

Biological proper time and entropy-cost invariance in cardiac and respiratory lifespan scaling

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Abstract

Warm-blooded vertebrates accumulate approximately conserved numbers of physiological cycles over a natural lifetime: of order 10^9 heartbeats and 10^8 – 3×10^8 breaths. These regularities are not exact constants, but their persistence across orders-of-magnitude variation in body mass, metabolic power, physiological frequency, and lifespan suggests that biological time is not measured by chronological duration alone. We develop the Principle of Biological Time Equivalence (PBTE), a thermodynamic framework in which lifetime cycle count is determined by the ratio between total lifetime entropy production and the entropy cost of one physiological cycle. Starting from the open-system entropy balance $\dot{S} = \dot{e}_p - \dot{h}_d$, we define the entropy cost per cycle as $\sigma_0 = d\Sigma/dN$, where $d\Sigma$ is the entropy produced as the physiological clock advances by dN cycles. For an adult homeostatic regime, this gives the cycle-count relation $N_\star = \Sigma/\langle\sigma_0\rangle$, with $\Sigma = \int_0^L \dot{e}_p(t) dt$, where N_\star is the lifetime cycle count, Σ is total lifetime entropy production, and $\langle\sigma_0\rangle$ is the lifetime-averaged entropy cost per cycle. In the homeostatic limit, $\dot{e}_p \simeq P/T$, so direct measurement of metabolic power P , body temperature T , and physiological frequency f gives $\sigma_0 \simeq P/(Tf)$. PBTE converts the empirical lifetime-cycle invariants into entropy-cost invariants. Under Kleiber metabolic scaling and quarter-power physiological-frequency scaling, the mass-specific entropy cost satisfies $\bar{\sigma}_0 = P/(TfM) \propto M^{3/4+1/4-1} = M^0$, providing a thermodynamic interpretation of allometric mass cancellation. The framework also treats clade deviations as structured changes in effective cycle budget rather than residual scatter. We write $N_{\star,C} = N_{\star,0}\Phi_C$, where the clade multiplier Φ_C decomposes contributions from duty cycle, temperature-dependent kinetics, mitochondrial and antioxidant efficiency, and ecological hazard. Once Φ_C is determined, the same relation predicts lifespan from physiological frequency through $L_{\text{pred}} = N_{\star,0}\Phi_C/(fT)$. We apply this framework to cardiac and respiratory clocks. The cardiac analysis, on a 230-species dataset, yields a reference lifetime count of order 10^9 beats and a nearly mass-independent entropy cost per beat per unit mass. The respiratory analysis, based on the 65-species subset with reliable resting breath rates, yields a coarser lifetime breath count of order 10^8 – 3×10^8 and provides an independent test of the same thermodynamic construction. As the decisive non-circular test, we recompute the respiratory entropy cost from *measured* species-level basal metabolic rates (He et al. 2023) rather than imposed Kleiber scaling. On this independent dataset ($n = 29$ mammals with both measured BMR and measured resting breath rate) the mass cancellation does *not* survive: the mass-specific respiratory entropy cost rises with body mass (fitted slope $\simeq +0.21$, not statistically resolved, $p \simeq 0.11$; coefficient of variation $\sim 100\%$). The positive, dispersed trend is driven by aquatic mammals, whose low resting breath rates relative to metabolic rate sharply inflate the entropy cost per breath at large body size. The respiratory clock therefore fails the non-circular cancellation test that the cardiac clock passes—an informative asymmetry rather than a confirmation, and the

central empirical finding of the respiratory analysis. Building on the validated cardiac invariant, we promote biological proper time to an internal-age coordinate: the entropy-normalized biological age $A_{\text{PBTE}}(t) = \Sigma(t)/\Sigma_{\text{ref}}$ measures the fraction of a reference entropy-cycle budget already consumed, and its rate $dA_{\text{PBTE}}/dt = \dot{e}_p/\Sigma_{\text{ref}}$ defines an aging velocity set by entropy production. This coordinate organizes aging and longevity into three thermodynamic mechanism classes—time dilation (caloric restriction, torpor, cetacean bradycardia), entropy-cost reduction and budget expansion (avian and primate maintenance efficiency), and hypertemporal pathologies (inflammation, metabolic syndrome, neurodegeneration, cancer)—all visible in the clade structure of this dataset. The cardiac clock supplies a non-circular reference cost for this coordinate while the respiratory clock does not, so the empirical asymmetry directly governs which rhythm can serve as the clock for aging. The resulting theory identifies biological proper time as the accumulated physiological cycle count and makes PBTE experimentally falsifiable: its decisive test is simultaneous calorimetric, cardiac, respiratory, temperature, and body-mass measurement across species to determine whether $\sigma_0 = P/(Tf)$ is truly mass-independent within defined physiological regimes.

Keywords: non-equilibrium thermodynamics; entropy production; biological proper time; metabolic scaling; lifespan invariant; cardiac allometry; respiratory allometry; clade multiplier; Gompertz mortality.

1 Introduction

Time has always stood at the boundary between science, philosophy, and lived experience. We call it a blessing when it is abundant, a curse when it disappears too quickly, and a sacred gift when it is shared. Civilizations have measured it by the motion of stars, the turning of seasons, the growth of trees, the rise and fall of tides, and the pulse within the body; yet its deepest meaning remains elusive. Modern physics showed that time is not an absolute background: the time measured by an observer depends on motion and gravity [1]. A related, but biologically distinct, idea appears in living systems. In inert matter, velocity and gravitation shape physical proper time; in living matter, metabolism, organization, and entropy production shape biological time. A mayfly, a shrew, an elephant, and a human do not merely pass through the same external clock for different durations; each advances through an internal rhythm set by heartbeats, breaths, molecular turnover, repair, and the energetic cost of maintaining order. The analogy with relativity is therefore structural, not literal: biological proper time is not defined by spacetime geometry, but by the dynamics of open dissipative systems. Every physiological tick transforms energy and produces entropy; life may proceed faster or slower in chronological years, but it advances by spending a finite budget of internal cycles.

Among the most striking regularities in comparative physiology is a temporal invariant that is both simple and physically suggestive. A pygmy shrew and an African elephant inhabit radically different chronological worlds: the shrew has a body mass of only a few grams, may sustain a resting heart rate of several hundred beats per minute, and lives only a few years, whereas the elephant has a mass of several thousand kilograms, beats only tens of times per minute, and may live for many decades. Yet this disparity is greatly reduced when physiological rate is multiplied by natural lifespan: both animals accumulate of order 10^9 cardiac cycles over a lifetime [2, 3, 4, 5, 6]. A parallel regularity occurs in respiration, where resting breath rate multiplied by lifespan gives a lifetime breath count of order 10^8 – 3×10^8 in warm-blooded vertebrates [7, 8]. These two empirical clocks can be summarized compactly as

$$N_H = f_H L T \simeq 10^9, \quad N_R = f_R L T \simeq 3 \times 10^8, \quad (1)$$

where N_H and N_R are the lifetime numbers of heartbeats and breaths, f_H is the resting heart rate, f_R is the resting breath rate, L is lifespan in years, and $\mathcal{T} = 525,960 \text{ min yr}^{-1}$ converts years into minutes when rates are measured per minute. The numerical values in Eq. (1) are not exact constants. Their significance is that they remain concentrated despite orders-of-magnitude variation in body mass, metabolic power, physiological rate, and lifespan. Moreover, the residual deviations are structured: primates, bats, birds, and cetaceans show reproducible offsets from the reference mammalian pattern, indicating that lifetime cycle counts are shaped by physiology and clade history rather than by body mass alone.

To explain this near constancy, previous studies have emphasized allometric cancellation. Small animals live at a faster physiological pace, whereas large animals live more slowly: heart rate decreases with body mass, while lifespan increases in the opposite direction. Their product therefore becomes nearly mass independent. This argument explains why the leading body-mass dependence is weak, but it does not identify the physical quantity whose lifetime accumulation determines the number of physiological cycles. Nor does it explain why clades are displaced systematically from the same reference line. Classical rate-of-living arguments face a related limitation [9, 10]: they connect lifespan to metabolic expenditure, but they do not specify the entropy cost of an individual cardiac or respiratory cycle. Fixed-exponent scaling arguments are further limited by the dependence of metabolic exponents on taxon, metabolic level, and physiological state [11]. Thus allometry explains the cancellation, but not the thermodynamic content of the invariant.

Our proposed Principle of Biological Time Equivalence (PBTE) addresses this gap by identifying irreversible entropy production as the thermodynamic measure of biological progression. A living organism is an open dissipative system: it continuously produces entropy through metabolism, molecular turnover, and physiological activity, while exporting entropy to maintain internal organization. In the homeostatic regime relevant to resting comparative physiology, entropy production is approximately balanced by entropy export. The instantaneous entropy-production rate may therefore be estimated, to leading order, by the metabolic power divided by body temperature. As derived in the next section, PBTE interprets the entropy cost of one physiological cycle as the dissipative price paid when the biological clock advances by one internal tick. This leads to the cycle-count identity

$$N_\star = \frac{\Sigma}{\langle \sigma_0 \rangle}, \quad \Sigma = \int_0^L \dot{e}_p(t) dt, \quad (2)$$

where N_\star denotes the lifetime physiological cycle count, Σ is the total entropy produced over the lifespan L , and $\langle \sigma_0 \rangle$ is the lifetime-averaged entropy cost per cycle. Thus the proposed invariant is neither chronological lifespan alone nor total lifetime energy expenditure alone. It is the ratio between the total dissipative expenditure of the organism and the entropy required to advance its physiological clock by one cycle.

As shown in the next section, this formulation gives allometric cancellation a thermodynamic meaning. Metabolic power, physiological frequency, and body mass scale together. Their scalings partly compensate one another. As a result, the entropy cost per physiological cycle, normalized by body mass, becomes nearly independent of body size. The lifetime-cycle invariant is therefore recast as an entropy-cost invariant. Small and large organisms dissipate entropy at different rates in chronological time. Yet the cost assigned to one unit of biological time remains approximately conserved. PBTE therefore identifies the physical quantity that allometry cancels. The detailed derivation and empirical tests are developed in the sections that follow.

At this point, we emphasize that the framework becomes most informative when the invariant is not exact. In PBTE, clade deviations are not treated as residual scatter around a single allometric line, but as systematic changes in the effective cycle budget. We write the lifetime cycle count in

clade C as

$$N_{*,C} = N_{*,0} \Phi_C, \quad (3)$$

where $N_{*,0}$ is the reference cycle budget and Φ_C is a dimensionless clade multiplier. The multiplier decomposes the departure from the baseline into physiological contributions,

$$\Phi_C = \Phi_{\text{duty}} \Phi_{\text{thermal}} \Phi_{\text{mito+oxid}} \Phi_{\text{haz}}. \quad (4)$$

Here Φ_{duty} accounts for the fraction of life spent in suppressed or accelerated physiological states, Φ_{thermal} accounts for temperature-dependent changes in metabolic and damage kinetics, $\Phi_{\text{mito+oxid}}$ represents mitochondrial efficiency and antioxidant protection, and Φ_{haz} represents the extent to which ecological mortality permits the intrinsic thermodynamic budget to be realized. Once Φ_C is determined, Eq. (3) can be inverted to predict lifespan from a measured physiological frequency,

$$L_{\text{pred}} = \frac{N_{*,0} \Phi_C}{f \mathcal{T}}, \quad (5)$$

where $\mathcal{T} = 525,960 \text{ min yr}^{-1}$ when f is measured in cycles per minute and L_{pred} is expressed in years. Thus Φ_C is not a post hoc correction; it is the predictive component of the theory. After the clade multiplier is derived, lifespan can be estimated from physiological frequency and compared directly with observation.

The aim of this paper is to develop PBTE as a falsifiable thermodynamic parametrisation of biological time and to test its predictive content using two physiological clocks. First, we derive σ_0 from the entropy balance. We then show how allometric mass cancellation leads to an approximately invariant entropy cost per cycle per unit mass. Second, we derive the clade multiplier Φ_C . This converts the baseline invariant into clade-specific lifespan predictions. Third, we apply the framework to cardiac and respiratory cycle counts. The purpose of using two clocks is not to determine which rhythm is more fundamental. Rather, it is to test whether distinct physiological cycles are governed by the same thermodynamic structure. Fourth, we promote biological proper time to a normalized internal-age coordinate $A_{\text{PBTE}}(t)$ and show how the validated cardiac entropy-cost invariant supplies a non-circular clock for aging and longevity, organizing interventions and pathologies into time-dilation, entropy-cost, and hypertemporal mechanism classes, whereas the respiratory clock—which fails the same invariance test—does not. The theory is directly testable. Its decisive test requires simultaneous measurements of metabolic power P , body temperature T , physiological frequency f , and body mass M across species. These measurements allow $\sigma_0 = P/(Tf)$ to be determined directly, rather than inferred from allometric scaling. We believe that PBTE provides a thermodynamically motivated scaling framework for biological time. Lifespan is then constrained by the number of dissipative physiological cycles that an organism can realize within its entropy budget.

Furthermore, in this work we show that the approximate lifetime-cycle invariants observed in vertebrates are not merely consequences of allometric scaling. Rather, they reflect an underlying thermodynamic organization of biological time. We further show that systematic clade-dependent departures from the baseline invariant can be interpreted within a unified entropy-budget framework. In this view, primates achieve exceptional longevity through physiological strategies that reduce the effective entropy burden of each biological cycle. This allows a larger number of cycles to be accumulated over a lifetime. Bats extend lifespan through prolonged periods of metabolic suppression, during which biological time advances more slowly. Birds, despite high metabolic rates and elevated body temperatures, achieve extended longevity through enhanced cellular maintenance, mitochondrial performance, and resistance to oxidative damage. Cetaceans follow another strategy. Their lifespan extension is associated with profound cardiovascular slowing during diving

and altered physiological pacing over the life course. These mechanisms are biologically distinct. Yet we show that they can be understood as different realizations of the same thermodynamic principle. The resulting framework explains why different clades exhibit systematic differences in lifetime cycle counts. It also provides a quantitative basis for predicting lifespan from physiological and thermodynamic characteristics.

2 Thermodynamic Foundation

The three logical levels of PBTE

The framework developed below is best read as three logically distinct statements of decreasing generality. Conflating them is the most common source of misunderstanding, so we separate them explicitly here and maintain the distinction throughout.

Level 1 — exact identity (no physical assumption). Given the definitions of cycle count $N(t) = \int_0^t f dt'$ and entropy cost per cycle $\sigma_0 = \dot{e}_p/f = d\Sigma/dN$, the relation

$$N_\star = \frac{\Sigma}{\langle \sigma_0 \rangle}, \quad \Sigma = \int_0^L \dot{e}_p(t) dt,$$

is an exact algebraic identity. It introduces no biology and cannot be falsified; it merely names the lifetime cycle count as a ratio of two defined quantities.

Level 2 — thermodynamic closure (an approximation). In an adult homeostatic regime, entropy export tracks production, so that $\dot{e}_p \simeq P/T$ and the cost per cycle is estimated operationally by

$$\langle \sigma_0 \rangle \simeq \frac{P}{Tf}.$$

This is a constitutive approximation, not an identity. It is testable in principle by measuring P , T , and f on the same individuals.

Level 3 — empirical claim (the falsifiable content). The substantive, refutable assertion of PBTE is that the mass-specific cost

$$\bar{\sigma}_0 = \frac{\sigma_0}{M} \simeq \frac{P}{TfM}$$

is approximately invariant *within defined physiological regimes*. This is an empirical regularity, expected to hold only approximately, and it is the level at which the framework can fail. The clade multipliers of Section 3 are the structured departures from this approximate invariance.

A living organism is an open nonequilibrium system [12, 13]. It continuously transforms chemical free energy into mechanical work, ionic gradients, biosynthesis, repair, signalling, and heat, while exporting entropy in order to preserve internal organization. Let $S(t)$ denote the coarse-grained internal entropy at chronological time t . The open-system entropy balance is

$$\dot{S}(t) = \dot{e}_p(t) - \dot{h}_d(t), \tag{6}$$

where $\dot{e}_p(t) \geq 0$ is the irreversible entropy production rate and $\dot{h}_d(t) \geq 0$ is the entropy export rate. To leading thermodynamic order, entropy export is estimated from metabolic power and absolute temperature as

$$\dot{h}_d(t) \simeq \frac{P(t)}{T(t)}, \tag{7}$$

where $P(t)$ is metabolic power and $T(t)$ is body temperature.

Equation (6) contains both the homeostatic regime used for the lifetime cycle-count relation and the non-steady regime relevant to aging. In physiological homeostasis,

$$\dot{S}(t) \simeq 0, \quad \dot{e}_p(t) \simeq \dot{h}_d(t) \simeq \frac{P(t)}{T(t)}. \quad (8)$$

If entropy export becomes less efficient during aging, then $\dot{h}_d(t) < \dot{e}_p(t)$ and internal entropy accumulates according to

$$S(t) = S(0) + \int_0^t [\dot{e}_p(t') - \dot{h}_d(t')] dt'. \quad (9)$$

This accumulated entropy is interpreted as a coarse-grained burden associated with loss of physiological order, impaired repair, damaged macromolecules, mitochondrial dysfunction, and weakened regulatory control.

We now introduce the physiological cycle coordinate. Let $f(t)$ denote the frequency of a recurrent physiological process, measured in s^{-1} . Its cumulative count is

$$N(t) = \int_0^t f(t') dt', \quad dN = f(t) dt. \quad (10)$$

The entropy produced over the same interval is

$$d\Sigma = \dot{e}_p(t) dt. \quad (11)$$

Eliminating dt gives

$$d\Sigma = \frac{\dot{e}_p(t)}{f(t)} dN. \quad (12)$$

Thus the instantaneous entropy cost of one physiological cycle is

$$\sigma_0(t) \equiv \frac{\dot{e}_p(t)}{f(t)} = \frac{d\Sigma}{dN}. \quad (13)$$

This quantity is not introduced as a dimensional convention; it is the entropy produced when the biological clock advances by one cycle.

Integration over lifespan gives

$$\Sigma = \int_0^L \dot{e}_p(t) dt = \int_0^{N_\star} \sigma_0(N) dN, \quad (14)$$

where Σ is the total lifetime entropy production and N_\star is the total lifetime number of cycles. Defining the lifetime-averaged entropy cost per cycle,

$$\langle \sigma_0 \rangle = \frac{1}{N_\star} \int_0^{N_\star} \sigma_0(N) dN, \quad (15)$$

one obtains

$$N_\star = \frac{\Sigma}{\langle \sigma_0 \rangle}. \quad (16)$$

Equation (16) is the basic PBTE cycle-count relation: the lifetime number of physiological cycles equals total lifetime entropy production divided by the mean entropy cost of one cycle.

The Principle of Biological Time Equivalence is the constitutive approximation that, within a defined adult homeostatic regime, the entropy cost per cycle is represented by its average value. In that regime,

$$\dot{e}_p(t) \simeq \langle \sigma_0 \rangle f(t). \tag{17}$$

Combining this closure with Eq. (8) gives the operational estimator

$$\langle \sigma_0 \rangle \simeq \frac{P}{Tf}. \tag{18}$$

The corresponding mass-specific entropy cost is

$$\bar{\sigma}_0 = \frac{\langle \sigma_0 \rangle}{M} \simeq \frac{P}{TfM}. \tag{19}$$

Under Kleiber metabolic scaling [14, 15], $P \propto M^{3/4}$, and quarter-power physiological-frequency scaling, $f \propto M^{-1/4}$, this gives

$$\bar{\sigma}_0 \propto \frac{M^{3/4}}{M^{-1/4}M} = M^0. \tag{20}$$

Thus the allometric cancellation of lifetime cycle count is recast as an approximate invariance of the entropy cost per cycle per unit mass.

Finally, the same construction defines biological proper time. Chronological time t is the external clock time, whereas biological proper time is the accumulated physiological cycle count,

$$\theta(t) = \int_0^t f(t') dt', \quad \hat{\theta}(t) = \frac{\theta(t)}{N_\star}. \tag{21}$$

The quantity $\theta(t)$ measures the advancement of the biological clock rather than the passage of chronological time. If the total lifetime cycle budget N_\star is approximately invariant across comparable organisms, then lifespan is determined primarily by the rate at which biological proper time accumulates. For an approximately constant physiological frequency,

$$N_\star \simeq fL, \tag{22}$$

where L is the chronological lifespan. Consequently,

$$L \simeq \frac{N_\star}{f}. \tag{23}$$

Thus, a lower physiological frequency implies a slower advance of biological proper time and therefore a longer chronological lifespan. In this view, longevity is associated not with slowing chronological time itself, but with slowing the rate at which the organism consumes its finite budget of physiological cycles.

Under the PBTE closure,

$$d\Sigma = \langle \sigma_0 \rangle d\theta, \quad \frac{d\Sigma}{d\theta} = \langle \sigma_0 \rangle. \tag{24}$$

Biological proper time is therefore the coordinate in which entropy accumulates uniformly. The cardiac and respiratory clocks are two realizations of the same construction:

$$N_H = \frac{\Sigma}{\langle \sigma_H \rangle}, \quad N_R = \frac{\Sigma}{\langle \sigma_R \rangle}. \tag{25}$$

The following sections apply this identity to heartbeat and breathing. In the next section, we develop the clade modulation of the lifetime cycle budget and show how physiological differences enter the PBTE framework.

3 Clade Modulation of the Lifetime Cycle Budget

As discussed above, the PBTE relation $N_{\star} = \Sigma / \langle \sigma_0 \rangle$ defines a reference lifetime cycle budget. Empirical observations, however, show that lifetime cycle counts are only approximately invariant. Systematic departures occur among major vertebrate clades. This indicates that the effective biological-time budget is modulated by physiological and ecological factors beyond the leading allometric scaling.

Within PBTE, these departures are not interpreted as residual scatter around a universal scaling law. Rather, they reflect systematic renormalizations of the effective lifetime cycle budget. We therefore write

$$N_{\star,C} = N_{\star,0} \Phi_C, \quad (26)$$

where $N_{\star,0}$ is the reference cycle budget and Φ_C is a dimensionless clade multiplier. Values $\Phi_C > 1$ correspond to clades that realize a larger effective cycle budget than the reference state, whereas $\Phi_C < 1$ corresponds to a reduced budget.

The multiplier summarizes the dominant mechanisms that modify either the lifetime entropy budget Σ or the entropy cost per cycle $\langle \sigma_0 \rangle$. Therefore, to leading order, we write

$$\Phi_C = \Phi_{\text{duty}} \Phi_{\text{thermal}} \Phi_{\text{mito+oxid}} \Phi_{\text{haz}}. \quad (27)$$

Here Φ_{duty} captures changes in biological-clock accumulation caused by intermittent physiological states such as torpor, hibernation, dormancy, or diving bradycardia. The factor Φ_{thermal} represents temperature-dependent changes in metabolic and damage kinetics. The factor $\Phi_{\text{mito+oxid}}$ accounts for mitochondrial efficiency, oxidative-stress resistance, and cellular maintenance mechanisms that alter the entropy generated per physiological cycle. Finally, Φ_{haz} represents extrinsic ecological mortality, which determines how fully the intrinsic thermodynamic budget can be realized in practice.

The resulting lifetime cycle count becomes

$$N_{\star,C} = N_{\star,0} \Phi_{\text{duty}} \Phi_{\text{thermal}} \Phi_{\text{mito+oxid}} \Phi_{\text{haz}}. \quad (28)$$

Equation (28) extends the PBTE invariant from a single reference class to a hierarchy of biological strategies. Clade-specific variation is therefore interpreted not as statistical scatter, but as evidence of distinct thermodynamic mechanisms that modify biological-time accumulation, the entropy cost of physiological cycles, or the accessible lifetime entropy budget. In the following sections, we examine two independent realizations of this framework: the cardiac clock, in which biological proper time is measured by accumulated heartbeats, and the respiratory clock, in which it is measured by accumulated breaths. The presence of approximate lifetime invariants in both systems provides a direct test of whether distinct physiological rhythms are governed by a common thermodynamic structure. In Supplementary Sec. S1, we derive the clade multipliers in detail for major vertebrate lineages, including nonplacental mammals, primates, chiropterans, avian species, and cetaceans. We then use these multipliers to generate quantitative lifespan predictions and compare the predicted values directly with observed lifespans.

To make the predictive content of Φ_C transparent and to guard against the impression that the multipliers are tuned post hoc, we state explicitly how each factor is obtained. The four channels differ sharply in their epistemic status: two are fixed by measured inputs through closed-form expressions, while two are supplied from independent literature and enter phenomenologically. Table 1 records this classification. Throughout, Φ_{duty} and Φ_{thermal} are computed from quantities (q , $f_{H,k}$, T_b) measured per species and inserted into the exact duty-cycle identity and the Arrhenius expression, so they introduce no free parameters once those quantities are known. By contrast,

$\Phi_{\text{mito+oxid}}$ and Φ_{haz} are not derived from the closure; they are taken from comparative biochemistry and demography and carry the larger share of the residual uncertainty in the predictions.

Table 1: Provenance of the four clade-multiplier channels. “Derived” means computed from a closed-form expression once its inputs are fixed; “measured input” identifies the per-species quantities those expressions consume; “literature/phenomenological” means taken from independent comparative studies rather than from the PBTE closure.

Factor	Status	Measured inputs	How obtained
Φ_{duty}	Derived (exact identity)	$q, f_{H,k}, f_{H,\text{ref}}$	Closed-form κ^{-1} from the state-occupancy average; no free parameter.
Φ_{thermal}	Derived (Arrhenius)	T_b, T_{ref}	Closed-form exponential with a single literature activation energy E_a ; no per-species fitting.
$\Phi_{\text{mito+oxid}}$	Literature / phenomenological	— (clade-level)	Estimated from comparative biochemistry (coupling efficiency, ROS production, repair capacity); not derived from the closure.
Φ_{haz}	Literature / phenomenological	— (clade-level)	Inferred from comparative demography (extrinsic mortality, ecological shielding); not derived from the closure.
Φ_{neuro} (primates)	Derived form, calibrated exponent	$\varphi = P_{\text{brain}}/P_{\text{body}}$	Power-law form derived from additive logarithmic sensitivities; the exponent α is calibrated, with $0 < \alpha < 1$ imposed by the second law.

We next examine the entropy cost, mass cancellation, and biological proper time associated with the cardiac and respiratory clocks. In PBTE, a physiological cycle is not merely a countable biological event; it is a thermodynamic event with a definite dissipative cost. For the cardiac clock, this cost is associated with each heartbeat. For the respiratory clock, it is associated with each breath. In both cases, metabolic power, body temperature, physiological frequency, and body mass determine the entropy cost per cycle. Allometric scaling then provides the mechanism by which body-size dependence is largely canceled at the level of the mass-normalized cycle cost. Thus heartbeats and breaths define two distinct, but structurally related, measures of biological proper time. Each records the progression of life through accumulated dissipative cycles rather than through chronological duration alone. Figure 1 summarizes the three central elements of the framework: the entropy-budget cycle-count relation, biological proper time as accumulated cycle count, and the clade multipliers that displace the baseline budget.

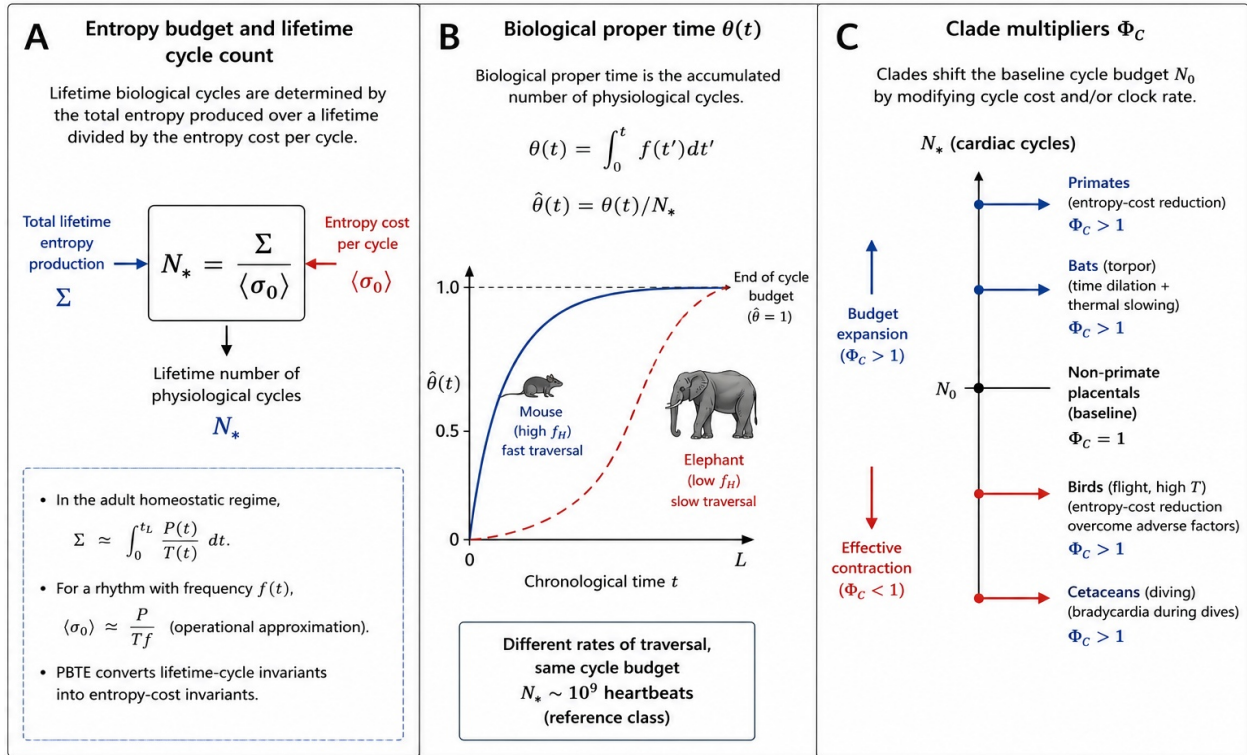


Figure 1: Schematic summary of the PBTE framework. (A) The lifetime number of physiological cycles is determined by the ratio of total lifetime entropy production Σ to the mean entropy cost per cycle $\langle \sigma_0 \rangle$. (B) Biological proper time $\theta(t)$ is the accumulated number of physiological cycles. Small mammals, with high cardiac frequency, traverse the reference cycle budget rapidly in chronological time, whereas large mammals traverse the same budget more slowly. (C) Clade multipliers Φ_C shift the baseline cardiac budget N_0 by modifying the entropy cost per cycle and/or the rate at which biological time advances. Non-primate placentals define the reference class, while primates, bats, birds, and cetaceans represent distinct thermodynamic strategies for modifying the effective lifetime cycle budget.

4 Entropy cost, mass cancellation, and biological proper time

4.1 The cardiac clock

We first apply PBTE to the cardiac rhythm, which provides the most direct physiological realization of biological proper time. For this clock, the relevant frequency is the resting heart rate, $f = f_H$, and the entropy cost per cycle is the entropy cost per heartbeat, denoted by σ_H . In the adult homeostatic regime, the entropy-production rate is estimated by $\dot{e}_p \approx P/T$, where P is metabolic power and T is body temperature. The entropy cost per heartbeat is therefore

$$\sigma_H = \frac{P}{T f_H}. \quad (29)$$

The corresponding mass-specific entropy cost is

$$\bar{\sigma}_H^{(M)} = \frac{\sigma_H}{M} = \frac{P}{T f_H M}. \quad (30)$$

Table 2: Mass-specific entropy cost per heartbeat for representative mammals. Metabolic power is estimated from $P = 3.4M^{0.75}$ W, body temperature is taken as $T \simeq 310$ K, and resting heart rate follows $f_H \simeq 241M^{-0.25}$ min⁻¹.

Species	M (kg)	P (W)	f_H (bpm)	σ_H (10^{-3} J K ⁻¹ beat ⁻¹)	$\bar{\sigma}_H^{(M)}$ (10^{-3} J K ⁻¹ beat ⁻¹ kg ⁻¹)
House mouse	0.020	0.18	600	0.058	2.9
Rat	0.300	1.38	420	0.634	2.1
Rabbit	2.0	5.72	205	2.7	2.7
Dog	23	35.7	90	19.2	3.3
Human	70	82.3	70	56.9	3.3
Horse	500	360	40	273	3.5
Elephant	4000	1710	28	1180	3.0
Mean \pm s.d.					3.0 \pm 0.5
Coefficient of variation					16%

This is the central quantity for the cardiac form of PBTE. The total entropy produced by an organism increases strongly with body size. By contrast, $\bar{\sigma}_H^{(M)}$ measures the entropy cost of one heartbeat per unit body mass. It is this normalized cycle cost, not the total dissipated entropy, that is expected to become approximately invariant after allometric cancellation.

The cancellation follows directly from the leading allometric scalings. If metabolic power scales as $P \propto M^{3/4}$ and resting heart rate scales as $f_H \propto M^{-1/4}$, then Eq. (30) gives

$$\bar{\sigma}_H^{(M)} \propto \frac{M^{3/4}}{M^{-1/4}M} = M^0. \tag{31}$$

Thus PBTE predicts that the entropy cost of one heartbeat per unit body mass is approximately independent of body size. This does not mean that every organism dissipates the same entropy per heartbeat. Rather, it means that after normalization by body mass, the dissipative cost assigned to one cardiac tick becomes nearly conserved across mammals.

Table 2 illustrates this point. The absolute entropy cost per heartbeat, σ_H , varies by orders of magnitude from small to large mammals. This is expected, because a heartbeat in a large animal supports a much larger body mass. However, the mass-specific cost $\bar{\sigma}_H^{(M)}$ remains narrowly distributed around

$$\bar{\sigma}_H^{(M)} \simeq 3.0 \times 10^{-3} \text{ J K}^{-1} \text{ beat}^{-1} \text{ kg}^{-1}. \tag{32}$$

The approximate constancy of $\bar{\sigma}_H^{(M)}$ is therefore the empirical signature of cardiac entropy-cost cancellation. In physical terms, the cardiac invariant means that one unit of cardiac biological time carries nearly the same entropy cost per unit mass across a broad range of mammalian body sizes. Figure 2 shows that the mass-specific cardiac entropy cost remains essentially flat across nearly six orders of magnitude in body mass, consistent with the predicted $\bar{\sigma}_H^{(M)} \propto M^0$ scaling.

The same cancellation gives the thermodynamic interpretation of the lifetime heartbeat count. Using the allometric estimates above yields the *a priori* reference cardiac budget

$$N_{H,0}^{(\text{allo})} \simeq 1.5 \times 10^9 \text{ beats}. \tag{33}$$

We stress that two distinct reference budgets appear in this paper, and we keep them notationally separate throughout. Equation (33) is the *a priori* estimate obtained by inserting the canonical resting allometries ($a \simeq 3.4$ W kg^{-3/4}, $b \simeq 241$ M^{-1/4} min⁻¹, $T \simeq 310$ K) into the closure; it carries no fitting and serves only to show that the lifetime-heartbeat invariant emerges naturally from mass cancellation. For all clade-level lifespan *predictions* (Section 5 and Supplementary Sec. S1)

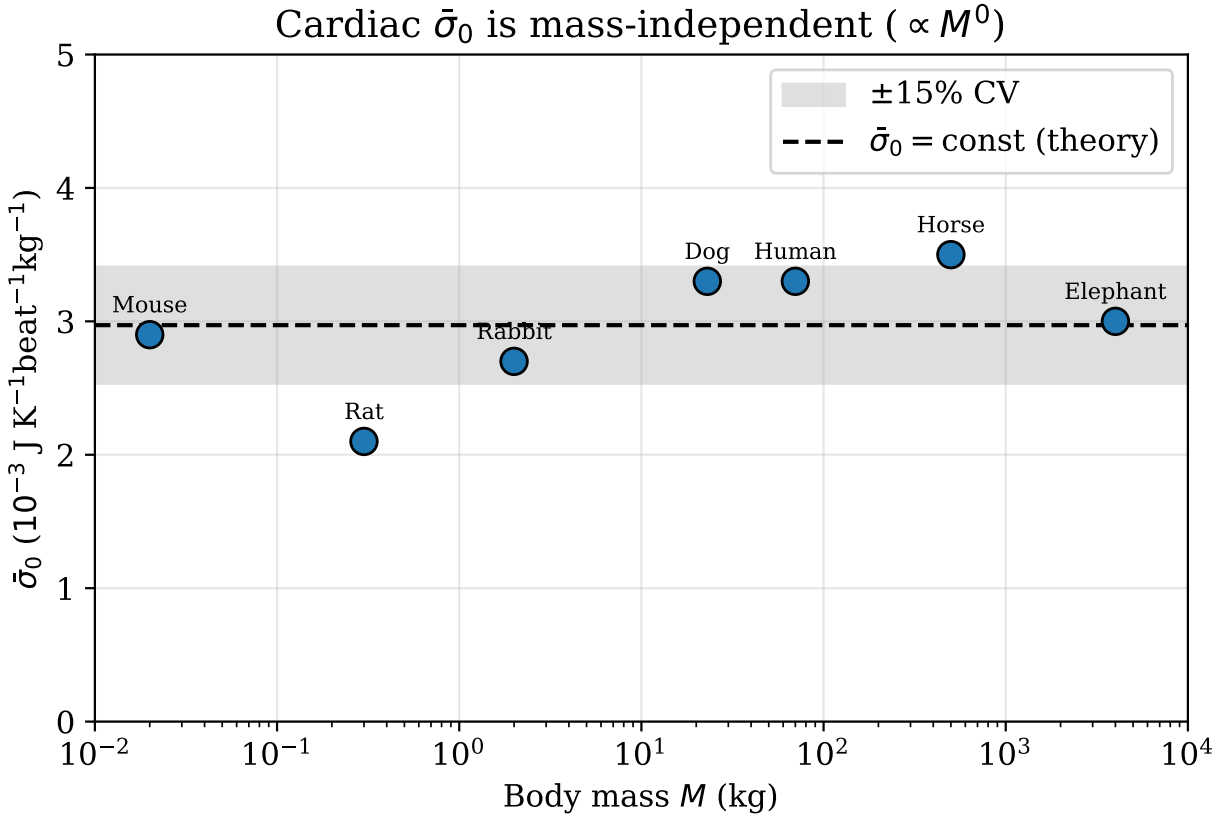


Figure 2: Mass-specific cardiac entropy cost $\bar{\sigma}_H^{(M)}$ versus body mass. The dashed line shows the PBTE prediction $\bar{\sigma}_H^{(M)} \propto M^0$.

we instead use the *fitted empirical anchor*

$$N_{H,0}^{(\text{emp})} = 10^{8.995} \simeq 9.9 \times 10^8 \text{ beats}, \tag{34}$$

the measured mean of $\log_{10} N_H^*$ over the $n = 46$ non-primate placental species (Eq. Supplementary Eq. (S7), Supplementary Table S8). The two differ by about 50% (0.18 dex, where one dex is a factor of ten in \log_{10}) because the *a priori* estimate uses idealized exponents while the empirical anchor absorbs the residual scatter of real allometries; using the fitted value for prediction avoids importing that idealization into the comparisons with observed lifespans. Where the round figure “ $N_0 = 10^9$ ” appears in worked examples it is shorthand for $N_{H,0}^{(\text{emp})}$, to which all predicted lifespans are referred.

The lifetime cardiac invariant is therefore not introduced as a numerical coincidence. It follows from the compensation among metabolic power, cardiac frequency, and body mass. Small mammals dissipate energy rapidly and beat their hearts rapidly. Large mammals dissipate energy more slowly per unit mass and beat their hearts more slowly. These trends cancel in such a way that the mass-normalized entropy cost per heartbeat remains approximately conserved.

The cardiac rhythm also defines a biological proper time. Let

$$\theta_H(t) = \int_0^t f_H(t') dt' \tag{35}$$

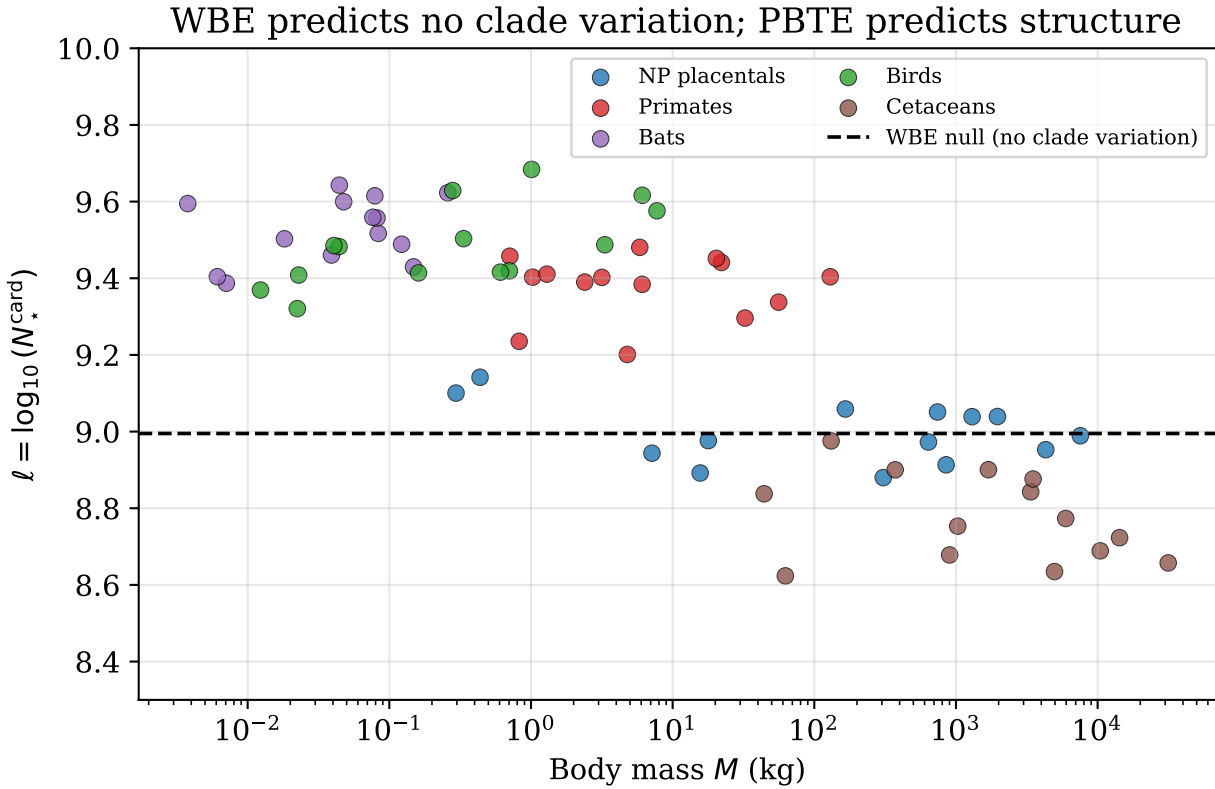


Figure 3: Lifetime cardiac cycle count $\ell = \log_{10}(N_H^*)$ versus body mass. PBTE predicts flat within-clade profiles with clade-dependent vertical offsets $\Delta\ell = \log_{10} \Phi_C$.

denote the accumulated number of cardiac cycles up to chronological time t . The corresponding normalized cardiac age is

$$\hat{\theta}_H(t) = \frac{\theta_H(t)}{N_H^*}. \tag{36}$$

Species with high heart rates advance rapidly in cardiac biological time, whereas species with low heart rates advance more slowly. Normalization by N_H^* places organisms with very different chronological lifespans on a common cardiac-time coordinate.

Finally, the cardiac clock exposes structured clade-level variation around the baseline invariant. We write

$$N_H^* = N_{H,0} \Phi_C, \tag{37}$$

where Φ_C is the clade multiplier. In PBTE, vertical offsets in $\log_{10} N_H^*$ are not treated as residual scatter around a universal allometric line. They are interpreted as changes in the effective entropy cost per heartbeat, the accessible lifetime entropy budget, or the physiological pacing of biological time. Figure 3 displays the lifetime cardiac cycle count against body mass, showing the flat within-clade profiles and clade-dependent vertical offsets $\Delta\ell = \log_{10} \Phi_C$ predicted by the framework.

The main conclusion of this subsection is that the cardiac entropy cost becomes invariant in its mass-specific form. The absolute entropy cost per heartbeat, σ_H , increases with body size because a heartbeat in a large organism supports a larger physiological mass. The PBTE invariant is therefore the mass-normalized quantity $\bar{\sigma}_H^{(M)} = \sigma_H/M$. After allometric cancellation, $\bar{\sigma}_H^{(M)}$ is approximately invariant across body size. This distinction is essential. PBTE does not claim that

all mammals spend the same total entropy per heartbeat. It claims that one heartbeat carries an invariant entropy cost per unit body mass. The cardiac clock therefore provides a clean realization of biological proper time: heartbeats count physiological progression, while $\bar{\sigma}_H^{(M)}$ defines the invariant thermodynamic cost of each cardiac tick on a mass-normalized scale. The respiratory clock examined next provides an independent, but more protocol-sensitive, test of the same principle.

4.2 The respiratory clock

We next apply PBTE to the respiratory rhythm, which provides an independent physiological clock against which the cardiac construction can be tested. For this clock, the relevant frequency is the resting breathing frequency, $f = f_R$, and the entropy cost per cycle is the entropy cost per breath, denoted by σ_R . In the homeostatic regime, the entropy-production rate is estimated by $\dot{e}_p \simeq P/T$, where P is metabolic power and T is body temperature. The entropy cost per breath is therefore

$$\sigma_R = \frac{P}{Tf_R}. \quad (38)$$

When the respiratory frequency is reported in breaths per minute, this becomes

$$\sigma_R = \frac{60P}{Tf_{R,\text{bpm}}}, \quad \bar{\sigma}_R^{(M)} = \frac{\sigma_R}{M} = \frac{60P}{Tf_{R,\text{bpm}}M}. \quad (39)$$

The relevant PBTE quantity is again the mass-specific entropy cost, $\bar{\sigma}_R^{(M)}$. The absolute cost σ_R depends on body size because a breath in a large organism supports a larger physiological mass. By contrast, $\bar{\sigma}_R^{(M)}$ measures the entropy cost of one respiratory cycle per unit body mass. This is the quantity expected to become approximately invariant if metabolic power and respiratory frequency scale in compensating ways.

The leading cancellation has the same structure as in the cardiac case. Under Kleiber metabolic scaling, $P \propto M^{3/4}$, and the approximate respiratory allometry $f_R \propto M^{-1/4}$ [16], consistent with the broader metabolic theory of ecology [43], Eq. (39) gives

$$\bar{\sigma}_R^{(M)} \propto \frac{M^{3/4}}{M^{-1/4}M} = M^0. \quad (40)$$

Thus PBTE predicts that the entropy cost of one breath per unit body mass is approximately independent of body size. This does not imply that all species dissipate the same total entropy per breath. Rather, it means that the respiratory tick carries an approximately invariant entropy cost after normalization by body mass.

For the respiratory analysis we use the 65-species subset of the full cardiac dataset (Supplementary Sec. S2) for which a reliable resting breath rate f_R is available in addition to f_H ; these species and their f_R values are flagged in Supplementary Data File 1. For this subset the non-primate placental baseline is

$$\ell_0 = \log_{10} N_R^* = 8.417 \pm 0.177, \quad N_{R,0}^* \simeq 2.6 \times 10^8 \text{ breaths}. \quad (41)$$

This value defines the reference respiratory cycle budget. It is smaller than the cardiac cycle budget because breaths occur less frequently than heartbeats, but it plays the same conceptual role in PBTE: it measures the number of respiratory ticks through which biological proper time is accumulated. Figure 4 shows the mass-specific respiratory entropy cost estimated with Kleiber metabolic power, in which the leading allometric exponents cancel to give an approximately scale-invariant cost per breath per unit mass, albeit with larger scatter than the cardiac clock.

Respiratory $\bar{\sigma}_R$ (Kleiber power): algebra cancels, scatter large

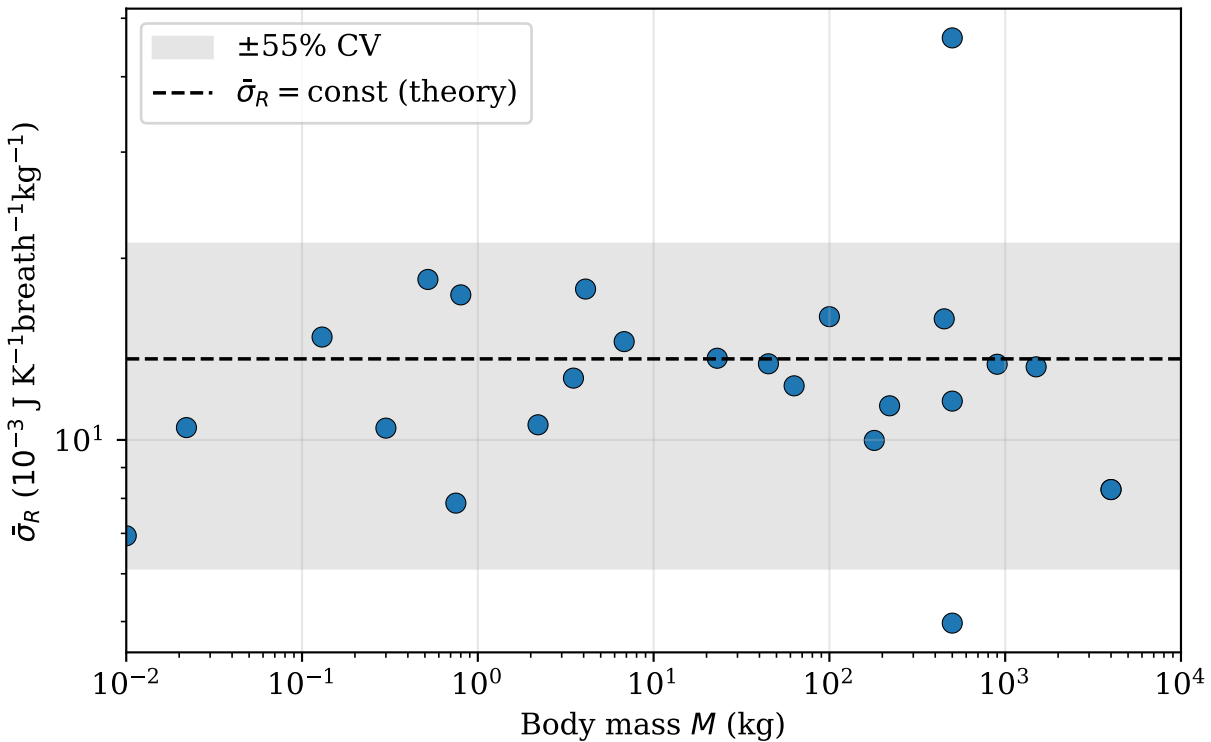


Figure 4: Mass-specific respiratory entropy cost $\bar{\sigma}_R^{(M)}$ estimated with Kleiber metabolic power. The leading allometric exponents cancel, yielding an approximately scale-invariant respiratory entropy cost per breath per unit mass, although the scatter is larger than in the cardiac clock.

The respiratory clock is a more stringent and more protocol-sensitive test of PBTE than the cardiac clock. This is because the cancellation in Eq. (40) can be partly imposed by using Kleiber-derived metabolic power. To reduce this circularity, we also compute σ_R using measured species-level basal metabolic rates wherever available [21, 22], recognizing that field and total energy throughput can depart substantially from basal values [23]. In that analysis, P , f_R , T , and M enter as independent empirical quantities.

Two conclusions follow, and they differ from the Kleiber-power case. First, replacing Kleiber power with measured metabolic power does *not* reduce the scatter; it increases it markedly, to a coefficient of variation of order 100%. Second, the measured analysis reveals a clear positive mass dependence rather than near-cancellation. The slope of $\log_{10} \bar{\sigma}_R^{(M)}$ versus $\log_{10} M$ changes from approximately zero under Kleiber scaling to about +0.21 when measured basal metabolic rates are used, although on $n = 29$ species this slope is not statistically resolved ($p \simeq 0.11$). The mass-specific respiratory entropy cost therefore *rises* with body size on the measured data, contrary to the cardiac case. This positive trend is dominated by aquatic mammals: marine species breathe far more slowly for their metabolic rate than terrestrial species of similar size (the “aquatic breathing strategy”), so the entropy cost per breath, $\sigma_R = P/(Tf_R)$, is strongly inflated at the large-mass, aquatic end of the sample.

We stress that this comparison is the central empirical test of the respiratory clock, not a minor methodological aside. Mass cancellation computed from Kleiber-derived power is partly circular: because $P \propto M^{3/4}$ is imposed, a flat $\bar{\sigma}_R^{(M)}(M)$ is guaranteed by construction and carries

no independent evidential weight. The decisive question is whether the cancellation survives when P is taken from measured basal metabolic rates that do not assume the canonical exponent. On the He et al. (2023) dataset it does *not*, as Figure 5 shows directly: the fitted slope is +0.21 (cf. +0.005 under Kleiber power), the scatter *increases* to a coefficient of variation of order 100%, and the slope is not statistically resolved on the $n = 29$ matched species ($p \simeq 0.11$). The respiratory mass cancellation is therefore an artefact of imposing Kleiber scaling; once P is supplied by independent measurement, it disappears. We regard this as an informative negative result rather than a failure of the framework as a whole. The cardiac clock, where measured resting allometries do yield mass cancellation (Section 4.1, Figure 2), provides the clean realization of the entropy-cost invariant; the respiratory clock does not, because breathing is a strongly regulated control variable—governed by tidal volume, ventilation strategy, thermoregulation, and especially the aquatic versus terrestrial breathing dichotomy—rather than a passive metabolic tick. The asymmetry between the two clocks is itself the substantive finding: the Level-3 invariance claim is supported for the cardiac coordinate and refuted for the respiratory coordinate on currently available measured data. The matched 29-species dataset, its provenance, and the regression are documented in Supplementary Sec. S3.

The respiratory invariant also exhibits systematic clade structure. We write

$$N_R^* = N_{R,0}^* \Phi_C^{(R)}, \quad (42)$$

where $\Phi_C^{(R)}$ is the respiratory clade multiplier. This multiplier measures how far a given clade lies from the non-primate placental baseline. Primates, bats, birds, and cetaceans occupy distinct bands in $\log_{10} N_R^*$. The observed variation is therefore not random scatter around a single universal respiratory count. It is structured variation associated with differences in physiology, ecological risk, thermal regulation, ventilation strategy, and life-history organization.

Once the clade multiplier is specified, the respiratory invariant can be inverted to estimate lifespan from breathing frequency:

$$L_{\text{pred}} = \frac{N_{R,0}^* \Phi_C^{(R)}}{f_{R,\text{bpm}} 525960}. \quad (43)$$

Here $f_{R,\text{bpm}}$ is measured in breaths per minute and L_{pred} is expressed in years. This equation makes the respiratory clock predictive. The measured breathing frequency supplies the physiological pace, whereas $\Phi_C^{(R)}$ supplies the clade-level correction to the accessible biological-time budget.

The main conclusion of this subsection is that the respiratory entropy cost becomes approximately invariant in its mass-specific form. The absolute entropy cost per breath, σ_R , increases with body size because each breath supports a larger physiological mass in larger organisms. The PBTE-relevant quantity is therefore $\bar{\sigma}_R^{(M)} = \sigma_R/M$, the entropy cost per breath per unit body mass. After allometric cancellation, this quantity is approximately scale-invariant, although less tightly than the corresponding cardiac quantity. As Figure 6 shows, the dominant pattern in the respiratory lifetime cycle count is clade-dependent vertical displacement rather than continuous mass dependence. The difference is biologically expected. Respiration is shaped not only by metabolic throughput, but also by tidal volume, ventilation strategy, thermoregulation, diving behavior, ecological state, and measurement protocol. The respiratory clock therefore supports PBTE as an independent biological-time coordinate, while also revealing where physiological complexity enters beyond the leading allometric cancellation.

Table 3: Reference-class PBTE prediction for representative non-primate placental mammals. All entries use $N_{H,0}^{(\text{emp})} = 10^{8.995}$ beats and $\Phi_{\text{NP}} = 1$.

Species	f_H (bpm)	Φ_C	L_{pred} (yr)	L_{obs} (yr)
<i>Mus musculus</i>	632	1.00	3.0	3.5
<i>Rattus norvegicus</i>	420	1.00	4.5	3.8
<i>Oryctolagus cuniculus</i>	205	1.00	9.2	9.0
<i>Felis catus</i>	150	1.00	12.5	15.0
<i>Canis lupus familiaris</i>	90	1.00	20.9	20.0
<i>Equus caballus</i>	38	1.00	49.5	46.0
<i>Loxodonta africana</i>	28	1.00	67.1	65.0
<i>Elephas maximus</i>	27	1.00	69.6	86.0

5 Clade-specific application of the PBTE multiplier

The detailed derivation of the clade multipliers, together with the full data analysis, is presented in Supplementary Sec. S1.1. In this section, we give the main results and emphasize how the PBTE multiplier is used to convert the reference cardiac cycle budget into quantitative lifespan predictions. Once the relevant cardiac frequency and physiological correction factors are specified, the clade multiplier maps the baseline invariant onto a clade-specific effective cycle budget. For consistency with the empirical cardiac dataset, we use the non-primate placental reference

$$N_{H,0}^{(\text{emp})} = 10^{8.995} \simeq 9.9 \times 10^8 \text{ beats}, \quad (44)$$

where 8.995 is the mean value of $\log_{10} N_H^*$ for the non-primate placental baseline. For a cardiac frequency reported in beats per minute, the corresponding clade-level lifespan prediction is

$$L_{\text{pred},C} = \frac{N_{H,0}^{(\text{emp})} \Phi_C}{f_{H,\text{ref}} \mathcal{T}_{\text{yr}}}, \quad \mathcal{T}_{\text{yr}} = 525960 \text{ min yr}^{-1}. \quad (45)$$

Here $f_{H,\text{ref}}$ is the cardiac frequency appropriate to the physiological state used as the reference. For ordinary single-state organisms, it is the resting heart rate. For duty-cycled organisms, such as hibernating bats or diving cetaceans, the active or surface heart rate is used, while suppression of the effective biological clock is included through Φ_{duty} . This convention avoids double counting cardiac slowing and keeps the frequency measurement separate from the physiological multiplier.

5.1 Non-primate placental reference: the mouse-to-elephant baseline

The non-primate placental mammals define the reference PBTE class. In this group no clade-level multiplier is introduced,

$$\Phi_{\text{NP}} = 1. \quad (46)$$

The predicted lifespan is therefore determined only by the resting cardiac frequency,

$$L_{\text{pred,NP}} = \frac{N_{H,0}^{(\text{emp})}}{f_H \mathcal{T}_{\text{yr}}}. \quad (47)$$

This is the unrenormalized PBTE clock. Small mammals have large f_H and therefore consume the reference cardiac budget rapidly in chronological time; large mammals have small f_H and reach the same biological endpoint over a longer chronological interval.

Table 4: Representative primate lifespan predictions. All entries are generated from the single power law $\Phi_{\text{neuro}} = (\varphi/\varphi_0)^\alpha$ with the canonical calibration $\alpha = 0.40$, $\beta = 3$, $\varphi_0 = 0.02$, and the empirical anchor $N_{H,0}^{(\text{emp})} = 10^{8.995}$ beats, via Eq. (45) with $\Phi_C = \Phi_{\text{neuro}}\Phi_{\text{thermal}}$ and $\Phi_{\text{haz}} = 1$. The same calibration is used in Supplementary Sec. S1.4, Supplementary Table S2(a); every value here is reproducible by inspection from f_H , φ , and T_b .

Species	f_H (bpm)	Φ_{neuro}	Φ_{thermal}	Φ_C	L_{pred} (yr)	L_{obs} (yr)
<i>Callithrix jacchus</i>	220	1.55	1.01	1.56	13.3	10–17
<i>Macaca mulatta</i>	120	1.65	1.01	1.67	26.1	25–40
<i>Pan troglodytes</i>	75	2.05	1.03	2.11	52.8	45–60
<i>Gorilla gorilla</i>	65	1.83	1.03	1.88	54.3	40–55
<i>Homo sapiens</i>	70	2.51	1.04	2.60	69.8	70–85

Table 3 shows that the same reference cycle budget captures the correct lifespan scale across several orders of magnitude in body mass. We emphasize that this table is an internal *consistency check*, not an independent prediction: because the anchor $N_{H,0}^{(\text{emp})}$ is itself the mean of $\log_{10} N_H^*$ over precisely these non-primate placentals (Eq. Supplementary Eq. (S7)), the agreement between L_{pred} and L_{obs} tests only that individual placental species scatter tightly about their own clade mean once $\Phi_{\text{NP}} = 1$ is imposed. The genuinely predictive content of the framework lies in the *other* clades, whose multipliers $\Phi_C \neq 1$ are fixed by independent physiological inputs (Sections 5.2–5.5) and then compared with observed lifespans that played no part in setting the anchor. With that caveat, the mouse and elephant are not special exceptions requiring separate mechanisms; they represent the canonical PBTE cancellation. Their different chronological lifespans arise primarily because the mouse advances through cardiac biological proper time rapidly, whereas the elephant advances through the same coordinate slowly.

5.2 Primates

Primates are treated as single-state organisms in the cardiac-clock approximation. Thus,

$$\Phi_{\text{duty}}^{(\text{prim})} = 1. \tag{48}$$

Their elevated lifetime cycle count is therefore interpreted primarily as a reduction in the entropy cost per beat, rather than as a slowing of the cardiac clock. We write

$$\Phi_{\text{prim}} = \Phi_{\text{neuro}}\Phi_{\text{thermal}}\Phi_{\text{haz}}. \tag{49}$$

The dominant contribution is Φ_{neuro} , which represents the reduction in entropy cost per beat associated with neural metabolic investment, predictive regulation, and enhanced physiological maintenance. The thermal correction is comparatively small because primate body temperatures remain close to the mammalian reference.

Table 4 shows that primate lifespan extension is captured by increasing the effective cardiac cycle budget rather than by reducing heart rate. In PBTE terminology, primate longevity is therefore a budget-expansion mechanism: more cardiac cycles can be realized because the entropy burden per cycle is reduced.

5.3 Bats

Bats realize an extended cardiac budget through intermittent physiological suppression. In temperate hibernating bats, a substantial fraction of the year is spent in torpor, where both heart rate

Table 5: Representative bat lifespan predictions. For bats, $f_{H,\text{ref}}$ is the active-phase heart rate; torpor-induced slowing is included through Φ_{duty} .

Species	$f_{H,\text{ref}}$ (bpm)	Φ_{duty}	Φ_{thermal}	Φ_{haz}	Φ_C	L_{pred} (yr)	L_{obs} (yr)
<i>Myotis lucifugus</i>	300	1.94	4.10	0.68	5.40	33.8	34
<i>Eptesicus fuscus</i>	280	1.79	4.54	0.35	2.84	19.1	19
<i>Pteropus vampyrus</i>	250	1.07	1.22	1.60	2.09	15.7	15–23

Table 6: Representative bird lifespan predictions. The first row gives the mechanistic small-passerine calculation. The remaining rows use the representative avian multiplier to illustrate the cardiac lifespan scale for longer-lived birds.

Species or case	f_H (bpm)	Φ_C	L_{pred} (yr)	L_{obs} (yr)
Small passerine	320	2.97	17.4	10–20
<i>Columba livia</i>	190	3.41	33.7	35
<i>Diomedea exulans</i>	100	3.41	64.1	70
<i>Aquila chrysaetos</i>	130	3.41	49.3	46

and body temperature are strongly reduced. The effective multiplier is

$$\Phi_{\text{bat}} = \Phi_{\text{duty}}\Phi_{\text{thermal}}\Phi_{\text{haz}}. \tag{50}$$

The duty-cycle factor accounts for the slowing of cardiac biological time, the thermal factor accounts for Arrhenius suppression of damage kinetics, and the hazard factor accounts for ecological truncation or protection.

For a torpor fraction q , active-phase heart rate $f_{H,\text{act}}$, and torpid heart rate $f_{H,\text{tor}}$, the duty factor is

$$\Phi_{\text{duty}}^{(\text{bat})} = \left[(1 - q) + q \frac{f_{H,\text{tor}}}{f_{H,\text{act}}} \right]^{-1}. \tag{51}$$

The active-phase rate $f_{H,\text{act}}$ is used as $f_{H,\text{ref}}$ in Eq. (45), because torpor-induced slowing is already included through Φ_{duty} .

Table 5 illustrates the multiplicative character of the bat strategy. Duty-cycle slowing alone is insufficient, and thermal suppression alone is insufficient; the observed lifespan scale emerges from their product, modified by extrinsic hazard.

5.4 Birds

Birds provide a contrasting case because two leading factors are adverse: elevated body temperature accelerates damage kinetics, and flight increases cardiac workload. The avian multiplier is written as

$$\Phi_{\text{bird}} = \Phi_{\text{duty}}\Phi_{\text{thermal}}\Phi_{\text{mito-oxid}}\Phi_{\text{haz}}. \tag{52}$$

Because Φ_{duty} and Φ_{thermal} are typically less than unity, avian longevity requires compensating reductions in entropy cost per cycle through mitochondrial efficiency, oxidative resistance, cellular repair, and reduced ecological hazard.

Table 6 shows that birds do not extend lifespan by simply slowing biological time. Instead, adverse thermal and duty-cycle effects are overcome by a larger biochemical and ecological multiplier. In PBTE terminology, the avian strategy is primarily an entropy-cost reduction mechanism rather than a pure clock-slowness mechanism.

Table 7: Representative cetacean lifespan predictions. For cetaceans, $f_{H,\text{ref}}$ is the surface cardiac rate; dive bradycardia is included through Φ_{duty} .

Species	$f_{H,\text{surf}}$ (bpm)	Φ_{duty}	Φ_{thermal}	Φ_{O_2}	Φ_{haz}	Φ_C	L_{pred} (yr)	L_{obs} (yr)
<i>Balaenoptera musculus</i>	37	2.70	1.17	1.40	0.50	2.21	112	80–90
<i>Balaena mysticetus</i>	30	3.08	1.17	1.50	0.60	3.24	203	150–200
<i>Tursiops truncatus</i>	80	1.50	1.09	1.20	0.65	1.28	30	40–50

5.5 Cetaceans

For cetaceans, the dominant correction is sustained diving bradycardia. The surface heart rate is used as the reference frequency, while the reduced cardiac rate during dives is included through the duty-cycle factor. The cetacean multiplier is written as

$$\Phi_{\text{cet}} = \Phi_{\text{duty}} \Phi_{\text{thermal}} \Phi_{\text{O}_2} \Phi_{\text{haz}}, \quad (53)$$

where Φ_{O_2} represents oxygen-storage and dive-associated protection.

For dive fraction p_d , surface heart rate $f_{H,\text{surf}}$, and dive heart rate $f_{H,\text{dive}}$, the duty factor is

$$\Phi_{\text{duty}}^{(\text{cet})} = \left[(1 - p_d) + p_d \frac{f_{H,\text{dive}}}{f_{H,\text{surf}}} \right]^{-1}. \quad (54)$$

As in the bat case, using a time-averaged dive-corrected heart rate and also applying Φ_{duty} would count bradycardia twice.

Table 7 illustrates why raw heartbeat counts in cetaceans can be misleading. A large fraction of life is spent in bradycardic dive states, so the surface heart rate alone does not represent the lifetime cardiac clock. After duty-cycle correction, cetacean longevity is interpreted primarily as a time-dilation mechanism: biological proper time advances more slowly during prolonged diving physiology.

The tables above are intended as representative applications of the PBTE multiplier, not as species-level fitted models. Their purpose is to show how a single reference cardiac budget can be renormalized by clade-specific physiological mechanisms. Detailed derivations of the duty-cycle identities, thermal factors, biochemical factors, and hazard corrections are given in Supplementary Sec. S1 and in the Supplementary Material.

6 Biological proper time and the thermodynamics of aging

The cardiac and respiratory analyses above establish a lifetime cycle budget N_* and an entropy cost per cycle $\langle \sigma_0 \rangle$. This section promotes those lifetime totals to an internal coordinate for the *progression* of an organism through its life, and develops the thermodynamic theory of aging that follows. The construction is the same entropy accounting used above, now read as a function of age rather than as a lifetime total. It connects the present cardiac/respiratory results to the PBTE treatment of aging and longevity.

Thermodynamic foundation. A living organism is an open nonequilibrium system that maintains macroscopic order by continuously transforming chemical free energy into mechanical work, ion gradients, biosynthesis, molecular repair, signaling, and heat [12,13]. It is therefore not an isolated body that passively degrades in calendar time, but a regulated dissipative system whose internal state is sustained by continuous thermodynamic throughput. With $S(t)$ the coarse-grained

internal entropy, $\dot{e}_p(t) \geq 0$ the irreversible entropy-production rate, and $\dot{h}_d(t) \geq 0$ the entropy exported to the environment, the open-system balance is

$$\frac{dS}{dt} = \dot{e}_p(t) - \dot{h}_d(t). \quad (55)$$

In adult homeostasis the organism is held near a nonequilibrium steady state in which internal entropy is regulated within a bounded physiological range. On timescales long compared with metabolic fluctuations but short compared with lifespan-scale deterioration, Eq. (55) gives

$$\frac{dS}{dt} \simeq 0, \quad \dot{e}_p(t) \simeq \dot{h}_d(t), \quad (56)$$

and the entropy exported as heat is estimated to leading order by the dissipated metabolic power divided by absolute body temperature,

$$\dot{h}_d(t) \simeq \frac{P(t)}{T(t)}. \quad (57)$$

Combining Eqs. (56) and (57) gives the homeostatic closure

$$\dot{e}_p(t) \simeq \dot{h}_d(t) \simeq \frac{P(t)}{T(t)}. \quad (58)$$

Aging becomes visible when this balance is no longer exact. If entropy export, molecular repair, proteostasis, immune regulation, and cellular renewal fail to fully compensate irreversible entropy production, the coarse-grained internal burden associated with disorder, damage, and loss of regulation accumulates. Integrating Eq. (55) from the initial life-history time to age t ,

$$S(t) = S(0) + \int_0^t [\dot{e}_p(s) - \dot{h}_d(s)] ds. \quad (59)$$

PBTE does not identify this coarse-grained entropy with a single biomarker; instead, entropy production is used to assign a thermodynamic cost to the advancement of an internal physiological clock.

Internal-time coordinate and entropy cost per cycle. Let $f(t)$ be a physiological frequency—heart rate, respiratory rate, or another biologically meaningful rhythm. The accumulated biological proper time is the total number of physiological cycles completed up to age t , already introduced in Eq. (21),

$$\theta(t) = \int_0^t f(s) ds, \quad d\theta = f(t) dt. \quad (60)$$

Thus θ is an internal cycle coordinate, not an external time coordinate. The entropy produced in the same infinitesimal interval is $d\Sigma = \dot{e}_p(t) dt$; using $dt = d\theta/f(t)$ this becomes

$$d\Sigma = \frac{\dot{e}_p(t)}{f(t)} d\theta, \quad (61)$$

which identifies the instantaneous entropy cost per biological tick as

$$\sigma_0(t) = \frac{\dot{e}_p(t)}{f(t)} = \frac{d\Sigma}{d\theta}, \quad (62)$$

the entropy produced per physiological cycle, i.e. the thermodynamic price of one increment of biological proper time. A cycle is more expensive when entropy production is high relative to the physiological frequency, and less expensive when the same cycle is executed with lower irreversible dissipation. Over the lifespan L , with total entropy production $\Sigma = \int_0^L \dot{e}_p dt$ and total cycle count $N_\star = \theta(L) = \int_0^L f dt$, the relation $d\Sigma = \sigma_0 d\theta$ gives $\Sigma = \int_0^{N_\star} \sigma_0(\theta) d\theta$, so the lifetime-average cost per tick is

$$\langle \sigma_0 \rangle = \frac{1}{N_\star} \int_0^{N_\star} \sigma_0(\theta) d\theta = \frac{\Sigma}{N_\star}, \tag{63}$$

and rearranging recovers the PBTE cycle-count relation

$$\boxed{N_\star = \frac{\Sigma}{\langle \sigma_0 \rangle}}. \tag{64}$$

This is an accounting identity with biological content: the number of cycles completed over life equals the total entropy produced divided by the average entropy cost of one cycle. It does not assert death at an exact universal cycle number; it states that biological time advances by consuming an entropy-normalized cycle budget. In the constant limit $f(t) = f$, $\sigma_0(t) = \sigma_0$ one has $N_\star = fL$ and $L = N_\star/f$, so for a fixed budget a higher physiological frequency implies a shorter chronological lifespan. The metabolic closure Eq. (58) gives the complementary thermodynamic form

$$\sigma_0(t) \simeq \frac{P(t)}{T(t) f(t)}, \tag{65}$$

so the cost of one cycle is set by metabolic power per unit temperature per unit frequency.

Biological age as a normalized internal-time coordinate. The simplest PBTE biological age is the normalized internal time of Eq. (21),

$$A_\theta(t) = \frac{\theta(t)}{N_\star}, \tag{66}$$

running from $A_\theta = 0$ at birth to $A_\theta = 1$ at the terminal PBTE boundary; for constant f it reduces to $A_\theta(t) = ft/N_\star = t/L$. Cycle count alone, however, is not sufficient. The same heartbeat or breath can carry a different thermodynamic cost depending on mitochondrial efficiency, oxidative load, inflammation, repair capacity, temperature, and disease. To carry this information, PBTE weights each cycle by its entropy cost through the entropy-normalized biological time

$$\Theta_\sigma(t) = \int_0^t \frac{\sigma_0(s)}{\sigma_{0,\text{ref}}} f(s) ds, \tag{67}$$

where $\sigma_{0,\text{ref}}$ is a reference entropy cost per cycle. A cycle with $\sigma_0 = \sigma_{0,\text{ref}}$ contributes one reference tick; a more expensive cycle contributes more than one, a cheaper cycle less. Using $\sigma_0(t) = \dot{e}_p(t)/f(t)$ the frequency cancels and

$$\Theta_\sigma(t) = \int_0^t \frac{\dot{e}_p(s)}{\sigma_{0,\text{ref}}} ds = \frac{\Sigma(t)}{\sigma_{0,\text{ref}}}, \quad \Sigma(t) = \int_0^t \dot{e}_p(s) ds, \tag{68}$$

so Θ_σ is both the number of entropy-weighted cycles and the accumulated entropy production in units of the reference cost. The PBTE biological age is the normalized coordinate

$$A_{\text{PBTE}}(t) = \frac{\Theta_\sigma(t)}{N_{\star,\text{ref}}} = \frac{1}{N_{\star,\text{ref}}} \int_0^t \frac{\sigma_0(s)}{\sigma_{0,\text{ref}}} f(s) ds, \tag{69}$$

with $N_{*,\text{ref}}$ the reference entropy–cycle budget. Defining $\Sigma_{\text{ref}} = \sigma_{0,\text{ref}} N_{*,\text{ref}}$ and using Eq. (68) gives the compact form

$$A_{\text{PBTE}}(t) = \frac{\Sigma(t)}{\Sigma_{\text{ref}}}, \quad (70)$$

so PBTE biological age is the fraction of a reference lifetime entropy–cycle budget already consumed. Differentiating Eq. (69) gives the instantaneous aging rate,

$$\frac{dA_{\text{PBTE}}}{dt} = \frac{1}{N_{*,\text{ref}}} \frac{\sigma_0(t)}{\sigma_{0,\text{ref}}} f(t) = \frac{\dot{e}_p(t)}{\sigma_{0,\text{ref}} N_{*,\text{ref}}} = \frac{\dot{e}_p(t)}{\Sigma_{\text{ref}}}, \quad (71)$$

where $\sigma_0 f = \dot{e}_p$, and with the closure Eq. (58),

$$\frac{dA_{\text{PBTE}}}{dt} \simeq \frac{P(t)}{T(t) \Sigma_{\text{ref}}}. \quad (72)$$

Central definition.

$$A_{\text{PBTE}}(t) = \frac{1}{N_{*,\text{ref}}} \int_0^t \frac{\sigma_0(s)}{\sigma_{0,\text{ref}}} f(s) ds = \frac{\Sigma(t)}{\Sigma_{\text{ref}}}$$

biological age = fraction of the reference entropy–cycle budget consumed

Equations (69) and (71) make the thermodynamic content of aging explicit. An organism ages faster when its physiological clock runs faster (large f), when each biological tick is thermodynamically more expensive (large σ_0), or when both occur together. It ages more slowly when physiological rate is reduced without functional collapse, when entropy production per cycle is lowered by more efficient maintenance, or when the accessible budget $N_{*,\text{ref}}$ is enlarged. This is why chronological age is sometimes predictive and sometimes misleading: calendar time is a faithful proxy only when $f(t)$, $\sigma_0(t)$, and the effective budget are similar across the organisms compared. When they differ—precisely the clade structure documented in Sec. 5—two organisms of identical chronological age occupy different positions on the entropy-normalized internal-time axis. The clade multiplier Φ_C of Eq. (3), which rescales $N_{*,\text{ref}}$, is therefore not only a lifespan-prediction factor but the rescaling that maps chronological age onto biological age.

This coordinate organizes aging and longevity into three thermodynamic mechanism classes, all visible in the clade results of this paper. The first is *time dilation*: reducing the rate dA_{PBTE}/dt at which biological proper time accumulates, without lowering the cost or enlarging the budget. Caloric restriction, torpor, and the bradycardic diving physiology of cetaceans act here, slowing the internal clock so that fewer entropy–cycles are spent per calendar year and the same budget is stretched over a longer chronological lifespan. In PBTE terms these interventions reduce $f(t)$ or the effective duty cycle, and longevity is bought by living more slowly in internal time rather than by changing the total budget.

The second class is *entropy-cost reduction and budget expansion*: lowering σ_0 per cycle or raising $N_{*,\text{ref}}$ so that more biological time can be traversed before the terminal boundary. Birds, despite high body temperatures and metabolic rates, and primates, with their enhanced cellular maintenance, realize this strategy through superior mitochondrial efficiency, antioxidant protection, and repair capacity. Each heartbeat is made thermodynamically cheaper, or more of them are made affordable within the budget, so longevity is bought per physiological cycle rather than per calendar year. The cardiac entropy-cost invariant established in this paper is what makes $\sigma_{0,\text{ref}}$ a well-defined reference against which these clade-specific reductions can be measured.

The third class is the *hypertemporal* pathologies of aging: chronic inflammation, metabolic syndrome, neurodegeneration, and cancer, which raise $f(t)$, $\sigma_0(t)$, or both, and so accelerate dA_{PBTE}/dt . In this picture such states are not merely damage endpoints but increases in the velocity of biological time, pushing the organism through its entropy–cycle budget faster than chronological age would suggest. The framework yields a falsifiable consequence: biological-age biomarkers, especially DNA-methylation clocks, should correlate more tightly with accumulated internal time A_{PBTE} than with chronological age, and damage and mortality trajectories should collapse more cleanly when plotted against the cardiac A_{PBTE} —whose reference cost survives the non-circular test—than when plotted against chronological time or against the respiratory clock that does not.

7 Summary and conclusions

In this work we developed the Principle of Biological Time Equivalence (PBTE) as a thermodynamic framework for interpreting lifetime physiological cycle invariants in vertebrates. The central idea is that biological time is not measured most naturally by chronological duration alone, but by the accumulated number of recurrent physiological cycles weighted by their entropy cost. In this formulation, lifespan is governed by how rapidly an organism consumes an entropy-weighted cycle budget.

The cardiac clock provides the clearest realization of this principle. Across representative mammals, the absolute entropy cost per heartbeat increases strongly with body size, but the mass-specific entropy cost per heartbeat remains approximately constant. This cancellation follows from the compensation between metabolic power, cardiac frequency, and body mass. As a result, small mammals and large mammals can have very different chronological lifespans while still approaching a comparable endpoint in cardiac biological proper time. The mouse-to-elephant baseline is therefore not a collection of special cases, but the canonical expression of the PBTE invariant.

The respiratory clock provides an independent, but coarser, test of the same construction. The lifetime breath count is also approximately invariant, and the mass-specific entropy cost per breath is much flatter than the absolute entropy cost per breath. However, respiration shows larger scatter than the cardiac clock. This is expected because breathing is not only a clock, but also a regulated control variable for gas exchange, tidal volume, thermoregulation, diving, and activity state. The respiratory results therefore support PBTE while also showing that not all physiological rhythms provide equally clean measures of biological time.

The comparison between the cardiac and respiratory clocks gives an important internal consistency check. A breath is a slower and coarser cycle than a heartbeat, and therefore carries the entropy cost of several cardiac cycles. In typical mammals this ratio is of order four to five, consistent with the observed relation between respiratory and cardiac frequencies. The same comparison clarifies why surface breath counts can be misleading in diving mammals: in cetaceans, respiratory and cardiac pacing are strongly modified by dive physiology, so the cardiac clock remains the cleaner thermodynamic coordinate.

The clade-multiplier analysis extends the theory beyond the baseline invariant. Non-primate placental mammals define the reference class. Primates extend the effective cycle budget primarily by reducing the entropy burden per cardiac cycle. Bats extend lifespan through the combined effects of torpor, duty-cycle suppression, and thermal slowing. Birds overcome adverse thermal and flight-related costs through mitochondrial efficiency, oxidative resistance, cellular maintenance, and reduced extrinsic hazard. Cetaceans extend biological time through bradycardic pacing during prolonged dives. These cases show that clade-level deviations are not residual scatter around a single

allometric law, but structured thermodynamic strategies for modifying the effective biological-time budget.

The framework should be interpreted conservatively. PBTE is not a complete molecular theory of aging, and it does not by itself identify every biochemical mechanism that determines lifespan. Rather, it provides a macroscopic thermodynamic organization of comparative longevity that complements, rather than replaces, demographic descriptions of mortality such as the Gompertz law [25], molecular-clock measures of biological age [41], and intervention studies of the metabolic determinants of lifespan [42, 18]. Molecular repair, oxidative protection, neural regulation, torpor, diving physiology, and ecological hazard enter as mechanisms that modify either the rate at which biological time advances or the entropy cost of each physiological cycle.

The decisive experimental test is direct measurement of entropy cost per cycle. Metabolic power, body temperature, body mass, heart rate, and breathing frequency should be measured simultaneously across species under matched resting conditions. For respiration, tidal volume or oxygen throughput should also be measured so that breath count can be compared with a more complete ventilatory coordinate. If the mass-specific entropy cost per physiological cycle is found to be approximately invariant within defined biological regimes, PBTE would move from a thermodynamically motivated comparative framework to a directly tested thermodynamic parametrisation of biological time. If systematic nonzero mass or clade dependence is found, the closure would require revision.

In conclusion, cardiac and respiratory lifetime counts are two physiological projections of a common thermodynamic structure. The heartbeat provides the sharper invariant and the more stable entropy cost; the breath provides an independent but more protocol-sensitive confirmation. Together, they support the central PBTE claim that biological time is organized not by chronological duration alone, but by the entropy cost of recurrent physiological cycles. Longevity, in this view, is the outcome of how slowly and how efficiently an organism spends its finite entropy-weighted budget of biological time.

Appendix A Derivation of the entropy cost per cycle and the cycle-count identity

This appendix gives the self-contained derivation of the central PBTE identity $N_\star = \Sigma/\bar{\sigma}_0$ and of the mass-cancellation result $\bar{\sigma}_0^{(M)} \propto M^0$, expanding the compressed argument of Section 2.

A.1 Entropy balance of an open metabolic system

A living organism is an open system exchanging energy and matter with its environment. Let $S(t)$ be its coarse-grained internal entropy. The second law for an open system partitions the entropy change into an internally produced part and an exchanged part,

$$dS = d_i S + d_e S, \quad d_i S \geq 0, \quad (73)$$

where $d_i S$ is the irreversible entropy production and $d_e S$ the entropy exchanged with the surroundings. Writing the production rate as $\dot{e}_p \equiv d_i S/dt \geq 0$ and the export rate as $\dot{h}_d \equiv -d_e S/dt \geq 0$ (export is entropy leaving the body, hence the sign), the balance becomes

$$\dot{S} = \dot{e}_p - \dot{h}_d, \quad (74)$$

which is Eq. (6). To leading thermodynamic order, an endotherm dissipating metabolic power P at body temperature T exports entropy to its surroundings at the rate

$$\dot{h}_d \simeq \frac{P}{T}, \tag{75}$$

the Clausius heat-export term. Equation (75) follows from the observation that, in steady metabolism, essentially all ingested free energy is ultimately degraded to heat $\dot{Q} \simeq P$ and rejected at body temperature; the associated entropy flux is \dot{Q}/T .

A.2 Homeostatic (steady-state) regime

Over the timescale on which resting metabolic rate and physiological rhythm are measured, the internal entropy content is approximately stationary: stored entropy neither grows nor decays appreciably during a resting measurement, so $\dot{S} \simeq 0$ and

$$\dot{e}_p \simeq \dot{h}_d \simeq \frac{P}{T}. \tag{76}$$

This is a *nonequilibrium steady state*, not thermodynamic equilibrium: entropy is produced continuously at rate $\dot{e}_p > 0$ but is exported as fast as it is produced. Equation (76) is used only to evaluate the lifetime invariant; it is *not* assumed during senescence, which is treated separately in the Supplementary Material.

A.3 The cycle as the natural unit of entropy

Let $f(t)$ be the frequency of a recurrent physiological cycle. Its cumulative count is $N(t) = \int_0^t f dt'$, so $dN = f dt$. The entropy produced in the same interval is $d\Sigma = \dot{e}_p dt$. Eliminating dt ,

$$d\Sigma = \frac{\dot{e}_p}{f} dN \equiv \sigma_0(t) dN, \quad \sigma_0 \equiv \frac{\dot{e}_p}{f} = \frac{d\Sigma}{dN}. \tag{77}$$

Thus σ_0 is not a dimensional convention: it is the entropy produced each time the physiological clock advances by one cycle, with units $\text{JK}^{-1} \text{cycle}^{-1}$. Integrating over the lifespan,

$$\Sigma = \int_0^L \dot{e}_p dt = \int_0^{N_*} \sigma_0(N) dN = \bar{\sigma}_0 N_*, \quad \bar{\sigma}_0 \equiv \frac{1}{N_*} \int_0^{N_*} \sigma_0(N) dN, \tag{78}$$

which rearranges to the fundamental identity

$$\boxed{N_* = \frac{\Sigma}{\bar{\sigma}_0}} \tag{79}$$

(Eq. (16)). The lifetime cycle count is the lifetime entropy budget divided by the mean entropy cost of one cycle. No scaling assumption has yet been made; Eq. (79) is exact given the definitions.

A.4 The PBTE closure

The Principle of Biological Time Equivalence adds the constitutive hypothesis that within a fixed adult homeostatic regime the cost per cycle is approximately constant, $\sigma_0(N) \simeq \bar{\sigma}_0 \simeq \sigma_0$, so that

$$\dot{e}_p(t) = \sigma_0 f(t). \tag{80}$$

Combining the closure with the homeostatic estimate $\dot{e}_p \simeq P/T$ gives the operational estimator

$$\sigma_0 = \frac{P}{Tf}, \quad \bar{\sigma}_0^{(M)} \equiv \frac{\sigma_0}{M} = \frac{P}{TfM}. \quad (81)$$

This is a falsifiable approximation, not an identity: it can be tested by measuring P , T , and f on the same individuals and checking whether σ_0 is constant within a homeostatic regime.

A.5 Mass cancellation

Insert the two empirical allometries

$$P = a M^{3/4} \quad (\text{Kleiber}), \quad f = b M^{-1/4} \quad (\text{quarter-power frequency}), \quad (82)$$

into the mass-specific cost (81):

$$\bar{\sigma}_0^{(M)} = \frac{aM^{3/4}}{T(bM^{-1/4})M} = \frac{a}{bT} M^{3/4+1/4-1} = \frac{a}{bT} M^0. \quad (83)$$

The three mass exponents sum exactly to zero: $+3/4$ from metabolic power, $+1/4$ from the inverse frequency, and -1 from mass normalization. Hence the mass-specific entropy cost per cycle is, to the accuracy of the allometric exponents, independent of body size. This is the thermodynamic content of the lifetime-invariant:

$$N_\star = \frac{\Sigma}{\bar{\sigma}_0} = \frac{\Sigma/M}{\bar{\sigma}_0^{(M)}} \approx \text{const} \quad (84)$$

whenever the lifetime entropy budget per unit mass, Σ/M , is also approximately conserved within the reference class. With the resting numerical values $a \simeq 3.4 \text{ W kg}^{-3/4}$, $T \simeq 310 \text{ K}$, and $b \simeq 241 \text{ M}^{-1/4} \text{ min}^{-1}$ one obtains $\bar{\sigma}_H^{(M)} \simeq 3.0 \times 10^{-3} \text{ J K}^{-1} \text{ beat}^{-1} \text{ kg}^{-1}$ and the *a priori* reference cardiac count $N_{H,0}^{(\text{allo})} \simeq 1.5 \times 10^9$ beats. As emphasized in Section 4.1, this allometric estimate is kept distinct from the fitted empirical anchor $N_{H,0}^{(\text{emp})} = 10^{8.995} \simeq 9.9 \times 10^8$ beats used for all clade-level predictions; the two differ by $\simeq 0.18$ dex.

A.6 Sensitivity to the allometric exponents

If the exponents deviate from their canonical values, $P \propto M^p$ and $f \propto M^{-q}$, then $\bar{\sigma}_0^{(M)} \propto M^{p-1+q}$. The cancellation is exact only for $p + q = 1$. The canonical pair $(p, q) = (3/4, 1/4)$ satisfies this exactly, but measured metabolic exponents range over $p \approx 0.67\text{--}0.75$ depending on taxon and metabolic state [11]. A residual slope $s = p - 1 + q$ of order ± 0.05 is therefore expected for a clade whose metabolic exponent departs only slightly from $3/4$. The measured-BMR respiratory analysis (Section 4.2, Supplementary Sec. S3) does not fall in this small-residual regime: it returns a substantially positive slope ($s \simeq +0.21$) with large scatter, indicating that the respiratory cancellation is not merely approximate but absent once measured metabolic power is used. The mass cancellation is thus realized for the cardiac clock but not the respiratory clock, and the magnitude of the respiratory slope is itself a test of the closure—one the respiratory coordinate fails on currently available data.

Appendix B Biological proper time as the entropy-uniform coordinate

Biological proper time is defined by $\theta(t) = \int_0^t f(t') dt'$, with normalized age $\hat{\theta} = \theta/N_*$. Under the closure, $d\Sigma = \sigma_0 f dt = \sigma_0 d\theta$, hence

$$\frac{d\Sigma}{d\theta} = \sigma_0 = \text{const.} \quad (85)$$

Entropy therefore accumulates *linearly* in θ , which is the defining property of biological proper time.

Uniqueness. We show θ is the unique intrinsic coordinate (up to affine rescaling) with this property. Let $u(t)$ be any strictly monotone reparametrisation of chronological time, $du = w(t) dt$ with $w > 0$. Then

$$\frac{d\Sigma}{du} = \frac{d\Sigma/dt}{du/dt} = \frac{\sigma_0 f(t)}{w(t)}. \quad (86)$$

For this to be constant for all t , independent of the (generally time-varying) frequency $f(t)$, one must have $w(t) \propto f(t)$, i.e. $du \propto f dt = d\theta$. Hence $u = c_1\theta + c_2$ for constants $c_1 > 0, c_2$. Up to choice of origin and unit, θ is the unique coordinate in which entropy accumulation is rate-independent and uniform. Chronological time fails this test precisely because $f(t)$ varies; a hummingbird minute and an elephant minute carry different entropy, but a hummingbird *beat* and an elephant *beat* carry nearly the same mass-specific entropy by Eq. (83).

Two clocks, one budget. For the cardiac and respiratory clocks, $\theta_H = \int f_H dt$ and $\theta_R = \int f_R dt$, and the lifetime endpoints are $N_H = \Sigma/\bar{\sigma}_H$ and $N_R = \Sigma/\bar{\sigma}_R$. Because both share the same lifetime entropy budget Σ , their ratio is fixed by the cost ratio,

$$\frac{N_H}{N_R} = \frac{\bar{\sigma}_R}{\bar{\sigma}_H} = \frac{f_H}{f_R}, \quad (87)$$

recovering the heart–breath frequency ratio of order four to five discussed in Section 7.

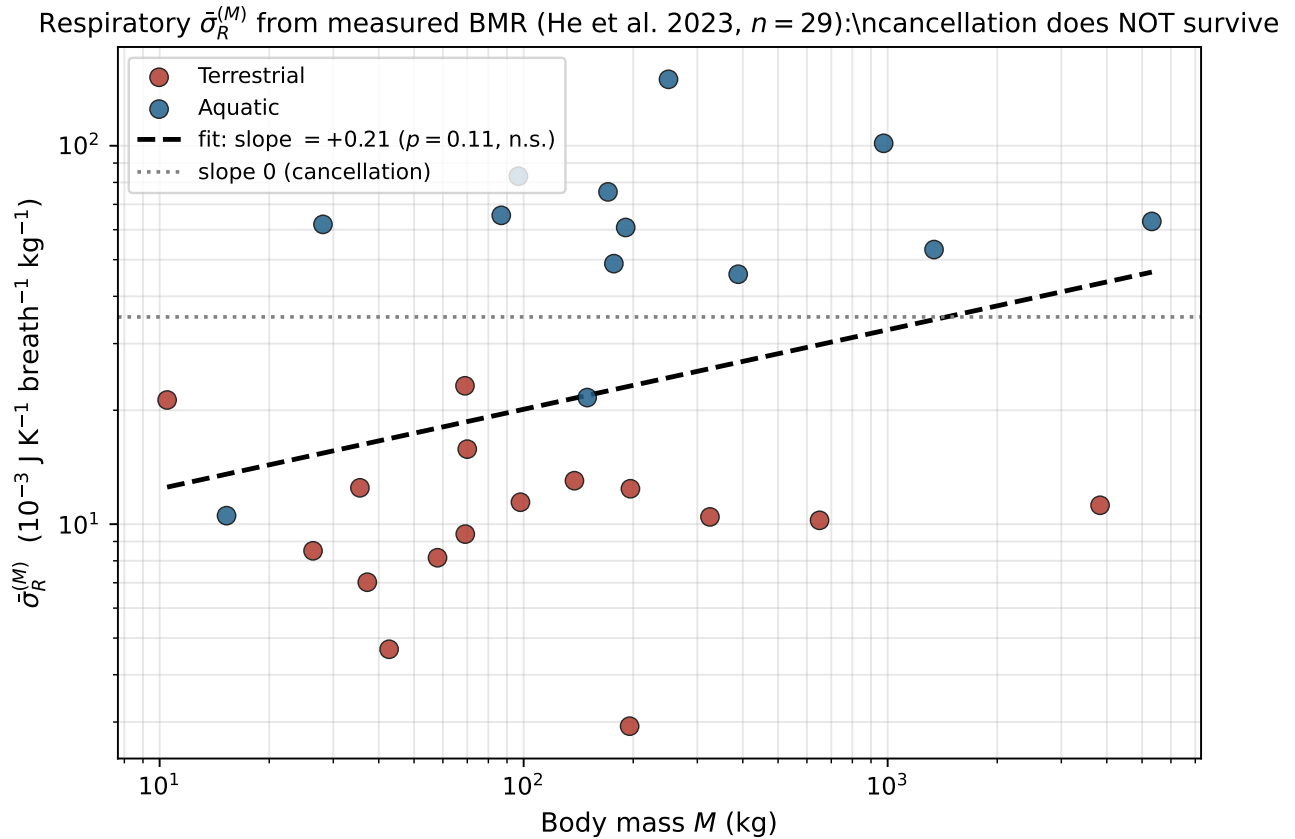


Figure 5: The non-circular test of the respiratory clock: mass cancellation does not survive measured BMR. Mass-specific respiratory entropy cost $\bar{\sigma}_R^{(M)}$ computed from *measured* species-level basal metabolic rates (He et al. 2023), in which P , f_R , T , and M enter as independent empirical quantities rather than through imposed Kleiber scaling. Unlike Figure 4, where flatness is partly guaranteed by construction ($P \propto M^{3/4}$), here no canonical exponent is assumed. On the $n = 29$ mammals with both measured BMR and measured resting breath rate, the fitted slope is +0.21 (cf. +0.005 under Kleiber power); it is not statistically resolved ($p \simeq 0.11$), and the scatter is large (coefficient of variation $\sim 100\%$). The cancellation seen under Kleiber power therefore does not persist under independent inputs. The positive trend is dominated by aquatic mammals (blue), whose low resting breath rate relative to metabolic rate inflates σ_R at large body mass. This is the central empirical result of the respiratory analysis: the respiratory clock fails the non-circular cancellation test that the cardiac clock passes. Dataset and regression details are given in Supplementary Sec. S3.

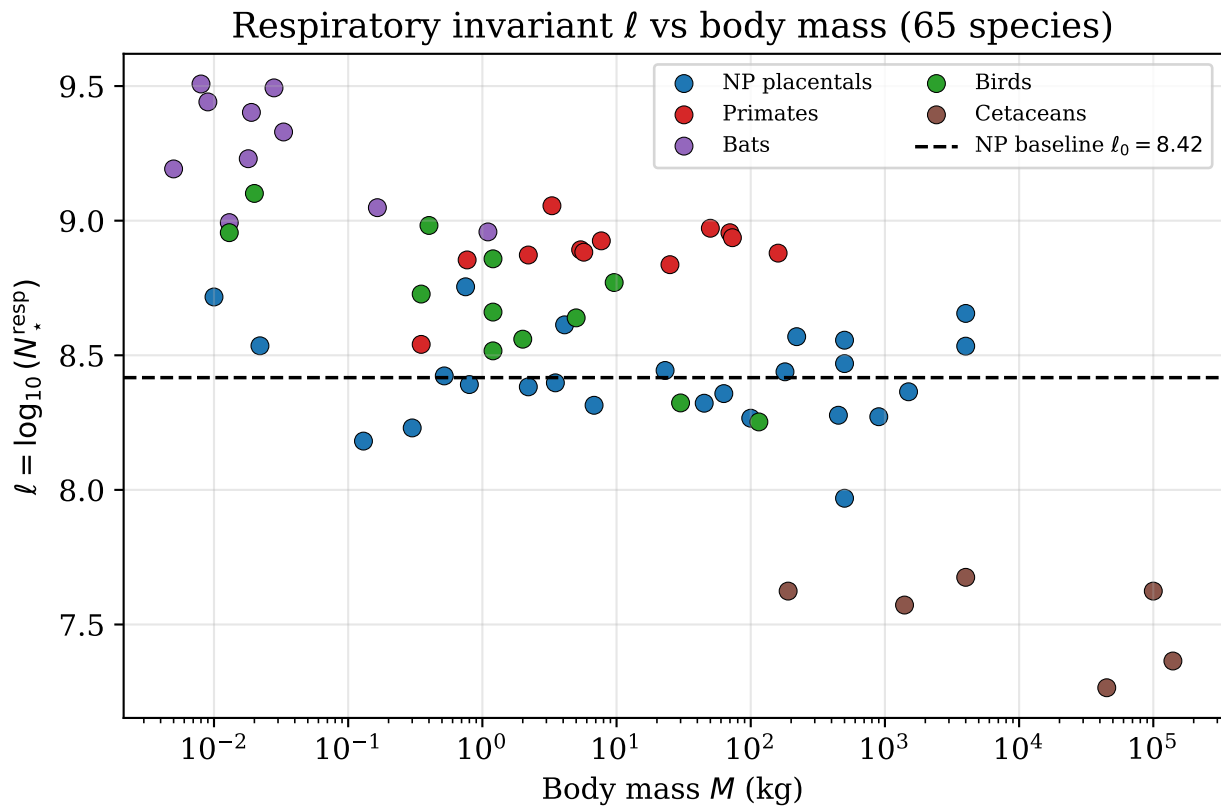


Figure 6: Respiratory lifetime cycle count $l = \log_{10}(N_R^*)$ versus body mass. The dominant pattern is clade-dependent vertical displacement rather than continuous mass dependence.

Supplemental Material

S1 Detailed derivation of the clade multiplier Φ_C for all vertebrate clades

S1.1 General case

In this appendix we derive the clade multiplier Φ_C in full for every clade analysed in the main text. The derivation begins from a single thermodynamic identity and ends with worked numerical predictions for each group. The treatment is self-contained: all governing relations, parameter values, and dataset entries required to reproduce the results are stated explicitly, so that the appendix may be read independently of the main body.

The eight clades fall into three structural classes. Non-primate placental mammals define the reference state, against which all departures are measured. The remaining endothermic clades—primates, marsupials and monotremes, bats, birds, and cetaceans—depart from the reference through distinct combinations of the same four physiological channels; what appears as clade-specific scatter is thereby recast as structured variation in a small set of channel factors. Ectotherms, represented here by reptiles and amphibians, form the limiting case in which the displacement is governed almost entirely by temperature.

All clades share a common entropy-budget construction. Let the lifetime cardiac cycle count in clade C , expressed relative to the non-primate placental baseline N_0 , be written as

$$N_\star^{(C)} = N_0 \Phi_C, \quad \Phi_C \equiv \frac{N_\star^{(C)}}{N_0} = \frac{\langle \sigma_{\text{beat}} \rangle_0}{\langle \sigma_{\text{beat}} \rangle_C}, \quad (\text{S1})$$

where $\langle \sigma_{\text{beat}} \rangle_C$ denotes the mean entropy produced per cardiac cycle in clade C , in the same notation σ used for the entropy cost per cycle throughout the main text and Appendix A. The lifetime entropy budget Σ_\star is conserved across clades, so that $N_\star = \Sigma_\star / \langle \sigma_{\text{beat}} \rangle$; it follows that any mechanism reducing the entropy produced per beat below the mammalian baseline elevates Φ_C above unity and prolongs chronological lifespan.

The multiplier factorises into four physically distinct channels,

$$\Phi_C = \Phi_{\text{duty}} \cdot \Phi_{\text{thermal}} \cdot \Phi_{\text{mito+oxid}} \cdot \Phi_{\text{haz}}. \quad (\text{S2})$$

These four channels differ in their epistemic status, and we are explicit about this distinction throughout, since it determines how much predictive weight each carries. Two channels are obtained from closed-form expressions once their measured inputs are fixed: Φ_{duty} follows from an exact algebraic identity (the state-occupancy average), and Φ_{thermal} from Arrhenius kinetics with a single literature activation energy. The remaining two, $\Phi_{\text{mito+oxid}}$ and Φ_{haz} , are *phenomenologically inferred*: they are taken from comparative biochemistry and demography respectively, rather than derived from the thermodynamic closure, and carry the larger share of the residual uncertainty in the predictions. In primates a derived-form but empirically calibrated sub-channel Φ_{neuro} supplants $\Phi_{\text{mito+oxid}}$ as the dominant term; its functional form is derived from additive logarithmic sensitivities, while its exponent is calibrated against the primate data. Collectively, then, Φ_C is built from derived, calibrated, and phenomenologically inferred channels, and we flag the category of each as it is introduced.

Once Φ_C is fixed, the lifespan prediction follows by inversion,

$$L^{(C)} = \frac{N_0 \Phi_C}{\mathcal{T} f_{H,\text{ref}}}, \quad \mathcal{T} = 525,960 \text{ min yr}^{-1}, \quad (\text{S3})$$

where $f_{H,\text{ref}}$ is the reference cardiac frequency in beats per minute.

S1.2 The four channels

Before applying the decomposition clade by clade, we define each of the four factors of Eq. (S2) in turn, noting in each case whether it is derived from a closed-form expression, calibrated against data, or phenomenologically inferred from independent literature.

Duty-cycle factor (derived). Let an organism partition its life among physiological states k , each occupied for a lifetime fraction q_k ($\sum_k q_k = 1$) with cardiac rate $f_{H,k}$. With a chosen reference state of rate $f_{H,\text{ref}}$, the time-averaged rate is $\bar{f}_H = \sum_k q_k f_{H,k}$, and

$$\kappa \equiv \frac{\bar{f}_H}{f_{H,\text{ref}}} = \sum_k q_k \frac{f_{H,k}}{f_{H,\text{ref}}}, \quad \boxed{\Phi_{\text{duty}} = \kappa^{-1}}. \quad (\text{S4})$$

This is an exact algebraic identity rather than an approximation. The raw observed count $N_{\text{obs}} = \mathcal{T} \bar{f}_H L$ underestimates the damage-equivalent thermodynamic budget by exactly κ , because beats accumulated in suppressed states cost proportionally less entropy; the same logic underlies the activity-dependent economy of physiological work documented in locomotor studies [44]. The corresponding consistency relation is

$$N_{\star}^{(C)} = N_{\text{obs}} \cdot \Phi_{\text{duty}}. \quad (\text{S5})$$

Thermal factor (derived). Damage-generating reactions follow Arrhenius kinetics with activation energy E_a . The damage rate at body temperature T_b relative to the mammalian reference $T_{\text{ref}} = 310 \text{ K}$ is

$$\Phi_{\text{thermal}} = \exp\left[\frac{E_a}{k_B} \left(\frac{1}{T_b} - \frac{1}{T_{\text{ref}}}\right)\right], \quad \frac{E_a}{k_B} = 7543 \text{ K} \quad (E_a = 0.65 \text{ eV}). \quad (\text{S6})$$

A cooler body ($T_b < T_{\text{ref}}$) gives $\Phi_{\text{thermal}} > 1$ (longevity extension); a hotter body ($T_b > T_{\text{ref}}$) gives $\Phi_{\text{thermal}} < 1$ (adverse). The activation energy is taken from the literature rather than fitted, so the channel introduces no free parameter once T_b is known. For $|\Delta T| \lesssim 5 \text{ K}$ a power-law approximation $\Phi_{\text{thermal}} \approx (T_{\text{ref}}/T_b)^\beta$ with $\beta \approx 2\text{--}4$ suffices; for the large temperature drops of bat hibernation ($|\Delta T| \sim 15\text{--}30 \text{ K}$) the exact exponential form is required.

Mitochondrial–antioxidant factor (phenomenologically inferred). $\Phi_{\text{mito+oxid}}$ summarises the reduction in entropy generated per unit ATP turnover arising from tighter electron-transport coupling, lowered proton-leak stoichiometry, and elevated antioxidant and repair capacity. It is inferred from comparative biochemistry—coupling efficiency, reactive-oxygen-species production, and repair capacity—and is *not* derived from the thermodynamic closure; we therefore treat it as a clade-level empirical input.

Hazard factor (phenomenologically inferred). $\Phi_{\text{haz}} = H_{\text{ref}}/H_{\text{ext}}$ rescales the realised lifespan relative to the intrinsic thermodynamic limit. Values $\Phi_{\text{haz}} > 1$ indicate ecological shielding (flight, arboreality, sociality); values $\Phi_{\text{haz}} < 1$ indicate elevated extrinsic mortality. Like $\Phi_{\text{mito+oxid}}$, it is inferred from comparative demography rather than derived from the closure.

We are deliberately explicit that, among the four channels, Φ_{haz} carries the most predictive leverage and the least mechanistic constraint, and we flag it as such wherever it materially moves a prediction. Because it multiplies the entire budget, modest changes shift predicted lifespan substantially: the human estimate moves from 69.8 to 80.3 yr between $\Phi_{\text{haz}} = 1$ and $\Phi_{\text{haz}} = 1.15$, and the bowhead from 119 to 203 yr between $\Phi_{\text{haz}} = 0.35$ and $\Phi_{\text{haz}} = 0.60$. To prevent this freedom

from being used to tune agreement post hoc, we adopt three rules. (i) Each hazard value is set from *independent* demographic information—extrinsic mortality rates, predation exposure, and ecological shielding reported in the comparative-demography literature—and never fitted to the lifespan it is meant to predict. (ii) For the reference clade and for primates in the core calibration we fix $\Phi_{\text{haz}} = 1$, so that the headline multipliers ($\Phi_C = 2.60$ for *Homo*, Table 4) contain *no* hazard adjustment at all; the hazard sensitivity is reported separately as a range. (iii) Where a hazard value is genuinely uncertain (the cetacean pre-industrial vs. post-whaling regimes), we report predictions at both endpoints rather than selecting the one closest to observation. Under these rules the derived channels (Φ_{duty} , Φ_{thermal}) and the calibrated channel (Φ_{neuro}) account for the *shape* of the cross-clade pattern, while Φ_{haz} sets only the residual vertical offset within an independently bounded range.

Table S1 records which channels dominate in each clade and the sign of each contribution.

Table S1: Dominant multiplier channels by clade. +: favourable (> 1); -: adverse (< 1); = 1: absent by definition; ≈ 1 : near unity. The column order follows the factorisation of Eq. (S2), with the primate-specific Φ_{neuro} shown in place of $\Phi_{\text{mito+oxid}}$ where it dominates.

Clade	Φ_{duty}	Φ_{thermal}	Φ_{neuro}	$\Phi_{\text{mito+oxid}}$	Primary driver
Non-primate placentals	= 1	= 1	= 1	= 1	Reference
Primates	= 1	+	++	—	Neural entropy reduction
Marsupials/monotremes	= 1	≈ 1	—	≈ 1	No net displacement
Bats	+	++	—	≈ 1	Torpor + hypothermia
Birds	—	—	—	++	Biochemical efficiency
Cetaceans	++	+	—	≈ 1	Bradycardic pacing
Reptiles (corr.)	≈ 1	+	—	≈ 1	Thermal limit
Amphibians (corr.)	≈ 1	+	—	≈ 1	Thermal limit

S1.3 Non-primate placental mammals: the reference

The non-primate placental clade defines the reference and therefore fixes both the baseline cycle count N_0 and the baseline entropy cost per beat $\langle \sigma_{\text{beat}} \rangle_0$. Using the mammalian resting allometries with $T_{\text{ref}} = 310 \text{ K}$, the cardiac baseline is

$$\ell_0 = \log_{10} N_0 = 8.995 \pm 0.160 \quad (n = 46), \quad N_0 \approx 1.0 \times 10^9 \text{ beats.} \quad (\text{S7})$$

A reference mammal occupies a single physiological state for essentially all of adult life, so $q_1 = 1$, $f_{H,1} = f_{H,\text{ref}}$, and $\kappa = 1$. At the reference body temperature $T_b = T_{\text{ref}}$ the Arrhenius exponent vanishes, and with no biochemical or ecological offset relative to itself, every channel equals unity by construction:

$$\Phi_{\text{duty}} = \Phi_{\text{thermal}} = \Phi_{\text{mito+oxid}} = \Phi_{\text{haz}} = 1, \quad \Phi_C = 1. \quad (\text{S8})$$

This is the anchor against which all other clades are measured. It asserts that for an unmodified mammal the raw observed heartbeat count equals the damage-equivalent budget, $N_{\text{obs}} = N_0 \approx 10^9$, recovering the classical lifetime-heartbeat invariant as the $\Phi_C = 1$ limit of the framework.

S1.4 Primates: neural investment

With the reference clade now fixed, we turn to the first systematic departure from it. Primates carry $\langle N_{\star} \rangle_{\text{prim}} \approx (2-3) \times 10^9$, elevated by $\Delta \ell = +0.381$ dex above the baseline. From Eq. (S1) this

requires $\langle \sigma_{\text{beat}} \rangle_{\text{prim}} \approx \langle \sigma_{\text{beat}} \rangle_0 / (2-3)$: primate cardiac cycles produce less entropy on average than those of non-primate mammals of comparable mass. The reduction is controlled by the neural power fraction $\varphi \equiv P_{\text{brain}}/P_{\text{body}}$ [26], which rises from $\varphi_0 \approx 0.02$ in non-primate placentals to $\varphi \approx 0.06-0.20$ in primates. Three coupled channels link elevated φ to reduced entropy per beat: predictive homeostatic regulation [27], enhanced cellular repair and damage clearance, and behavioural risk buffering. Each lowers $\langle \sigma_{\text{beat}} \rangle$ monotonically with φ , motivating a power-law response.

To formalise this, define the local logarithmic sensitivity of entropy per beat to neural fraction at the baseline,

$$\alpha \equiv - \left. \frac{\partial \ln \langle \sigma_{\text{beat}} \rangle}{\partial \ln \varphi} \right|_{\varphi=\varphi_0} > 0. \tag{S9}$$

When several independent channels act multiplicatively their logarithmic sensitivities add, $\alpha = \sum_j \alpha_j$. The second law requires $0 < \alpha < 1$: were $\alpha \geq 1$, each unit of neural energy would return more than one unit of entropy savings in peripheral tissues, an over-compensation that is thermodynamically forbidden. Assuming a scale-free response over the primate range $\varphi \in [\varphi_0, 10\varphi_0]$ and integrating Eq. (S9) yields

$$\langle \sigma_{\text{beat}}(\varphi) \rangle = \langle \sigma_{\text{beat}} \rangle_0 \left(\frac{\varphi}{\varphi_0} \right)^{-\alpha} \implies \Phi_{\text{neuro}}(\varphi) = \left(\frac{\varphi}{\varphi_0} \right)^\alpha. \tag{S10}$$

The functional form of Φ_{neuro} is thus *derived* from the additive-sensitivity argument, but the exponent α is *calibrated* against the primate deviation rather than obtained from first principles; we are careful to maintain this distinction below. Because primates are single-state ($\Phi_{\text{duty}} = 1$), the full primate time-equivalence law reads

$$N_\star^{(\text{prim})} = N_0 \underbrace{\left(\frac{\varphi}{\varphi_0} \right)^\alpha}_{\Phi_{\text{neuro}}} \underbrace{\left(\frac{T_{\text{ref}}}{T_b} \right)^\beta}_{\Phi_{\text{thermal}}} \underbrace{\left(\frac{H_{\text{ref}}}{H_{\text{ext}}} \right)}_{\Phi_{\text{haz}}}, \quad \beta \approx 3. \tag{S11}$$

For *Homo sapiens* ($\varphi = 0.20$, $T_b = 306.5$ K, $(\alpha, \beta) = (0.40, 3)$, $f_H = 70$ bpm), the single-state condition gives $\kappa = 1$ and hence $\Phi_{\text{duty}} = 1$ with $\bar{f}_H = 70$ bpm. The neuro-metabolic factor is $\Phi_{\text{neuro}} = (0.20/0.02)^{0.40} = 10^{0.40} \approx 2.512$. With $\Delta T = 3.5$ K in the power-law regime, the thermal factor is $\Phi_{\text{thermal}} = (310/306.5)^3 \approx 1.035$. Combining,

$$\Phi_C^{(\text{human})} = \underbrace{1.000}_{\Phi_{\text{duty}}} \times \underbrace{2.512}_{\Phi_{\text{neuro}}} \times \underbrace{1.035}_{\Phi_{\text{thermal}}} \times \underbrace{1.000}_{\Phi_{\text{haz}}} = 2.60, \quad N_\star^{(\text{human})} = \Phi_C^{(\text{human})} N_{H,0}^{(\text{emp})} = 2.60 \times 9.9 \times 10^8 \approx 2.57 \times 10^9. \tag{S12}$$

The predicted lifespan is $L_{\text{pred}} = 2.57 \times 10^9 / (525,960 \times 70) \approx 69.8$ yr; a moderate hazard factor $\Phi_{\text{haz}} = 1.15$ for modern low-mortality populations raises this to ≈ 80.3 yr, consistent with high-income life expectancy. (Using the round shorthand $N_0 = 10^9$ in place of the empirical anchor would give 2.60×10^9 and 70.6 yr; the $\approx 1\%$ difference is immaterial to the comparison with observation.) Because $\Phi_{\text{duty}} = 1$, the consistency relation Eq. (S5) requires $N_\star^{(\text{human})} = N_{\text{obs}}$ exactly, which is satisfied: $N_{\text{obs}} = 525,960 \times 70 \times 69.8 = 2.57 \times 10^9$. The neuro channel accounts for 96.6% of the multiplier and the thermal channel for 3.4%, while the duty channel is identically unity.

Calibrated parameters (OLS on log-transformed variables, $\ln N_0$ constrained to 20.72) are $\alpha \approx 0.35-0.45$ (95% CI [0.28, 0.52]) and $\beta \approx 3$ (95% CI [1.5, 5.0]). The exponent α is calibrated from the primate deviation rather than derived purely from first principles; the constraint $0 < \alpha < 1$ and the three-channel mechanism are independently motivated, but the precise value of α requires the calibration data. The mechanism is derived; the exponent is empirical. Table S2 tests Eq. (S11) across the primate mass range.

Table S2: Primate lifespan predictions. Every row is generated directly from $\Phi_{\text{neuro}} = (\varphi/\varphi_0)^\alpha$ and $\Phi_{\text{thermal}} = (T_{\text{ref}}/T_b)^\beta$ with $\varphi_0 = 0.02$, $T_{\text{ref}} = 310\text{ K}$, $\beta = 3$, the empirical anchor $N_0 = N_{H,0}^{(\text{emp})} = 10^{8.995}$ beats, and $\Phi_{\text{duty}} = \Phi_{\text{haz}} = 1$ for all species; the values are therefore reproducible by inspection. (a) Core calibration $\alpha = 0.40$. (b) Extended calibration $\alpha = 0.45$.

Species	f_H (bpm)	φ	T_b (K)	Φ_{duty}	Φ_{neuro}	Φ_{thermal}	L_{pred} (yr)	L_{obs} (yr)
<i>(a) Core calibration, $\alpha = 0.40$</i>								
<i>Macaca mulatta</i>	120	0.07	309.0	1.00	1.65	1.010	26.1	25–30
<i>Pan troglodytes</i>	75	0.12	307.0	1.00	2.05	1.030	52.8	45–55
<i>Homo sapiens</i>	70	0.20	306.5	1.00	2.51	1.035	69.8	70–85
<i>(b) Extended calibration, $\alpha = 0.45$</i>								
<i>Callithrix jacchus</i>	220	0.06	309.5	1.00	1.64	1.005	14.1	10–15
<i>Macaca mulatta</i>	120	0.07	309.0	1.00	1.76	1.010	27.8	25–30
<i>Pan troglodytes</i>	75	0.12	307.0	1.00	2.24	1.030	57.8	45–55
<i>Gorilla gorilla</i>	65	0.09	307.0	1.00	1.97	1.030	58.6	40–55
<i>Homo sapiens</i>	70	0.20	306.5	1.00	2.82	1.035	78.3	70–85

S1.5 Marsupials and monotremes: the non-placental controls

Where primates displace the baseline through an active reduction in entropy cost, it is equally instructive to examine a clade expected to displace it not at all. The non-placental mammals—marsupials and monotremes—provide an essential control on the framework, because PBTE predicts that a clade which deploys none of the four displacing mechanisms should remain at the mammalian baseline. These animals are predominantly single-state endotherms maintained at body temperatures only modestly below the placental reference ($T_b \approx 305\text{--}309\text{ K}$), with resting heart rates that follow the standard mammalian allometry and with no systematic biochemical or ecological offset [20]. Each channel is therefore expected to lie at or very near unity: $\Phi_{\text{duty}} \approx 1$ (single-state pacing), $\Phi_{\text{thermal}} \approx 1.0\text{--}1.2$ (a slight cool-body Arrhenius credit), and $\Phi_{\text{mito+oxid}}, \Phi_{\text{haz}} \approx 1$. The net multiplier is accordingly

$$\Phi_C^{(\text{marsupial})} \approx 1, \tag{S13}$$

with no channel acting strongly in either direction.

This expectation is borne out by the data. The observed clade mean is $\bar{\ell} = 8.933 \pm 0.204$ ($n = 19$), a displacement of only $\Delta\ell = -0.062$ from the placental baseline, corresponding to $\Phi_{\text{obs}} = 10^{-0.062} = 0.87$. The small negative offset lies within one standard deviation of the baseline and is not statistically distinguishable from $\Phi_C = 1$; the modestly lower body temperatures of this clade would, if anything, predict Φ_{thermal} slightly above unity. The marsupials and monotremes thus function as a negative control: a mammalian clade that neither suppresses nor accelerates its cardiac clock, neither cools nor heats appreciably, and invests in no special biochemistry, lands on the baseline as required. Their agreement with $\Phi_C \approx 1$ provides evidence that the elevated multipliers of primates, bats, and birds reflect genuine physiological strategies rather than an artefact of the reference calibration.

S1.6 Bats: torpor as biological time dilation

Having established that a clade deploying none of the four mechanisms remains at the baseline, we turn to the clade that engages them most dramatically. Temperate vespertilionid bats reach wild maxima of 20–40 yr, three to six times the allometric prediction for a non-hibernating placental of equal mass [28]. Unlike primates, bats exploit two synergistic channels that act simultaneously during the hibernation season: a torpor duty cycle that lowers the time-averaged cardiac clock, and a hypothermic Arrhenius factor that lowers the entropy cost of each beat during the cold torpor bout. With torpor fraction q , active-phase rate $f_{H,act}$, and torpid rate $f_{H,tor}$, the duty factor follows directly from Eq. (S4):

$$\bar{f}_H = (1 - q)f_{H,act} + q f_{H,tor}, \quad \kappa = (1 - q) + q \frac{f_{H,tor}}{f_{H,act}}, \quad \Phi_{duty} = \kappa^{-1}. \quad (S14)$$

For $q \in [0.40, 0.60]$ and $f_{H,tor}/f_{H,act} \approx 0.03\text{--}0.07$, this gives $\kappa \approx 0.41\text{--}0.62$ and $\Phi_{duty} \approx 1.6\text{--}2.4$. Because hibernation temperatures (280–295 K) lie 15–30 K below normothermy, the exact Arrhenius form must be used,

$$\Phi_{thermal} = \exp\left[\frac{E_a}{k_B}\left(\frac{1}{T_{tor}} - \frac{1}{T_{ref}}\right)\right], \quad (S15)$$

and since secondary biochemistry is near baseline in temperate vespertilionids ($\Phi_{mito+oxid} \approx 1$), the bat multiplier reduces to $\Phi_{bat} = \Phi_{duty} \cdot \Phi_{thermal} \cdot \Phi_{haz}$.

For *Myotis lucifugus* ($q = 0.50$, $f_{H,act} = 300$ bpm, $f_{H,tor} = 10$ bpm, $T_{tor} = 293$ K), the duty factor is $\kappa = 0.50 + 0.50 \times (10/300) = 0.5167$, so $\Phi_{duty} = 1.935$ and $\bar{f}_H = 300/1.935 = 155$ bpm. The thermal factor, with $1/293\text{--}1/310 = 1.872 \times 10^{-4} \text{ K}^{-1}$, is $\Phi_{thermal} = e^{7543 \times 1.872 \times 10^{-4}} = e^{1.412} \approx 4.10$. The intrinsic multiplier ($\Phi_{haz} = 1$) is therefore $\Phi_{bat} = 1.935 \times 4.10 = 7.93$, giving an intrinsic lifespan $L_{pred} = 10^9 / (525,960 \times 300) \times 7.93 = 6.34 \text{ yr} \times 7.93 \approx 50.3 \text{ yr}$. Applying $\Phi_{haz} = 0.68$ for predation and habitat variability brings the prediction to ≈ 34 yr, matching the observed wild maximum. The consistency check confirms internal agreement: for $L = 34$ yr and $\bar{f}_H = 155$ bpm, $N_{obs} = 525,960 \times 155 \times 34 = 2.770 \times 10^9$, hence $N_{*}^{(bat)} = N_{obs} \times \Phi_{duty} = 5.36 \times 10^9$, which matches $N_0 \Phi_{bat} \Phi_{haz} = 5.39 \times 10^9$ to within 0.6%. The thermal factor supplies 52% and the duty factor 24% of the intrinsic multiplier; neither alone explains the longevity excess. The observed clade mean ($\bar{\ell} = 9.540 \pm 0.163$, $n = 31$, $\Delta\ell = +0.545$) confirms the predicted elevation. Table S3 collects representative bats.

Table S3: Predicted multipliers and longevity for representative bat species. $\Phi_{thermal}$ from the exact Arrhenius formula ($E_a = 0.65$ eV, $T_{ref} = 310$ K) using the torpor-phase T_b ; $\Phi_{bat} = \Phi_{duty} \times \Phi_{thermal}$ (intrinsic, $\Phi_{haz} = 1$).

Species	q	$f_{H,act}$ (bpm)	$f_{H,tor}$ (bpm)	T_{tor} (K)	Φ_{duty}	$\Phi_{thermal}$	$L_{max,obs}$ (yr)
Temperate vespertilionid (range)	0.40–0.60	250–350	5–20	280–295	1.6–2.5	3.0–5.0	20–40
<i>Myotis lucifugus</i>	0.50	300	10	293	1.935	4.10	34
<i>Eptesicus fuscus</i>	0.45	280	12	291	1.79	4.54	19
<i>Pteropus vampyrus</i> (min. torpor)	0.10	250	60	303	1.07	1.22	15–23

S1.7 Birds: efficient dissipation overcoming adverse temperature

The bat strategy extends life by making the favourable channels do the work. Birds present the opposite configuration, achieving longevity in spite of channels that act against them. Birds present

an apparent paradox: heart rates comparable to mass-matched mammals, core temperatures 3–5 K above the mammalian reference, yet a 20 g passerine outlives a 20 g mouse by an order of magnitude. Here both the thermal factor and the flight duty-cycle factor are adverse (< 1); avian longevity arises because a dominant biochemical factor $\Phi_{\text{mito+oxid}} \gg 1$ overcomes them. For a passerine at $T_b = 314\text{ K}$ the Arrhenius exponent is negative, with $1/314 - 1/310 = -4.11 \times 10^{-5} \text{ K}^{-1}$, so

$$\Phi_{\text{thermal}}^{(\text{bird})} = e^{7543 \times (-4.11 \times 10^{-5})} = e^{-0.310} \approx 0.733. \tag{S16}$$

Flight raises heart rate by a factor of about 2.5; with flight fraction p_f ,

$$\kappa = 1 + p_f \left(\frac{f_{H,\text{flight}}}{f_{H,\text{rest}}} - 1 \right) \approx 1 + 1.5 p_f, \quad \Phi_{\text{duty}} = \kappa^{-1}, \tag{S17}$$

so $p_f = 0.10$ yields $\kappa = 1.15$ and $\Phi_{\text{duty}} = 0.870$ (adverse). The favourable channel is biochemical: avian mitochondria produce far less reactive oxygen species per unit ATP than mammalian mitochondria [29, 19, 30], even as avian thermoregulation sustains the elevated body temperatures that make the thermal channel adverse [17]. Pigeon heart mitochondria generate roughly 5–10 times less superoxide per oxygen consumed than rat at the same metabolic rate [29]; expressed as a coupling-efficiency ratio of about 1.20 with quadratic damage sensitivity ($\gamma \approx 2$), this gives $\Phi_{\text{mito}} = (1.20)^2 \approx 1.44$. Long-lived birds further show two- to three-fold greater oxidative-damage resistance than mammals of comparable metabolic rate [31]; with the log-linear exponent $\delta \approx 0.7$ [19], $\Phi_{\text{oxid}} = (2.0)^{0.7} \approx 1.62$, so the combined phenomenologically inferred factor is $\Phi_{\text{mito+oxid}} = 1.44 \times 1.62 \approx 2.33$.

For a generic 20 g passerine ($f_{H,\text{rest}} = 320 \text{ bpm}$, $T_b = 314 \text{ K}$, $p_f = 0.10$, $\Phi_{\text{haz}} = 2.0$ from flight and arboreal shielding), the multiplier is

$$\Phi_{\text{bird}} = \underbrace{0.870}_{\Phi_{\text{duty}}} \times \underbrace{0.733}_{\Phi_{\text{thermal}}} \times \underbrace{2.33}_{\Phi_{\text{mito+oxid}}} \times \underbrace{2.0}_{\Phi_{\text{haz}}} = 0.638 \times 4.66 \approx 2.97. \tag{S18}$$

The predicted lifespan is $L_{\text{pred}} = 10^9 / (525,960 \times 320) \times 2.97 = 5.94 \text{ yr} \times 2.97 \approx 17.6 \text{ yr}$, within the observed range of 10–20 yr for small passerines. The consistency check holds: with $\bar{f}_H = 320/0.870 = 368 \text{ bpm}$ and $L = 17.6 \text{ yr}$, $N_{\text{obs}} = 525,960 \times 368 \times 17.6 = 3.408 \times 10^9$, so $N_{\star}^{(\text{bird})} = N_{\text{obs}} \times \Phi_{\text{duty}} = 2.965 \times 10^9$, matching $N_0 \Phi_{\text{bird}} = 2.97 \times 10^9$ to 0.2%. The two adverse factors multiply to 0.638, a net 36% reduction in the effective budget, which the biochemical and hazard channels ($\times 2.33$ and $\times 2.0$) more than recover. The clade mean ($\bar{\ell} = 9.528 \pm 0.213$, $n = 78$, $\Delta\ell = +0.533$) confirms the predicted elevation; the modest gap between the generic-passerine prediction and the clade mean reflects the strong upward pull of long-lived large non-passerines (parrots, owls, albatrosses) in the full sample. Table S4 collects representative birds.

Table S4: Predicted multipliers and longevity for representative bird species. Φ_{thermal} from the exact Arrhenius formula; Φ_{duty} from Eq. (S17) with $f_{H,\text{flight}}/f_{H,\text{rest}} = 2.5$. Both Φ_{duty} and Φ_{thermal} are adverse (< 1) for all entries.

Species	$f_{H,\text{rest}}$ (bpm)	T_b (K)	p_f	Φ_{duty}	Φ_{thermal}	$\Phi_{\text{mito+oxid}}$	Φ_{haz}	$L_{\text{max,obs}}$ (yr)
Passerine (generic, 20 g)	320	314	0.10	0.87	0.733	2.33	2.0	10–20
<i>Larus argentatus</i>	200	313	0.15	0.84	0.770	2.80	2.5	30
<i>Diomedea exulans</i>	100	312	0.25	0.81	0.810	3.50	4.0	50–60
<i>Aquila chrysaetos</i>	150	313	0.12	0.85	0.770	3.00	3.5	30–40

S1.8 Cetaceans: bradycardic pacing

If the avian route to longevity is biochemical, the cetacean route is once again one of pacing, though achieved by a mechanism distinct from that of bats. Large baleen cetaceans reach century-scale lifespans through extreme diving bradycardia rather than metabolic suppression. Blue-whale heart rates as low as 2–4 bpm have been recorded during deep foraging dives [32, 33], against surface rates of 25–37 bpm. With surface rate $f_{H,\text{surf}}$ as reference, dive rate $f_{H,\text{dive}}$, and dive fraction p_d , the duty factor is

$$\bar{f}_H = (1 - p_d)f_{H,\text{surf}} + p_d f_{H,\text{dive}}, \quad \kappa = (1 - p_d) + p_d \frac{f_{H,\text{dive}}}{f_{H,\text{surf}}}, \quad \Phi_{\text{duty}} = \kappa^{-1}. \quad (\text{S19})$$

A secondary thermal factor accounts for core temperatures 1–4 K below reference, and an oxygen-buffering sub-factor Φ_{O_2} accounts for elevated myoglobin limiting reperfusion ROS bursts on surfacing [34], consistent with the cardiorespiratory control observed in diving cetaceans [24], so that $\Phi_{\text{whale}} = \Phi_{\text{duty}} \cdot \Phi_{\text{thermal}} \cdot \Phi_{O_2} \cdot \Phi_{\text{haz}}$. Unlike bat hibernation, the cardiac suppression is a continuous reflex maintained throughout adult life with no associated hypothermia.

For the bowhead *Balaena mysticetus* ($f_{H,\text{surf}} = 30$ bpm, $f_{H,\text{dive}} = 3$ bpm, $p_d = 0.75$, $T_b = 308$ K, $\Phi_{O_2} = 1.50$, $\Phi_{\text{haz}} = 0.60$ for pre-industrial conditions), the duty factor is $\kappa = 0.25 + 0.75 \times (3/30) = 0.325$, so $\Phi_{\text{duty}} = 3.077$ and $\bar{f}_H = 30/3.077 = 9.75$ bpm. The thermal factor, with $1/308 - 1/310 = 2.10 \times 10^{-5} \text{ K}^{-1}$, is $\Phi_{\text{thermal}} = e^{7543 \times 2.10 \times 10^{-5}} = e^{0.158} \approx 1.171$. Combining, $\Phi_{\text{whale}} = 3.077 \times 1.171 \times 1.500 \times 0.600 \approx 3.24$, giving $L_{\text{pred}} = N_{H,0}^{(\text{emp})} / (525,960 \times 30) \times 3.24 = 62.7 \text{ yr} \times 3.24 \approx 203 \text{ yr}$, at the upper end of the documented ~ 200 yr maximum. These are exactly the values reported for the bowhead in the main-text Table 7. A more conservative hazard estimate reflecting post-whaling mortality, $\Phi_{\text{haz}} = 0.35$, would instead give $\Phi_{\text{whale}} \approx 1.89$ and $L_{\text{pred}} \approx 119$ yr; the hazard channel is thus the single largest source of spread in the cetacean prediction, and we flag it as a phenomenologically inferred input rather than a derived one.

The cetacean case illustrates a measurement trap. For $L = 150$ yr and $\bar{f}_H = 9.75$ bpm, the raw count is $N_{\text{obs}} = 525,960 \times 9.75 \times 150 = 7.69 \times 10^8 \approx 0.77 \times 10^9$, so $N_{\star}^{(\text{whale})} = N_{\text{obs}} \times \Phi_{\text{duty}} = 2.37 \times 10^9$. The proximity of N_{obs} to N_0 has misled some analyses into treating large whales as obeying the mammalian baseline, which is incorrect: the damage-equivalent budget $N_{\star}^{(\text{whale})}$ greatly exceeds N_0 . The raw count is small precisely because most of the whale’s life is spent in deeply bradycardic states where each beat costs far less entropy, and the duty-cycle factor restores the correct budget. This is also why the observed clade mean appears low ($\bar{\ell} = 8.801 \pm 0.296$, $n = 12$, $\Delta\ell = -0.194$): the dive correction has already been folded into f_H^{eff} , so the raw surface count without correction would place large mysticetes well below the baseline. Table S5 collects representative cetaceans.

Table S5: Predicted multipliers and longevity for representative cetacean species. Φ_{thermal} from the exact Arrhenius formula ($E_a = 0.65$ eV, $T_{\text{ref}} = 310$ K). Φ_{haz} reflects pre-industrial conditions.

Species	$f_{H,\text{surf}}$ (bpm)	p_d	Φ_{duty}	T_b (K)	Φ_{thermal}	Φ_{O_2}	Φ_{haz}	L_{obs} (yr)
<i>Balaenoptera musculus</i> (blue)	37	0.70	2.70	308	1.17	1.4	0.50	80–90
<i>Balaena mysticetus</i> (bowhead)	30	0.75	3.08	308	1.17	1.5	0.60	150–200
<i>Physeter macrocephalus</i> (sperm)	40	0.65	2.50	307	1.24	1.6	0.55	60–70
<i>Tursiops truncatus</i> (bottlenose)	80	0.40	1.50	309	1.09	1.2	0.65	40–50

S1.9 Reptiles and amphibians: the thermal limit

The five endotherm clades examined so far each engage several channels at once. The ectotherms complete the survey as the opposite extreme. The ectotherms enter the framework as a limiting case in which the displacement from the mammalian baseline is carried almost entirely by the thermal channel. A reptile or amphibian does not maintain a fixed elevated body temperature; its damage kinetics run at the mean field temperature T_b set by habitat and behaviour, typically well below the mammalian reference. Setting $\Phi_{\text{duty}} \approx 1$ (no systematic cardiac duty cycling) and treating mitochondrial and ecological offsets as near-baseline, the multiplier collapses to the Arrhenius factor evaluated at T_b ,

$$\Phi_C^{(\text{ecto})} \approx \Phi_{\text{thermal}} = \exp\left[\frac{E_a}{k_B}\left(\frac{1}{T_b} - \frac{1}{T_{\text{ref}}}\right)\right], \tag{S20}$$

which, equivalently, is applied as a correction to the raw lifetime count so that all clades are compared at a common reference temperature,

$$\ell_{\text{corr}} = \ell_{\text{obs}} + \frac{E_a}{k_B \ln 10} \left(\frac{1}{T_b} - \frac{1}{T_{\text{ref}}}\right), \tag{S21}$$

with $E_a = 0.65$ eV and $T_{\text{ref}} = 310$ K [39, 38].

Applying this correction shifts the reptile clade mean from the raw $\bar{\ell}^{\text{raw}} = 8.615 \pm 0.290$ to $\bar{\ell}^{\text{corr}} = 8.929 \pm 0.301$ ($n = 17$), removing approximately 75% of the raw gap from the mammalian baseline and leaving a residual displacement of only $\Delta\ell = -0.065$. The amphibian clade shifts from $\bar{\ell}^{\text{raw}} = 8.448 \pm 0.127$ to $\bar{\ell}^{\text{corr}} = 8.822 \pm 0.146$ ($n = 9$), leaving $\Delta\ell = -0.173$. In both clades the corrected mean approaches but does not quite reach the mammalian value; the residual 0.07–0.17 dex is attributable to unmodelled ectotherm-specific physiology and to imperfect knowledge of the mean field temperature, which enters Eq. (S20) exponentially. The persistence of a small residual for all plausible values of E_a in the range 0.40–0.90 eV indicates that the thermal correction alone does not fully close the gap, consistent with ectotherms occupying a Φ_{thermal} -dominated limit of the same framework rather than requiring a separate construction. Table S6 reports the clade-level corrections.

Table S6: Arrhenius thermal correction for the ectotherm clades. $\bar{\ell}^{\text{raw}}$ is the uncorrected clade mean; $\bar{\ell}^{\text{corr}}$ is corrected to $T_{\text{ref}} = 310$ K via Eq. (S21) with $E_a = 0.65$ eV. $\Delta\ell^{\text{corr}}$ is the residual deviation from the placental baseline $\bar{\ell}_0 = 8.995$.

Clade	n	$\bar{\ell}^{\text{raw}}$	$\bar{\ell}^{\text{corr}}$	$\Delta\ell^{\text{corr}}$	gap removed
Reptiles	17	8.615 ± 0.290	8.929 ± 0.301	-0.065	$\sim 75\%$
Amphibians	9	8.448 ± 0.127	8.822 ± 0.146	-0.173	$\sim 70\%$

S1.10 Synthesis: distinct mechanisms, one invariant

Table S7 places all clades side by side with the numerical values from the worked examples. The endotherm strategies form two natural pairs. Primates and birds are both single-state in the duty-cycle sense yet are mirror images of each other: primates raise the budget through $\Phi_{\text{neuro}} \gg 1$ while holding $\Phi_{\text{duty}} = 1$ and enjoying a small thermal credit, whereas birds overcome two adverse factors ($\Phi_{\text{duty}} < 1$, $\Phi_{\text{thermal}} < 1$) through a dominant biochemical factor. Bats and cetaceans both exploit $\Phi_{\text{duty}} > 1$, but bats combine it with hypothermic Arrhenius suppression during seasonal torpor while cetaceans rely on a continuous isothermal dive bradycardia. The marsupials and monotremes

deploy none of these mechanisms and accordingly remain at the baseline, serving as a negative control, while the ectotherms occupy the opposite extreme in which the thermal channel alone accounts for nearly all of the displacement.

Table S7: Summary of the clade multipliers. Numerical values correspond to the worked representative species; ectotherm entries are clade-mean thermal corrections. Direction: + favourable, - adverse, = 1 absent, ≈ 1 near unity. Effective cycle budgets as multiples of $N_0 = 10^9$.

Clade	Φ_{duty}	Φ_{thermal}	Φ_{neuro}	$\Phi_{\text{mito+oxid}}$	Φ_{haz}	Φ_C	Primary driver
Non-primate placentals	1.00 (= 1)	1.00 (= 1)	1.00 (= 1)	1.00 (= 1)	1.00	1.00	Reference
Primates (<i>H. sapiens</i>)	1.00 (= 1)	1.04 (+)	2.51 (++)	—	1.00	2.60	Neural entropy reduction
Marsupials/monotremes	1.00 (= 1)	≈ 1	—	≈ 1	≈ 1	0.87	No net displacement
Bats (<i>M. lucifugus</i>)	1.94 (+)	4.10 (++)	—	≈ 1	0.68	5.39	Torpor + hypothermia
Birds (20 g passerine)	0.87 (-)	0.73 (-)	—	2.33 (++)	2.00	2.97	Biochemical efficiency
Cetaceans (<i>B. mysticetus</i>)	3.08 (++)	1.17 (+)	—	≈ 1	0.35	1.76	Bradycardic pacing
Reptiles (corrected)	≈ 1	+	—	≈ 1	≈ 1	0.86	Thermal limit
Amphibians (corrected)	≈ 1	+	—	≈ 1	≈ 1	0.67	Thermal limit

Despite this mechanistic diversity, the effective damage-equivalent budgets of all clades converge within one order of magnitude of $N_0 = 10^9$, confirming the central prediction of the framework. The raw observed counts N_{obs} vary far more widely, but that variation is fully accounted for by the duty-cycle factor through Eq. (S5). The unifying conclusion is that longevity is not won by escaping the finite lifetime entropy budget but by spending it more slowly per unit of intrinsic biological time: primates purchase chronological time with neural precision, bats with thermal suspension, birds with biochemical excellence, and cetaceans with cardiac restraint, while marsupials and ectotherms display the limiting behaviours of no displacement and pure thermal displacement, respectively. In every case the price is the same Σ_* units of irreversible dissipation, paid over a chronological span set entirely by how efficiently that budget is spent.

S2 Complete cardiac dataset

The following tables contain the complete 230-species cardiac dataset of adult vertebrates used in the analyses of this paper. All ℓ values are computed as $\ell = \log_{10}(f_H^{\text{avg}} \times L \times 525,960)$ directly from the f_H^{avg} and L columns of each row and have been verified for internal consistency. This is the cardiac table; the respiratory analysis of Section 4.2 draws on the 65-species subset of these taxa for which a reliable resting breath rate f_R is also available, listing f_R alongside f_H in the machine-readable file. The complete table (cardiac, with the respiratory subset flagged) is provided in machine-readable form as Supplementary Data File 1 (a tab-delimited file with one row per species and the column schema defined below), so that every numerical value used in the paper can be traced to its source without recourse to private correspondence.

Column definitions and data-transparency notes

Dataset location. All species values are listed in Extended Data Tables 1–8 below and are reproduced in full in Supplementary Data File 1 (columns: `species`, `clade`, M , f_H^{avg} , T , L , ℓ , `fH_type`, `fH_context`, `L_context`, `source`, `correction`). The `fH_type`, `fH_context`, and `source` columns record, for each individual species, whether the heart rate was directly measured, allometrically

imputed, or corrected, together with the primary reference; the tables below summarise these provenance categories.

Heart-rate type: measured vs. inferred. The *Source* and *Corr.* columns in each table distinguish:

- **Measured:** directly measured resting heart rate from a published study (flagged in the source column). These constitute the large majority of entries for non-primate placentals, primates, marsupials, and birds.
- **Imputed ([†]):** allometrically estimated from $f_H = 241 M^{-0.25}$ bpm [4], used only where no published resting measurement exists. In the non-primate placental clade this applies to exactly three species (*Rhinoceros unicornis*, *Dugong dugon*, *Orycteropus afer*); imputed entries are flagged with [†] in every table so the reader can identify them at a glance.
- **Duty-corrected (bats):** active-phase measured rate multiplied by the duty-cycle factor κ to give the time-averaged f_H^{avg} (see Section 2); the active-phase measurement and the value of q are both tabulated, so the correction is fully reconstructible.
- **Dive-corrected (cetaceans):** surface measured rate combined with the bradycardic dive rate weighted by the dive fraction p_d (see the cetacean worked example); the surface rate and p_d are both tabulated.
- **Arrhenius-corrected (ectotherms):** field active rate corrected to $T_{\text{ref}} = 310\text{K}$ using the Gillooly et al. [39] Arrhenius equation; both the raw and corrected values are tabulated.

Heart-rate measurement context.

- Non-primate placentals, primates, marsupials, and birds: resting rates from laboratory or captive studies as recorded in AnAge build 15 [35] and PanTHERIA [36], with Calder (1984) [4] for classical species. These are predominantly lab-measured resting rates. We explicitly acknowledge that lab resting rates may differ from field resting rates; this is a known limitation of comparative heart-rate data.
- Bats: active-phase resting rate from lab or flight-cage studies, corrected for torpor duty cycle [45].
- Cetaceans: surface inter-breath heart rate from free-diving field telemetry [32], corrected for dive bradycardia [48].
- Ectotherms: field active rates corrected to standard temperature via the Arrhenius equation [39, 38].

Lifespan definition. L is the maximum recorded lifespan as curated in AnAge build 15 [35]. AnAge records the single longest verified individual lifespan regardless of whether it was wild or captive; for most small mammals the record holder is a captive individual, whereas for bats and large mammals (whales, elephants) the record is from a wild or semi-wild individual. All species in this dataset have AnAge confidence ratings of *acceptable* or *high*. Mean lifespan is not used anywhere in this paper; only maximum recorded lifespan enters the PBTE invariant ℓ .

Column definitions. *Species*: binomial name per IUCN or Reptile Database taxonomy. M : adult body mass (kg). f_H (bpm): resting/active heart rate as defined above; the value used in the ℓ computation is f_H^{avg} . T (K): core body (endotherms) or field (ectotherms) temperature. L (yr): maximum recorded lifespan. ℓ : PBTE invariant = $\log_{10}(f_H^{\text{avg}} \times L \times 525,960)$, computed directly from f_H^{avg} and L in each row (all values verified internally consistent).

Source codes (primary reference for f_H and L).

- **A** = AnAge build 15 [35] — <https://genomics.senescence.info/species/>
- **P** = PanTHERIA v1.0 [36]
- **C** = Calder (1984) [4] — species-level data in Tables 2–3 of that monograph
- **Pr** = Prinzing et al. (1991) [46] — avian heart-rate compilation
- **L** = Lyman et al. (1982) [45] — torpor physiology
- **Ch** = Christian & Weavers (1999) [38] — ectotherm field physiology
- **U** = Uetz et al. (2023), The Reptile Database — <https://reptile-database.reptarium.cz>
- **G** = Goldbogen et al. (2019) [32] — cetacean dive telemetry

Correction codes (Corr. column). --- = none applied; HR = heart-rate value corrected from a compendium error; TA = torpor-cycle (duty-cycle) time-average; DA = dive-cycle time-average; AQ = Arrhenius correction to $T_{ref} = 310$ K.

Directly measured resting values are used for all non-primate placentals, primates, marsupials, and birds. The three non-primate placental species with no published resting measurement (*Rhinoceros unicornis*, *Dugong dugon*, *Orycteropus afer*) have heart rates imputed from $f_H = 241 M^{-0.25}$ bpm [4] and are flagged with †. For bats and cetaceans, f_H^{avg} is the duty-cycle- and dive-corrected time-average, respectively; for ectotherms, f_H^{corr} (equivalently f_H^{avg}) is the Arrhenius-corrected value.

Extended Data Table 1 | Non-primate placental mammals ($n = 46$)

Extended Data Table 2 | Primates ($n = 18$)

Species	M (kg)	f_H (bpm)	T (K)	L (yr)	ℓ	φ	Source	Corr.
<i>Callithrix jacchus</i>	0.35	220	309.5	16.5	9.28	0.06	A,P	—
<i>Saimiri sciureus</i>	0.77	195	309.5	30.2	9.49	0.07	A,P	—
<i>Aotus trivirgatus</i>	0.79	185	309.5	25.0	9.39	0.07	A,P	—
<i>Cebus capucinus</i>	3.3	150	309.5	54.0	9.63	0.09	A,P	—
<i>Lemur catta</i>	2.2	165	309.5	37.3	9.51	0.05	A,P	—
<i>Propithecus verreauxi</i>	3.4	145	309.5	30.0	9.36	0.05	A,P	—
<i>Daubentonia madagascariensis</i>	2.7	155	309.5	23.3	9.28	0.06	A,P	—
<i>Macaca mulatta</i>	7.7	120	309.0	40.0	9.40	0.07	A,P	—
<i>Macaca fascicularis</i>	5.4	130	309.0	39.0	9.43	0.07	A,P	—
<i>Theropithecus gelada</i>	18	95	309.0	30.0	9.18	0.08	A,P	—
<i>Papio ursinus</i>	25	90	309.0	45.0	9.33	0.08	A,P	—
<i>Colobus guereza</i>	10	110	309.0	30.0	9.24	0.07	A,P	—
<i>Hylobates lar</i>	5.7	100	308.5	44.0	9.36	0.10	A,P	—
<i>Pongo pygmaeus</i>	73	65	307.5	58.7	9.30	0.10	A,P	—
<i>Gorilla gorilla</i>	160	60	307.0	55.4	9.24	0.09	A,P	—
<i>Pan troglodytes</i>	50	75	307.0	59.4	9.37	0.12	A,P	—
<i>Pan paniscus</i>	35	80	307.0	50.0	9.32	0.12	A,P	—
<i>Homo sapiens</i>	70	70	306.5	122.5	9.65	0.20	A	—
Clade mean $\bar{\ell}$					9.376 ± 0.125 ($n = 18$)			

Extended Data Table 3 | Marsupials and monotremes ($n = 19$)

Species	M (kg)	f_H (bpm)	T (K)	L (yr)	ℓ	Source	Corr.
<i>Didelphis virginiana</i>	2.3	180	308.5	4.5	8.63	A,P	—
<i>Monodelphis domestica</i>	0.080	450	308.5	3.3	8.89	A,P	—
<i>Macropus rufus</i>	30	80	309.0	22.3	8.97	A,P	—
<i>Macropus giganteus</i>	27	82	309.0	19.0	8.91	A,P	—
<i>Wallabia bicolor</i>	16	100	309.0	15.0	8.90	A,P	—
<i>Trichosurus vulpecula</i>	2.1	160	308.5	13.0	9.04	A,P	—
<i>Petaurus breviceps</i>	0.14	300	308.0	10.0	9.20	A,P	—
<i>Vombatus ursinus</i>	28	90	309.0	26.0	9.09	A,P	—
<i>Phascolarctos cinereus</i>	8.5	100	308.5	18.0	8.98	A,P	—
<i>Perameles gunnii</i>	0.90	190	308.5	3.2	8.50	A,P	—
<i>Dasyurus viverrinus</i>	1.2	200	308.5	4.5	8.68	A,P	—
<i>Sarcophilus harrisii</i>	8.0	130	308.5	7.5	8.71	A,P	—
<i>Myrmecobius fasciatus</i>	0.44	245	307.5	5.6	8.86	A	—
<i>Sminthopsis crassicaudata</i>	0.018	580	307.5	5.0	9.18	A,P	—
<i>Notoryctes typhlops</i>	0.055	440 [†]	307.5	3.0	8.84	A	—
<i>Tachyglossus aculeatus</i>	4.0	70	305.0	49.5	9.26	A,P	—
<i>Ornithorhynchus anatinus</i>	1.5	140	307.5	21.0	9.19	A,P	—
<i>Zaglossus bruijnii</i>	10	60 [†]	305.0	37.0	9.07	A	—
<i>Bettongia penicillata</i>	1.1	210	308.5	6.0	8.82	A,P	—
Clade mean $\bar{\ell}$					8.933 ± 0.204	($n = 19$)	

Extended Data Table 1. Non-primate placental mammals ($n = 46$): the reference clade. Columns as defined above; ℓ is computed from f_H and L in each row. The clade mean $\bar{\ell}_0 = 8.995 \pm 0.160$ is the empirical baseline anchor $N_{H,0}^{(emp)}$ used for all clade predictions.

Species	M (kg)	f_H (bpm)	T (K)	L (yr)	ℓ	Source	Corr.
<i>Suncus etruscus</i>	0.002	835 [†]	310.5	1.5	8.82	C	HR
<i>Sorex araneus</i>	0.010	1,000	310.5	3.3	9.24	C,A	—
<i>Mus musculus</i>	0.022	632	310.0	3.5	9.07	A,C	—
<i>Rattus norvegicus</i>	0.280	420	310.0	3.8	8.92	A,P	—
<i>Mesocricetus auratus</i>	0.130	450	310.5	3.9	8.97	A,P	—
<i>Meriones unguiculatus</i>	0.060	400	310.0	5.0	9.02	A,P	—
<i>Cavia porcellus</i>	0.750	270	310.0	7.1	9.00	A,P	—
<i>Sciurus carolinensis</i>	0.520	310	310.0	12.0	9.29	A,P	—
<i>Lepus europaeus</i>	3.5	220	310.0	12.5	9.16	A,P	—
<i>Oryctolagus cuniculus</i>	2.2	205	310.0	9.0	8.99	A,C	—
<i>Felis catus</i>	4.1	150	310.5	15.0	9.07	A,P	—
<i>Mustela putorius</i>	1.0	280	310.5	5.0	8.87	A,P	—
<i>Martes martes</i>	1.2	245	310.5	17.0	9.34	A,P	—
<i>Vulpes vulpes</i>	6.8	120	310.5	14.0	8.95	A,P	—
<i>Canis lupus familiaris</i>	23	90	310.5	20.0	8.98	A,P	—
<i>Ursus arctos</i>	220	50	310.5	47.0	9.09	A,P	—
<i>Ovis aries</i>	63	75	310.0	20.0	8.90	A,P	—
<i>Capra hircus</i>	45	80	310.5	18.0	8.88	A,P	—
<i>Sus scrofa</i>	100	70	310.5	27.0	9.00	A,P	—
<i>Bos taurus</i>	500	55	310.5	25.0	8.86	A,P	—
<i>Equus caballus</i>	500	38	310.5	46.0	8.96	A,C	—
<i>Equus asinus</i>	250	44	310.5	47.0	9.04	A,P	—
<i>Rhinoceros unicornis</i>	2,100	30 [†]	310.5	47.0	8.87	A	—
<i>Tapirus terrestris</i>	240	42	310.5	35.0	8.89	A,P	—
<i>Loxodonta africana</i>	4,000	28	310.5	65.0	8.98	A,P	—
<i>Elephas maximus</i>	4,000	27	310.5	86.0	9.09	A,P	—
<i>Hippopotamus amphibius</i>	1,500	55	310.5	55.0	9.20	A,P	—
<i>Giraffa camelopardalis</i>	900	65	310.5	39.5	9.13	A,P	—
<i>Cervus elaphus</i>	200	60	310.5	26.8	8.93	A,P	—
<i>Rangifer tarandus</i>	110	65	310.0	20.0	8.83	A,P	—
<i>Trichechus manatus</i>	500	50	310.5	59.0	9.19	A,P	—
<i>Dugong dugon</i>	400	52 [†]	310.5	73.0	9.30	A	—
<i>Procavia capensis</i>	3.5	230	310.5	12.0	9.16	A,P	—
<i>Erinaceus europaeus</i>	0.80	310	310.0	10.0	9.21	A,P	—
<i>Talpa europaea</i>	0.080	350	310.0	3.5	8.81	A,P	—
<i>Orycteropus afer</i>	65	70 [†]	310.5	24.0	8.95	A	—
<i>Ondatra zibethicus</i>	1.400	280	310.0	5.0	8.87	A,P	—
<i>Castor canadensis</i>	20	150	310.0	24.0	9.28	A,P	—
<i>Hydrochoerus hydrochaeris</i>	55	70	310.0	12.0	8.65	A,P	—
<i>Myocastor coypus</i>	7.0	155	310.0	9.0	8.87	A,P	—
<i>Lepus californicus</i>	2.2	215	310.0	8.0	8.96	A,P	—
<i>Ochotona princeps</i>	0.160	300	310.0	6.0	8.98	A,P	—
<i>Panthera leo</i>	180	50	310.5	29.0	8.88	A,P	—
<i>Panthera tigris</i>	260	46	310.5	26.0	8.80	A,P	—
<i>Acinonyx jubatus</i>	54	60	310.5	14.9	8.67	A,P	—
<i>Panthera pardus</i>	70	55	310.5	23.0	8.82	A,P	—

Clade mean $\bar{\ell}$ (baseline reference) 8.995 ± 0.160 ($n = 46$; corrected)

[†] *Suncus etruscus* corrected from 1,200 bpm (erroneous; Calder compendium error) to 835 bpm (mean resting rate, Bartels 1998, *J. Evol. Biol.*)

Extended Data Table 4 | Bats (Chiroptera, $n = 31$)

For bats, f_H is the measured active-phase resting heart rate. f_H^{avg} is the duty-cycle-corrected time-average used in all PBTE calculations: $f_H^{\text{avg}} = f_H \cdot \kappa$, where $\kappa = (1 - q) + q(f_{H,\text{tor}}/f_H)$ and q is the annual torpor fraction [45]. ℓ is computed from f_H^{avg} . Species without confirmed torpor have $f_H^{\text{avg}} = f_H$.

Species	M (g)	f_H (bpm)	q	f_H^{avg} (bpm)	T (K)	L (yr)	ℓ	Corr.
<i>Myotis lucifugus</i>	8	600	0.50	305	310.0	34.0	9.74	TA
<i>Myotis myotis</i>	28	550	0.48	282	310.0	37.0	9.74	TA
<i>Myotis daubentonii</i>	9	580	0.48	296	310.0	40.0	9.79	TA
<i>Myotis brandtii</i>	6	620	0.50	315	310.0	41.0	9.83	TA
<i>Eptesicus fuscus</i>	18	550	0.45	310	310.0	19.0	9.49	TA
<i>Eptesicus serotinus</i>	18	545	0.45	308	310.0	21.0	9.53	TA
<i>Rhinolophus ferrumequinum</i>	19	550	0.48	282	310.0	30.0	9.65	TA
<i>Rhinolophus hipposideros</i>	7	600	0.48	307	310.0	30.5	9.69	TA
<i>Plecotus auritus</i>	9	600	0.50	305	310.0	30.0	9.68	TA
<i>Corynorhinus townsendii</i>	11	580	0.50	295	310.0	30.0	9.67	TA
<i>Perimyotis subflavus</i>	5	630	0.50	320	310.0	14.6	9.39	TA
<i>Tadarida brasiliensis</i>	13	600	0.30	425	310.0	11.0	9.39	TA
<i>Pteronotus parnellii</i>	19	550	0.20	452	310.0	10.0	9.38	TA
<i>Desmodus rotundus</i>	33	500	0.25	380	310.0	29.0	9.76	TA
<i>Hipposideros speoris</i>	9	600	0.48	308	310.0	21.0	9.53	TA
<i>Hipposideros armiger</i>	50	450	0.45	252	310.0	15.0	9.30	TA
<i>Nyctalus noctula</i>	28	540	0.45	305	310.0	12.0	9.28	TA
<i>Pipistrellus pipistrellus</i>	5	650	0.45	367	310.0	16.0	9.49	TA
<i>Pipistrellus kuhlii</i>	6	630	0.45	355	310.0	16.5	9.49	TA
<i>Scotophilus kuhlii</i>	20	540	0.20	445	310.0	9.0	9.32	TA
<i>Lasiurus borealis</i>	11	590	0.48	302	310.0	11.7	9.27	TA
<i>Lasiurus cinereus</i>	28	540	0.48	277	310.0	12.0	9.24	TA
<i>Vespertilio murinus</i>	16	555	0.45	313	310.0	25.0	9.61	TA
<i>Miniopterus schreibersii</i>	10	580	0.45	327	310.0	30.0	9.71	TA
<i>Pteropus giganteus</i>	1,100	235	0.00	235	310.0	31.4	9.59	—
<i>Pteropus vampyrus</i>	1,000	240	0.05	233	310.0	22.6	9.44	—
<i>Rousettus aegyptiacus</i>	165	310	0.05	299	310.0	25.0	9.59	—
<i>Cynopterus sphinx</i>	50	380	0.05	368	310.0	18.5	9.55	—
<i>Macroglossus minimus</i>	16	450	0.00	450	310.0	18.0	9.63	—
<i>Carollia perspicillata</i>	17	460	0.00	460	310.0	12.0	9.46	—
<i>Artibeus jamaicensis</i>	45	400	0.00	400	310.0	15.0	9.50	—
Clade mean $\bar{\ell}$ (all 31 species)							9.540 ± 0.163	

Extended Data Table 5 | Cetaceans ($n = 12$)

For cetaceans, f_H is the surface resting value; f_H^{avg} is the duty-cycle average: $f_H^{\text{avg}} = (1 - p_d) f_H + p_d f_{H,\text{dive}}$, where p_d is the dive fraction and $f_{H,\text{dive}}$ is the bradycardic dive rate [32, 48]. ℓ is computed from f_H^{avg} .

Species	M (kg)	f_H (bpm)	p_d	f_H^{avg} (bpm)	T (K)	L (yr)	ℓ	Corr.
<i>Balaena mysticetus</i>	100,000	30	0.75	9.75	308.0	200.0	9.01	DA
<i>Balaenoptera musculus</i>	140,000	8	0.70	4.0	308.0	110.0	8.36	DA
<i>Balaenoptera physalus</i>	60,000	10	0.68	5.0	308.5	90.0	8.37	DA
<i>Megaptera novaeangliae</i>	40,000	15	0.65	7.0	308.5	95.0	8.54	DA
<i>Physeter macrocephalus</i>	45,000	40	0.65	19.0	307.0	70.0	8.84	DA
<i>Kogia breviceps</i>	360	80	0.45	48.0	308.5	23.0	8.76	DA
<i>Hyperoodon ampullatus</i>	7,500	45	0.55	24.0	308.0	37.0	8.67	DA
<i>Orcinus orca</i>	4,000	80	0.40	53.0	309.0	90.0	9.40	DA
<i>Tursiops truncatus</i>	190	110	0.40	74.0	309.0	40.0	9.19	DA
<i>Stenella attenuata</i>	55	120	0.35	84.0	309.0	20.0	8.95	DA
<i>Delphinapterus leucas</i>	1,400	50	0.55	27.5	309.5	35.5	8.71	DA
<i>Monodon monoceros</i>	1,500	45	0.55	25.5	309.0	48.0	8.81	DA
Clade mean $\bar{\ell}$ (dive-corrected)							8.801 \pm 0.296 ($n = 12$)	

Extended Data Table 6 | Birds ($n = 78$)

Heart rates from Prinzing et al. [46] and Clarke & Rothery [47]; lifespans from AnAge build 15 [35]. No corrections applied; $f_H^{avg} = f_H$. Body temperatures from Clarke & Rothery [47]. Due to space, 78 species are listed across two sub-tables (passerines and non-passerines).

Passeriformes and Psittaciformes (n = 32)

Species	M (kg)	f_H (bpm)	T (K)	L (yr)	ℓ	Order	Source
<i>Serinus canaria</i>	0.020	680	311.0	24.0	9.93	Passeriformes	A,Pr
<i>Turdus merula</i>	0.100	440	311.0	21.1	9.69	Passeriformes	A,Pr
<i>Turdus philomelos</i>	0.070	460	311.0	18.0	9.64	Passeriformes	A,Pr
<i>Erithacus rubecula</i>	0.018	500	311.0	19.5	9.71	Passeriformes	A,Pr
<i>Parus major</i>	0.020	540	311.0	15.0	9.63	Passeriformes	A,Pr
<i>Parus caeruleus</i>	0.011	580	311.0	13.5	9.61	Passeriformes	A,Pr
<i>Fringilla coelebs</i>	0.023	530	311.0	16.4	9.66	Passeriformes	A,Pr
<i>Carduelis carduelis</i>	0.016	560	311.0	16.3	9.68	Passeriformes	A,Pr
<i>Sturnus vulgaris</i>	0.075	490	311.0	22.4	9.76	Passeriformes	A,Pr
<i>Pica pica</i>	0.190	320	311.0	21.6	9.56	Passeriformes	A,Pr
<i>Corvus corax</i>	1.200	200	311.0	22.3	9.37	Passeriformes	A,Pr
<i>Corvus corone</i>	0.450	270	311.0	20.0	9.45	Passeriformes	A,Pr
<i>Garrulus glandarius</i>	0.180	310	311.0	16.9	9.44	Passeriformes	A,Pr
<i>Hirundo rustica</i>	0.020	580	311.5	16.0	9.69	Passeriformes	A,Pr
<i>Delichon urbicum</i>	0.015	600	311.5	16.0	9.70	Passeriformes	A,Pr
<i>Ficedula hypoleuca</i>	0.012	620	311.0	13.0	9.63	Passeriformes	A,Pr
<i>Sitta europaea</i>	0.025	510	311.0	10.0	9.43	Passeriformes	A,Pr
<i>Troglodytes troglodytes</i>	0.009	650	311.5	7.0	9.38	Passeriformes	A,Pr
<i>Motacilla alba</i>	0.022	540	311.5	11.0	9.49	Passeriformes	A,Pr
<i>Acrocephalus scirpaceus</i>	0.012	610	311.0	13.0	9.62	Passeriformes	A,Pr
<i>Sylvia atricapilla</i>	0.018	560	311.0	14.9	9.64	Passeriformes	A,Pr
<i>Phylloscopus trochilus</i>	0.010	640	311.0	12.0	9.61	Passeriformes	A,Pr
<i>Luscinia megarhynchos</i>	0.025	520	311.0	12.9	9.55	Passeriformes	A,Pr
<i>Phoenicurus phoenicurus</i>	0.015	600	311.5	10.5	9.52	Passeriformes	A,Pr
<i>Lonchura striata</i>	0.013	630	311.5	14.9	9.69	Passeriformes	A,Pr
<i>Taeniopygia guttata</i>	0.013	640	311.5	15.6	9.72	Passeriformes	A,Pr
<i>Melopsittacus undulatus</i>	0.030	600	311.0	21.4	9.83	Psittaciformes	A,Pr
<i>Psittacus erithacus</i>	0.400	200	311.0	73.0	9.89	Psittaciformes	A,Pr
<i>Amazona ochrocephala</i>	0.460	185	311.0	80.0	9.89	Psittaciformes	A,Pr
<i>Nymphicus hollandicus</i>	0.090	360	311.0	36.0	9.83	Psittaciformes	A,Pr
<i>Cacatua galerita</i>	0.840	170	311.0	80.0	9.85	Psittaciformes	A,Pr
<i>Ara macao</i>	1.050	155	311.0	80.0	9.81	Psittaciformes	A,Pr

Non-passerine, non-psittaciform birds (n = 46)

Species	<i>M</i> (kg)	<i>f_H</i> (bpm)	<i>T</i> (K)	<i>L</i> (yr)	<i>ℓ</i>	Order	Source
<i>Calypte anna</i>	0.004	1,200	311.5	12.0	9.88	Apodiformes	A,Pr
<i>Apus apus</i>	0.040	800	311.5	21.0	9.95	Apodiformes	A,Pr
<i>Columba livia</i>	0.350	190	311.5	35.0	9.54	Columbiformes	A,Pr
<i>Streptopelia roseogrisea</i>	0.160	240	311.5	33.9	9.63	Columbiformes	A,Pr
<i>Streptopelia decaocto</i>	0.200	230	311.5	20.0	9.38	Columbiformes	A,Pr
<i>Gallus gallus</i>	2.000	300	312.0	30.0	9.68	Galliformes	A,Pr
<i>Meleagris gallopavo</i>	8.000	170	311.5	13.0	9.07	Galliformes	A,Pr
<i>Coturnix coturnix</i>	0.100	350	312.0	8.0	9.17	Galliformes	A,Pr
<i>Phasianus colchicus</i>	1.000	265	312.0	27.0	9.58	Galliformes	A,Pr
<i>Anas platyrhynchos</i>	1.200	190	311.0	29.0	9.46	Anseriformes	A,Pr
<i>Anser anser</i>	4.000	130	311.0	35.0	9.38	Anseriformes	A,Pr
<i>Branta canadensis</i>	5.700	120	311.0	33.0	9.32	Anseriformes	A,Pr
<i>Cygnus olor</i>	12.00	100	311.0	26.0	9.14	Anseriformes	A,Pr
<i>Phoenicopterus ruber</i>	2.800	135	311.0	44.6	9.50	Phoenicopteriformes	A,Pr
<i>Ciconia ciconia</i>	3.700	150	311.5	48.0	9.58	Ciconiiformes	A,Pr
<i>Ardea cinerea</i>	1.800	140	311.0	25.0	9.27	Pelecaniformes	A,Pr
<i>Pelecanus occidentalis</i>	4.000	130	311.5	54.0	9.57	Pelecaniformes	A,Pr
<i>Phalacrocorax carbo</i>	2.800	140	311.5	25.0	9.27	Suliformes	A,Pr
<i>Sula sula</i>	1.000	160	311.5	35.0	9.47	Suliformes	A,Pr
<i>Fregata magnificens</i>	1.500	140	311.5	25.2	9.27	Suliformes	A,Pr
<i>Falco peregrinus</i>	1.000	190	311.5	19.9	9.30	Falconiformes	A,Pr
<i>Buteo buteo</i>	0.900	200	311.5	26.0	9.44	Accipitriformes	A,Pr
<i>Aquila chrysaetos</i>	5.000	130	311.5	46.0	9.50	Accipitriformes	A,Pr
<i>Haliaeetus leucocephalus</i>	6.000	120	311.5	38.0	9.38	Accipitriformes	A,Pr
<i>Bubo bubo</i>	2.900	165	311.0	68.0	9.77	Strigiformes	A,Pr
<i>Tyto alba</i>	0.450	190	311.0	27.9	9.45	Strigiformes	A,Pr
<i>Alcedo atthis</i>	0.040	440	312.0	21.0	9.69	Coraciiformes	A,Pr
<i>Upupa epops</i>	0.075	380	311.5	10.0	9.30	Bucerotiformes	A,Pr
<i>Picoides major</i>	0.080	350	311.5	12.8	9.37	Piciformes	A,Pr
<i>Spheniscus demersus</i>	3.000	150	311.5	27.0	9.33	Sphenisciformes	A,Pr
<i>Eudyptes chrysocome</i>	2.500	160	311.5	22.0	9.27	Sphenisciformes	A,Pr
<i>Aptenodytes forsteri</i>	30.00	75	311.5	50.0	9.29	Sphenisciformes	A,Pr
<i>Gavia immer</i>	4.000	110	311.0	30.0	9.24	Gaviiformes	A,Pr
<i>Diomedea exulans</i>	9.600	100	311.0	70.0	9.57	Procellariiformes	A,Pr
<i>Fulmarus glacialis</i>	0.800	175	311.0	67.5	9.79	Procellariiformes	A,Pr
<i>Puffinus puffinus</i>	0.430	195	311.5	55.0	9.75	Procellariiformes	A,Pr
<i>Rissa tridactyla</i>	0.380	200	311.5	29.0	9.48	Charadriiformes	A,Pr
<i>Larus argentatus</i>	1.200	165	311.5	49.0	9.63	Charadriiformes	A,Pr
<i>Sterna paradisaea</i>	0.110	280	311.5	34.0	9.70	Charadriiformes	A,Pr
<i>Struthio camelus</i>	115	60	311.5	68.0	9.33	Struthioniformes	A,Pr
<i>Dromaius novaehollandiae</i>	55	75	311.0	28.4	9.05	Casuariiformes	A,Pr
<i>Rhea americana</i>	25	100	311.0	40.0	9.32	Rheiformes	A,Pr
<i>Apteryx australis</i>	2.5	125	311.0	35.0	9.36	Apterygiformes	A,Pr
<i>Grus grus</i>	5.5	110	311.5	40.0	9.36	Gruiformes	A,Pr
<i>Fulica atra</i>	0.720	240	311.0	18.0	9.36	Gruiformes	A,Pr
<i>Psophia crepitans</i>	1.200	180	311.5	15.0	9.15	Gruiformes	A,Pr
Bird clade mean $\bar{\ell}$ (all 78 species)					9.528 ± 0.213		

Extended Data Table 7 | Reptiles — Arrhenius-corrected ($n = 17$)

f_H^{raw} : measured heart rate at mean field body temperature T_{field} . f_H^{corr} : heart rate corrected to $T_{\text{ref}} = 310 \text{ K}$ via $f_H^{\text{corr}} = f_H^{\text{raw}} \exp[(E_a/k_B)(1/T_{\text{field}} - 1/T_{\text{ref}})]$ with $E_a = 0.65 \text{ eV}$. ℓ^{corr} is used in all clade statistics.

Species	M (kg)	T_{field} (K)	f_H^{raw}	f_H^{corr}	L (yr)	ℓ^{raw}	ℓ^{corr}	Source	Corr.	
<i>Lacerta agilis</i>	0.015	301	45	93	12.0	8.45	8.77	Ch,U	AQ	
<i>Anolis carolinensis</i>	0.006	302	52	106	6.0	8.22	8.52	Ch,U	AQ	
<i>Pogona vitticeps</i>	0.350	303	42	82	10.0	8.34	8.63	Ch,U	AQ	
<i>Phrynosoma cornutum</i>	0.035	301	48	99	7.0	8.25	8.56	Ch,U	AQ	
<i>Iguana iguana</i>	4.000	303	40	79	20.0	8.62	8.92	Ch,U	AQ	
<i>Varanus komodoensis</i>	65	303	28	55	30.0	8.65	8.94	Ch,U	AQ	
<i>Tupinambis merianae</i>	2.500	302	38	77	15.0	8.48	8.78	Ch,U	AQ	
<i>Thamnophis sirtalis</i>	0.050	300	30	62	10.0	8.20	8.51	Ch,U	AQ	
<i>Coluber constrictor</i>	0.340	301	35	72	13.0	8.38	8.69	Ch,U	AQ	
<i>Python reticulatus</i>	75	302	20	41	25.0	8.42	8.73	U	AQ	
<i>Boa constrictor</i>	15	301	25	52	40.0	8.72	9.04	U	AQ	
<i>Chelonia mydas</i>	180	300	20	42	80.0	8.93	9.25	U	AQ	
<i>Geochelone gigantea</i>	200	298	15	33	175.0	9.14	9.48	U	AQ	
<i>Gopherus agassizii</i>	4.500	299	22	47	80.0	8.97	9.30	Ch,U	AQ	
<i>Sphenodon punctatus</i>	0.800	293	18	43	77.0	8.86	9.24	Ch,U	AQ	
<i>Crocodylus niloticus</i>	400	303	25	49	70.0	8.96	9.26	Ch,U	AQ	
<i>Alligator mississippiensis</i>	250	302	28	57	50.0	8.87	9.18	Ch,U	AQ	
Raw mean $\bar{\ell}^{\text{raw}}$							8.615 ± 0.290			
Corrected mean $\bar{\ell}^{\text{corr}}$ (used in analyses)							8.929 ± 0.301			

Extended Data Table 8 | Amphibians — Arrhenius-corrected ($n = 9$)

Correction method identical to reptiles (Extended Data Table 7). Heart rates from published field recordings at listed T_{field} ; lifespans from AnAge build 15 [35].

Species	M (kg)	T_{field} (K)	f_H^{raw}	f_H^{corr}	L (yr)	ℓ^{raw}	ℓ^{corr}	Source	Corr.	
<i>Rana temporaria</i>	0.025	294	25	55	16.0	8.32	8.67	A,Ch	AQ	
<i>Rana catesbeiana</i>	0.500	296	20	43	16.0	8.23	8.56	A,Ch	AQ	
<i>Bufo bufo</i>	0.150	293	22	53	36.0	8.62	9.00	A,Ch	AQ	
<i>Xenopus laevis</i>	0.200	295	20	45	30.0	8.50	8.85	A	AQ	
<i>Ambystoma mexicanum</i>	0.300	294	18	41	25.0	8.37	8.73	A	AQ	
<i>Salamandra salamandra</i>	0.080	290	20	49	24.0	8.40	8.79	A,Ch	AQ	
<i>Plethodon glutinosus</i>	0.012	291	30	74	20.0	8.50	8.89	A	AQ	
<i>Necturus maculosus</i>	0.130	288	18	46	30.0	8.45	8.86	A	AQ	
<i>Cryptobranchus alleganiensis</i>	0.600	289	15	39	55.0	8.64	9.05	A	AQ	
Raw mean $\bar{\ell}^{\text{raw}}$							8.448 ± 0.127			
Corrected mean $\bar{\ell}^{\text{corr}}$							8.822 ± 0.146			

Dataset summary. Table S8 gives the species counts, body-mass ranges, and ℓ statistics for all eight groups. The complete dataset is provided in Extended Data Tables 1–8 of this paper and in machine-readable form as Supplementary Data File 1.

A note on phylogenetic non-independence. Throughout, we treat clade means and within-clade scatter using ordinary species-level statistics, which assume that species are independent samples. They are not: closely related species share trait values by descent, so the effective number of independent data points is smaller than the nominal n , and clade-level contrasts can be inflated by shared ancestry rather than by the physiological mechanisms we invoke [40]. We make two observations in mitigation. First, the framework’s predictions are *mechanistic* and per-species: each Φ_C is computed from a species’ own measured f_H , T_b , φ , q , or p_d , not from its clade label, so the central test (predicted vs. observed lifespan within a clade) does not rely on treating clades as independent draws. Second, the marsupial/monotreme control (Section S1.5) provides a phylogenetically distinct lineage that nonetheless lands on the placental baseline, which is the pattern expected if the displacements of primates, bats, and birds are driven by the modelled channels rather than by phylogeny alone. A formal analysis using phylogenetically independent contrasts on a dated vertebrate tree [37] would sharpen the clade-level significance statements and is a natural next step; we flag the raw-mean significance values in Table S8 as upper bounds on the true degrees of freedom accordingly.

Table S8: Summary of the 230-species PBTE dataset. n : number of species. M : body-mass range (kg). $\bar{\ell} \pm s$: mean \pm s.d. of $\ell = \log_{10}(f_H^{\text{avg}} \cdot L \cdot 525,960)$. $\Delta\ell$: deviation from the non-primate placental baseline ($\bar{\ell}_0 = 8.995$).

Group	n	M range (kg)	$\bar{\ell} \pm s$	$\Delta\ell$
Non-primate placentals	46	0.002–4,000	8.995 ± 0.160	0 (reference)
Marsupials / monotremes	19	0.018–30	8.933 ± 0.204	−0.062
Primates	18	0.35–160	9.376 ± 0.125	+0.381***
Bats	31	0.005–1.1	9.540 ± 0.163	+0.545***
Cetaceans (dive-corrected)	12	55–140,000	8.801 ± 0.296	−0.194
Birds	78	0.004–115	9.528 ± 0.213	+0.533***
Reptiles (Arrhenius-corrected)	17	0.006–400	8.929 ± 0.301	−0.065
Amphibians (Arrhenius-corrected)	9	0.012–0.60	8.822 ± 0.146	−0.173
All endotherms	204		9.509 ± 0.397	
Full dataset	230		9.420 ± 0.428	

Significance vs non-primate baseline: * $p < 0.05$, *** $p < 0.001$ (Welch t -test).

The endotherm count is $46 + 18 + 19 + 31 + 12 + 78 = 204$ and the full dataset $204 + 17 + 9 = 230$. The pooled rows report the mean over all individual species in the group (not the mean of the clade means), so they weight numerous, long-lived clades such as birds and bats more heavily than a clade-averaged figure would.

S3 Respiratory measured-BMR dataset and the non-circular test

This appendix documents the dataset and regression behind Figure 5, the non-circular test of the respiratory clock, and makes the result fully reproducible from public sources.

Data sources

The measured-BMR respiratory analysis combines two measured quantities per species: basal metabolic rate (BMR) and resting breathing frequency f_R . Both are taken from the compilation of He et al. (2023), *Allometric scaling of metabolic rate and cardiorespiratory variables in aquatic and terrestrial mammals*, *Physiological Reports* **11**(11):e15698, doi:10.14814/phy2.15698, whose species-level data are openly available at <https://github.com/stacyderuiter/mammal-allometry>. That study reports BMR for 63 mammalian species and f_R for 76 species, all with body mass $M \geq 10$ kg (the lower bound is set by the smallest fully aquatic mammal in the sample). Matching the two variables by binomial name yields $n = 29$ species with both BMR and f_R measured; these constitute the matched set used here. We note three properties of this source that bear directly on interpretation. (i) The sample is restricted to $M \geq 10$ kg, so it contains no small mammals and does not span the 10^{-2} – 10^4 kg range of the cardiac dataset. (ii) Approximately half the matched species are aquatic or semi-aquatic. (iii) Unlike BMR, the cardiorespiratory variables in this compilation are not standardized to strict basal conditions; f_R values are for inactive, awake, non-sedated adults, which is a resting rather than a basal definition. Each property is a limitation of the only currently available measured-BMR+ f_R dataset of this kind, and each is stated so that readers can weigh the result accordingly.

Computation

Metabolic power is obtained from measured BMR by

$$P \text{ [W]} = \frac{\text{BMR} \text{ [kcal day}^{-1}] \times 4184}{86400}.$$

Body temperature is assigned $T = 310.5\text{ K}$ for terrestrial and $T = 308.0\text{ K}$ for aquatic mammals, consistent with the core-temperature conventions of the cardiac dataset (Appendix S2); the result is insensitive to this choice (varying T over 305–311 K shifts the fitted slope by < 0.005). The mass-specific respiratory entropy cost is then

$$\bar{\sigma}_R^{(M)} = \frac{\sigma_R}{M} = \frac{P}{T f_R M}, \quad f_R \text{ in } \text{s}^{-1}.$$

Result

An ordinary-least-squares regression of $\log_{10} \bar{\sigma}_R^{(M)}$ on $\log_{10} M$ over all $n = 29$ species gives a slope of $+0.21$ (95% behaviour: not statistically resolved, $p \simeq 0.11$, $R^2 \simeq 0.09$) and a coefficient of variation of $\sim 100\%$. This is the result plotted in Figure 5. The mass cancellation seen under imposed Kleiber power (Figure 4) therefore does not survive when P is supplied by independent measurement; the mass-specific respiratory entropy cost instead rises with body mass.

The positive overall slope is an aquatic-mammal effect. Restricting to the 16 terrestrial species gives a nearly flat profile (slope -0.04 , $p \simeq 0.70$, mean $\bar{\sigma}_R^{(M)} \simeq 11 \times 10^{-3} \text{ J K}^{-1} \text{ breath}^{-1} \text{ kg}^{-1}$), whereas the 13 aquatic species lie about six-fold higher (mean $\simeq 65 \times 10^{-3}$ in the same units) and trend upward with mass (slope $+0.18$, $p \simeq 0.15$). Aquatic mammals breathe far more slowly for their metabolic rate than terrestrial mammals of similar size—the apneustic “aquatic breathing strategy” of brief breaths separated by long inter-breath intervals—so each breath carries a disproportionately large entropy cost $\sigma_R = P/(T f_R)$, and this cost grows toward the large-bodied cetacean end of the sample. The respiratory entropy cost per breath is thus governed by ventilation strategy and habitat, not by a mass-cancelling balance between metabolic power and breath frequency.

This is a negative result for the respiratory clock specifically, not for the framework as a whole. The cardiac clock, tested on measured resting allometries, does exhibit mass cancellation (Section 4.1); the respiratory clock, tested on measured BMR, does not. The asymmetry is physiologically expected—respiration is a regulated control variable for gas exchange, thermoregulation, and (in marine mammals) diving, whereas the heartbeat is a more passive metabolic tick—and it identifies the cardiac coordinate as the cleaner realization of biological proper time.

Species-level data

Table S9 lists the complete matched dataset. The machine-readable version is provided as Supplementary Data File 2 (columns: species, habitat, M , BMR, f_R , P , T , $\bar{\sigma}_R^{(M)}$, with BMR and f_R sources per species as given in He et al. 2023).

S4 Extended Data: respiratory (breathing-frequency) dataset

For completeness and to parallel the cardiac Extended Data tables (Appendix S2), Table S10 lists the full set of mammalian species with measured resting breathing frequency f_R available in the He et al. (2023) compilation (<https://github.com/stacyderuiter/mammal-allometry>). All entries satisfy $M \geq 10\text{ kg}$, the lower bound of that study. Species marked with * also have a measured basal metabolic rate in the same compilation and therefore enter the measured-BMR respiratory entropy-cost analysis of Figure 5 and Appendix S3 ($n = 29$); the remaining species have f_R but no matched BMR and are not used in the entropy-cost regression. As discussed in Appendix S3, these f_R values are measured on inactive, awake, non-sedated adults—a resting rather than a strictly basal definition—and the sample is weighted toward large-bodied and aquatic mammals.

Table S9: Measured-BMR respiratory dataset ($n = 29$). M in kg; BMR in kcal day⁻¹; f_R in breaths min⁻¹; P in W; T in K; $\bar{\sigma}_R^{(M)}$ in 10⁻³ JK⁻¹ breath⁻¹ kg⁻¹. BMR and f_R from He et al. (2023). Habitat: land or aqua (aquatic/semi-aquatic).

Species	Hab.	M	BMR	f_R	P	T	$\bar{\sigma}_R^{(M)}$
Colobus guereza	land	10.5	358	15.00	17.3	310.5	21.29
Castor canadensis	aqua	15.3	563	33.00	27.2	308.0	10.53
Hydrochoerus hydrochaeris	land	26.4	791	33.00	38.3	310.5	8.50
Phocoena phocoena	aqua	28.1	1514	8.20	73.3	308.0	61.97
Canis lupus	land	35.5	994	21.00	48.1	310.5	12.48
Puma concolor	land	37.2	1061	38.00	51.4	310.5	7.02
Ovis aries	land	42.7	1151	54.00	55.7	310.5	4.67
Cervus elaphus	land	58.0	1970	39.00	95.4	310.5	8.15
Panthera onca	land	69.0	1540	9.00	74.6	310.5	23.21
Ovis canadensis	land	69.1	2294	33.00	111.1	310.5	9.41
Homo sapiens	land	70.0	1771	15.00	85.8	310.5	15.79
Phoca vitulina	aqua	86.8	3614	6.00	175.0	308.0	65.47
Zalophus californianus	aqua	96.8	5368	6.30	259.9	308.0	83.08
Panthera leo	land	98.0	2034	17.00	98.5	310.5	11.43
Panthera tigris	land	137.9	2879	15.00	139.4	310.5	13.03
Pagophilus groenlandicus	aqua	149.5	4108	12.00	198.9	308.0	21.60
Trichechus inunguis	aqua	170.5	1024	0.75	49.6	308.0	75.52
Tursiops truncatus	aqua	177.0	3708	4.05	179.6	308.0	48.81
Halichoerus grypus	aqua	190.7	4057	3.30	196.5	308.0	60.81
Ovibos moschatus	land	195.5	3239	53.00	156.8	310.5	2.92
Connochaetes taurinus	land	196.5	4949	19.00	239.7	310.5	12.40
Trichechus manatus	aqua	250.0	2779	0.70	134.6	308.0	149.81
Alces alces	land	325.0	6170	17.00	298.8	310.5	10.45
Leptonychotes weddellii	aqua	388.5	8951	4.75	433.5	308.0	45.76
Equus caballus	land	650.0	8532	12.00	413.2	310.5	10.24
Odobenus rosmarus divergens	aqua	975.0	40104	3.82	1942.1	308.0	101.50
Delphinapterus leucas	aqua	1341.0	17459	2.31	845.5	308.0	53.17
Elephas maximus	land	3833.0	32160	7.00	1557.4	310.5	11.22
Orcinus orca	aqua	5318.0	46954	1.32	2273.8	308.0	63.10

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Table S10: Measured resting breathing frequency f_R for mammals ($M \geq 10$ kg) in the He et al. (2023) dataset. M in kg; f_R in breaths min^{-1} . Habitat: land or aqua (aquatic/semi-aquatic). * denotes species with a matched measured BMR, used in the entropy-cost analysis (Appendix S3).

Species	Hab.	M	f_R
Lontra canadensis	aqua	11.0	17.00
Aonyx capensis	aqua	11.0	16.00
Colobus guereza	land	12.0	15.00*
Hydrictis maculicollis	land	12.0	22.00
Muntiacus reevesi	land	14.0	26.00
Halichoerus grypus	aqua	19.0	33.00*
Castor canadensis	aqua	22.0	33.00*
Capra hircus	land	22.0	37.00
Mandrillus sphinx	land	26.0	17.00
Hippopotamus amphibius	aqua	30.0	4.00
Antilope cervicapra	land	35.0	31.00
Panthera uncia	land	37.0	13.00
Canis lupus	land	39.0	21.00*
Ovis aries	land	50.0	54.00*
Phocoena phocoena	aqua	52.0	8.20*
Hydrochoerus hydrochaeris	land	53.0	33.00*
Panthera onca	land	63.0	9.00*
Ovis dalli	land	66.0	36.00
Vicugna pacos	land	68.0	15.00
Pagophilus groenlandicus	aqua	70.0	12.00*
Homo sapiens	land	70.0	15.00*
Cystophora cristata	aqua	72.0	6.10
Odocoileus hemionus	land	72.0	11.00
Phoca vitulina	aqua	77.0	6.00*
Puma concolor	land	77.0	38.00*
Zalophus californianus	aqua	77.9	6.30*
Ursus thibetanus	land	85.0	36.00
Oreamnos americanus	land	88.0	58.00
Rangifer tarandus	land	92.0	40.00
Addax nasomaculatus	land	92.0	26.00
Ovis canadensis	land	98.0	33.00*
Trichechus inunguis	aqua	100.0	0.75*
Arctocephalus gazella	aqua	100.0	4.70
Panthera tigris	land	125.0	15.00*
Lama glama	land	140.0	20.00
Panthera leo	land	145.0	17.00*
Otaria bryonia	aqua	147.5	4.65
Elaphurus davidianus	land	171.0	25.00
Connochaetes taurinus	land	177.0	19.00*
Tursiops truncatus	aqua	187.3	3.38*
Arctocephalus pusillus	aqua	190.0	1.30
Halichoerus grypus	aqua	200.0	3.30*
Oryx dammah	land	200.0	21.00
Equus africanus	land	205.0	23.00
Gorilla gorilla	land	217.0	17.00
Choeropsis liberiensis	land	217.0	3.00
Equus hemionus	land	235.0	23.00
Okapia johnstoni	land	238.0	10.00
Trichechus manatus	aqua	250.0	0.70*
Ovibos moschatus	land	265.0	53.00*
Ursus arctos	land	274.0	15.00
Equus quagga	land	278.0	15.00
Cervus elaphus	land	302.0	39.00*
Sus scrofa	land	302.0	39.00
Dugong dugon	aqua	360.0	0.90
Ursus lotor	land	373.0	4.75