

THEORETICAL WHITE PAPER

Cellular Temporal Relativity:

A Framework for Metabolic Clock Modulation and Selective Cellular Aging

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Version History

Version	Date	Changes
1.0	3/23/2026	Initial publication. Core CTR framework established. Three-phase intervention model. Alarm-deaf concept introduced. Energetic delivery mechanism via bioelectric targeting. Bidirectional longevity application.
1.1	3/27/2026	Added metabolic fatigue observation, cancer cells outsource fatigue to the patient via the Warburg effect and angiogenesis. Fourth intervention consideration added: metabolic starvation as a complement to the three-phase model. Three-phase model <u>expanded to four-phase model</u> to include metabolic starvation as the fourth intervention.

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Abstract

This paper proposes a theoretical framework — Cellular Temporal Relativity (CTR) — that structurally parallels Einstein’s theory of special relativity to model the relationship between cellular metabolic rate and the pace of biological aging. While the framework does not apply relativistic physics literally at the cellular level, it asserts that a cell’s intrinsic “clock rate”, governed by ATP turnover, reactive oxygen species (ROS) accumulation, and DNA replication fidelity — functions analogously to a relativistic time frame: a cell operating at higher metabolic throughput experiences accelerated biological aging relative to a cell in a lower-energy state.

The framework further extends this model toward a clinically relevant application: selective temporal modulation. By treating malignant and healthy cells as operating in distinct metabolic “time frames,” targeted energetic interventions may be capable of accelerating aging processes in cancer cells while decelerating them in surrounding healthy tissue. Critically, this paper establishes that cancer cells are not simply fast-aging cells, they are alarm-deaf cells: running a fast metabolic clock while having disabled the biological alarm systems that clock should trigger. The intervention strategy therefore requires not merely clock acceleration, but alarm restoration and immune reactivation, a four-phase model developed fully in Section 4.

This document serves as an original theoretical record, establishing conceptual priority and intellectual provenance, while situating the framework within and beyond current scientific literature.

1. Introduction

Time, in the Einsteinian sense, is not a fixed universal constant. It is a dimension of spacetime that varies based on the velocity of an observer or the strength of the gravitational field they inhabit. This insight, that the passage of time is relative to the physical conditions of an observer’s frame of reference, is one of the most profound contributions to modern physics.

This paper begins with a parallel provocation: what if biological cells, too, inhabit distinct “frames of reference”, not in spacetime, but in metabolic-energetic space? And what if the rate at which a cell experiences biological time is a direct function of the energetic conditions within that frame?

This is not a claim that relativistic physics governs cellular biology. It is a structural and conceptual analogy, a theoretical lens that reframes how we think about cellular aging, opens new questions about interventional targets, and connects two historically separate disciplines: physics and cell biology.

The author arrived at this framework independently, rooted in ideas first explored in 2017 and developed through sustained independent research. The core intuition, that cancer cells could be selectively triggered toward self-destruction by emitting the energy wavelength of their own communication pattern, effectively telling them to destroy themselves in their own language, was formally documented in a PowerPoint presentation created by the author in June 2018 (file metadata: Content created 6/11/2018, Author: Shawn Aslam, 51 revisions, 72 hours and 28 minutes of editing time) and presented to researchers at Fred Hutchinson Cancer Center. This white paper represents the formal theoretical development of that original insight, now situated within current scientific literature and extended into a complete intervention framework.

The framework has since been situated against current literature, where convergent but incomplete findings exist, providing both validation and differentiation of this approach.

2. Background and Prior Art

2.1 Einsteinian Time Dilation

Special relativity holds that an object moving at high velocity experiences time more slowly than a stationary observer, a phenomenon known as time dilation. General relativity extends this: objects in stronger gravitational fields experience slower time. Both effects have been empirically confirmed. The GPS satellite network, for example, must continuously correct for relativistic time discrepancies to maintain accuracy.

The conceptual core of this framework borrows from relativity's most fundamental insight: that time is not experienced uniformly, and that the conditions of a system's "frame" determine how fast its internal clock runs.

2.2 Cellular Aging Mechanisms

Cellular aging is governed by several well-characterized mechanisms:

- Telomere shortening with each replication cycle, eventually triggering senescence
- Mitochondrial dysfunction and accumulating reactive oxygen species (ROS) damage
- Epigenetic drift - measurable as the "epigenetic clock" via DNA methylation patterns
- Accumulation of senescent cells exhibiting the senescence-associated secretory phenotype (SASP)
- Declining proteostasis - failure of protein quality control systems

An important distinction must be made regarding rapidly dividing cells such as malignant cells. Under the CTR framework, rapid division should theoretically accelerate biological aging and trigger cell death, yet cancer persists. This apparent contradiction is resolved by understanding that cancer cells actively disable the very mechanisms that rapid division should trigger. Through telomerase reactivation, cancer cells rebuild telomere length after each division, resetting what should be a shortening death clock. Through p53 mutation, they silence the protein responsible for detecting damage and initiating programmed cell death. Through apoptosis evasion, they learn to ignore the death signals that oxidative stress and rapid replication would normally generate. In other words, cancer cells are running a fast metabolic clock but have systematically disconnected the alarms that clock should trigger. This refinement sharpens the CTR intervention strategy considerably - the goal is not merely to accelerate the cancer cell clock further, but to reconnect the disabled alarm systems while doing so, forcing the cell to finally hear what its own biology has been saying all along. Making them not immortal, but alarm-deaf.

2.3 Metabolic Rate and Aging: Existing Literature

The relationship between metabolic rate and aging is an active area of research. The allometric theory of metabolism proposes that aging rate scales with organism size and metabolic throughput. At the cellular level, Columbia University research (Picard et al., bioRxiv 2022/2023) has empirically demonstrated that metabolic rate and pharmacological manipulations predictably accelerate or decelerate biological aging rates in primary human fibroblasts.

Separately, research into circadian biology (ScienceDirect, 2025; Frontiers in Physiology, 2025) has established that metabolic rhythms and circadian clocks are deeply intertwined, and that modulating metabolic inputs can alter the pace of cellular aging. Molecular aging clocks, computational models trained on metabolomic, epigenomic, and proteomic biomarkers, have been shown to outperform chronological age as predictors of health outcomes (Nature npj Metabolic Health, 2025).

What is notably absent from this literature is:

- A unifying physics-derived theoretical framework that treats metabolic clock rate as a relativistic analog
- A model that explicitly derives from this framework a strategy for differential clock rate modulation between cell populations
- An energetic targeting approach (distinct from pharmacological or genetic intervention) as the primary modulation mechanism
- A four-phase intervention model integrating clock acceleration, alarm restoration, immune reactivation, metabolic starvation

These gaps define the original contribution of this paper.

3. The Cellular Temporal Relativity Framework

3.1 Core Thesis

Cellular aging is a function of a cell's intrinsic metabolic clock rate, and that clock rate can be modulated through targeted energetic inputs. Understanding the biophysics of how energy states govern molecular aging processes opens a pathway to selective temporal manipulation at the cellular level.

Just as a fast-moving object in special relativity experiences less time relative to a stationary observer, a cell in a lower metabolic rate state experiences less biological aging relative to a cell operating at full metabolic throughput. The “rate of time” for that cell is functionally slower.

Formally, let M represent a cell's metabolic clock rate — a composite function of ATP turnover frequency, ROS accumulation rate, and replication cycle throughput. The biological aging rate A of that cell is proportional to M :

$$A \propto M(\text{ATP turnover, ROS flux, replication rate})$$

This relationship holds when cellular alarm systems — telomere signaling, p53 response, and apoptotic pathways — remain intact. In malignant cells these systems are disabled, decoupling

M from its natural consequence. The aging markers accumulate; the death response does not follow. This is why the CTR intervention strategy must address not only M itself, but the restoration of the alarm systems that M is supposed to activate.

This relationship implies that M is a tunable parameter — and that interventions capable of selectively modulating M in a targeted cell population could effectively “slow” or “accelerate” that population’s biological clock.

3.2 The Relativistic Analogy Unpacked

The structural parallel to relativity is as follows:

In Special Relativity:

- The "frame of reference" is defined by velocity and gravitational field
- Higher velocity → greater time dilation → slower clock relative to external observer
- The laws of physics are the same in all inertial frames; only the rate of experienced time differs

In Cellular Temporal Relativity:

- The "frame of reference" is defined by metabolic energy state and throughput
- Higher metabolic rate → greater aging rate → faster biological clock relative to low-M cells → senescence or death, where alarm systems are intact"
- Cellular biochemistry operates by the same rules in all cells; only the rate of experienced aging differs

The analogy is structural, not literal. There is no spacetime curvature in cellular biology. But the logical architecture — a rate-dependent, frame-specific experience of time, is directly transferable as a conceptual and modeling tool.

3.3 Biological Support for the Framework

Multiple independent observations from biology align with the CTR framework:

- **Metabolic scaling:** Species with higher mass-specific metabolic rates generally have shorter lifespans, a shrew lives approximately 2 years while an elephant lives approximately 70. However this relationship breaks down in humans, who are relatively small mammals yet live significantly longer than the simple metabolic scaling rule would predict. This suggests that additional regulatory mechanisms, neurological complexity, social bonding, stress regulation, and psychological factors such as meaning and purpose, modulate M independently of body mass. Under CTR this is a profound observation: humans may possess a unique capacity to consciously and socially influence their own metabolic clock rate in ways no other species can. The implications for intentional clock modulation are significant.

- **Caloric restriction:** Reducing caloric intake - and by extension, metabolic throughput, consistently extends lifespan across model organisms from yeast to mammals.
- **Hibernation:** Animals in torpor compress years of biological aging into a fraction of calendar time by dramatically reducing M.
- **mTOR inhibition:** Rapamycin, a pharmacological mTOR inhibitor, slows the metabolic signaling clock and extends healthy lifespan in mice, a chemical reduction of M.
- **In vitro hypermetabolism:** Cells isolated from the human body and grown in culture exhibit elevated growth rates and accelerated telomere shortening, consistent with CTR's prediction that elevated M accelerates aging markers. Notably, these cells still have intact alarm systems, unlike malignant cells, and therefore do eventually reach senescence.

4. Application: Selective Temporal Modulation in Oncology

4.1 Cancer Cells as Alarm-Deaf Hypermetabolic Frames

Malignant cells are, by their nature, hypermetabolic. The Warburg effect describes cancer cells' preferential use of glycolysis even in the presence of oxygen, a metabolic signature that results in elevated ATP turnover, increased ROS generation, and accelerated replication cycles. Under the CTR framework, cancer cells already inhabit a faster-running metabolic clock frame than surrounding healthy tissue.

However, as established in Section 2.2, cancer cells have disabled the alarm systems that their fast clock should trigger. They are not simply aging faster and dying - they are accumulating the biological markers of aging while remaining deaf to the death signals those markers should generate.

This has a critical implication: the metabolic differential between malignant and healthy cells is not merely a biochemical curiosity - it is a targetable temporal gradient. Cancer cells are already running a faster metabolic clock in the CTR sense, but have disabled the alarms that clock should trigger. The question therefore becomes twofold: can we further accelerate M in malignant cells while simultaneously restoring the alarm systems they have silenced? Acceleration without alarm restoration simply produces more rapidly dividing cells. Alarm restoration without acceleration may be insufficient alone. It is the combination, a forced reckoning between a running clock and a reconnected alarm, that represents the true CTR intervention target.

A critical question arises, if malignant cells are running a fast metabolic clock, why don't they fatigue and die from energy exhaustion? The answer lies in the Warburg effect. Cancer cells bypass normal mitochondrial energy production in favor of rapid glycolysis, essentially becoming glucose addicts. They sustain themselves not through efficiency but through theft, hijacking the body's blood supply via angiogenesis to feed their constant energy demand. The fatigue the cancer cell should feel is instead experienced by the patient, the exhaustion, the weight loss, the weakness that characterizes advanced cancer are in part the body being drained to sustain the tumor's relentless energy consumption. This observation adds a fourth

intervention consideration to the CTR framework: metabolic starvation. Cut the energy supply while sending the self-destruct signal. Starve the clock while reconnecting the alarm.

4.2 The Four-Phase Intervention Model

The CTR framework suggests a four-phase intervention strategy:

- **Accelerate M** in malignant cells - push their already-elevated metabolic clock to a rate that creates unsustainable biological stress
- **Restore disabled alarm systems** - reactivate p53 signaling, telomere death response, and apoptotic pathways so the cell can finally respond to what its own biology is telling it
- **Reactivate immune visibility** - trigger the surface markers that the body's own immune surveillance system looks for, allowing natural killer cells and T lymphocytes to complete the kill condition
- **Metabolic starvation** - cut the cancer cell's energy supply by disrupting its glucose dependency and blocking angiogenesis, starving the clock of the fuel it needs to sustain itself while the other three phases force the death reckoning.

Importantly, phases one and two of this intervention model are not purely theoretical. p53 restoration compounds such as Eprenetapopt (APR-246) are already in clinical trials and have received FDA breakthrough therapy designation for certain cancers. The scientific community already knows what needs to happen inside the cell. The unsolved problem is not the mechanism — it is the targeting. Systemic delivery of p53 reactivating agents affects healthy cells alongside malignant ones, creating toxicity that limits therapeutic application. The CTR framework's contribution is not to reinvent the mechanism but to propose the missing delivery layer: energetic signature targeting as the basis for selective, cell-population-specific intervention. Science has the key. CTR proposes the lock it fits.

This four-phase model gains additional power when considered alongside the body's own immune surveillance system. Healthy cells under biological stress display surface markers that attract natural killer cells and T lymphocytes - the immune system's native scanner for damaged and abnormal cells. Cancer cells evade this scanner by downregulating these markers and creating immunosuppressive environments around tumors. Under the CTR framework, restoring alarm systems in malignant cells does double duty: it reconnects the cell's internal death response AND relights the beacon that immune surveillance looks for.

The body's scanner was never broken, the cancer cells learned to hide from it. A successful CTR intervention does not just fix the alarm inside the cell. It makes the cell visible again. And once visible, the immune system, the most sophisticated cancer-killing apparatus in existence, can finish the job it was always designed to do.

4.3 Energetic Intervention as the Delivery Mechanism

The key enabling requirement for this four-phase strategy is selectivity, the ability to distinguish between cell populations and deliver differentiated energetic signals. This is precisely the research objective of the SubstrateAI.tech energy signature targeting platform, which explores the hypothesis that malignant and healthy cells exhibit distinct energetic resonance signatures that can serve as the basis for differential targeting.

Malignant cells differ from healthy cells in measurable energetic ways: they exhibit more depolarized membrane potentials, distinct bioelectric field signatures, different electromagnetic emission patterns at the molecular level, and unique metabolic heat signatures attributable to the Warburg effect. These are not merely biochemical differences — they are physical, energetic differences that function as a cellular address.

The CTR framework proposes that this address is not merely a passive identifier to route a drug to a location. It is the intervention itself. A precisely tuned energetic pulse — matched to the resonance signature of malignant cells — could trigger p53 restoration, apoptotic signaling, and immune visibility simultaneously, using the cell's own energetic language against it. This is the principle first articulated by the author in 2018: send the energy wavelength of self-destruction to cancer cells in their own communication frequency. The science of bioelectric medicine, pioneered by researchers such as Michael Levin at Tufts University, is now beginning to validate that cells respond to electrical and energetic signals in ways that can fundamentally alter their fate — including triggering apoptosis.

Prior theoretical work under the SubstrateAI platform has explored the concept that biological cells emit and respond to energy signatures at the molecular level — signatures that are altered in malignant cells due to genomic instability, membrane potential changes, and metabolic reprogramming. The CTR framework provides a temporal interpretation of this energetic difference: malignant cells are not merely biochemically different; they are temporally offset — running at a faster metabolic clock rate with disabled alarms, making them selectively vulnerable to the four-phase intervention.

This convergence of the CTR framework with energy signature targeting theory represents the most novel intersection in this body of work, and is the primary direction for further development.

5. Novelty and Differentiation from Existing Work

The CTR framework is differentiated from existing research in the following dimensions:

What exists in the literature:

- Metabolic rate correlates with aging rate (allometric theory, Picard et al.)
- Epigenetic/metabolomic clocks measure biological age
- mTOR, caloric restriction, and senolytics modulate aging at the pharmacological level
- Circadian metabolite pacemakers influence aging trajectories
- p53 restoration compounds such as Eprexapopt are in clinical trials
- Immunotherapy approaches attempt to reactivate immune response against tumors
- Bioelectric medicine (Levin et al.) demonstrates cells respond to energetic signals in fate-altering ways

What this framework contributes:

- A physics-derived conceptual model that unifies these observations under a single structural framework
- The explicit framing of metabolic clock rate as a “frame of reference” enabling differential temporal modulation
- The alarm-deaf concept — cancer cells as fast-clock cells with disabled death responses, not simply immortal cells
- The identification that the unsolved problem in p53 restoration is targeting, not mechanism, and the proposal of energetic signature targeting as the missing delivery layer
- The derivation of a four-phase oncological intervention strategy: clock acceleration, alarm restoration, immune reactivation, metabolic starvation
- The identification of the human metabolic anomaly neurological, social, and psychological modulation of M, as evidence that clock rate is consciously influenceable
- The connection between temporal modulation and energetic signature targeting as the intervention delivery mechanism, first proposed by the author in 2018

6. Limitations and Open Questions

This paper presents a theoretical framework, not experimental findings. The following limitations and open questions are acknowledged:

- The relativistic analogy is structural, not physical. Literal time dilation effects are negligible at biological scales and velocities. The framework’s value is conceptual and modeling-oriented.
- The composite metabolic clock rate variable M requires formal operationalization. A rigorous mathematical model mapping ATP turnover, ROS flux, and replication rate to a single aging rate variable is a necessary next step.
- Selectivity of energetic intervention remains the central unresolved technical challenge. The ability to differentiate malignant from healthy cells via energy signature alone has not been empirically demonstrated at scale.
- The four-phase intervention model - clock acceleration, alarm restoration, immune reactivation, metabolic starvation - requires each phase to be independently validated before integration.
- Safety and off-target effects of metabolic clock manipulation in healthy tissue must be characterized before any clinical translation.
- The framework does not yet account for heterogeneity within tumor cell populations, which may exhibit a spectrum of metabolic clock rates and varying degrees of alarm system disablement.

7. Future Directions

The following research directions are proposed for further development under LivesComeFirst.org and SubstrateAI.tech:

- Formal mathematical modeling of the metabolic clock rate variable M and its relationship to established aging biomarkers (telomere length, DNA methylation age, mitochondrial membrane potential)
- Computational simulation of the four-phase intervention model in malignant versus healthy cell populations, building on the existing SubstratePulseDemo Blazor WebAssembly platform
- Literature synthesis mapping energy signature research to CTR predictions — identifying which known malignant cell energy signatures correspond to elevated M values and disabled alarm states
- Exploration of pharmacological proxies for the four phases — identifying existing compounds that differentially affect M , p53 restoration, immune visibility, and glucose metabolism/angiogenesis disruption as a validation pathway. For the fourth phase specifically, existing metabolic therapies such as metformin, 2-DG (2-Deoxyglucose), and anti-angiogenic agents like bevacizumab represent candidate proxies worth mapping to the CTR metabolic starvation hypothesis.
- Investigation of the human M -modulation anomaly, specifically whether meaning, purpose, and social connection can be operationalized as measurable influences on cellular metabolic clock rate
- Engagement with the bioelectric medicine research community, particularly work on energetic signal-based cellular fate modulation, as the most proximate scientific neighborhood for CTR's delivery mechanism hypothesis
- Collaboration with oncology researchers and biophysicists to design in vitro validation experiments
- Theoretical exploration of CTR-based cellular immortality in healthy tissue, distinct from the pathological immortality observed in malignant cells. Cancer achieves immortality through telomerase reactivation combined with disabled alarm systems, producing unstoppable division without repair. CTR suggests an alternative pathway: dramatic deceleration of M in healthy cells while maintaining fully intact alarm systems and repair mechanisms. This would produce not a runaway cell but a stable one, a slow clock with perfect maintenance. The biological, ethical, and philosophical implications of this direction are profound and warrant dedicated interdisciplinary exploration spanning cell biology, bioethics, and philosophy of human longevity.

8. Conclusion

The Cellular Temporal Relativity framework proposes that the pace of biological aging is a frame-dependent phenomenon, determined not by calendar time, but by the metabolic clock rate of individual cells. By drawing a structural parallel to Einstein's theory of relativity, it reframes the central question of cellular aging from "how much time has passed?" to "how fast is this cell's clock running, and is it listening?"

Cancer cells run fast clocks and deaf alarms. They have accelerated their own aging markers while systematically silencing every mechanism that should convert those markers into death. The CTR framework names this condition precisely — alarm-deaf — and derives from it a four-phase intervention strategy: accelerate the clock further, restore the alarms, relight the beacon that the immune system was always designed to find, and starve the clock of the fuel it needs to sustain itself. Cut the energy. Reconnect the alarm. Make the cell visible. Force the reckoning.

The framework is original, is not replicated in existing literature in this form, and sits at the intersection of biophysics, oncology, immunology, and the energy signature research program that has been the author's primary research focus since 2018. The core intuition, that cancer cells could be told to self-destruct in their own energetic language, was first documented in June 2018. This white paper is its formal scientific expression.

While this paper focuses on oncological application, the CTR framework is inherently bidirectional. The same principles that suggest accelerating M in malignant cells to force a death reckoning also suggest that controlled deceleration of M in healthy cells could meaningfully extend biological healthspan. The cancer application is the proving ground. Longevity is the horizon.

References and Prior Art Acknowledgment

The following works were consulted in situating this framework within the existing literature. Their inclusion constitutes acknowledgment that adjacent work exists, and that the CTR framework builds upon and extends — rather than duplicates — these contributions.

Author's Prior Work — Original Source Documents

Aslam, S. (2018). "Cure for Cancer" PowerPoint presentation. File metadata: Content created 6/11/2018 11:11 PM, Last saved 6/26/2018, Author: Shawn Aslam, 51 revisions, 72 hours 28 minutes total editing time. Presented to researchers at Fred Hutchinson Cancer Center, Seattle, WA. Original concept: emitting the energy wavelength of cellular self-destruction to selectively target malignant cells.

Columbia University / UCSF — Metabolic Aging Rate

Picard et al. (bioRxiv, 2022/2023). "Accelerating the clock: Interconnected speedup of energetic and molecular dynamics during aging in cultured human cells." Demonstrates that metabolic rate manipulations predictably accelerate or decelerate biological aging rates.

ScienceDirect — Circadian-Metabolic Intersection

Dong et al. (Journal of Genetics and Genomics, 2025). “The circadian clock at the intersection of metabolism and aging — emerging roles of metabolites.” Establishes metabolite pacemakers as largely unexplored aging modulators.

Frontiers in Physiology — Clock Genes and Aging

(Frontiers, 2025). “Progress in understanding how clock genes regulate aging and associated metabolic processes.” Characterizes SIRT1/NAMPT/NAD⁺ feedback loop as a metabolic oscillator linked to telomere maintenance.

PMC — Cellular Models of Aging

(PMC, 2025). “Cellular Models of Aging and Senescence.” Distinguishes chronological time from biological clock and reviews partial epigenetic reprogramming strategies.

Nature npj — Metabolomic Aging Clocks

(Nature npj Metabolic Health and Disease, 2025). “Metabolomic-based aging clocks.” Demonstrates that metabolic biomarker-based clocks outperform chronological age in predicting health outcomes.

Molecular Cancer — Senescence and Oncology

(Molecular Cancer, 2025). “Reprogramming cellular senescence and aging clocks for advanced cancer immunotherapy.” Reviews epigenetic and metabolic clocks in tumor biology and immune evasion mechanisms.

Tufts University — Bioelectric Medicine

Levin, M. et al. Ongoing research into bioelectric signaling and cellular fate modulation. Demonstrates that cells respond to electrical and energetic signals in ways that can alter fundamental cellular behavior including apoptosis.

Intellectual Property Notice

This document constitutes an original theoretical contribution by the author. The Cellular Temporal Relativity framework, the alarm-deaf concept, the four-phase intervention model, the energetic self-destruction delivery hypothesis, and the connection to energy signature targeting are the intellectual property of the author.

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