Relativistic PBTE: Biological Proper Time Along the Worldline

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Abstract

Biological aging is conventionally indexed by chronological time, yet every organism is a physical system tracing a worldline through spacetime, so the time available to its metabolism is not coordinate time t but relativistic proper time τ. Building on the established Principle of Biological Time Equivalence (PBTE), whose thermodynamic foundation and aging dynamics are taken here as prior results [32–34], we ask how internal physiological time relates to the proper time of physics. The central result is that biological age is an entropy-production functional evaluated along the proper-time worldline,

$$\mathcal{A}_{\mathrm{PBTE}} = \frac{1}{\Sigma_{\mathrm{ref}}} \int_{\tau_0}^{\tau_1} \dot{\Sigma}_p(\tau)\, d\tau, \qquad \frac{d\mathcal{A}_{\mathrm{PBTE}}}{dt} = \frac{\dot{\Sigma}_p}{\gamma\, \Sigma_{\mathrm{ref}}},$$

where $\dot{\Sigma}_p$ is entropy production per unit proper time in the local rest frame and γ the Lorentz factor. The consequence is that relativistic time dilation is not caused by physiology: physiology, like every physical process, is parametrized by proper time—a fact established on muons, atomic and optical clocks, and GPS, none of them living [1, 3, 5, 13]—and PBTE only fixes the irreversible biological history accumulated per unit of it. The word *relativity* is used here in two compatible senses: the standard relativity of proper time, supplied by spacetime geometry, and a distinct *PBTE relativity* that is relational rather than geometric—two organisms exposed to the same external interval need not accumulate the same biological interval, because their characteristic physiological frequencies, entropy costs per cycle, and effective lifetime budgets differ, with internal times transformed by $d\vartheta_i/d\vartheta_j = f_i/f_j$. The framework preserves Einstein relativity intact, separates physiological from geometric contributions to externally observed aging, and yields falsifiable predictions for organisms in relativistic motion or differing gravitational potentials. More broadly, it reframes the comparative question of biology from how many years an organism has existed to how much internally generated biological history it has traversed.

Keywords: biological proper time; PBTE; relativity; proper time; time dilation; entropy production; biological aging.

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1 Introduction

Time enters modern science through several logically distinct concepts. In Newtonian mechanics it is an absolute external parameter, assumed to advance uniformly for every observer [25]. Special and general relativity replace this universal background with proper time: the invariant duration accumulated by a physical system along its worldline through spacetime [11, 12, 24, 28]. Thermodynamics introduces a different structure, the arrow of time, through the macroscopic irreversibility associated with entropy production [10, 27, 31]. Biology adds another level. Living systems possess internal rhythms—cardiac cycles, respiration, circadian oscillations, cellular division, and molecular turnover—whose cumulative progression need not remain proportional to calendar time across organisms or physiological states [4, 19, 29].

The Principle of Biological Time Equivalence (PBTE) was introduced to formalize this distinction. Its empirical point of departure is the approximate compensation between physiological frequency and lifespan. A mouse lives for only a few years and possesses a rapid heart rate, whereas an elephant lives for several decades and possesses a much slower heart rate. Although their chronological lifespans differ by more than an order of magnitude, their lifetime cardiac counts are both of order 10^9 cycles [6, 17]. PBTE interprets this regularity by defining biological proper time as the accumulated number of intrinsic physiological events and by treating aging as the irreversible thermodynamic expenditure associated with those events [32–34, 36].

It is useful to fix at the outset the sense in which the present work speaks of *relativity*, because two compatible meanings are involved. Relativistic proper time retains its standard physical meaning: the invariant duration accumulated along a worldline and fixed by spacetime geometry. PBTE introduces no alternative spacetime, no biological Lorentz group, and no modification of special or general relativity. Its additional claim is relational rather than geometric: biological duration is relative to the internal physiological clock by which a living system progresses through its own organized history. Two organisms exposed to the same external interval need not accumulate the same biological interval, because their characteristic physiological frequencies, entropy costs per cycle, and effective lifetime budgets may differ. In this restricted but physically meaningful sense PBTE defines a biological relativity principle, in which the transformation between organisms is governed not by velocity but by the ratio of their intrinsic clock rates, $d\vartheta_i/d\vartheta_j = f_i/f_j$, and, in the entropy-normalized formulation, by the irreversible cost assigned to those cycles. The mouse–elephant comparison gives this an operational meaning. If the cardiac frequency of the mouse is approximately twenty times that of the elephant, then during the same calendar day the mouse accumulates approximately twenty times as many cardiac cycles; one mouse day contains about the same cardiac-cycle distance as twenty elephant days. The two organisms inhabit the same astronomical day but do not spend the same amount of biological time within it. PBTE therefore shifts the fundamental comparative question from how many years an organism has existed to how much internally generated biological distance it has traversed.

The empirical invariant, its thermodynamic closure, and its application to biological aging have previously been formulated relative to terrestrial chronological time. That approximation is

sufficient for conventional comparative biology, because relativistic differences between terrestrial organisms are negligible. It is not, however, a fundamental formulation. Every organism is also a physical system following a worldline, and every local physical process within that organism evolves according to the proper time accumulated along that worldline. A complete formulation of PBTE must therefore distinguish the external coordinate time t, the relativistic proper time τ, the accumulated biological cycle count ϑ, and the entropy-normalized biological age A_{PBTE}.

This distinction resolves the central causal question addressed in the present work. A rapidly moving organism does not age more slowly because uniform velocity directly suppresses its physiology. In the organism's local rest frame, metabolism, cardiac activity, chemical reactions, molecular turnover, and cognition proceed normally. The difference observed from another frame arises because different worldlines contain different amounts of proper time between specified comparison events. Relativistic time dilation is therefore prior to physiology rather than produced by it. This conclusion follows from the experimentally established behavior of particle lifetimes, transported atomic clocks, optical clocks, and satellite timing systems, none of which requires a biological mechanism [1, 3, 5, 13, 14].

The contribution of this paper is to place PBTE consistently within that proper-time structure. Biological age is defined as an entropy-production functional evaluated along the organism's worldline. The resulting formulation separates the geometric and physiological contributions to externally observed aging:

spacetime geometry determines $d\tau$; local physiology determines $d\Sigma_{\mathrm{p}}$;
PBTE converts $d\Sigma_{\mathrm{p}}$ into biological age. (1)

The central law, derived in Section 3, expresses biological age as an entropy-production functional evaluated along the proper-time worldline,

$$A_{\mathrm{PBTE}} = \frac{1}{\Sigma_{\mathrm{ref}}} \int_{\tau_0}^{\tau_1} \dot{\Sigma}_{\mathrm{p}}(\tau)\, d\tau, \qquad \dot{\Sigma}_{\mathrm{p}} \equiv \frac{d\Sigma_{\mathrm{p}}}{d\tau}, \tag{2}$$

in which A_{PBTE} is the entropy-normalized biological age, $\dot{\Sigma}_{\mathrm{p}}$ the irreversible entropy-production rate per unit proper time measured in the organism's local rest frame, Σ_{ref} a reference lifetime entropy budget, and τ the relativistic proper time. We do not modify relativity; we identify the temporal parameter on which the previously developed PBTE aging functional must be evaluated. The mathematical operation is elementary, yet its physical content is consequential: spacetime fixes the proper duration available to the organism, whereas PBTE fixes the irreversible biological history accumulated during that duration. The remainder of the paper makes this precise. Section 2 restates the PBTE foundations—the lifetime-cycle relation, its thermodynamic closure, and the resulting entropy-normalized age—and isolates the proper-time problem that motivates the present work. Section 3 develops the covariant relativistic functional of Eq. (2) and its physical consequences, and Section 5 states the resulting temporal hierarchy, the limitations of the framework, and its falsifiable predictions.

2 PBTE Foundations and the Relativistic Proper-Time Problem

The present study extends the Principle of Biological Time Equivalence (PBTE) to systems whose motion through spacetime cannot be described by a single universal time coordinate. It does not propose an alternative theory of special relativity, nor does it attempt to explain relativistic time dilation through physiology. Its purpose is more specific. Previous PBTE work formulated biological progression and thermodynamic expenditure relative to an external chronological parameter t. Relativity, however, establishes that the duration locally accumulated by any physical system is the proper time τ along its worldline. The problem addressed here is therefore one of temporal consistency: how should PBTE be formulated when coordinate time and proper time are no longer interchangeable?

The empirical foundation of PBTE is the lifetime-cycle relation. Let $f_i(t)$ denote the instantaneous frequency of a recurrent physiological process in organism i. Depending on the biological system under consideration, this frequency may represent cardiac activity, respiration, cell division, metabolic cycling, or another recurrent process that provides an operational measure of physiological progression. The accumulated number of intrinsic cycles is defined as biological proper time,

$$\vartheta_i(t) = \int_0^t f_i(s)\, ds, \qquad d\vartheta_i = f_i(t)\, dt. \tag{3}$$

The term "proper time" is used here in a biological rather than a spacetime sense. The quantity ϑ_i is dimensionless and counts intrinsic biological events. It is not a coordinate of spacetime and should not be identified with the relativistic proper time τ_i. The distinction between these two quantities is central to the development below.

If L_i denotes the chronological lifespan of the organism, its total lifetime biological time is

$$N_i \equiv \vartheta_i(L_i) = \int_0^{L_i} f_i(t)\, dt. \tag{4}$$

Defining the lifetime-averaged physiological frequency as

$$\bar{f}_i \equiv \frac{1}{L_i} \int_0^{L_i} f_i(t)\, dt, \tag{5}$$

one obtains the exact identity

$$N_i = \bar{f}_i L_i. \tag{6}$$

The identity itself is elementary: frequency integrated over time produces a cycle count. The empirical content of PBTE lies elsewhere. It is the observation that, within an appropriately specified biological domain, N_i does not vary in direct proportion to either lifespan or physiological frequency. Instead, it concentrates around a characteristic lifetime scale,

$$N_i \simeq N_{\star,C}, \tag{7}$$

where C denotes the relevant clade, physiological clock, and normalization convention. Thus,

$$\bar{f}_{\mathrm{i}} L_{\mathrm{i}} \simeq N_{\star,\mathrm{C}}. \tag{8}$$

Equation (8) is the elementary form of PBTE. Organisms with rapid intrinsic clocks tend to traverse the lifetime cycle budget over short chronological intervals, whereas organisms with slower clocks distribute a comparable number of cycles over longer intervals. For the cardiac clock of many non-primate placental mammals, the characteristic scale is of order

$$N_{\star,\mathrm{C}} \sim 10^{9} \quad \text{cardiac cycles}. \tag{9}$$

PBTE does not require that every living system possess precisely the same numerical value of $N_{\star}$, nor that the cardiac clock be the appropriate clock for every form of life. Rather, it proposes that lifetime biological duration is constrained in the space of accumulated intrinsic events. Different clades, physiological architectures, and environmental regimes may require different effective clocks or explicit correction factors. The near-billion-cycle cardiac scale is therefore a well-defined empirical regularity within a particular biological domain, not an unrestricted numerical constant applying without qualification to all living systems [17, 29, 34].

The mouse–elephant comparison illustrates the physical meaning of this relation. Representative values are

$$\bar{f}_{\mathrm{mouse}} \simeq 600 \ \mathrm{min}^{-1}, \qquad L_{\mathrm{mouse}} \simeq 3 \ \mathrm{yr}, \tag{10}$$

and

$$\bar{f}_{\mathrm{elephant}} \simeq 30 \ \mathrm{min}^{-1}, \qquad L_{\mathrm{elephant}} \simeq 70 \ \mathrm{yr}. \tag{11}$$

Using

$$1 \ \mathrm{yr} = 525{,}960 \ \mathrm{min}, \tag{12}$$

the corresponding lifetime cardiac counts are approximately

$$\begin{aligned} N_{\mathrm{mouse}} &\simeq 600 \times 3 \times 525{,}960 \\ &\simeq 9.47 \times 10^{8}, \end{aligned} \tag{13}$$

$$\begin{aligned} N_{\mathrm{elephant}} &\simeq 30 \times 70 \times 525{,}960 \\ &\simeq 1.10 \times 10^{9}. \end{aligned} \tag{14}$$

The mouse heart therefore operates approximately twenty times faster than the elephant heart,

$$\frac{\bar{f}_{\text{mouse}}}{\bar{f}_{\text{elephant}}} \simeq 20, \tag{15}$$

whereas the elephant lives for approximately twenty-three times longer,

$$\frac{L_{\text{elephant}}}{L_{\text{mouse}}} \simeq 23. \tag{16}$$

The two ratios approximately compensate. In chronological units the lives of the two organisms are radically unequal; in accumulated cardiac cycles they are of the same order. The mouse traverses its biological trajectory rapidly, while the elephant traverses a comparable intrinsic interval more slowly. PBTE interprets this compensation as evidence that physiological history is more naturally represented by accumulated internal activity than by chronological duration alone.

For two organisms described relative to the same external time parameter,

$$\frac{d\vartheta_{\text{i}}}{d\vartheta_{\text{j}}} = \frac{f_{\text{i}}(t)}{f_{\text{j}}(t)}. \tag{17}$$

This is a multiplicative transformation between biological rates. It is not a Lorentz transformation. It contains no invariant limiting speed, no relativity of simultaneity, and no spacetime metric. The relation expresses only that two biological systems may accumulate intrinsic events at different rates while sharing the same environmental time coordinate. Figure 4 presents this chronological PBTE picture: different organisms traverse comparable lifetime cycle budgets at markedly different calendar rates.

The thermodynamic formulation developed in our previous work gives the cycle count a physical interpretation. A living organism is an open nonequilibrium system that maintains local organization by consuming free energy and producing entropy. Let $\Sigma_{\text{p,i}}$ denote cumulative irreversible entropy production and let

$$\dot{\Sigma}_{\text{p,i}} \equiv \frac{d\Sigma_{\text{p,i}}}{dt} \tag{18}$$

denote the corresponding entropy-production rate. The entropy produced per physiological cycle is

$$\sigma_{\text{i}} \equiv \frac{d\Sigma_{\text{p,i}}}{d\vartheta_{\text{i}}} = \frac{\dot{\Sigma}_{\text{p,i}}}{f_{\text{i}}}. \tag{19}$$

Because whole-organism entropy production scales strongly with body mass, cross-species comparisons are naturally expressed in mass-specific form. For an organism of mass M_{i}, define

$$q^{*} \equiv \frac{1}{M_{\text{i}}}\frac{d\Sigma_{\text{p,i}}}{d\vartheta_{\text{i}}} = \frac{\dot{\Sigma}_{\text{p,i}}}{M_{\text{i}} f_{\text{i}}}. \tag{20}$$

If P_{i} denotes metabolic power and T_{i} the characteristic temperature at which dissipation occurs, the leading thermodynamic estimate is

$$\dot{\Sigma}_{\mathrm{p,i}} \simeq \frac{P_{\mathrm{i}}}{T_{\mathrm{i}}}. \tag{21}$$

Consequently,

$$\sigma^{*}_{\mathrm{i}} \simeq \frac{P_{\mathrm{i}}/M_{\mathrm{i}}}{T_{\mathrm{i}} f_{\mathrm{i}}}. \tag{22}$$

The differential thermodynamic closure of PBTE can therefore be written as

$$\frac{d\Sigma_{\mathrm{p,i}}}{M_{\mathrm{i}}} = \sigma^{*}_{\mathrm{i}}\, d\vartheta_{\mathrm{i}}. \tag{23}$$

Using $d\vartheta_{\mathrm{i}} = f_{\mathrm{i}}(t)\, dt$,

$$\frac{d\Sigma_{\mathrm{p,i}}}{M_{\mathrm{i}}} = \sigma^{*}_{\mathrm{i}}(t) f_{\mathrm{i}}(t)\, dt. \tag{24}$$

Integration over the lifetime gives

$$\frac{\Sigma^{\mathrm{life}}_{\mathrm{p,i}}}{M_{\mathrm{i}}} = \int_{0}^{N_i} \sigma^{*}_{\mathrm{i}}(\vartheta)\, d\vartheta. \tag{25}$$

If the cycle-averaged mass-specific entropy cost is defined by

$$\bar{\sigma}^{*}_{\mathrm{i}} \equiv \frac{1}{N_{\mathrm{i}}} \int_{0}^{N_i} \sigma^{*}_{\mathrm{i}}(\vartheta)\, d\vartheta, \tag{26}$$

then

$$\frac{\Sigma^{\mathrm{life}}_{\mathrm{p,i}}}{M_{\mathrm{i}}} = \bar{\sigma}^{*}_{\mathrm{i}} N_{\mathrm{i}}. \tag{27}$$

Under the previously stated PBTE closure

$$\bar{q}^{*} \simeq q^{*}_{0,\mathrm{C}}, \tag{28}$$

together with the empirical lifetime relation $N_{\mathrm{i}} \simeq N_{\star,\mathrm{C}}$, one obtains

$$\frac{\Sigma^{\mathrm{life}}_{\mathrm{p,i}}}{M_{\mathrm{i}}} \simeq \sigma^{*}_{0,\mathrm{C}} N_{\star,\mathrm{C}}. \tag{29}$$

The cycle invariant and the thermodynamic invariant are therefore two descriptions of the same lifetime constraint. The former counts biological events; the latter weights those events by their irreversible entropy cost [33, 36]. Here $\Sigma_{\mathrm{p,i}}$ denotes accumulated entropy production rather than entropy stored within the organism. Living systems maintain internal organization by exporting entropy, and PBTE concerns the irreversible throughput associated with that maintenance.

An entropy-normalized biological age may consequently be defined as

$$A_{\mathrm{PBTE,i}}(t) = \frac{1}{N_{\star,\mathrm{ref}}} \int_0^t \frac{\sigma_{\mathrm{i}}^*(s)}{\sigma_{0,\mathrm{ref}}^*} f_{\mathrm{i}}(s)\, ds. \tag{30}$$

Introducing the reference mass-specific lifetime entropy budget

$$\Sigma_{\mathrm{ref}}^* \equiv \sigma_{0,\mathrm{ref}}^* N_{\star,\mathrm{ref}}, \tag{31}$$

Eq. (30) is equivalently

$$A_{\mathrm{PBTE,i}}(t) = \frac{\Sigma_{\mathrm{p,i}}(t)/M_{\mathrm{i}}}{\Sigma_{\mathrm{ref}}^*}. \tag{32}$$

The physiological and aging consequences of this functional were developed in the preceding PBTE work and are not re-derived here. The point relevant to the present paper is that Eqs. (3) and (30) have so far been parametrized by an external time coordinate t.

For ordinary terrestrial comparisons this convention is adequate because the difference between terrestrial coordinate time and local proper time is negligible at the level of biological measurement. It is not, however, the fundamental formulation. Special relativity replaces the Newtonian notion of a universal elapsed time with proper time attached to an individual worldline. For an object moving at speed v_{i} relative to an inertial coordinate system,

$$d\tau_{\mathrm{i}} = dt\sqrt{1 - \frac{v_{\mathrm{i}}^2}{c^2}} = \frac{dt}{\gamma_{\mathrm{i}}}, \qquad \gamma_{\mathrm{i}} = \frac{1}{\sqrt{1 - v_{\mathrm{i}}^2/c^2}}. \tag{33}$$

The elapsed proper time along the moving worldline is therefore smaller than the elapsed coordinate time assigned by the external inertial frame. This does not mean that the traveler experiences a local slowing of physiology. In the traveler's instantaneous rest frame, atomic transitions, chemical reactions, metabolism, cardiac activity, and cognition proceed normally. Time dilation arises when elapsed durations along distinct worldlines are compared.

The physical status of Eq. (33) is independent of PBTE. The relation is supported by measurements of relativistic particle lifetimes, transported atomic clocks, precision optical clocks, and satellite timing systems [1, 3, 5, 13, 14]. These observations are not presented as experimental confirmations of biological time equivalence. Their relevance is more fundamental: they establish that local physical processes are parametrized by proper time. Muon decay, atomic oscillation, chemical change, and physiological activity do not obey separate relativistic laws.

The same principle admits a geometric expression in a general spacetime. For a timelike worldline x_{i}^{μ}, with metric signature (−, +, +, +),

$$d\tau_{\mathrm{i}} = \frac{1}{c}\sqrt{-g_{\mu\nu}\, dx_{\mathrm{i}}^{\mu} dx_{\mathrm{i}}^{\nu}}. \tag{34}$$

Equation (33) is the flat-spacetime inertial limit of Eq. (34). The special-relativistic case is sufficient to expose the central PBTE issue: if physiology evolves locally according to proper time,

then the expression

$$d\vartheta_i = f_i(t)\, dt \tag{35}$$

is only a nonrelativistic shorthand. Its invariant replacement is

$$d\vartheta_i = f_i(\tau_i)\, d\tau_i. \tag{36}$$

Correspondingly, the thermodynamic increment becomes

$$\frac{d\Sigma_{p,i}}{M_i} = \sigma^*(\tau_i) f_i(\tau_i)\, d\tau_i. \tag{37}$$

Relative to an external coordinate time t,

$$\frac{d\vartheta_i}{dt} = f_i(\tau_i)\frac{d\tau_i}{dt}. \tag{38}$$

For uniform motion in flat spacetime this reduces to

$$\frac{d\vartheta_i}{dt} = \frac{f_i(\tau_i)}{\gamma_i}. \tag{39}$$

This relation establishes the division of physical roles on which the remainder of the paper is based. Relativity determines the amount of proper time accumulated along an organism's world-line. PBTE determines the amount of physiological progression and irreversible thermodynamic expenditure accumulated per unit proper time. Relativistic biological time dilation is therefore not produced by a separate slowing of metabolism. It is the consequence of expressing an intrinsically proper-time-parametrized biological process relative to an external coordinate clock.

3 Covariant Relativistic PBTE and Its Physical Consequences

The preceding formulation identifies two intrinsic quantities associated with a living system. Relativistic proper time, τ, measures the invariant duration accumulated along the system's spacetime worldline, whereas biological proper time, ϑ, measures the accumulated number of physiologically meaningful events occurring during that duration. The relativistic completion of PBTE follows by requiring every biological and thermodynamic rate to be defined locally with respect to τ, rather than with respect to an observer-dependent coordinate time t.

Let Σ_p denote the cumulative irreversible entropy production of the organism and let

$$\dot{\Sigma}_p(\tau) \equiv \frac{d\Sigma_p}{d\tau} \tag{40}$$

denote the entropy-production rate measured in its local rest frame. The entropy-normalized PBTE age is then

$$\boxed{A_{\mathrm{PBTE}}(\tau) = \frac{1}{\Sigma_{\mathrm{ref}}} \int_{\tau_0}^{\tau} \dot{\Sigma}_{\mathrm{p}}(\tau')\, d\tau'} \tag{41}$$

where Σ_{ref} is the specified reference lifetime entropy budget. Equation (41) is the central result of the present work. It states that biological age is an irreversible thermodynamic functional evaluated along the organism's worldline. The value of A_{PBTE} is therefore independent of the coordinate system used to describe that worldline.

The corresponding aging rate relative to an external coordinate time follows directly from the chain rule:

$$\frac{dA_{\mathrm{PBTE}}}{dt} = \frac{1}{\Sigma_{\mathrm{ref}}} \frac{d\Sigma_{\mathrm{p}}}{d\tau} \frac{d\tau}{dt}. \tag{42}$$

For uniform inertial motion in flat spacetime,

$$\frac{d\tau}{dt} = \sqrt{1 - \frac{v^2}{c^2}} = \frac{1}{\gamma}, \qquad \gamma = \frac{1}{\sqrt{1 - v^2/c^2}}, \tag{43}$$

and Eq. (42) becomes

$$\boxed{\frac{dA_{\mathrm{PBTE}}}{dt} = \frac{\dot{\Sigma}_{\mathrm{p}}}{\gamma \Sigma_{\mathrm{ref}}}} \tag{44}$$

with $\dot{\Sigma}_{\mathrm{p}} = d\Sigma_{\mathrm{p}}/d\tau$ evaluated in the organism's local rest frame. In the weak-field and slow-motion limit,

$$\frac{dA_{\mathrm{PBTE}}}{dt} \simeq \frac{\dot{\Sigma}_{\mathrm{p}}}{\Sigma_{\mathrm{ref}}} \left(1 + \frac{\Phi}{c^2} - \frac{v^2}{2c^2}\right), \tag{45}$$

where Φ is the Newtonian gravitational potential along the worldline. Equations (44) and (45) separate two physically distinct contributions to externally observed biological aging. The local entropy-production rate describes the physiological and thermodynamic state of the organism, whereas $d\tau/dt$ describes the geometry of its worldline. Neither contribution is reducible to the other.

This distinction resolves a common causal misconception. A moving organism does not age more slowly because velocity directly suppresses its metabolism. In its instantaneous local rest frame, metabolism, cardiac activity, molecular turnover, neural activity, and every other internal process proceed normally. The reduction observed from another frame arises because less proper time accumulates along the moving worldline between the chosen comparison events. The local relation

$$\frac{dA_{\mathrm{PBTE}}}{d\tau} = \frac{\dot{\Sigma}_{\mathrm{p}}}{\Sigma_{\mathrm{ref}}} \tag{46}$$

is unchanged by uniform inertial motion. What changes is the conversion between local proper

time and external coordinate time.

The same reasoning applies to the physiological cycle count. The nonrelativistic expression $d\vartheta = f(t)\,dt$ is replaced by

$$d\vartheta = f(\tau)\,d\tau, \tag{47}$$

where $f(\tau)$ is the local physiological frequency measured per unit proper time. Relative to an external coordinate time,

$$\frac{d\vartheta}{dt} = f(\tau)\frac{d\tau}{dt}, \tag{48}$$

and for inertial motion,

$$\frac{d\vartheta}{dt} = \frac{f(\tau)}{\gamma}. \tag{49}$$

Every local biological clock is therefore assigned the same global relativistic factor when expressed relative to the same external coordinate time. Heart rate, respiration, circadian phase, cell division, chemical turnover, and molecular aging are not independently dilated. They are locally normal processes sharing a common proper-time parameterization.

The distinction between PBTE scaling and relativistic time dilation can now be stated precisely. The mouse–elephant comparison concerns two organisms with different intrinsic physiological frequencies occupying approximately the same spacetime environment. Their difference is primarily biological:

$$\frac{d\vartheta_{\text{mouse}}}{d\vartheta_{\text{elephant}}} \simeq \frac{f_{\text{mouse}}}{f_{\text{elephant}}} \simeq 20. \tag{50}$$

By contrast, the relativistic comparison concerns systems following different spacetime worldlines. Their difference is geometric and is expressed through the ratio of their proper-time rates. For two organisms i and j described in a common coordinate system, both effects combine as

$$\frac{d\vartheta_i/dt}{d\vartheta_j/dt} = \underbrace{\frac{f_i(\tau_i)}{f_j(\tau_j)}}_{\text{physiological factor}} \times \underbrace{\frac{d\tau_i/dt}{d\tau_j/dt}}_{\text{spacetime factor}}. \tag{51}$$

Equation (51) provides a compact unification without confusing the two mechanisms. If the organisms occupy the same terrestrial environment, the proper-time ratio is effectively unity and the relation reduces to ordinary PBTE scaling. If identical organisms follow different worldlines while maintaining the same local physiology, the physiological ratio is unity and only the relativistic factor remains. If both physiology and worldline differ, their effects compose multiplicatively.

A simple example makes the interpretation transparent. Consider an organism traveling at $v = 0.8c$ relative to Earth. The Lorentz factor is

$$\gamma = \frac{1}{\sqrt{1 - 0.8^2}} = \frac{5}{3}, \qquad \frac{d\tau}{dt} = 0.6. \tag{52}$$

During one year of Earth coordinate time, the traveler accumulates 0.6 years of proper time. If its locally measured resting heart rate is 70 min^{-1}, its accumulated cardiac count is

$$\Delta\vartheta_{\text{traveler}} = 70 \times 0.6 \times 525{,}960 \simeq 2.21 \times 10^7 \tag{53}$$

beats. An otherwise identical organism remaining at rest relative to Earth accumulates

$$\Delta\vartheta_{\text{Earth}} = 70 \times 525{,}960 \simeq 3.68 \times 10^7 \tag{54}$$

beats during the same Earth-coordinate interval. The ratio is

$$\frac{\Delta\vartheta_{\text{traveler}}}{\Delta\vartheta_{\text{Earth}}} = 0.6 = \frac{1}{\gamma}. \tag{55}$$

The traveler nevertheless measures a normal local heart rate of 70 min^{-1}. After correcting for signal-propagation and Doppler effects, Earth assigns a rate of 42 beats per Earth-coordinate minute. The reduced accumulated heartbeat count is thus a consequence of the smaller proper-time interval, not of impaired cardiac function.

Gravity produces the corresponding effect through gravitational time dilation. For a stationary worldline at radius r in the Schwarzschild geometry,

$$d\tau = dt\sqrt{1 - \frac{2GM}{rc^2}}. \tag{56}$$

An organism deeper in the gravitational potential accumulates less proper time per unit of the distant observer's coordinate time. If its local physiological state remains unchanged, its externally assigned PBTE aging rate is reduced by the same factor. Near Earth's surface, two stationary systems separated vertically by a small height h have the approximate fractional clock-rate difference

$$\frac{\Delta(d\tau/dt)}{d\tau/dt} \simeq \frac{gh}{c^2}. \tag{57}$$

For $h = 1$ m, this quantity is approximately 1.1×10^{-16}. The effect is measurable with modern optical clocks but overwhelmingly smaller than ordinary biological variability. Its importance here is therefore conceptual rather than biomedical: even at laboratory scales, the fundamental temporal variable remains proper time.

The proper-time formulation may also be written covariantly. Let s^μ denote the entropy four-current of the biological system. The local second law is expressed as

$$\nabla_\mu s^\mu = \sigma \geq 0, \tag{58}$$

where σ is the local entropy-production density. For an organism whose spatial extent is small

relative to the relevant curvature scale, a representative center-of-mass worldline and an instantaneous local rest-frame volume $V(\tau)$ may be used. The total entropy-production rate is then

$$\dot{\Sigma}_{\mathrm{p}}(\tau) = \int_{V(\tau)} \sigma(x, \tau)\, dV_{\mathrm{proper}}. \tag{59}$$

Substitution into Eq. (41) gives

$$\boxed{\mathcal{A}_{\mathrm{PBTE}} = \frac{1}{\Sigma_{\mathrm{ref}}} \int_{\tau_0}^{\tau_1} \int_{V(\tau)} \nabla_\mu s^\mu\, dV_{\mathrm{proper}}\, d\tau} \tag{60}$$

which is independent of the coordinates chosen to describe the worldtube. Equation (60) is the manifestly covariant form of relativistic PBTE. It reduces to the ordinary terrestrial expression when spacetime curvature is weak, velocities are small, and $d\tau \simeq dt$.

The geometrical content required for this construction is correspondingly modest. Biological proper time may be represented by the one-dimensional line element

$$d\ell_{\mathrm{B}} = f(\tau)\, d\tau = d\vartheta, \tag{61}$$

so that the accumulated biological path length is

$$\ell_{\mathrm{B}} = \int f(\tau)\, d\tau. \tag{62}$$

This representation is useful because it distinguishes the amount of proper time supplied by spacetime from the number of intrinsic events accumulated during that interval. It does not define an additional spacetime metric, introduce Lorentz symmetry into biology, or imply an invariant biological speed analogous to c. The relativistic metric is fundamental; the biological line element is a state-dependent measure constructed on top of it.

The framework yields several clear physical consequences. Uniform inertial motion produces no locally detectable physiological change. Identical local biological clocks following different worldlines acquire the same relativistic dilation factor when compared in a common external coordinate system. Physiological perturbations and relativistic effects remain separable:

$$\frac{d\mathcal{A}_{\mathrm{PBTE}}}{dt} = \frac{\dot{\Sigma}_{\mathrm{p}}^{\mathrm{local}}}{\Sigma_{\mathrm{ref}}} \frac{d\tau}{dt}. \tag{63}$$

Radiation, microgravity, inflammation, circadian disruption, stress, or disease may alter the first factor, while velocity and gravitational potential alter the second. Real spaceflight can therefore accelerate local physiological deterioration even while relativistic motion reduces the amount of proper time accumulated per Earth year. The net biological outcome is determined by their product, not by either contribution alone.

Equation (63) also states the principal empirical consistency condition of the theory. After local environmental and physiological effects are accounted for, independent biological clocks should

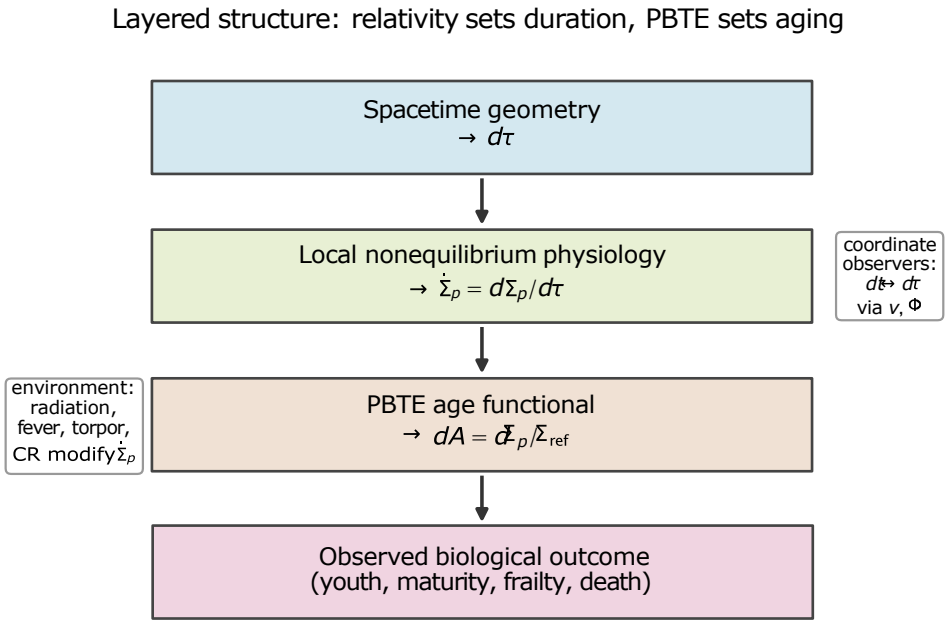

Figure 1: Logical structure of relativistic PBTE. Spacetime geometry determines the proper-time increment $d\tau$. Local physiological state determines the intrinsic biological frequency $f(\tau)$ and entropy production rate $\dot{\Sigma}_p(\tau)$. Biological proper time and PBTE age are then accumulated as $d\vartheta = f\, d\tau$ and $dA_{PBTE} = \dot{\Sigma}_p\, d\tau/\Sigma_{ref}$. Velocity and gravitational potential modify the mapping between coordinate time and proper time; environmental and pathological conditions modify the local physiological terms.

remain mutually synchronized in proper time and should acquire the same global relativistic factor relative to an external coordinate time. A violation in which different isolated internal processes acquired different velocity-dependent factors would contradict the proper-time formulation. Direct biological tests at relativistically significant velocities are not currently feasible, and terrestrial gravitational effects are far below normal biological noise. The experimentally established particle and atomic-clock evidence therefore validates the proper-time factor used here, but it does not by itself validate the biological invariant, the entropy-per-cycle closure, or the PBTE aging functional. Those remain independent biological hypotheses requiring their own comparative and physiological tests.

4 Deviation From Temporal Balance: Localised Symmetry Breaking and Applications

The relativistic functional of Section 3 describes an organism that traverses its biological history along a single worldline. Sections 2–3 treated the normal case, in which an organism completes life having consumed an approximately clade-invariant intrinsic budget. We now ask a complementary

question that the proper-time formulation makes precise: how should one describe systems that depart from that balance, and how is the description modified when the relevant rates are referred to proper time rather than coordinate time? The constructions below are advanced as testable PBTE hypotheses rather than established results; aging itself, and its damage dynamics, are treated in the companion work [33] and are not re-examined here. Our focus is the parametrisation of deviation along the worldline and two applications—inflammatory bias and viral latency—that are natural consequences of it.

4.1 A temporal order parameter referred to proper time

To quantify how much of an organism's intrinsic budget has been spent, it is convenient to introduce a single dimensionless coordinate. Let $N(\tau) = \int_{\tau_0}^{\tau} f(\tau')\, d\tau'$ be the intrinsic cycle count accumulated along the worldline, with f the local physiological frequency per unit proper time of Eq. (47). Define the *temporal order parameter*

$$\zeta(\tau) = \frac{N(\tau)}{N_\star} = \frac{1}{N_\star} \int_{\tau_0}^{\tau} f(\tau')\, d\tau', \tag{64}$$

in which $N_\star$ is the reference intrinsic budget introduced in Section 2. The quantity ζ is a biological odometer: $\zeta = 0$ at the start of the trajectory and, on the reference manifold, $\zeta = 1$ at its end. Because the integral in Eq. (64) is taken over proper time, ζ is itself an invariant of the worldline, independent of the external coordinate used to describe it, exactly as A_{PBTE} was shown to be in Eq. (41). Using the thermodynamic closure of Section 2, $d\Sigma_{\mathrm{p}} = \sigma_0\, d\vartheta$, and the lifetime budget $\Sigma_\star = \sigma_0 N_\star$, Eq. (64) acquires an equivalent thermodynamic form,

$$\zeta(\tau) = \frac{\Sigma_{\mathrm{p}}(\tau)}{\Sigma_\star}, \tag{65}$$

so that ζ is simultaneously the fraction of the intrinsic cycle budget and the fraction of the lifetime entropy budget consumed. Comparison with Eq. (41) shows that ζ and the PBTE age A_{PBTE} coincide when the reference budgets are chosen consistently; ζ is therefore the natural state variable in which deviations from temporal balance are expressed. Three regimes follow directly from Eq. (64): a balanced trajectory with $\zeta \to 1$ as the worldline ends, a *hypertemporal* trajectory with $\zeta > 1$ in which the budget is over-consumed, and a *hypotemporal* trajectory with $\zeta < 1$ in which it is under-consumed.

4.2 Relaxation dynamics, physiological noise, and inflammatory bias

A healthy organism does not merely possess a value of ζ; it actively maintains ζ near its reference trajectory against perturbation. To represent this regulation, define the fractional deviation $\phi(\tau) = \zeta(\tau) - \tau/\tau_{\mathrm{exp}}$ from the expected reference path, where τ_{exp} is the proper time at which a balanced organism reaches $\zeta = 1$. Near the reference manifold the simplest dynamics consistent with

homeostatic restoration are linear, giving an Ornstein–Uhlenbeck relaxation along the worldline,

$$\frac{d\phi}{d\tau} = -\frac{\phi(\tau)}{\tau_\zeta} + \eta(\tau) + u(\tau), \tag{66}$$

in which each term has a definite role. The restoring term $-\phi/\tau_\zeta$ pulls the system back toward balance, and the *temporal resilience time* $\tau_\zeta > 0$ sets the rate of recovery: small τ_ζ denotes a resilient system that corrects perturbations quickly, large τ_ζ a fragile one. The term $\eta(\tau)$ represents zero-mean stochastic physiological fluctuations, $\langle \eta(\tau) \rangle = 0$, $\langle \eta(\tau)\eta(\tau') \rangle = 2D_\eta\, \delta(\tau - \tau')$, with noise intensity D_η. The term $u(\tau)$ is a sustained deterministic bias—an external drive that displaces the organism systematically from balance. Chronic inflammation is the paradigmatic positive bias: by raising metabolic demand and repair load it acts in Eq. (66) as a persistent $u > 0$ that pushes ϕ upward, driving the system toward the hypertemporal regime; a sustained suppression of metabolic rate enters instead as $u < 0$. The advantage of expressing Eq. (66) in proper time is that the resilience time τ_ζ and the bias u are then intrinsic properties of the organism, cleanly separable from the relativistic factor $d\tau/dt$ that governs how the worldline is observed externally.

Equation (66) makes a falsifiable prediction. Its stationary fluctuation spectrum is the Lorentzian

$$S_\phi(\omega) = \frac{2D_\eta\, \tau_\zeta^2}{1 + \omega^2 \tau_\zeta^2}, \tag{67}$$

where ω is the angular frequency conjugate to proper time. As the resilience time τ_ζ increases, the corner frequency $\omega_c = 1/\tau_\zeta$ of Eq. (67) shifts to lower values and fluctuations become slower and more strongly correlated. Figure 2 plots Eq. (67) for three resilience times and shows this leftward migration of spectral weight. A loss of temporal resilience is therefore expected to announce itself, before any change in mean physiological state, as a slowing and low-frequency amplification of fluctuations in measurable signals such as heart-rate variability, temperature, glucose, sleep rhythm, and inflammatory markers—a biological analogue of critical slowing down near a transition.

4.3 Localised symmetry breaking: a spatially resolved order parameter

In a multicellular organism the whole-organism worldline may remain balanced while a localised tissue or an embedded pathogen runs an internal clock that departs sharply from the organism-wide average. To represent this, promote the order parameter to a field over the proper-time slices of the worldtube,

$$\zeta(x, \tau) = \frac{f(x, \tau)\, \tau_{\mathrm{loc}}(x, \tau)}{N_\star}, \tag{68}$$

where $f(x, \tau)$ is the local intrinsic frequency and τ_{loc} the local persistence time at proper-time-labelled position x. Homeostatic coupling between neighbouring tissue, together with a local pathological drive, gives the reaction–diffusion dynamics

$$\tau_\zeta\, \partial_\tau \zeta = -(\zeta - 1) + D_\zeta \nabla^2 \zeta + S(x, \tau), \tag{69}$$

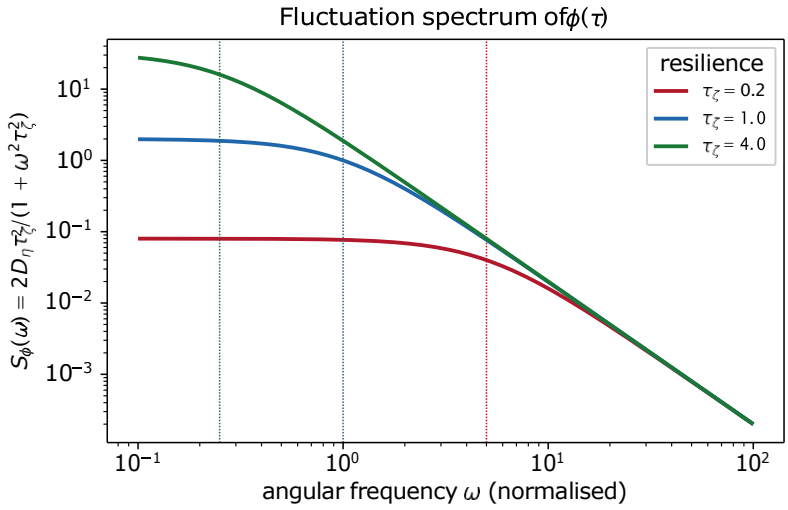

Figure 2: Fluctuation spectrum of the temporal-deviation variable ϕ. The Lorentzian spectrum of Eq. (67) is shown for three values of the resilience time τ_ζ; dotted lines mark the corner frequencies $\omega_c = 1/\tau_\zeta$. As τ_ζ grows, spectral weight migrates to low frequencies, so that a fragile system recovering slowly from perturbation displays slower, more correlated fluctuations. The prediction that such low-frequency amplification precedes a transition from temporal balance to the hypertemporal regime is directly testable in physiological time series.

whose three terms are, in order, relaxation of the local clock toward the homeostatic value $\zeta = 1$, spatial coupling that synchronises adjacent tissue with effective diffusivity D_ζ, and a source $S(x, \tau)$ encoding persistent local drivers of temporal dysregulation. The balance of the first two terms defines a characteristic healing length $\ell_\zeta = \sqrt{D_\zeta \tau_\zeta}$ over which a local defect in biological time is repaired by its surroundings. Equation (69) reduces to the whole-organism relaxation (66), without noise, when ζ is spatially uniform and S plays the role of the bias u. Figure 3(a) shows two opposite stationary profiles of Eq. (69) that organise the applications below.

4.4 Application: localised acceleration and viral latency

Two limiting forms of Eq. (69) are of direct biological interest, and both are forms of spontaneous temporal symmetry breaking. In the first, a localised population sustains a source $S > 0$ strong enough to hold $\zeta(x, \tau) \gg 1$ within a bounded region while the surrounding tissue remains near $\zeta = 1$. The accompanying elevation of local entropy production per cell—of which the preferential use of aerobic glycolysis is a recognised signature [38]—and the steep spatial gradients $\nabla\zeta$ at the domain boundary disrupt the intercellular temporal coordination that normally maintains tissue homeostasis. Proliferative pathology is, in this language, a domain of locally accelerated biological time embedded in a balanced organism; we state this as a PBTE interpretation to be tested, not as a mechanistic claim about causation.

In the second limit the local frequency is driven nearly to zero. During the latent phase of an

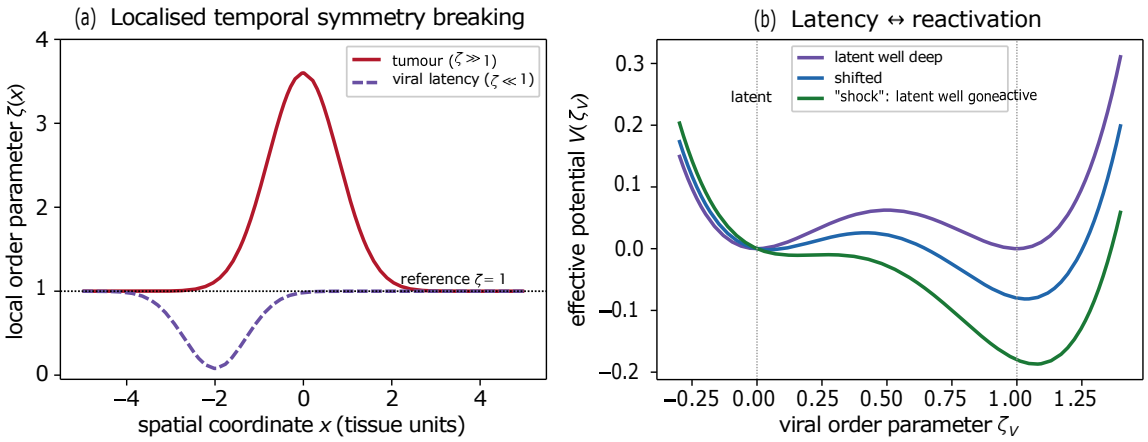

Figure 3: Localised temporal symmetry breaking. (a) Stationary spatial profiles of the local order parameter $\zeta(x)$ from Eq. (69): a domain of accelerated local time ($\zeta \gg 1$, solid) and a domain of near-arrested local time ($\zeta \ll 1$, dashed), relaxing toward the homeostatic value $\zeta = 1$ over the healing length $\ell_\zeta = \sqrt{D_\zeta \tau_\zeta}$. (b) Effective bistable potential $V(\zeta_V)$ for an embedded pathogen: a deep latent well near $\zeta_V \approx 0$ and an active-replication state near $\zeta_V \approx 1$ are separated by a barrier; a latency-reversal drive shifts the potential so that the latent well disappears.

infection the replication frequency $f_V \approx 0$, so that, from Eq. (68),

$$\zeta_V^{(\ell)} = \frac{f_V \, \tau_{\mathrm{loc}}}{N_\star} \ll 1, \tag{70}$$

signalling near-arrest of the pathogen's biological time while the host's proper time continues to advance normally. Latency is thus a state in which two coupled systems sharing the same spacetime worldtube occupy opposite extremes of the order parameter. Reactivation is the escape of ζ_V from the latent value toward $\zeta_V \approx 1$, naturally modelled as a transition in the bistable effective potential of Figure 3(b): a deep well near $\zeta_V \approx 0$ (latency) and a second minimum near $\zeta_V \approx 1$ (active replication) separated by a barrier. Latency-reversal strategies correspond to deforming that potential until the latent well becomes unstable, driving the deliberate transition $\zeta_V \ll 1 \to \zeta_V \approx 1$ [8]. The same construction places host–pathogen encounters in the language of interacting clocks: productive infection requires the pathogen and the host response to occupy commensurate values of ζ within a finite window, a condition expressed compactly by the proper-time-referred frequencies of Section 3.

4.5 Application: restoration as optimal control

If deviation from temporal balance is undesirable, its correction can be posed as an optimal control problem, which is the practical payoff of expressing biological state through ζ. One seeks the

intervention $u(\tau)$ in Eq. (66) that minimises the quadratic cost

$$J[u] = \int_{\tau_0}^{\tau_1} \left[\phi(\tau)^2 + \lambda u(\tau)^2 \right] d\tau, \tag{71}$$

in which the first term penalises residual deviation of the biological clock from its reference trajectory and the second penalises the burden of intervention, with $\lambda > 0$ setting their relative weight. The minimiser of Eq. (71) is a linear state feedback, $u^\star(\tau) \propto -\phi(\tau)$: one measures how far the system has drifted and applies the smallest correction that restores balance. Candidate physiological channels through which such a control could act include circadian entrainment by timed light and feeding, intermittent caloric restriction, mild thermal modulation, and, for the latency application, latency-reversal agents that reshape the potential of Figure 3(b). Because ζ is estimable in principle from wearable physiological data, Eqs. (66) and (71) together suggest a closed-loop architecture—continuous estimation of ζ, comparison with the reference trajectory, and delivery of a timed intervention—in which relativistic corrections enter only through the conversion $d\tau/dt$ of Section 3 and are negligible for terrestrial application. These proposals define an empirical programme; their clinical status remains to be established.

5 Discussion and Conclusion

The purpose of the present work is not to construct a biological alternative to special or general relativity. It is to place PBTE within the temporal structure already established by relativity. This distinction prevents two opposite conceptual errors. The first is to regard biological proper time as merely metaphorical. In PBTE, biological proper time is an operational quantity: it is the accumulated count of specified intrinsic events,

$$\vartheta = \int f(\tau)\, d\tau. \tag{72}$$

The second error is to identify that quantity with Einsteinian proper time. The two are not identical. Relativistic proper time is a geometric duration determined by the spacetime metric; biological proper time is a state-dependent accumulation of physiological events occurring during that duration. The correct relation is hierarchical:

$$t \longrightarrow \tau \longrightarrow \vartheta \longrightarrow A_{\mathrm{PBTE}}. \tag{73}$$

Coordinate time t describes events relative to a chosen reference system. Proper time τ gives the invariant duration along the organism's worldline. Biological proper time ϑ counts internal physiological events accumulated along that worldline. PBTE age A_{PBTE} weights irreversible physiological activity relative to a reference lifetime budget.

The principal theoretical advance is therefore a covariant completion of the PBTE functional. In its nonrelativistic form, biological age was expressed as an integral over terrestrial chronological

time. That expression is adequate whenever $d\tau \simeq dt$, as it is for ordinary comparative biology. It is not, however, invariant under changes of spacetime trajectory. Equation (41) removes this dependence by defining biological age directly along the organism's proper-time worldline.

This result clarifies the relationship between the familiar mouse–elephant comparison and Einsteinian time dilation. A mouse and an elephant accumulate biological events at different rates because their local physiological frequencies differ. Identical twins following different relativistic worldlines accumulate different amounts of biological history because their elapsed proper times differ. The first is a physiological rescaling; the second is a geometric effect. They meet in Eq. (51), which shows that biological and relativistic clock-rate differences compose multiplicatively while remaining conceptually and experimentally distinguishable.

The formulation also places an important limit on the interpretation of entropy within PBTE. Entropy production does not generate the spacetime metric, and it is not presented here as the cause of relativistic time dilation. Atomic clocks and unstable particles accumulate proper time without possessing biological entropy budgets. Conversely, living systems are distinctive not because they alone possess time, but because they transform proper-time duration into organized physiological history, memory, repair, adaptation, damage, and eventually loss of function. Spacetime supplies duration; irreversible thermodynamics supplies direction; biological organization converts irreversible activity into age.

The phrase "time is entropy's arrow; life is entropy's clock" is therefore defensible only when interpreted with this hierarchy in mind. The thermodynamic arrow distinguishes irreversible histories, while the PBTE clock records a biologically selected subset of entropy-producing events. A nonliving object may undergo entropy production and physical change without possessing a regulated lifetime cycle budget. A living organism, by contrast, maintains itself through continuous energetic throughput and thereby converts irreversible expenditure into an internally structured trajectory.

Nature more generally contains a hierarchy of characteristic internal times. A star possesses a nuclear-burning timescale, a mountain a geological weathering timescale, a radioactive nucleus a decay timescale, a cell a division-and-repair timescale, and a virus a host-dependent replication timescale. These are not separate spacetime dimensions but process coordinates generated by the characteristic rates of the systems concerned. PBTE identifies a more specific structure within living matter. Living systems do not merely undergo change; they actively maintain a low-entropy organization through continuous free-energy consumption, repair, molecular turnover, regulation, and entropy export. Their internal time is therefore simultaneously kinematic and thermodynamic: physiological cycles supply the ticks, while entropy production supplies the irreversible cost of advancing them. A multicellular organism moreover contains many such clocks at once—cardiac, respiratory, circadian, immune, neural, metabolic, reproductive, and molecular—which need not remain perfectly synchronized. PBTE does not require that one clock be universally dominant; it provides a framework in which these clocks may be coarse-grained, compared, and weighted according to their contribution to organismal maintenance and irreversible biological burden.

Several claims lie outside the present theory. The framework does not claim that only living systems measure time, that a body causes relativistic time dilation through metabolic slowing, that zero entropy production implies the cessation of proper time, or that the approximate lifetime invariant $N_\star$ has the exact status of a fundamental constant. Relativistic proper time is fundamental within the spacetime theory; PBTE is an emergent biological model whose invariant, closure relations, and reference budgets must be evaluated empirically within specified domains.

For ordinary terrestrial organisms, relativistic corrections are negligible compared with physiological variation. Differences caused by sleep, infection, temperature, stress, diet, activity, inflammation, and genetic background exceed the kinematic and gravitational factors by many orders of magnitude. The covariant formulation is nevertheless necessary at the conceptual level. A proposed law of biological age should identify the correct independent temporal variable even when the correction is too small to matter in everyday practice.

This interpretation has concrete consequences, because it decomposes biological aging into three experimentally distinguishable elements: the rate of internal ticking, the irreversible cost associated with each tick, and the effective lifetime budget over which those costs can be sustained. Torpor and hibernation primarily lower the local accumulation rate of physiological cycles; improved mitochondrial coupling may reduce entropy production per effective cycle; enhanced repair and damage clearance may change the fraction of dissipation that contributes to persistent aging; and evolutionary adaptation may alter the effective budget itself. These mechanisms should not be treated as interchangeable merely because each can extend chronological lifespan, since each generates a different PBTE signature and therefore different empirical predictions. The same decomposition allows disease to be read as temporal dysregulation: chronic inflammation, cancer, sustained sympathetic activation, metabolic disease, or repeated repair failure may create a hypertemporal state by raising physiological rate, raising irreversible cost per cycle, or accelerating the conversion of energetic throughput into persistent damage. A clinical assessment would then require both a state measure—how much biological distance has already been traversed—and a rate measure—how rapidly the remaining trajectory is being consumed. The framework extends naturally to spaceflight, where environmental stressors modify local physiology while velocity and gravitational potential independently modify the available proper time, the two contributions remaining separable through the factorized relativistic PBTE law of Eq. (63).

The wider implication is a change in the status assigned to calendar time throughout biology. Chronological time remains indispensable as an external coordinate, but it is no longer assumed to be a complete measure of development, aging, disease progression, or ecological interaction. Equal calendar intervals can contain unequal quantities of physiological work, entropy production, repair, replication, and accumulated biological history. Comparative experiments may therefore become more informative when organisms are matched not only by chronological age but also by physiological-cycle count or entropy-normalized PBTE age. Host–pathogen dynamics can be viewed as interactions between clocks running at different intrinsic rates, in which immune success or failure may depend on whether those rates synchronize within a finite response window; ecological

relationships similarly couple organisms whose reproductive, metabolic, and seasonal clocks occupy different parts of a temporal hierarchy. At the conceptual level, PBTE removes the presumption of a single universal biological present. Spacetime supplies proper duration, thermodynamics supplies irreversible direction, physiology supplies intrinsic ticks, and PBTE supplies the transformation and normalization through which different living times become comparable. What changes is not the physics of time but the biological meaning assigned to its passage: the same external hour can contain radically different amounts of life.

The resulting synthesis can be stated compactly. Relativity determines how much proper time is available along a worldline. Local physiology determines how rapidly biological cycles and entropy production accumulate during that proper time. PBTE converts the resulting irreversible expenditure into a normalized aging coordinate:

$$\boxed{A_{\mathrm{PBTE}} = \frac{1}{\Sigma_{\mathrm{ref}}} \int_{\tau_0}^{\tau_1} \dot{\Sigma}_{\mathrm{p}}(\tau)\, d\tau} \tag{74}$$

with the coordinate-time representation

$$\boxed{\frac{dA_{\mathrm{PBTE}}}{dt} = \frac{\dot{\Sigma}_{\mathrm{p}}}{\Sigma_{\mathrm{ref}}} \frac{d\tau}{dt}.} \tag{75}$$

The organism does not make relativistic time run slowly. It undergoes physiological change during the proper time accumulated along its worldline. Relativity determines the duration of that worldline; PBTE describes the irreversible biological history written within it.

Declarations

Data availability. No new empirical dataset is analyzed in this theoretical manuscript. Figures are generated from the analytic relations defined in the text.

Competing interests. The author declares no competing interests.

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Ethics statement. No new research involving human participants or animals was conducted.

A Supplementary Information

This Supplementary Information collects the graphical illustrations, algebraic details, numerical checks, and falsifiability conditions directly required by the relativistic formulation of PBTE. It is intentionally restricted to material that supports the central result of the main text: biological proper time and entropy-normalized biological age must be integrated with respect to relativistic proper time along the organism's worldline.

Figure 4 illustrates the ordinary nonrelativistic PBTE picture. Organisms with different intrinsic physiological frequencies accumulate biological proper time at different rates when described relative to the same terrestrial coordinate time. For an approximately constant cardiac frequency f_{H},

$$\vartheta(t) = f_{\mathrm{H}}\, t\, C_{\mathrm{yr}}, \qquad C_{\mathrm{yr}} = 525{,}960 \text{ min yr}^{-1}, \tag{76}$$

when f_{H} is expressed in beats per minute and t in years. Normalizing by the reference lifetime count $N_0 = 10^9$ gives the fraction of the reference cardiac-cycle budget traversed by chronological age t.

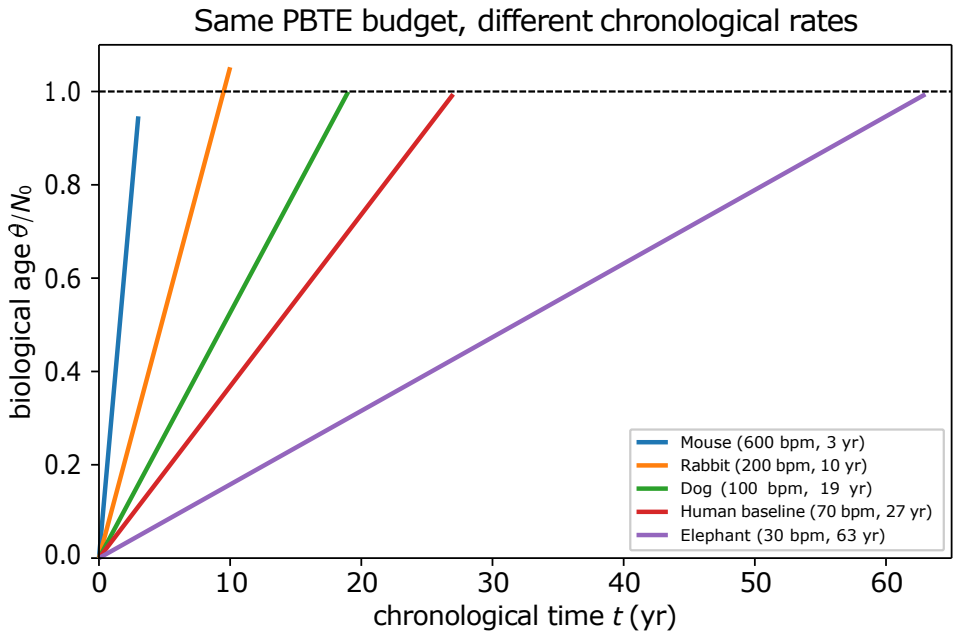

Figure 4: PBTE trajectories for representative organisms in a common terrestrial coordinate time. The ordinate is the normalized accumulated cycle count $\vartheta(t)/N_0$. High-frequency organisms traverse the reference cycle budget rapidly, whereas low-frequency organisms distribute a comparable intrinsic path length over a longer chronological interval. The dashed horizontal line denotes $\vartheta/N_0 = 1$. These trajectories represent physiological rate differences and not relativistic time dilation.

Figure 5 shows the distinct relativistic effect. Consider an organism whose local entropy-production rate per unit proper time remains fixed. Relative to an inertial coordinate time t, its PBTE aging rate is

$$\frac{dA_{\mathrm{PBTE}}}{dt} = \frac{\dot{\Sigma}_{\mathrm{p}}}{\Sigma_{\mathrm{ref}}} \frac{d\tau}{dt}, \tag{77}$$

where $\dot{\Sigma}_{\mathrm{p}} = d\Sigma_{\mathrm{p}}/d\tau$. For uniform motion in flat spacetime,

$$\frac{d\tau}{dt} = \sqrt{1 - \frac{v^2}{c^2}} = \frac{1}{\gamma}, \tag{78}$$

and therefore

$$\frac{dA_{\mathrm{PBTE}}}{dt} = \frac{\dot{\Sigma}_{\mathrm{p}}}{\gamma \Sigma_{\mathrm{ref}}}. \tag{79}$$

The decrease shown in Figure 5 is not a local suppression of metabolism. It is the reduction in proper time accumulated per unit external coordinate time.

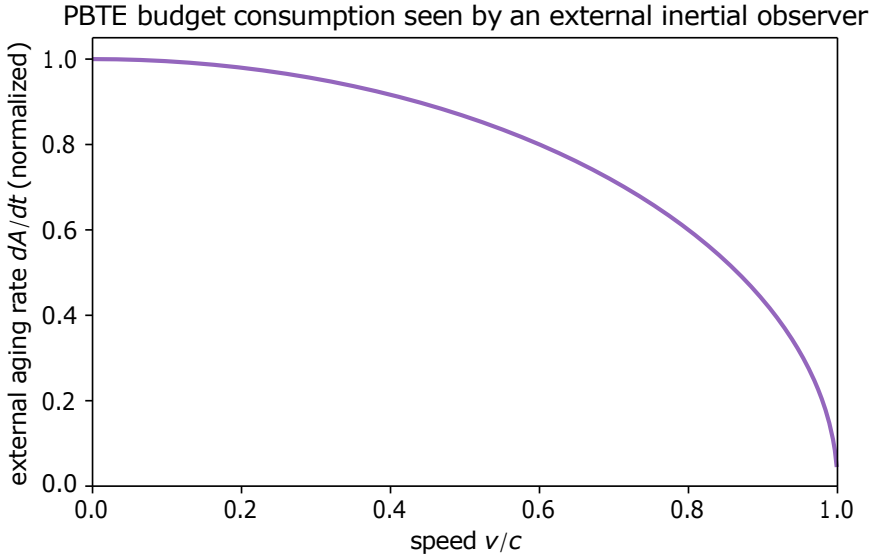

Figure 5: Externally assigned PBTE aging rate for an organism with fixed local entropy production per unit proper time. The plotted quantity is the rate relative to the same organism at rest, $(dA/dt)_v/(dA/dt)_0 = 1/\gamma$. The traveler experiences normal local physiology; the reduction arises because less proper time accumulates along the moving worldline during a fixed interval of external coordinate time.

A third illustration is useful only to preserve the distinction between physiological and relativistic clock-rate changes. In torpor or hibernation, the organism alters its local physiological frequency. This is a genuine biological slowing of cycle accumulation per unit local proper time. It is therefore conceptually different from Lorentz dilation, for which the local frequency remains unchanged and only the relation between $d\tau$ and dt changes.

The combined transformation law follows directly from the separation of these two effects. Let organism i follow the timelike worldline x_i^μ. Its relativistic proper-time increment is

$$d\tau_i = \frac{1}{c}\sqrt{-g_{\mu\nu}\, dx_i^\mu dx_i^\nu}, \tag{80}$$

for metric signature $(-, +, +, +)$. Biological proper time is accumulated locally according to

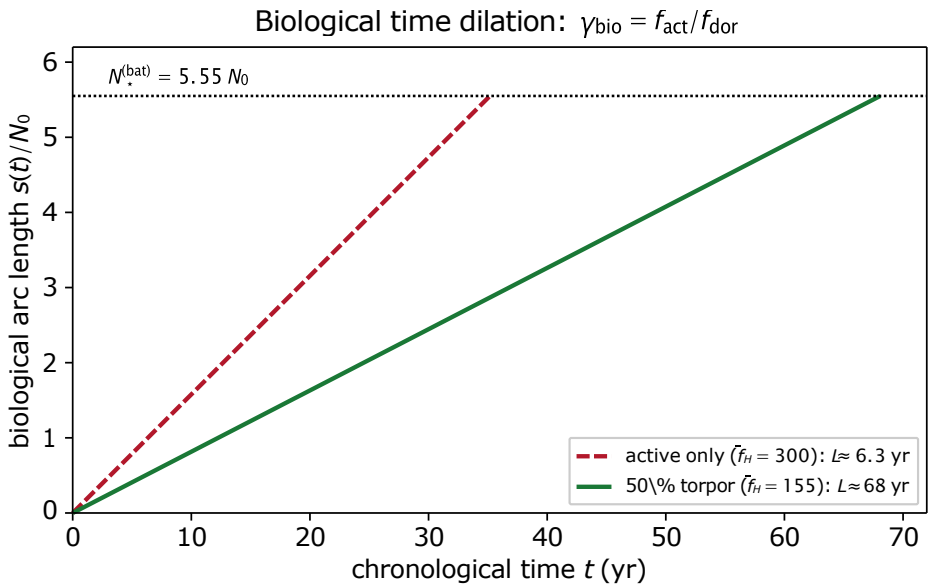

Figure 6: Physiological extension of chronological lifespan in a two-state active–torpor model. The dormant state lowers the locally measured physiological frequency and therefore reduces $d\vartheta/d\tau$. The same cycle budget may consequently be distributed over a longer chronological interval. This mechanism is biological rather than relativistic: torpor changes the local clock rate $f(\tau)$, whereas relativistic dilation changes $d\tau/dt$ while leaving local physiology unchanged.

$$d\vartheta_i = f_i(\tau_i)\, d\tau_i. \tag{81}$$

If the worldline is parametrized by a coordinate time t, then

$$\frac{d\vartheta_i}{dt} = f_i(\tau_i)\frac{d\tau_i}{dt}. \tag{82}$$

For two organisms i and j described in the same coordinate system,

$$\boxed{\frac{d\vartheta_i/dt}{d\vartheta_j/dt} = \frac{f_i(\tau_i)}{f_j(\tau_j)}\,\frac{d\tau_i/dt}{d\tau_i/dt}} \tag{83}$$

follows without additional assumptions. The first ratio is physiological; the second is geometric. If the organisms occupy the same terrestrial environment, then $d\tau_i/dt \simeq d\tau_j/dt$, and Eq. (83) reduces to the ordinary PBTE clock-rate relation. If identical organisms maintain the same local physiology while following different worldlines, then $f_i = f_j$, and the expression reduces to the relativistic proper-time ratio. When physiology and worldline both differ, the two factors compose multiplicatively.

The same derivation applies to entropy-normalized biological age. For

$$A_i = \frac{1}{\Sigma_{ref}} \int \dot{\Sigma}_{p,i}(\tau_i)\, d\tau_i, \tag{84}$$

one obtains

$$\frac{dA_i}{dt} = \frac{\dot{\Sigma}_{p,i}(\tau_i)}{\Sigma_{ref}} \frac{d\tau_i}{dt}. \tag{85}$$

Consequently, the externally assigned aging-rate ratio of two organisms is

$$\frac{dA_i/dt}{dA_j/dt} = \frac{\dot{\Sigma}_{p,i}(\tau_i)}{\dot{\Sigma}_{p,j}(\tau_j)} \frac{d\tau_i/dt}{d\tau_j/dt}, \tag{86}$$

provided the same reference normalization is used. This factorization is the formal basis for separating environmental and physiological influences from relativistic effects.

Table 1 gives the special-relativistic proper-time factor for representative velocities. The final column may be read either as the proper time accumulated during one unit of external coordinate time or as the external PBTE aging rate relative to an identical organism at rest, assuming equal local entropy production.

Table 1: Special-relativistic proper-time and PBTE rate factors. The normalized rate assumes that $dA/d\tau$ is identical at all velocities.

v/c	γ	$d\tau/dt = 1/\gamma$	$(dA/dt)_v/(dA/dt)_0$
0.10	1.005	0.995	0.995
0.50	1.155	0.866	0.866
0.80	1.667	0.600	0.600
0.90	2.294	0.436	0.436
0.99	7.089	0.141	0.141

At $v = 0.8c$, for example, $d\tau/dt = 0.6$. During one Earth year of uniform inertial motion, the traveler accumulates 0.6 years of proper time. A locally measured cardiac frequency of 70 min^{-1} therefore produces

$$\Delta\vartheta_{traveler} = 70 \times 0.6 \times 525{,}960 \simeq 2.21 \times 10^7 \tag{87}$$

cardiac cycles. An otherwise identical organism at rest relative to Earth accumulates

$$\Delta\vartheta_{Earth} = 70 \times 525{,}960 \simeq 3.68 \times 10^7 \tag{88}$$

cycles over the same Earth-coordinate interval. Their ratio is $0.6 = 1/\gamma$. The traveler nevertheless measures a normal local frequency of 70 min^{-1}.

The relativistic extension also imposes explicit consistency and falsifiability conditions. First, the biological foundation remains independently testable: within a prespecified domain, the lifetime quantity

$$N_i = \int_0^{L_i} f_i(t)\, dt \tag{89}$$

must exhibit substantially weaker systematic variation than either frequency or lifespan separately. A strong residual dependence on body mass, temperature, or phylogeny after the stated PBTE corrections would falsify the proposed invariant for that domain.

Second, the thermodynamic closure requires the independently estimated entropy cost per effective physiological cycle,

$$\sigma^*_{\mathrm{i}} = \frac{1}{M_{\mathrm{i}}} \frac{d\Sigma_{\mathrm{p,i}}}{d\vartheta_{\mathrm{i}}}, \tag{90}$$

to remain sufficiently concentrated within the population or clade for which approximate constancy is claimed. Systematic variation over several orders of magnitude would invalidate the simple closure and would require a different physiological weighting.

Third, the relativistic component predicts no locally detectable effect of uniform inertial motion. An organism enclosed in a freely moving laboratory must not infer its uniform velocity from local metabolism, cardiac rhythm, chemical reaction rates, or any other internal experiment. A model predicting direct velocity-dependent physiological suppression in the local rest frame would violate the principle of relativity.

Fourth, after local physiological perturbations are controlled, all internal clocks must inherit the same global proper-time factor relative to a common external coordinate time. Cardiac cycles, respiration, molecular turnover, circadian phase, and other local processes may possess different intrinsic frequencies, but relativity cannot assign them different Lorentz factors. Any such process-specific kinematic dilation would contradict the proper-time formulation.

Fifth, environmental effects must remain separate from spacetime effects. Radiation, microgravity, altered exercise, confinement, circadian disruption, stress, inflammation, and disease may change the local entropy-production rate $\dot{\Sigma}_{\mathrm{p}}$. Velocity and gravitational potential change $d\tau/dt$. The observable aging rate is their product,

$$\frac{dA_{\mathrm{PBTE}}}{dt} = \left(\frac{\dot{\Sigma}_{\mathrm{p}}^{\mathrm{local}}}{\Sigma_{\mathrm{ref}}} \right)^{!} \frac{d\tau}{dt}. \tag{91}$$

Real spaceflight may therefore increase local physiological burden while relativistic motion decreases the amount of proper time accumulated per Earth year. Those effects are physically distinct and may, in principle, oppose one another.

Finally, the present framework does not claim that PBTE has independently confirmed special or general relativity. Particle-decay measurements, atomic clocks, optical clocks, and satellite timing establish the proper-time structure adopted here. They do not establish the PBTE lifetime invariant, the entropy-per-cycle closure, or the biological aging functional. Conversely, evidence for PBTE would not constitute a new test of Lorentz invariance unless a biological clock were compared under conditions in which relativistic effects could be isolated from local physiological perturbations. The contribution of the present theory is the consistent composition of these independently testable structures.

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