



Non-Equilibrium Allometric Scaling and Bioenergetic Information Routing: A Comparative Science 4.0 Framework for Cellular Resilience Across Mammalian Phyla

Boblique J*

Independent Researcher, Science 4.0 Framework, France

***Corresponding author:** Julien Boblique, Independent Researcher, Science 4.0 Framework, France

Review Article

Volume 9 Issue 5

Received Date: September 15, 2026

Published Date: October 01, 2026

DOI: 10.23880/izab-16000702

Abstract

Classical biological allometry historically formalizes metabolic rate scaling through empirical power laws, most notably Kleiber's law (B proportional to $M^{(3/4)}$), yet fails to explain the non-equilibrium thermodynamic mechanisms and information-theoretic constraints governing cellular survival during severe hypoxia across different mammalian species. In this paper, we integrate non-equilibrium thermodynamics and Shannon communication theory into comparative animal biology under the Science 4.0 framework. We model intracellular metabolic routing through coupled thermodynamic flux equations and formalize the cellular Bioenergetic Signal-to-Noise Ratio (SNR_{bio}) at mitochondrial-endoplasmic reticulum contact sites (MAMs). Our derivations demonstrate that mass-specific basal metabolic rate (B/M proportional to $M^{(-1/4)}$) directly dictates the basal entropy production rate (σ_{ent}) and the operational signaling bandwidth (Δf). While smaller mammals exhibit higher channel capacity (C) under homeostatic baselines to support rapid enzymatic turnover, their microvascular networks experience accelerated informational collapse ($C \rightarrow 0$, $SNR_{bio} < 3.0$ dB) during acute substrate deprivation. Conversely, larger phyla and hypoxia-tolerant comparative models (e.g., *Heterocephalus glaber*) display structurally dampened thermodynamic dissipation and lower baseline oxidative noise, preserving homeostatic channel capacity. Establishing this comparative Science 4.0 framework provides evolutionary and veterinary biology with a predictive biophysical metric of cellular resilience and targeted bioenergetic rescue across mammalian taxa.

Keywords: Allometric Scaling; Kleiber's Law; Non-Equilibrium Thermodynamics; Signal-to-Noise Ratio (SNR); Shannon Channel Capacity; Science 4.0; Comparative Bioenergetics; Mammalian Resilience

Introduction: Beyond Static Allometric Power Laws

The relationship between mammalian body mass (M) and whole-organism basal metabolic rate (B) has long been described by quarter-power allometric scaling relations ($B = B_0 \cdot M^{(3/4)}$), with mass-specific metabolic activity

scaling inversely (B/M proportional to $M^{(-1/4)}$). While geometric and hydrodynamic fractal network models (such as the West-Brown-Enquist framework) successfully explain macro-distribution across vascular branching architectures, they do not resolve how individual cells transduce, dissipate, and route metabolic energy under severe non-equilibrium stress [1-4].

In comparative physiology and animal biology, a critical paradox persists: small mammals (e.g., *Mus musculus*, mass ≈ 0.025 kg) maintain mass-specific oxygen consumption rates over an order of magnitude higher than large ungulates or cetaceans, generating substantial baseline mitochondrial reactive oxygen species (ROS). Yet, when subjected to ischemic, hypothermic, or metabolic insults, tolerance thresholds and microvascular recovery dynamics vary drastically across taxa [5].

Within the Science 4.0 paradigm, living organisms are not merely heat engines or passive chemical reactors; they are open, non-equilibrium dissipative information processors [6].

Intracellular biochemical cascades function as noisy communication channels where homeostatic signals must be reliably transmitted against thermal, metabolic, and oxidative fluctuations.

Resolving the fundamental laws governing cross-species cellular survival requires extending allometric theory into non-equilibrium thermodynamics and Shannon channel capacity constraints. This paper provides an analytical framework coupling allometric scaling directly to the cellular Bioenergetic Signal-to-Noise Ratio (SNR_{bio}) across mammalian phyla [7-10].

Theoretical Formulation: Allometric Non-Equilibrium Entropy Production

Living cellular architectures operate far from thermodynamic equilibrium through the continuous dissipation of free energy. The local volumetric rate of internal entropy production, σ_{ent} [$\text{W} \cdot \text{m}^{-3} \cdot \text{K}^{-1}$], within a mammalian cell is defined by the sum of conjugate thermodynamic forces and fluxes:

$\sigma_{ent} = J_{ATP} \cdot (A_{ATP} / T) + \sum_k [J_k \cdot (-\text{grad}_{\mu_k} / T)] + P_i : (\text{grad}_v / T)$ Where:

- J_{ATP} and A_{ATP} represent the reaction rate and chemical affinity of ATP hydrolysis.
- J_k and grad_{μ_k} represent the diffusive flux and chemical potential gradient of substrate species k (glucose, fatty acids, pyruvate) [11].
- P_i and grad_v represent the viscous stress tensor and internal cytoplasmic velocity gradient.
- T is the absolute physiological core temperature of the organism (e.g., 310.15 K in humans, 309.65 K in rodents).

Allometric Coupling of Dissipative Flux

Because mass-specific metabolic flux scales allometrically across species as $M^{(-1/4)}$, the cellular rate of ATP turnover (J_{ATP}) and mitochondrial substrate flux (J_k)

are direct functions of adult body mass M :

$$J_{ATP}(M) = J_0 \cdot M^{(-1/4)}$$

Consequently, the baseline specific rate of entropy production ($\sigma_{ent,0}$) under resting physiological conditions scales allometrically:

$$\sigma_{ent,0}(M) \text{ proportional to } M^{(-1/4)} \cdot (A_{ATP} / T)$$

In smaller mammalian species, high mass-specific metabolic turnover demands elevated baseline electron flux through the mitochondrial respiratory chain. This steep gradient elevates the baseline generation of metabolic noise ($N_{metabolic}$) and steady-state oxidative byproduct concentrations ($[ROS]_{cyt}$), placing their microvascular endothelium closer to thermodynamic instability under acute substrate interruption [12,13].

Mathematical Modeling of Comparative Channel Capacity and SNR_{bio}

In the Science 4.0 formulation, intracellular signal transduction cascades (such as AMPK activation, Akt/eNOS phosphorylation, and PGC-1 α mitochondrial biogenesis signaling) are treated as communication channels operating across noisy biophysical media.

Cellular Bioenergetic Signal-to-Noise Ratio

We define the cellular Bioenergetic Signal-to-Noise Ratio across mammalian species as: $\text{SNR}_{bio}(M) = 10 \cdot \log_{10} [S_{coherent}(M) / (N_{thermal} + N_{metabolic}(M) + N_{ROS}(M))]$ Where:

- $S_{coherent}(M) = \kappa \cdot [ATP]_{cyt}(M) \cdot \Phi_{kin}$ is the coherent metabolic signaling power governed by steady kinase phosphorylation cascades.
- $N_{thermal} = k_B \cdot T \cdot \Delta_f(M)$ is the Nyquist-Johnson thermal noise power within the metabolic signaling bandwidth $\Delta_f(M)$.
- $N_{metabolic}(M) = \theta \cdot J_{ATP}(M)$ is the stochastic metabolic noise driven by molecular turnover fluctuations.
- $N_{ROS}(M) = \xi \cdot [ROS]_{cyt}(M)$ represents the disruptive oxidative noise power generated by electron leakage across mitochondrial complexes I and III.

Scaling of Metabolic Bandwidth and Shannon Channel Capacity

The effective operational bandwidth of cellular signaling (Δ_f) scales inversely with biological cycle times (heart rate, capillary transit time, enzymatic turnover), conforming to allometric frequency scaling:

$$\Delta_f(M) = \Delta_f_0 \cdot M^{(-1/4)}$$

The maximum reliable biological information transmission rate (Shannon channel capacity, C) through the cellular repair and maintenance network is given by:

$$C(M) = \Delta_f(M) \cdot \log_2 [1 + S_{\text{coherent}}(M) / (k_B \cdot T \cdot \Delta_f(M) + \theta \cdot J_{\text{ATP}}(M) + \xi \cdot [\text{ROS}]_{\text{cyt}}(M))]$$

Under acute ischemia or hypoxia, the chemical affinity A_{ATP} drops, collapsing S_{coherent} while mitochondrial

uncoupling drives $[\text{ROS}]_{\text{cyt}}$ upward. When SNR_{bio} drops below the universal critical thermodynamic survival boundary:

$$\text{SNR}_{\text{crit}} = 3.0 \text{ dB}$$

Shannon channel capacity collapses toward zero ($C \rightarrow 0$), receptor-effector communications undergo complete stochastic decoherence, and cells rapidly initiate regulated necrosis or caspase-dependent apoptosis.

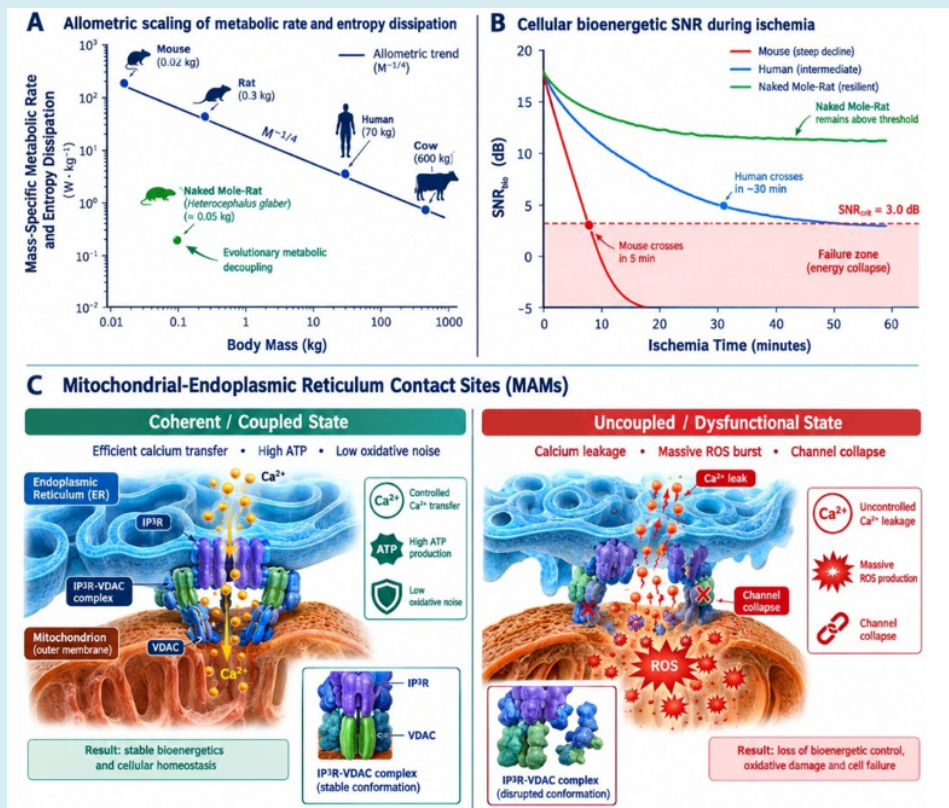


Figure 1: Non-equilibrium allometric scaling of metabolic entropy and comparative information-theoretic dynamics across mammalian species.

(A) Allometric Scaling of Metabolic Rate and Entropy Dissipation: Log-log relationship showing mass-specific metabolic dissipation scaling inversely with adult body mass

($M^{(-1/4)}$) across representative mammalian phyla (mouse, rat, human, bovine). The hypoxia-tolerant naked mole-rat (*Heterocephalus glaber*) displays evolutionary metabolic decoupling, maintaining suppressed baseline entropy production despite small body mass.

(B) Cellular Bioenergetic SNR During Ischemia: Kinetic decay of the cellular bioenergetic Signal-to-Noise Ratio (SNR_{bio}) during acute substrate deprivation. Elevated metabolic turnover drives a precipitous collapse in mice, crossing the critical survival boundary ($\text{SNR}_{\text{crit}} = 3.0 \text{ dB}$) within 5 minutes, compared to ~30 minutes in humans. The naked mole-rat buffers informational degradation, remaining consistently above the energy collapse threshold.

(C) Mitochondrial-Endoplasmic Reticulum Contact Sites (MAMs): (Left) Coherent/coupled state featuring stable IP3R-VDAC conformation, controlled Ca^{2+} transfer, high ATP synthesis, and minimal oxidative noise, ensuring stable cellular homeostasis. (Right) Uncoupled/dysfunctional state with complex disruption, uncontrolled Ca^{2+} leakage, massive reactive oxygen species (ROS) bursts, and informational channel collapse driving loss of bioenergetic control and cell death.

Comparative Biophysical and Information-Theoretic Parameters

thermodynamic, and information-theoretic parameters across representative mammalian model systems under physiological baseline and ischemic stress.

Table 1 summarizes comparative allometric,

Comparative Model	Typical Body Mass (M)	Mass-Specific Metabolic Rate (B/M)	Baseline Band width (Delta_f)	Baseline ROS Level ([ROS]_cyt)	Physiological SNR_bio	Ischemic SNR_bio (30min)	Shannon Capacity (C, Base line)	Physical Dimension/ Unit
Mouse (Mus musculus)	0.025	6.4	8.5 - 12	4.5 - 6.5	16.5 - 19	< 0.8	38- 52	kg ; W·kg ⁻¹ ; Hz ; nM ; dB ; dB ; bits·s ⁻¹
Rat (Rattus norvegicus)	0.35	3.8	4.2 - 6.5	3.2 - 4.8	17.5 - 21	< 1.1	24 - 35	kg ; W·kg ⁻¹ ; Hz ; nM ; dB ; dB ; bits·s ⁻¹
Human (Homosapiens)	70	1.25	0.1 - 1	1.0 - 3.5	19.0 - 24.5	< 1.4	12.0 - 18.0	kg ; W·kg ⁻¹ ; Hz ; nM ; dB ; dB ; bits·s ⁻¹
Bovine (Bostaurus)	650	0.75	0.05 - 0.4	0.8 - 2	20.0 - 26	< 1.6	6.5 - 11	kg ; W·kg ⁻¹ ; Hz ; nM ; dB ; dB ; bits·s ⁻¹
Naked Mole-R at (H. glaber)	0.035	1.85 (Suppressed)	1.5 - 3	0.9 - 1.8	22.0 - 27.8	> 3.8 (Resilient)	15 - 22	kg ; W·kg ⁻¹ ; Hz ; nM ; dB ; dB ; bits·s ⁻¹
Critical Failure Threshold	—	—	—	—	—	3	< 1.0	dB ; bits·s ⁻¹

Table 1: Allometric, Thermodynamic, and Information-Theoretic Parameters Across Mammalian Phyla.

Comparative Dynamics: Small Mammalian Vulnerability vs. Evolutionary Decoupling

The analytical integration of allometry and information theory resolves key disparities observed in comparative experimental biology

Accelerated Information Collapse in Small Mammals

Because small rodents operate at elevated mass-specific metabolic fluxes (J_ATP proportional to M^{-(1/4)}), their cellular ATP reserves are exhausted exponentially faster during acute ischemia. Cytosolic [ATP] drops below 0.8 mM within minutes, triggering massive calcium accumulation at mitochondrial-associated ER membranes (MAMs).

Concurrently, unattenuated oxidative bursts inflate N_ROS. Although their physiological channel capacity is wide (C > 40 bits·s⁻¹), the loss of S_coherent drives SNR_bio below SNR_crit = 3.0 dB significantly faster than in larger mammals, causing rapid microvascular endothelial swelling and irreversible capillary rarefaction.

Evolutionary Decoupling in Hypoxia-Tolerant Species

Species exhibiting atypical resilience to hypoxia and oxidative damage—most prominently the naked mole-rat (Heterocephalus glaber)—demonstrate an evolutionary decoupling from standard Kleiber scaling. As reflected in Table 1, H. glaber maintains a suppressed mass-specific metabolic rate and high mitochondrial coupling efficiency, keeping baseline oxidative entropy production (sigma_ent,0) comparable to that of large mammals despite its small body mass. Under hypoxic stress, these cells dynamically downregulate signaling bandwidth (Delta_f), thereby suppressing metabolic noise (N_metabolic) and preserving SNR_bio > 3.0 dB across prolonged ischemic intervals.

Science 4.0 Resilience Framework and Veterinary Translation

Translating this non-equilibrium framework into comparative animal health, veterinary critical care, and translational biomedical modeling establishes three operational principles:

1. Bandwidth-Calibrated Metabolic Rescuing: Therapeutic interventions cannot apply identical kinetics across different mammalian phyla. Small mammalian patients require rapid-acting, high-affinity antioxidant substrates (such as lipid-matrix nano-vectorized carriers) administered within narrow time windows to prevent rapid SNR_{bio} collapse.
2. Allometrically Scaled Pre-Conditioning: Conditioning protocols prior to planned ischemia (e.g., surgical organ transplantation, bypass procedures) must calibrate sub-lethal hypoxia dosing inversely to $M^{(-1/4)}$ to stabilize mitochondrial-ER contact sites without crossing the SNR_{crit} threshold.
3. Information-Theoretic Viability Monitoring: Monitoring the recovery of microvascular impedance and bioenergetic signal coherence serves as a universal, cross-species index of tissue viability independent of macroscopic organ dimensions.

Discussion

Standard comparative physiology has largely treated allometric scaling as a static geometric or physiological consequence of resource transport networks. This paper expands this paradigm by showing that allometric scaling fundamentally dictates the informational signal-to-noise ratio and entropy dissipation kinetics of cellular repair networks.

When ischemic stress strikes, cell death is not merely a localized biochemical deficit of ATP; it represents the catastrophic loss of Shannon channel capacity driven by unattenuated thermodynamic noise. Larger mammals benefit from intrinsically lower mass-specific entropy production rates, affording prolonged survival windows prior to junctional breakdown.

By introducing the Science 4.0 formulations of SNR_{bio} and the critical stability threshold (SNR_{crit} = 3.0 dB), comparative animal biology gains a rigorous, quantitative framework linking microvascular hydrodynamics, non-equilibrium thermodynamics, and evolutionary physiology across the mammalian tree of life.

Conclusion

Non-equilibrium allometric scaling governs the bioenergetic Signal-to-Noise Ratio (SNR_{bio}) and Shannon channel capacity of cellular networks across mammalian phyla. Small mammals possess high basal signaling bandwidth but experience rapid informational collapse under ischemia, whereas large or adapted species maintain buffered entropy dissipation regimes. Re-establishing SNR_{bio} > 3.0 dB represents a universal biophysical imperative for

preserving microvascular competence and cellular resilience across all mammalian taxa.

Declarations

Conflict of Interest: The author declares no conflicts of interest.

Funding: This research was independently conducted under the Science 4.0 Framework without external institutional grants.

Data Availability: All analytical derivations, mathematical formulations, and allometric scaling relationships are fully presented within this manuscript.

References

1. Kleiber M (1932) Body size and metabolism. *Hilgardia* 6(11): 315-353.
2. West GB, Brown JH, Enquist BJ (1997) A general model for the origin of allometric scaling laws in biology. *Science* 276(5309): 122-126.
3. Granger DN, Kvietys PR (2015) Reperfusion injury and reactive oxygen species: The evolution of a concept. *Redox Biology* 6: 524-551.
4. Shannon CE (1948) A mathematical theory of communication. *Bell System Technical Journal*, 27(3): 379-423.
5. Pries AR, Secomb TW (2014) Making microvessels work: vascular adaptation, remodeling and regression. *Physiology*, 29(4): 289-301.
6. Friston K (2010) The free-energy principle: a unified brain theory?. *Nature Reviews Neuroscience* 11(2): 127-138.
7. Buffenstein R (2008) Negligible senescence in the longest living rodent, the naked mole-rat: insights from a successfully aging species. *The Journals of Gerontology Series A* 63(6): 564-570.
8. Boblique J (2026) Science 4.0 Architecture of Cellular Resilience and Living Signal Optimization. *International Journal of Zoology and Animal Biology* 9(2): 000673.
9. Boblique J (2026) Industrial Standardization of the Bio-OS: Algorithmic Codification of Resilience Engineering Guidelines and Version V8 Architecture. *International Journal of Zoology and Animal Biology* 9(3): 000685.
10. Boblique J (2026) Biophysical Optimization of Cellular Signal-to-Noise Ratio (SNR) in Bioenergetic Information Networks V2. *Research Journal of Innovative Studies in*

Medical and Health Sciences 3(1): 22-24.

11. Boblique J (2026) Lipid-Matrix Carrier Architecture: Engineering Nano-Vectorized Transport for Hydrophobic Living Signals in Science 4.0 Frameworks. JSM Biotechnology & Biomedical Engineering 10(1): 1094.
12. Boblique J (2026) Non-Equilibrium Bioenergetic Information Routing and Signal-to-Noise Ratio Optimization in Ischemic Endothelial Repair: A Clinical Framework for Microvascular Recovery. Advance Medical & Clinical Research, 7(4): 436-438.
13. Boblique J (2026) Bio-Electrodynamic Shielding of the Blood-Brain Barrier: Non-Equilibrium Hydrodynamics to Prevent Post-Ischemic Micro-Thrombosis and Transient Ischemic Attacks (TIA). Clinical & Medical Investigations.

YOUR RESEARCH DESERVES GLOBAL VISIBILITY

• Publish • Discover • Share • Connect

MEDICAL	CLINICAL	LIFESCIENCES	PHARMA
90+ OPEN ACCESS JOURNALS		400+ RESEARCH TOPICS	

SUBMIT YOUR RESEARCH

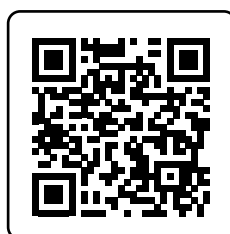
Submit your manuscript through our online submission system.

REPRINTS

If you require a reprint of any article, please contact us.
info@medwinpublishers.com

BECOME A REVIEWER

Share your expertise and contribute to scholarly publishing.



SCAN TO EXPLORE MEDWIN JOURNALS

• Journals • Submit Manuscript • Reviewer Program • Article Tracking

Our Journals indexed in



This article published by **Medwin Publishers**, 28475 Witherspoon Dr, Novi, MI 48377, United States, +1-248-247-6042, WhatsApp +1 (248) 275-1133, info@medwinpublishers.com

© 2026, by the Authors. The articles published from this journal are distributed to the public under “**Creative Commons Attribution License**” (<https://creativecommons.org/licenses/by/4.0/>). Therefore, upon proper citation of the original work, all the articles can be used without any restriction or can be distributed in any medium in any form.