Information-Theoretic Origins of Universality in Stochastic Biological Systems

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Metabolic rate scales with body size, however its universality remains debated and unresolved. We show that such universal scaling may arise from information neutrality in stochastic cell dynamics. Using a stochastic ontogenetic growth model of cellular dynamics, we identify an optimal microscopic noise structure where organism level metabolic fluctuations are least sensitive to the underlying microscopic cellular noise and have maximal dependence on organism size. At this point, the macroscopic scaling exponent collapses to a universal value across species size close to Kleiber's law. These results reveal a noncritical RG-like behavior, suggesting that universality emerges here from an information-theoretic optimum of stochastic metabolic fluctuations.

I. INTRODUCTION

Power-law distributions are prevalent in natural complex systems. This distribution is often interpreted as a signature of phase transitions which emerge when a control parameter is tuned to a critical threshold [31]. This underlies the concept of self-organized criticality, i.e. when natural complex systems evolve towards a critical threshold in order to achieve maximal information processing [17, 23]. Since "life at the edge of chaos" argument has gained traction in biology over the past decades, many biological processes have been observed to reside close to phase transitions [15, 32]. However, various mechanisms can generate power laws without criticality [40], such as aggregation [27], preferential attachment [4], or stochastic processes [33, 42]. Thus identifying the generative process remains crucial for understanding the biological significance and interpretation of such scaling laws.

Scaling laws are widespread across biological hierarchies. Traits including metabolic rate, growth, lifespan, and population density exhibit systematic relationships with organism size [3, 6, 38, 46]. These regularities suggest that biological systems obey underlying constraints governing how resources, fluctuations, and information propagate across organizational levels. Among these relationships, metabolic scaling is one of the most extensively studied examples of biological scaling laws, which describes the sublinear scaling between organisms' body size and metabolic rate rooted in an empirical observation called the Kleiber's law [5, 20]. Since body size is a master trait, metabolic scaling belongs to a broader class of biological allometries [38] with life-history traits, population growth rates, and energy fluxes [7] to name a few. This suggests that living systems obey internal constraints as a consequence of historical contingencies and functional and structural rules [1, 41]. The universality principle of this relationship has been up for debate for a long time due to largely varying observations [9, 11, 18], however the most popular explanations include noncritical phenomena such as fractal-like structures of distribution networks [44], surface-to-volume ratio of the organisms [16, 37] or life-history optimisation [47]. In biological contexts, such scale invariance may reflect constraints on how fluctuations and information propagate across biological hierarchy [43]. Coarse-grained variables such as metabolic rate are therefore the most informative macroscopic descriptors that captures the system behavior as a whole.

Information theory provides a natural framework for quantifying how microscopic fluctuations influence macroscopic observables and how information about microscopic parameters is preserved or lost under coarse-graining [10, 43]. In complex systems, coarse-graining often leads to regimes in which macroscopic observables become insensitive to microscopic details, a property associated with universality in statistical physics [40, 48]. Recent work shows that metabolic fluctuations can successfully reproduce the observed variability of the metabolic scaling exponents across and within taxa [42]. Biological systems are inherently stochastic that may arise from various cellular processes such as birth–death dynamics, metabolic bursts due to thermodynamic fluctuations, noisy biochemical reactions, and stochastic gene expression [12, 28, 36, 39]. Such microscopic fluctuations can propagate across organizational levels, generating macroscopic variability even without environmental forcing. Similarly, demographic noise in ecological systems arises from discrete birth-death processes, and is a fundamental source of intrinsic stochasticity [22, 34], which can generate macroscopic population fluctuations even without environmental forcings [29]. This raises a question under what conditions do macroscopic patterns, in this case the allometric exponents, become universal given the underlying microscopic noise structure.

In this study we investigate whether universal metabolic scaling can emerge from statistical properties of stochastic cellular dynamics. We present a stochastic ontogenetic growth model in which cellular bursts generate

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metabolic fluctuations whose magnitude scales with organism size. Using an information-theoretic analysis, we identify a regime where organism-level fluctuations become minimally sensitive to microscopic fluctuations, i.e, an information neutral regime, indicating that coarse-grained variables retain minimal information about the underlying microscopic parameters. At this point the macroscopic metabolic scaling exponent collapses to a universal value across species sizes. These results suggest that universal biological scaling laws may arise from information-theoretic constraints rooted in stochastic cellular processes rather than from criticality.

II. METHODS

A. Stochastic metabolic model of cellular bursts

The ontogenetic growth is modeled at the cellular level. First, we model cell dynamics which is assumed to be a bursty stochastic process. The cell dynamics regulates the metabolic rate and the body mass of the organism. Cell dynamics is modeled as a stochastic jump process where the jumps J(t) are approximated as a symmetric exponential jumps and sampled from a Laplace distribution. Cellular activity often exhibits bursty fluctuations due to discrete biological events such as cell division or death and synchronized metabolic activity. To capture these effects we model cell dynamics as a stochastic jump process, where abrupt changes in cellular activity are represented by random bursts. The cell population evolves according to a stochastic logistic model [42]:

$$\frac{dN_c}{dt} = aN_c\left(1 - \frac{N_c}{N}\right) + J(t), \tag{1}$$

where a is the cellular growth rate, N is the total number of cells at maturity, $J(t) \sim \text{Laplace}(0, b)$, is the jump process and the magnitude throughout the life cycle can be written as:

$$b = N_c^{\alpha/2}\left[\phi + (1-\phi)\left(1 - \frac{N_c}{N}\right)^{\theta}\right]. \tag{2}$$

where α is the correlation exponent, ϕ is the fluctuation coefficient. The exponent $\alpha/2$ derives from the assumption that the variance of the stochastic burst term scales with system size (Fig.1A). For the Laplace distribution $Var(J) = 2b^2$, and we assume $Var(J) \propto N_c^{\alpha}$ [40]. This formulation provides a tunable representation of the correlation structure of cellular bursts. If cellular bursts occur independently, the variance of the fluctuations grows linearly with system size ($\alpha = 1$). In contrast, perfectly synchronized cellular activity produces fluctuations that scale quadratically with system size ($\alpha = 2$). Intermediate values $1 < \alpha < 2$ correspond to partially correlated cellular dynamics, which may arise from shared metabolic networks or resource constraints linking cellular activity. In this framework, values of $\alpha > 1$ lead to sublinear allometric scaling, whereas $\alpha < 1$ can generate superlinear scaling exponents β (Fig. S1). We assume that the magnitude of fluctuations are higher in the growth phase pf the organism compared to the adult phase, which gradually decays over time. This decay is modulated by θ. The magnitude of the jump process is proportional to organism size and at the adult stage it simplifies to:

$$b = \phi N^{\alpha/2} \tag{3}$$

Then, we estimate organism-level metabolism as the sum of maintenance costs of each individual cell (E_M), the growth costs (E_G) and an overhead cost of stochastic cellular dynamics (H) [42].:

$$B(t) = B_M(t) + B_G(t) + H(t). \tag{4}$$

The maintenance component is proportional to the number of cells:

$$B_M(t) = N_c(t)B_c, \tag{5}$$

where B_c is the maintenance power expenditure per cell ($B_c = 1.08e-7$ W [46]). The growth component accounts for the energetic cost of creating new cells:

$$B_G = \begin{cases} \dfrac{E_c}{\eta}\dfrac{dN_c}{dt}, & \text{if } \dfrac{dN_c}{dt} \geq 0, \\ 0, & \text{if } \dfrac{dN_c}{dt} < 0 \end{cases} \tag{6}$$

where E_c is the energy required for cell birth ($E_c = 3.7e^{-7}$ J [35]), and η is the growth efficiency ($\eta = 0.7$). The stochastic inefficiency component H represents energetic losses associated with burst-like cellular activity. Rapid fluctuations in cell number or metabolic activity introduce additional energetic costs, which are modeled as a dissipation term proportional to the magnitude of stochastic bursts [13, 39]:

$$H = \begin{cases} (1-\eta)E_c\dfrac{dN_c}{dt} + \gamma\left|J(t)\right|, & \text{if } \dfrac{dN_c}{dt} \geq 0, \\ \kappa E_c\left|\dfrac{dN_c}{dt}\right| + \gamma\left|J(t)\right|, & \text{if } \dfrac{dN_c}{dt} < 0 \end{cases} \tag{7}$$

where γ is a stochastic overhead cost that converts the stochastic cellular burst into energy dissipation ($\gamma = 1$ J/cell) and κ is the apoptosis cost set to 0.3. Metabolic fluctuations often exhibit Laplace-like distributions [21], which arises from the underlying bursty cellular processes. The total body mass evolves over time as the

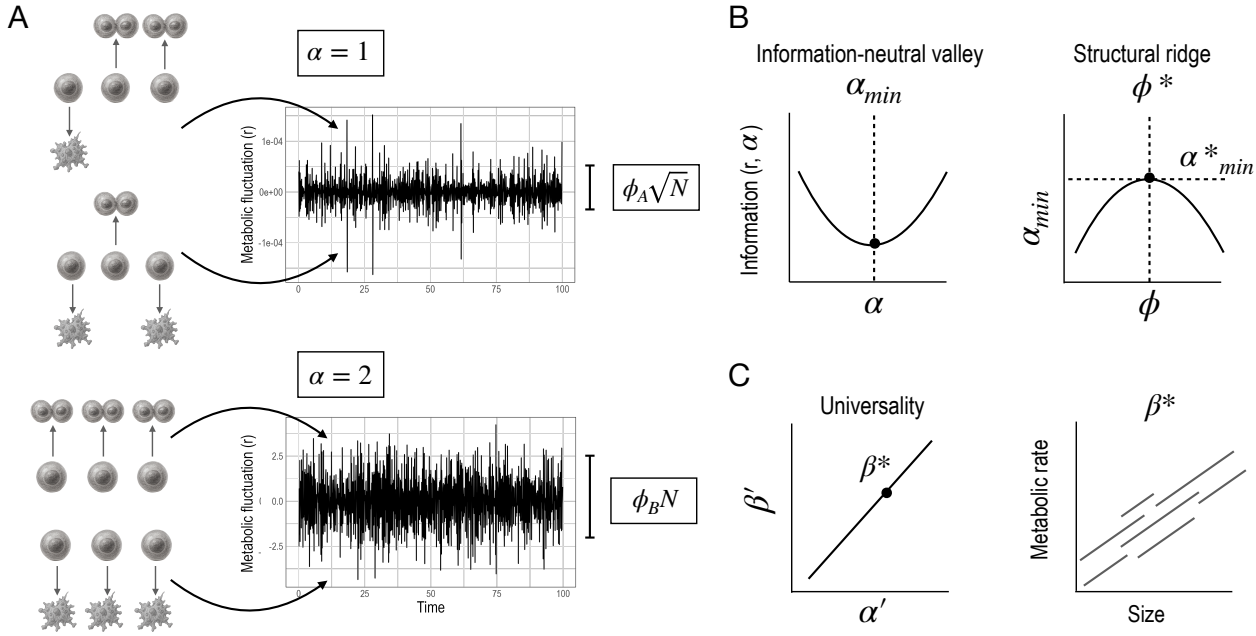

Figure 1 **Conceptual framework of the information-neutral scaling mechanism.** (A) Schematic illustration of metabolic fluctuations generated by stochastic cellular bursts. Uncorrelated cellular dynamics (top) produce weakly structured fluctuations, whereas correlated bursts (bottom) generate larger collective fluctuations at the organism level. (B) Identification of the information-neutral correlation exponent. For each value of the microscopic correlation exponent α, the distribution of metabolic fluctuations $p(r|\alpha)$ is compared with the pooled distribution $p(r)$ using the KL divergence $D_{\mathrm{KL}}(\alpha, r)$. The minimum of the KL divergence identifies the information-neutral exponent $\alpha_{\min}$, where macroscopic fluctuations are least sensitive to microscopic parameters. Second, we determine the optimal noise scale. The information-neutral exponent $\alpha_{\min}$ is evaluated across different fluctuation amplitudes ϕ. The maximum of $\alpha_{\min}(\phi)$ defines the structural ridge corresponding to the optimal fluctuation scale ϕ^*, which produces a universal metabolic scaling exponent β^* across species sizes. (C) Then using the identified optimal noise structure, we calculate the corresponding scaling exponent $\beta^*(\alpha^*_{\min}, \phi^*)$.

net energy available for biomass production, after subtracting maintenance and stochastic inefficiency [45]:

$$\frac{dm}{dt} = \frac{m_c}{E_c}(B - H) - \frac{mB_c}{E_c}. \tag{8}$$

where m_c is the average mass of a cell ($m_c = 1e^{-12}$ kg [26]). This final form describes the dynamics of body mass based on energy input, metabolic noise, and maintenance cost.

B. Estimation of species-level metabolic fluctuations

We derive analytical formulas for the expected species-level metabolic rate, body mass and the allometric scaling exponent in the adult phase (steady state). The metabolic rate in the adult phase simplifies to:

$$B(t) = NB_c \; + \; c_+ \, J_+ \; + \; c_- \, J_-, \tag{9}$$

$$X \sim \mathrm{Laplace}(0, s), \qquad s = \phi N^{\alpha/2}\sqrt{\frac{\Delta t}{2}}. \tag{10}$$

where $J_+ = \max(X, 0)$ and $J_- = \max(-X, 0)$ are the positive and negative parts of the jump term (J). The noise amplitude ϕ controls the microscopic variability, while α governs how fluctuations scale with system size. Positive and negative bursts are weighted by

$$c_+ = E_c\left(\frac{1}{2\eta} + \frac{1 - \eta + \kappa}{2}\right) + \gamma, \tag{11}$$

$$c_- = \kappa E_c + \gamma, \tag{12}$$

The metabolic fluctuations r driven by the microscopic noise structure (ϕ, α) is defined as:

$$r(t) = \log\left(\frac{B_{t+1}}{B_t}\right) \tag{13}$$

where the only difference between B_t and B_{t+1} arises from a stochastic jump in cell number. In this study, we focus on the noise structure, quantified by stochasticity magnitude ϕ, and the correlation exponent α and all other model parameters are kept fixed. For each $\alpha \in [0.9, 2]$, $\phi \in [10^{-6}, 10^{-2}]$ and $N \in [10^{10}, 10^{16}]$ grid, we compute the allometric scaling exponent β. We simulate $n_{\text{sim}} = 10^5$ stochastic trajectories for each (N, α, ϕ) parameter combination using Monte Carlo simulations. For each parameter set we draw $X_1, X_2 \overset{\text{iid}}{\sim} \text{Laplace}(0, b)$, to construct B_t and B_{t+1} and compute the sample of r. This procedure yields unbiased estimates that match the simulated time-series statistics for all species sizes.

C. Estimation of the allometric exponent at steady state

At adult steady state the stochastic growth dynamics reach a statistical equilibrium in which the expected metabolic rate and body mass depend only on organism size and the noise structure. In this regime we derive analytical expressions for the expected metabolism and body mass and compute the metabolic scaling exponent as the logarithmic derivative of metabolism with respect to body mass.

$$\mathbb{E}[B_{\text{adult}}] = A\,N + C\,N^{\alpha/2}, \tag{14}$$

$$A = B_c, \tag{15}$$

$$C = \phi\sqrt{\frac{dt}{2}}\left[E_c\left(\frac{1}{2\eta} + \frac{1-\eta+\kappa}{2}\right) + \gamma\right]. \tag{16}$$

Similarly, the expected adult mass is

$$\mathbb{E}[m_{\text{adult}}] = m_c N + m_c E N^{\alpha/2}, \tag{17}$$

$$E = \phi\sqrt{\frac{dt}{2}}\left(\frac{E_c}{2\eta B_c}\right), \tag{18}$$

where the second term is negligible for large N. Then, we define the metabolic scaling exponent β as the fraction of the derivatives of the expected values of adult metabolism and mass.

$$\beta = \frac{d\ln \mathbb{E}[B_{\text{adult}}]}{d\ln \mathbb{E}[m_{\text{adult}}]} \tag{19}$$

After differentiating each quantity with respect to N, the steady-state allometric exponent can be calculated as:

$$\beta = \frac{AN + \frac{\alpha}{2}CN^{\alpha/2}}{AN + CN^{\alpha/2}} \cdot \frac{N + EN^{\alpha/2}}{N + \frac{\alpha}{2}EN^{\alpha/2}} \tag{20}$$

The maintenance term scales linearly with cell number, $B_M \propto N_c$, while the stochastic term scales as $H \propto N_c^{\alpha/2}$, producing an effective nonlinear allometric relationship between total metabolism and body mass. The exponent β interpolates continuously between sublinear and superlinear regimes as a mixture of $aN + bN^{\alpha/2}$. This simple mechanism generates scaling naturally from the interplay between deterministic processes and stochastic fluctuations and reproduces known biological scaling laws and can be generalized to other stochastic systems such as economic and informational systems.

D. Information-theoretic analysis

In order to find the optimal noise structure and the corresponding allometric exponent, first we measure for each α the Kullback-Leibler (KL) divergence between the conditional fluctuation distribution of the mesoscopic fluctuations r and α and the pooled distribution of r across α:

$$\begin{aligned} D_{\text{KL}}(\alpha, r) &= D_{\text{KL}}\big(p(r \mid \alpha) \parallel p(r)\big) \\ &= \sum_{i=1}^{n_{\text{bins}}} p(r_i \mid \alpha)\,\log\frac{p(r_i \mid \alpha)}{p(r_i)}. \end{aligned} \tag{21}$$

Here $p(r|\alpha)$ denotes the distribution of metabolic fluctuations generated under a given microscopic correlation exponent α, while $p(r)$ represents the pooled distribution across all correlation exponents. The KL divergence therefore quantifies how strongly macroscopic fluctuations depend on the microscopic correlation structure. For each species size (i.e., each N), we evaluate $D_{\text{KL}}(\alpha, r)$ across all α values between [0.9, 2]. Note that here we define optimal as maximal robustness of macroscopic fluctuations to microscopic noise. The optimization proceeds in two stages. First, we identify the correlation exponent where macroscopic fluctuations become least sensitive to microscopic parameters (information neutrality). Then, we determine the noise amplitude for which this information-neutral exponent reaches its maximum across species sizes (Fig.1B). This criterion selects the noise structure that simultaneously maximizes robustness and cross-scale correlations.

$$\alpha_{\min}(N, \phi) = \arg\min_{\alpha} D_{\text{KL}}(\alpha, r) \tag{22}$$

At the information neutrality point, α has minimal effect on the distribution of r. We define the information-neutral valley as the region in parameter space where the distribution of macroscopic fluctuations is least sensitive to variations in the microscopic correlation exponent α, quantified by a minimum in the $D_{KL}(\alpha, r)$. This minimum defines the most robust correlation exponent α_{min}. To reduce Monte Carlo noise, we smoothed (α, D_{KL}) trajectories before locating the minimum. Then, we calculate the corresponding allometric exponents $\beta(\alpha_{min})$ for each body size across all ϕ values.

The goal is to determine whether there exists a species–independent optimal noise scale ϕ^* at which the allometric scaling exponent is universal. To do so, we calculate the overlap of the species-specific $\alpha_{min} - \phi$ curve representing the universal crossover. This value or interval represent the point where all species exhibit maximal robustness to microscopic noise and at the same time maximally correlated microscopic noise structure. For each species we identify the noise values ϕ at which robustness is maximal, i.e. when $\alpha_{\min}(\phi, N)$ achieves its maximum over all possible ϕ values:

$$\phi^*(N) = \max_{\phi'} \alpha_{\min}(\phi, N) \tag{23}$$

which defines the structural ridge or crossover region. Then, for each ϕ^*, we compute the corresponding allometric scaling exponent β^*.

In this framework universality corresponds to a regime where coarse-grained observables become insensitive to microscopic parameters. To check for universality across species trajectories, we apply a min-max normalization ($x' = \frac{x - x_{\min}}{x_{\max} - x_{\min}}$) of α and β, performed independently for each species size (N). This procedure is consistent with the linear reparametrization of coupling space under the renormalization group [8, 48], and produces a dimensionless coordinate in which the trajectories collapse onto a universal curve. Although the Renormalization Group (RG) terminology used here is phenomenological, we explicitly test the stability of the information-neutral landscape under blocking transformations (see for more details in Supplementary Material).

As a complementary analysis, we quantify the sensitivity of the mesoscopic observable r to the scaling exponent α using the Fisher Information:

$$I_r(\alpha) = \sum_{i=1}^{n_{\text{bins}}} \frac{\left(\partial_\alpha p(r_i \mid \alpha)\right)^2}{p(r_i \mid \alpha)}. \tag{24}$$

$$\partial_\alpha p(r_i \mid \alpha) \approx \frac{p(r_i \mid \alpha + \delta) - p(r_i \mid \alpha - \delta)}{2\delta}, \tag{25}$$

where is set to $\delta = 0.02$. Then, calculating $I_r(\alpha_{min})$ gives the sensitivity of the most robust correlation exponent to microscopic fluctuations.

III. RESULTS

Our results reveal how information mapping from microscopic bursty cell dynamics to organism-level fluctuations shapes the species-level allometric exponent. First, we computed the KL divergence between the α and the organism-level metabolic fluctuations r across a α-ϕ grid for each species sizes (Fig.2A). In each parameter combination, a correlation exponent α_{min} can be identified, where the KL divergence reached its minimum (information-neutral valley). These minimum correlation exponents are in the range of $\sim$ 1.4–1.8 across species sizes. At α_{min} the metabolic fluctuations $r(t)$ retain the least sensitivity to the microscopic parameter α, indicating that the coarse-grained description is maximally insensitive to microscopic details.

To test for universality, we use the normalised trajectories of the exponents $\beta'(\alpha_{min})$ and α', which closely collapse across species, revealing a universal scaling law emerging at the point of minimal dependence on α (Fig.2B). This is consistent with the renormalization group theory, i.e. values of the control parameter at which the coarse-grained statistics become invariant under changes in microscopic parameters. In this framework, species size (N) plays the role of a system-size, where increasing N corresponds to moving toward the macroscopic limit, where microscopic details become irrelevant. These invariant structures are identified through the behavior of the KL divergence, therefore reflects an information-theoretic signature of universality.

As a next step, we calculated the crossover region where the minimum correlation exponents reach their maximum, which identifies the optimal fluctuation coefficient ϕ^* (Fig.2C). This represents the structural ridge where the correlation exponents are minimal and maximally size-dependent and correlated. This minimax criterion identifies the most robust noise structure. Within the crossover region, the metabolic scaling exponent is narrowly distributed around $\beta^* = 0.77$ with a standard deviation of 0.02. This indicates that, once coarse-grained to the crossover regime, species spanning six orders of magnitude in maximum cell number share an effectively universal metabolic scaling exponent that is in the vicinity of the 3/4 Kleiber's law. The tight clustering of β^* values across species in the universal region provides direct evidence for universality that does not rely on criticality.

Furthermore, we analysed the sensitivity of r with respect to α quantified by the Fisher Information. On the joint information plane where the KL divergence $D_{\text{KL}}[\alpha]$ has its minimum, the Fisher information is not divergent and of intermediate magnitude (Fig.2D). For large ϕ, the dynamics amplify noise, the Fisher and KL divergence decrease and the system becomes nearly insensitive to microscopic noise leading to diminishing allometric exponents.

IV. DISCUSSION

The presented analysis confirms that universality can arise from a simple information neutrality criterion in biological systems without phase transition. Contrary to the Edge of Chaos assumption [23], our results show that

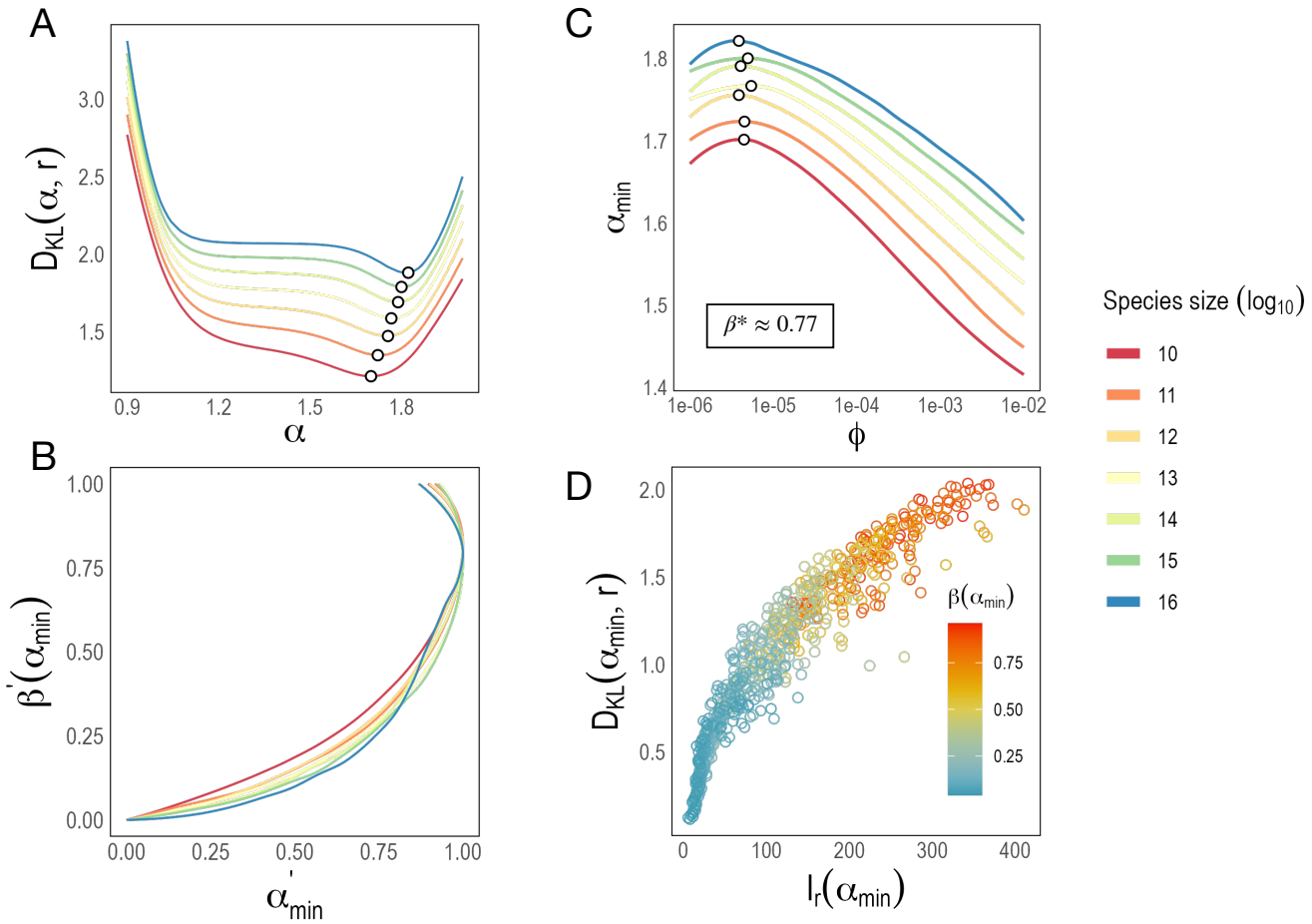

Figure 2 **Sensitivity of stochastic metabolic fluctuations across species sizes.** (A) An example of the KL divergence $D_{\mathrm{KL}}(\alpha, r)$ as a function of the microscopic correlation exponent α for a fixed fluctuation amplitude ($\log_{10}(\phi) = -5.3$). The minimum identifies the information-neutral exponent $\alpha_{\min}$ (black circles), where macroscopic metabolic fluctuations are least sensitive to the microscopic correlation structure. (B) Trajectories of the normalized scaling exponents $\beta'(\alpha_{\min})$ and α' across different noise amplitudes ϕ. Each curve corresponds to a species size, and the collapse of trajectories indicates universal behavior. (C) The maximized $\alpha_{\min}(\phi)$ identifies the structural ridge (black circles) corresponding to the optimal fluctuation scale ϕ^*, where microscopic correlations are strongest while remaining information-neutral. The corresponding metabolic scaling exponent β^* is shown. (D) Sensitivity of macroscopic fluctuations to the correlation exponent measured by the Fisher information.

metabolic scaling does not operate at a point of maximal information transmission or maximal sensitivity. Instead, universality emerges where the information transmission is minimal. The identified information-neutral valley corresponds to a noncritical crossover point or region where Fisher information is low to moderate and where organism-level metabolic fluctuations are maximally robust to microscopic stochasticity and species converge onto a universal scaling exponent. This indicates that universal metabolic scaling is the consequence of evolution tuning itself rather towards a point or plateau of maximal robustness then to critical boundary. Interestingly, the canonical 3/4 scaling exponent [20] emerges close to this robust crossover region.

These results correspond to a phenomenological RG, which emerges from coarse-graining of microscopic cellular bursts into mesoscopic metabolic fluctuations and consequently to macroscopic scaling laws. Importantly, the universality observed, i.e. the collapse of normalised α' and β' across species sizes, reflects a non-critical RG-like behavior. Note that here RG-like is used in a phenomenological sense, referring to the collapse of normalized trajectories under coarse-graining, rather than to a formally defined renormalization transformation. Such noncritical universal behaviors are increasingly recognized in biology. They appear in developmental scaling [24], neural population dynamics [30], biochemical reaction networks, protein aggregation, and scaling laws in ecosystems [2]. In these systems, robustness and universality emerge not from being near a phase transition, but

rather from the natural elimination of microscopic detail as biological units are aggregated into larger scales [27]. These findings are similar to a previous model, where universal metabolic scaling arises from life-history optimisation [47].

The large variance in observed empirical diversity is more likely a result of a locally optimised trait space rather than global optimisation. Evolution can be formalised as a stochastic adaptive walk on rugged landscapes [19, 25] constrained by historical contingencies and developmental architecture [1, 14, 41], which prevent natural selection from globally optimizing traits. The average scaling exponent of ~ 0.77 recovered in this analysis therefore represents a purely theoretical optimum closely coinciding with the observed Kleiber's law. Note that a local minimum appears around $\alpha \approx 1$, which corresponds to uncorrelated noise structure.

This study offers a new theoretical perspective on how information neutrality can give rise to universal scaling laws. More broadly, the framework developed here suggests that information neutrality principles may underlie the universal scaling behavior of noisy processes. This model is generalizable to other complex systems.

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Supplementary Materials for:
Information-Theoretic Origins of Universality in Stochastic Biological Systems

S1 Coarse-graining of mesoscopic fluctuations

To assess whether the information-neutral regime is stable under coarse-graining, we performed a blocking analysis of the mesoscopic fluctuation observable. Note that this analysis is intended only as a diagnostic test of RG-like phenomenology rather than as a formal renormalization-group analysis.

First, we define the coarse-grained fluctuation of the kth step by block summation,

$$R_k(t) = \sum_{i=0}^{k-1} r(t+i) = \log \frac{B(t+k)}{B(t)}. \tag{1}$$

This corresponds to aggregating fluctuations over k consecutive steps and is analogous to blocking transformations used in coarse-graining analyses.

For independent or weakly correlated increments, one would generically expect the variance of R_k to grow with k. However, across the explored parameter ranges we find that the standard deviation of R_k is constant over all block sizes. This happens because $r(t)$ is defined as a relative increment of a stationary process, such that aggregation does not lead to systematic broadening of the distribution. Therefore, no rescaling of R_k (e.g. z-score or $\sqrt{k}$ normalisation) is required to compare distributions across different coarse-graining levels.

Using the coarse-grained variable R_k, we computed the $D_{KL}(\alpha, r)$,

$$D_{\mathrm{KL}}(p(R_k \mid \alpha) \,\|\, p(R_k)) \,, \tag{2}$$

where $p(R_k)$ denotes the distribution pooled across α. We find that the KL landscape as a function of α is invariant under coarse-graining: both the shape of the landscape and the location of its minimum are unchanged as k is increased (Figure S2). This invariance indicates that the information-neutral exponent $\alpha_{\min}$ identified is stable under coarse-graining of mesoscopic fluctuations and therefore supports the phenomenological RG interpretation.

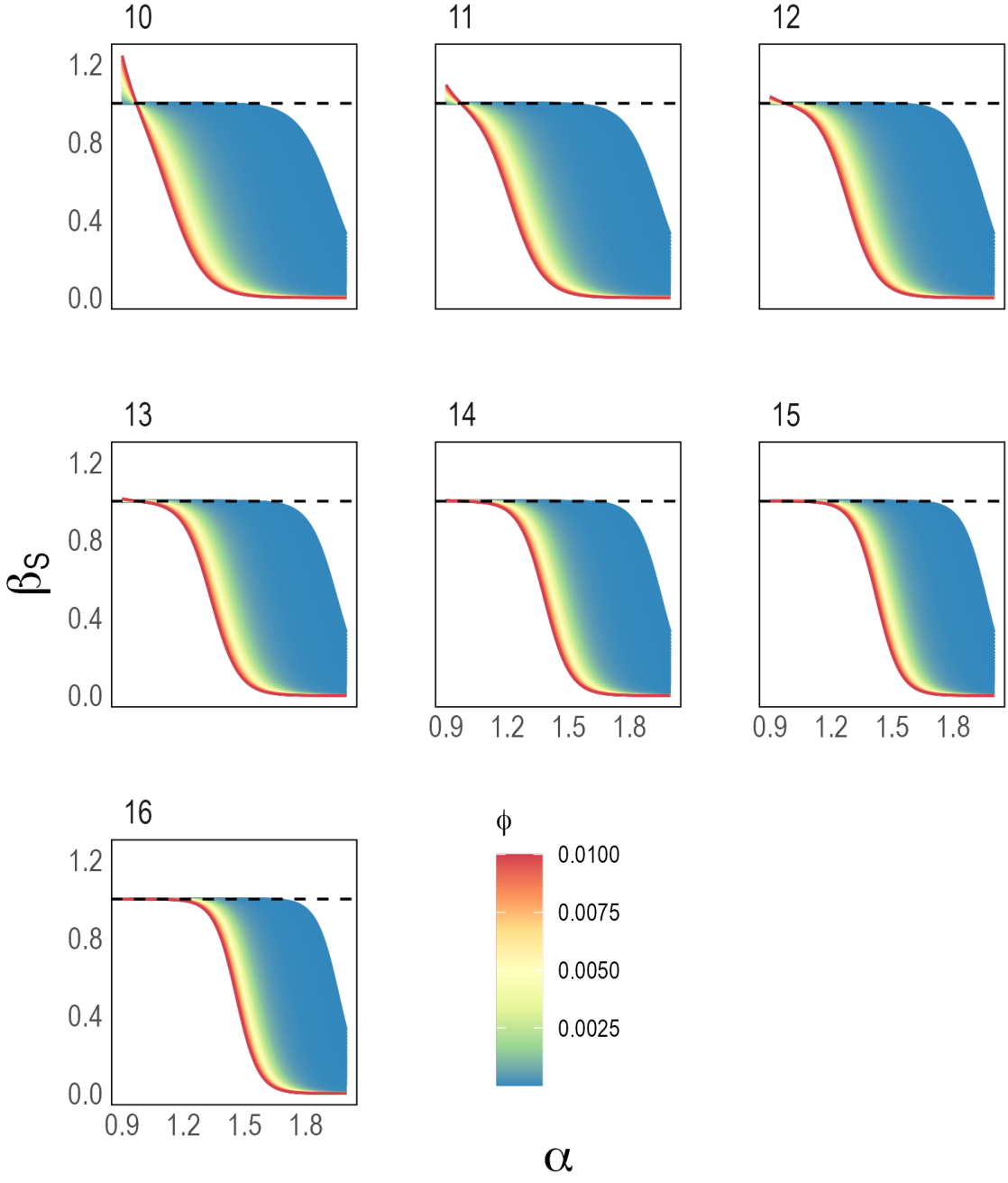

Figure S1: **Species allometric exponents by organism size across different noise structures (α and ϕ).**

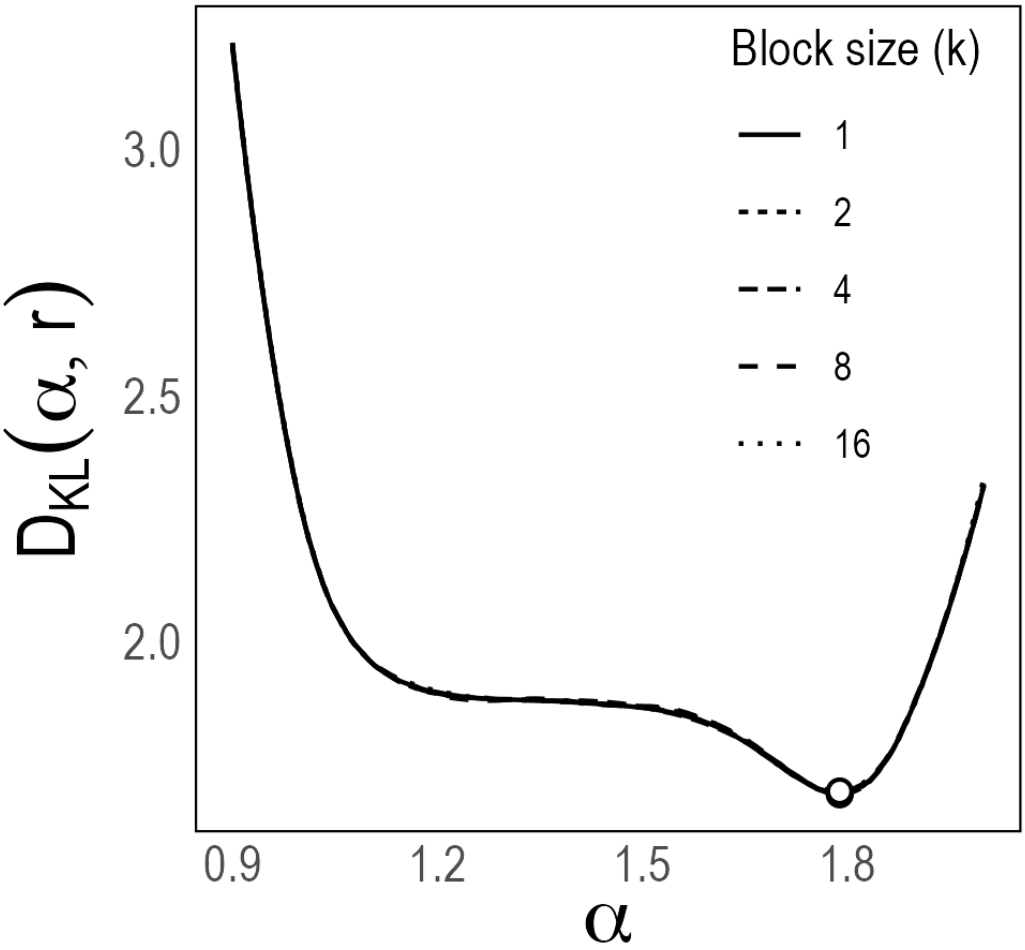

Figure S2: **Coarse-grained fluctuations.**

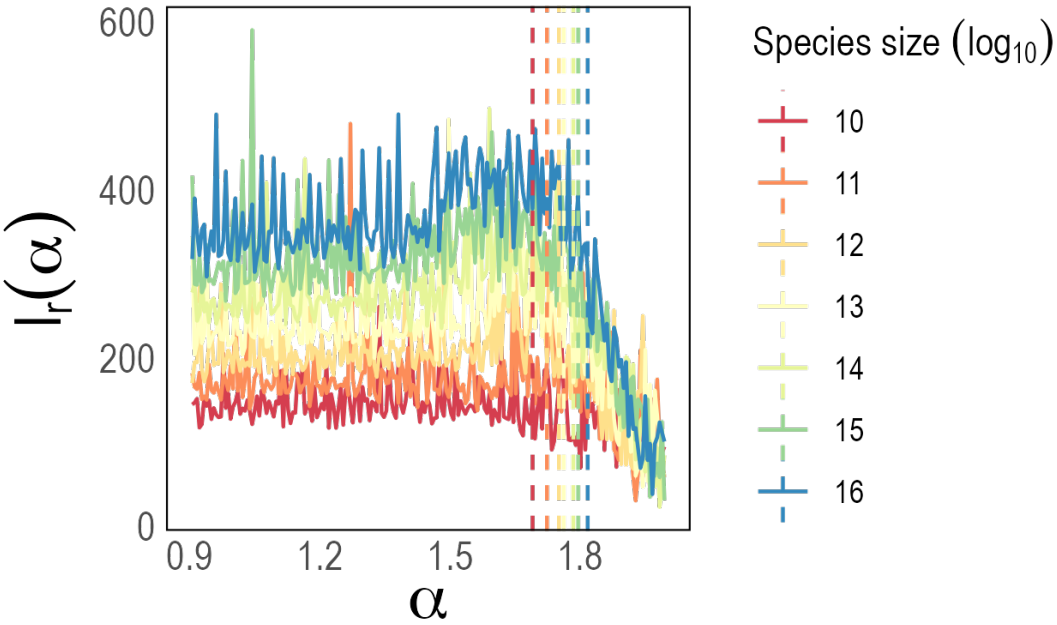

Figure S3: **Example of Fisher Information across** α. The dashed lines correspond to the α_{min} values, i.e. the minimum of D_{KL} curve with $\phi = 10^{-5.3}$. At that point, the Fisher Information metrics take an intermediate value.