Sleep elasticity: accessory, useful and vital components of a single state

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Abstract

Sleep is universal, homeostatically defended and apparently costly, and for half a century this combination has been read as evidence of a vital, conserved function still awaiting identification. The search has not converged. Instead, it has accumulated a dozen candidate functions, each defensible but none commanding assent. I argue that the difficulty lies with the question rather than with the answers to it. Evolution optimises fitness, not function, and asking what fitness sleep confers, and to whom, removes the expectation of a single answer. Reviewing the comparative, experimental and epidemiological evidence, I propose the sleep elasticity hypothesis: that sleep is not one thing but up to three components of separate evolutionary origin, superimposed into a single state. An accessory component arose first as an ecological buffer, keeping an animal safely and economically inactive during the hours in which activity would not repay itself; it discharges no dedicated cellular transaction, yet carries most of the sevenfold interspecific variation in sleep duration, the elasticity that gives the hypothesis its name. A useful component comprises the lineage-specific processes subsequently exapted onto that pre-existing state, so that what a species does with its sleep becomes a fact about the species rather than about sleep. A vital component, if it exists, is a small and still unidentified residue, which I hold open as a hypothesis rather than deny. The three observations conventionally taken to establish indispensability, that deprivation kills, that sleep is costly, and that no sleepless animal exists, do not carry the weight placed on them, and the literature on the harms of sleep loss, in animals and humans alike, is confounded throughout by stress. Sleep elasticity instead accommodates what single-function accounts must explain away: the migrating bird that sheds sleep for weeks and never repays it, the cavefish that has lost most of its sleep but kept the homeostat that would defend it, the fur seal that abandons one sleep state at sea while defending the other, and the captive sloth that sleeps six hours longer than its free-living counterpart. What makes inactivity sustainable is the elevated arousal threshold, and depth measured that way, rather than the slow wave, offers the most promising route to separating the three components experimentally.

I. INTRODUCTION

Modern sleep science inherited a strong prior. Following Rechtschaffen, much of the field has proceeded on the assumption that sleep serves an absolutely vital function that we have not yet identified: a function so important that its absence would be lethal, and so fundamental that it justifies the apparent recklessness of spending a third of one’s life insensible to the world’s perils and opportunities[1–5]. The wager rests on three observations. The first is that sleep deprivation kills:

if prolonged wakefulness is followed by death, then sleep must be supplying something the body cannot do without[6–8]. The second is that being asleep is costly: if a sleeping animal can neither forage, nor mate, nor watch for predators, then this conspicuous cost must be repaid by a benefit equally large and universal[9–11]. The third is that no sleepless animal exists: despite a century of comparative work, no species has been shown to forgo sleep entirely[12–14]. Examined individually, none of these observations supports the conclusion it is asked to carry.

The central terms (what is sleep, and what is vital) need pinning down first, because much of the disagreement here is definitional. Behavioural sleep, defined by reversible quiescence with an elevated arousal threshold and homeostatic regulation, has now been described in almost every animal in which it has been sought, from mammals and birds to fish, flies, worms and cnidarians[12,13,15,16], and the evolutionary framing of that ubiquity is itself a live question[17]. Yet "sleep is vital" can mean two quite different things, often allowed to substitute for one another:

Physiological indispensability, in which sleep is believed to discharge a biological transaction that cannot occur in any other state, such that without it some essential process fails outright and the animal dies. This is the strong claim, akin to a "sleep calorie", and the one I wish to question; throughout this paper I reserve the term *physiologically indispensable* for it.

Necessity under ordinary ecological challenge, in which a non-sleeping animal may function adequately in a protected environment and yet die facing real-world challenges. Nothing fails inside the cell; the animal dies, killed by an environment it can no longer meet.

To say that sleep is not physiologically indispensable is emphatically *not* to say that losing it is harmless, that it is functionless, or that its loss is without cost. It is not the same thing as a vital transaction, and the evidence for the one has too often been offered as evidence for the other. With that distinction in hand, the three observations can be taken in turn.

II. THE EVIDENCE FOR INDISPENSABILITY

(1) Does sleep deprivation kill?

The claim that sleep loss is lethal rests on a surprisingly small and largely dated set of experiments. Marie de Manacéïne's puppies[6] and the disk-over-water rats of Rechtschaffen and colleagues[7,18,19] form the core of this literature, and both showed that animals subjected to enforced wakefulness deteriorate and die. In both paradigms the animals were kept awake by continuous, inescapable,

physically stressful intervention, and in neither was the cause of death established. In dogs and rats, death arrives as systemic collapse rather than organ failure: runaway hypermetabolism, thermoregulatory failure, negative energy balance despite ravenous overeating, and finally breakdown of the gut barrier, spilling bacteria into the bloodstream. That is the syndrome of the sleep-deprived animal, and almost line for line the syndrome of one that has merely been stressed: sustained glucocorticoid elevation drives the same hypermetabolism, tissue catabolism and weight loss despite maintained food intake[20,21], and raises intestinal permeability enough to drive the same bacterial translocation across the gut wall[22–24] (even in animals that have lost no sleep at all). When deprivation is engineered to minimise stress, the signatures of stress fail to appear, most notably activation of the hypothalamic–pituitary–adrenal (HPA) axis, the body's principal glucocorticoid response to threat[25,26]. Forced locomotion, gentle handling, novel-object exposure and optogenetic arousal impose different stress loads, and the reported consequences of "sleep deprivation" vary accordingly[27,28]. Applied to pigeons, which lose substantial sleep and rebound clearly, the disk-over-water produces no lethal syndrome, no hyperphagia, no weight loss, none of the metabolic collapse[29].

This confound is not a relic of the past. Sang and colleagues recently reported that mice die within four days of sleep deprivation, tracing the death to a neutrophilic cytokine storm[30]. Yet their mouse stands in eight millimetres of water, without bedding, for ninety-six hours, woken whenever its nose touches the surface, its fur wet throughout, unable to get out, and it dies hypothermic[30]. Inescapability, immersion and thermal load are the classic ingredients of the severest rodent stress paradigms, and this protocol applies all three for four days without remission and without a yoked control. The authors concede the environment "is still stressful" and that they "cannot fully rule out a role for stress". This is a stress experiment in which the animals also lose sleep.

Invertebrate studies follow the same line. In cockroaches *Blaberus giganteus* the "sleep deprivation" method is again forced locomotion[31], and prolonging it raises metabolic rate[32], and stress by itself, with no sleep lost, is enough to kill given that animals placed in social conflict die of it[33].

Drosophila shows the same pattern but through its genetics also provides glimpses of the other side of the coin. Shaw and colleagues were the first to find sleep deprivation quickly fatal to flies, yet showing that stress-response genes protect against it[34]. The apparent conflict dissolves once method is held constant. Lifelong, quantitatively verified sleep restriction, imposed by automated tracking on a closed-loop principle[35], leaves flies close to a normal lifespan, with only a moderate, sex-dependent effect on female longevity[36]. Deprivation triggered only when a fly attempts to sleep,

avoiding the chronic forced arousal every earlier paradigm relied on, produces neither the lethality nor the intestinal reactive-oxygen-species accumulation that had been attributed to sleep loss[8]; the damage and the mortality track the method before the lost sleep[37].

On the side of survival stand the many short-sleeping mutants and the artificially selected lines that sleep a fraction of normal amounts and live full lives. The *Drosophila* dopamine-transporter mutant *fumin* sleeps almost not at all yet lives a normal lifespan[38] with normal fecundity, and short-sleeping variants recur at the tail of the natural distribution in flies, rodents and humans. In mammals, mutations in *DEC2*, the β1-adrenergic receptor or the neuropeptide-S receptor cut sleep sharply without evident harm, and neuropeptide-S-receptor mice consolidate memory normally on their reduced sleep[39–41]. Under artificial selection, fly lines reached a few hours' sleep with no cost to lifespan or egg-to-adult viability[42]. Extreme short sleep was assembled from ordinary alleles at no vital price, though partly reverted once relaxed, hinting that short sleep trades against something other than survival. There are counter-cases: *shaker*, *sleepless* and *insomniac* fly mutants sleep little and die young[43–45], but these are lesions in pleiotropic genes, with no clear evidence that lethality is consequential to lost sleep. The same confound afflicts the one human case. Fatal familial insomnia, invariably cited as proof that sleep loss kills, is a prion disease caused by a *PRNP* mutation, in which thalamic degeneration produces insomnia as a symptom of the underlying neurodegeneration. It is a prionopathy before it is a sleep disorder, and all prionopathies are lethal. In fact, a transgenic mouse carrying the mutation develops the misfolded-prion pathology and neurological disease with only a very mild sleep phenotype[46]. No clean human evidence shows sleep deprivation to be lethal in isolation, and for ethical reasons there never will be.

The point is Popperian. Indispensability is a universal claim, refutable by a single counterexample[47]: no number of white swans proves all swans white but one black swan settles the matter. Similarly, some short-sleeping mutants that die young cannot show sleep is indispensable but one that barely sleeps and lives fully shows it is not. The many sleepless mutants, the evolutionary-selected lines, and the mammalian short-sleeper alleles are the black swans any vital-function account must first accommodate.

(2) The cost of being asleep

The second pillar treats sleep as self-evidently costly and reasons backwards that a behaviour this expensive must be repaid by a benefit equally large. It would be easy, and it has been attempted, to answer that the costs of sleep are largely illusory[48]. That answer is only half right, and unsafe as an argument. The instructive question is when sleep costs an animal something, not whether. The

expected return of being awake is not constant, and animals pay, refuse and renegotiate the cost of sleep as ecology dictates: with light, prey, temperature and the presence of a receptive mate, on timescales from the hour to the season. Sleep expands to fill the hours in which that return falls below the return of safe inactivity, and contracts when it rises. Sleep duration, on this view, is a residual: what is left of the day once the hours worth being awake for have been spent. The argument is borrowed from foraging theory, where Charnov's marginal-value theorem holds that an animal should quit a patch when its return falls to the average available elsewhere[49]. Lesku and colleagues put it almost in these words: sleep "enforces adaptive inactivity to conserve energy when activity is unproductive", so that "animals may evolve the ability to dispense with sleep when ecological demands favor wakefulness"[50]. It has even been formalised as an extension of the two-process model[51].

The commonly argued costs can now be taken in turn. The first two, predation and exposure, are arguably not costs at all. The canonical image is of an animal lying exposed and defenceless, at the mercy of anything that finds it, but how many animals sleep this way? Sleep is preceded, near-universally, by the search for or construction of a refuge. Across primates, sleeping-site selection is driven principally by antipredator considerations, with parasite avoidance close behind: animals choose tall or inaccessible trees, cliffs and concealed, escapable positions, and build nests to those ends[52–54]. The same logic governs burrowing, denning and communal roosting across the rest of the animal kingdom[55,56], and sleep is timed, in most species, to the phase when the animal could least spot a predator anyway. Hidden and immobile in a defended refuge, a sleeping animal is less exposed than a waking one in the open[10]. Often, those sites also shelter against cold, heat, wind and rain[53,54], as does social huddling[57]. With the reduced metabolic rate of the sleeping state[58,59], this makes sleep a thermal and energetic gain, not an expenditure. Refuges serve waking needs too (shelter, safety, the rearing of young), and an animal that spent its unprofitable hours awake would need one anyway. The refuge is the price of being inactive in a dangerous world, not of being asleep.

The third cost is reproduction, and it is this pillar's best case. Mating is not continuous (most animals breed seasonally, and oestrus is brief[60–62]) but territory defence and mate-searching run year-round in many taxa, and insects are not gated at all. The easy reply, that sleep therefore steals little, does not survive scrutiny: its cost in offspring is the reproductive return an animal forgoes by staying asleep, and that varies with circumstance. The decisive case is an animal whose chance to breed never pauses, so that every hour it sleeps is an hour of mating given up. The pectoral sandpiper (*Calidris melanotos*) is that animal. Outside the breeding season it sleeps 16 hours a day.

During the three mating weeks, when receptive females are continuously available, sleep collapses: males remain active for up to 95% of nineteen consecutive days, sleeping less than 10 minutes a day[50]. Sleep is surrendered when its price rises and resumed when the price falls, male activity dropping again as soon as fertile females are gone, with no noticeable rebound. The same dissociation can be observed in an insect. A male *Drosophila* housed with a female is virtually sleepless (1% of the time against 40% in controls) and the cellular markers of sleep pressure rise in his brain as they do after mechanical deprivation. Then the rebound does not come[63,64]. It is eliminated rather than postponed, and female pheromones alone abolish it, through a defined neuronal circuit[64]. A bird and a fly, six hundred million years apart, forgo sleep because something better is on offer reminding us that sleep does not occupy a privileged seat in the hierarchy of ecological priorities.

This goes beyond sex: starve an animal and it stops sleeping and starts searching, through a genetic programme separable from the general stress response[65]; feed it, and sleep returns[66]. The decisive experiment combines the two. A well-fed male paired with a female suppresses almost all of his sleep to court her; a male deprived of dietary protein courts her just as much, but sleeps considerably more[67]. His courtship drive is untouched; what has changed is the price in wakefulness he will pay to pursue it. Flies selected for starvation resistance over many generations sleep more, not less[68], reversing the acute effect of hunger. A lineage adapted to chronic scarcity, for which searching no longer pays, does best to remain inactive and conserve energy.

The pattern needs no manipulation to be seen. Captive animals sleep far more than wild ones. Between species, with phylogeny and recording conditions controlled, sleep is not predicted by brain mass and correlates negatively with metabolic rate; what survives is a trade-off with foraging time, herbivores sleeping least[69]. Wild elephants (*Loxodonta africana*) sleep about two hours[70] because sixteen to eighteen are spent eating.

The last cost often invoked is lost productive time, but an opportunity cost is only a cost where an opportunity exists. Foraging returns diminish, night foraging is often unprofitable or dangerous, and an animal that is awake is frequently producing nothing either. The Hadza of northern Tanzania, among the best available models of how humans lived before agriculture, spend roughly ten hours of the day non-ambulatory, resting and mostly squatting[71]; and when sleep is measured objectively, three such pre-industrial populations average between five and a half and seven hours a night[72], which is not the profile of a hoarded resource. Nor are their nights wasted: staggered chronotypes leave almost no moment of a Hadza night unattended, so individual sleep and collective vigilance are traded against one another[73]. For most organisms most of the time, extra waking hours would be

idle hours, into which sleep expands. Give an animal or a human a magic sleepless pill, and they would not know what to do with that extra time.

Sleep carries a price, but one that varies with circumstance, and animals shed it the moment that price rises. A cost borne cheerfully while it is cheap and shed the instant it begins to bite is, in the language of economics, met at the margin, where an optimum requires only that marginal benefit match marginal cost, not that either be large. A quantity that answers to its price in this way is, in the same language, elastic[74].

(3) The absence of sleepless animals

The third pillar holding the wager is the strongest, and the only one that is close to straightforwardly true: no sleepless animal species has ever been found and even when animals reduce sleep for ecological reasons, they never reduce to nought. The inference drawn from that, that sleep must therefore be vital, is nonetheless a non sequitur.

Circadian rhythms are more deeply and more universally conserved than sleep, and they confer a clear and pervasive fitness advantage. Yet they are not vital in the physiological sense at stake: many animals with a genetically disabled clock survive, reproduce and live out a normal lifespan in the laboratory[75,76].

The instructive part is what happens when clockless animals are returned to the world. Free-living chipmunks (*Tamias striatus*) and antelope ground squirrels (*Ammospermophilus leucurus*) whose circadian pacemaker has been ablated are killed by predators at markedly higher rates than intact controls, because an arrhythmic animal is active at the wrong hours and betrays itself to weasels and cats[77,78]. Cyanobacteria strains whose free-running period resonates with the ambient light–dark cycle competitively displace strains whose period does not, though each grows perfectly well cultured alone[79,80]. Plants show it too, clock-matched genotypes fixing more carbon, growing faster and out-competing mismatched ones[81]. A clock's fitness value may be invisible in the incubator and decisive in the world, which warns against reading "non-vital" as "unimportant": a trait can be under intense selection, and its loss can reliably kill, without either fact revealing a vital cellular transaction. If ubiquity proved indispensability, the clock would be the first example, and it is not one.

The datum that most needs explaining and that we hid for too long is not that all animals sleep, but that they sleep in such radically different amounts. Half a century of phylogenetic and ecological analysis shows it tracking constitutional and ecological variables (body size, diet, metabolic rate, predation risk, infection, parasite exposure) in the manner of an adaptive trait, not a conserved

physiological constant[9,12,69,82,83]. Any account of sleep function that cannot explain this variation has not explained sleep.

Before these differences can carry such weight, the numbers must be examined: the most quoted do not survive scrutiny. The two highest mammalian values are heterotherms in warm laboratories: twenty hours in the little brown bat (*Myotis lucifugus*) falls to eleven at 26°C rather than 33°C, and is inseparable from torpor[84]. The 20.4 hours of the armadillo (*Chaetophractus villosus*), the database's highest, come from five animals recorded once each, in a study of penile erection reporting sleep as an aside[85]. Not every long value is an artefact: in the pocket mouse (*Perognathus longimembris*), slow-wave sleep grades into shallow torpor[86,87], yet its tabulated twenty hours are genuine euthermic sleep. What survives, method held constant, is a roughly sevenfold range, from some nineteen hours in the opossum (*Didelphis marsupialis*), by EEG across full 24-hour cycles[88], to under three in the horse (*Equus caballus*): still far too large for any account treating sleep as the repayment of a conserved physiological debt.

The variation is not only large but dynamic. Some animals down-regulate sleep drastically, for weeks, whenever ecology demands, without the deterioration a vital-function account predicts: migrating white-crowned sparrows (*Zonotrichia leucophrys*) and pectoral sandpipers cut sleep to a fraction of baseline, and in the sandpiper the least-sleeping males sire the most offspring, making the loss a reproductive strategy rather than a pathology[50,89]; frigatebirds (*Fregata minor*) sleep in seconds-long snatches on the wing for days at a time[90], and marine mammals all but abolish particular sleep states at sea for weeks without repaying them[91–93]. A state shed at ecological convenience for weeks and then not recovered is hard to construe as intrinsically lethal.

There are two ways to reconcile a universal vital function with this variability. Either evolution knows a trick to pack into two hours what other species need twenty (in which case, given a real cost of sleep, the long sleepers should all have evolved to two hours), or sleep does different things in different lineages, abandoning the premise of a single conserved function. The second must be correct.

III. THE SLEEP ELASTICITY HYPOTHESIS

Rechtschaffen was right to insist that sleep be understood through evolution, but wrong to phrase the demand in terms of function: evolution does not optimise functions, it optimises fitness, which need not be vital, only, on balance, advantageous. Tinbergen[94] distinguished four questions of any trait (mechanism, ontogeny, function and phylogeny) that do not substitute for one another; Mayr[95] drew the parallel between proximate and ultimate causation, still neglected across the behavioural

sciences[96,97]. The sleep-function literature often conflates them, reading evidence about mechanism, or about the laboratory consequences of sleep loss, as evidence about why sleep evolved. A trait can be strongly conserved because it is useful, without any single component being indispensable. The question should never have been "what is the function of sleep?" but "what fitness does sleep confer, and to whom?" Posed that way it stops demanding one answer: different advantages can accrue to different lineages, layered, of different evolutionary vintage.

The sleep elasticity hypothesis states that sleep is a layered adaptation of three components that arose at different times, answer to different pressures and are held to different standards of necessity. An accessory component (i), the largest in most species, is adaptive inactivity: an ecological buffer keeping an animal safely and economically out of the way during the hours in which activity would not repay itself. A useful component (ii) comprises the species-specific processes that came, over evolutionary time, to be discharged during that pre-existing state. A vital component (iii) would be whatever, if anything, is physiologically indispensable in the strict sense defined in Section I. Any instance of an animal's sleep mixes the three, in proportions varying by species, individual and circumstance. Most of the field's paradoxes dissipate once that mixture is resolved into its parts (Figure 1). I borrow elasticity from economics, where it measures how strongly one quantity responds to a change in another: sleep is elastic in that its duration responds to the opportunity cost of being awake, most of it stretching and contracting with ecology instead of meeting a fixed quota. The tripartite decomposition is the architecture that elasticity implies, and the vital layer is the part I hold most loosely: if it proves empty, the hypothesis stands with two layers.

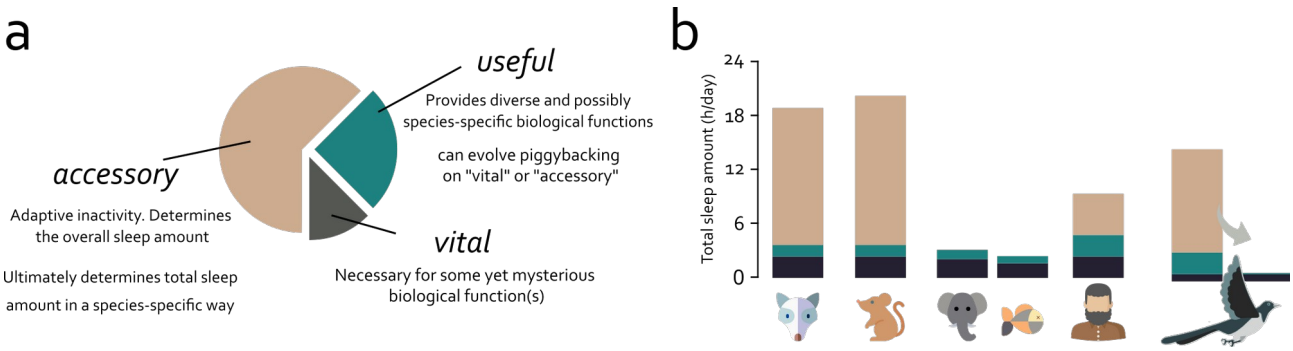

Figure 1. Sleep elasticity and the tripartite decomposition. (a) Sleep as three superimposed components of distinct evolutionary origin (defined in the text): the accessory buffer of adaptive inactivity (tan), the useful, largely lineage-specific processes exapted onto it (teal), and the hypothesised vital core (navy). Areas are illustrative. (b) The framework's central prediction: the sevenfold interspecific variation in total sleep duration is carried overwhelmingly by the accessory

buffer, while the useful and vital components stay small and stable across species. Approximate daily sleep is shown for an opossum, a pocket mouse, an elephant, a cavefish, a human and a bird, and the arrow marks the collapse of sleep during migration, when the buffer is shed and little but the core retained. The bat and the armadillo, conventional occupants of the long-sleeping end, are omitted as heterotherms (see text). The partitioning within each bar is schematic, not a measurement.

(1) Accessory sleep: the primordial buffer

The first component of the trine is what has been previously coined adaptive inactivity[11,98,99]: sleep as a state that keeps an animal safe, economically inactive when there is nothing more fitness-enhancing to do. On this view sleep evolved as a scheduling device to keep visually dependent animals out of the dangerous dark, desert insects out of the lethal afternoon, to conserve energy when foraging would not repay itself, and to synchronise activity so that feeding and mating happen when others are active. Its evolutionary value is genuine and often large, but it has no dedicated consequence at the level of the cell, tissue, or organ.

In 1975, Meddis[98] first proposed that sleep is an immobiliser, a state that preserves an animal's inactivity and "does not supply any unique physiological benefit"; that the time a species spends asleep "is limited by the time normally available after these essential activities have been completed", which is the residual account of sleep duration defended here. Horne[100] later divided sleep into an obligatory core and a dispensable remainder; Rial and colleagues[101] argued that sleep in mammals and birds may be "trivial", with any genuine functions "secondarily added"; and Siegel[11] recast the intuition as adaptive inactivity and assembled the modern comparative evidence for it.

That view is eliminativist: from the fact that sleep need not restore anything it concludes that sleep restores nothing, an unnecessary last step that has made the account easy to dismiss. I treat the useful functions instead as a genuine evolutionary stratum, keep the vital component as a testable hypothesis with a defined place in the stack, and state the account so that the relative size of the three components becomes an empirical question. The sleep elasticity hypothesis builds on evidence Meddis did not have: sleep measured in the wild, the phylogenetic lability of its regulation, and a molecular dissection of sleep pressure. Reading sleep through phylogeny and ecology, rather than mammalian physiology alone, is now a substantial literature[13,14,102] and can no longer be ignored.

The accessory component is easily misread as a null category, a residue left once the real functions are subtracted. It is nothing of the kind. Adaptive inactivity is a positive adaptation: a solution, under strong and continuous selection, to the problem of what an animal should do with the hours in which activity is unprofitable or dangerous. It has a fitness rationale, a mechanism (the raised arousal threshold that makes inactivity stable, discussed below), a regulatory system that defends it, and observable comparative signatures. To call it accessory is to say it discharges no dedicated cellular transaction, not that it does nothing.

Untied to a fixed physiological debt, the buffer can expand to fill most of the day in a small insectivore with a cheap, calorie-dense diet and a brief window in which being awake pays[9], and contract to minutes in a migrating bird[90], or in cave-adapted *Astyanax mexicanus* that evolved away most of their ancestral sleep once perpetual darkness removed the ecological rationale for quiescence[103]. This fits the long-noted correlation between sleep amount, body size and diet[69], whereby large herbivores that must forage continuously sleep little while small carnivores taking their calories in brief meals can afford to sleep most of the day. Much of what has been measured as an animal's "sleep need" is better described as an economical way to sit out the unprofitable hours[58,59,104] than as the repayment of a cellular debt[105,106].

Total sleep duration should therefore not be treated as the amount of an indispensable process. If much of an animal's sleep is elastic ballast, the hours on the clock are a compound: amount shaped by ecology and opportunity, timing by circadian organisation, depth by physiological operations and threat trade-offs, and a residue that bounds any indispensable core. Reading the total as one quantity, then asking what it is for, is the error beneath most of the field's paradoxes.

The strongest evidence for this buffering role comes from the recent turn towards measuring sleep in the wild[107]. Almost the entire comparative database on which vital-function arguments rest came from captive animals[12,108], and captivity distorts the measurement in the direction sleep elasticity predicts[109,110]. The first EEG recordings of a wild mammal found free-ranging three-toed sloths (*Bradypus variegatus*) sleeping 9.6 hours a day, more than six hours less than the ~15.9 recorded for the same species in captivity[111]; the result has been replicated in fifteen wild sloths across two species[109]. Removed from the demands of foraging and the risk of predation, the captive animal expands its accessory buffer to fill time that in the wild would be spent alert and alive; no fixed vital requirement could nearly double when an animal is moved into a cage.

(2) Useful sleep: species-specific exaptations

The accessory component is primordial: the ground state onto which the useful and vital layers were later added. Onto it evolution layered functions, and these are the processes most sleep research has actually studied: energy conservation, changes in extracellular transport and metabolite clearance, prefrontal restitution, mood regulation, memory-consolidation, synaptic and network maintenance, and immune support, among others[112]. What they share matters more than which of them is correct: all are exaptations of a pre-existing state, in the sense of Gould and Vrba[113]: a character that arose for one reason, or for none, and was later co-opted to another use. Accessory sleep supplies a long, reliable, sensorily disengaged window, and any process that benefits from one is under selection to migrate into it. Memory consolidation, synaptic renormalisation, clearance, and immune signalling[4,114,115] are, on this reading, exaptations of the sleep state, not the reasons it exists. As Williams[116] insisted, showing a trait to be useful does not show it to be an adaptation for that use.

Dreaming illustrates the relationship most clearly, because the direction of dependence is not in question. A dream is state-dependent: it can occur only during sleep, and no one has proposed that sleep evolved so that animals might dream. Whatever dreams do, they do because sleep already exists. Yet the case that they do something is not negligible. Dreaming has been argued to rehearse the recognition and avoidance of threat[117], to process and defuse emotional memory[118], to participate in the consolidation and reorganisation of experience[119], and, on a recent account, to protect the brain from overfitting to its own training data by injecting noise into its models of the world[120].

Flanagan[121,122] proposed that dreams are the spandrels of sleep, borrowing from Gould and Lewontin the term for a structure that arises as a by-product and is later put to use, and concluded that dreams have no function of their own; Revonsuo[117] replied with an explicitly adaptationist account. This reading declines both extremes as it declines them for sleep as a whole: dreams need not be functionless to be non-vital, and need not be indispensable to be useful.

That sleep’s functions are layered, and were multiplied over phylogeny, is not itself new. Krueger and colleagues[123] proposed sleep as a fundamental property of neuronal assemblies onto which higher functions accrued, and Bódizs[112] frames sleep as a multilevel system with older and newer functions and "no really ultimate function." Their base layer is still a function (energy homeostasis, timing); the accessory base I propose has no cellular function at all, only the ecological value that lets it serve as the elastic buffer explaining interspecific variability, the problem the multilevel view leaves untouched.

The useful layer should also show itself in development. Mammalian neonates sleep two thirds of the day, disproportionately in REM; an eight-week-old human infant may spend more than eight

hours a day in REM, several times the adult figure, a profile classically proposed to supply endogenous stimulation to a nervous system whose sensory experience is still limited[124,125]. Whether that mechanism survives modern scrutiny is beside the point: a function active in particular clades at particular ages, layered on the same buffer adults occupy more modestly, is what the useful layer predicts.

The crucial corollary is that, because these functions did not create sleep and do not drive its conservation, they need not be shared across lineages: what is useful to a large-brained mammal may be irrelevant to a fly or a jellyfish whose fitness never depended on it. This is what the divergent, species-specific homeostatic regulation across *Drosophila* species implies[126]: the defended, useful layer of sleep is evolutionarily labile, not conserved. Treating any one of these functions (synaptic homeostasis, clearance, proteostasis, *etc.*) as *the* function of sleep is the category error the field has repeatedly made. Each is a genuine benefit in the species where it obtains and none is the universal engine of sleep's conservation.

(3) Vital sleep: a hypothesis held open

The third component is the one the field assumed at the outset, and the verdict is unproven but not refuted. Is there a genuinely indispensable component, a "sleep calorie" that must occur during sleep and can occur nowhere else? The evidence is thin, and what keeps the question open is the observation that, pushed to the limit, animals compress sleep but never take it to zero. It is partitioned across hemispheres in cetaceans and many birds[127,128], fragmented into bouts of a few seconds in chinstrap penguins (*Pygoscelis antarcticus*)[129], or driven into wakefulness itself as local OFF periods in sleep-deprived rats[130], and microsleeps in drowsy humans[131]. The migrating sandpiper compresses sleep to minutes but keeps them. Why maintain a residue at all, sometimes at the cost of specialised machinery such as unihemispheric sleep[127], if the residue bought nothing? The temptation is to read it as the vital core showing through. But a useful function under strong selection would defend a residue just as fiercely: the sandpiper may keep its minutes because a navigator that lets its brain blur mid-ocean loses the path, and with it the race. That is a useful function as defined above, cognitive and fitness-critical, without being a sleep calorie.

The vital hypothesis has also protected itself by shrinking: each time a smaller residue is found, the vital function is quietly redefined to fit inside it. The claim survives every observation by rescaling the thing claimed, converging on a tautology: a vital function defined as whatever survives in the smallest residue can never be disproven, and an undisprovable claim does no scientific work. The remedy is a commitment the programme has never made: to state the floor in advance. How much

sleep does the transaction need, in what state, and what fails without it? Until that number is on the table the hypothesis is being accommodated, not tested. And even discharged in seconds of local cortical silence, a vital function could not explain why animals spend a third of their lives asleep.

IV. HOMEOSTATIC REGULATION AND SLEEP PRESSURE

One recurring confusion props up the vital-function assumption. A homeostatic control system is routinely offered as evidence that sleep discharges a vital function: we feel strong pressure to recover lost sleep, so that sleep must have been doing something essential. That sleep is homeostatically regulated is not in dispute. Slow-wave activity rises with time awake and dissipates with time asleep, and the two-process model of a homeostatic process and a circadian pacemaker is among the most successful descriptive frameworks in the field[132–134], with the set-point under demonstrable genetic control[135]. But regulation should not be conflated with function: a beneficial side effect is not thereby a function, any more than the mood lift from a good meal is the function of eating[112]. Any quantity held at a species-specific set-point needs a controller, but the controller states how much is maintained, not why. A homeostatic sleep controller likewise tells us that a set amount is defended and that falling short is punished with sleepiness, and says nothing about what that amount is for. The sleepiness that enforces it is a signal strong enough not to be ignored, a mechanism keeping an animal at its adaptive amount of accessory inactivity, felt as a need whether or not any vital transaction is pending.

There is also an observation the debt intuition cannot accommodate: animals do not repay most of the sleep they lose. Acute total deprivation is rarely followed by a rebound compensating the lost time in full[136]; rats given unlimited recovery after chronic restriction reclaim only a small fraction of what they lost[137]; and the most celebrated human case recovered from 264 hours of continuous wakefulness in a single night of under fifteen hours[138]. A deficit of some eighty hours was, in effect, written off. The very same is true in flies[36], and in the wild: free-ranging baboons that lose sleep in unfamiliar or crowded roosts do not compensate the following day[139].

The standard reply is that the debt is settled in intensity, not duration: recovery sleep is deeper, and slow-wave activity is the true currency of sleep pressure. This is the observation from which the two-process model was built[132,140], but it relocates the debt without rescuing it, and two difficulties follow. Firstly, intensity is not reliably defended: after chronic restriction rats show no compensatory rise in sleep intensity, despite a substantial accumulated loss[137]. And secondly, no exchange rate between hours and intensity has ever been specified. Without one, any shortfall in duration can be absorbed by a postulated increase in depth, and the claim that the debt is always

repaid becomes unfalsifiable. The elasticity framework predicts this. Its useful and vital components form a small, protected core, discharged in deep, early, high-intensity sleep whilst its accessory component is a large, dispensable buffer filling the lighter remainder. If deprivation removes both, recovery should then restore the core and abandon the buffer: intensity defended, duration shed. That is what is observed.

The founding observation of the two-process model is therefore its clearest empirical signature, not an objection to an elastic framework, and the same signature appears in the architecture of undisturbed sleep, where the deep, early portion is protected and the light, late portion shed.

The same reading answers an objection raised almost every time this argument is presented aloud. If most sleep is only accessory, why does losing a little feel so bad, the grogginess, the flattened mood, the blunted attention after a short night? Because sleepiness is a controller output, not a damage readout, and the two need not be proportional. Its proximate substrate is well understood: extracellular adenosine accumulates in the basal forebrain across waking and dissipates during sleep, and is sufficient to induce sleepiness independently of any damage it might index[141,142].

Consider what would happen without the aversive signal. If accessory sleep were a large, non-vital buffer carrying no penalty for skipping, an animal would rationally trade it away at the first competing incentive (forage a little longer, play a little more) and, night after night, grind down through the buffer to the useful and vital minima beneath. Natural selection cannot leave a defended set-point to voluntary cost–benefit accounting, because the incentives to skip sleep are immediate and chronic while the costs are deferred and probabilistic. So it installs a drive that makes falling short feel bad at once, whether or not anything vital was pending. The unpleasantness is the mechanism that stops the loss from being chosen, not the report of one. Being tired is how the set-point defends itself, and it is not the same thing as sleep deprivation. The logic is the one Nesse[143] named “the smoke detector principle”: when a protective response is cheap relative to the harm of failing to respond, the optimal system is tuned to fire readily, generating false alarms. A drive that switched on only when something vital was at stake would let the animal run its buffer to the bone every night before complaining. This is a general principle, not special pleading for sleep. Hunger is agonising long before starvation, and the neurons that signal it transmit an explicitly aversive signal whose relief, rather than any nutritional state, is what the animal learns to seek[144]; air hunger becomes unbearable at carbon-dioxide levels nowhere near lethal[145]. In each case the discomfort is engineered to force a behaviour reliably, and its severity reflects how badly that behaviour must be defended, not how close the body is to damage. Sleepiness is a motivational drive of this kind[146], one that, when it rises, actively suppresses the motivation to do anything else.

There is also a mechanistic reason to separate signal from damage. In sleep-deprived humans, cognitive lapses are immediately preceded by local sleep-like silencing of neurons in task-relevant cortex, so that the failure is a state intrusion rather than a depletion of any substrate[147–149]. Much of the "cognitive decline" of mild loss is plausibly the enforcement signal itself, sleepiness degrading attention and mood to make waking costly and sleep attractive[146]. The felt decline may, in large part, be the whip rather than the wound.

If the impairment is the signal, feeling tired and having lost sleep can be driven apart, which is what the fragmentation literature finds. Sleep broken by brief, repeated arousals produces next-day sleepiness and cognitive impairment comparable to outright sleep loss even when total sleep time is barely reduced, and when the disturbances are too slight to register as visible awakenings in the EEG[150–152]. The clinical counterpart is obstructive sleep apnoea, where the defining insult is fragmentation and daytime impairment tracks the arousal index[152]. The controller defends a consolidated quantity, not merely a quantity: even in the fly, minimal disturbance that removes hardly any sleep elicits a significant rebound[36].

This must not be pushed too far. If the felt impairment were only the signal, it should climb and fall with how sleepy people feel, and under chronic partial restriction it does not. Night after night, objective vigilance degrades, through an accumulating instability of the waking state rather than a uniform slowing[153,154], while the felt sleepiness levels off: people grow steadily more impaired while reporting themselves only moderately tired, and are strikingly poor judges of their own decline[155,156]. Signal and deficit are therefore distinct, dissociable both ways: the acute tiredness of a broken night is the controller demanding its set-point back, while the slow decline under chronic restriction is the useful layer degrading, impaired but not abolished. Both the whip and the wound are real; my claim is only that the field has often read the first as evidence of the second.

V. SHORT SLEEP IN HUMAN EPIDEMIOLOGY

Short sleep is associated, in populations numbering in the millions, with all-cause mortality, cardiovascular disease and dementia[157–159]. The associations are real, but less direct than they are made to seem, and the most telling results abandon duration altogether. Measured objectively across the UK Biobank, by accelerometry rather than questionnaire, what predicts survival is how regularly people sleep, not how much: the most regular sleepers die at some 30% lower all-cause and 40% lower cardiometabolic rates than the most irregular, the Sleep Regularity Index outpredicting duration outright[160], and the same holding for major cardiovascular events[161].

Human epidemiology carries the confound this paper has already met in the animal literature, where subjects lose sleep and are stressed at once and the two are hardly ever separated. Most human short sleep is imposed rather than chosen, by insecure work, money, illness, caring for someone, a failing marriage. Psychosocial stress is a replicated prospective cause of disturbed sleep[162], and it independently causes coronary disease at nearly the magnitude of the association it confounds: job strain raises coronary risk by about a quarter[163], and in INTERHEART psychosocial factors carried a population-attributable risk of 32%, behind only lipids and smoking[164,165]. Yet only two of the fifteen cohorts pooled in the principal cardiovascular-sleep meta-analysis adjusted for anything resembling stress[158] and where anyone has looked, the result is strong. In a thirty-year follow-up of five thousand Danish men, short sleep predicted death from ischaemic heart disease only among those taking tranquillisers or hypnotics, and neither short sleep nor medication was a risk factor alone, only their interaction[166]. In Whitehall II, short sleep raised coronary risk only in those also reporting disturbed rest[167], measured with a psychological-distress questionnaire. When Kivimäki's own group asked whether sleep is the pathway by which job insecurity reaches the heart, the effect ran instead through psychological distress, with none through sleep[168]. The most direct test dates from 2004. The JACC cohort followed 104,010 Japanese adults for a decade and set out, unusually, to examine sleep and mortality "after adjusting for several covariates, mental condition in particular"[169]. In that cohort, high mental stress falls monotonically with sleep duration, from 36% of men sleeping four hours or less to 14% of those sleeping ten or more. Adjusting for age, perceived mental stress and depressive symptoms (and nothing else) cuts the excess mortality of the shortest-sleeping men from 1.62 (1.26–2.09) to 1.29 (0.86–1.93). Adding the remaining covariates cuts it to 0.88. A single question about how stressful daily life feels is enough to abolish the association. Two qualifications: in women the association survived, and the non-mental covariates did as much work as the mental ones, so stress cannot be isolated as the culprit. Long sleep survived every model in both sexes, and the authors' own conclusion is this section's: that sleep duration "could be influenced by unknown physical and psychological conditions", in which case changing it "would not necessarily" reduce mortality. The second such test points the same way: Heslop and colleagues followed six thousand Scottish workers for twenty-five years[170]. Higher perceived stress predicted shorter sleep; short sleepers smoked and drank more, exercised less, were poorer and less often married; and once those correlates entered the model, the association between persistent short sleep and mortality lost significance in both sexes, as did that with cardiovascular death.

Adjustment can always be accused of missing something, which is why Mendelian randomisation is offered as the escape: alleles are dealt at conception and cannot be confounded by a hard life. But

the genetic instrument for short sleep is correlated with insomnia at rg = 0.73, and significantly with neuroticism and with depressive symptoms[171]. To a first approximation it is an instrument for stress-reactivity, and no study has conditioned on those traits, so pleiotropy through the stress axis is unexamined, not excluded. Applied across fifteen hundred phenotypes at once, the genetic evidence suggests instead that changes in sleep are predominantly the consequence of other conditions rather than their cause[172].

One observation is hardest of all to reconcile with the vital view. The first-line treatment for chronic insomnia, cognitive behavioural therapy, targets what the field's own validated instrument calls dysfunctional beliefs about sleep. Two of the beliefs it scores as dysfunctional, and directs the clinician to correct — *(i)* that one needs eight hours to function, and *(ii)* that poor sleep will damage one's health[173] , are almost verbatim the two propositions sleep science broadcasts to the public. Yet the therapy works without restoring sleep, producing a large fall in insomnia severity while adding, on average, eight minutes a night[174,175], and its most effective component does so by deliberately imposing a sleep debt of fifteen to twenty hours over four weeks[176]. Conversely, telling a person they slept badly, when they did not, measurably impairs their cognition the following day[177,178] and the canonical cognitive model of insomnia has held since 2002 that worry about the consequences of poor sleep is itself sufficient to produce a real deficit[179].

VI. THE STABILITY OF INACTIVITY

If the point is to keep an animal safely inactive, why go offline, why not rest quietly awake, conscious and responsive and get the best of both worlds, *i.e.* the safety that comes from refuge and the alertness that comes from wakefulness? Because quiet wakefulness is only metastable: stable enough to rest in, but easily broken. Keeping the sensory and motor systems online leaves the animal liable to be tipped into full wakefulness by the next salient stimulus. Sleep's defining feature, the elevated arousal threshold[180,181] exists to hold inactivity in place. Withdrawing responsiveness is not a side effect of the accessory state; it is what makes sustained inactivity possible. Rial and colleagues put the same intuition in words, arguing that sleep is an upgrade of wakeful rest because rest can be abandoned the moment something intrudes, whereas sleep enforces immobility[182].

This can be made precise and tested on real data. Figure 2 models rest as a state leaking to wakefulness at random, at a rate (its hazard, λ) fixed by the arousal threshold (θ): the higher the threshold, the slower the leak and the longer a bout survives. A quietly awake animal cannot hold inactivity for more than a few minutes; a sleeping one, exposed to the same stimuli with a raised

threshold, holds it for tens of minutes, keeping nearly all of the inactive window (Figure 2a,b). Scored by hidden Markov model across seven *Drosophila* species (close to a million bouts – new analysis on data from[126]), quiet wakefulness averages 2.2 minutes and never exceeds twenty-one, while deep sleep averages 27 and runs to twelve hours (Figure 2f). Quiet wakefulness cannot be sustained. Two further findings were not assumed by the model. First, a fly knocked out of rest takes about half an hour to settle again (not the few minutes the model grants), so resting while awake preserves even less of the night than estimated. Second, deep sleep, once entered, is not shed at random. No bout is shorter than twelve minutes, and a bout grows likelier to end only as it ages, the committed pattern seen in mammalian sleep, now in an insect. Sleep therefore holds inactivity more firmly than the bare model assumes, and quiet wakefulness less. In the fly, quiet wakefulness slips into sleep eleven times for every once it wakes: left to itself, rest rolls downhill into sleep.

Why not raise the threshold without limit? Because the same gate that filters benign stimuli filters genuine ones: raising the threshold suppresses false alarms but also lowers the chance of waking to a real one. Since a looming predator is more salient than a rustling leaf — the sleeping brain rouses far more readily to one's own name than to matched neutral sound[183–185], thalamic circuits gating the transition[186] — a band of raised thresholds suppresses false alarms while preserving threat detection. That band is sleep: staying quietly awake is always the worse choice, and so is bottomless sleep; the optimum sits between them (Figure 2c).

The same threshold predicts how sleep depth varies with ecology: the more dangerous the environment, the lighter the sleep (Figure 2d). Mallards (*Anas platyrhynchos*) at a group's exposed edge sleep one-eyed, that eye outward, more so when risk is higher[187]; rats restructure sleep under predator cues[55]; and sleep architecture now reads as a risk-sensitive trait rather than a fixed requirement[188]. What Rattenborg and colleagues read as proof of an essential function (the vulnerability of a sleeping animal) is on this account where selection settles the trade-off between sleeping deeply and staying alert: as deep as ecology allows, and no deeper. The metastability is not a modelling convenience: the sleep–wake switch is built from mutually inhibitory circuitry that makes the two states bistable, each self-reinforcing once entered[189,190], with the threshold setting how deeply the resting state sits. And both rates can be measured directly: in *Drosophila* by arousal-threshold probing[126,185,191] and in mammals by classical acoustics[180,181].

Energy saving is not the alternative: metabolic rate in human sleep falls only modestly below quiet waking[192]. To whatever extent full disconnection buys something inactivity cannot, that is the upper layers at work: accessory sleep sets how much and when, while the useful and vital components set how deep. That difference may be the framework's best hope of telling them apart. That division

predicts the architecture within an episode: the hard-to-skip components should be discharged first, so sleep should begin deep and lighten. It does: across *Drosophila*, sleep depth, scored by the model and checked against a robotically measured arousal threshold, peaks early in the night and then declines[126] (Figure 2e), as slow-wave activity does in mammals[114,140], the lightening being the threshold falling as the night wears on, the same signature the homeostat shows after deprivation, when it defends intensity and lets duration go.

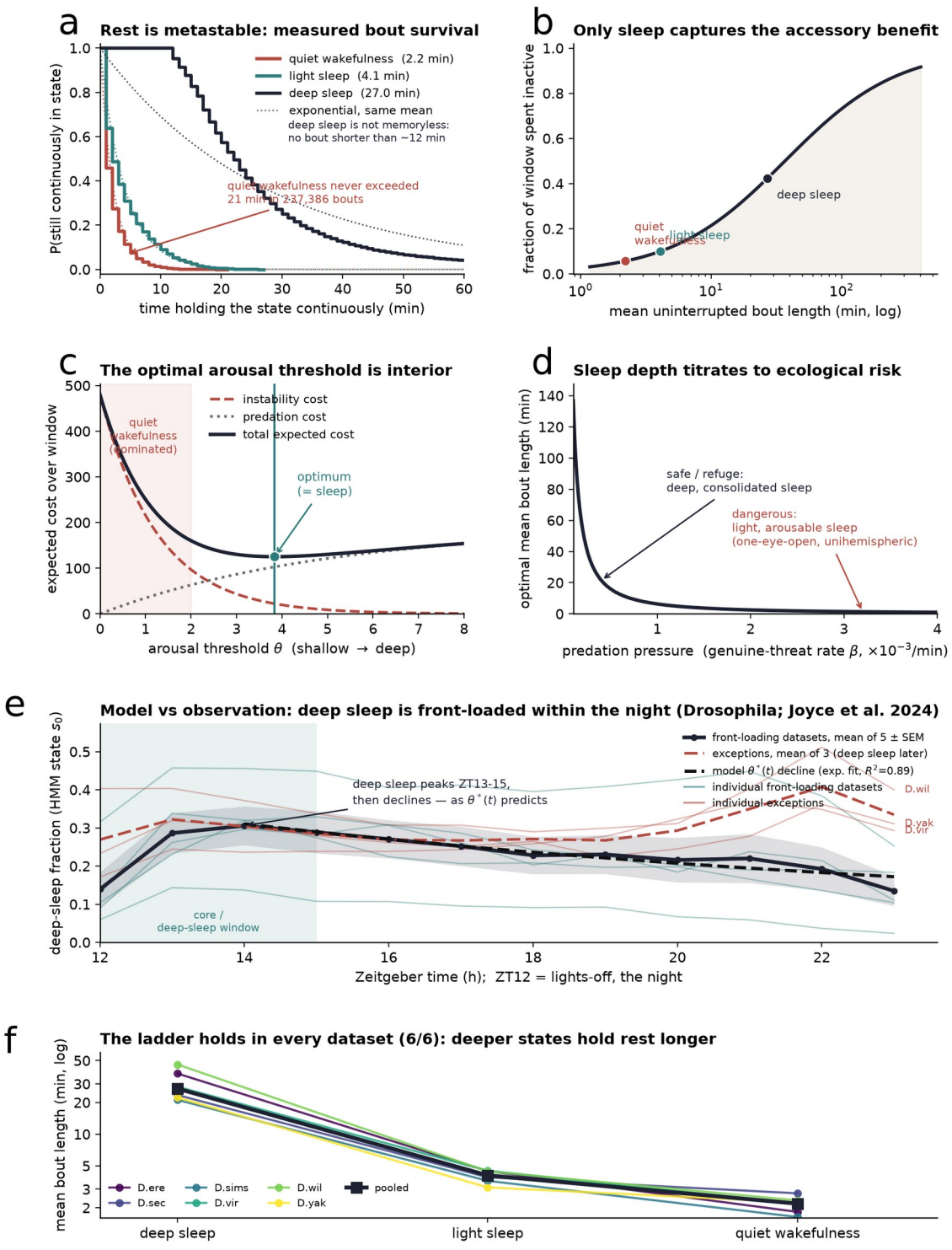

Figure 2. Why sleep rather than quiet wakefulness: a metastability model of the accessory state. Rest decays to active wakefulness as a Poisson process with hazard λ set by the arousal threshold θ (deeper sleep = higher θ = lower λ). (a) Measured bout survival across the *Drosophila* datasets (Kaplan–Meier over 516,543 Viterbi-decoded HMM bouts; provenance, exclusions and censoring in the Supporting Information), with the exponential of the same mean dotted. Quiet wakefulness lasts 2.2 min on average and never exceeded 21 min; deep sleep lasts 27 min, is never shorter than

12, and runs to 12 h. Its hazard rises with time, so deep sleep is not memoryless and holds inactivity better than the model assumes. (b) Fraction of the rest window spent inactive, the accessory benefit, against mean bout length, using the measured resettling time after an ejection (37 min, the mean full-awake bout): quiet wakefulness banks 6% of the window, deep sleep 42%. (c) Expected cost, decomposed into an instability term (false-alarm arousals, decreasing in θ) and a predation term (missed threats, increasing in θ). Because genuine threats are more salient, the total has an interior optimum in the sleep range and quiet wakefulness is strictly dominated. (d) Optimal sleep depth titrates to ecological risk. (e) Deep-sleep prevalence across the night, the ordinate effectively θ (state scoring and depth validation from Joyce et al., 2024). Thin lines are the eight datasets: five front-load deep sleep (teal, mean ± SEM in navy, fitted bounded exponential dashed), while three (rust; *D. virilis*, *D. willistoni*, *D. yakuba*) place it in the second half of the night. (f) The ladder holds in every dataset. Cost parameters in (c) and (d) are illustrative.

VII. COMPARATIVE EVIDENCE FOR A LAYERED ACCOUNT

A framework earns its place by explaining what its rivals cannot. If sleep serves one vital function, that function must be present and defended wherever sleep is found; if sleep is a layered, exapted, elastic state, we should instead find different loads on the same cart. The comparative record answers at every scale the comparison can be controlled. Within a genus, the rebound after deprivation is present in *Drosophila melanogaster* and absent in six close relatives[126]. Within a clade, two lizards as different as the bearded dragon (*Pogona vitticeps*) and the tegu (*Salvator merianae*) have sleep architectures that cannot be reconciled[193]. Within a single skull, the northern fur seal (*Callorhinus ursinus*) at sea suppresses REM by 96% for a fortnight and never repays it, while slow-wave sleep is defended and rebounds sharply[92]: one state written off, the other reclaimed. The clincher is a double dissociation: cavefish lose most of their sleep but keep the homeostat that would defend it[194], while flies keep the sleep and lose the homeostat. What is conserved is general machinery. The LKB1–SIK3–HDAC cassette sets sleep need across mice, flies and worms[195,196], but it is a repurposed nutrient-sensing module of the AMPK family, not a sleep-dedicated organ; even adenosine, the mammalian somnogen, is dispensable in the fly[197,198]. The cart is conserved, not its load, which is the reading the comparative data have supported for fifty years. Others glimpsed it: Anafi, Kayser and Raizen, that what is conserved may be "a temporal structure and organizational principle" rather than a gene[14]; Joiner, that sleep’s functions were co-opted onto "the inactive state that lies at the core of sleep"[102]. What was missing was the architecture. A field

that keeps discovering new functions for sleep is not closing in on the function of sleep, but finding that different animals have loaded different things onto the same cart.

VIII. PREDICTIONS AND POTENTIAL REFUTATIONS

Table 1 sets out the principal claims of the sleep elasticity hypothesis with the evidence for each, the leading alternative reading, the confidence each warrants, and the observation that would decide it. Two points deserve more than a table entry. The first is the kind of evidence that could settle any of this. Most of the case made here, and almost all of the case made by the field, rests on comparisons between species, which are exposed to phylogenetic non-independence, allometry, and the fact that sleep is measured by different methods, in different conditions, in different clades. The decisive tests are within-species manipulations: varying foraging demand, predation cues, thermal stress, refuge quality, mating opportunity and social environment in one species, while measuring depth, architecture, rebound, duration, and subsequent performance, survival and reproductive output. The framework predicts that sleep amount will move substantially and reversibly with these variables while some residue proves refractory to all of them, and that the useful and vital functions will track that residue, not the total. *Drosophila suits the programme, since every one of those variables is manipulable and sleep can be measured at scale and at depth by high-throughput video tracking with closed-loop stimulus delivery*[35,126]; the design generalises, and miniaturised biologgers and remote EEG now make versions of it feasible in free-living animals[107,199]. Field studies of wild populations, long marginal to a discipline organised around laboratory models and human health, will have to carry much of this.

The second is that the framework's central quantities are not yet measurable, which is its principal weakness. No method exists for decomposing an animal's sleep into accessory, useful and vital components. The most promising handle is depth, taken as the arousal threshold. This may be why the search has failed. For decades the field has hunted the function of NREM, transfixed by the slow-wave signature and forced to explain why so striking a feature appears in only some species. But the slow wave is the electrophysiological correlate of a brain at a high arousal threshold, and it is the threshold, not the signature, that is conserved. Arousal threshold is the tree and the EEG its shadow. Asking instead what fitness deep sleep confers dissolves the evolutionary paradox, since every animal has been shown to have gradations of arousal. Since every increment of arousal threshold is paid for in vigilance, the depth an animal reaches measures what the sleep at that depth is worth to it: the shallow end, costing least, holds the accessory buffer, and the deep, defended end is where any useful or vital load must be discharged. The prediction is sharp enough to fail:

manipulate the ecology, and the depth that shifts is accessory, while any that holds is not. Devising such a method, through selective ablation of each layer, the ecological manipulations above, or the depth-resolved arousal measurements that high-throughput tracking now makes possible[126,181], is the framework's most important experimental challenge. Until then the proportions in Figure 1 remain a hypothesis, not a result.

One quantity, at least, is already well defined. Economists estimate an elasticity as a ratio of proportional changes, and sleep elasticity can be estimated the same way: the proportional change in sleep duration over the proportional change in the return to being awake. The within-species manipulations proposed above supply both terms, since each varies that return while sleep is measured. A component whose elasticity is near zero is one ecology cannot move; a large elasticity marks the accessory buffer. Economics has a century of technique for estimating elasticities when the driving variable cannot be set directly, and sleep science would do better to borrow those tools than to derive them again.

Claim	Principal evidence	Main alternative reading	Confidence	Crucial test
Sleep contains an ecologically elastic component	wild/captive contrasts; migratory and breeding compression; predation-linked architecture	measurement artefact; state misclassification in the wild	strong	controlled within-species ecological release and imposition
That component is large — often most of sleep	extreme compressions tolerated without collapse	compressed sleep becomes more intense or efficient rather than shorter	moderate	decomposition of sleep into components; intensity-corrected accounting
The accessory layer is evolutionarily primordial	conserved circadian organisation with labile homeostasis across *Drosophila*; quiescence in basal metazoans	stabilising selection; secondary divergence; basal "sleep" may not be homologous	weak	broad ancestral-state reconstruction; functional assays in basal metazoans
Known functions of sleep are lineage-specific exaptations	divergent architecture and homeostatic regulation across related species	one conserved function with different implementations	moderate	homologous functional assays across species
Quiet wakefulness is metastable, and sleep depth titrates to risk	the arousal threshold is the control parameter of a bistable switch; ecological titration of sleep depth	quiet rest is as stable as sleep; depth is set by physiology alone	moderate	measure the quiet-wake-to-wake and sleep-to-wake hazards for matched stimuli; manipulate predation cues
Sleep architecture follows the order of importance	deep sleep front-loaded and dissipating (Figure 2e); recovery restores intensity, not duration	slow-wave dissipation reflects a single unitary process	moderate	curtail sleep and ask which portion is shed; deprive and ask which portion returns
Sleepiness is a set-point enforcer, not a damage readout	motivational-drive data; fragmentation costs; rebound after minimal perturbation	impairment is direct disruption of useful sleep	moderate	dissociation of signal from deficit
A physiologically indispensable core exists	irreducible residue in extreme sleep-reducers	the residue discharges a rapid *useful* process; micro-sleep	unresolved	clean, stress-free deprivation below the residue

Table 1. The framework's claims, and the confidence they warrant.

IX. SYNTHESIS

The search for the function of sleep has been shaped, for fifty years, by a question that presupposed its own answer. Begin by assuming sleep must be vital, and variability becomes a nuisance to explain away, extreme sleepers become errors to dismiss, and every newly discovered benefit becomes a candidate for the function. Begin instead by asking what fitness sleep confers, and the picture reorganises itself. Sleep looks less like a single indispensable process than a layered adaptation: an ancient, flexible buffer of adaptive inactivity; a set of useful, species-specific functions exapted onto it; and perhaps a small vital core whose existence remains an open question, not a foundational assumption. This reorders the functions already on the table into an arrangement that accommodates the comparative data as they are.

If the reader takes one claim from this paper, let it be the narrowest and best supported: total sleep duration is not a direct measure of one universal physiological necessity. Everything else is a gradient of decreasing confidence built on that foundation: that the elastic component is large (credible), that it came first (suggestive), and that no physiologically indispensable core exists (unresolved, and not asserted). A reader who believes a vital core will eventually be found is not refuting the framework but locating themselves within it. Whether such a core sits at the bottom of the stack is worth pursuing, and I would be glad to see it answered either way. But it should be pursued as a hypothesis, tested at last against the variability that has always been sleep's most important and most ignored fact, instead of assumed as the price of admission to the field.

X. CONCLUSIONS

(1) The three observations conventionally taken to establish that sleep is physiologically indispensable do not carry that weight. The lethality literature is confounded by stress throughout, in invertebrates and mammals alike, and the same confound runs through the human epidemiology.

(2) Sleep is costly only intermittently. Predation and exposure are largely met by the refuge an inactive animal needs in any case, and the reproductive cost is paid only where breeding opportunity is continuous.

(3) The ubiquity of sleep does not establish its indispensability. Circadian rhythms are more widely conserved and confer large fitness advantages, yet many animals with a disabled clock live and breed normally in the laboratory.

(4) Total sleep duration should not be treated as the amount of any indispensable process.

(5) Sleep is better read as a layered adaptation of three components: an accessory buffer of adaptive inactivity, useful lineage-specific processes exapted onto that pre-existing state, and a vital core whose existence remains an open question.
(6) The accessory component carries most of the sevenfold interspecific variation in sleep duration, and expands in captivity once foraging demand and predation risk are removed.
(7) Homeostatic regulation establishes that an amount is defended, not what that amount is for. Sleepiness is a controller output calibrated to the behavioural control problem rather than a readout of damage.
(8) Quiet wakefulness cannot substitute for sleep because it is only metastable. The elevated arousal threshold is what makes sustained inactivity possible, and the threshold, not the slow wave, is the conserved quantity.
(9) The framework's central quantities are not yet measurable. No method exists for decomposing an animal's sleep into accessory, useful and vital components, and devising one is its most important experimental challenge.

XI. ACKNOWLEDGEMENTS

The elastic, tripartite model of sleep was first sketched in 2018 and has been under continuous development since. It has been improved beyond recognition in the intervening years by all the members of my laboratory and the many colleagues who engaged with it, at meetings, in seminars and in argument, and who offered it both their constructive criticism and their excitement. I am grateful to all of them; the errors that remain are mine.

Competing interests

The author declares no competing interests.

Data and code availability

All primary behavioural data come from our own previously published *Drosophila* recordings[126]. The derived data and the Python script that generates Figure 2 are supplied as supplementary files, described in the accompanying README. The analysis in Figure 2 is original to this article: the hidden Markov segmentation of the recordings, the survival analysis of 516,543 decoded bouts and the metastability model fitted to them are reported here for the first time.

XII. REFERENCES

1. Rechtschaffen, A. The control of sleep. in *Human Behavior and Its Control* (ed. Hunt, W. A.) 75–92 (Schenkman, Cambridge, MA, 1971).
2. Rechtschaffen, A. Current Perspectives on the Function of Sleep. *Perspect. Biol. Med.* **41**, 359–390 (1998).
3. Cirelli, C. & Tononi, G. Is Sleep Essential? *PLOS Biol.* **6**, e216 (2008).
4. Tononi, G. & Cirelli, C. Sleep and the Price of Plasticity: From Synaptic and Cellular Homeostasis to Memory Consolidation and Integration. *Neuron* **81**, 12–34 (2014).
5. Sharon, O., Ben Simon, E., Shah, V. D., Desel, T. & Walker, M. P. The new science of sleep: From cells to large-scale societies. *PLOS Biol.* **22**, e3002684 (2024).
6. de Manacéïne, M. Quelques Observations Experimentales sur L'Influence de L'Insomnie Absolue. *Arch. Ital. Biol.* **21**, 322–325 (1894).
7. Everson, C. A., Bergmann, B. M. & Rechtschaffen, A. Sleep Deprivation in the Rat: III. Total Sleep Deprivation. *Sleep* **12**, 13–21 (1989).
8. Vaccaro, A. *et al.* Sleep Loss Can Cause Death through Accumulation of Reactive Oxygen Species in the Gut. *Cell* **181**, 1307-1328.e15 (2020).
9. Allison, T. & Cicchetti, D. V. Sleep in mammals: ecological and constitutional correlates. *Science* **194**, 732–734 (1976).
10. Lima, S. L., Rattenborg, N. C., Lesku, J. A. & Amlaner, C. J. Sleeping under the risk of predation. *Anim. Behav.* **70**, 723–736 (2005).
11. Siegel, J. M. Sleep viewed as a state of adaptive inactivity. *Nat. Rev. Neurosci.* **10**, 747–753 (2009).
12. Campbell, S. S. & Tobler, I. Animal sleep: A review of sleep duration across phylogeny. *Neurosci. Biobehav. Rev.* **8**, 269–300 (1984).
13. Keene, A. C. & Duboue, E. R. The origins and evolution of sleep. *J. Exp. Biol.* **221**, jeb159533 (2018).
14. Anafi, R. C., Kayser, M. S. & Raizen, D. M. Exploring phylogeny to find the function of sleep. *Nat. Rev. Neurosci.* **20**, 109–116 (2018).

15. Tobler, I. & Neuner-Jehle, M. 24-h variation of vigilance in the cockroach *Blaberus giganteus*. *J. Sleep Res.* **1**, 231–239 (1992).
16. Nath, R. D. *et al.* The Jellyfish *Cassiopea* Exhibits a Sleep-like State. *Curr. Biol.* **27**, 2984-2990.e3 (2017).
17. Nunn, C. L., Samson, D. R. & Krystal, A. D. Shining evolutionary light on human sleep and sleep disorders. *Evol. Med. Public Health* **2016**, 227–243 (2016).
18. Bergmann, B. M. *et al.* Sleep Deprivation in the Rat: V. Energy Use and Mediation. *Sleep* **12**, 31–41 (1989).
19. Kushida, C. A., Bergmann, B. M. & Rechtschaffen, A. Sleep Deprivation in the Rat: IV. Paradoxical Sleep Deprivation. *Sleep* **12**, 22–30 (1989).
20. Selye, H. A Syndrome produced by Diverse Nocuous Agents. *Nature* **138**, 32–32 (1936).
21. Harris, R. B. S. Chronic and acute effects of stress on energy balance: are there appropriate animal models? *Am. J. Physiol.-Regul. Integr. Comp. Physiol.* **308**, R250–R265 (2015).
22. Meddings, J. B. & Swain, M. G. Environmental stress–induced gastrointestinal permeability is mediated by endogenous glucocorticoids in the rat. *Gastroenterology* **119**, 1019–1028 (2000).
23. Söderholm, J. D. & Perdue, M. H. II. Stress and intestinal barrier function. *Am. J. Physiol.-Gastrointest. Liver Physiol.* **280**, G7–G13 (2001).
24. Zareie, M. *et al.* Probiotics prevent bacterial translocation and improve intestinal barrier function in rats following chronic psychological stress. *Gut* **55**, 1553–1560 (2006).
25. Nollet, M., Wisden, W. & Franks, N. P. Sleep deprivation and stress: a reciprocal relationship. *Interface Focus* **10**, 20190092 (2020).
26. Meerlo, P., Sgoifo, A. & Suchecki, D. Restricted and disrupted sleep: Effects on autonomic function, neuroendocrine stress systems and stress responsivity. *Sleep Med. Rev.* **12**, 197–210 (2008).
27. Longordo, F., Kopp, C. & Lüthi, A. Consequences of sleep deprivation on neurotransmitter receptor expression and function. *Eur. J. Neurosci.* **29**, 1810–1819 (2009).

28. Colavito, V. *et al.* Experimental sleep deprivation as a tool to test memory deficits in rodents. *Front. Syst. Neurosci.* **7**, 106 (2013).
29. Newman, S. M., Paletz, E. M., Obermeyer, W. H. & Benca, R. M. Sleep Deprivation in Pigeons and Rats Using Motion Detection. *Sleep* **32**, 1299–1312 (2009).
30. Sang, D. *et al.* Prolonged sleep deprivation induces a cytokine-storm-like syndrome in mammals. *Cell* **186**, 5500-5516.e21 (2023).
31. Tobler, I. Effect of forced locomotion on the rest-activity cycle of the cockroach. *Behav. Brain Res.* **8**, 351–360 (1983).
32. Stephenson, R., Chu, K. M. & Lee, J. Prolonged deprivation of sleep-like rest raises metabolic rate in the Pacific beetle cockroach, Diploptera punctata (Eschscholtz). *J. Exp. Biol.* **210**, 2540–2547 (2007).
33. Ewing, L. S. Fighting and Death from Stress in a Cockroach. *Science* **155**, 1035–1036 (1967).
34. Shaw, P. J., Tononi, G., Greenspan, R. J. & Robinson, D. F. Stress response genes protect against lethal effects of sleep deprivation in *Drosophila*. *Nature* **417**, 287–291 (2002).
35. Geissmann, Q. *et al.* Ethoscopes: An Open Platform For High-Throughput Ethomics. *PLOS Biol.* **15**, e2003026 (2017).
36. Geissmann, Q., Beckwith, E. J. & Gilestro, G. F. Most sleep does not serve a vital function: Evidence from *Drosophila melanogaster. Sci. Adv.* **5**, eaau9253 (2019).
37. Guo, L., Ferretti, V. & Gilestro, G. F. Sleep Deprivation Primes Synaptic Vulnerability Without Inducing Oxidative Damage: A Mechanistic Reappraisal. 2025.09.05.674430 Preprint at https://doi.org/10.1101/2025.09.05.674430 (2025).
38. Kume, K. A *Drosophila* dopamine transporter mutant, fumin (fmn), is defective in arousal regulation. *Sleep Biol. Rhythms* **4**, 263–273 (2006).
39. He, Y. *et al.* The Transcriptional Repressor DEC2 Regulates Sleep Length in Mammals. *Science* **325**, 866–870 (2009).
40. Shi, G. *et al.* A Rare Mutation of β1-Adrenergic Receptor Affects Sleep/Wake Behaviors. *Neuron* **103**, 1044-1055.e7 (2019).

41. Xing, L. *et al.* Mutant neuropeptide S receptor reduces sleep duration with preserved memory consolidation. *Sci. Transl. Med.* **11**, eaax2014 (2019).
42. Harbison, S. T., Negron, Y. L. S., Hansen, N. F. & Lobell, A. S. Selection for long and short sleep duration in *Drosophila melanogaster* reveals the complex genetic network underlying natural variation in sleep. *PLOS Genet.* **13**, e1007098 (2017).
43. Cirelli, C. *et al.* Reduced sleep in *Drosophila* Shaker mutants. *Nature* **434**, 1087–1092 (2005).
44. Koh, K. *et al.* Identification of SLEEPLESS, a Sleep-Promoting Factor. *Science* **321**, 372–376 (2008).
45. Stavropoulos, N. & Young, M. W. insomniac and Cullin-3 Regulate Sleep and Wakefulness in *Drosophila*. *Neuron* **72**, 964–976 (2011).
46. Bouybayoune, I. *et al.* Transgenic Fatal Familial Insomnia Mice Indicate Prion Infectivity-Independent Mechanisms of Pathogenesis and Phenotypic Expression of Disease. *PLOS Pathog.* **11**, e1004796 (2015).
47. Popper, K. R. *The Logic of Scientific Discovery.* (Hutchinson, London, 1959).
48. Akaarir, M. *et al.* The Disputable Costs of Sleeping. *Biology* **14**, 352 (2025).
49. Charnov, E. L. Optimal foraging, the marginal value theorem. *Theor. Popul. Biol.* **9**, 129–136 (1976).
50. Lesku, J. A. *et al.* Adaptive sleep loss in polygynous pectoral sandpipers. *Science* **337**, 1654–1658 (2012).
51. Cardon, J. H., Eide, E. R., Phillips, K. L. & Showalter, M. H. Interacting circadian and homeostatic processes with opportunity cost: A mathematical model of sleep with application to two mammalian species. *PLOS ONE* **13**, e0208043 (2018).
52. Anderson, J. R. Sleep, sleeping sites, and sleep-related activities: Awakening to their significance. *Am. J. Primatol.* **46**, 63–75 (1998).
53. Ellison, G. *et al.* Sleeping Site Selection in the Nocturnal Northern Lesser Galago (*Galago senegalensis*) Supports Antipredator and Thermoregulatory Hypotheses. *Int. J. Primatol.* **40**, 276–296 (2019).

54. Pozzi, L., Voskamp, M. & Kappeler, P. M. The effects of body size, activity, and phylogeny on primate sleeping ecology. *Am. J. Biol. Anthropol.* **179**, 598–608 (2022).
55. Lesku, J. A. *et al.* Predator-induced plasticity in sleep architecture in wild-caught Norway rats (*Rattus norvegicus*). *Behav. Brain Res.* **189**, 298–305 (2008).
56. Lima, S. L. & Rattenborg, N. C. A behavioural shutdown can make sleeping safer: a strategic perspective on the function of sleep. *Anim. Behav.* **74**, 189–197 (2007).
57. Gilbert, C. *et al.* One for all and all for one: the energetic benefits of huddling in endotherms. *Biol. Rev.* **85**, 545–569 (2010).
58. Berger, R. & Philips, N. Sleep and Energy Conservation. *Physiology* **8**, 276–281 (1993).
59. Schmidt, M. H. The energy allocation function of sleep: A unifying theory of sleep, torpor, and continuous wakefulness. *Neurosci. Biobehav. Rev.* **47**, 122–153 (2014).
60. Bronson, F. H. *Mammalian Reproductive Biology.* (University of Chicago Press, Chicago, 1989).
61. Bronson, F. H. Climate change and seasonal reproduction in mammals. *Philos. Trans. R. Soc. B Biol. Sci.* **364**, 3331–3340 (2009).
62. Nakane, Y. & Yoshimura, T. Photoperiodic Regulation of Reproduction in Vertebrates. *Annu. Rev. Anim. Biosci.* **7**, 173–194 (2019).
63. Machado, D. R. *et al.* Identification of octopaminergic neurons that modulate sleep suppression by male sex drive. *eLife* **6**, e23130 (2017).
64. Beckwith, E. J., Geissmann, Q., French, A. S. & Gilestro, G. F. Regulation of sleep homeostasis by sexual arousal. *eLife* **6**, e27445 (2017).
65. Keene, A. C. *et al.* Clock and cycle Limit Starvation-Induced Sleep Loss in *Drosophila*. *Curr. Biol.* **20**, 1209–1215 (2010).
66. Murphy, K. R. *et al.* Postprandial sleep mechanics in *Drosophila*. *eLife* **5**, e19334 (2016).
67. Duhart, J. M., Baccini, V., Zhang, Y., Machado, D. R. & Koh, K. Modulation of sleep-courtship balance by nutritional status in *Drosophila*. *eLife* **9**, e60853 (2020).
68. Slocumb, M. E. *et al.* Enhanced Sleep Is an Evolutionarily Adaptive Response to Starvation Stress in *Drosophila*. *PLOS ONE* **10**, e0131275 (2015).

69. Capellini, I., Barton, R. A., McNamara, P., Preston, B. T. & Nunn, C. L. PHYLOGENETIC ANALYSIS OF THE ECOLOGY AND EVOLUTION OF MAMMALIAN SLEEP. *Evolution* **62**, 1764–1776 (2008).
70. Gravett, N. *et al.* Inactivity/sleep in two wild free-roaming African elephant matriarchs – Does large body size make elephants the shortest mammalian sleepers? *PLOS ONE* **12**, e0171903 (2017).
71. Raichlen, D. A. *et al.* Sitting, squatting, and the evolutionary biology of human inactivity. *Proc. Natl. Acad. Sci.* **117**, 7115–7121 (2020).
72. Yetish, G. *et al.* Natural Sleep and Its Seasonal Variations in Three Pre-industrial Societies. *Curr. Biol.* **25**, 2862–2868 (2015).
73. Samson, D. R., Crittenden, A. N., Mabulla, I. A., Mabulla, A. Z. P. & Nunn, C. L. Chronotype variation drives night-time sentinel-like behaviour in hunter–gatherers. *Proc. R. Soc. B Biol. Sci.* **284**, 20170967 (2017).
74. Marshall, A. *Principles of Economics*. (Macmillan, London, 1890).
75. Konopka, R. J. & Benzer, S. Clock mutants of *Drosophila melanogaster. Proc. Natl. Acad. Sci. U. S. A.* **68**, 2112–2116 (1971).
76. van der Horst, G. T. J. *et al.* Mammalian Cry1 and Cry2 are essential for maintenance of circadian rhythms. *Nature* **398**, 627–630 (1999).
77. DeCoursey, P. J., Krulas, J. R., Mele, G. & Holley, D. C. Circadian Performance of Suprachiasmatic Nuclei (SCN)-Lesioned Antelope Ground Squirrels in a Desert Enclosure. *Physiol. Behav.* **62**, 1099–1108 (1997).
78. DeCoursey, P. J., Walker, J. K. & Smith, S. A. A circadian pacemaker in free-living chipmunks: essential for survival? *J. Comp. Physiol. [A]* **186**, 169–180 (2000).
79. Ouyang, Y., Andersson, C. R., Kondo, T., Golden, S. S. & Johnson, C. H. Resonating circadian clocks enhance fitness in cyanobacteria. *Proc. Natl. Acad. Sci.* **95**, 8660–8664 (1998).

80. Woelfle, M. A., Ouyang, Y., Phanvijhitsiri, K. & Johnson, C. H. The Adaptive Value of Circadian Clocks: An Experimental Assessment in Cyanobacteria. *Curr. Biol.* **14**, 1481–1486 (2004).

81. Dodd, A. N. *et al.* Plant Circadian Clocks Increase Photosynthesis, Growth, Survival, and Competitive Advantage. *Science* **309**, 630–633 (2005).

82. Lesku, J. A., Roth II, T. C., Amlaner, C. J. & Lima, S. L. A Phylogenetic Analysis of Sleep Architecture in Mammals: The Integration of Anatomy, Physiology, and Ecology. *Am. Nat.* **168**, 441–453 (2006).

83. Lesku, J. A., Roth, T. C., Rattenborg, N. C., Amlaner, C. J. & Lima, S. L. Phylogenetics and the correlates of mammalian sleep: A reappraisal. *Sleep Med. Rev.* **12**, 229–244 (2008).

84. Harding, C. D., Yovel, Y., Peirson, S. N., Hackett, T. D. & Vyazovskiy, V. V. Re-examining extreme sleep duration in bats: implications for sleep phylogeny, ecology, and function. *Sleep* **45**, zsac064 (2022).

85. Affanni, J. M., Cervino, C. O. & Marcos, H. J. A. Absence of penile erections during paradoxical sleep. Peculiar penile events during wakefulness and slow wave sleep in the armadillo. *J. Sleep Res.* **10**, 219–228 (2001).

86. Harris, D. V., Walker, J. M. & Berger, R. J. A Continuum of Slow-Wave Sleep and Shallow Torpor in the Pocket Mouse *Perognathus longimembris*. *Physiol. Zool.* **57**, 428–434 (1984).

87. Walker, J. M., Walker, L. E., Harris, D. V. & Berger, R. J. Cessation of thermoregulation during REM sleep in the pocket mouse. *Am. J. Physiol.-Regul. Integr. Comp. Physiol.* **244**, R114–R118 (1983).

88. Van Twyver, H. & Allison, T. Sleep in the opossum *Didelphis marsupialis*. *Electroencephalogr. Clin. Neurophysiol.* **29**, 181–189 (1970).

89. Rattenborg, N. C. *et al.* Migratory Sleeplessness in the White-Crowned Sparrow (*Zonotrichia leucophrys gambelii*). *PLOS Biol.* **2**, e212 (2004).

90. Rattenborg, N. C. *et al.* Evidence that birds sleep in mid-flight. *Nat. Commun.* **7**, 12468 (2016).

91. Mukhametov, L. M. Sleep in Marine Mammals. in *Experimental Brain Research Supplementum* 227–238 (Springer Berlin Heidelberg, Berlin, 1984). doi:10.1007/978-3-642-69554-4_17.

92. Lyamin, O. I. *et al.* Fur Seals Suppress REM Sleep for Very Long Periods without Subsequent Rebound. *Curr. Biol.* **28**, 2000-2005.e2 (2018).

93. Kendall-Bar, J. M. *et al.* Brain activity of diving seals reveals short sleep cycles at depth. *Science* **380**, 260–265 (2023).

94. Tinbergen, N. On aims and methods of Ethology. *Anim. Biol.* **55**, 297–321 (2005).

95. Mayr, E. Cause and Effect in Biology: Kinds of causes, predictability, and teleology are viewed by a practicing biologist. *Science* **134**, 1501–1506 (1961).

96. Bateson, P. & Laland, K. N. Tinbergen's four questions: an appreciation and an update. *Trends Ecol. Evol.* **28**, 712–718 (2013).

97. Nesse, R. M. Tinbergen's four questions, organized: a response to Bateson and Laland. *Trends Ecol. Evol.* **28**, 681–682 (2013).

98. Meddis, R. On the function of sleep. *Anim. Behav.* **23**, 676–691 (1975).

99. Siegel, J. M. Clues to the functions of mammalian sleep. *Nature* **437**, 1264–1271 (2005).

100. Horne, J. A. *Why We Sleep: The Functions of Sleep in Humans and Other Mammals*. (Oxford University Press, Oxford, 1988).

101. Rial, R. V. *et al.* The trivial function of sleep. *Sleep Med. Rev.* **11**, 311–325 (2007).

102. Joiner, W. J. Unraveling the Evolutionary Determinants of Sleep. *Curr. Biol.* **26**, R1073–R1087 (2016).

103. Duboué, E. R., Keene, A. C. & Borowsky, R. L. Evolutionary Convergence on Sleep Loss in Cavefish Populations. *Curr. Biol.* **21**, 671–676 (2011).

104. Schmidt, M. H., Swang, T. W., Hamilton, I. M. & Best, J. A. State-dependent metabolic partitioning and energy conservation: A theoretical framework for understanding the function of sleep. *PLOS ONE* **12**, e0185746 (2017).

105. Häfker, N. S. *et al.* Circadian Clock Involvement in Zooplankton Diel Vertical Migration. *Curr. Biol.* **27**, 2194-2201.e3 (2017).

106. Davis-Walton, J. & Sherman, P. W. Sleep arrhythmia in the eusocial naked mole-rat. *Naturwissenschaften* **81**, 272–275 (1994).
107. Rattenborg, N. C. *et al.* Sleep research goes wild: new methods and approaches to investigate the ecology, evolution and functions of sleep. *Philos. Trans. R. Soc. B Biol. Sci.* **372**, 20160251 (2017).
108. McNamara, P. *et al.* The Phylogeny of Sleep Database: A New Resource for Sleep Scientists. *Open Sleep J.* **1**, 11–14 (2007).
109. Voirin, B. *et al.* Ecology and Neurophysiology of Sleep in Two Wild Sloth Species. *Sleep* **37**, 753–761 (2014).
110. Davimes, J. G. *et al.* Seasonal variations in sleep of free-ranging Arabian oryx (*Oryx leucoryx*) under natural hyperarid conditions. *Sleep* **41**, zsy038 (2018).
111. Rattenborg, N. C. *et al.* Sleeping outside the box: electroencephalographic measures of sleep in sloths inhabiting a rainforest. *Biol. Lett.* **4**, 402–405 (2008).
112. Bódizs, R. Theories on the functions of sleep. in *Sleep Medicine Textbook* (European Sleep Research Society, Regensburg, 2021).
113. Gould, S. J. & Vrba, E. S. Exaptation—a Missing Term in the Science of Form. *Paleobiology* **8**, 4–15 (1982).
114. Rasch, B. & Born, J. About Sleep's Role in Memory. *Physiol. Rev.* **93**, 681–766 (2013).
115. Besedovsky, L., Lange, T. & Haack, M. The Sleep-Immune Crosstalk in Health and Disease. *Physiol. Rev.* **99**, 1325–1380 (2019).
116. Williams, G. C. *Adaptation and Natural Selection*. (Princeton University Press, Princeton, NJ, 1966).
117. Revonsuo, A. The reinterpretation of dreams: An evolutionary hypothesis of the function of dreaming. in *Sleep and Dreaming* 85–110 (Cambridge University Press, Cambridge, 2001). doi:10.1017/cbo9780511615511.007.
118. Walker, M. P. & van der Helm, E. Overnight therapy? The role of sleep in emotional brain processing. *Psychol. Bull.* **135**, 731–748 (2009).

119. Wamsley, E. J. & Stickgold, R. Memory, Sleep, and Dreaming: Experiencing Consolidation. *Sleep Med. Clin.* **6**, 97–108 (2011).
120. Hoel, E. The overfitted brain: Dreams evolved to assist generalization. *Patterns* **2**, 100244 (2021).
121. Flanagan, O. Deconstructing Dreams: The Spandrels of Sleep. in *Toward a Science of Consciousness* 63–84 (The MIT Press, Cambridge, MA, 1996). doi:10.7551/mitpress/6860.003.0007.
122. Flanagan, O. *Dreaming Souls: Sleep, Dreams, and the Evolution of the Conscious Mind*. (Oxford University Press, New York, 2000).
123. Krueger, J. M. *et al.* Sleep as a fundamental property of neuronal assemblies. *Nat. Rev. Neurosci.* **9**, 910–919 (2008).
124. Frank, M. G. & Heller, H. C. The ontogeny of mammalian sleep: a reappraisal of alternative hypotheses. *J. Sleep Res.* **12**, 25–34 (2003).
125. Roffwarg, H. P., Muzio, J. N. & Dement, W. C. Ontogenetic Development of the Human Sleep-Dream Cycle. *Science* **152**, 604–619 (1966).
126. Joyce, M. *et al.* Divergent evolution of sleep in *Drosophila* species. *Nat. Commun.* **15**, 5091 (2024).
127. Rattenborg, N. C., Amlaner, C. J. & Lima, S. L. Behavioral, neurophysiological and evolutionary perspectives on unihemispheric sleep. *Neurosci. Biobehav. Rev.* **24**, 817–842 (2000).
128. Lyamin, O. I., Manger, P. R., Ridgway, S. H., Mukhametov, L. M. & Siegel, J. M. Cetacean sleep: An unusual form of mammalian sleep. *Neurosci. Biobehav. Rev.* **32**, 1451–1484 (2008).
129. Libourel, P.-A. *et al.* Nesting chinstrap penguins accrue large quantities of sleep through seconds-long microsleeps. *Science* **382**, 1026–1031 (2023).
130. Vyazovskiy, V. V. *et al.* Local sleep in awake rats. *Nature* **472**, 443–447 (2011).
131. Poudel, G. R., Innes, C. R. H., Bones, P. J., Watts, R. & Jones, R. D. Losing the struggle to stay awake: Divergent thalamic and cortical activity during microsleeps: Neural Activity During Microsleeps. *Hum. Brain Mapp.* **35**, 257–269 (2014).

132. Borbély, A. A. A two process model of sleep regulation. *Hum. Neurobiol.* **1**, 195–204 (1982).
133. Daan, S., Beersma, D. G. & Borbély, A. A. Timing of human sleep: recovery process gated by a circadian pacemaker. *Am. J. Physiol.* **246**, R161-183 (1984).
134. Achermann, P. Mathematical models of sleep regulation. *Front. Biosci.* **8**, s683–s693 (2003).
135. Franken, P., Chollet, D. & Tafti, M. The Homeostatic Regulation of Sleep Need Is under Genetic Control. *J. Neurosci.* **21**, 2610–2621 (2001).
136. Rechtschaffen, A., Bergmann, B. M., Gilliland, M. A. & Bauer, K. Effects of Method, Duration, and Sleep Stage on Rebounds from Sleep Deprivation in the Rat. *Sleep* **22**, 11–31 (1999).
137. Kim, Y., Laposky, A. D., Bergmann, B. M. & Turek, F. W. Repeated sleep restriction in rats leads to homeostatic and allostatic responses during recovery sleep. *Proc. Natl. Acad. Sci.* **104**, 10697–10702 (2007).
138. Gulevich, G., Dement, W. & Johnson, L. Psychiatric and EEG Observations on a Case of Prolonged (264 Hours) Wakefulness. *Arch. Gen. Psychiatry* **15**, 29–35 (1966).
139. Loftus, J. C., Harel, R., Núñez, C. L. & Crofoot, M. C. Ecological and social pressures interfere with homeostatic sleep regulation in the wild. *eLife* **11**, e73695 (2022).
140. Borbély, A. A., Daan, S., Wirz-Justice, A. & Deboer, T. The two-process model of sleep regulation: a reappraisal. *J. Sleep Res.* **25**, 131–143 (2016).
141. Porkka-Heiskanen, T. *et al.* Adenosine: A Mediator of the Sleep-Inducing Effects of Prolonged Wakefulness. *Science* **276**, 1265–1268 (1997).
142. Bjorness, T. E. & Greene, R. W. Adenosine and Sleep. *Curr. Neuropharmacol.* **7**, 238–245 (2009).
143. Nesse, R. M. The Smoke Detector Principle. *Ann. N. Y. Acad. Sci.* **935**, 75–85 (2001).
144. Betley, J. N. *et al.* Neurons for hunger and thirst transmit a negative-valence teaching signal. *Nature* **521**, 180–185 (2015).
145. Lansing, R. W., Gracely, R. H. & Banzett, R. B. The multiple dimensions of dyspnea: Review and hypotheses. *Respir. Physiol. Neurobiol.* **167**, 53–60 (2009).

146. Axelsson, J. *et al.* Sleepiness as motivation: a potential mechanism for how sleep deprivation affects behavior. *Sleep* **43**, zsz291 (2020).
147. Nir, Y. *et al.* Selective neuronal lapses precede human cognitive lapses following sleep deprivation. *Nat. Med.* **23**, 1474–1480 (2017).
148. Kirszenblat, L. & van Swinderen, B. The Yin and Yang of Sleep and Attention. *Trends Neurosci.* **38**, 776–786 (2015).
149. Andrillon, T. *et al.* Does the Mind Wander When the Brain Takes a Break? Local Sleep in Wakefulness, Attentional Lapses and Mind-Wandering. *Front. Neurosci.* **13**, 949 (2019).
150. Bonnet, M. H. Effect of Sleep Disruption on Sleep, Performance, and Mood. *Sleep* **8**, 11–19 (1985).
151. Martin, S. E., Wraith, P. K., Deary, I. J. & Douglas, N. J. The Effect of Nonvisible Sleep Fragmentation on Daytime Function. *Am. J. Respir. Crit. Care Med.* **155**, 1596–1601 (1997).
152. Bonnet, M. H. & Arand, D. L. Clinical effects of sleep fragmentation versus sleep deprivation. *Sleep Med. Rev.* **7**, 297–310 (2003).
153. Doran, S. M., Van Dongen, H. P. A. & Dinges, D. F. Sustained attention performance during sleep deprivation: Evidence of state instability. *Arch. Ital. Biol.* **139**, 253–267 (2001).
154. Lim, J. & Dinges, D. F. A meta-analysis of the impact of short-term sleep deprivation on cognitive variables. *Psychol. Bull.* **136**, 375–389 (2010).
155. Van Dongen, H. P. A., Maislin, G., Mullington, J. M. & Dinges, D. F. The Cumulative Cost of Additional Wakefulness: Dose-Response Effects on Neurobehavioral Functions and Sleep Physiology From Chronic Sleep Restriction and Total Sleep Deprivation. *Sleep* **26**, 117–126 (2003).
156. Belenky, G. *et al.* Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: a sleep dose-response study. *J. Sleep Res.* **12**, 1–12 (2003).
157. Cappuccio, F. P., D'Elia, L., Strazzullo, P. & Miller, M. A. Sleep Duration and All-Cause Mortality: A Systematic Review and Meta-Analysis of Prospective Studies. *Sleep* **33**, 585–592 (2010).

158. Cappuccio, F. P., Cooper, D., D'Elia, L., Strazzullo, P. & Miller, M. A. Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. *Eur. Heart J.* **32**, 1484–1492 (2011).

159. Sabia, S. *et al.* Association of sleep duration in middle and old age with incidence of dementia. *Nat. Commun.* **12**, 2289 (2021).

160. Windred, D. P. *et al.* Sleep regularity is a stronger predictor of mortality risk than sleep duration: A prospective cohort study. *Sleep* **47**, zsad253 (2024).

161. Chaput, J.-P. *et al.* Sleep regularity and major adverse cardiovascular events: a device-based prospective study in 72 269 UK adults. *J. Epidemiol. Community Health* **79**, 257–264 (2025).

162. Linton, S. J. *et al.* The effect of the work environment on future sleep disturbances: a systematic review. *Sleep Med. Rev.* **23**, 10–19 (2015).

163. Kivimäki, M. *et al.* Job strain as a risk factor for coronary heart disease: a collaborative meta-analysis of individual participant data. *The Lancet* **380**, 1491–1497 (2012).

164. Yusuf, S. *et al.* Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *The Lancet* **364**, 937 –952 (2004).

165. Rosengren, A. *et al.* Association of psychosocial risk factors with risk of acute myocardial infarction in 11 119 cases and 13 648 controls from 52 countries (the INTERHEART study): case-control study. *The Lancet* **364**, 953–962 (2004).

166. Garde, A. H., Hansen, Å. M., Holtermann, A., Gyntelberg, F. & Suadicani, P. Sleep duration and ischemic heart disease and all-cause mortality: Prospective cohort study on effects of tranquilizers/hypnotics and perceived stress. *Scand. J. Work. Environ. Health* **39**, 550–558 (2013).

167. Chandola, T., Ferrie, J. E., Perski, A., Akbaraly, T. & Marmot, M. G. The Effect of Short Sleep Duration on Coronary Heart Disease Risk is Greatest Among Those with Sleep Disturbance: A Prospective Study from the Whitehall II Cohort. *Sleep* **33**, 739–744 (2010).

168. Magnusson Hanson, L. L. *et al.* Job insecurity and risk of coronary heart disease: Mediation analyses of health behaviors, sleep problems, physiological and psychological factors. *Psychoneuroendocrinology* **118**, 104706 (2020).
169. Tamakoshi, A. & Ohno, Y. Self-Reported Sleep Duration as a Predictor of All-Cause Mortality: Results from the JACC Study, Japan. *Sleep* **27**, 51–54 (2004).
170. Heslop, P., Smith, G. D., Metcalfe, C., Macleod, J. & Hart, C. Sleep duration and mortality: the effect of short or long sleep duration on cardiovascular and all-cause mortality in working men and women. *Sleep Med.* **3**, 305–314 (2002).
171. Dashti, H. S. *et al.* Genome-wide association study identifies genetic loci for self-reported habitual sleep duration supported by accelerometer-derived estimates. *Nat. Commun.* **10**, 1100 (2019).
172. García-Marín, L. M., Campos, A. I., Martin, N. G., Cuéllar-Partida, G. & Rentería, M. E. Inference of causal relationships between sleep-related traits and 1,527 phenotypes using genetic data. *Sleep* **44**, zsaa154 (2021).
173. Morin, C. M., Vallières, A. & Ivers, H. Dysfunctional Beliefs and Attitudes about Sleep (DBAS): Validation of a Brief Version (DBAS-16). *Sleep* **30**, 1547–1554 (2007).
174. Trauer, J. M., Qian, M. Y., Doyle, J. S., Rajaratnam, S. M. W. & Cunnington, D. Cognitive Behavioral Therapy for Chronic Insomnia. *Ann. Intern. Med.* **163**, 191–204 (2015).
175. van Straten, A. *et al.* Cognitive and behavioral therapies in the treatment of insomnia: A meta-analysis. *Sleep Med. Rev.* **38**, 3–16 (2018).
176. Maurer, L. F., Espie, C. A., Omlin, X., Emsley, R. & Kyle, S. D. The effect of sleep restriction therapy for insomnia on sleep pressure and arousal: a randomized controlled mechanistic trial. *Sleep* **45**, zsab223 (2022).
177. Draganich, C. & Erdal, K. Placebo sleep affects cognitive functioning. *J. Exp. Psychol. Learn. Mem. Cogn.* **40**, 857–864 (2014).
178. Gavriloff, D. *et al.* Sham sleep feedback delivered via actigraphy biases daytime symptom reports in people with insomnia: Implications for insomnia disorder and wearable devices. *J. Sleep Res.* **27**, e12726 (2018).

179. Harvey, A. G. A cognitive model of insomnia. *Behav. Res. Ther.* **40**, 869–893 (2002).
180. Rechtschaffen, A., Hauri, P. & Zeitlin, M. Auditory Awakening Thresholds in Rem and Nrem Sleep Stages. *Percept. Mot. Skills* **22**, 927–942 (1966).
181. Neckelmann, D. & Ursin, R. Sleep Stages and EEG Power Spectrum in Relation to Acoustical Stimulus Arousal Threshold in the Rat. *Sleep* **16**, 467–477 (1993).
182. Rial, R. V. *et al.* The Birth of the Mammalian Sleep. *Biology* **11**, 734 (2022).
183. Oswald, I., Taylor, A. M. & Treisman, M. Discriminative responses to stimulation during human sleep. *Brain* **83**, 440–453 (1960).
184. Portas, C. M. *et al.* Auditory Processing across the Sleep-Wake Cycle: Simultaneous EEG and fMRI Monitoring in Humans. *Neuron* **28**, 991–999 (2000).
185. French, A. S., Geissmann, Q., Beckwith, E. J. & Gilestro, G. F. Sensory processing during sleep in *Drosophila melanogaster. Nature* **598**, 479–482 (2021).
186. Coenen, A. Sensory gating and gaining in sleep: the balance between the protection of sleep and the safeness of life (a review). *J. Sleep Res.* **33**, e14152 (2024).
187. Rattenborg, N. C., Lima, S. L. & Amlaner, C. J. Half-awake to the risk of predation. *Nature* **397**, 397–398 (1999).
188. Aulsebrook, A. E., Jones, T. M., Rattenborg, N. C., Roth, T. C. & Lesku, J. A. Sleep Ecophysiology: Integrating Neuroscience and Ecology. *Trends Ecol. Evol.* **31**, 590–599 (2016).
189. Saper, C. B., Fuller, P. M., Pedersen, N. P., Lu, J. & Scammell, T. E. Sleep State Switching. *Neuron* **68**, 1023–1042 (2010).
190. Scammell, T. E., Arrigoni, E. & Lipton, J. O. Neural Circuitry of Wakefulness and Sleep. *Neuron* **93**, 747–765 (2017).
191. Alphen, B. van, Yap, M. H. W., Kirszenblat, L., Kottler, B. & Swinderen, B. van. A Dynamic Deep Sleep Stage in *Drosophila*. *J. Neurosci.* **33**, 6917–6927 (2013).
192. Jung, C. M. *et al.* Energy expenditure during sleep, sleep deprivation and sleep following sleep deprivation in adult humans. *J. Physiol.* **589**, 235–244 (2011).

193. Libourel, P.-A. *et al.* Partial homologies between sleep states in lizards, mammals, and birds suggest a complex evolution of sleep states in amniotes. *PLOS Biol.* **16**, e2005982 (2018).
194. McGaugh, S. E., Passow, C. N., Jaggard, J. B., Stahl, B. A. & Keene, A. C. Unique transcriptional signatures of sleep loss across independently evolved cavefish populations. *J. Exp. Zoolog. B Mol. Dev. Evol.* **334**, 497–510 (2020).
195. Funato, H. *et al.* Forward-genetics analysis of sleep in randomly mutagenized mice. *Nature* **539**, 378–383 (2016).
196. Grubbs, J. J., Lopes, L. E., Linden, A. M. van der & Raizen, D. M. A salt-induced kinase is required for the metabolic regulation of sleep. *PLOS Biol.* **18**, e3000220 (2020).
197. Wu, M. N. *et al.* The Effects of Caffeine on Sleep in *Drosophila* Require PKA Activity, But Not the Adenosine Receptor. *J. Neurosci.* **29**, 11029–11037 (2009).
198. Andretic, R., Kim, Y.-C., Jones, F. S., Han, K.-A. & Greenspan, R. J. *Drosophila* D1 dopamine receptor mediates caffeine-induced arousal. *Proc. Natl. Acad. Sci.* **105**, 20392–20397 (2008).
199. Vyssotski, A. L. *et al.* EEG Responses to Visual Landmarks in Flying Pigeons. *Curr. Biol.* **19**, 1159–1166 (2009).

XIII. SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Appendix S1. Data and code reproducing Figure 2: the metastability model script, the Viterbi-decoded bout durations, the fitted hidden Markov models and the hourly sleep-state summaries, with a README describing each file.