

# The dynamics of starvation and recovery

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The eco-evolutionary dynamics of species are fundamentally linked to the energetic constraints of its constituent individuals. Of particular importance are the tradeoffs between reproduction and the dynamics of starvation and recovery. We introduce a minimal nutritional state-structured model that incorporates two classes of consumer: nutritionally replete, reproducing consumers, and undernourished, non-reproducing consumers. We obtain strong constraints on starvation and recovery rates by deriving allometric scaling relationships and find that population dynamics are typically driven to a steady state. Moreover, we find that these rates fall within a ‘refuge’ in parameter space, where the probability of extinction of the consumer population is minimized. We also show that our model provides a natural framework that predicts maximum body size for mammals by determining the relative stability of an otherwise homogeneous population to a mutant population with altered percent body fat, providing a principled mechanism for a within-lineage driver of Cope’s rule.

The behavioral ecology of all organisms is influenced by the energetic state of individuals, which directly influences how they invest reserves in uncertain environments. Such behaviors are generally manifested as tradeoffs between investing in somatic maintenance and growth, or allocating energy towards reproduction<sup>1–3</sup>. The timing of these behaviors responds to selective pressure, as the choice of the investment impacts future fitness<sup>4–6</sup>. The influence of resource limitation on an organism’s ability to maintain its nutritional stores may lead to repeated delays or shifts in reproduction over the course of an organism’s life.

The balance between (a) somatic growth and maintenance, and (b) reproduction depends on resource availability<sup>7</sup>. For example, reindeer invest less in calves born after harsh winters (when the mother’s energetic state is depleted) than in calves born after moderate winters<sup>8</sup>. Many bird species invest differently in broods during periods of resource scarcity compared to normal periods<sup>9,10</sup>, sometimes delaying or even foregoing reproduction for a breeding season<sup>1,11,12</sup>. Even freshwater and marine zooplankton have been observed to avoid reproduction under nutritional stress<sup>13</sup>, and those that do reproduce have lower survival rates<sup>2</sup>. Organisms may also separate maintenance and growth from reproduction over space and time: many salmonids, birds, and some mammals return to migratory breeding grounds to reproduce after one or multiple seasons in resource-rich environments where they accumulate nutritional reserves<sup>14–16</sup>.

Physiology also plays an important role in regulating reproductive expenditures during periods of resource limitation. The data collected thus far has shown that diverse mammals (47 species in 10 families) exhibit delayed implantation, whereby females postpone fetal development (blastocyst implantation) until nutritional reserves can be accumulated<sup>17,18</sup>. Many other species (including humans) suffer irregular menstrual cycling and higher abortion rates during periods of nutritional stress<sup>19,20</sup>.

In the extreme case of unicellular organisms, nutrition is unavoidably linked to reproduction because the nutritional state of the cell regulates all aspects of the cell cycle<sup>21</sup>. The existence of so many independently evolved mechanisms across such a diverse suite of organisms highlights the importance and universality of the fundamental tradeoff between somatic and reproductive investment. However the general dynamic implications of these constraints are unknown.

Though straightforward conceptually, incorporating the energetic dynamics of individuals<sup>22</sup> into a population-level framework<sup>22,23</sup> presents numerous mathematical obstacles<sup>24</sup>. An alternative approach involves modeling the macroscale relations that guide somatic versus reproductive investment in a consumer-resource system. For example, macroscale Lotka-Volterra models assume that the growth rate of the consumer population depends on resource density, thus *implicitly* incorporating the requirement of resource availability for reproduction<sup>25</sup>.

In this work, we adopt an alternative approach in which we *explicitly* account for resource limitation and the subsequent effects of starvation. Namely, only individuals with sufficient energetic reserves can reproduce. Such a constraint leads to reproductive time lags due to some members of the population going hungry and then recovering. Additionally, we incorporate the idea that reproduction is strongly constrained allometrically<sup>3</sup>, and as we shall show, these constraints influence the ensuing population dynamics in dramatic ways.

## Nutritional state-structured model (NSM)

We begin by defining a minimal Nutritional State-structured population Model (NSM), where the consumer population is partitioned into two states: (a) an energetically replete (full) state  $F$ , where the consumer reproduces at a constant rate  $\lambda$  and does not die from starvation, and (b) an energetically deficient (hungry) state

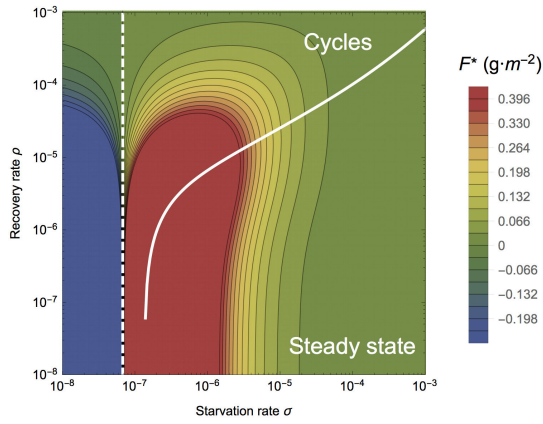


Figure 1: The transcritical (TC; dashed line) and Hopf bifurcation (solid line) as a function of the starvation rate  $\sigma$  and recovery rate  $\rho$  for a 100g consumer. These bifurcation conditions separate parameter space into unphysical (left of the TC), cyclic, and steady state dynamic regimes. The colors show the steady state densities for the energetically replete consumers  $F^*$ .

$H$ , where the consumer does not reproduce but dies by starvation at rate  $\mu$ . The underlying resource  $R$  evolves by logistic growth with an intrinsic growth rate  $\alpha$  and a carrying capacity  $C$ . The rate at which consumers transition between states and consume resources is dependent on their overall abundance, the abundance of resources, the efficiency of converting resources into metabolism, and how that metabolism is partitioned between maintenance and growth purposes. In the Supplementary Information we provide a fully mechanistic model for each of these dynamics and constants, and show that the system produces a simple non-dimensional form which we describe below.

Consumers transition from the full state  $F$  to the hungry state  $H$  at a rate  $\sigma$ —the starvation rate—and also in proportion to the absence of resources  $(1 - R)$ . Conversely, consumers recover from state  $H$  to state  $F$  at rate  $\xi\rho$  and in proportion to  $R$ , where  $\xi$  represents a ratio between maximal resource consumption and the carrying capacity of the resource. Resources are eaten by the hungry consumers at rate  $\rho R + \delta$ , that accounts for their somatic growth ( $\rho R$ ) and maintenance ( $\delta$ ). Full consumers eat resources at a constant rate  $\beta$  that accounts for maximal maintenance and somatic growth (see Supplementary Information for mechanistic derivations of these rates from resource energetics). The NSM represents an ecologically motivated fundamental extension of the idealized starving random walk model of foraging, which focuses on resource depletion, to include reproduction and resource replenishment<sup>26–28</sup>, and is a more general formulation than previous models incorporating starvation<sup>29</sup>.

In the mean-field approximation, in which the consumers and resources are perfectly mixed, their densities evolve according to the rate equations

$$\begin{aligned}\dot{F} &= \lambda F + \xi\rho R H - \sigma(1 - R)F, \\ \dot{H} &= \sigma(1 - R)F - \xi\rho R H - \mu H, \\ \dot{R} &= \alpha(1 - R)R - (\rho R + \delta)H - \beta F\end{aligned}\quad (1)$$

This system of nondimensional equations follows from a set of first-principle relationships for resource consumption and growth (see Supplementary Information for a full derivation and the dimensional form). Notice that the total consumer density  $F + H$  evolves according to  $\dot{F} + \dot{H} = \lambda F - \mu H$ . This resembles the equation of motion for the predator density in the classic Lotka-Volterra model<sup>30</sup>, except that the resource density does not appear in the growth term. The rate of reproduction is independent of resource density because it is assumed that the satiated state of the full consumer allows it to partition a constant amount of energy towards reproduction, whereas a starved consumer partitions no energy towards reproduction. The rate of reproduction for the total consumer density is dependent on resource density, which determines the size of the full and starved portions of the consumer population. Similarly, the consumer maintenance terms ( $\delta H$  and  $\beta F$ ) are independent of resource density because they represent a minimal energetic requirement for consumers in the  $H$  and  $F$  state, respectively. It follows that model predictions are robust only when  $R \gg 0$ , which holds for all cases that we explore.

From the solution to the single internal fixed point (Eq. (2), see Methods), an obvious constraint on the NSM is that the reproduction rate  $\lambda$  must be less than the starvation rate  $\sigma$ , so that the consumer and resource densities are positive. The condition  $\sigma = \lambda$  thus represents a transcritical (TC) bifurcation<sup>31</sup> that demarcates a physical from an unphysical regime where all steady-state densities become negative after intersecting the trivial fixed point  $(F^*, H^*, R^*) = (0, 0, 0)$ . The biological implication of the constraint  $\lambda < \sigma$  has a simple interpretation—the rate at which a macroscopic organism loses mass due to lack of resources is generally much faster than the rate of reproduction. As we will discuss below, this inequality is a natural consequence of allometric constraints<sup>3</sup> for organisms within empirically observed body size ranges.

In the physical regime of  $\lambda < \sigma$ , the fixed point (2) may either be a stable node or a limit cycle (Fig. 1). In continuous-time systems, a limit cycle arises when a pair of complex conjugate eigenvalues crosses the imaginary axis to attain positive real parts<sup>32</sup>. This Hopf bifurcation is defined by  $\text{Det}(\mathbf{S}) = 0$ , with  $\mathbf{S}$  the Sylvester matrix, which is composed of the coefficients of the characteristic polynomial of the Jacobian matrix<sup>33</sup>. As the system parameters are tuned to be within the stable regime, but close to the Hopf bifurcation, the amplitude of the transient cycles becomes large. Given that ecological systems are constantly being perturbed<sup>34</sup>, the onset of transient cycles, even though they decay with time in the mean-field description, can increase extinction risk<sup>35–37</sup>.

When the starvation rate  $\sigma \gg \lambda$ , a substantial frac-

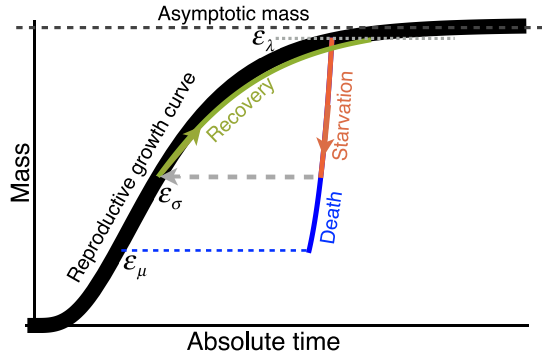


Figure 2: The growth trajectory over absolute time of an individual organism as a function of body mass. Initial growth follows the black trajectory to an energetically replete reproductive adult mass  $m = \epsilon_\lambda M$ . Starvation follows the red trajectory to  $m = \epsilon_\sigma \epsilon_\lambda M$ . Recovery follows the green curve to the replete adult mass, where this trajectory differs from the original growth because only fat is being regrown which requires a longer time to reach  $\epsilon_\lambda M$ . Alternatively, death from starvation follows the blue trajectory to  $m = \epsilon_\mu \epsilon_\lambda M$ .

tion of the consumers are driven to the hungry non-reproducing state. Because reproduction is inhibited, there is a low steady-state consumer density and a high steady-state resource density. However, if  $\sigma/\lambda \rightarrow 1$  from above, the population is overloaded with energetically-replete (reproducing) individuals, thereby promoting transient oscillations between the consumer and resource densities (Fig. 1). If the starvation rate is low enough that the Hopf bifurcation is crossed, these oscillations become stable over time. This threshold occurs at higher values of the starvation rate as the recovery rate  $\rho$  increases, such that the range of parameter space giving rise to cyclic dynamics also increases with higher recovery rates.

While there are no *a priori* constraints on the parameters in the NSM, we expect that each species should be restricted to a distinct portion of the parameter space. We use allometric scaling relations to constrain the covariation of rates in a principled and biologically meaningful manner (see Methods). Allometric scaling relations highlight common constraints and average trends across large ranges in body size and species diversity. Many of these relations can be derived from a small set of assumptions and in the Methods we describe our framework to determine the covariation of timescales and rates across a range of body sizes for each of the key parameters of our model (cf. ref.<sup>38</sup>).

Nearly all of the rates described in the NSM are determined by consumer metabolism, which can be used to describe a variety of organismal features<sup>39</sup>. We derive relationships for the rates of reproduction, starvation, recovery, and mortality based on first principles, and as a function of an organism's body size and metabolic rate (see Methods). Because we aim to explore the

starvation-recovery dynamics as a function of an organism's body mass  $M$ , we parameterize these rates in terms of the *percent* gain and loss of the asymptotic (maximum) body mass,  $\epsilon M$ , where different values of  $\epsilon$  define different states of the consumer (Fig. 2; see Methods for derivations of allometrically constrained rate equations). Although the rate equations (1) are general and can in principle be used to explore the starvation recovery dynamics for most organisms, here we focus on allometric relationships for terrestrial-bound lower trophic level endotherms (see Supplementary Information for values), specifically herbivorous mammals, which range from a minimum of  $M \approx 1\text{g}$  (the Etruscan shrew *Suncus etruscus*) to a maximum of  $M \approx 10^7\text{g}$  (the early Oligocene Indricotheriinae and the Miocene Deinotheriinae). Investigating other classes of organisms would simply involve altering the metabolic exponents and scalings associate with  $\epsilon$ . Moreover, we emphasize that our allometric equations describe mean relationships, and do not account for the (sometimes considerable) variance associated with individual species.

## Extinction risk

As the allometric derivations of the NSM rate laws reveal (see Methods), starvation and recovery rates are not independent parameters, and the biologically relevant portion of the phase space shown in Fig. 1 is constrained via covarying parameters. Given the parameters of terrestrial endotherms, we find that the starvation rate  $\sigma$  and the recovery rate  $\rho$  are constrained to lie within a small region of potential values for the known range of body sizes  $M$ . Indeed, starvation and recovery rates across all values of  $M$  fall squarely in the steady state region at some distance from the Hopf bifurcation. This suggests that cyclic population dynamics should be rare, particularly in environments where resources are limiting.

Higher rates of starvation result in a larger flux of the population to the hungry state. In this state, reproduction is absent, thus increasing the likelihood of extinction. From the perspective of population survival, it is the rate of starvation relative to the rate of recovery that determines the long-term dynamics of the various species (Fig. 1). We therefore examine the competing effects of cyclic dynamics vs. changes in steady-state density on extinction risk, both as functions of  $\sigma$  and  $\rho$ . To this end, we computed the probability of extinction, where we define extinction as a population trajectory falling below one fifth of the allometrically constrained steady state at any time between  $t = 10^8$  and  $t = 10^{10}$ . This procedure is repeated for 50 replicates of the continuous-time system shown in Eq. 1 for organisms with mass ranging from  $10^2$  to  $10^6$  grams. In each replicate the initial densities are chosen to be  $(XF^*, XH^*, R^*)$ , with  $X$  a random variable that is uniformly distributed in  $[0, 2]$ . By allowing the rate of starvation to vary, we assessed extinction risk across a range of values for  $\sigma$  and  $\rho$  between ca.  $10^{-7}$  to  $10^{-3}$ . As expected, higher rates of extinction correlate with both high values of  $\sigma$  if  $\rho$  is small, and high values

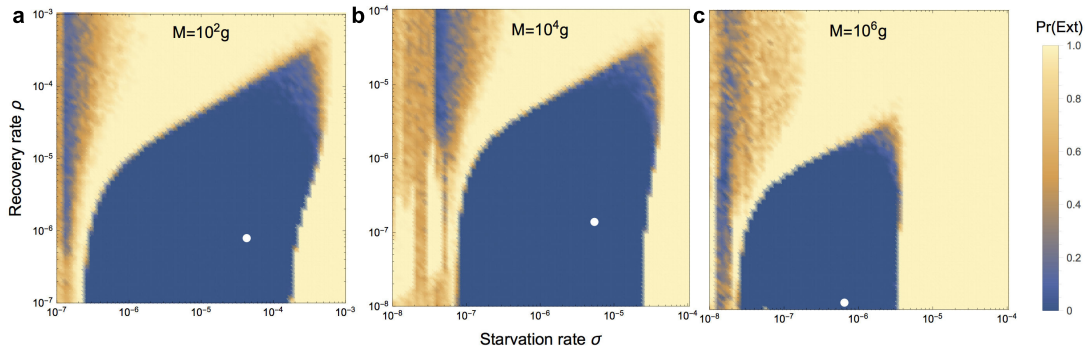


Figure 3: Probability of extinction for a consumer with (a)  $M = 10^2\text{g}$ , (b)  $M = 10^4\text{g}$ , and (c)  $M = 10^6\text{g}$  as a function of the starvation rate  $\sigma$  and recovery rate  $\rho$ , where the initial density is given as  $(XF^*, XH^*, R^*)$ , where  $X$  is a random uniform variable in  $[0, 2]$ . Note the change in scale for  $M = 10^4$  and  $M = 10^6\text{g}$ . Extinction is defined as the population trajectory falling below  $0.2 \times$  the allometrically constrained steady state. The white points denote the allometrically constrained starvation and recovery rate.

of  $\rho$  if  $\sigma$  is small. For low values of  $\sigma$  and high values of  $\rho$ , the increased extinction risk results from transient cycles with larger amplitudes as the system nears the Hopf bifurcation (Fig. 3). For high values of  $\sigma$  and low values of  $\rho$ , increased extinction risk arises because of the decrease in the steady-state consumer population density (Figs. 1b, 3). This interplay creates an ‘extinction refuge’, such that for a constrained range of  $\sigma$  and  $\rho$ , extinction probabilities are minimized.

We find that the allometrically constrained values of  $\sigma$  and  $\rho$  fall squarely within the extinction refuge across a range of  $M$  (Fig. 3a-c, white points). These values are close enough to the Hopf bifurcation to avoid low steady-state densities, and far enough away to avoid large-amplitude transient cycles. The feature that allometric values of  $\sigma$  and  $\rho$  fall within this relatively small window supports the possibility that a selective mechanism has constrained the physiological conditions that drive starvation and recovery rates within populations. Such a mechanism would select for organism physiology that generates appropriate  $\sigma$  and  $\rho$  values that serve to minimize extinction risk. This selection could occur via the tuning of body fat percentages, metabolic rates, and biomass maintenance efficiencies. We also find that as body size increases, the amount of low extinction risk parameter space becomes smaller (Fig. 3a-c), suggesting that the population dynamics of larger organisms are more sensitive to variability in physiological rates controlling starvation and recovery. This finding is in accordance with, and may serve as contributing support for, observations of increased extinction risk among larger mammals<sup>40</sup>. To summarize, our finding that the allometrically-determined parameters fall within this low extinction probability region suggests that the NSM dynamics may both drive—and constrain—natural animal populations.

## Discussion

Metabolite transport constraints are widely thought to

place strict boundaries on biological scaling<sup>39,41,42</sup> and thereby lead to specific predictions on the minimum possible body size for organisms<sup>43</sup>. Above this bound, a number of energetic and evolutionary mechanisms have been explored to assess the costs and benefits associated with larger body masses, particularly for mammals. One important such example is the *fasting endurance hypothesis*, which contends that larger body size, with consequent lower metabolic rates and increased ability to maintain more endogenous energetic reserves, may buffer organisms against environmental fluctuations in resource availability<sup>44</sup>. Over evolutionary time, terrestrial mammalian lineages show a significant trend towards larger body size (known as Cope’s rule)<sup>45–48</sup>, and it is thought that within-lineage drivers generate selection towards an optimal upper bound of roughly  $10^7$  grams<sup>45</sup>, a value that is likely limited by higher extinction risk for large taxa over longer timescales<sup>46</sup>. These trends are thought to be driven by a combination of climate change and niche availability<sup>48</sup>; however the underpinning energetic costs and benefits of larger body sizes, and how they influence dynamics over ecological timescales, have not been explored. We argue that the NSM provides a suitable framework to explore these issues.

The NSM correctly predicts that species with smaller masses have larger steady-state population densities (Fig. 4a). Similar predictions have been made for carnivore populations using alternative consumer-resource models<sup>49</sup>. Moreover, we show that the NSM provides independent theoretical support for the energy equivalence hypothesis and Damuth’s Law<sup>50–52</sup>. The energy equivalence hypothesis argues that the total energy use,  $B_{\text{tot}}$ , of a population is constant independent of species size<sup>50–52</sup>. This hypothesis is based on observations showing that the steady state abundance,  $N^*$ , of a species is proportional to the inverse of individual metabolism, such that  $N^* \propto M^{-3/4}/B_0$ <sup>51,52</sup>. This relationship implies that  $B_{\text{tot}} = N^*B(M) = Q$ , where  $Q$  is a constant, and has been shown to hold in both mammalian and vas-



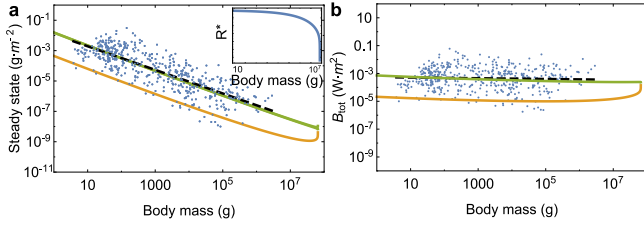


Figure 4: (a) Consumer steady states  $F^*$  (green) and  $H^*$  (orange) as a function of body mass. Inset: Resource steady state  $R^*$  as a function of consumer body mass. (b) Total energetic use  $B_{\text{tot}}$  of consumer populations at the steady state as a function of body mass. The data are from Damuth<sup>50</sup> and have been converted to total population metabolism using the allometric relationships for metabolic rate (please see Supplementary Information and Refs.<sup>53–55</sup>).

340 cular plant communities<sup>50–52</sup>. Figure 4a shows that both  
 341  $F^*$  and  $H^*$  scale as  $M^{-\eta}$  over a wide range of organism  
 342 sizes and Figure 4b shows that  $F^*B$  is nearly constant  
 343 over this same range. This result is remarkable because it  
 344 illustrates that the steady state values of the NSM com-  
 345 bined with the derived timescales naturally give rise to  
 346 energy equivalence.

347 Our model shows that the equivalence breaks down at  
 348 large  $M$  suggesting that this maximum is a hard limit  
 349 where deviations outside of this range are energetically  
 350 suboptimal. In the framework of our model, the total  
 351 metabolic rate of  $F$  and  $H$  becomes infinite at a finite  
 352 mass, and occur at the same scale where the steady state  
 353 resources vanish (Fig. 4). This asymptotic behavior is  
 354 governed by body sizes at which  $\epsilon_\mu$  and  $\epsilon_\lambda$  equal zero,  
 355 causing the timescales to become infinite and the rates  
 356  $\mu$  and  $\lambda$  to equal zero. The  $\mu = 0$  asymptote occurs  
 357 first when  $f_0 M^{\gamma-1} + u_0 M^{\zeta-1} = 1$ , and corresponds to  
 358  $(F^*, H^*, R^*) = (0, 0, 0)$ . This point predicts a strong  
 359 upper bound on mammalian body size and occurs at  
 360  $M_{\text{max}} = 6.54 \times 10^7$ . Moreover,  $M_{\text{max}}$ , which is entirely  
 361 determined by the population-level consequences of  
 362 energetic constraints, is within an order of magnitude of  
 363 the maximum body size observed in the North American  
 364 mammalian fossil record<sup>45</sup>, as well as the mass predicted  
 365 from an evolutionary model of body size evolution<sup>46</sup>.  
 366 It should be noted that the asymptotic behavior and  
 367 predicted upper bound depend only on the scaling of  
 368 body composition and are independent of the resource  
 369 parameters. We also note that the prediction of an  
 370 asymptotic limit on mammalian size parallels work  
 371 on microbial life where an upper and lower bound  
 372 on bacterial size, and an upper bound on single cell  
 373 eukaryotic size, is predicted from similar growth and  
 374 energetic scaling relationships<sup>3,56</sup>.

376 The NSM predicts that the steady state resource  
 377 density  $R^*$  decreases with increasing body size of the  
 378 consumer population (Fig. 4a, inset), and classic re-  
 379 source competition theory predicts that the species sur-

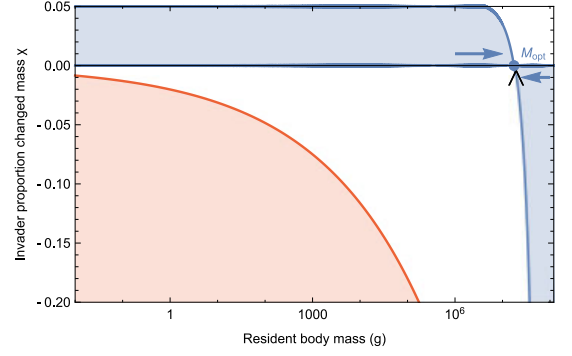


Figure 5: Invasion feasibility for organisms with a proportional change in mass  $\chi$  against a population with a resident body mass  $M$ . The blue region denotes proportions of modified mass  $\chi$  resulting in successful invasion. The red region denotes values of  $\chi$  that result in a mass that is below the starvation threshold and are thus infeasible. Arrows point to the predicted optimal mass from our model  $M_{\text{opt}} = 1.748 \times 10^7$ , which may serve as an evolutionary attractor for body mass. The black wedge points to the largest body mass known for terrestrial mammals (*Deinotherium* spp.) at  $1.74 \times 10^7$  g<sup>47</sup>.

380 living on the lowest resource abundance will outcom-  
 381 pete others<sup>57–59</sup>. Thus, the combined NSM steady-state  
 382 dynamics and allometric timescales predict that larger  
 383 mammals have an intrinsic competitive advantage given  
 384 a common resource, but these absolute limits do not offer  
 385 a within-lineage mechanism by which larger body sizes  
 386 are selected for or against. We will now show that the  
 387 NSM indeed provides a mechanistic understanding of the  
 388 energetic dynamics that give rise to both observed limi-  
 389 tations on mammalian body size, as well as the observed  
 390 trend towards larger body size over evolutionary time  
 391 known as Cope’s Rule.

392 To examine whether the NSM could provide such a  
 393 mechanism, we begin by noting that a theoretical upper  
 394 bound on mammalian body size is given by  $\epsilon_\sigma = 0$ , where  
 395 mammals are entirely composed of metabolic reserves,  
 396 and this occurs at  $M = 8.3 \times 10^8$ , or 120 times the mass of  
 397 a male African elephant. Next we examine to what extent  
 398 a more realistic upper bound to body mass may serve  
 399 as an evolutionary attractor, thus providing a suitable  
 400 within-lineage mechanism for Cope’s rule.

401 We directly assess the susceptibility of an otherwise  
 402 homogeneous population to invasion by a mutated sub-  
 403 set of the population (denoted by  $'$ ) where individuals  
 404 have a modified proportion of body fat  $M' = M(1 + \chi)$ .  
 405 If  $\chi < 0$ , individuals within the invading mutant pop-  
 406 ulation have fewer metabolic reserves, and if  $\chi > 0$ ,  
 407 individuals have more metabolic reserves than the res-  
 408 ident population. For the allowable values of  $\chi$  the ad-  
 409 justed mass should exceed the minimal amount of body  
 410 fat,  $1 + \chi > \epsilon_\sigma$ , and the adjusted time to reproduce  
 411 must be positive, which given Equation 4, implies that  
 412  $1 - \epsilon_\lambda^{1-\eta} (1 + \chi)^{1-\eta} > 0$ . Together these conditions imply  
 413 that  $\chi \in (-f_0 M^{\gamma-1}, 1/\epsilon_\lambda - 1)$  where the upper bound  
 414 approximately equals 0.05 and the lower bound is mass-

dependent. The modified mass adjusts our model via the altered rates of starvation  $\sigma(M')$ , recovery  $\rho(M')$ , and the maintenance of both starving  $\delta(M')$  and full consumers  $\beta(M')$ . Importantly,  $\epsilon_\sigma$ , which determines the point along the growth curve that defines the body composition of starved foragers, is assumed to remain unchanged for the invader population (see Supplementary Information for detailed derivations of invader rates).

To assess the susceptibility of the resident population to invasion, we determine which consumer pushes the steady-state resource density to lower values for a given value of  $\chi$ , with the expectation that populations able to survive on lower resource densities have a competitive advantage<sup>57</sup>. We find that for  $M \leq 1.748 \times 10^7$ g, having additional body fat ( $\chi > 0$ ) results in a lower steady state resource density ( $R'^* < R^*$ ), such that the invader has an intrinsic competitive advantage over the resident population (Fig. 5). However, for  $M > 1.748 \times 10^7$ g, leaner individuals ( $\chi < 0$ ) have lower resource steady state densities, switching the advantage from having more metabolic reserves to having less.

The observed switch in susceptibility as a function of  $\chi$  at  $M_{\text{opt}} = 1.748 \times 10^7$ g thus serves as an attractor, such that the NSM predicts organismal mass to increase if  $M < M_{\text{opt}}$  and decrease if  $M > M_{\text{opt}}$ . This value is close to but smaller than the asymptotic upper bound for terrestrial mammal body size predicted by the NSM, however it is remarkably close to independent estimates of the largest land mammals, the early Oligocene *Indricotherium* at ca.  $1.5 \times 10^7$ g and the late Miocene *Deinotherium* at ca.  $1.74 \times 10^7$ g<sup>47</sup>. Additionally, our calculation of  $M_{\text{opt}}$  as a function of mass-dependent physiological rates is similar to theoretical estimates of maximum body size<sup>46</sup>, and provides independent theoretical support for the observation of a ‘maximum body size attractor’ for North American mammals outlined by Alroy<sup>45</sup>. While the state of the environment, as well as the competitive landscape, will determine whether specific body sizes are selected for or against<sup>48</sup>, we propose that the dynamics of starvation and recovery described in the NSM provide a general within-lineage mechanism for the evolution of larger body size among terrestrial mammals.

The energetics associated with somatic maintenance, growth, and reproduction are important elements that influence the dynamics of all populations<sup>11</sup>. The NSM is a minimal and general model that incorporates the dynamics of starvation and recovery that are expected to occur in resource-limited environments. By incorporating allometric relations between the rates in the NSM, we found: (i) different organismal masses have distinct population dynamic regimes, (ii) allometrically-determined rates of starvation and recovery appear to minimize extinction risk, and (iii) the dynamic consequences of these rates may introduce additional drivers and hard boundaries on the evolution of maximum body size. We suggest that the NSM offers a means by which the dynamic consequences of energetic constraints can be assessed using macroscale interactions between and among species.

Future efforts will involve exploring the consequences of these dynamics in a spatially explicit framework, thus incorporating elements such as movement costs and spatial heterogeneity, which may elucidate additional tradeoffs associated with the dynamics of starvation and recovery.

## Methods

**Analytical solution to the NSM** Equation (1) has three fixed points: two trivial fixed points at  $(F^*, H^*, R^*) = (0, 0, 0)$  and  $(0, 0, 1)$ , and one non-trivial, internal fixed point at

$$\begin{aligned} F^* &= (\sigma - \lambda) \frac{\alpha \lambda \mu^2 (\mu + \xi \rho)}{A(\lambda \rho B + \mu \sigma (\beta \mu + \lambda (\delta + \rho)))}, \\ H^* &= (\sigma - \lambda) \frac{\alpha \lambda^2 \mu (\mu + \xi \rho)}{A(\lambda \rho B + \mu \sigma (\beta \mu + \lambda (\delta + \rho)))}, \\ R^* &= (\sigma - \lambda) \frac{\mu}{A}. \end{aligned} \quad (2)$$

where  $A = (\lambda \xi \rho + \mu \sigma)$  and  $B = (\beta \mu \xi + \delta \lambda \xi - \lambda \mu)$ . The stability of this fixed point is determined by the Jacobian matrix  $\mathbf{J}$ , where each matrix element  $J_{ij} = \partial \dot{X}_i / \partial X_j$  when evaluated at the internal fixed point, and  $\mathbf{X}$  is the vector  $(F, H, R)$ . The parameters in Eq. (1) are such that the real part of the largest eigenvalue of  $\mathbf{J}$  is negative, so that the system is stable with respect to small perturbations from the fixed point. Because this fixed point is unique, it is the global attractor for all population trajectories for any initial condition where the resource and consumer densities are both nonzero.

**Metabolic scaling relationships** The scaling relation between an organism’s metabolic rate  $B$  and its body mass  $M$  at reproductive maturity is known to scale as  $B = B_0 M^\eta$ <sup>60</sup>, where the scaling exponent  $\eta$  is typically close to 2/3 or 3/4 for metazoans (e.g., ref.<sup>39</sup>), and has taxonomic shifts for unicellular species between  $\eta \approx 1$  in eukaryotes and  $\eta \approx 1.76$  in bacteria<sup>3,61</sup>.

Several efforts have shown how a partitioning of  $B$  between growth and maintenance purposes can be used to derive a general equation for both the growth trajectories and growth rates of organisms ranging from bacteria to metazoans<sup>3,53–55,62</sup>. This relation is derived from the simple balance condition  $B_0 m^\eta = E_m \dot{m} + B_m m$ ,<sup>3,53–55,62</sup> where  $E_m$  is the energy needed to synthesize a unit of mass,  $B_m$  is the metabolic rate to support an existing unit of mass, and  $m$  is the mass of the organism at any point in its development. This balance has the general solution<sup>3,63</sup>

$$\left( \frac{m(t)}{M} \right)^{1-\eta} = 1 - \left[ 1 - \left( \frac{m_0}{M} \right)^{1-\eta} \right] e^{-a(1-\eta)t/M^{1-\eta}}, \quad (3)$$

where, for  $\eta < 1$ ,  $M = (B_0/B_m)^{1/(1-\eta)}$  is the asymptotic mass,  $a = B_0/E_m$ , and  $m_0$  is mass at birth, itself varying allometrically (see Supplementary Information). We now use this solution to define the timescale for reproduction and recovery from starvation (Fig. 2; see<sup>55</sup> for a detailed presentation of these timescales). The time that it takes to reach a particular mass  $\epsilon M$  is given by the timescale

$$\tau(\epsilon) = \ln \left[ \frac{1 - (m_0/M)^{1-\eta}}{1 - \epsilon^{1-\eta}} \right] \frac{M^{1-\eta}}{a(1-\eta)}, \quad (4)$$

where we will define values of  $\epsilon$  to describe a set of rates within our model. For the time to reproduce,  $t_\lambda = \tau(\epsilon_\lambda)$ , where  $\epsilon_\lambda$

is the fraction of the asymptotic mass where an organism is reproductively mature and should be close to one (typically  $\epsilon_\lambda \approx 0.95$ ; <sup>53</sup>). The growth rate is then given by  $\lambda = \ln(v)/t_\lambda$  where  $v$  is the number of offspring produced, and for any constant value of  $\epsilon_\lambda$ , this rate will scale as  $\lambda \propto M^{\eta-1}$  for  $M \gg m_0$  <sup>3,53–55,62</sup>.

The rate of recovery  $\rho = 1/t_\rho$  requires that an organism accrues sufficient tissue to transition from the hungry to the full state. Since only certain tissues can be digested for energy (for example the brain cannot be degraded to fuel metabolism), we define the rates for starvation, death, and recovery by the timescales required to reach, or return from, specific fractions of the replete-state mass (see Supplementary Information, Table I for parameterizations). We define  $m_\sigma = \epsilon_\sigma M$ , where  $\epsilon_\sigma < 1$  is the fraction of replete-state mass where reproduction ceases. This fraction will deviate from a constant if tissue composition systematically scales with adult mass. For example, making use of the observation that body fat in mammals scales with overall body size according to  $M_{\text{fat}} = f_0 M^\gamma$  and assuming that once this mass is fully digested the organism starves, this would imply that  $\epsilon_\sigma = 1 - f_0 M^\gamma/M$ . It follows that the recovery timescale,  $t_\rho$ , is the time to go from  $m = \epsilon_\sigma \epsilon_\lambda M$  to  $m = \epsilon_\lambda M$  (Fig. 2). Using Eqs. (3) and (4) this timescale is given by simply considering an adjusted starting mass of  $m'_0 = \epsilon_\sigma \epsilon_\lambda M$ , in which case

$$t_\rho = \ln \left[ \frac{1 - (\epsilon_\sigma \epsilon_\lambda)^{1-\eta}}{1 - \epsilon_\lambda^{1-\eta}} \right] \frac{M^{1-\eta}}{a' (1-\eta)} \quad (5)$$

where  $a' = B_0/E'_m$  accounts for possible deviations in the biosynthetic energetics during recovery (see Supplementary Information). It should be noted that more complicated ontogenetic models explicitly handle storage<sup>54</sup>, whereas this feature is implicitly covered by the body fat scaling in our framework.

To determine the starvation rate,  $\sigma$ , we are interested in the time required for an organism to go from a mature adult that reproduces at rate  $\lambda$ , to a reduced-mass hungry state where reproduction is impossible. For starving individuals we assume that an organism must meet its maintenance requirements by using the digestion of existing mass as the sole energy source. This assumption implies the following simple metabolic balance  $\dot{m}E'_m = -B_m m$  or  $\dot{m} = -a' m/M^{1-\eta}$  where  $E'_m$  is the amount of energy stored in a unit of existing body mass, which differs from  $E_m$ , the energy required to synthesize a unit of biomass<sup>54</sup>. Given the replete mass,  $M$ , of an organism, the above energy balance prescribes the mass trajectory of a non-consuming organism:  $m(t) = M e^{-a't/M^{1-\eta}}$ . The timescale for starvation is given by the time it takes  $m(t)$  to reach  $\epsilon_\sigma M$ , which gives

$$t_\sigma = -\frac{M^{1-\eta}}{a'} \ln(\epsilon_\sigma). \quad (6)$$

The starvation rate is then  $\sigma = 1/t_\sigma$ , which scales with replete-state mass as  $1/M^{1-\eta} \ln(1 - f_0 M^\gamma/M)$ . An important feature is that  $\sigma$  does not have a simple scaling dependence on  $\lambda$ , which is important for the dynamics that we later discuss.

The time to death should follow a similar relation, but defined by a lower fraction of replete-state mass,  $m_\mu = \epsilon_\mu M$  where  $\epsilon_\mu < \epsilon_\sigma$ . Suppose, for example, that an organism dies once it has digested all fat and muscle tissues, and that muscle tissue scales with body mass according to  $M_{\text{muscle}} = u_0 M^\zeta$ . This gives  $\epsilon_\mu = 1 - (f_0 M^\gamma + u_0 M^\zeta)/M$ . Muscle mass has been shown to be roughly proportional to body mass<sup>64</sup> in mammals and thus  $\epsilon_\mu$  is merely  $\epsilon_\sigma$  minus a constant. The time to go from starvation to death is the total time to reach  $\epsilon_\mu M$  minus the time to starve, or  $t_\mu = -M^{1-\eta} \ln(\epsilon_\mu)/a' - t_\sigma$ , and  $\mu = 1/t_\mu$ .

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