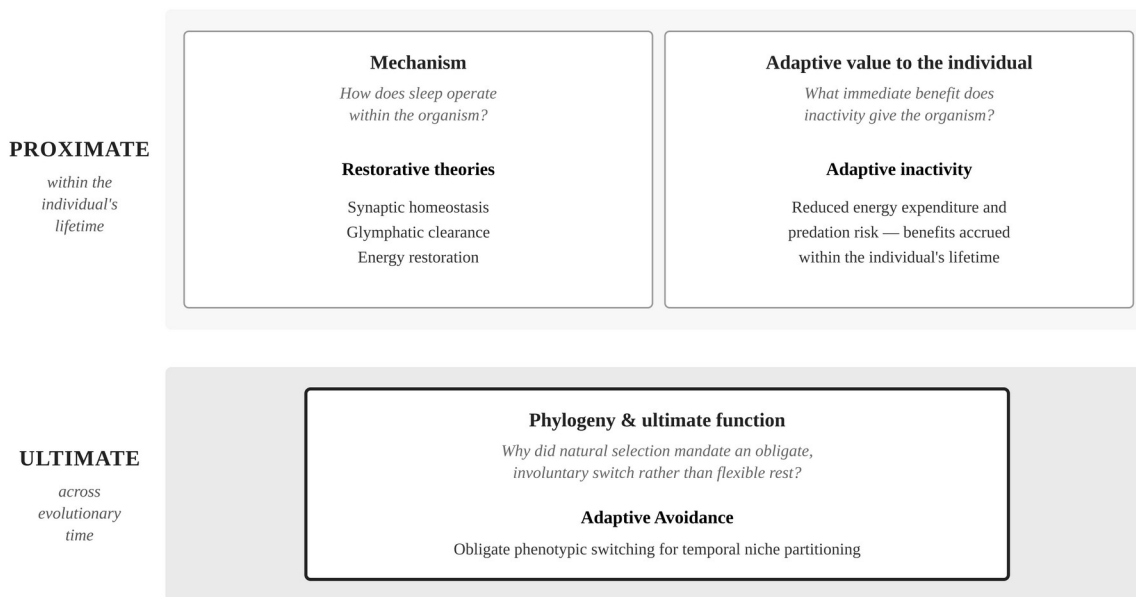
three kinds of change; the daily row recruits only a physiological-state change, and that change is the most complete switch in the table.

Table 2. Comparative predictions of three classes of sleep theory. Rows indicate specific predictions; columns indicate whether each theory makes the prediction (Yes), does not address it (No prediction), or predicts the opposite (No). Adaptive Avoidance generates predictions 1, 4, and 5 that are not shared by restorative or adaptive inactivity theories.

Table 3. Proximate mechanisms and the ultimate functions they serve. Each row pairs a proximate mechanism with the adaptive problem it evolved to solve. In the first five rows the pairing is uncontroversial; the final row states the pairing proposed here for sleep.

Figure 1. Levels of explanation applied to sleep theories

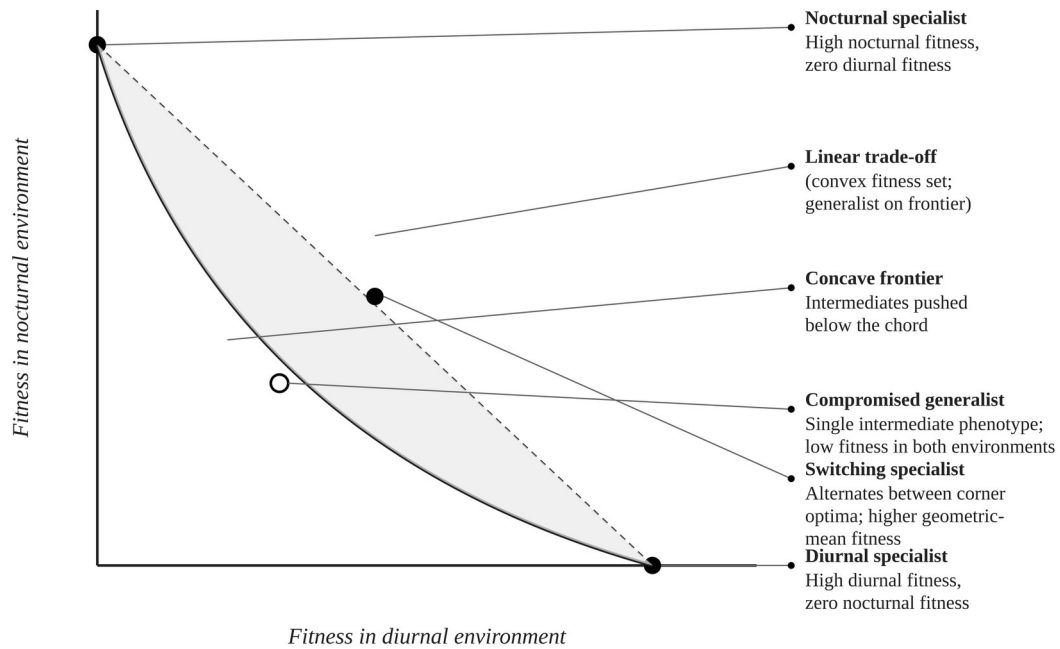
The Mayr–Tinbergen framework: proximate accounts of sleep vs. the ultimate evolutionary question



Restorative theories and adaptive inactivity are both *proximate* accounts: the first explains sleep's mechanism, the second its immediate adaptive value to the individual. Adaptive Avoidance addresses the distinct *ultimate* question — why selection made the inactive state obligate. Proximate accounts describe what sleep does within a lifetime; the ultimate account explains why the switch evolved.

Figure 1. Levels of explanation applied to sleep theories. Sleep theories placed within the Mayr/Tinbergen 2×2 matrix. Columns partition questions by how they are asked: static (how does the process operate within the organism?) versus dynamic (why does it exist, and how did it come to exist?). Rows partition them by the level at which they are answered: proximate (within the individual organism) versus ultimate (across evolutionary time). Restorative theories (synaptic homeostasis, glymphatic clearance, energy restoration) address proximate mechanism. Adaptive inactivity addresses proximate adaptive value. Adaptive Avoidance addresses the ultimate phylogenetic question: why natural selection mandated the obligate state change itself.

Figure 2. Fitness-set analysis of temporal niche specialization



Axes: fitness in each temporal environment. Specialist phenotypes (solid points) occupy the corner optima. The concave fitness frontier (solid curve) falls below the linear chord (dashed), leaving a generalist-penalty zone (shaded): any single compromise phenotype is disproportionately worse than what a linear trade-off would predict. A switching specialist alternating between corner optima achieves higher geometric-mean fitness than the compromised generalist. Under coarse-grained, predictable environmental cycling, selection favors switching over generalism. Adapted from Levins (1968).

Figure 2. Fitness-set analysis of temporal niche specialization, adapted from Levins (1968). Axes represent fitness in the diurnal and nocturnal environments. A linear trade-off between specialist optima defines a convex fitness set (dashed chord), within which any compromise generalist lies on the frontier. A concave fitness set (solid curve) bows inward: the Pareto frontier falls below the chord, and any single intermediate phenotype lies inside the penalty zone (shaded). Under coarse-grained, predictable environmental cycling, a switching specialist alternating between the corner optima achieves higher geometric-mean fitness than any compromised generalist on the concave frontier. The day–night trade-off is plausibly concave: sensory, thermoregulatory, and predator-avoidance phenotypes cannot be linearly interpolated without nonlinear performance penalties.

Figure 3. Convergent evolution of sleep loss in Mexican cavefish (*Astyanax mexicanus*)

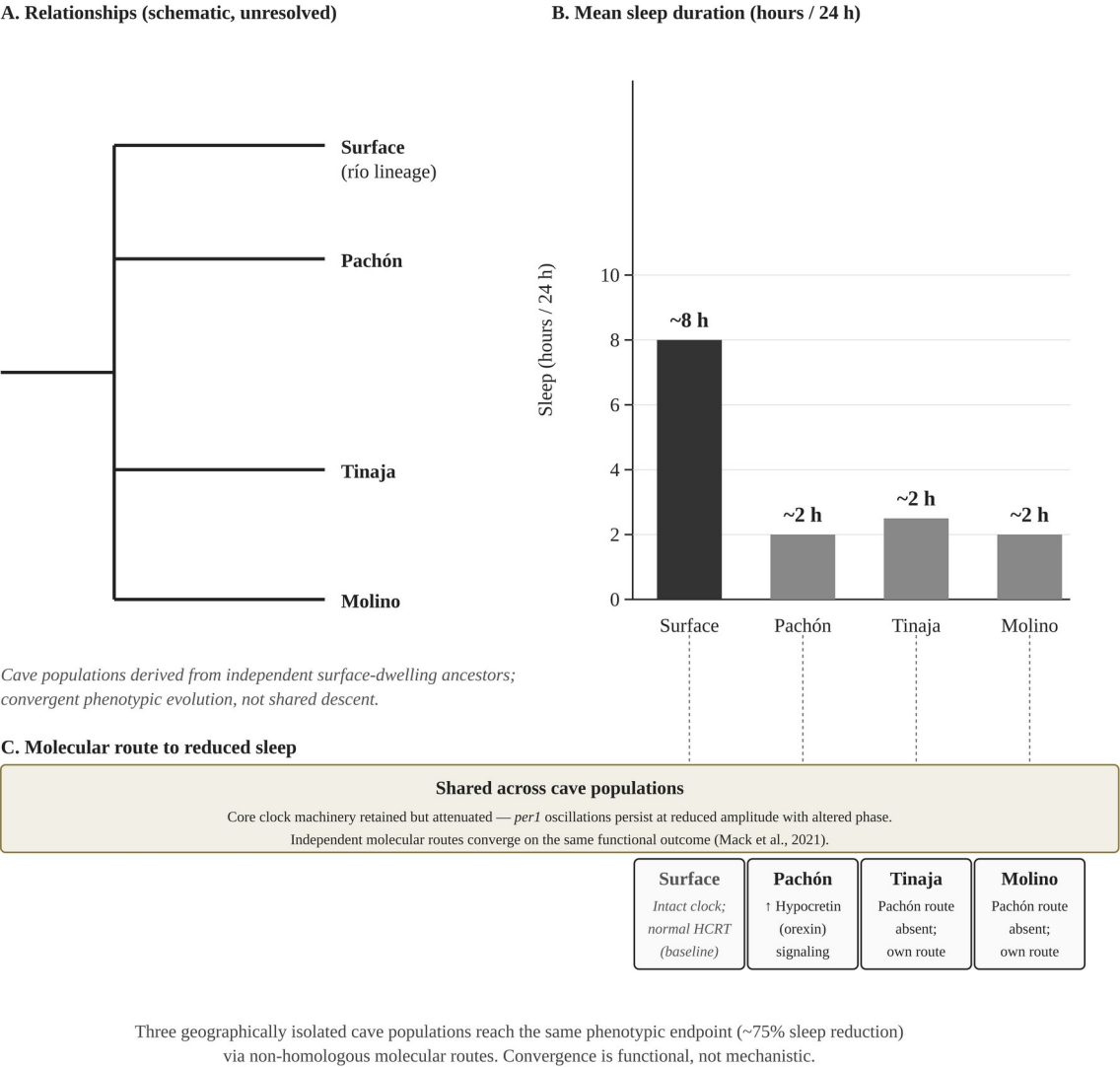


Figure 3. Cavefish evidence for Adaptive Avoidance. (A) Schematic relationships among surface-dwelling *Astyanax mexicanus* and three independently derived cave populations (Pachón, Tinaja, Molino); the cave populations derive from independent surface-dwelling ancestors, indicating convergent phenotypic evolution rather than shared descent; branching order among the populations is contested and is drawn unresolved. (B) Mean sleep duration (hours per 24 h) for each population. Surface fish sleep approximately 8 h; all three cave populations sleep approximately 2 h. (C) Molecular routes to reduced sleep. A shared substrate is evident across cave populations: core clock machinery is retained but attenuated, with *per1* oscillations persisting at reduced amplitude and altered phase; independent molecular routes converge on the same functional outcome (Mack et al., 2021). Pachón fish show increased hypocretin (orexin) signaling, and HCRT receptor blockade partially restores sleep (Jaggard et al., 2018). The Pachón hypocretin/lateral-line route was tested directly and found not to operate in the other populations — lateral-line ablation alters sleep in Pachón but not in Molino, Tinaja, or Chica (Jaggard et al., 2018) — and clock dysregulation has evolved repeatedly through distinct routes (Mack et al., 2021), so Tinaja and Molino reach the same

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Figure 4. One evolutionary principle across timescales

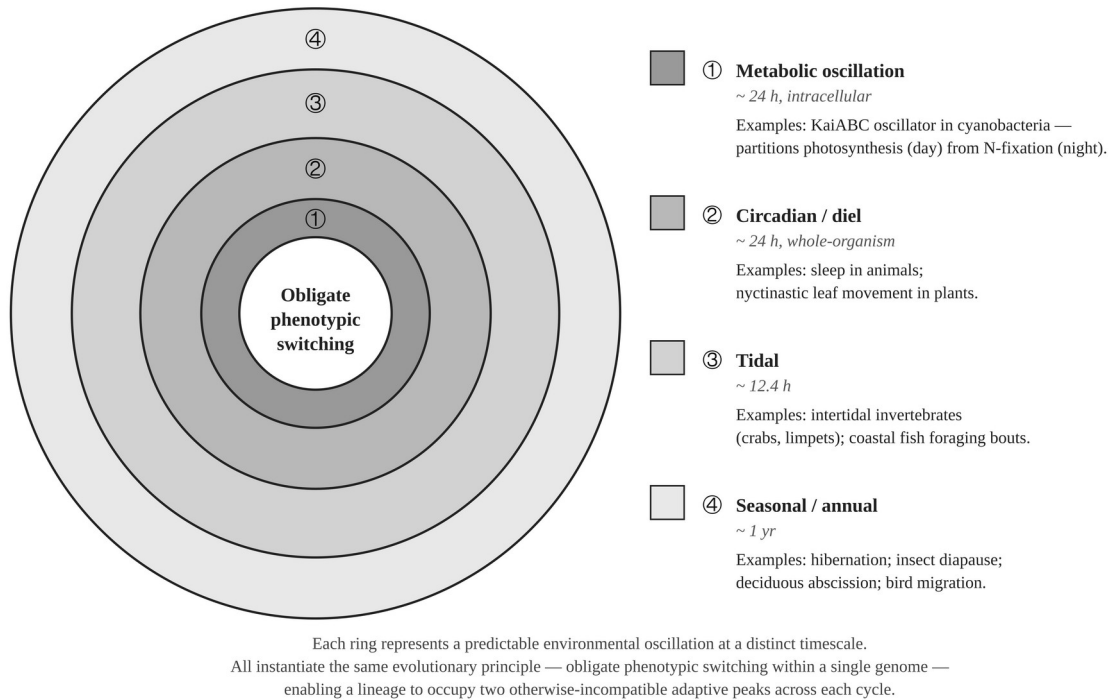


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